SPEECH CHARACTERISTICS IN INDIVIDUALS WITH AUDITORY DYS-SYNCHRONY

Pooja
Register No.: 07AUD012

A Dissertation Submitted as Part of Fulfillment of
Final year M.Sc (Audiology)
University of Mysore, Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING
MANASAGANGOTHRI, MYSORE - 570 006
May 2009

CERTIFICATE

This is to certify that this dissertation entitled ***'Speech characteristics in individuals with auditory dys-synchrony'*** is the bonafide work submitted as part of fulfillment for the degree of Master of Science (Audiology) of the student with registration no. 07AUD012. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Date: 25-May-2009

Place: Mysore

Dr. Vijayalakshmi Basavaraj
Director
All India Institute of Speech and Hearing
Manasagangothri,
Mysore-570006

CERTIFICATE

This is to certify that the dissertation entitled ***'Speech characteristics in individuals with auditory dys-synchrony'*** has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any Diploma or Degree.

Guide

Place: Mysore

Date: 25-May-2009

Mr. Sandeep. M.
Lecturer in Audiology
Department of Audiology
All India Institute of Speech and Hearing
Manasagangothri
Mysore -570006

DECLARATION

This is to certify that this dissertation entitled ***'Speech characteristics in individuals with auditory dys-synchrony'*** is the result of my own study under the guidance of Mr. Sandeep. M, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted in any other University for the award of any Diploma or Degree.

Place: Mysore

Date: 25-May-2009 Register No. 07AUD012

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Chapter 1

INTRODUCTION

Perception is a delicate chain of events including conversion of a sensory stimulus into electrical signals at the receptor level, transmission of the electrical signals via the peripheral nerve, and processing and interpretation of the electrical signal in the CNS. Any breakdown in the process could have significant consequences in perception. In audition, perceptual consequences of both peripheral and central auditory disorders have been studied extensively. For example, peripheral damage in the inner ear and the auditory nerve leads to threshold elevation, abnormal loudness, pitch, and temporal processing (Buss, Labadie, Brown, Gross, Grose, & Pillsbury, 1998; Formby 1986; Moore 1996; Moore & Oxenham 1998; Nienhuys & Clark 1978; Oxenham & Bacon 2003; Prosen, Moody, Stebbins, & Hawkins, 1981; Ryan & Dallos 1975) whereas, central disorders produce complex processing deficits in speech and sound object recognition (Cacace & McFarland 1998; Gordon-Salant & Fitzgibbons 1999; Levine et al. 1993; Wright, Lombardino, King, Puranik, Leonard, & Merzenich, 1997).

The perceptual consequences of a recently defined hearing disorder that preserves the outer hair cell function but apparently disrupts auditory nerve activity was first described in one single subject and considered to involve a dysfunction of the auditory nerve (Starr et al. 1991). Subsequently, 10 subjects with similar symptoms were identified. Since eight of them had accompanying peripheral neuropathy, the term ‘Auditory neuropathy’ (AN) was coined (Starr et al. 1991). AN may result from a loss of inner hair cells (IHC), dysfunction of the IHC-nerve synapses, neural demyelination, axonal loss, or a possible combination of multiple sites. These pathologies may be mixed with the traditional cochlear loss involving outer hair cells and/or central processing disorders involving the brainstem and cortex, complicating the classification of AN (Rapin & Gravel, 2003). Because one possible neural mechanism underlying AN symptoms is desynchronized discharges in the auditory nerve fibers, it has been suggested that AN be termed as 'Auditory dys-synchrony' (Berlin, Morlet, & Hood, 2003). Therefore preferred terms are 'Auditory dys-synchrony' (AD) (Berlin, Li, Hood, Morlet, Rose, & Brashears, 2002), Auditory de-synchrony' or 'Auditory mismatch' as they attempt to merely describe, or reflect better what is happening in the auditory system, without ascribing a specific locus of pathology. Whereas, the term AD would include both true AN (i.e., a true neural abnormality) and other possible underlying mechanisms resulting in neural dys- synchrony, as well as delayed maturation of the lower level auditory pathway.

Auditory dys-synchrony is a clinical syndrome characterized by the presence of otoacoustic emissions and/or cochlear microphonics suggesting normal outer hair cell function in conjunction with absent or grossly abnormal auditory brain stem responses (Starr, Picton, Sininger, Hood, & Berlin, 1996).

The exact site of lesion and pathophysiology of AD is not yet completely understood. These individuals have preserved cochlear amplification, but disturbed normal synchronous activity of the auditory nerve. Discharges of the auditory nerve are presumed to be asynchronous as is evident from normal otoacoustic emissions in the presence of absent or abnormal auditory brainstem responses. These individuals typically have speech recognition deficits that are not in consonance with their pure tone hearing thresholds. They usually do not benefit from conventional amplification.

Poor speech perception abilities in these patients are attributed to abnormal temporal coding and asynchrony (Kraus et al, 2000; Rance, McKay, & Grayden, 2004; Zeng Oba, Garde, Sininger, & Starr, 1999; Zeng, Kong, Michalewski, & Starr, 2004). Etiologies of AD are just beginning to be appreciated and appear diverse. It has often appeared in clinical reports that neonates, at risk for hyperbilirubinemia and anoxia seem to be at risk for AD as well (Rance et al 1999). Genetic factors have also been identified. Starr et al (2003) reported a novel mutation in MPZ gene in a family with hereditary motor sensory neuropathy and deafness.

Need for the Study

Disruptions in the perception of temporal cues have been demonstrated in children as well as adults with AD (Kraus et al., 2000; Michalewski, Starr, Nguyen, Kong, & Zeng, 2005; Rance, et al., 2004; Starr et al., 1991; Zeng et al., 1999; Zeng, Kong, Michalewski, & Starr, 2005). In addition to distortion of the spectral information that is seen in cochlear hearing impaired individuals (Moore, 1995; Rance et al., 2004), individuals with AD possess distortion in temporal information (Rance et al., 2004; Zeng et al., 1999; 2005; Kraus et al., 2000). Hence the input signal in the auditory system is lot more distorted in individuals with AD compared to those with cochlear pathologies. This is supported by the findings of earlier studies who have recorded speech perception in individuals with AD (Ajith & Jayaram, 2006; Rance et al., 2004; Starr et al., 1996; Starr, Sininger, & Pratt, 2000; Zeng, Oba, & Starr, 2001; Zeng & Liu, 2006). Speech intelligibility problems reported in consequence of AD are often out of proportion with their behavioral audiograms (Starr et al., 1996; Starr et al., 2000; Zeng et al., 2001; Zeng & Liu, 2006).

The present study attempts to understand the production characteristics in long term AD. It is based on the hypothesis that production characteristics would deviate over time according to the degree and type of perceptual characteristics. Individual with perceptual deficits shall be using correction strategies in their production that helps them monitor their own speech. Production characteristics of individuals with cochlear hearing loss have been well established. However, AD differs from this group in the degree and type of perceptual deficits as well as its pathophysiology. Hence, there is a need to study characteristics of speech production of adults with AD.

Objectives of the Study

The objectives of the present study were:

1. To characterize the speech production of adults with long-term AD through perceptual analysis.
2. To characterize the speech production of adults with long-term AD through acoustic analysis.

Chapter 2

REVIEW OF LITERATURE

Auditory dys-synchrony (AD) is a clinical syndrome in which outer hair cell function is spared, but afferent neural transmission is disordered (Starr, et al., 1996) A typical person with AN has the following profile: elevated thresholds on pure tone audiogram by air and bone conduction, very poor speech discrimination for degree of loss, no acoustic reflex in any configuration for any stimuli, no auditory brainstem response (ABR), and presents robust Otoacoustic emissions (OAEs).

One of the first reports on AD by Starr et al. (1991) was a single case study. An 11 year old had an absence of sensory components of auditory evoked potentials (brainstem, middle and long latency) to click and tone burst stimuli that she could hear clearly. Psychoacoustic test revealed a marked impairment of those auditory perceptions dependent on temporal cues; lateralization of binaural clicks, change of binaural masked threshold with changes in signal phase, binaural beats, detection of paired monaural clicks, monaural detection of a silent gap in sound, and monaural threshold elevation of short duration tones. Pure tone audiometry showed a moderate (50 dB) bilateral hearing loss with a disproportionate severe loss of word intelligibility. Those auditory evoked potentials that were preserved included cochlear microphonics, and long latency cognitive components. Both the evoked potential and perceptual deficits were attributed to changes in temporal encoding of acoustic signals occurring at the synapse between hair cells and eighth nerve dendrites.

Incidence and Prevalence of AD

Davis and Hirsh (1979) reported that 1 in 200 hearing impaired children exhibit an audiological picture that is consistent with the contemporary diagnosis of AD. Rance et al., (1999) and Tang, Mcpherson, Yuen, Wong, and Lee (2004) reported that the overall incidence rate varies from 11% to 1.83% of the hearing impaired population. Ajith (2006) estimated the prevalence of AD in Mysore district. The prevalence of AD was around 1 in 183 (0.54%) among individuals with sensoryneural hearing loss.

Approximately 1 to 3 infants per 10,000 births are reported to have AD (Dolphin, 2004). This rate increased to 0.94% in the group at risk for hearing loss (Foerst, Beutner, Lang-Roth, Huttenbrink, Wedel, & Walger, 2006). Typically, AD is bilateral (96%) and shows no gender preference (Sininger & Starr, 2001). The female to male ratio of AD was reported to be 2:1 (Ajith & Jayaram, 2006).

Etiology of AD

The following etiologies have been attributed to AD. Medical conditions encompassed by the AD umbrella are:

- Anoxia
- Hyperbilurubinemia
- Infectious processes (Ex. Mumps)
- Immune disorders (ex: Guiliar-prarre syndrome)
- Genetic & Syndromal
- Neurological disorders (Ex. Fried Reich's ataxia)

- Perinatal diseases
- Hyperbilirubinemia
- Hypoxic insults
- Ischemic insults
- Prematurity

Neurological disorders

- Demyelinating diseases
- Hydrocephalus
- Immune disorders (Gullian-Barre Syndrome)
- Inflammatory neuropathies
- Severe developmental delay

Genetic and Hereditary Etiologies

- Family history
- Connexin mutations
- Otoferlin (OTOF) gene
- Wardenberg's syndrome
- Neurodegenerative diseases (Freidreich's ataxia)
- Leber's hereditary optic neuropathy
- Hereditary motor sensory neuropathies (Charcot-Marie-Tooth syndrome)
- Delayed visual maturation

Pathophysiology of AD

In the auditory system, neurons can generate action potentials synchronized to stimulus frequency up to several thousands of Hertz (Johnson, 1980), and preserve the relative timing of these action potentials even after passing several synaptic stages (Trussell, 1999). Synchronization of neural discharges carries important information for perception (Barinaga, 1998; Riehle, Grun, Diesmann, & Aertsen, 1997; Stopfer, Bhagavan, Smith, & Laurent, 1997). The synchronous activities in auditory neurons may encode basic auditory percepts such as loudness and pitch (Srulovicz & Goldstein, 1983; Zeng & Shannon, 1994), and extract complex sound features such as spectral peaks and waveform envelopes for speech perception (Joris & Yin, 1992; Shannon, Zeng, Wygonski, Kamath, & Ekelid, 1995; Young & Sachs, 1979).

AD may affect the functioning of inner hair cells, synaptic junctions between the inner hair cells and auditory nerve, or the auditory nerve itself (Starr et al., 1996). In a histopathological study of cochlea and auditory nerve in an individual with AD, Starr et al. (2003) found that the organ of corti was normal throughout the cochlea except at the apical turn, where about 30% loss of outer hair cells was observed. The inner hair cells were normal throughout the length of the cochlea. However, there was a profound loss of ganglion cells (>95%). Furthermore, the myelin sheath on the surviving auditory nerve fibers was thin indicating incomplete remyelinization attributed the reduced neural input due to axonal loss to be the reason for loss of acoustic reflexes, that is, middle ear muscle and olivocochlear reflexes (Starr, Picton & Kim, 2001).

Persons with AD may also manifest asynchronous firing of the auditory nerve fibers due to demyelinization (Starr et al., 2001). Demyelinization affects salutatory conduction and thereby slows down the conduction velocity of the nerve fibers. If the extent of slowing varies from one fiber to next (due to different degrees of demyelinization), then it leads to temporal asynchrony in the firing of the auditory nerve fibers thereby reducing the compound action potential of the auditory nerve.

Asynchronization not only affects ABRs, but also influences auditory perception that depends on temporal cues (Kraus et al., 2000; Rance, et al., 2004; Starr et al., 1991; Starr et al., 1996: Zeng, et al., 1999; Zeng, et al., 2005). Axonal loss and demyelinization can occur together.

Therefore, it has been hypothesized that lesions in the inner hair cells, the synapse between the inner hair cell and the auditory nerve, and the auditory nerve itself may account for the clinical findings (Berlin, Hood, Morlet, Rose, & Brashears, 2003; Berlin, Morlet, & Hood, 2003; Fuchs, Glowatzki, & Moser, 2003; Starr et al., 1996).

Audiological Profile of AD

Patients with the physiologic characteristics that have been broadly categorized as AD can present with a range of clinical symptoms. The variability in the clinical features seen in this group may represent differing degrees of the same pathology or may be the result of a range of distinct auditory pathway disorders.

The general audiological findings in these patients suggest that responses which require intact auditory nerve or brainstem pathways like the acoustic reflex, auditory brainstem response (ABRs), masking level difference and efferent suppression of otoacoustic emissions (OAEs) are abnormal. As said earlier, ABRs are generally absent, but when present, are severely abnormal. The extent of abnormality is disproportionate to the subject’s audiometric thresholds for puretones (Starr et al., 1996). On the other hand, cochlear responses like OAEs and cochlear microphonics that are a result of normally functioning outer hair cells are normal (Berlin, 1999; Santarelli & Arslan, 2002; Starr et al, 1996).

Audiograms

Patients with AD present with all degrees of hearing loss from minimal to profound. The majority of patients (82%) had symmetric losses, however, 14% had bilateral asymmetric losses and 4% were unilateral. Overall 43% of patient show a flat audiometric shape, and 28% have a reverse sloping loss with higher thresholds for low-frequency stimuli than for high frequencies. The reverse slope configuration of hearing loss provides evidence that the underlying etiology of the hearing loss in AD is neural rather than cochlear. The laws of basilar membrane mechanics do not provide a viable explanation for significant loss of low frequency sensitivity in light of much better high- frequency hearing. (Sininger & Oba, 2001).

Change in Hearing over Time

Hearing loss progression in patients with AD has a different pattern than seen with sensory loss. 29% of patients with AD show more than 10 dB pure tone average fluctuation in hearing level over a series of hearing tests whereas 14% showed progressive hearing loss. Many patients with AD appear to have moment to moment fluctuations in hearing that can create the illusion of lack of cooperation or even malingering. This inconsistency in neural representations of signals may be particularly disruptive to auditory learning and speech perception in an infant or young child with AD.

Speech Discrimination / Recognition

Patients with AD have dysfunction of speech perception that is out of proportion with their pure tone loss (Sininger Hood, Starr, Berlin, & Picton, 1995; Starr et al. 1996). Speech perception further deteriorates in adverse listening conditions like ipsilateral noise (Zeng & Liu, 2006). Similar findings have been reported by in school going children with AD (Rance, Barker, Mok, Dowell, Rincon, & Garratt, 2007)

Acoustic Reflexes

Most patients with AD have no brain-stem auditory reflexes including acoustic (middle ear muscle) and Olivocochlear reflex. About of AD 93.5% showed absent reflexes and 6.5% showed elevated reflexes in a study by Sininger and Oba, 2001.

Otoacoustic Emissions and Cochlear Microphonics

In a sample, 80% of the patients with AD have a clear OAEs. Only 9% have never shown an OAE during evaluation, and in 11% the OAE disappeared over time (Sininger & Oba, 2001). Others have reported that patients with AD may have but later lose OAEs over time (Deltenre et al. 1999). The reason for loss of OAEs over time is unclear. In general studies reported that OAEs are stronger in patients with AD than that in normals (Nortons & Widen, 1990).

Cochlear microphonics that are robust and are present for several milliseconds after a transient click, have been recorded from individuals with AD (Berlin, 1999; Deltenre, et al., 1999; Santarelli & Arslan, 2002; Starr, et al., 1996; Starr et al., 2000). Berlin (1999) reported that in approximately 50% of the individuals (N=33) with AD, the amplitude of cochlear microphonics increased compared to those with normal hearing. It was speculated that this finding of increased cochlear microphonics in patients with AD reflected specific outer hair cell changes that were secondary to alterations of the auditory nerve input. Sininger and Oba (2001) also could record cochlear microphonic in cases with AN, even though OAEs were elevated.

The presence of cochlear microphonics, measured through ECochG, has been considered indicative of at least some degree of outer hair cell function and therefore considered suggestive of neural transmission abnormality in ears with absent or disrupted brainstem potentials (Berlin, Hood, Cecola, Jacson & Szabo, 1993; Berlin et al., 1998; Chisin, Pearman, & Sohmer, 1979; Starr et al., 1991; Starr et al. 1996). The presence of a clear cochlear microphonic can supplant the presence of OAEs, evidence of normal hair cell function.

Similarly, Duan and Wang (2002) reported that summating potential was present patients with AD whom they tested and its amplitudes were significantly larger than those of normal subjects. These results suggest that at least in this group individuals with presence of summating potential, the lesion may be localized in the retro-outer hair cell region.

Auditory Brainstem Response

Sininger and Oba (2001) reported that out of 59 patients, 70% had complete absence of auditory brainstem response (ABR) regardless of the level of the stimulus. In their study, 19% showed wave V only, which was poorly defined with abnormal latency while 6% had abnormal ABR but Wave III & V present. These patients with absent ABR showed the poorest pure tone average thresholds and those with several peaks in the waveform (called abnormal ABR) had the best thresholds. In all cases of

AD, the threshold of the ABR was unrelated to the hearing threshold. ABR cannot be used to estimate hearing thresholds in a patient with AN.

Auditory Middle and Late Latency Responses

Berlin et al., (2005) reported absent middle latency response (MLRs) and present N1-P2 responses in a patient with AD related to Charcot-Marie-Tooth disease. Starr et al (1996) found MLRs in one of six patients but observed none on later evaluation of this patient. Late latency response (N1-P2 and P300) were observed in one of six patients, and abnormal N1-P2 responses in another patient aged 15-49 years. However, responses were reported as small as and often slightly later than normal. Kumar & Vanaja (2008) also reported recordable LLRs in individuals with auditory neuropathy and reported a reasonably good relation between amplitude of LLR and speech identification scores.

Although there are studies that report abnormality in TEN (Vinay & Moore, 2007), psychophysical tuning curves, Auditory Steady State Response (Toca & López, 2005) and efferent suppression (Abdala, Sininger, & Starr, 2000; Lalaki, 2003) in individuals with AD, they are not of relevance to the present study and hence are not discussed in detail.

Deficits in Psychophysical Perception in Patients with AD and their Implication to Speech Perception

Intensity Processing

Zeng et al. (2001) analyzed the loudness growth function in one subject with AD using magnitude estimation and loudness scaling techniques. Results showed that the subject demonstrated a larger compressive loudness function than the normal control subject. In another study, Rance et al. (2004) demonstrated that persons with AD show slightly larger difference limens at low sensation levels than normals, but it approached normal values at high sensation levels. Because subjects with AD often have problems in speech recognition even if speech is presented at high levels, this suggested that intensity processing is not a major factor contributing to their speech discrimination.

Frequency Processing

Frequency discrimination ability of patients with AD is significantly poorer compared to that of normal hearing subjects, particularly at low frequencies (Rance et al., 2004; Starr et al., 1991; Starr et al., 1996; Zeng et al., 2005). Rance, et al. measured the frequency difference limen and frequency modulation detection limen for 500 Hz and 4 kHz in children with AD. Results showed that both the difference limens were better at 4 kHz compared to those at 500 Hz in individuals with AD. Similar results were reported by Zeng et al. (2005). Furthermore, frequency difference limens were better compared to frequency modulated difference limen scores. Also, frequency discrimination abilities were strongly correlated with speech perception scores at low frequencies. Studies had shown that the greater the loss at low frequencies, the greater is the severity of temporal asynchrony which in turn reduces speech perception abilities. The poorer frequency discrimination in the middle frequency region (1000-3000 Hz) may pose some problem for discerning the second formant frequencies of two spectrally closed spaced vowels but should not prevent subjects with AD from distinguishing other speech sounds, such as fricatives, in which spectral cues are not as fine as the vowels.

Temporal Processing

Several investigators have explored the temporal processing abilities of individuals with AD. Zeng et al. (2005) evaluated several time-related functions like temporal integration, gap detection, temporal modulation detection, and masking (backward, forward and simultaneous) in individuals with AD. They found improvement in thresholds with increase in signal duration in individuals with AD as is the case with normals. However, the slope of the integration function was slightly elevated in individuals with AD (-9 dB per doubling of duration) than in normal hearing subjects (-3 dB per doubling of the duration). Similar results were reported by Starr et al. (1991).

Zeng et al. (2005) reported poor gap detection thresholds in individuals with AD. Normal hearing individuals required a silent interval of around 50 ms to detect a gap at 5 dB SL. However, the detection threshold improved to 3 ms at higher sensation levels (30 to 40 dB SL). Individuals with AD performed similar to normal hearing subjects at low sensation levels, but unlike normals, required significantly larger gap to detect at higher sensation levels. This defies any explanation. However, Zeng et al. (1999, 2005) gave a phenomenological model to explain the abnormal gap detection thresholds in individuals with AD. This model assumes that the main effect of dys-synchronous activity is a smeared temporal representation of the acoustic stimulus (Figure 2.1). The sharp temporal changes in the physical representation of the stimulus are lost in the internal neural representation due to smearing of the waveform. If the listening task is to merely detect the sound, then this smeared representation will not affect perception. However, if the task is to discriminate between sounds - one with a gap and one without; then the smearing of the internal representation makes the task more difficult.

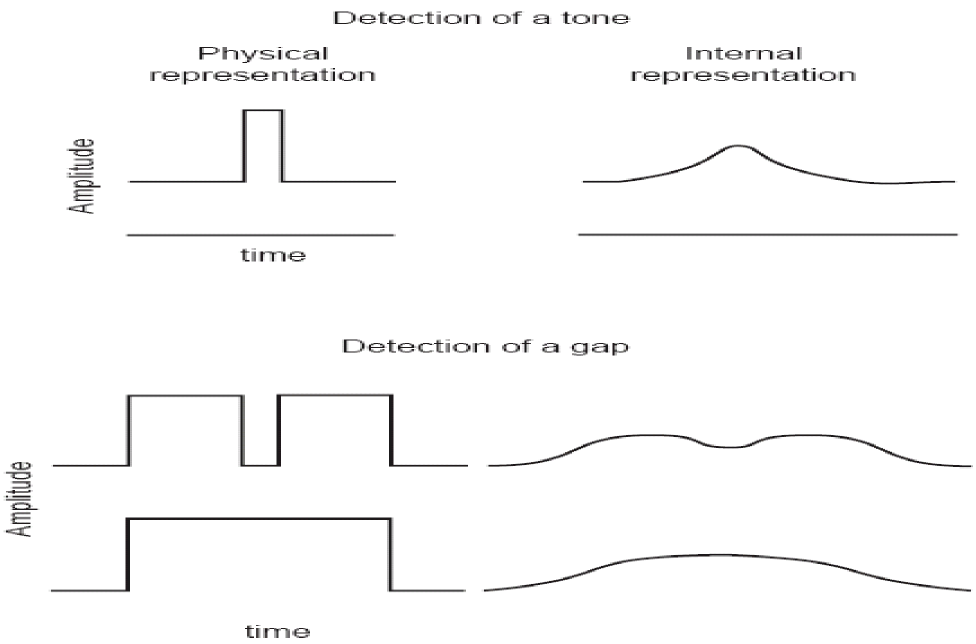

Figure 2.1: A phenomenological model of AD (Zeng et al., 1999).

Smearing of the temporal envelope does not affect the detection of a tone (top panel) because this task requires an all-or-none decision. However, smearing causes major problem in gap detection (bottom panel) as the task requires finer discrimination of two different waveforms.

Difficulty in detecting short duration acoustic events (as shown by psychoacoustic experiments on gap detection and temporal integration) may pose problems to persons with AD in processing brief, but critical events of speech. It is known that many critical elements for speech perception such as transition, burst and VOT last only up to a few milliseconds. The studies of Zeng et al. (1999, 2001, & 2005) have amply demonstrated that individuals with AD have severe problems in processing silent intervals of even 20-30 ms as compared to normals and individuals with cochlear hearing loss. Also, it has been shown that thresholds for short duration signals are significantly higher in individuals with AD than in normals or individuals with cochlear hearing loss. Consonants which contribute predominantly to the intelligibility of speech are cued by dynamic cues like burst and transition. If the processing of these cues is abnormal in individuals with AD, speech identification is expected to deteriorate.

Temporal Modulation Transfer Function in AD

Another temporal process that has been reported to be abnormal in individuals with AD is the temporal modulation transfer function (Ajith & Jayaram, 2005; Rance et al., 2004; Zeng et al., 1999; Zeng et al., 2005). Temporal modulation transfer function is a measure of sensitivity to amplitude fluctuation over a range of modulation frequencies. This measures one's ability to perceive changes in stimuli over time. Ajith & Jayaram, (2005) reported that subjects with AD were most sensitive to slow temporal fluctuation and became less sensitive as the fluctuation rate was increased. At higher modulation frequencies 12 subjects did not even detect a modulation of depth of 0 dB (100%). This showed that all subjects with AD had severe temporal processing deficits. The result of reduced modulation may be implied to poor speech perception. Speech, particularly in the presence of noise is perceived based on their temporal modulations. If modulation sensitivity is reduced, it should evident as poor speech identification, atleast in the presence of noise. Rance et al. (2004) also reported significant differences in modulation detection thresholds between individual with AD who have good and poor speech perception scores. Individuals with AD with speech identification scores less than 30% had poorer modulation detection thresholds compared to subjects who had more than 30% speech identification score.

Effect of Masking in AD

Kraus et al., (2000) reported exaggerated masking effect in one patient with AD who had near normal hearing thresholds. Temporal masking and simultaneous masking paradigms have shown that individuals with AD have difficulty in separating sounds that occur successively as well as in detecting signal in noise (Zeng et al., 2005). In forward masking, individuals with AD showed 60% masking even when signal and masker were separated by as much as 100 ms while normal controls showed only 15% masking at a signal delay of <20ms. This shows that individuals with AD have difficulty in separating sounds that occur in close succession. This may produce difficulty in perceiving voice onset time, burst or transition (that occurs in close succession) as discrete components and may probably be smeared in turn affecting the speech perception.

Speech Perception in AD

Deficits in speech perception is the prime feature of AD. Speech perception ability in adults diagnosed with AD is shown to have no correlation with the pure-tone audiogram (Starr et al., 2000; Zeng et al., 2001). Starr et al., (1996) presented open-set speech perception findings in 8 of their 10 subjects with AD. Word recognition scores ranged from 0% to 92% and were significantly lower in 12 of the 16 ears than that predicted from the norms generated by Yellin, Jerger, and Fifer (1989) for ears with sensorineural hearing loss. Similarly, Sininger and Oba (2001) reported speech discrimination scores (CID W-22 lists) for 36 subjects with AD. Results showed that 25 (69%) of them fell below the normative range given by Yellin et al. (1989). Similar results have been reported by other studies (Berlin et al., 1993; Berlin, Hood, Hurley, & Wen, 1996; Jerger, Ali, & Fong, 1992; Sininger et al., 1995; Starr et al., 2000; Starr et al., 2003).

Speech perception difficulties in AD are reported to be more serious in adverse listening conditions. Shallop (2002) reported a case of a woman diagnosed with hearing thresholds in the mild-to-moderate range when in her late 20s, but who had reported difficulties in noise throughout childhood. Hearing in Noise Test (HINT) sentence testing in this case also showed 100% perception in quiet listening conditions but extreme difficulty in noise. Word identification for this subject fell to 25% at a +15 dB signal-to-noise ratio and to 0% at +12 dB. This was due to the compromised redundancy in the presence of noise. Although speech perception difficulties in background noise are not unique to AD, effects of noise in AD tend to be extreme. Zeng and Liu (2006) studied in detail the perception of 14 (mostly adult) subjects and found consistent reductions in speech recognition ability, even at signal-to-noise ratios that show little or no effect on subjects with normal hearing (+10 to +15 dB).

These speech perception abilities in individuals with AD are related to temporal processing abilities (Rance et al., 2004; Zeng et al. 1999; Zeng, et al., 2005). Kraus et al. (2000) systematically examined fine-grained speech perception abilities in an adult with AD. They measured just noticeable differences (JNDs) for three CV continua: /ba-wa/, /da-ga/ and another /da-ga/ continuum in which amplitude of formant transitions had been enhanced. Results showed that on /ba-wa/ continuum, the JND of the patient with AD was comparable to that of normal hearing subjects. However, JND for /da-ga/ continuum in auditory dys-synchronics was poorer than in normal hearing adults. Normal hearing adults could discriminate between stimuli in which the onset frequency of the 3rd formant frequency differed by 80 Hz in the two stimuli, while individual with AD required a difference of almost 120 Hz between the stimuli. When the amplitude of formant transition was enhanced in /da-ga/ continuum relative to vowel segment, the subject with AD showed even greater difficulty in discriminating along the continuum. The data of Kraus et al. (2000) amply demonstrate that individuals with AD perform similar to normals on speech perception if the rate of change of temporal features is slow (as in /ba-wa/ continuum), but their speech processing mechanism breaks down while dealing with rapid spectro-temporal changes. Enhancement of intensity related information in the formant frequencies did not improve speech perception in the subject with AD. However, the issue is, majority of the phonemes have rapid spectro-temporal changes that act as the primary cues for speech perception.

In the same subject, Kraus et al. (2000) measured word identification by manipulating three factors, namely, signal to noise ratio, lexical difficulty, and number of talkers (single vs. multiple talkers) to investigate the effect of multiple sources of variability and signal degradation on speech perception. The subject with AD showed marked effect of noise on speech identification compared to normal hearing subjects. However, on tasks where the number of talkers and lexical difficulty was varied, the performance of patient with AD was similar to that of normal hearing subjects.

Ajith (2006) studied fine grained speech perception in individuals with AD. Just noticeable differences were measured for transition duration, burst duration & VOT. The data was obtained on 14 individuals with AD (16 to 30 years with the mean age of 23 years) and compared against that from a normal group of 30 individuals. Open set speech identification scores were also obtained for both the groups. Results showed that the JNDs of subjects with AD were almost three to four times larger than those for normal hearing listeners. Stimulus- Response Matrix for Unmodified Speech Sounds was in the study also made & revealed that speech identification scores did not exceed 50%. Other observations from this matrix are as follows:

1. Individuals with AD perceived /dha/ better in relation to other speech sounds. This was closely followed by the identification of syllable /ga/.
2. No consistent grouping among phoneme categories was evident in the stimulus-response matrix. The exceptions were /ba/ which was frequently confused with its unvoiced cognate /pa/, and /dha/ which was confused with /ga/.
3. The phonemes /ta/ and /pa/ were rarely identified correctly.

Elevated JNDs for these temporal parameters of speech were accounted to lead to difficulty in discriminating speech sounds that differ in temporal aspects.

Subjects in whom cortical evoked potentials could be recorded generally are reported to show better speech perception scores (Rance, Cone-Wesson, Wunderlich, & Dowell, 2002). Cortical potentials require lesser synchrony among the nerve fibers. Hence, the presence of cortical potentials in some of these individuals probably goes to prove that synchrony is still preserved to some an extent in these individuals. Rance et al. showed that cortical evoked potentials could be elicited using both tonal and speech stimuli in children with AD. Presence or absence of cortical evoked potentials with age appropriate latency and morphology seemed to be related to open set speech identification scores and benefit derived by subjects from amplification.

The neural dys-synchrony seen in individuals with AD, apart from severely distorting the timing information, could also distort the spectral information of the incoming signal. Rance et al. (1999) reported impairment in the tonotopicity of the neural signals reaching higher centers of the auditory system which in turn can cause poor speech discrimination due to spectral distortion. These deficits may lead to difficulty in processing vowels as well as consonants.

Relation between Perception and Production

A close relationship has traditionally been assumed between speech perception and the development of speech production skills (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967). Self-hearing has an essential role to play in mapping the systematic (language-determined) relationship between the sounds and the articulatory activity produced, while hearing others may serve primarily to establish linguistic significance in terms of the meaning of the sounds (Fowler & Saltzman, 1993). Likewise, maintaining intelligible speech on long-term requires normal feedback of one's speech through the auditory mode. Earlier studies have shown that the hearing loss in the early years of life can negatively influence the development of speech and language (Culbertson & Kricos, 2002; Dunn & Newton, 1986; Hudgins & Numbers, 1942; Smith, 1982). This could be in terms of delayed or deviance language and defective speech in terms of articulation, voice and fluency. These speech production deficits are attributed to the defective auditory feedback secondary to hearing loss (Binnie, Daniloff, & Buckingham, 1982; Cowie, Douglas-Cowie, & Kerr, 1982; Elman, 1981; Kirchner & Suzuki, 1968; Penn, 1955; Ramsden, 1981, Zimmermann & Rettaliata, 1981).

There are different articulatory errors reported as typical errors in individuals with cochlear hearing loss. Hudgins and Numbers (1942) reported the main speech production errors in hearing-impaired children as deletion of initial and final consonants, consonant cluster errors, voicing and nasality errors, consonant substitutions, and vowel distortions. Many similar studies agreed on the complex and varied nature of speech errors present in hearing-impaired speech (Culbertson & Kricos, 2002; Dunn & Newton, 1986; Smith, 1982). There is a well-documented relationship between the severity of hearing loss and intelligibility of speech (Boothroyd, 1984; Perkell, Matthies, & Lane, 1997; Smith, 1982). That is as the severity of the hearing loss increases, speech intelligibility reduces.

The relationship between hearing ability and speech intelligibility supports the acoustic theory of speech production (Kuhl, 1981; Stevens, 2002), which claims that the acoustic patterns of a speech signal are processed and organized into an internal map that can be distorted if the acoustic patterns have not been adequately received during the input process. With a compromised input process, such as that associated with a hearing loss, the incorrect mapping will result in distorted or deleted speech sounds in speech production (Stevens, 2002). For instance, children with mild-moderate losses tend to develop intelligible speech, but have production errors mainly involving affricates, fricatives and blends (Elfenbein, Hardin-Jones & Davis, 1994). In contrast, children with severe to profound hearing losses have significantly reduced intelligibility due to difficulty with consonant, vowel and diphthong production, as well abnormal voice production (Culbertson & Kricos, 2002).

Furthemore, Dunn and Newton (1986) described the speech of people with severe to profound hearing impairments as including suprasegmental errors also along with the segmental errors. Suprasegmental errors typically seen were slow speech rate, slow articulatory transitions, poor breath control, inappropriate stress patterns, and poor resonance.

Although the effects of hearing loss are more serious in the early years of life, many perceptual studies have reported that long-term auditory deprivation even in adventitiously deaf results in flat, unmodulated, and dysprosodic voice with segmental speech deterioration (Binnie et al, 1982; Cowie et al, 1982; Elman, 1981; Kirchner & Suzuki, 1968; Penn, 1955; Ramsden, 1981). Ramsden (1981) reported that speech of the adventitiously deaf degenerated systematically over time, indicating that auditory information plays an important role in the maintenance of normal speech. Zimmermann and Rettaliata (1981) investigated in greater detail the systemic longitudinal degeneration of speech. They concluded that the adventitiously deaf speaker's speech degenerated slowly due to overlearned motor patterns, errors made without knowledge of the errors occurring, and that it takes many instances of exceeding the normal range of variability to change production. Articulatory movement patterns were less efficiently maintained over time when only nonauditory sensory systems were available. Consequently, auditory information is not used for moment-to-moment monitoring, but periodically to update and calibrate the system. Houde and Jordan, (2002) reported that compensatory changes in individual sound production can be induced over time by systematically altering auditory feedback to indicate inaccurate articulation.

Even the acoustic studies have reported significant speech deterioration considered to be a result of lack of auditory feedback. Specifically, higher speaking fundamental frequency (Leder, Spitzer, & Kirchner, 1987a), greater intensity (Leder, Spitzer, Milner, Flevaris-Philips, Kirchner, & Richardson, 1987b) and lower speaking rate (Leder, Spitzer, Kirchner, Flevaris-Philips, Milner, & Richardson, 1987c) than that of age-matched, normal-hearing subjects. Various investigators have observed that postlingually deafened adults have significantly longer sentence duration (Kirk &

Edgerton, 1983; Lane & Webster, 1991; Lane et al., 1998) which is a result of significantly longer syllables (Lane & Webster, 1991; Leder et al., 1987b), pause duration (Lane & Webster, 1991), and vowel duration (Waldstein, 1990).

In contrast to the aforementioned studies, Leder and Spitzer (1990) reported that segmental parameters are affected when there is failure to detect certain phonemes and failure to demonstrate discrimination between phonemes. These difficulties are in turn reported to manifest as articulatory errors such as substitution and distortions. Their findings didn't show widespeard articulation errors in adventitiously deaf adults. Similarly, Goehl & Kaufman (1984) reported that no clinically significant deterioration of speech sound production (i.e., segmental errors). The fact that individuals with profound postlingual hearing loss maintain highly intelligible speech suggests that mature phonemic motor patterns are quite robust, and do not depend heavily on auditory feedback. These researchers argue in favor of a predominantly open-loop speech motor control system, i.e., the speaker ‘‘knows’’ the relationship between motor commands and resulting sound output and uses this knowledge to compute the motor sequence for producing the desired speech output (Matthies, Svirsky, Perkell, & Lane, 1996).

Among the Indian studies, Grover (1998) reported that rate of speech was slower in hearing impaired individuals. Jayaradha (2001) reported that speed of transition was reduced significantly in hearing impaired compared to normals. Sluggish movements of tongue and imprecision in attaining actual articulatory target attribute as the reason for reduced Speech of transition in hearing impaired.

The changes in speech production observed in deafened adults appear to have little effect on speech intelligibility in cases where onset of deafness occurred in adulthood. In general, earlier the onset of deafness, the greater the effect of hearing loss on intelligibility (Binnie et al., 1982; Cowie et al., 1982).

Individuals with AD are proved to have more serious deficits in speech processing and perception as evident through psychophysical and perceptual studies. If individuals with long standing cochlear hearing loss who have relatively better speech identification are prone to have speech production deficits, it is logical to assume that individuals with longstanding AD should also possess speech production errors. Only one relevant report could be traced in this direction in the literature. Rance, Barker, Sarant, and Ching, (2007) reported that school aged children with AD hearing loss are developing spoken language more slowly than would be expected for children with normal hearing.

However, to the best of the information, there is no study to characterize the speech production errors in adolescent or adults with long-standing AD. The severity as well as pattern of speech perception deficits is different between cochlear hearing loss and AD. Based on the closed loop models, it could be hypothesized that speech deficits are seen even in acquired AD. Hence, production needs to be characterized in these individuals. Hence the present study was taken up.

Chapter 3

METHOD

The present study was aimed to characterize the speech production of adults with long-term AD (AD). The study used a standard group comparison research design. The study was done in the 2 phases. Phase 1 included the perceptual analysis of speech of adults with long-term AD, while the phase 2 included acoustic analysis of the same speech samples.

Subjects

Two groups of subjects were included in the study; an experimental group that had individuals with confirmed diagnosis of AD and a control group that had age matched normal hearing individuals.

Experimental Group

The study was conducted on 12 individuals (8 females & 4 males) with acquired long-term AD. Long-term AD was operationally defined as AD for more than 5 years. All the subjects had visited the department of audiology, All India Institute of Speech and Hearing, Mysore once earlier (5 years back). They were contacted through letters and were called for a follow up evaluation. This way the duration of AD was confirmed. The diagnosis of AD was based on the results of Auditory brainstem responses (absent responses) and Oto acoustic emissions (robust responses). They had showed no evidence of space occupying lesion on neurological examination. Age of the subjects in this group ranged between 17 and 30 years. Hearing loss among these subjects ranged between mild and severe degree.

Control Group

Control group had 20 age matched individuals with normal hearing sensitivity. All of them had normal Oto acoustic emissions (OAEs) as well as normal Auditory brainstem response (ABRs). Speech identification scores were normal in quiet as well as at 0 dB signal to noise ratio. They did not have any past/present history of neurologic or otologic disorders.

Subjects in both the groups were native speakers of Kannada and belonged to same geographical location (Mysore city or places within Mysore district). As reported by the parents and also as observed in the informal testing, all the subjects in the present study had normal speech and language development. Oral mechanism examination was done to rule out the presence of any structural abnormality. A written consent about willingness to participate in the study was obtained from all the subjects.

Instrumentation

Following instruments were used in the present study:

1. A calibrated two channel diagnostic audiometer (Orbiter 922) was used to estimate pure tone thresholds, speech reception threshold and speech identification scores.
2. Samsung digital voice recorder- Voice Yepp*TM VY-H350 was used to record the speech samples.

3. A computer with Pratt software was used for the acoustic analysis of speech samples.

Test Materials

Two speech samples were collected from all the subjects. The first of these was during reading of a Standardardized passage in Kannada on Bengalooru (had total of 39 words containing only voiced sounds) and is given in Appendix 1. Second was during a description of a standardized picture depicting a picnic situation (given in Appendix 2).

Recording of Speech Samples

The speech samples were recorded in a room that was sound treated as per the guidelines of ANSI (1991). The samples were recorded using a Samsung digital voice recorder- Voice Yepp*TM VY- H350 with an inbuilt unidirectional dynamic microphone that was kept at 6 inches from the speaker's mouth. The VU meter in the recorder was monitored at optimum levels during the recording. Each subject was instructed to read the standard passage and to describe the given picture. This sample was recorded into the Samsung Digital Voice Recorder- Voice Yepp*TM VY- H350. The recorded sample was then digitized and stored into the computer with Pratt software.

Test Procedure

Phase 1: Perceptual Analysis

The speech samples of the subjects were subjected to perceptual judgments. Thirteen sophisticated listeners (native speaker of Kannada) who were blindfolded to the purpose of the study were asked to independently rate the speech samples of subjects in the control and experimental groups. The randomized samples of recorded speech were played to the listeners using an audio deck in a sound treated room. The parameters that were perceptually analyzed are - voice, articulation, prosody, rate of speech & overall naturalness. The listeners categorized each of these parameters as perceptually normal or abnormal. Overall intelligibility of speech was then rated using speech intelligibility rating scale given by Markides (1986), for each of the speech samples. A score between 1 and 7 was determined for each sample to the following description:

1. Normal
2. Very easy to follow
3. Fairly easy to follow
4. Rather difficult to follow
5. Very difficult to follow
6. Unintelligible
7. Non-existive

The intra-subject reliability was checked by comparing the 2 ratings of the same listener but with a time gap of 1 month. This was done with three listeners.

Phase 2: Acoustic Analysis

The stored signal of the speech samples of each subject was analyzed to obtain the temporal parameters. Pratt software package was used for data analysis of the speech samples. Each word was analyzed for the following parameters - preceding vowel duration, following vowel duration, voice onset time, burst duration, word duration, transition duration and speed of transition. Only temporal parameters were considered in the study as individuals with AD primarily have temporal processing deficits.

The following parameters were measured:

1. *Word Duration*

Word duration is the time taken between initiation and termination of the word. It was measured directly from the speech waveform. The waveform was displayed on the computer monitor. The words were identified based upon the continuity of the waveform. The word duration was considered to extend from the beginning of the periodic signal to the end of the periodic signals. This duration was highlighted using the cursor. The highlighted portion was played back through head phones to confirm that it contained the word under study. Once this was confirmed, the duration of the highlighted portion was read from the display and considered as the duration of that particular word. Thirty-nine words from standard reading passage & nine words from picture description task were considered. The following figure 3.1 shows the duration of the representative word /dodda/.

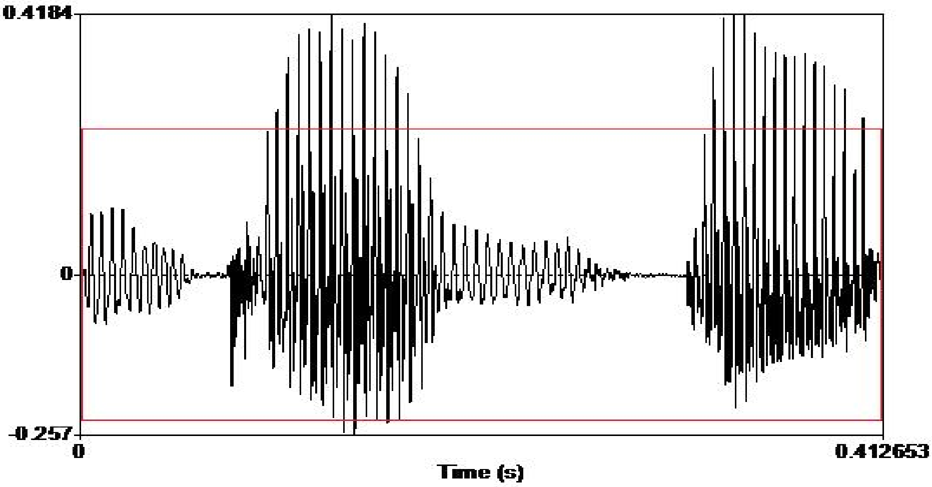

Figure 3.1: Word duration for word /dodda/

2. Vowel Duration

The vowel duration was defined as the time for which a vowel is sustained (House, 1961; Whitehead & Jones, 1978). The vowel duration was measured directly from the speech waveform and spectrogram as the time duration between the initial regular glottal vibrations to the final regular vibrations. This duration was highlighted using the cursors. The highlighted portion was played back through head phones to confirm that it contained the vowel under study. Once this was confirmed, the duration of the highlighted portion was read from the display. The unit of vowel duration was in milliseconds (ms). The preceding vowel duration (PVD) and following vowel duration (FVD) was measured in the three target words (/ondu/, /idu/ & /u:ru/). The figure 3.1 & figure 3.2 show the PVD for vowel /i/ & FVD for vowel /u/ in word /idu/ respectively. The transition duration for /d/ in the word /dodda/ is depicted in figure 3.6.

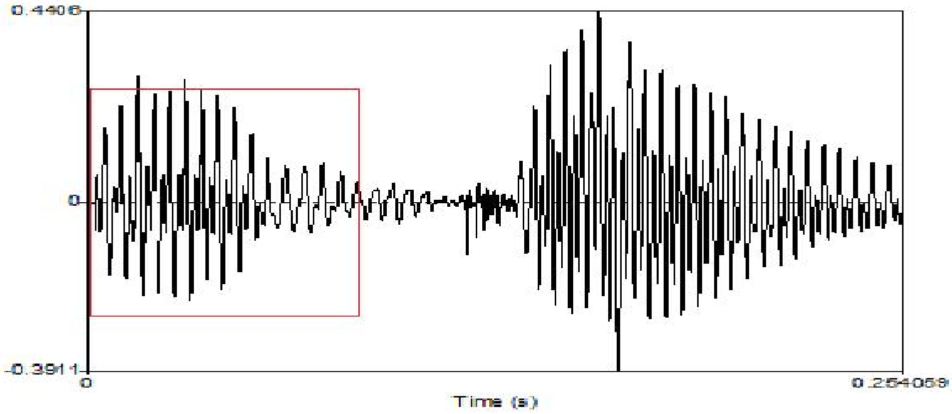

Figure 3.2: Preceding vowel duration for vowel /i/ in word /idu/

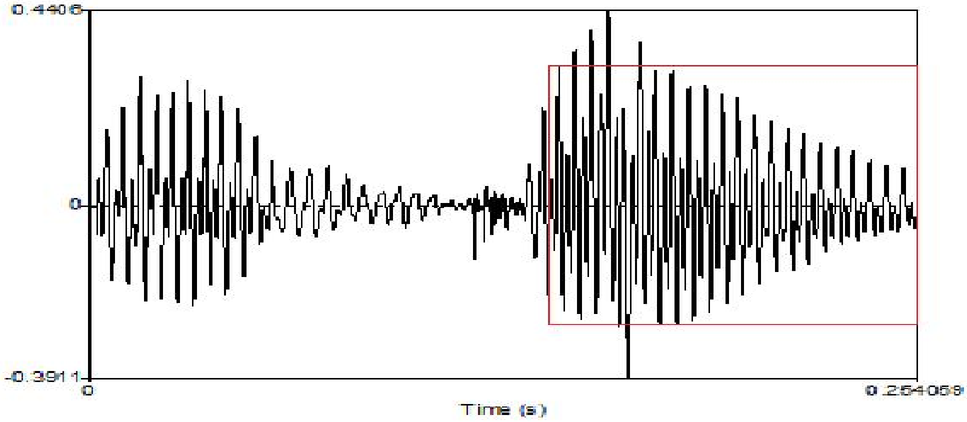

Figure 3.3: Following vowel duration for vowel /u/ in word /idu/

3. Voice Onset Time

Voice onset time was defined as the time equivalent space from the onset of the stop release burst in the first vertical striation representing glottal pulsing. The VOT was measured directly from the speech waveform and spectrogram as the first indication of energy associated with the oral release of stop and the point was marked. The cursor was moved to the point before the burst begins. This duration was highlighted using the cursor. The real time value (in ms) between these two markings provided the VOT. The VOT was measured in the eight stop consonants of the target words. The target words included were /dodda/ (2 occurences) and /be:re/ (4 occurences) from the standard reading passage; and /ga:lipəta/ and /ba:vuta/ (1 occurrence each) from the picture description. The VOT for /d/ in the word /dodda/ is depicted in figure 3.4.

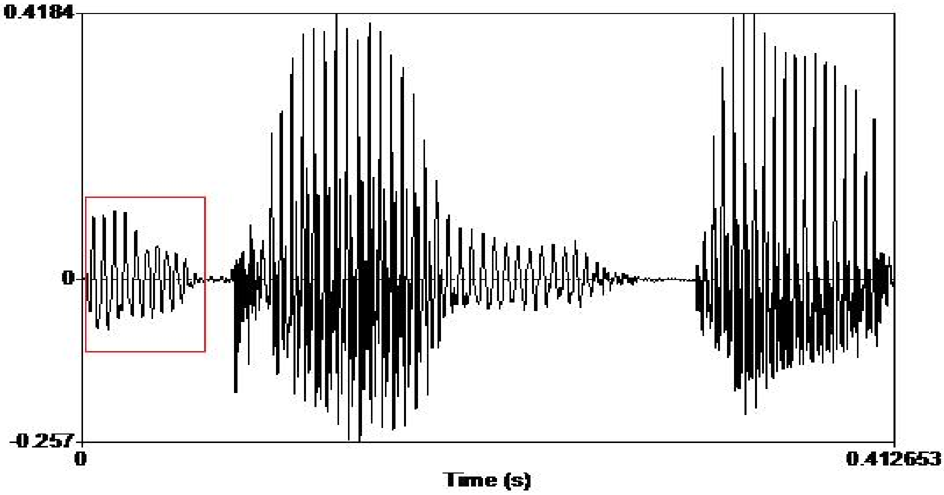

Figure 3.4: VOT for /d/ in word /dodda/

4. Burst Duration

Burst duration is the interval between the onset of the burst and the release of articulators. It is one of the shortest acoustic events that are commonly analyzed in speech. Burst duration was measured as the time interval between onset of burst & onset of second formant transition. The waveform was displayed on the computer monitor. The words were identified based upon the continuity of the waveform. The point was marked at the first indication of energy associated with the oral release of stop. The cursor was then moved to the point where regularly appearing waveform of the vowel following that stop starts. The real time value (in ms) between these two markings provided the burst duration. Burst duration was measured in the eight stop consonants of the target words which were same as that used for VOT measurement. The burst duration for /d/ in the word /dodda/ is depicted in figure 3.5.

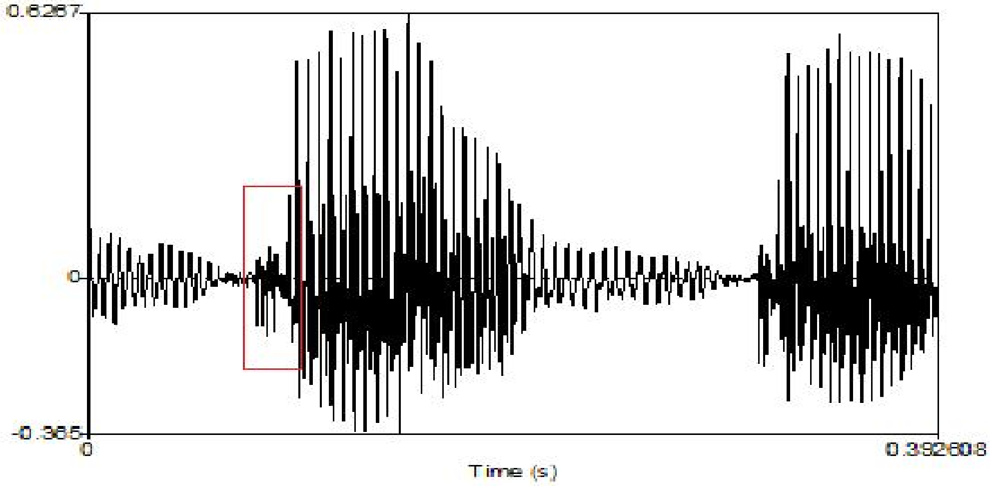

Figure 3.5: Burst duration for /d/ in word /dodda/

5. Transition Duration (TD)

Transition duration (TD) is the time taken to complete the transitional segment. TD was estimated by calculating the duration from the onset of F2 frequency till the offset of F2 frequency in milliseconds. The waveform was displayed on the computer monitor. The words were identified based upon the continuity of the waveform. The cursor was moved from the onset of F2 frequency after the burst duration till the offset of F2 frequency where regularly appearing waveform of the vowel following that stop starts. The real time value (in ms) between these two markings provided the transition duration. Transition duration was measured in the eight stop consonants of the target words which were same as that used for VOT measurement. The transition duration for /d/ in the word /dodda/ is depicted in figure 3.6.

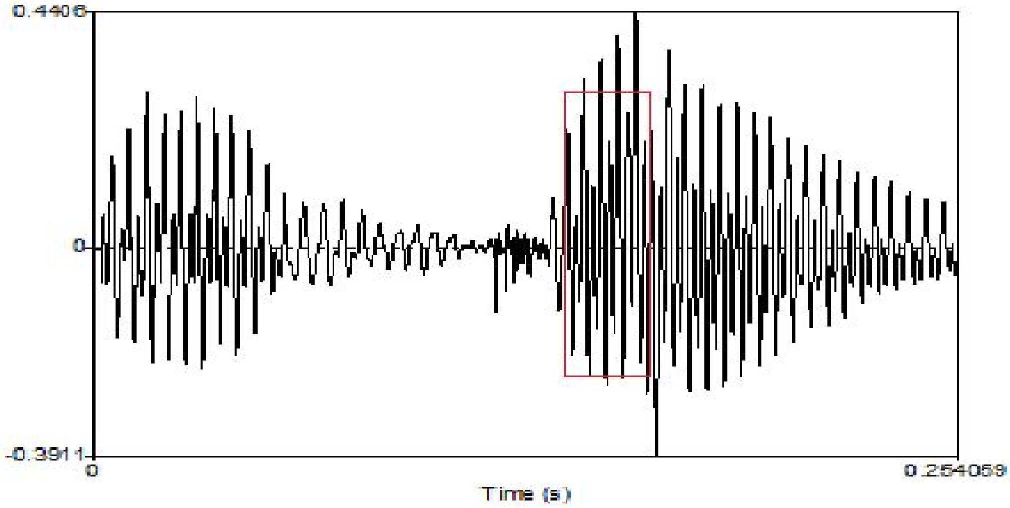

Figure 3.6: Transition duration for /d/ in word /dodda/

6. *Speed of Transition or Formant Transition Rate*

The frequency change in F2 transitions (Hz/ms) was measured to assess speed of transition (F2 transition rate). Speed of transition was estimated by calculating the difference in frequencies between the onset and offset of F2 frequency (Hz) divided by the duration of the transition (ms). This was measured for the stop consonants in the target words /dodda/, /be:re/, /ga:lipəta/ and /ba:vuta/. The speed of transition for /d/ in the word /dodda/ is depicted in figure 3.7.

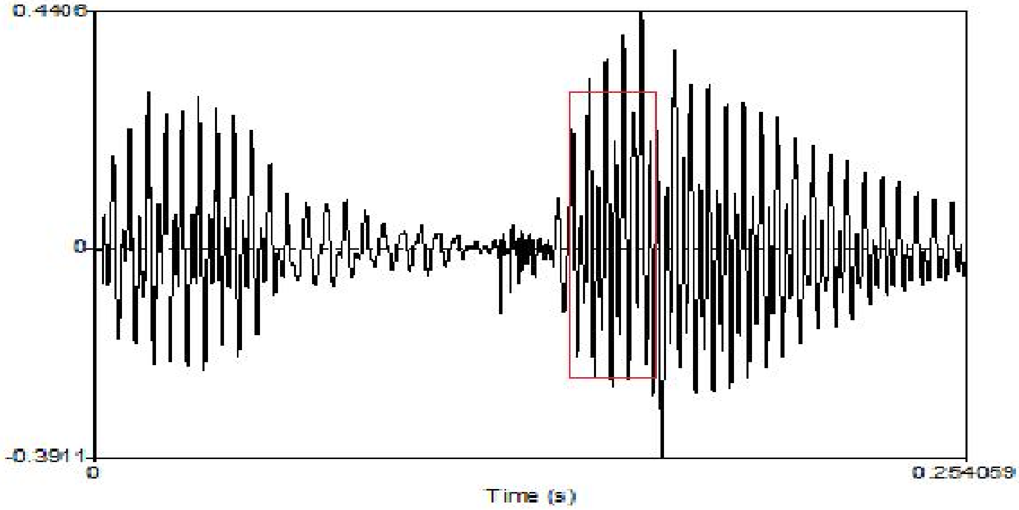

Figure 3.7: Speed of transition for /d/ in word /dodda/

Statistical Analysis

The perceptual and acoustical data thus obtained was tabulated. Descriptive statistics like mean and standard deviation of the data were obtained for all the parameters analyzed. Independent *t* test, Mann-Whitney tests and Equality of proportion were applied to check whether there were any significant differences between normal group and AD group. Majority of the statistical analysis was carried out using the Statistical Package for the Social Sciences software (version 17).

Chapter 4

RESULTS

The data obtained was subjected to the following analysis:

a) Comparison of perceptual rating assigned for speech of normal individuals and individuals with AD by 13 sophisticated listeners for speech of using Equality of proportions test.

b) Correlation between speech identification scores and overall intelligibility in individuals with AD.

c) Comparison of temporal parameters of speech (word duration, voice onset time, burst duration, transition duration and speed of transition, preceding vowel duration , following vowel duration) of normal individuals and individuals with AD, using Independent *t* test.

Phase 1: Perceptual Analysis

Thirteen judges rated the speech samples of each subject in terms of voice, articulation, prosody, rate of speech and overall naturalness. The judges were blindfolded to the purpose of the study and judged the samples independently. The samples were judged as either normal or abnormal. Mean percentage calculation was calculated for both 'Normal' and 'Abnormal' judgments using the following formula.

$$\textit{Mean \% percentage} = \frac{\textit{No. of 'Normal/ Abnormal' judgments in each parameter}}{\textit{Total no. of judgments}} \times 100$$

Where,

The total number of judgments was derived by multiplying the number judges and number of subjects in the particular group.

Number of ‘Normal/ Abnormal’ judgments in each parameter referred to the total number normal/abnormal judgments across all the subjects in a particular group and across judges

4.1 Comparison of Perceptual Rating for ‘Normal’ Judgments

Figure 4.1 shows the mean percentage of ‘Normal’ judgments among the samples collected from control and experimental groups. According to Figure 4.1, majority (above 95%) of the speech samples collected from individuals in the control group was judged as Normal by most of the judges. Whereas, less than 50% of the speech samples collected from individuals with AD were judged as ‘Normal’. The result was true for voice, articulation, prosody, rate of speech and overall naturalness.

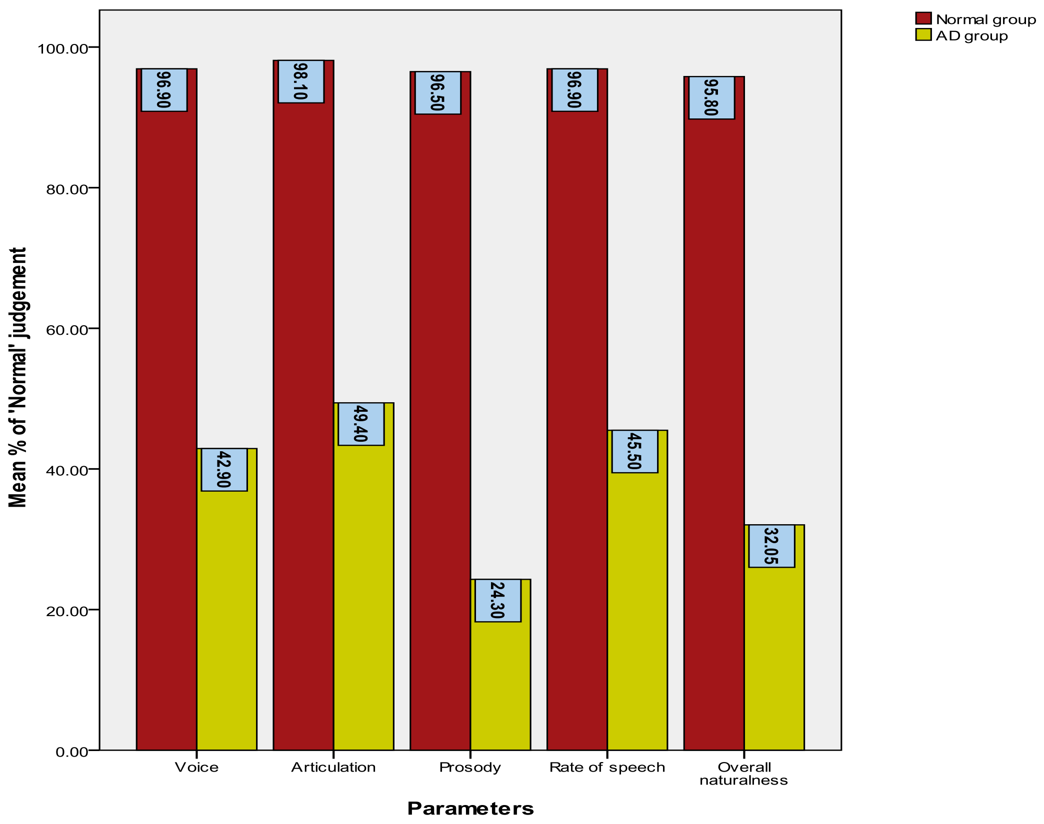

Figure 4.1 : Mean percentage of 'Normal' judgments in each parameter of speech in normal and AD group.

To verify whether there was significant difference between control and experimental groups in terms of the mean percentage of 'Normal' judgments, Equality of proportion was tested and Z scores were calculated. Results of Z scores are given in Table 4.1. Results showed a significant difference between the 2 groups in all the parameters at 0.05 level of significance.

Table 4.1: *Z scores showing the significance of difference between the mean percentage of 'Normal' judgments in normal and AD group*

Parameter	*Z*
Voice	10.59*
Articulation	11.90*
Prosody	19.93*
Rate of speech	12.44*
Overall naturalness	16.18*

*Note: * - $p < 0.05$*

4.2 Comparison of Perceptual Rating for 'Abnormal' Judgments

Figure 4.2 shows the mean percentage of 'Abnormal' judgments assigned for the speech samples of control and AD group in each parameter of speech. The trend observed was opposite of that in the Figure 4.1. The mean percentage of 'Abnormal' judgments was higher in AD group (above 50%) compared to control group (below 5%). The result was true for voice, articulation, prosody, rate of speech and overall naturalness. Within in the different parameters of speech, the mean percentage of abnormality was highest for prosody and lowest for articulation.

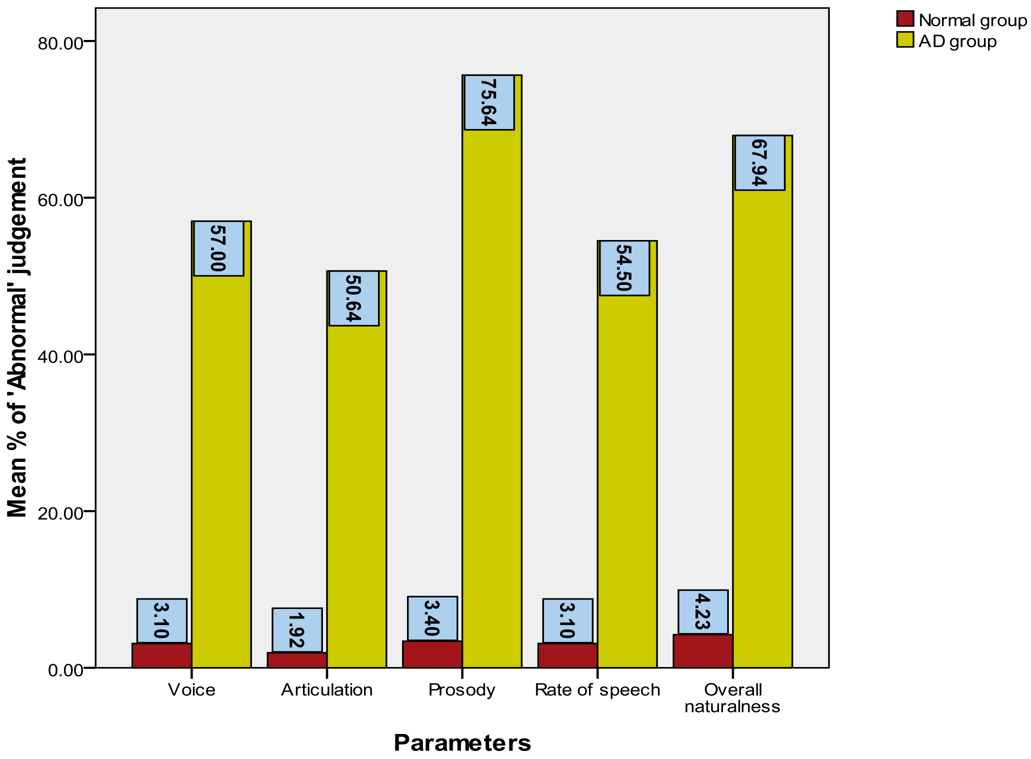

Figure 4.2: Mean percentage of 'Abnormal' judgment in each parameter of speech in normal and AD group.

To verify whether the difference in the mean percentage between the two groups was statistically significant, Z scores were calculated. Results of Z scores are given in Table 4.2. Results showed a significant difference between the 2 groups in all the parameters at 0.05 level of significance.

Table 4.2: *Z scores showing the significance of difference between the mean percentage of 'Abnormal' judgments in normal and AD group*

Parameter	*Z*
Voice	13.18*
Articulation	11.91*
Prosody	19.93*
Rate of speech	12.83*
Overall naturalness	16.71*

*Note: * - $p < 0.05$*

4.3 Comparison of Perceptual Rating Assigned for Overall Intelligibility

A seven point rating scale was used to rate the overall intelligibility of speech samples collected from both the groups. Mean percentage was calculated for overall intelligibility using the following formula.

$$\text{Mean \% percentage of judgments} = \frac{\text{No. of subjects rated under a particular scale}}{\text{Total no. of judgments}} \times 100$$

Figure 4.3 shows the mean percentage calculated for overall intelligibility for both the groups. In general, the AD group showed higher intelligibility rating compared to normals. This means intelligibility of speech of AD was poorer compared to normals. The overall intelligibility ranged from 2 -5 in AD group and 1-3 in normal group.

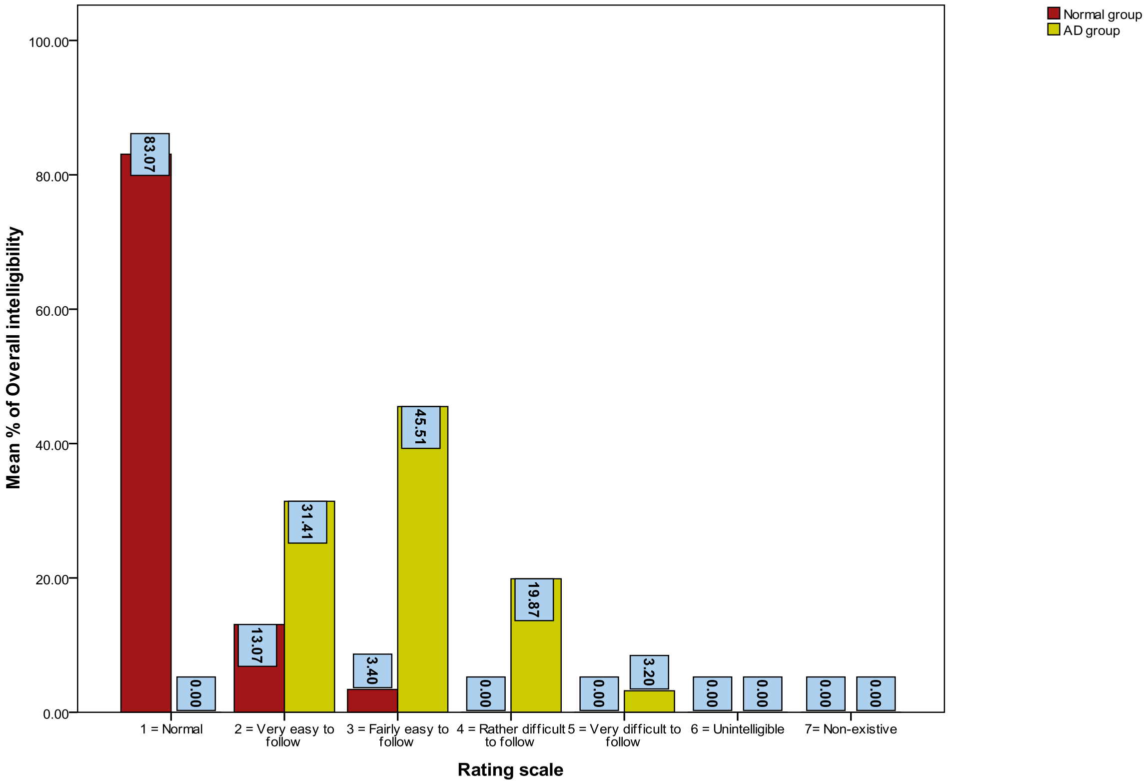

Figure 4.3: Mean percentage of overall intelligibility rating in normal and AD groups.

4.4 Correlation between Speech Identification Scores and Overall Intelligibility

Speech identification scores and ratings of overall intelligibility were correlated using the data obtained from AD group. Correlation was not carried out for the data from normal group. Correlation was done for the identification scores of the better ear as well as poorer ear. Spearman's rho test was used for the task and the results are as given in Table 4.3. The correlation coefficient for the better and poorer ear scores are given separately. Results showed a significant negative correlation ($p<0.05$) between speech identification scores and overall intelligibility ratings. That is, as the speech identification scores decreased, overall speech intelligibility reduced. The result was same with both better and poorer ear scores.

Table 4.3: *Results of correlation between overall intelligibility and speech identification scores of better and poorer ear*

Ear	*N*	*r*
Results of better ear	12	-0.624*
Results of poorer ear	12	-0.673*

*Note: * - p < 0.05*

Phase 2: Acoustic Analysis

The present study considered only the temporal parameters of speech & didn't study the spectral parameters. The parameters were word duration, voice onset time, burst duration, transition duration and speed of transition, preceding vowel duration, and following vowel duration. Descriptive statistical data was obtained in terms of the mean, and standard deviation. The two target groups were compared using independent *t*-test and Mann-Whitney test.

Word Duration

The words for this purpose were chosen from speech samples of passage reading as well as picture description task.

Task 1: Standard Passage

The words were categorized based on the number syllables as bisyllabic, trisyllabic, foursyllabic, fivesyllabic and sixsyllabic words. The number of words available in each category in standard passage is given in Table 4.4. The comparison between the 2 target groups was done separately in each word categories.

Table 4.4: *Number of words in different word categories*

Word Category	*Number of words*
Bisyllabic	15
Trisyllabic	10
Foursyllabic	8
Fivesyllabic	4
Sixsyllabic	2

Table 4.5 gives the Mean and standard deviation of word duration in different word categories in the two groups. In general, the mean word duration was longer in AD group compared to normal group in all the word categories. The AD group had greater variations in the word duration than that of normal group. To verify whether these differences in word duration were significantly different, the two groups were compared using independent *t* test.

Table 4.5: *Mean and Standard Deviation (SD) of word duration in different word categories*

Word Category	*Subject*	*N*	*Mean (in ms)*	*SD*
Bisyllabic	Normal	20	290.59	29.62
	AD	12	348.77	44.83
Trisyllabic	Normal	20	440.67	46.62
	AD	12	509.71	79.48
Foursyllabic	Normal	20	547.85	64.89
	AD	12	607.27	102.58
Fivesyllabic	Normal	20	622.92	74.11
	AD	12	684.54	102.18
Sixsyllabic	Normal	19	774.55	84.73
	AD	12	862.33	153.15

Table 4.6 gives results of independent *t* test. Results showed a significant difference in the word duration between the two groups. Looking at the mean values it can be interpreted that the mean word duration in AD group was significantly longer compared to normal group. However, this statistical difference ($p<0.05$) was seen only in bisyllabic and trisyllabic words. There was no difference ($p>0.05$) in the mean word duration between the two groups in four syllabic, five syllabic and six syllabic words.

Table 4.6: *Results of independent t test in different word categories*

Word	*t*	*df*
Bisyllabic	4.431*	30
Trisyllabic	3.111*	30
Foursyllabic	2.014	30
Fivesyllabic	1.974	30
Sixsyllabic	1.982	29

*Note: * - $p < 0.05$*

Task 2: Picture Description

The words chosen from the picture description were again categorized based on the number syllables; bisyllabic, trisyllabic, and foursyllabic words. The number of words in each category is given in Table 4.7.

Table 4.7: *Number of words in different word categories*

Word Catogery	*Number of words*
Bisyllabic	6
Trisyllabic	2
Foursyllabic	1

Table 4.8 gives the Mean and standard deviation of word duration separately for the two groups. In general, the mean word duration was longer in AD group compared

to normal group in all the word categories. The AD group had greater variations in word duration than that of normal group.

Table 4.8: *Mean and standard deviation (SD) of word duration in different word categories*

Word	*Group*	*N*	*Mean (in ms)*	*SD*
Bisyllabic	Normal	19	3.44	38.34
	AD	11	4.30	70.24
Trisyllabic	Normal	14	4.11	85.18
	AD	8	4.80	106.49
Foursyllabic	Normal	16	5.84	77.30
	AD	5	5.94	52.43

Table 4.9 gives the results of independent *t*-test. Results show a significant difference ($p<0.05$) in the word duration between the two groups in bisyllabic words and trisyllabic words. However, between the groups, there was no significant difference ($p>0.05$) in the mean word duration of foursyllabic words. Since the data available for foursyllabic words in the clinical group was less, Mann Whitney test was done to cross check the results of Independent *t*-test. Results of Mann-Whitney test is given in Table 4.10. Results showed no significant difference ($p>0.05$) between the two groups.

Table 4.9: *Results of Independent t test in different word categories*

Word	*t*	*df*
Bisyllabic	4.353*	28
Trisyllabic	1.671*	20
Foursyllabic	0.279	19

*Note: * - $p < 0.05$*

Table 4.10: *Results of Mann-Whitney test for foursyllabic words*

	Z	*p*
Foursyllabic	-.165	0.869

Other Temporal Parameter

The words for this purpose were chosen from speech samples of passage reading as well as picture description task. Temporal parameters calculated were voice onset time (VOT), burst duration (BD), transition duration (TD) and speed of transition (STD), preceding vowel duration (PVD), following vowel duration (FVD). The comparison between the 2 target groups was done separately in each parameter. The two target groups were compared using independent *t*-test and Mann-Whitney test.

Task 1: Standard Passage Reading

Table 4.11 gives the Mean and standard deviation of data of each temporal parameter in both the groups. In general, the mean duration for VOT, BD, PVD and FVD were longer in AD group compared to normal group. STD was faster in AD groups compared to normal groups. However, the mean duration for TD was shorter in AD group compared to normal group. The AD group had greater variation in all parameters than that of normal group. To verify whether these differences in all parameters were significantly different, the two groups were compared using independent *t* test.

Table 4.11: *Mean and Standard deviation (SD) of data of different temporal parameter in two groups*

Parameter	*Group*	*N*	*Mean (in ms)*	*SD*
VOT	Normal	20	48.15	7.94
	AD	12	60.29	7.82
BD	Normal	20	9.24	1.31
	AD	12	12.64	1.68
TD	Normal	20	38.10	6.02
	AD	12	26.47	7.48
STD	Normal	20	8.90	2.91
	AD	12	14.01	6.11
PVD	Normal	20	87.11	18.88
	AD	12	109.28	16.49
PVD	Normal	20	76.88	14.43
	AD	12	114.37	27.74

Table 4.12 gives results of Independent *t*-test. Results show a significant difference ($p<0.05$) in all the parameters; VOT, BD, TD, STD, PVD and FVD between two groups.

Table 4.12: *Results of Independent t test in different temporal parameter of speech*

Parameter	*t*	*df*
VOT	3.744*	30
BD	6.381*	30
TD	4.827*	30
STD	3.201*	30
PVD	3.365*	30
FVD	5.045*	30

*Note: * - $p < 0.05$*

Task 2: Picture Description

Table 4.13 gives Mean and standard deviation data for each parameter in both the groups. The words spoken by both the groups were not the same as those selected for analysis. In general, the mean duration of VOT, BD and TD were longer in AD group compared to normal group. However, the mean duration for STD was faster in AD group compared to normal group. The AD group had greater variation in all parameters than that of normal group. To verify whether these differences were significantly different, the two groups were compared using independent *t* test.

Table 4.13: *Mean & Standard deviation (SD) of data of different temporal parameter in two groups*

Parameter	*Group*	*N*	*Mean (in ms)*	*SD(in ms)*
VOT	Normal	19	65.57	11.64
	AD	7	81.78	21.78
BD	Normal	19	9.14	1.94
	AD	5	13.20	1.78
TD	Normal	19	45.01	5.25
	AD	8	50.91	5.46
STD	Normal	19	6.87	3.32
	AD	8	4.11	2.65

Table 4.14 gives results of independent *t* test. Results show a significant difference between the two groups in all the parameters. Since the data available for all parameters in the clinical group was less, Mann Whitney test was done to cross check the results of Independent *t* test. Table 4.15 gives results of Mann-Whitney test done for all parameters for picture description task. Results showed a significant difference ($p<0.05$) between the two groups in VOT, BD, TD, and STD.

Table 4.14: *Results of Independent t test for each parameter*

Parameter	*t*	*df*
VOT	2.469*	24
BD	4.194*	22
TD	2.634*	25
STD	2.080*	25

*Note: * - $p < 0.05$*

Table 4.15: *Results of Mann-Whitney test for each parameter for both groups*

Parameter	*Z*	*p*
VOT	1.706	0.008*
BD	2.898	0.004*
TD	2.154	0.031*
STD	2.177	0.029*

*Note: * - $p < 0.05$*

Chapter 5

DISCUSSION

Major findings of the present study can be summarized as follows

- Results of the perceptual experiment showed that, majority of speech samples of auditory dyssynchrony (AD) were perceptually abnormal. All the parameters of speech (voice, articulation, prosody, rate of speech & overall naturalness) showed abnormality, although the prosody was found to be maximally affected.
- Overall intelligibility of speech of AD was found to be poorer. In individuals with AD, there was a significant high correlation between speech identification scores and, the severity of abnormality in speech intelligibility.
- Acoustically, there were deviations observed in the speech of AD. Speech of AD showed lengthened VOT, BD, PVD and FVD. Speech of transition was faster in AD group compared to normals. Results of Transition duration were different between the two tasks. In individuals with AD, TD was shorter in the reading task whereas, it was lengthened in the picture description task.

The main purpose of the present study was to characterize the speech of AD. The study was planned with a hypothesis that speech perception deficits when it is long-standing could lead to speech production deficits. The basis for such a hypothesis was the results of earlier studies that characterized the speech production of individuals with longstanding cochlear hearing loss (Binnie et al, 1982; Cowie et al, 1982; Elman, 1981; Kirchner & Suzuki, 1968; Penn, 1955; Ramsden, 1981, Ramsden, 1981). These studies had shown regression of speech both in terms of segmental as well as suprasegmentals (Leder, Spitzer, & Kirchner, 1987a).

In the present study, the mean speech identification scores in AD group were 24.58 % and 32.67 % in the poorer & better ear respectively. These scores are evidence for the presence of severe speech perceptual deficit in the study group. The individuals in the AD group had the same diagnosis five years back and they were contacted through mail and recruited. This way, longstanding (atleast for 5 years) nature of the disorder was confirmed. In the literature, a close relationship has been established between speech perception, and the development as well as maintainance of speech production skills (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967). Self-hearing has been proved to play an essential role in maintaining intelligible speech by mapping the systematic (language-determined) relationship between the sounds and the articulatory activity produced. Considering these results, it is expected that individuals in the AD group show errors in the speech production due to defective auditory feedback. Results of the present study seem to support this notion.

Results of Perceptual Analysis

Speech production was abnormal in individuals with longstanding AD. Perceptually, all the parameters of speech were found to be abnormal. The result of abnormal production is in agreement with earlier studies in individuals with cochlear hearing loss. Houde and Jordan, (2002) reported that compensatory changes in individual sound production can be induced over time by systematically altering auditory feedback to indicate inaccurate articulation. Similar findings have been reported by other investigators (Binnie et al, 1982; Cowie et al, 1982; Elman, 1981; Kirchner & Suzuki, 1968; Penn, 1955; Ramsden, 1981, Zimmermann & Rettaliata, 1981). Auditory feedback helps in moment-to-moment monitoring, periodic update and calibration of the system. These results seem to support closed loop models of speech production.

The notion of reduced auditory feedback being the reason for abnormal articulation has been refuted by researchers who argue in favor of a predominantly open-loop speech motor control system. That is, the speaker ‘‘knows’’ the relationship between motor commands and resulting sound output and uses this knowledge to compute the motor sequence for producing the desired speech output (Matthies et al., 1996). Consequently, based on their experience with auditory and nonauditory information, they could maintain their articulatory moments. This is in agreement with studies done by Leder and Spitzer (1990) and, Goehl and Kaufman (1984), who had reported that no clinically significant deterioration of speech sound production (i.e., segmental errors) in adventitiously deaf adult’s speech. The fact that individuals with profound postlingual cochlear hearing loss maintain highly intelligible speech (Matthies et al., 1996) suggests that mature phonemic motor patterns are quite robust, and do not depend heavily on auditory feedback.

The results of the present study do not support the open loop models as it was found that 5 years of disrupted auditory feedback, due to AD, resulted in abnormal production. Furthermore, in the present study, it was found that articulation was least affected compared to prosody. This could be accounted by the difference in the feedback involved in articulation and prosody. In articulation, feedback is available from both auditory and non-auditory (tactile) modes. So if the feedback from the auditory system is disrupted due to AD, the tactile system will still be cueing to some an extent. Whereas, in prosody, the feedback is only from the auditory mode. Because there is no alternate mode, in instances of disrupted auditory feedback due to AD, prosody should get affected to a greater extent than articulation. Hence, the presence of tactile feedback (of articulators) is helping to reduce the regression in articulation, in turn supporting the closed loop models.

Results of the present study showed that the speech produced by individuals with AD was less intelligible compared to normals. Also, there was a significant correlation between speech identification scores and the over intelligibility of speech of AD. That means ones who had relatively better speech identification, produced more intelligible speech compared to ones who had poorer speech identification. This result again supports the importance of auditory feedback to speech production skills. More the disruption in auditory feedback, more likely of errors in speech production. In contrast, Binnie et al. (1982) and others (Cowie et al., 1982; Plant, 1984) had reported that the changes in speech production observed in deafened adults appear to have little effect on speech intelligibility in cases where onset of deafness occurred in adulthood. The earlier the onset of deafness, the greater the effect of hearing loss on intelligibility.

Results of Acoustic Analysis

Earlier studies on speech perception in AD (Ajith, 2006) had shown increased just noticeable differences in the VOT, burst duration and transition duration. Considering these results, it was hypothesized that there shall be changes in the temporal parameters of speech of longstanding AD. Also, results of perceptual analysis in the present seemed to support the closed loop models of speech production. In such a case, the type of errors seen in the speech production in some way related to type of perceptual deficit. That is, if individuals with AD require more temporal and spectral difference to discriminate between phonemes, in their own production they may be using compensatory changes and producing the phonemes accordingly different. This was probed through acoustic analysis of speech of AD. The parameters analyzed were word duration, voice onset time, transition duration, burst duration, speed of duration, preceding vowel duration, and following vowel duration.

The mean word duration was longer in AD group compared to normal group in all the word categories. Statistical difference between the groups in mean word duration was present in bisyllabic and trisyllabic words. This could be because of their slower articulation of speech. Slower articulation results in lengthened temporal cues of speech. Probably, this is a compensatory strategy they adopt over time to facilitate the auditory feedback. This is in agreement with studies done by various investigators who have observed that postlingually deafened adults have significantly longer sentence duration (Kirk & Edgerton, 1983; Lane & Webster, 1991; Lane et al., 1998) which is a result of significantly longer syllables (Lane & Webster, 1991; Leder et al., 1987b), pause duration (Lane & Webster, 1991), and vowel duration (Waldstein, 1990). However, the duration of four, five and six syllabic words did not differ between the two groups which may be attributed to the higher standard deviation in these word categories.

The increase in the mean word duration could have been either due to increase in consonantal duration, vowel duration or both. To probe into this issue additional temporal parameters were considered that includes voice onset time, burst duration, transition duration, speed of transition, preceding vowel duration and following vowel duration. The results were compared between normal and AD group.

Results showed a significant difference between the groups in all the temporal parameters. For standard passage reading task, the mean duration for VOT, burst duration, PVD and FVD were longer in AD group compared to normal group. Hence, increased word duration is contributed by increased consonantal as well as vowel duration. The above mentioned temporal parameters cue for the perception of place as well as manner of different phonemes (Kumar, 2005). The fact that these are lengthened indicates that individuals with AD are making modifications in their own production to get better feedback on place and manner of articulation. Unlike anticipated, speed of transition was faster and TD was shorter in AD group. This could not be furnished with any logical explanation. In the picture description task, all the parameters; VOT, burst duration and TD were longer in AD group compared to normal group. However, STD was faster in AD group compared to normal group.

Overall, there was agreement between the perceptual deficits and the speech production characteristics. This supports the closed loop models of speech production and refutes the open loop models. There was also a good agreement between perceptual deficits in individuals with AD (as reported in studies in the literature) and the characteristics of speech of AD. In general, there was lengthened temporal cues of speech and this could be probably a compensatory strategy used by individuals with AD to facilitate the perception.

Chapter 6

SUMMARY AND CONCLUSIONS

Auditory dys-synchrony (AD) is a clinical syndrome in which outer hair cell function is spared, but afferent neural transmission is disordered. Psychophysical and perceptual studies have shown that individuals with AD are proved to have more serious deficits in speech processing and perception. These deficits further deteriorate in adverse listening conditions.

Studies had shown that there is a close relationship between speech perception and the development of speech production skills. These studies had shown regression of speech both in terms of segmental as well as suprasegmentals. Rance, Barker, Sarant, and Ching, (2007) reported that school aged children with AD hearing loss are developing spoken language more slowly than would be expected for children with normal hearing. This is expected as the perceptual deficits occur before the development of the speech and language. However, it was interesting to study whether the speech errors are present in postlingually acquired AD. There was no study to characterize the speech production errors in adolescent or adults with long-standing AD. Hence, the purpose of the present study was to characterize the speech production of adults with long-term AD.

The study was conducted in 12 individuals (8 females & 4 males) with acquired long-term AD and 20 age matched individuals (10 males & 10 females) with normal hearing sensitivity. This study was carried out in the 2 phases. Phase 1 included the perceptual analysis of speech of adults with long-term AD, while the phase 2 included acoustic analysis of the same speech samples. In phase 1, perceptual rating assigned for all the parameters (voice, articulation, prosody, rate of speech & overall intelligibility) of speech of normal individuals and individuals with AD were compared between the two groups. Overall intelligibility was rated on speech intelligibility rating scale given by Markides (1986). Speech identification scores and overall intelligibility in individuals with AD was correlated.

In the phase 2, acoustic analysis was done & temporal parameters of speech (word duration, voice onset time, burst duration, transition duration and speed of transition, preceding vowel duration, & following vowel duration) of normal individuals and individuals with AD were statistically compared.

Results of the perceptual experiment showed that, majority of speech samples of auditory dyssynchrony (AD) were perceptually abnormal. All the parameters of speech (voice, articulation, prosody, rate of speech & overall naturalness) showed abnormality, although the prosody was found to be maximally affected. The overall intelligibility of speech of AD was found to be poorer. In individuals with AD, there was a significant high correlation between speech identification scores and, the severity of abnormality in speech intelligibility. These results further validate the need of auditory feedback in maintaining the speech production skills. In instances of disordered auditory feedback, there is deterioration of speech over time. Also, more speech perception difficulties were found to be associated with more abnormalities in speech production. The results show a close relationship between speech perception and speech production skills in turn supporting the closed loop models of speech production.

Results also showed deviations in speech production in the acoustical analysis. In general, there were lengthened temporal cues of speech. Earlier studies had shown increased JNDs in AD. Hence, lengthened temporal cues could be probably a compensatory strategy used by individuals with AD to facilitate the perception of their own speech.

REFERENCES

Abdala, C., Sininger, Y., & Starr, A. (2000). Distortion product otoacoustic emission suppression in subjects with auditory neuropathy. *Ear and Hearing, 21,* 542-53.

Ajith, K. U. & Jayaram, M. (2006). Prevalence and audiological characteristics in individuals with auditory neuropathy/AD. *International Journal of Audiology, 45,* 360-366.

Ajith, K. U. (2006). *Perception of some temporal parameters of speech in individuals with AD.* Unpublished Dissertaion submitted to University of Mysore.

Barinaga, M. (1998). Listening in on the brain. *Science, 280,* 376-378.

Berlin, C. I. (1999). Auditory Neuropathy: Using OAEs and ABRs from screening to management. *Seminars in Hearing, 29,* 307-315.

Berlin, C. I., Bordelon, J., St John, P., Wilensky, D., Hurleym, A., Kluka, E., & Hood, L.J. (1998). Reversing click polarity may uncover auditory neuropathy in infants. *Ear and Hearing, 19,* 37-47.

Berlin, C. I., Hood, L. J. Morlet, T., Wilensky, D., St John, P. Montgomery, E., & Thibodaux, M. (2005). Absent or elevated middle ear muscle reflexes in the presence of normal otoacoustic emissions: A universal finding in 136 cases of auditory neuropathy/dys-synchrony. *Journal of American Academy Audiology, 16,* 546-553.

Berlin, C. I., Hood, L. J., Cecola, R. P., Jackson, D. F., & Szabo, P. P. (1993). Does type II afferent neuron dysfunction reveals itself through lack of efferent suppression. *Hearing Research, 65,* 40-50.

Berlin, C. I., Hood, L., Morlet, T., Rose, K., & Brashears, S. (2003). Auditory neuropathy/dys-synchrony: diagnosis and management. *Mental Retardation and Developmental Disabilities Research Reviews, 9,* 225–231.

Berlin, C. I., Morlet, T., & Hood, L. J. (2003). Auditory neuropathy/ dyssynchrony: its diagnosis and management. *Pediatric Clinics of North America, 50*, 331–340.

Berlin, C., Li, L., Hood, L., Morlet, T., *&* Brashears, S. (2002). Auditory Neuropathy/Dys-Synchrony: After the Diagnosis, then what? *Seminars in Hearing, 23,* 209-214.

Berlin, C.I., Hood, L.J., Hurley, M.S., & Wen, H. (1996). Hearing aids: Only for hearing impaired patients with abnormal otoacoustic emissions. In Berlin CI (ed): Hair Cells and Hearing Aids. San Diego: Singular Publishing Group, 99-111.

Binnie, C.A., Daniloff, R.G., & Buckingham, H.W. (1982). Phonetic disintegration in a five-year-old following sudden hearing loss. *Journal of Speech Hearing Research, 47,* 181-189.

Boothroyd, A. (1984). Auditory perception of speech contrasts by subjects with sensorineural hearing loss. *Journal of Speech and Hearing Research, 27,* 134-144.

Buss, E., Labadie, R.F., Brown, C.J., Gross, A.J., Grose, J.H., & Pillsbury, H.C. (2002). Outcome of cochlear implantation in pediatric auditory neuropathy. *Otology Neurotology, 23,* 328–332.

Cacace, A.T. & McFarland, D.J. (1998). Central auditory processing disorder in school-aged children: a critical review. *Journal of Speech Language Hearing Research, 41,* 355–373.

Chisin, R., Pearman, M., & Sohmer, H. (1979). Cochlear and brainstem responses in hearing loss following neonatal hyperbilirubinemia. *Annals of Otology Rhinology & Laryngology, 88,* 352-357.

Cowie, R., Douglas-Cowie, E., & Kerr, A.G. (1982). A study of speech deterioration in post-lingually deafened adults. *Journal of Speech Hearing Disorder, 96,* 101-112.

Culbertson, D.N. & Kricos, P.B. (2002). Language and speech of the deaf and hard of hearing. In R.L. Schow & M.A. Ling (Eds.), *Introduction to Audiologic Rehabilitation* (4th Ed.). (pp.183-224). Boston: Allyn and Bacon.

Davis, H., & Hirsh, S. (1979). A slow brainstem response for low-frequency audiometry. *Audiology, 25*, 84-91.

Deltenre, P., Mansbach, A.L., Bozet, C., Christiaens, F., Barthelemy, P., Paulissen, D., & Renglet,T. (1999). Auditory neuropathy with preserved cochlear microphonics and secondary loss of otoacoustic emissions. *Audiology*, *38*, 187-195.

Dolphin, W.F. (2004). Auditory neuropathy and configured hearing loss: the case for two-stage screening. *Hearing Review*. *3*, 28-32.

Duan, J. & Wang, J. (2002). The ECochG in patients with auditory neuropathy. *Lin Chuang Er Bi Yan Hou Ke a Zhi*, *16,* 605-606.

Dunn, C. & Newton, L. (1986). A comprehensive model for speech development in hearing impaired children. *Topics in Language Disorders*, *6(3),* 25-46.

Elfenbein, J.L., Hardin-Jones, M.A., & Davis, L.M. (1994). Oral communication skills of children who are hard of hearing. *Journal of Speech and Hearing Research*, 37, 216-226.

Elman, J. (1981). Effect of frequency shifted feedback on the pitch of vocal production. *Journal of the Acoustical Society of America*, *70,* 45-50.

Foerst, A., Beutner, D., Lang-Roth, R., Huttenbrink, K.B., von Wedel, H., & Walger, M. (2006). Prevalence of auditory neuropathy/ synaptopathy in a population of children with profound hearing loss. *International Journal of Pediatric Otorhinolaryngology*. *70,* 1415-1422.

Formby, C. (1986). Modulation detection by patients with eighth-nerve tumors. *Journal of Speech and Hearing Research*, *29,* 413–419.

Fowler, C.A. & Saltzman, E. (1993). Coordination and coarticulation in speech production. *Language and Speech*, *36,* 171-195.

Fuchs, P. A., Glowatzki, E., & Moser, T. (2003). The afferent synapse of cochlear hair cells. *Current Opinion in Neurobiology, 13,* 452–458.

Gordon-Salant, S. & Fitzgibbons, P.J. (1999). Profile of auditory temporal processing in older listeners. *Journal of Speech Language and Hearing Research, 42*, 300–311.

Goehl, H., & Kaufman, D. (1984). Do the effects of adventitious deafness include disordered speech? *Journal of Speech and Hearing Disorder, 49,* 58-64.

Grover, S. (1998). *Acoustic analysis of prosody of punjabi-in deaf children,* Unpublished Dissertaion submitted to University of Mysore, as part of fulfillment of Master degree in Speech and Hearing..

Houde, J. F. & Jordan, M. I. (1998). Sensorimotor adaptation in speech production. *Science 279,* 1213–1216.

House, A. S. (1961). On vowel duration in English. *Journal of the Acoustical Society of America*, *33,* 1174-1178.

Hudgins, C. & Numbers, F. (1942). An investigation of the intelligibility of the speech of deaf and normal subjects. *Genetic Psychology Monographs*, *25*, 286-392.

Jayaradha S. (2001). *Coarticulation in the speech of prelingually profound hearing impaired and normal children.* Unpublished Dissertaion submitted to University of Mysore, as part of fulfillment of Master degree in Speech and Hearing..

Jerger, J.F., Ali, A., Fong, K., et al. (1992). Otoacoustic emissions,audiometric sensitivity loss, and speech understanding: A case study. *Journal of American Academy of Audiology, 1,* 283-286.

Johnson, D.H. (1980). The relationship between spike rate and synchrony in response of auditory-nerve fibers to single tones. *Journal of the Acoustical Society of America 68,* 1115-1122.

Joris, P.X., & Yin, T.C.T. (1992). Response to amplitude-modulated tones in the auditory nerve of the cat. *Journal of the Acoustical Society of America, 91,* 215-232.

Kirchner, S. & Suzuki, Y. (1968). Laryngeal reflexes and voice production. *NY academy Science, 155,* 98-129.

Kirk, K. & Edgerton, B. J. (1983). The effects of cochlear implant use on voice parameters. *Otolaryngologic Clinics of North America, 16,* 281–292.

Kraus, N., Bradlow, A. R., Cheatham, J., Cunningham, C. D., King, D.B., Kouch, T. G., Nicol, T.J., McGee, L. K., Stein, L. K., & Wright, B. A. (2000). Consequence of neural asynchrony: A case study of auditory neuropathy. *Journal of the Association for Research in Otolaryngology, 1,* 33-45.

Kraus, N., Bradlow, A.R., Cheatham, M.A., Cunningham, J., King, C.D., & Koch, C.D. (2000). Consequences of neural asynchrony: A case of auditory neuropathy. *Journal of Association for Research in Otolaryngology, 1,* 33-45.

Kuhl, P.K. (1981). Discrimination of speech by nonhuman animals: Basic auditory sensitivities conducive to the perception of speech-sound categories. *Journal of the Acoustical Society of America, 70,* 340-349.

Kumar, V. & Vanaja, C. S. (2008) Speech identification and cortical potentials in individuals with auditory neuropathy *Behavioral and Brain Functions, 4,* 1-5.

Lalaki P. (2003). *Otoacoustic emissions in the assessment of the efferent auditory system: clinical and experimental protocols.* Thesis, Aristotle University, Thessaloniki, Greece.

Lane, H. L. & Webster, J. (1991). Speech deterioration in postlingually deafened adults *Journal of the Acoustical Society of America, 89,* 859–866.

Lane, H. L., Perkell, J., Wozniak, J., Manzella, J., Guiod, P., Matthies, M., MacCollin, M., & Vick, J. (1998). The effect of changes in hearing status on speech sound level and speech breathing: A study conducted with cochlear implant users and NF-2 patients. *Journal of the Acoustical Society of America, 104,* 3059–3069.

Leder, S.B. & Spitzer, J.B. (1990). A perceptual evaluation of the speech of adventitiously deaf adult males. *Amplification and Aural Rehabilitation, 11,* 169-175.

Leder, S.B., Spitzer, J.B, Kirchner, J.C., Flevaris-Philips, C., Milner, P., & Richardson, F. (1987c). Speaking rate of adventitiously deaf male cochlear implant candidates. *Journal of the Acoustical Society of America, 82,* 843-846.

Leder, S.B., Spitzer, J.B., & Kirchner, J.C. (1987a). Speaking fundamental frequency of adventitiously profoundly deaf adult males. *Anna Oto Rhino Laryngologie, 96*, 322-324.

Leder, S.B., Spitzer, J.B., Milner, P., Flevaris-Philips, C., Kirchner, J.C., & Richardson, F. (1987b). Voice intensity of prospective cochlear implant candidates and normal- hearing adult males. *Laryngoscope*, *97*, 224-227.

Levine, R.A., Gardner, J.C., Fullerton, B.C., Stufflebeam, S.M., Carlisle, E.W., Furst, M., Rosen, B.R., & Kiang, N.Y. (1993). Effects of multiple sclerosis brainstem lesions on sound lateralization and brainstem auditory evoked potentials. *Hearing Research*, *68,* 73–88.

Liberman, A. M., Cooper, F. S., Shankweiler, D. P., & Studdert-Kennedy, M. (1967). Perception of the speech code. *Psychology Review, 74,* 431-461.

Markides, A. (1986). Age at fitting of hearing aids and speech intelligibility. *British Journal of Audiology*, *20,* 165-167.

Matthies, M. L., Svirsky, M. A., Perkell, J. S., & Lane, H. L. (1996). Acoustic and articulatory measures of sibilant production with and without auditory feedback from a cochlear implant, *Journal of Speech and Hearing Disorder, 39,* 936–946.

Michalewski, H. J., Starr, A., Nguyen, T. T., Kong, Y. Y., & Zeng, F. G. (2005). Auditory temporal processes in normal- hearing individuals and in patients with auditory neuropathy. *Clinical Neurophysiology, 116*, 669-680.

Moore, B. C. J. (1995). Speech perception in people with cochlear damage. In *Perceptual consequences of cochlear damage* 147-172.

Moore, B.C. & Oxenham, A.J. (1998). Psychoacoustic consequences of compression in the peripheral auditory system. *Psychology Review, 105,* 108–124.

Moore, B.C., (1996). Perceptual consequences of cochlear hearing loss and their implications for the design of hearing aids. *Ear and Hearing, 17,* 133–161.

Nienhuys, T.G. & Clark, G.M. (1978). Frequency discrimination following the selective destruction of cochlear inner and outer hair cells. *Science, 199,* 1356–1357.

Norton, S. J. & Widen, J. E. (1990). Evoked otoacoustic emissions in normal infants and children: Emerging data and issues. *Ear and Hearing, 11,* 121-127.

Oxenham, A.J. & Bacon, S.P. (2003).Cochlear compression: perceptual measures and implications for normal and impaired hearing. *Ear and Hearing, 24,* 352–366.

Penn, G. (1955). Voice and speech patterns in the hard of hearing. *Acta Otolaryngology, 124,* 320-326.

Perkell J, Matthies M, & Lane H. (1997). Speech motor control: acoustic goals, saturation effects, auditory feedback and internal models. *Speech Communication, 22,* 227-249.

Prosen, C.A., Moody, D.B., Stebbins, W.C., & Hawkins, J.E. (1981). Auditory intensity discrimination after selective loss of cochlear outer hair cells. *Science, 212,* 1286–1288.

Ramsden, R.T. (1981). Rehabilitation of the suddenly deafened adults. *Ear Nose Throat Journal, 60,* 49-54.

Rance, G., Barker, E. J., Sarant, J. Z., & Ching, Y.C. (2007). Receptive language and speech production in children with Auditory neuropathy/ Dyssynchrony type hearing loss. *Ear and hearing, 28,* 694-702.

Rance, G., Barker, E., Mok, M., Dowell, R., Rincon, A., & Garratt, R.U. (2007). Speech perception in noise for children with auditory neuropathy/dys-synchrony type hearing loss, *Ear and Hearing, 28(3),* 351-360.

Rance, G., Beer, D., Cone-Wesson, B., Shepard, R. K., Dowell, R. C., King, A. M., Rickards, F. W., & Clark, G. M. (1999). Clinical findings for a group of infants and young children with auditory neuropathy. *Ear & Hearing, 20,* 238–263.

Rance, G., Cone-Wesson, B., Wunderlich, J., & Dowell, R. (2002). Speech perceptionand cortical event related potentials in children with auditory neuropathy. *Ear and Hearing, 23, 239*-253.

Rance, G., McKay, C., & Grayden, D. (2004). Perceptual characterization of children with auditory neuropathy. *Ear and Hearing, 25,* 34-46.

Rance, G., McKay, C., & Grayden, D. (2004). Perceptual characterstics of children with auditory neuropathy. *Ear and Hearing, 23,* 239-253.

Rapin, I. & Gravel, J. (2003). Auditory neuropathy : physiologic and pathologic evidence calls for more diagnostic specificity. *International Journal of Pediatric Otorhinolaryngol*ogy, *67,* 707–728.

Riehle, A., Grun, S., Diesmann, M., & Aertsen, A. (1997). Spike Synchronization and Rate Modulation Differentially Involved in Motor Cortical Function. *Science, 278,* 1950-1953.

Ryan, A. & Dallos, P. (1975). Effect of absence of cochlear outer hair cells on behavioural auditory threshold. *Nature, 253,* 44–46.

Santarelli, R. & Arslan, E. (2002). Electrocochleography in auditory neuropathy. *Hearing Research, 170,* 32-47.

Shallop, J.K. (2002). Auditory neuropathy/dys-synchrony in adults and children. *Seminar in Hearing, 23(3),* 215-223.

Shannon, R.V., Zeng, F.G., Wygonski, J., Kamath, V., & Ekelid, M. (1995**)**. Speech recognition with primarily temporal cues, *Science, 270,* 303-304.

Sininger, Y. & Oba, S. (2001). Patients with auditory neuropathy: Who are they and what can they hear? In Y. Sininger, & A. Starr (Eds.), *Auditory neuropathy: A new perspective on hearing disorder* (pp. 15-36). Canada: Singular publishing group.

Sininger, Y. & Starr, A. (2001). *Auditory Neuropathy: A New Perspective on Hearing Disorders,* Thomson publishing DL, San Diego.

Sininger, Y.S., Hood, L. J., Starr, A., Berlin, C.I., & Picton, T.W. (1995). Hearing loss due to auditory neuropathy. *Audiology Today, 7, 10*-13.

Smith, B.L. (1982). Some observations concerning pre-meaningful vocalization of hearing impaired infants. *Journal of Speech and Hearing Disorders*, *47*, 439-441.

Srulovicz, P. & Goldstein, J.L. (1983). A central spectrum model: a synthesis of auditory nerve timing and place cues in monoaural communication of frequency spectrum. *Journal of the Acoustical Society of America, 73,* 1266-1276.

Starr, A., McPherson, D., Patterson, J., Don, M., Luxford, W., Shannon, R., Sininger, Y., Tonakawa, L., & Waring, M. (1991). Absence of both auditory evoked potentials and auditory percepts dependent on timing cues. *Brain, 114,* 1157-1180.

Starr, A., Michalewski, H.J., Zeng, F.G., Brooks, S.F., Linthicum, F. Kim, C.S., Winnier, D., & Keats, B. (2003). Pathology and physiology of auditory neuropathy with a novel mutation in the MPZ gene**.** *Brain, 126***;** 1604-1619.

Starr, A., Picton, T.W., & Kim, R. (2001). Pathophysiology of auditory neuropathy. In: Y. Sininger, & A. Starr (Eds.), *Auditory neuropathy: A new perspective on hearing disorder* (pp. 67-82). Canada: Singular publishing group.

Starr, A., Picton, T.W., Sininger, Y., Hood, L., & Berlin, C.I. (1996). Auditory neuropathy. *Brain*, *119,* 741-753.

Starr, A., Sininger, Y. S., & Pratt, H. (2000). The varieties of auditory neuropathy. *Journal of Basic and Clinical Pharmacology, 11*, 215–230.

Stevens, K.N. (2002). Toward a model for lexical access based on acoustic landmarks and distinctive features. *Journal of the Acoustical Society of America, 111*, 1872-1891.

Stopfer, M., Bhagavan, S., Smith, B.H., & Laurent, G. (1997). Impaired odour discrimination on desynchronization of odour-encoding neural assemblies. *Nature, 390,* 70-74.

Tang, T.P., Mcpherson, B., Yuen, K.C., Wong, L.L., & Lee, J.S. (2004). Auditory neuropathy/AD in school children with hearing loss: Frequency of occurrence. *International Journal of Pediatric Otolaryngology, 168,* 175-183.

Tapia Toca M.C., & Savio López, G. (2005). Steady-state auditory evoked potentials in the study of two patients with auditory neuropathy *Acta Otorrinolaringologia Esp; 56,* 240-245.

Trussell, L. O. (1999). Synaptic mechanisms for coding timing in auditory neurons. *Annual Review of Physiology, 61,* 477-496.

Vinay, S.N. & Moore, B.C. (2007). Ten (HL)-test results and psychophysical tuning curves for subjects with auditory neuropathy, *International Journal of Audiology, 46,* 39-46.

Whitehead, R.L. & Jones, K.O. (1978). The effect of vowel environment on duration of consonants produced by normal-hearing, hearing-impaired and deaf adult speakers. *Journal of Phonetics, 6,* 77-81.

Wright, B.A., Lombardino, L.J., King, W.M., Puranik, C.S., Leonard, C.M., & Merzenich, M.M. (1997). Deficits in auditory temporal and spectral resolution in language-impaired children. *Nature, 387,* 176-178.

Yellin, M.W., Jerger, J., & Fifer, R.C. (1989). Norms for disproportionate loss in speech intelligibility. *Ear and Hearing, 10(4),* 231-234.

Young, E.D., & Sachs, M.B. (1979). Representation of steady-state vowel in the temporal aspects of the discharge patterns of populations of auditory nerve fibers. *Journal of the Acoustical Society of America, 66,* 1381-1392.

Zeng, F. G. & Liu, S. (2006). Speech perception in individuals with auditory neuropathy. *Journal of Speech, Language, and Hearing Research, 49,* 367-380.

Zeng, F. G., Kong, Y. Y., Michalewski, H. J., & Starr, A. (2005). Perceptual consequences of disrupted auditory nerve activity. *Journal of Neurophysiology, 93,* 3050-3063.

Zeng, F. G., Oba, S., & Starr, A. (2001). Supra threshold processing deficits due to desynchronous neural activities in auditory neuropathy. In D.J. Breebaart, A. J. M. Houstma, A. Kohlrausch, V.F. Prijis & R. Schoonhoven (Eds.): *Physiological and Psychophysical Bases of Auditory Function.* Shaker publishing BV, Maastricht, The Netherlands. 365-372.

Zeng, F. G., Oba, S., Garde, S., Sininger, Y., & Starr, A. (1999). Temporal and speech processing deficits in auditory neuropathy. *NeuroReport, 10,* 3429-3435.

Zeng, F.G., & Shannon, R.V. (1994). Loudness-coding mechanisms inferred from Electric stimulation of Human auditory system. *Science, 264,* 564-566.

Zeng, F.G., Kong, Y.Y., Michalewski, H.J. & Starr, A. (2004). Perceptual consequences of disrupted auditory nerve activity. *Journal of Neurophysiology, 93,* 3050-3063.

Zeng, F.G., Kong, Y.Y., Michalewski, H.J., & Starr, A. (2005). Perceptual consequences of disrupted auditory nerve activity. *Journal of Neurophysiology, 93,* 3050-3063.

Zeng, F.G., Oba, S., Garde, S., Sininger, Y., & Starr, A. (2001). Psychoacoustics and speech perception in auditory neuropathy. In: Y. Sininger, & A. Starr (Eds.), *Auditory neuropathy: A new perspective on hearing disorder* (pp. 141-164). Canada: Singular publishing group.

Zimmermann, G.N., & Rettaliata, P. (1981). Articulatory pattern of an adventitiously deaf speaker: implication for the role of auditory information in speech production. *Journal of Speech Hearing Research, 24,* 169-179.

Appendix- 1

Standard Reading Passage

Passage ‘B’ (Voiced)

¨ÉAUÀ¼ÀÆgÀÄ £ÀªÀÄä gÁdåzÀ MAzÀÄ zÉÆqÀØ HgÀÄ. F HgÀ£ÀÄß £ÀªÀÄä gÁdåzÀ “¨ÉÆA¨Á¬Ä” J£ÀÄßªÀgÀÄ. EArAiÀiÁzÀ zÉÆqÀØ £ÀUÀgÀUÀ¼À°è EzÀÆ MAzÀÄ. F HgÀ£ÀÄß £ÉÆÃqÀ®Ä d£ÀgÀÄ ¨ÉÃgÉ ¨ÉÃgÉ gÁdåUÀ½AzÀ, ¨ÉÃgÉ ¨ÉÃgÉ HgÀÄUÀ½AzÀ §gÀÄªÀgÀÄ. EzÀ®èzÉÃ £ÀªÀÄä gÁdåzÀ°ègÀÄªÀ ¨ÉÃ®Ægï, eÉÆÃUï, £ÀA¢ EªÀÅUÀ¼À£ÀÄß £ÉÆÃqÀ®Ä d£ÀgÀÄ §gÀÄªÀgÀÄ. F £Ár£À°è gÉÃµÉäAiÀÄ£ÀÆß ¨É¼ÉAiÀÄÄªÀgÀÄ.

bangʌlu:ru nʌnnʌ ra:dzyʌdʌ ondu doddʌ u:ru i u:rʌnnu

. .

nʌmmʌ ra:dzyʌdʌ bombai ennuvʌru. indija:dʌ doddʌ

¬ ¬ ¬ . .

nʌgʌragnlʌlli idu ondu i: u:rʌnnu no:dnʃu dznnʌru

. ¬ ¬ .

be:re be: re ra:dzjʌgʌlindʌ be:re be: re u:rugʌlindʌ

¬ ¬

bʌrnvʌru. idʌlʌde nʌmmʌ ra:dzjadʌlliruvʌ be:lur dzo:g

¬ ¬ ¬

nʌndi i:vugʌlʌnnu no:dʌlu dznnʌru bʌruvʌru. i:

¬ .

ha:dinʌlli re:ʃmejʌnnu belejuvʌru.

Appendix -2

WAB (Picnic - Picture)