Psycho-Acoustical Profile in Normals and Individuals with Auditory Dys-Synchrony

DOCTORAL THESIS

Animesh Barman

Under the Guidance of Prof. Asha Yathiraj

Submitted to the University of Mysore in 2007

CERTIFICATE

This is to certify that the thesis entitled 'Psycho-Acoustical Profile in Normals and Individuals with Auditory Dys-Synchrony' submitted by Animesh Barman, for the Degree of Doctor of Philosophy in Speech and Hearing to the University of Mysore, Mysore, was carried out at the All India Institute of Speech and Hearing, Mysore, under my guidance.

Asha Yathiraj

Place: Mysore

Date: 28/12/07

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DECLARATION

I declare that this thesis entitled 'Psycho-Acoustical Profile in Normals and Individuals with Auditory Dys-Synchrony' which is submitted herewith for the award of the Degree of Doctor of Philosophy in the field of Speech and Hearing to the University of Mysore, Mysore, is the result of work carried out by me at the All India Institute of Speech and Hearing, Mysore, under the guidance of Prof. Asha Yathiraj, Professor of Audiology, All India Institute of Speech and Heraing, Mysore. I further declare that the results of this work have not been previously submitted for any other Degree.

Place: Mysore

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Date: 28/12/07

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1. INTRODUCTION

The ear is one of the most important links in the speech chain and is essential for communication. All information from the peripheral receptor organ (cochlea) is carried to the brain for analysis through afferent auditory pathways. Deficits anywhere in these structures and pathways will lead to a hearing impairment. The major effect of hearing impairment is loss of some or all of the important acoustic cues which in turn affect speech perception and communication (Denes & Pinson, 1973). Even a slight hearing impairment can affect fine-grained auditory discrimination. Thus, if a child cannot hear phonetic distinctions, he or she is at significant risk for language learning problems (Leonard, 1991). Even elderly persons with hearing loss typically complain of their inability to understand speech.

The impact of hearing loss has been found to vary depending on the type of hearing loss. A conductive hearing loss has been thought to just attenuate the incoming signal (Moore, 2003). Individuals with a conductive hearing loss usually do not have problem in discriminating or understanding speech if spoken to loudly (Roeser, Valente & Hosford-Dunn, 2000). However, a long standing conductive hearing loss, especially in children, can affect the development of the central auditory pathway. It has been reported that sensory stimulation of the auditory centres of the brain is critically important and influences the actual organization of auditory brain pathways (Boothroyd, 1997; Chermak & Musiek, 1997; Musiek & Berge, 1998).

A sensory neural hearing loss is reported to have a severe impact on the perception of auditory stimuli. Individuals with sensorineural hearing loss, along with reduced hearing sensitivity, often have difficulty in understanding speech, especially in noisy environments. The particular difficulties experienced by the sufferer depend on

which part of the system is affected i.e. whether the cochlea or retro-cochlear (Moore, 2003). Lesions affecting the auditory nerve or the cochlear nucleus are generally associated more with loss of sensitivity than lesion in more rostral areas of the central auditory nervous system (CANS). Difficulty in understanding speech, especially in the presence of noise are associated with CANS disorders (Musiek, Baran & Pinheiro, 1994). Most often clients with central auditory lesions have been found not to have a loss of hearing sensitivity. They often complain of difficulty in understanding speech in noisy situation, difficulty following complex auditory directions, poor utilization of prosodic cues, difficulty localizing sounds sources and marked decrease in the appreciation of music (Musiek et al., 1994).

Auditory neuropathy, more recently referred to as auditory dys-synchrony, by Berlin, Hood and Ross (2001) is known to be a retro-outer-hair-cell disorder, where the patient displays characteristics consistent with normal outer hair cell function and abnormal function at the level of the VIII nerve (Starr, Picton, Sininger, Hood & Berlin, 1996; Berlin et al., 2001). It has been observed at any age group (Sininger & Oba, 2001). The proposed etiologies of auditory neuropathy have been diverse and include neonatal hyperbilirubinemia (Stein et al., 1996), severe illness during the neonatal period (Deltenere, Mansbach, Bozet, Clercx & Hecox, 1997) and a part of a generalized metabolic toxic or inflammatory neuropathy (Berlin, Hood, Cecola, Jackson & Szabo, 1993; Starr et al., 1996). Some patients may also have an accompanying generalized neuropathy affecting other cranial and or peripheral nerves (Starr et al., 1996). The other etiologies, which have been reported to result in auditory neuropathies include genetic factors as in hereditary sensory motor neuropathy (Musiek, Weider & Muller, 1982;

Raglan, Prasher, Trinder & Rudge, 1987), hereditary sensory and autonomic neuropathy (Hallpike, Harriman & Wells, 1980; Wright & Dyck, 1995), and the neuropathy accompanying Friedrich's ataxia (Cassandro, Mosca, Sequino, De Falco & Campanella, 1986). The demyelinating neuropathy of the Guillian-Barre syndromes have been found to include auditory neuropathy according to Rooper and Chiappa (1986). The histological findings from animal studies (Salvi, Wang, Ding, Stecker & Arnold, 1999; Harrison, 1998) showed that auditory neuropathy could arise from scattered inner hair cell loss.

It was almost 30 years ago that audiologist began to report about patients with absent auditory brainstem response (ABR), but normal or near normal audiograms. Davis and Hirsh (1979), Worthington and Peters (1980) and Lenhardt (1981) were among the first few to report of clients with absent ABRs and normal or near normal hearing threshold. Kraus, Ozdamar, Stein and Reed (1984) also reported of such audiological findings. Starr et al. (1991) carried out several psycho-acoustical tests in a single case with absent ABR and presence of OAE without assigning any name to the condition. The disorder was eventually named auditory neuropathy by Starr et al. (1996) and renamed as Auditory Neuropathy/Dys-Synchrony (AN/AD) by Berlin et al. (2001).

The nature of the problem has been usually reported to be progressive (Sininger, Hood, Starr, Berlin, & Picton, 1995; Starr et al., 1996; Deltenere et al., 1997; Rance et al., 1999). A majority of these patients have been found to have bilateral low frequency sensorineural hearing loss (Starr et al., 1996). Speech identification scores were noted to be generally poorer both in quiet as well as in noise when compared to those obtained from patients with comparable pure tone loss due to cochlear damage (Starr et al., 1996;

Hood, 1998). The auditory neuropathy has been found to occur either in isolation or as part of a generalized neuropathic process (Starr et al., 1996; Hood, 1998). However, Sheykholeslami, Kaga, Murofushi and Hughes (2000) reported that in patients with isolated auditory neuropathy, the vestibular branch of the VIII nerve and its innervated structures may also be affected, leading to dys-equilibrium.

The characteristics that have been observed in clinical audiological tests were normal otoacoustic emissions (OAEs) and absent or severely abnormal ABRs (Hood, 1998). The ABR was noted to be absent or severely abnormal, not corresponding to the subject's audiometric threshold (Starr et al., 1996; Deltenere et al., 1997; Hood, 1998; Berlin, 1999; Rance et al., 1999).

The site of lesion based on the general clinical findings in these patients have been reported to be the auditory nerve or brainstem pathways which could result in abnormality in the acoustic reflex, the auditory brainstem response and efferent suppression of otoacoustic emissions (Starr et al., 1996; Hood, 1998; Berlin, 1999; Rance et al., 1999). However, cochlear responses that involve outer hair cell function, which includes OAEs and cochlear microphonics, were reported to be normal (Starr et al., 1996; Berlin, 1999; Rance et al., 1999). The presence of OAEs has been considered to indicate the functioning of the outer hair cells and that the abnormality could be at the level of inner hair cells and their dendrites, the spiral ganglion, eighth nerve fibers or a combination of any of the above (Hood, 1998).

Further, Starr et al. (1996) earlier reported that auditory dys-synchrony may affect the functioning of the inner hair cells, synaptic junctions between the inner hair cells and auditory nerve, or the auditory nerve itself. This has been found to result in

some degree of peripheral hearing loss. In such cases, it was noted by Chermak and Musiek (1997) that the results of tests such as dichotic CV would be affected more than cortical auditory evoked potentials such as late latency responses (LLR). They observed that this could be due to the greater complexity of intensity and frequency interactions, especially over a restricted range, thus leading to greater peripheral influence. Starr et al. (1996) reported that the cortical auditory evoked potentials might be absent in some individuals with AN. In some case these potentials were found to be normal. However, speech elicited cortical potentials (LLR and MMN) were reported to be present (Kraus et al., 2000).

Rance, Cone-Wesson, Wunderlich and Dowell (2002) observed that a subgroup of children with auditory dys-synchrony, who had recordable cortical evoked potentials, performed well on an open-set speech identification task and derived significant benefit from amplification. In contrast, subjects who had no recordable cortical evoked potentials performed poorly on speech identification tasks.

Persons with auditory dys-synchrony have been reported to often complain that they hear, but that they do not understand speech. Furthermore, their problem in understanding speech is aggravated under listening situations where noise and reverberation is present to a greater degree than usual. The speech understanding deficits of individuals with auditory dys-synchrony have been found to be disproportionate to their degree of hearing loss unlike those with cochlear hearing loss (Starr et al., 1996; Li, Wang, Chen & Liang, 2005).

It has been shown that performance of individuals with auditory dys-synchrony is similar to normal hearing individuals on perception of intensity related information such

as sound localization based on interaural level difference and loudness discrimination (Zeng, Kong, Michalewski, & Starr, 2005). In contrast the frequency discrimination ability of patients with auditory dys-synchrony has been noted to be significantly poorer compared to that of normal hearing subjects, particularly at low frequencies (Starr et al., 1991; Starr et al., 1996; Rance, McKay & Grayden, 2004; Zeng et al., 2005).

In addition, Zeng, Oba, Garde, Sininger and Starr (1999) and Zeng et al. (2005) noted that individuals with AN/AD exhibited severe problems in temporal perception like temporal integration, gap detection, temporal modulation detection, backward and forward masking, and sound localization using interaural time differences. Individuals with auditory dys-synchrony were also found to have difficulty in detecting short duration acoustic signals, but not longer ones.

Further, Zeng et al. (2005) found improvement in thresholds with increase in signal duration in individuals with auditory dys-synchrony as was the case with normal hearing individuals. However, the slope of the integration function was seen to be slightly elevated in individuals with auditory dys-synchrony compared to normal hearing subjects (Starr et al., 1991 & Zeng et al., 2005). In contrast, Zeng et al. (1999, 2001) reported of normal or near normal temporal integration functions in individuals with auditory dys-synchrony. Abnormal gap detection (identification of silent period embedded within a noise burst) has been reported in individuals with auditory dys-synchrony (Zeng et al., 1999, 2001 & Zeng et al., 2005).

Rance et al. (2004) also reported poor performance on tasks involving timing cues in a group of children with auditory dys-synchrony. Specifically, processing abnormalities on these temporal tasks were significantly correlated with speech

identification scores. Rance et al. attributed these disproportionate speech identification scores to deficits in the processing of temporal information.

The studies reported in the literature indicate that impetus has been paid to determining the basic audiological findings or responses to electrophysiological measures. These studies bring to light the diverse audiological findings have been reported in individuals with AD, within a study as well as between studies. There is need to tap the exact perceptual deficit in clients with AD. This information would be of immense help while deciding the line of treatment that should be recommended for them. The information in literature regarding the psycho-physical perception is sparse, making it essentials to study this aspect.

1.1. Need for the study

Need to study frequency, intensity and temporal perception

The multiple cues in speech have been considered its most remarkable quality. Ainsworth and Greenberg (2006) reported that not only are its spectrum, pitch and amplitude constantly changing, but the variations in these properties occur largely independent of each other. The spectrum of speech signal is seen to change over time, sometimes slowly, often quickly (Kewley-Port & Neel, 2006). van Wieringen and Pols (1998) observed that these dynamic properties provide information essential for distinguishing among phonemes. It is also important to understanding how speech is processed in the auditory system over time, not only in terms of spectral changes, but also in terms of energy changes. Such energy fluctuations have been considered as important as spectral variations (Kollmeier & Koch, 1994; Shannon, Zeng, Kamath, Wygonski &

Ekelid, 1995). This is considered to provide crucial information for segmentation of speech, particularly at the syllabic level (Shastri, Chang & Greenberg, 1999).

However, less is known about the dynamics of speech stimuli. This is partly because of the difficulty to examining perceptual cues of sounds that consist of three covarying dimensions (frequency, duration and rate of frequency change), and partly because other physical parameters, such as amplitude and bandwidth, are difficult to control in dynamic sounds (van Wieringen & Pols, 2006). It is difficult to isolate or manipulate specific properties of speech signals. Hence, under such conditions it is necessary to make use of non-speech analogs such as tone stimuli.

Need to study psycho-acoustical tests in the Indian population

It has been established that auditory perceptual abilities can differ from race to race. Beasley and Beasley (1973) reported that there are differences in the auditory reassembly ability of black and white children. The hearing acuity was found to be better in Negros than whites (Post, 1964). In contrast, Berlin and Dill (1967) reported that the Negro children were poorer listeners than the lower class white children. Differences in auditory discrimination ability in black and white children have also been reported by Shirns, Ruder and Tew (1973). Whitehead, Kamal, Lonsbury-Martin and Martin (1993) noted that the presence of spontaneous otoacoustic emissions were more in Negros than the Asians. All these studies support the fact that there could be some differences in physiological and perceptual abilities in different races. In addition, it is always advisable to compare the data of a clinical population with that of normal individuals from the same geographical location. Thus, it is necessary to investigate the fine grained

discrimination ability of normal hearing individuals and compare the findings with that reported in western countries.

Further, only a relatively few researchers in India have made attempts to study the psycho-physical discriminative ability for intensity (Iyengar, 2000), frequency (Kamath, 1989), duration (Shylaja, 2005) and gap detection threshold (Shivaprakash, 2003) in the Indian population. The findings of these studies are not comparable due to the variations in method used. Turner, Zwislocki and Filion (1989) compared the different methods to obtained difference limen (DL) and reported differences in values. Hence, there is a need for a database, collected from normal hearing individuals in India, using a standard method. This would serve as a reference against which data from subjects with auditory dys-synchrony could be compared. This would also serve as a reference for further research on other clinical populations who may exhibit auditory perception deficits.

Need to study psycho-acoustical performance in individuals with Auditory Dys-synchrony

Studies published in literature show that frequency, intensity and temporal cues are coded in the auditory nerve. Liberman and Kiang (1978) reported that a majority of auditory nerve fibers have thresholds in the bottom 10-15 dB range. They have also shown that there are a significant proportion of fibers with higher threshold. The phase locking, that was seen in the auditory nerve fibers for stimuli below 5 kHz was present even at higher intensities, inspite of a saturation of the firing rate. Further, single auditory nerve fibers have been found to behave as band-pass filters with an asymmetric filter shape. The frequency selectivity has been found to be similar to that of the basilar membrane and the hair cells (Russell & Sellick, 1978; Sellick, Patuzzi, & Johnstone

1982). There is a need to determine whether individuals with auditory dys-synchrony show poorer performance in finer discrimination of frequency, intensity and temporal cues, as the properties of these parameters change the neural output of the afferent fibers that innervate the inner haircells (Yost, 2000). It is necessary to assess the ability to discriminate frequency, intensity and temporal cues in these individuals, as their major problem has been found to lie in the auditory nerve. Such an assessment would bring to light whether individuals with auditory dys-synchrony are impaired in the fine grained discrimination of frequency, intensity and /or temporal information. This information would help to determine the acoustical parameters that pose greater problem for these individuals.

Studies on a large group of individuals having AD are few. This is essential since Starr et al. (1991), Starr et al. (1996), Zeng et al. (2001) and Zeng et al. (2005) have observed high inter-subject variability in individuals with AD. Also Rance et al. (2004) carried out a series of psycho-acoustical tests on children and found that the responses were highly subjective. Thus, it is essential to carry out such studies on a large population to validate the data and confirm the results obtained from earlier studies.

Need to study psycho-acoustical tests at different sensation level

Several researchers reported that the just noticeable difference (JND) values obtained at different sensation levels are not the same. The researchers observed that difference limen (DL) seems to decrease at higher sensation levels (Plomp, 1964b; Jesteadt, Wier, Green, 1977; Wier, Jesteadt & Green, 1977; Starr et al., 1991; Starr et al., 1996; Zeng et al., 2001 & Zeng et al., 2005). Investigators have also indicated that

subjects with cochlear impairment show slightly elevated gap detection thresholds at moderate sound levels but reach the normal range of values higher sound levels (Florentine & Buus, 1984; Fitzgibbons & Gordon-Salant, 1987; Moore & Glasberg, 1988). Thus, DL obtained at different sensation levels might provide information as to whether individuals with auditory neuropathy function differently at softer and louder levels. This information in-turn would help making judgements about how they would perform in a real-life situation when they have to respond to softer and louder signals.

Need to study perception in a sound field condition

Most of the experiments reported in literature have been carried out in a monaural condition under headphones (Plomp, 1964b; Jesteadt et al., 1977; Wier, et al., 1977). However, in a day-to-day situation, listening does not take place under phones, or in a monaural condition. It has been reported that there is an improvement in speech intelligibility in sound field conditions, especially in the presence of noise (Plomp, 1976; Plomp & Mimpen, 1981). Shaw (1974) also reported that there is a change in the power spectrum of the signal in the ear canal depending upon the direction of the source. Though the experiments conducted under earphones provide better control of stimuli, it is difficult to generalize the results to a binaural listening condition in the real world. Unlike studies done under earphones, research done in a sound field condition would be more practical. Butler (1975) observed that the external and the middle ear could also influence the auditory discrimination. Sound field-testing would also allow researchers to test, without altering the physiology of the external ear.

Further, to improve the speech perception ability of individuals with AD in quiet and in noise is of great concerned. Auditory training under sound field might help individuals with auditory dys-synchrony to improve their speech perception ability. This is based on the information that normal hearing individuals perform better in sound field than under head phones, which could be due to a squelch effect (Dunn, Tyler & Witt, 2005). Thus, it might improve their communication skills, especially in those who have lesser degree of hearing loss. Hence, there is a need to investigate the ability of individuals with AD to discriminate frequency, intensity and temporal aspects of acoustic signals in a sound field condition. This information could serve as a baseline performance for those who undergo training to improve their perception in a real-life situation.

If the information regarding the specific perceptual problems of individuals with auditory dys-synchrony is available, then a therapy program can be designed to help them overcome their perceptual difficulties. Without such information, the therapy activities may not focus on the specific perceptual problems faced by these individuals. The therapy would be on a trial and error method and would be ineffective. The information obtained would also help in monitoring the progress during a therapy program and provide a feedback to the clients.

Thus, it is a challenging task for an audiologist to rehabilitate individuals with auditory dys-synchrony, especially since their ability to understand speech is poor. Hence, it is essential to know whether they exhibit deficits in finer discrimination of different acoustical parameters, which are essential to understand speech. It would also help in knowing the exact perceptual deficit exhibited by an individual to design an

effective therapy plan and also monitor progress during and after training. Keeping in view the above needs, the objectives of the study would be as mentioned in the next section.

1.2. Objectives of the study

The main objectives of this study were:

- To investigate the ability of normal hearing individuals and individuals with auditory dys-synchrony to carry out the following fine-grained behavioural discrimination for:
    - Frequency at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz at 10 and 40 dB SL,
    - Intensity at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz at 10 and 40 dB SL,
    - Duration at 1000 Hz for two anchor duration stimuli of 50 ms and 500 ms at 10 and 40 dB SL, and
    - Gap detection for white noise at 10 and 40 dB SL.

- To compare the fine grained behavioural discrimination abilities of a group of normal hearing individuals with individuals having auditory dys-synchrony with respect to:
    - Frequency at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz at 10 and 40 dB SL,
    - Intensity at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz at 10 and 40 dB SL,
    - Duration at 1000 Hz for two anchor duration stimuli of 50 ms and 500 ms at 10 and 40 dB SL, and
    - Gap detection for white noise at 10 and 40 dB SL.

- To compare the temporal integration of a group of normal hearing individuals with individuals having auditory dys-synchrony.
- To compare the MLD obtained from a group of normal hearing individuals with individuals having auditory dys-synchrony.
- To know which acoustic parameter (frequency, intensity and temporal) is more affected in individuals with auditory dys-synchrony.

Prior to studying these objectives, a review of literature was carried out regarding auditory dys-synchrony. Information on studies related to different psycho-physical tests carried out in normal hearing individuals and individuals having auditory dys-synchrony were also gathered.

2. REVIEW OF LITERATURE

The term auditory neuropathy (AN)/auditory dys-synchrony (AD) has been used to describe a type of hearing impairment due to a dysfunction of the auditory nerve in the presence of preserved cochlear outer hair cell functioning (Starr, Picton, Sininger, Hood & Berlin, 1996; Berlin, Hood & Ross, 2001). This clinical entity was first described in detail by Starr et al. (1991) in a single case study where the problem was attributed to the involvement of the auditory nerve dysfunction. Subsequently ten subjects with similar symptoms were reported by Starr et al. (1996) and they coined the term 'auditory neuropathy', as eight of the participants had accompanying peripheral neuropathies. However, later Berlin et al. (2001) felt that the term auditory dys-synchrony was more appropriate for this disorder due to two primary reasons: that the auditory nerve may not be the only part affected; and that the term auditory neuropathy may lead clinicians not to consider cochlear implants as a management option. Ensuring research highlighted that cochlear implants are beneficial in many individuals with auditory neuropathy (Berlin, Hood, Morlet, Rose & Brashears, 2003; Berlin, Morlet & Hood, 2003; Peterson et al., 2003).

It has been reported by Sininger and Oba (2001) that to diagnose an individual as having auditory dys-synchrony, the client must have the three clinical features. These include evidence of poor auditory functioning, where the patient must have difficulty in understanding speech at least in some situations regardless of pure tone hearing thresholds; evidence of poor auditory neural functioning, where the patient must have abnormal or absent auditory brainstem responses and elevated/absent acoustic auditory

brainstem reflexes such as stapedial reflex and medial olivocochlear bundle reflex; and evidence of normal outer hair cell function where the patients must show presence of either cochlear microphonics or otoacoustic emissions.

Similarly, Kraus (2001) also provided a clinical definition of auditory neuropathy. She considered the condition to be present if the client had normal otoacoustic emissions and cochlear microphonic, but absent or severely abnormal ABR.

Interest in the area of auditory neuropathy has gained momentum since 1996 after Starr et al. first coined the term and described the typical paradoxical findings. The focus of the research has been to determine the aetiology, audiological characteristics and psycho-acoustical findings.

2.1. Demographics of Auditory Neuropathy

Information regarding the age of onset, gender specificity and incidence and prevalence of the auditory neuropathy have been reported in literature. This reviewed information is discussed below.

2.1.1. Age of Onset

Considerable discrepancies regarding the age of onset of auditory dys-synchrony has been reported in the literature. Sininger and Oba (2001) provided a systematic report regarding the onset of AD. They observed that the mean age of onset of auditory dys-synchrony symptoms was nine years in a group of 59 patients. The onset of such symptom was found to occur at any age between births to 60 years of age. Seventy-five percent of the individuals were below ten years of age when the symptoms were first

seen, with the largest group showing the onset before two years of age. It has been observed that only one out of four cases with auditory dys-synchrony might be older than 10 years of age (Starr, Sininger & Pratt, 2000). Kumar and Jayaram (2006) reported the mean age of onset in the Indian population to be around 16 years. They observed that the onset of auditory dys-synchrony was from one year to 31 years, with the majority of subjects having an onset at around 24 years of age.

There are equivocal reports regarding the age of onset of auditory dys-synchrony. However, the majority of the studies indicate that the onset of auditory dys-synchrony could be during early childhood or adulthood.

2.1.2. Gender specificity

Sininger and Oba (2001) reported that 55% of their subjects with auditory neuropathy were males and 45% were females. Starr (2001) also noticed a similar trend. He found that 54% of his subjects with auditory neuropathy were males and 46% were females. In contrast, Kumar and Jayaram (2006) reported a strong gender bias, with the female to male ratio being of 2:1 in the 61 clients they studied. Except for the study reported by Kumar and Jayaram, the finding reported in literature show no specific gender bias. Both males and females are almost equally affected.

2.1.3. Incidence and Prevalence

The incidence and prevalence of auditory dys-synchrony is still not clearly known. Much before the term auditory neuropathy/dys-synchrony was coined, several authors reported of cases with ABR being absent and OAEs being present. Davis and

Hirsh (1979) could be the first to observe such a paradox with an incidence of 0.5% in their clinical population. Kraus, Ozdamar, Stein and Reed (1984) reported that 14% of their cases had absent ABR and that 1.3% of their total population of 543 children evaluated for hearing loss had evidence of tests results that could have been present in cases with auditory neuropathy.

Berlin et al. (1998) reported that five out of their 60 (12%) children who were diagnosed as deaf had auditory neuropathy. Similarly, Stein et al., (1996) identified four infants with auditory dys-synchrony among 100 infants at-risk for hearing impairment. Psarommatis, Tsakanikos, Kontorgianni, Ntouniadakis and Apostolopoulos (1997) found two infants with AD out of 102 infants with risk factors. From a group of 5199 children who were at-risk for hearing loss and auditory dys-synchrony, Rance et al. (1999) found the prevalence to be one in 433 (0.23%). However, among children with hearing impairment the prevalence was one in 109 (11.01%). Berlin et al. (2000) reported that 87 children had AD out of 1000 children with hearing loss (8.7%). Among school-going children with hearing loss, the frequency of occurrence of AD was found to be 1.78% by Tang, McPherson, Yuen, Wong and Lee (2004). Kumar and Jayaram (2006) reported an incidence rate of 1 in 348 (0.29%) within a population with hearing-impairment. When they considered only individuals with sensori-neural hearing loss, the prevalence increased to 1 in 184 (0.54%).

From the studies reported in literature, it is evident that the incidence/prevalence of individuals with AD could be as low as 0.29% and as high as 12%. Despite the variation across the studies, it can be construed that the incidence and the prevalence of AD is not very high.

2.2. Aetiology

Much after reports of the audiological findings in clients with AN, information regarding the aetiology of the condition has been published. A number of different aetiologies have been associated with auditory neuropathy/dys-synchrony. These conditions can be broadly categorized as *genetic factors* (Starr et al., 2003; Wang, Gu, Han & Yang, 2003; Wang et al., 2005), *neonatal risk factors* (Deltenre, Mansbach, Bozet, Clercx & Hecox, 1997; Rance et al., 1999; Simmons & Beauchaine, 2000; Akman et al., 2004), and different *syndromic conditions* and *peripheral neuropathies* (Starr et al., 1996; Jutras, Russell, Hurteau & Chapdelaine, 2003). However, approximately 50% of the patients have been found to have no defined aetiology (Starr et al., 2000). The aetiologies reported in literature are further discussed below.

2.2.1. Genetic and Syndromic conditions

Auditory neuropathy/dys-synchrony has often been found to occur as a part of a generalized neuropathic disorder. It has been reported with syndromes that affect the central nervous system like Charcot-Marie-Tooth syndrome and hereditary sensory motor neuropathy (Starr et al., 1996; Starr et al., 2003), and Wardenburg's syndrome (Jutras et al., 2003). Hereditary motor and sensory neuropathies (HMSN) such as Charcot-Marie-Tooth Syndrome (Type I & II) have been reported to make-up a relatively high proportion of the adult clients with AN/AD cases reported to-date (Starr et al.,1996; Butinar et al., 1999; Leonardis et al., 2000; Sininger & Oba, 2001; Starr et al., 2003).

Sininger and Oba (2001) reported that 8 of their 13 patients with symptoms of AN/AD at an age of 10 years or above were confirmed HMSN sufferers. Charcot-Marie-Tooth syndrome, a form of HMSN has been reported to be a genetic disorder, which involves the degeneration of the myelin sheaths. It has been related to an abnormality in the peripheral myelin protein 22 [PMP-22] on chromosome 17p 11.2 (Kovach, Lin & Boyajiev, 1999) or a mutation of MPZ gene (Starr et al., 2003). Loss of axons of the distal portions of the peripheral nerves has also been reported with this condition (Chance & Fishbeck, 1994; Ouvrier, 1996). Auditory brainstem responses have been found to be absent or grossly abnormal in patients with Charcot-Marie-Tooth syndrome (Cassandro, Mosca, Sequino, De Falco & Campanella, 1986). Histopathological results have shown evidence of cochlear hair cell survival while there is a loss of cochlear spiral ganglion cells and evidence of demyelinating processes in the VIII nerve (Nadol, 2001).

Hereditary motor and sensory neuropathies have also been linked to auditory neuropathy/dys-synchrony in studies involving Slovene, Italian and Bulgarian Gypsy families (Butinar et al., 1999; Leonardis et al., 2000). The autosomal recessive condition, which in these cases produced both myelin and axonal damage, was mapped to the long arm of chromosome 8 (8q24). The disease process with this form of neuropathy tended to produce severe, progressive motor disabilities in early childhood and auditory pathway affects in adolescence.

Another inherited disease that is relatively commonly noted to be associated with auditory neuropathy/dys-synchrony is Friedreich's Ataxia. Four cases of this autosomal recessive condition were described by Sininger and Oba (2001) among the cases seen by them. Auditory brainstem response assessments in patients with Friedreich's Ataxia have

typically shown either complete response absence (Cassandro et al., 1986) or the presence of wave I and absent later responses (Jabbari, Schwartz, MacNeil & Coker, 1983). Histopathology reports by Spoendlin (1974) have indicated that the cochlear neurons and spiral ganglion cells are affected in Friedrich's Ataxia whereas the cochlear structures (organ of corti and hair cells) are unimpaired. Gait ataxia and chorea are also reported to be associated with AN/AD by Starr et al. (1996).

AN/AD has been observed to be a part of other genetic disorders. These include Ehlers-Danlos syndrome, an autosomal dominant connective tissue condition related to serious vascular abnormalities (Sininger & Oba, 2001) and Stevens-Johnson syndrome which is a rare cutaneous disease typically triggered by drug therapy (Doyle, Sininger & Starr, 1998). Auditory neuropathy/dys-synchrony has also been reported to be associated with syndromes affecting the immune system like Guillain-Barre syndrome and mitochondrial enzymes (Deltenre et al., 1997; Corley & Crabbe, 1999).

Kim et al. (2004) also reported an autosomal dominance pattern of inheritance of dys-synchrony, mapped to a novel locus called auditory neuropathy dominant 1 (AUNA1) on chromosome 13q 14-21 in a large kindred spanning 7 generations. They reported that the hearing impairment in the initial stage expressed as a disorder of the auditory nerve function in presence of normal outer hair cell activity. As the hearing loss progressed, outer hair cell activity (as measured from otoacoustic emissions) in the high and mid frequency cochlear region became impaired. Finally, affected family members in the 5^{th} and 6^{th} decade were completely deaf and lost all outer hair cell and neural function.

Starr et al. (2003) identified the presence of missense mutation in the MPZ gene in three individuals with auditory dys-synchrony. This A to C mutation at position 434, resulted in an amino acid change from tyrosin to scrine at codon 145. Starr et al. did not observe this mutation in the 100 control chromosomes of unaffected individuals. Pathological examination of the cochlea in one of the family members with this mutation revealed marked loss of auditory ganglion cells and auditory nerve fibres within the cochlea. However, it was noted that the outer hair cells and inner hair cells were normal in number and appearance.

A study on 72 members belonging to large kindred with hearing impairment was carried out by Starr et al. (2004). They reported that auditory dys-synchrony was inherited in autosomal dominant pattern with 100% penetrance in the family studied. Consanguineous marriage did not increase the risk or severity of the phenotype in any of the offsprings. They also reported of a marked improvement in auditory function in three affected family members after a cochlear implant surgery, with the recovery of electrically evoked ABR, auditory temporal processing and speech perception skills.

Further, Wang et al. (2005) reported of mutations in the mitochondrial DNA, particularly T-to-C transition at 1095 (T1095C) in the 12srRNA gene. This was noticed in a Chinese family with auditory dys-synchrony.

The studies on genes in individuals with AD provide evidence to show that the condition could be genetic based. It has been found to either be associated with other syndromic condition or occur in isolation.

2.2.2. Neonatal illness

The most commonly reported neonatal conditions associated with auditory neuropathy/dys-synchrony are anoxia and hyperbilirubinemia (Stein et al., 1996; Berlin, Bordelon, Hurley, Hood & Parkins, 1997; Deltenre et al., 1999; Rance et al., 1999; Simmons & Beauchaine, 2000; Starr et al., 2000; Sininger & Oba, 2001; Franck, Rainey, Montoya & Gerdes, 2002; Madden, Rutter, Hilbert, Greinwald Jr., & Daniel, 2002; Dunkley, Farnsworth, Mason, Dodd & Gibbin, 2003). Over 50% with early-onset AN/AD reported in the literature so far, have recorded one or both of these conditions in their neonatal histories.

Severe hyperbilirubinemia has often been noted to result in hearing impairment. Some of these report have specifically noted that it causes auditory dyd-synchrony (Deltenre et al., 1997; Berlin et al. 1998; Rance et al., 1999; Simmons & Beauchaine, 2000; Akman et al., 2004; Rance, McKay & Grayden, 2004). Thirteen of the 20 children with auditory neuropathy/dys-synchrony, described by Rance et al. (1999), presented with serious neonatal health concerns. Rance et al. (1999) reported that 10 of the 20 children with auditory dys-synchrony had bilirubin concentration levels more than 350 µmol/l. Subsequent findings presented by Sininger and Oba (2001) confirmed this result. Approximately 80% of their 59 cases with AD/AN, who had an onset of less than two years, presented with neonatal and/or familial risk factors. Further, Sininger and Oba found that almost half of their infant cases had both genetic and neonatal health factors, and suggested that some children may be pre-disposed towards developing auditory neuropathy/dys-synchrony if they suffer some form of neonatal insult.

Akman et al. (2004) also reported that 7 of their 19 babies with hyperbilirubinemia showed indications of auditory dys-synchrony. Of these 7 infants, 6 had serum bilirubin values more than 25mg/dl. Babies with hyperbilirubinemia, who passed ABR testing, had significantly less serum neuron specific enolase. This was in comparison with babies who were diagnosed as auditory dys-synchrony. Auditory dys-synchrony has also been reported in other neonatal risk factors such as anoxia, hypoxia, pneumonia, neonatal meningitis, hydrocephalus and metabolic abnormalities (Deltenre et al., 1997; Doyle et al., 1998; Rance et al., 1999; Rance et al., 2004).

Even short term episodes of hyperbilirubinemia have been shown to result in both temporary and permanent evoked potential abnormalities, including elevated ABR thresholds (Hung, 1989) and prolonged ABR wave (I-V) latencies (Nakamura et al., 1985; Tan, Skurr & Yip, 1992). This suggests that both the peripheral and central auditory systems are vulnerable to bilirubin insult.

Infection related causes of auditory neuropathy/dys-synchrony other than hyperbilirubinemia have also been reported in a small but significant number of cases reported in literature. Starr et al. (2000) estimated that post-viral infectious processes were involved in 10% of the 67 patients from their AN/AD database. Specific etiologic details were not presented for these subjects, but other studies have reported that both mumps (Prieve, Gorga & Neely, 1991) and meningitis (Sininger, Hood, Starr, Berlin & Picton, 1995; Rance et al., 1999) can be associated with the auditory neuropathy/dys-synchrony.

It can be observed from above studies that hyperbilirubinemia and anoxia have been found to be present often in infants and toddler having AN/AD. In addition, with

these risk factors, genetic causes have also been found to coexist in several children. Less commonly, other risk factors such as pneumonia, meningitis, hydrocephalus, mumps and metabolic abnormalities have been reported.

2.3. Pathophysiology of auditory dys-synchrony

A limited number of researchers have studied the pathophysiology of auditory dys-synchrony directly. Indirect information is available through the findings of audiological tests. These studies have indicated that the possible sites of lesion for AD could be the cochlear inner hair cells (IHCs), the synapse between the IHCs and Type 1 auditory nerve fibres, and the auditory nerve itself (Starr et al., 1996; Rance et al., 1999; Amatuzzi et al., 2001). Details regarding the different possible sites are discussed in greater depth in the below given section.

2.3.1. Inner Hair Cell Loss

Information regarding the functioning of the inner hair cells in live individuals with AD has been speculated using auditory brainstem responses. The earlier ABR waves (including Wave I which represents the first action potential in the auditory nerve) are reported to be absent in cases with auditory dys-synchrony. Also, a specific inner hair cell abnormality was reported to result in a decrement of the entire ABR complex, with the preservation of outer hair cell responses (Rance, 2005).

Though there are no suitable diagnostic tests to check the integrity of the inner hair cell function in live patients, biological precedents for selective inner hair cell loss in the Bronx Waltzer mouse (Lenoir & Pujol, 1984; Schrott, Stephan & Spoendlin, 1989)

and the Beethoven mouse models (Bussoli, Kelly & Steel, 1997) are reported. Furthermore, the auditory neuropathy/dys-synchrony physiologic profile has been chemically induced in animals (chinchillas) treated with antineoplastic agents (carboplatin) that produced selective inner hair cell lesion (Takeno, Harrison, Ibrahim, Wake & Mount, 1994, Wake, Anderson, Takeno, Mount & Harrison, 1996; Liberman, Chesney & Kujawa, 1997; Harrison, 1998; Salvi, Wang, Ding, Stecker & Arnold, 1999). They reported of ABR threshold disruption in these animals that was considered to be due to a diminution in response amplitude (resulting from a reduction in the number of elements contributing to the volume-conducted potential) rather than an increase in firing threshold for the surviving elements. This conclusion was made as single unit responses from the inferior colliculus neurons showed normal response thresholds. These findings suggest a mechanism whereby patients with AN/AD type hearing loss could demonstrate normal or near normal behavioural hearing thresholds, as has been reported in many human cases, in conjunction with severely disordered evoked potential findings.

More recent findings presented by Amatuzzi et al. (2001) have confirmed that selective inner hair cell loss can occur in human subjects. These authors carried out a detailed histological evaluation of fifteen non-survivors from a neonatal intensive care unit and identified two babies with loss of both inner and outer hair cells, two with loss of outer hair cells alone, and three cases with selective inner hair cell loss. Each of the cases with specific inner hair cell loss had been assessed for the presence of ABR before they died which showed no response at screening levels (40 dB nHL). None of these cases showed any evidence of cochlear neuron damage.

Fit with recent animal histological findings, studies suggest that certain types of cochlear insult can have a greater effect on inner than outer hair cell survival (Bohne, 1976; Shirane & Harrison, 1987; Billet, Thorne & Gavin, 1989). The insult was notably those due to prolonged hypoxia.

Thus, directly or indirectly, studies have shown that AD does result in specific loss of inner hair cells. While the direct evidence have been provided by histological evaluations the indirect information has been provided through ABR findings.

2.3.2. The Synapse between Inner Hair Cells and Auditory Nerve Terminals

A disorder at the synapse between the cochlear inner hair cells and Type 1 auditory nerve fibres has also been proposed as a mechanism that could produce symptoms of auditory neuropathy/dys-synchrony (Starr et al., 1991). It has been reported that at the base of the inner hair cell are anatomic structures involved in the storage and release of neurotransmitters. These neurotransmitters have been found to act upon receptor sites in the auditory nerve dendrites and initiate the generation of action potentials. Disorders at the pre-synaptic site have been found to affect the release of neurotransmitters, while disorders at the post-synaptic sites affect the ability of the receptor sites on the auditory nerve dendrite to respond these substances (Starr et al., 2000). Mechanisms by which synaptic disruption might occur in the auditory pathway in human subjects are yet to be determined. However, genetic dysfunction involving disruption of the otoferlin protein, which affects transmitter release, has been found in the IHCs. It has been identified in subjects presenting audiological results observed in individuals with AN/AD (Varga et al., 2003).

The above review suggests that the abnormality seen in individuals with AN/AD could be at the level of synapse between the inner hair cells and the auditory nerve. This could result in abnormal transmission of the neurotransmitters.

2.3.3. Auditory Nerve Abnormality

As the term 'auditory neuropathy' suggests, the affected site in many patients is thought to be the auditory nerve itself. Starr et al. (1996) coined the expression as 8 of their 10 subjects had evidence of other peripheral nerve abnormality in addition to hearing loss. Generalized neuropathic disorders have been indicated in approximately 30-40% of reported cases with AN/AD, in all age groups, and around 80% of patients with symptom onset over the age of 15 years. The site of the disorder affecting the auditory nerve and auditory brainstem in these cases has been found to be the myelin sheath, or the neuron itself.

2.3.3.1. Myelin Disorder

Partial or complete loss of myelin has been reported to have profound effects on the generation and propagation of action potentials within the auditory nerve fibres. Demyelination has been noted to result in an increase in membrane capacitance and a decrease in membrane resistance, leading to a delayed excitation, a reduction in the velocity of action potential propagation and an increase in conduction vulnerability (McDonald & Sears, 1970; Rasminsky & Sears, 1972; Pender & Sears, 1984). Fibres that were demyelinated to differing degrees were observed to conduct neural signals at different speeds, and the synchrony of discharges was found to be affected. While

neurons that were not entirely myelinated were capable of conducting action potentials, they did so with prolonged refractory periods and impaired ability to transmit high frequency pulse trains (McDonald & Sears, 1970; Rasminsky & Sears, 1972; Pender & Sears, 1984). Rasminsky and Sears (1972) found that repetitive activation of demyelinated fibres resulted in a progressive increase in the conduction time of the action potential and lead to an intermittent or total block in their propagation, also termed as conduction block.

Rance (2005) reported that the pathophysiological changes in neural conduction properties associated with demyelination would result in reductions in the temporal synchrony of demyelinized VIII nerve fibres, leading to a significant reduction in the amplitude of the averaged auditory evoked responses. Further, with more advanced lesions, the propagation of the action potential was speculated to become increasingly vulnerable, with the risk of a depolarisation block increasing. This was considered to especially occur for repetitive stimuli used to generate auditory brainstem responses.

Starr, Picton and Kim (2001) reported the result of a biopsy of the sural nerve in 6 patients with auditory dys-synchrony and a concomitant peripheral neuropathy. They observed axonal neuropathy resulting in a loss of large myelinated fibres. Three other subjects had axonal loss with evidence of secondary demyelinization and remyelinization of remaining fibres. In two other subjects, the sural nerve biopsy showed extensive loss of both axons and myelin sheath.

Later in 2003, Starr et al. reported the histopathological findings of the cochlea and auditory nerve in a patient with auditory dys-synchrony. The organ of corti was normal throughout the cochlea except for the apical turn, where a 30% loss of outer hair

cells was found. The inner hair cells were normal throughout the length of the cochlea. There was a profound loss of ganglion cells (> 95%). Only around 1161 and 1548 surviving ganglion cells were found in the right and left ear respectively, against a count of 23,000 for age matched normal individuals. The central auditory nerves adjacent to the cochlear nucleus also showed marked reduction of the number of auditory nerve fibres. The myelin sheath on the surviving auditory nerve fibres was thin indicating incomplete demyelinization.

The demyeliniting neuropathies have been found to slow or block the nerve conduction and produce motor or sensory symptoms distal to the site of demyeilinization. Demyelinated fibres have been noted to be poor conductors of rapid trains of action potential. Normally, high discharge rates were found to occur in response to intense acoustic stimuli, contributing to the reflex activation of middle ear muscles. However, acoustic middle ear muscle reflexes have been typically found to be absent in subjects with auditory dys-synchrony. The mechanism underlying this has been considered responsible for the failure of the auditory nerve fibres to develop sufficiently high discharge rates to activate the motor neurons of the stapedial muscle (Starr et al., 2001).

Another reason for slow or blocked nerve conduction reported in literature could be due to its sensitivity to the physiological temperature. Such temperature sensitive hearing impairment has been reported in individuals with clinical symptoms of auditory dys-synchrony (Starr et al., 1998).

A third reason reported for a conduction block is the sensitive to ephaptic transmission (cross-talk) between fibres, with one active fibre setting off discharges in

adjacent fibres. If this occurs in auditory nerve fibres, Starr et al. (2001) noted that there would be severe distortion in the coding of speech.

2.3.3.2. Axonal Neuropathy

Axonal neuropathies have been reported to reduce the number of neural elements but not directly affect conduction speed. The refractory periods of surviving elements have also been noted to be normal, allowing reasonably unimpaired response to high rate stimuli (Kuwabara et al., 1999). The reported classical signs of axonal neuropathy in the auditory pathway are normal compound action potentials and reduction in the amplitude of the whole nerve action potential (Starr et al., 2001; Rance, 2005). Furthermore, Rance (2005) observed that ABR wave amplitude reduced instead of resulting in an increase in latency or the broadening of these potentials, as noted in myelin related disorders. He also found evoked brainstem responses were absent in most cases having auditory neuropathy/dys-synchrony, making them clinically indistinguishable from myelin related neuropathies. Accurate differentiations between axonal and demyelinating neuropathies have been found possible only if the axon itself was affected in the former condition. Starr et al. (2001) reported that the hallmark of many axonal neuropathies was retrograde degeneration of the distal portion of peripheral nerves. This was observed to happen because of inadequate transport of metabolic substrate and growth factors between the neuronal cell body and distal portion of its axon. Hence, the nerve fibres functioned normally in terms of speed of conduction, though they were reduced in number. This was found to result in relatively normal conduction velocity but reduced amplitudes of compound action potential. The longest fibres, which originated from the apex of the cochlea, were found to be more susceptible to axonal neuropathies. These fibres

mediated the lower frequencies. The shortest fibres, responsible for the middle frequencies, were found to originate from the second half of the first cochlear turn. The fibres having an intermediate length started from the basal part of the cochlea, which mediated high frequencies. Starr et al. (2001) concluded that if the auditory dys-synchrony were to exhibit a dying back pathophysiology, then the mid frequencies would be affected less than the low and high frequencies. These types of the audiograms have been reported to be most common in patients with auditory dys-synchrony (Sininger & Oba, 2001; Kumar & Jayaram, 2006).

It can be concluded that neuropathic disorders of the peripheral nervous system, including the auditory nerve, can result in varying degrees of axon loss and myelin damage. Abnormal function in the auditory system, resulting in the auditory neuropathy/dys-synchrony pattern, may therefore be related to disrupted neural synchrony on account of myelin damage, a reduction in the number of functioning fibres due to axonal loss, or in many cases, a combination of both.

2.4. Clinical Profile of clients with Auditory Dys-Synchrony

The clinical profile of clients having auditory dys-synchrony has been discussed in depth in literature. The studies have described audiological characteristics with reference to the degree and symmetry of hearing loss; audiogram configuration; stability of behavioural threshold and response to specific audiological tests. The unique audiological characteristics of clients with AD are discussed further in section below.

2.4.1. Degree and symmetry of hearing loss

The majority of reports on auditory neuropathy/dys-synchrony published prior to the mid-1990s described subjects having a mild/moderate hearing loss (Davis & Hirsh, 1979; Worthington & Peters, 1980; Lenhardt, 1981; Kraus et al., 1984). The presence of losses of lesser degree may reflect the fact that many of these early patients were only identified as a result of the inconsistency between behavioural and electrophysiological findings. However, recent literature suggests that clients with auditory dys-synchrony present all degrees of hearing loss. Pure-tone thresholds have been found to range anywhere from normal hearing sensitivity to profound hearing loss. It has been observed in the studies reported in the literature that the degree of hearing loss varies across the two ears in individuals with auditory dys-synchrony.

Starr et al. (2000) found 31% of the ears with AN had an average hearing levels of less than 35 dB HL, 39% of the ears had average thresholds 35 and 70 dB HL and 30%, 70 dB HL. Madden et al. (2002), in their group of 18 affected children, also found variation in the degree hearing loss. Six subjects (33%) had audiograms in the normal to mild range, six in the moderate to severe range, and six in the profound hearing loss range.

Sininger and Oba (2001) noted that 82% of their clients with AN had bilateral symmetrical hearing loss, 14% of them had bilateral asymmetrical and only 4% of them had unilateral hearing loss. Starr (2001) also reported that 96% of the AN population had bilateral hearing loss. All the 61 clients with AN reported by Kumar and Jayaram (2006) had bilateral hearing loss. Most of their subjects had symmetrical loss and only a few of them had asymmetrical hearing loss.

Thus, it is evident from the literature that individuals having AN could have pure-tone thresholds ranging from normal hearing levels to levels indicating profound hearing loss. This loss has been found to be symmetrical, asymmetrical or unilateral.

2.4.2. Configuration of the audiogram

Reports on patients with auditory dys-synchrony have described variable configurations of audiogram. Rance et al. (1999) noted that the audiometric configurations varied with the degree of hearing loss. Ears with normal or near normal hearing acuity showed equal sensitivities at all of the frequencies. However, subjects with thresholds in the mild to severe range, had audiograms that showed poorer hearing sensitivity in low and mid frequencies and better auditory thresholds in the high frequencies. Starr et al. (2000), in their database of 67 patients with auditory dys-synchrony, observed flat audiograms in 41% clients, reverse sloping audiogram in 29%, an irregular saw-tooth pattern in 9%, a 'U' shaped audiogram in 5%, and a tent shaped audiogram with a peak usually at 2 kHz in 5%. Only 11% had high frequency sloping hearing loss, which has been found to be the typical pattern of sensory hearing loss. Likewise, Sininger and Oba (2001) reported that overall 43% of their clients had a flat audiometric shape and 28% had reverse sloping loss with higher thresholds for low frequency stimuli than for high frequency stimuli. However, Kumar and Jayaram (2006) observed that around 42% of their participants with AN had peaked audiograms (sharp peak at a single frequency with worsening of thresholds at immediately adjacent frequencies). The clients with peaked audiograms demonstrated better hearing thresholds

at 2 kHz. Only 5% of the individuals demonstrated a sloping hearing loss among the 61 individuals they studied.

The peaked or reverse sloping audiogram that has been observed in individuals with auditory dys-synchrony has been considered as evidence that the underlying aetiology of the hearing loss in auditory dys-synchrony is neural and not cochlear (Starr et al., 2001). They made this observation based on the fact that the laws of the basilar membrane mechanisms do not provide a viable explanation for significant loss in the low frequencies. Frequent occurrence of peaked audiograms might be due to anatomico-physiological make-up of the auditory nerve. The longest fibres, rising from the apex of the cochlea and mediating low frequencies, have been found to be most susceptible to pathology. The shortest fibres, rising from the second half of the first cochlear turn were found to mediate middle frequencies. The fibres originating from the basal parts of the cochlea, with an intermediate length, were observed to be responsible for high frequencies. Due to the placement of the nerve fibres, the mid frequencies were less likely to be affected than the low and high frequencies.

Thus, individuals with AN most commonly exhibit reverse sloping, flat or peak type audiogram. High frequency or sloping hearing loss have been reported to occur more rarely in these clients.

2.4.3. Stability of behavioural threshold

Fluctuation of hearing sensitivity has been observed to be a common feature in clients with auditory neuropathy. Rance et al. (1999) reported that five out of their 14 children with AN, for whom repeated measures were available, showed significant

hearing level fluctuations. The threshold variances noted by them was approximately 20 dB. These fluctuations were not as dramatic as those reported by Gorga, Stelmachowicz, Barlow and Brookhouser (1995) and Starr et al. (1998) on clients with temperature sensitive neuropathy. They also observed relieving type of auditory dys-synchrony. These authors found only one patient who showed a 15-20 dB threshold improvement over time. Sininger and Oba (2001) and Starr et al. (2000) subsequently noted that a similar proportion (29%) of ears had significant hearing level fluctuations. Stockard, Stockard and Coen (1983), Kileny and Robertson (1985), Stein et al. (1996) and Berlin et al., (1997) also observed improvements in hearing sensitivity in their database of clients with AN.

Madden et al. (2002) presented evidence of spontaneous hearing recovery in 9 of the 22 children with AN/AD in their sample. In the majority of cases the behavioural audiogram improved from a profound to a moderate or severe range, but in 4 cases hearing thresholds reportedly improved to normal or near-normal levels. Hearing recovery was more likely in this group amongst the subjects who had suffered neonatal hyperbilirubinemia, and in all cases, had occurred before the age of 25 months. Kumar and Jayaram (2006) observed in their study that many patients with auditory dys-synchrony appeared to have moment-to-moment fluctuations in the hearing sensitivity that could create the illusion of inconsistent responses during testing.

However, Starr et al. (2000) found that approximately 15% of the subjects with AN in their database had deterioration of hearing sensitivity greater than 10 dB at three or more test frequencies over a series of hearing evaluations. Likewise, Sininger and Oba (2001) observed that 14% of such clients showed progressive hearing loss. Three clients

were reported to have temperature sensitive auditory dys-synchrony where the hearing sensitivity worsened with the increase in body temperature and improved with a decrease in body temperature.

The literature bring to light that fluctuation in hearing sensitivity is common in individuals with AN. While some studies report of an improvement in threshold over time, others report of deterioration over time. Individuals with temperature sensitive AN were likely show improvement in hearing sensitivity with a reduction in temperature and similarly worsening in threshold with rise in body temperature.

2.4.4. Speech perception

It has been noted that patients with auditory dys-synchrony have speech perception abilities that are not in proportion with their pure-tone hearing loss (Starr et al., 1996; Li, Wang, Chen & Liang, 2005; Kumar & Jayaram, 2006). Speech perception abilities of patients with auditory dys-synchrony have been found to vary considerably. Some studies have reported of clients performing at the levels expected for cochlear hearing loss of same degree (Li et al., 2005; Kumar & Jayaram, 2006), while Starr et al. (1996) reported of little or no measurable speech identification despite adequate sound detection. Furthermore, this discrepancy between sound detection and speech identification was observed to be related to supra-threshold distortion of temporal cues (Zeng, Oba, Garde, Sininger & Starr, 1999; Rance et al., 2004; Zeng, Kong, Michalewski & Starr, 2005; Kumar & Jayaram, 2006).

2.4.5. Audiological test findings (Physiological)

Different physiological tests have been carried out on individuals having AD to get a better understanding of their specific hearing problems. These test include acoustic reflexes, otoacoustic emissions, electrocochleography (ECochG), auditory brainstem responses (ABR) and cortical auditory evoked potentials. The findings reported for each of these test in literature are discussed further.

2.4.5.1. Acoustic reflexes

Absent acoustic reflexes has been commonly reported in both children and adults with auditory dys-synchrony. Only three out of 44 subjects (6.5%) in a study by Sininger and Oba (2001) had presence or elevated acoustic reflexes, whereas 93.5% of their cases did not have acoustic reflexes. Deltenre et al. (1997) observed the presence of acoustic reflexes only in one of their subject. Acoustic reflexes have been reported to be absent for both ipsilateral and contralateral stimulation in almost all published cases, including those with normal or near-normal audiometric thresholds (Deltenre et al., 1997; Kraus, 2001; Sininger & Oba, 2001; Starr, 2001). However, non-acoustic middle ear muscle reflexes have been elicited in clients with auditory neuropathy by tactile stimulation to the face. This has been considered to suggest that the efferent components of the reflex arc (facial nerve and stapedius muscle) are intact (Gorga et al., 1995; Starr et al., 1998). Furthermore, Konradsson (1996) in his study involving four children with unilateral auditory neuropathy/dys-synchrony, found that an acoustic reflex in the AN/AD ear could be elicited by contralateral stimulation, but neither the ipsilateral nor contralateral responses could be seen when the stimulus was directed to the affected side. Hood and

Berlin (2001) also reported absence of acoustic reflexes in the affected ear, whereas non-acoustic reflexes were present. Earlier, Starr et al. (1998) inferred that in patients with auditory neuropathy, the afferent auditory pathway is not able to provide sufficiently high or sufficiently synchronized rates of discharge to activate the motor neurons of the stapedius muscle.

Hence, it can be concluded that stapedial acoustic reflexes are likely to be absent in most individuals with AN. However, non-acoustic reflexes elicited by tactile stimulation might be present in them.

2.4.5.2. Otoacoustic emissions

The otoacoustic emission responses have been considered to provide an indirect measure of the function of the cochlear amplifier and outer hair cells, offering a means of differentiating between sensory and auditory dys-synchrony types of hearing loss (Starr et al.,1996; Berlin et al., 2001). Ears with absent auditory brainstem responses due to sensorineural hearing loss have been found to typically show the presence of otoacoustic emission indicating the presence of AN/AD rather than a sensory type of hearing loss (Rance, 2005).

Otoacoustic emissions have been reported to be present in most individuals with dys-synchrony. Sininger and Oba (2001) reported that about 80% of the patients with dys-synchrony had clear OAE. In 11 to 16% of the patients with auditory dys-synchrony, OAEs disappeared over time and only 9% did not show OAE in the initial evaluation. This suggests that outer hair cells can be affected in dys-synchrony over time. Deltenre et al. (1999) also described two pre-lingual children with dys-synchrony who lost OAE

over time. One of the client's was successfully fitted with hearing aids soon after she lost OAE. The authors suggested that conventional amplification could benefit pre-lingual dys-synchrony children once they lost their OAEs. However, it is dangerous to make such a conclusion since this was found in just one child. In addition, no physiological basis was provided by Deltenre et al. (1999) regarding the usefulness of conventional hearing aids once the OAEs were lost. Thus, there is possibly no relation between disappearing of OAEs and hearing aid use.

The studies on OAEs in individuals with AD highlight that the test response is often present in this condition. However, it is not uncommon for OAEs to be present in the initial stage of the condition and disappear with time, indicating a progress of the problem.

2.4.5.3. Electrocochleography

The cochlear microphonics, through their ability to reflect the integrity of the cochlear hair cells, have been observed to play a significant role in the identification of ears with auditory neuropathy/dys-synchrony. 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 (Chisin, Pearman, & Sohmer, 1979; Starr et al., 1991; Berlin, Hood, Cecola, Jacson & Szabo, 1993; Starr et al. 1996; Berlin et al., 1998).

Cochlear microphonics that are robust and are present for several milliseconds after a transient click, have been recorded from individuals with auditory dys-synchrony

(Starr, et al., 1996; Berlin, 1999; Deltenre, et al., 1999; Starr et al., 2000; Santarelli & Arslan, 2002). Berlin (1999) reported that in approximately 50% of the individuals (N=33) with auditory dys-synchrony, 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 auditory dys-synchrony, 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 otoacoustic emissions were elevated.

Santarelli and Arslan (2002) reported ECochG findings in five patients with auditory dys-synchrony. Cochlear microphonics and summating potential was present with normal amplitude and thresholds in all but one subject. This finding led them to conclude that inner hair cells retain normal functions in patients with dys-synchrony, as they are believed to be the main source of summating potential generation (Durrant, Wang, Ding & Salvi, 1998). Santarelli and Arslan found the compound action potential component of ECochG recordings to be present in three out of five patients with auditory dys-synchrony. The N_1 component of the compound action potential showed variable degrees of dys-synchronization, from a broad response to a low amplitude delayed activity in all the three patients in whom compound action potentials were present. The authors hypothesized that these alterations in the compound action potential may have resulted from a lesion, localized in a more proximal portion of the auditory nerve where compound action potential is believed to be generated.

Similarly, Duan and Wang (2002) reported that summating potential was present in all patients with auditory dys-synchrony whom they tested and its amplitudes were

significantly larger than those of normal subjects. These results suggest that at least in a group of auditory dys-synchrony individuals with summating potential present, the lesion may be localized in the retro-outer hair cell region.

Thus, it can be concluded that cochlear microphonics are present in almost all individuals with AN even though otoacoustic emissions are absent or elevated. Hence, ECochG could be a better tool to find out integrity of outer hair cell function than OAEs measurements in clients with AN.

2.4.5.4. Auditory brainstem responses

In ears with auditory neuropathy/dys-synchrony, auditory brainstem responses (ABR) have been noted to be absent or grossly abnormal at maximum stimulus presentation levels, regardless of the behavioural hearing level (Starr et al., 1996; Rance et al., 1999; Sininger & Oba, 2001).

Starr et al. (2000) reported that 70% of their cases with auditory dys-synchrony had complete absence of any ABR component regardless of the level of the stimulus. 19% showed abnormal wave V, with most of them having a clearly defined peak but abnormal amplitude and latency. 6% showed wave III and V but wave morphology including amplitude and latency were abnormal. A common feature noted in patients who had ABR present, was their increased sensitivity to increase in stimulation rate. At higher stimulation rates none of the patients with dys-synchrony were found to have any ABR component. It was also noticed that patients who had ABR component had hearing loss that was 20 dB better than that of patients without ABR components. However, in

all patients with auditory dys-synchrony, behaviour thresholds did not correlate with ABR thresholds (Starr et al., 1996; Starr et al., 2000).

Disruption of ABR is thought be the result of either a reduction in the number of neural elements available to contribute to the response, or a disruption in the temporal integrity of the neural signal. Various authors have suggested that a dys-synchrony in the neural firing of the order of fractions of a millisecond is sufficient to disrupt the response and render the averaged potentials unrecognizable (Starr et al., 1991; Sininger et al., 1995; Kraus et al., 2000).

Berlin et al. (1998) reported that in some patients with dys-synchrony, cochlear potential to 100 µs condensation or rarefaction clicks persisted after the cessation of click and simulate the peaks of ABRs at high intensities. In these clients, when stimulus polarity was changed, the polarity of the waveform changed. Furthermore, the waveform did not shift in latency as the intensity of the stimulus was reduced. These findings confirm that potentials observed were cochlear rather than neural. Therefore, Berlin et al. (1998) recommended that responses of condensation and rarefaction clicks should be compared in order to separate ABRs from cochlear potentials.

From the studies reported in literature, it can be observed that absent or abnormal ABR is one of the important feature to identify individuals with AN. However, it has been recommended that caution should be exerted to separate ABRs from cochlear potentials, when interpreting the results.

2.4.5.5. Cortical auditory evoked potentials

One of the signature features reported regarding an AN/AD profile is the absence or severe disruption of the auditory brainstem response. It might be expected that more central evoked responses such as the middle latency response (MLR) and cortical auditory evoked potential (CAEP) would be similarly affected. Yet many of the cases reported in the literature have shown clearly identifiable responses with reasonably normal morphology and response latency (Gorga et al., 1995; Hood, 1999; Kraus et al., 2000; Rance, Cone-Wesson, Wunderlich & Dowell, 2002; Zeng & Liu, 2006). Rance et al. (2002) observed similarities between averaged CAEP waveforms obtained for a group of children with AN/AD with those from cohorts of age matched normal hearing children.

It has been speculated by Rance (2005) that cortical auditory evoked potentials may be recordable in some cases of auditory neuropathy/dys-synchrony because they are less dependent on synchronous neural firing than auditory brainstem responses. The peaks in the normal ABR waveform are reported to be biphasic and are usually only separated by approximately 1 ms. Small variations in the timing of responses to individual stimuli have been found to result in cancellation in the averaged signal. In contrast, it was found that the component peaks in the CAEP waveform, were more resistant to subtle fluctuations in the timing of individual responses. They were much broader and separated by 50 –100 ms in adults and longer in children. This was confirmed in a study by Michalewski, Prasher and Starr (1986). They determined the latency of various cortical event related potentials including N1 and P2 in normal adult subjects for individual stimulus trials. It was found that the standard deviations of the

peak latency were approximately 17 ms for the N1 potential, and 22 ms for the P2 potential. These individual trials, when subjected to conventional signal averaging procedures produced robust waveforms.

Hence, the findings of the studies in the literature suggests that cortical evoked potentials might be present in individuals with AN. The findings also suggests that synchronous firing, which is a main concern for clients with AN, is likely to have lesser influence on higher auditory evoked potentials.

2.5. Psychophysics in clients with auditory dys-synchrony

The abilities of individuals to perceive frequency, intensity and temporal aspect of acoustic signals have instigated considerable interest among researchers. These perceptual abilities in those have hearing problems have been compared with the abilities of the normal hearing individuals. Details regarding the perception of frequency, intensity and temporal cues in individuals with AD are further discussed in the next section.

2.5.1. Frequency Processing

Frequency discrimination has been generally studied by determining the ability of a person to perceive changes in frequency (or pitch) over time. There are several studies on frequency discrimination abilities in normal hearing individuals (Shower & Biddulph, 1931; Harris, 1952; Rosenblith & Stevens, 1953; Henning, 1966; Nordmak, 1968; Moore, 1973a; 1973b & Wier, Jesteadt & Green, 1977). All the studies are in agreement that the ΔF increases with the increase in frequency in normal hearing individuals.

Wier et al. (1977) have reported that difference limen (DL) values at 200 Hz to 800 Hz to be approximately 1 to 1.5 Hz and at 1000 Hz to 2000 Hz to be approximately 2 to 3 Hz. The DL value increased to 16 to 18 Hz in the frequency region of 4000 Hz. However, they found that the sensation level affected frequency discrimination more in the low frequencies than in the high frequencies.

Kamath (1989) obtained frequency modulated difference limen (FMDL) from 40 normal hearing Indians in the frequencies of 250 Hz, 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz. The psychophysical test was carried out at 20, 40, 60 and 80 dB SL. FMDL values obtained across the five frequencies and four sensation levels did not different significantly from each other. The normal FMDL values ranged between 1% to 1.25 % of the base frequency at any sensation levels.

For steady state (pure-tone) stimuli at 1 kHz and above, frequency discrimination has been thought to depend primarily on place mechanisms based on spatial changes in the basilar membrane excitation pattern (Moore, 1973b; Sek & Moore, 1995). In contrast, discrimination of stimuli less than 1 kHz is thought to be enhanced by the use of temporal information (Moore, 1973a; 1973b; Sek & Moore, 1995). It has been hypothesized that neural phase locking plays an important role in the fine-tuning of discrimination abilities in this range, whereas for higher frequencies, limitations in neural refractory period prevent phase-related responses (Rance, 2005). This has been substantiated by Sek and Moore (1995), who showed that models of frequency discrimination based solely on excitation pattern information, not taking into account phase locking, could not explain the variation of difference limens for frequency (DLF)

across anchor frequencies. Further, these authors found that low frequency difference limens were significantly smaller than predicted by the place of excitation models.

Several studies have measured frequency discrimination limens to fixed tonal stimuli in adults with cochlear hearing loss (Tyler, Summerfield, Wood & Fernandes, 1982; Freyman & Nelson, 1986; 1991; Moore & Peters, 1992). A high degree of inter-subject variability has been reported, but overall the findings indicated that discrimination ability was degraded by cochlear damage. In addition they also revealed that, frequency difference limens were not strongly correlated with either the subjects' hearing levels or frequency resolution ability (Tyler et al., 1982; Moore & Peters, 1992), suggesting that, as with normally hearing subjects, temporal cues play an important role in the discrimination process.

Frequency discrimination abilities in subjects with auditory neuropathy are yet to be thoroughly investigated but the data that has been presented thus far suggests extreme perceptual deficits in this regard. Frequency discrimination abilities in patients with auditory dys-synchrony are significantly poorer compared to normal hearing subjects (Starr, et al., 1991; Starr, et al., 1996; Zeng, Oba, Garde, Sininger & Starr, 2001; Rance et al., 2004; Zeng et al., 2005).

Starr et al. (1991) measured the monaural just noticeable differences (JNDs) for pairs of tone bursts in their 11 year old subject with AN. Pair of tone bursts, having duration of 500 ms with a rise and fall time of 10 ms, at octave frequencies from 200 Hz to 8 kHz were used. The pairs were separated by 400 ms and presented with an interval of 5 to 10 seconds. The subjects were asked to say whether the two tones were same or different. Catch trials were also presented to avoid false positive or negative responses.

Frequency discrimination results in this AD subject was consistently depressed. The JNDs were approximately 3 to 15 times higher across test frequencies, compared to a group of five age-matched normal children.

Starr et al. (1996) determined psycho-acoustical frequency discrimination task of subjects with AD, one being a participant of an earlier study by Starr et at. (1991). They described in detail the psychacoustical findings of one subject. The subject heard three stimuli from which the stimulus that was different from the other two, had to be selected. The difference between the stimuli was reduced and a bracketing method was used to establish monaural discrimination threshold. The 1000Hz anchor stimulus had a duration of 750 ms with a rise and fall time of 5 ms. The task was carried out at 60 dB HL. To discriminate the stimuli this subject required a difference of 172 Hz and 235 Hz between the anchor and variable tone, for the right and left ear respectively. This is in contrast with the findings in their normal hearing subjects who required only 2 to 17 Hz to differentiate between two frequency tones. This findings suggested that frequency discrimination ability was affected severely in cases with AN.

Zeng et al. (2001) also found impaired frequency discrimination abilities in three individuals with auditory neuropathy, compared to normal controls. Frequency difference limens were obtained at octave frequencies from 125 Hz to 8 kHz tone at the most comfortable level. The results indicated that the difference limen for frequency in listener with normal hearing increased monotonically as the frequency increased. In contrast, the JND for frequency was a non-monotonic function of frequency for the cases with auditory neuropathy. Their performances were considerably poorer than those obtained for individuals with normal hearing at low frequencies (below 2000 Hz). The

JND for frequency was found to continue to improve as a function of frequency and reached a normal value at 8000 Hz. The details of their results can be seen in Figure 2.1.

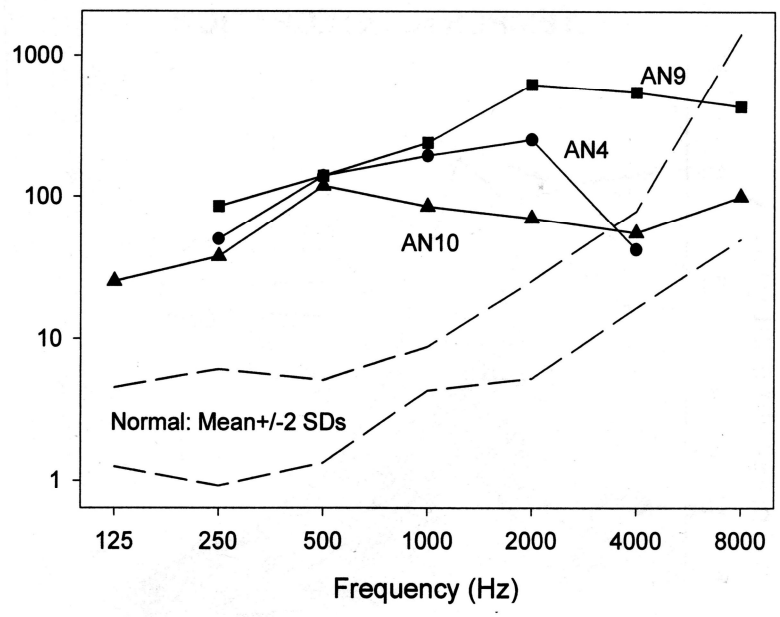

Figure. 2.1. Findings of Zeng et al. (2001) for frequency discrimination in subjects with normal hearing (marked by dashed lines) and in three subjects with neuropathy (symbols and solid lines). (Figure reprinted with permission from Delmar Learning, a division of Thomson Learning).

Frequency discrimination ability was similarly impaired in the group of children with auditory neuropathy presented in the study by Rance et al. (2004). In this study, the mean difference limen for a 4 kHz pure tone was 4.5 times the normal value, whereas discrimination at 500 Hz was on an average 11 times poorer than that observed in their normally hearing cohorts. Further, they compared the frequency discrimination limen for frequency modulated tones, which did not offer phase locking cues and pure tones, which

required phase locking. This comparison revealed that the children with auditory neuropathy were less able to use phase locking cues than subjects with normal hearing.

In a similar study, Zeng et al. (2005) measured the frequency discrimination ability of 12 cases having auditory dys-synchrony and compared their finding with the values obtained from 4 normal controls. This was done for octave frequencies from 250 to 8,000 Hz in octave steps. These tones were presented at the maximal comfortable loudness level. The result indicated a significant difference in performance between the subjects with AD and the normal controls. The normal controls required less than10 Hz to discriminate a pitch difference for frequencies below 1,000 Hz, and the subjects with AD required a difference that was about two orders of magnitude higher than the normal difference limen. Further, the difference between the two groups reduced with frequency and was not significant at 8,000 Hz (Figure 2.2). This result suggested that individuals with AD had profound impairment in pitch discrimination at low frequencies ($\leq$ 4000 Hz) but not at high frequencies ($\geq$ 4000 Hz). Hence, it was inferred that individuals with AN have a disruption in the processing of the low frequency temporal discrimination.

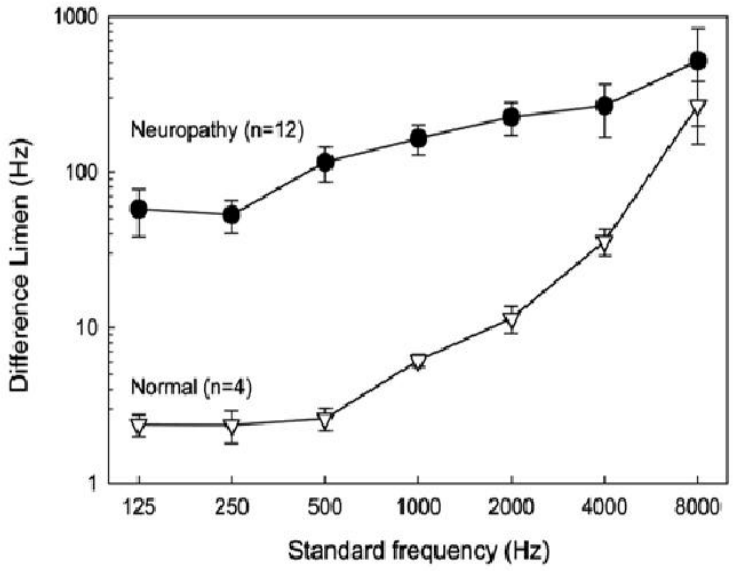

Figure.2.2. Findings of Zeng et al. (2005) for frequency discrimination in subject with AD and normal hearing. (Figure reprinted with permission from The American Physiological Society).

The results of frequency discrimination are explained on the basis of differential mechanisms of frequency coding at the high and low frequencies. For frequencies above 4 kHz frequency discrimination is thought to be dependent on spatial changes in the excitation pattern along the basilar membrane (Sek & Moore, 1995). In contrast, discrimination of frequencies below 4 kHz is considered to be enhanced by the use of neural phase locking cues (Goldberg & Brownell, 1973; Blackburn & Sachs, 1989; Winter & Palmer, 1990). The findings of the studies indicate that individuals with auditory dys-synchrony cannot use the phase locking cues to the same extent as normally hearing subjects. Hence, their performance on high frequency discrimination tasks that did not involve phase locking cues was relatively spared compared to the discrimination tasks that utilized phase locking cues.

The majority of the studies on frequency discrimination in individuals with AD, making use of a gated pedestal method highlighted how a disrupted temporal processing, led to poor DLF especially in the low frequencies. Across the studies, there was a consensus that those with AD have a greater problem in frequency discrimination in the lower frequencies, than in the higher frequencies. However, the effect of sensation level on DLF across frequencies needs to be addressed.

2.5.2. Intensity Processing

The normal auditory system is considered remarkable both in terms of its absolute hearing sensitivity and its ability in detecting small changes in intensity. Gelfand (2004) reported that DLs for intensity becomes smaller as the sensation level (SL) increases for mid-frequency stimuli.

Iyenger (2000) found SL not to affect difference limen for intensity (DLI). She obtained DLI for a1000 Hz tone at 10 and 40 dB SL using a 'yes-no' procedure. Twenty normal hearing Indian participants were evaluated in a monaural condition under earphones. She reported of a mean DLI value of 3.84 dB and 2.87 dB at 10 and 40 dB SL respectively. The range for these two intensities was 1 to 7 dB and 1 to 6 dB. The psychophysical data was compared with DLI obtained using MMN. However, the effect of presentation levels on psychophysical DLI was not statistically analysed.

Much earlier Riesz (1928) reported the modulation detection value of DLI to be 1.5 dB at 20 dB SL, 0.7 dB at 40 dB SL and 0.3 dB at 80 dB SL. Likewise, Miller (1947) had reported that the just detectable change in level was constant regardless of the

absolute level. The value was about 0.5-1 dB for white noise, presented at 20 dB to 100 dB above threshold in normal hearing individuals.

Studies have been carried out to determine the effect of DLI across different anchor frequencies. Jesteadt, Wier and Green (1977) did not find any frequency effect on DLI. Rather, they reported that ΔI/I was constant across frequency at any given sensation level. In contrast, Florentine, Buus and Mason (1987) reported a weaker frequency effect on DLI. This could be due to the methodological difference they used.

Turner, Zwislocki and Filion (1989) compared a continuous method with a gated method and reported smaller intensity DL for the continuous increment detection method. Further, the change in DL with level has been found to be somewhat less for pulsed tones than modulated tones (Moore, 1995a). He observed that if the ΔI (in dB) was plotted against I (in dB), a straight line would be obtained with a slope of about 0.9. A Weber fraction would result in a slope of 1.0. This has been called the 'near miss' to Weber Law which would suggest improvement in DL at high presentation levels (Moore, 1995a).

Loudness sensations evoked by sounds was usually thought to help detect the changes in intensity or to compare the intensity of two separate sounds. Loudness growth was reported to be usually more in individuals with cochlear damage than normal hearing individuals for given changes in intensity. Thus, Moore (1995b) opined that cases with a cochlear hearing loss would have better discrimination abilities for intensity than the normal hearing population. Such a findings was reported by Buus, Florentine and Ridden (1982 a, b), who found an improvement in the ability to detect changes in intensity in individuals with cochlear damage. Difference limen for intensity values was reported to

be smaller for clients with cochlear damage than normal hearing individuals when testing was done at equal sensation levels. In contrast, Glasberg and Moore, (1989) and Turner et al. (1989) reported that at equal SPLs, DLI values were similar in both the groups.

Starr et al. (1991) were the first to study the intensity discrimination ability of an 11 year old girl with auditory dys-synchrony. Monaural just noticeable difference (JND) for a 1000 Hz tone at 20 and 40 dB SL were obtained using paired stimuli. The intensity of a second stimulus was varied in 1 dB increments. The subject was asked to indicated whether the two tones were the same or different. The data obtained were compared with the data obtained from five age-matched children with normal hearing. The subject with AN required intensity increments that were approximately twice (10 dB) that obtained from her normal counterparts (4 dB).

Starr et al. (1996) carried out a psycho-acoustical intensity discrimination task on two clients with AN, one being a subject in the earlier study done by Starr et al. (1991). The clients had to discriminate one among a triad of stimuli, where one stimulus was different from the other two. The difference between the stimuli was reduced and a bracketing method was used to establish monaural discrimination thresholds. It was carried out for a 1000 Hz anchor stimulus having a duration of 750 ms with a rise and fall time of 5 ms. The test was carried out at 60 dB HL. The second patient, for whom the findings were described in detail, required 3 dB and 6 dB increment for the right and left ear respectively to discriminate between the stimuli. On the contrary, the norms obtained in their lab indicated that normal hearing individuals required less that 1 dB increment to differentiate in intensity. This suggests that clients with AN required relatively more intensity differences than what was required by the normal hearing individuals to perceive the difference.

Zeng et al. (2001) reported the loudness growth function in one subject with auditory dys-synchrony using a magnitude estimation and loudness scaling technique. Results showed that the subject with AD demonstrated a much more compressive loudness growth function than did their normal subjects. They also collected data regarding intensity discrimination from five subjects with auditory dys-synchrony and from normal controls. Two out of five subjects with AD showed slightly larger difference limen at low sensation levels than did the normal controls, which can be seen in Figure 2.3. The performance on intensity discrimination was not significantly different from that of the normal hearing individuals. Intensity difference limens decreased as a function of stimulus intensity in a similar fashion that could be seen even in the normal hearing individuals.

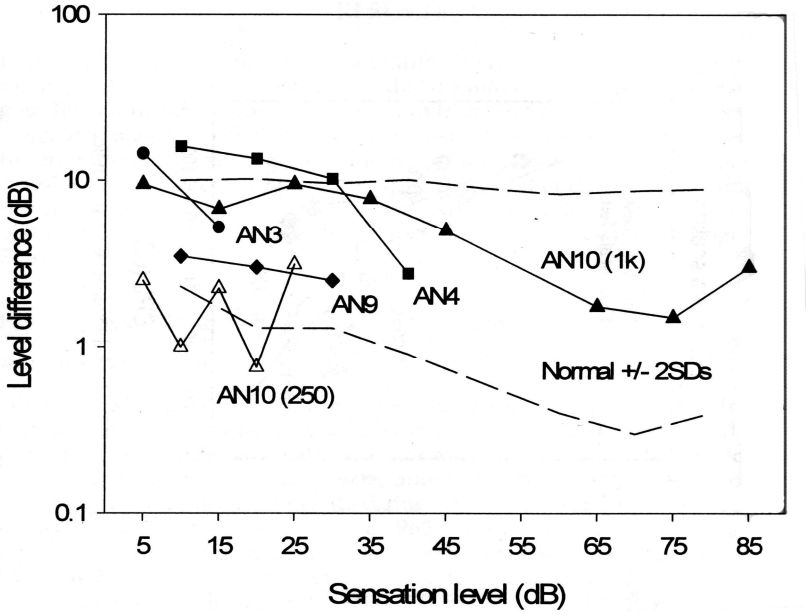

Figure 2.3. Findings of Zeng et al. (2001) for intensity discrimination in subjects with normal hearing (mean $\pm$ 2 SD, marked by dashed lines) and subjects with neuropathy (symbols and solid lines). (Figure reprinted with permission from Delmar Learning, a division of Thomson Learning).

Zeng et al. (2005) measured intensity discrimination as a function of level from near threshold to the maximal comfortable loudness for a 200 ms, 1000-Hz tone in eight subjects with AN and eight normal controls. An adaptive, three-interval, three-alternative, forced-choice, two-down and one-up procedure was employed to track correct responses, as suggested by Levitt (1971). The results indicated that the individuals with AD required higher intensity differences till 40 dB SL than the normal hearing individuals, which can be seen in Figure 2.4. Although the all the subjects showed slightly larger difference limens at low levels compared to the normal controls, no significant main effect was observed between groups. Thus, the authors concluded that the subjects with AN encounter no significant difficulty in performing pure-tone intensity discrimination.

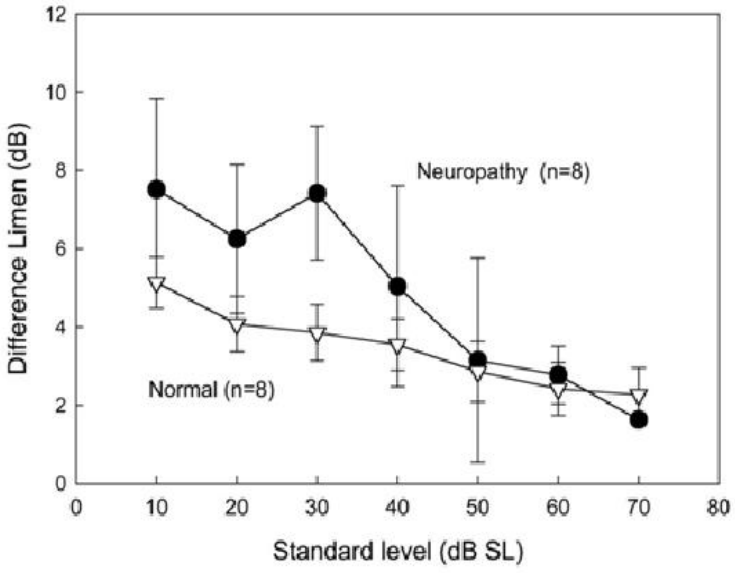

Figure 2.4. Findings of Zeng et al. (2005) for intensity discrimination in subject with AD and the normal hearing subjects as a function of standard level (dB SPL). (Figure reprinted with permission from The American Physiological Society).

It can be observed from the above studies that individuals with auditory neuropathy required more intensity difference to detect the difference between two stimuli in comparison to normal controls. However, there is considerable variability in findings from one study to another. This could be due to heterogeneity between the subjects with auditory neuropathy. Thus, it highlights the requirement of an extensive study in a large population.

2.5.3. Temporal Processing

Several investigators have explored the temporal processing abilities in individuals with auditory dys-synchrony. Different aspects of temporal processing have been studied and these include duration discrimination, temporal gap detection, temporal integration, masking level difference, temporal masking and temporal modulation transfer function. These studies are discussed below.

2.5.3.1. Duration discrimination

Duration discrimination, the ability of the auditory system to detect minute changes in duration of acoustic stimuli, has been examined by several authors. Studies on normal hearing individuals have been carried out for several decades. Creelman (1962) reported that the smallest detectable change in duration of a stimulus, ΔT, increased with increase in baseline duration (T) of a stimulus. Abel (1972) found that for stimuli with baseline durations (T) of 10, 100 and 1000 ms, ΔT was about 4, 15 and 60 ms respectively. The results were relatively independent of the overall level of the

stimuli and also were similar for noise bursts of various band widths and 1000 Hz tone burst. However, Moore (2003) reported that the ΔT increased at low sound levels.

Shylaja (2005) studied duration discrimination ability of 18 normal hearing Indians in a monaural condition using insert phones. The testing was done using a 1000 Hz anchor tone having a duration of 50 ms. Stimuli were presented at 40 dB SL using a gated method and discrimination threshold was obtained using a bracketing method. The results indicated that the normal hearing participants could differentiate 15 to 25 ms difference in duration between the two stimuli.

A few studies have evaluated duration discrimination in individuals having AN/AD. Starr et al. (1991) measured the JND for duration monaurally in their single case with AN and the data was compared with the data obtained from five control subjects. The JND for duration was obtained for 50 and 500 ms standard duration 1000 Hz tone burst pairs with a rise and fall time of 10 ms. These JNDs were measured at 40 SL. Each pair was separated by a 500 ms duration and the interval between the presentations of two pairs was 5-10 s. For the 50 ms standard tone, the second tone was varied with increments of 5 ms while for the 500 ms standard tone it was 50 ms. The subjects were asked to indicate whether the two tones were same or different in terms of duration. The results indicated that for the 50 ms duration tone the mean increment required was 20 ms for the normal hearing subjects, whereas for the client with AN the increment required was 20-30 ms. For the 500 ms duration tone, the normal hearing individuals required a mean duration increment of 140 ms whereas the subject with AN required higher changes in duration of 200 to 300 ms. Thus, they concluded that both the normal hearing subjects and the subject with AN seemed to required comparable

increments of duration of tone to perceive the difference.

In a continuation study, Starr et al. (1996) carried out a psycho-acoustical duration discrimination task on two subjects, one being a subject in the study by Starr et al. (1991). The clients had to identify the stimulus that differed in a set of three stimuli. The difference between the stimuli was reduced and a bracketing method was used to establish monaural discrimination threshold. It was carried out for a 1000 Hz anchor stimulus of 750 ms duration with a rise and fall time of 5 ms. The test was carried out at 60 dB HL. The result indicated that both subjects had poorer ΔT compared to the normal hearing individuals. The second subject of the study required duration differences between the anchor and variable tone of 118 ms and 145 ms for the right and left ear respectively. In contrast, the norms developed by them in their laboratory showed that normal hearing individuals required just 25 to 50 ms difference to identify the difference.

Thus, the review of the literature brings to light that the ability to discriminate two short duration stimuli in individuals with auditory dys-synchrony is relatively poor compared to normal controls. However, to generalise these findings, a larger database would be required.

2.5.3.2. Temporal gap detection

The normal auditory system has been found to have the ability to perceive minute changes in stimuli over time such as to detect a brief gap between sounds (Moore, 2003). In an attempt to measure the threshold for detecting a gap in narrow-band noise, Shailer and Moore (1987) observed that it was about 5 ms for centre frequencies of 400, 1000 and 2000 Hz. Later Moore, Peters and Glasberg (1993) also reported of a constant gap threshold of 6-8 ms over the frequency range of 400-2000 Hz. However, they found that

it increased at low sound levels. Plomp (1964) and Penner (1977) observed a gap threshold of 2-3 ms for broad band noise at high SLs. They also noted that it was almost constant for moderate to high levels.

Slightly higher gap detection thresholds were obtained by Shivaprakash (2003) in a group of normal hearing Indian children and adults. He used noise bursts of 300 ms duration with a silence of different duration to obtained gap detection threshold (GDT) at 40 dB SL. A bracketing method was used to track the gap detection threshold. The result indicated that normal hearing adults could detect a mean gap of 3.3 ms and children aged 7 years could detect a gap of 4.05 ms. However, the significance of difference between children and adults was not obtained.

The gap-detection thresholds were found to be insensitive to changes in level (above 30 dB SL), total duration and temporal position within a white noise (Moore, 1995a). However, the gap threshold was observed to increase at very low sound levels, when the noise level approached the absolute threshold (Moore, 2003).

In individuals with a cochlear hearing loss, Moore (1995b) recorded a larger gap detection threshold than in normal hearing individuals. He obtained a gap threshold in different envelop conditions and found the geometric mean to be 12.8 ms for a normal ear and 27.2 ms for a cochlear hearing loss ear for a normal envelop condition.

Starr et al. (1991) were the first to measure monaural gap detection thresholds in a subject with AN. They used 1000 Hz tone-pairs presented at a comfortably loud level. The durations of the tone burst, for which gap detection was measured, were 5, 10, 20, 50, 55, 60, 80 and 200 ms. In the middle of one of the tone-pairs, a silence interval was introduced in a random schedule which varied adaptively to achieve a 79% correct

performance. The tone-pairs were separated by 500 ms. The subject was asked to press one of the two buttons to indicate whether the first or second tone had the gap. They assessed only the right ear of the subject. It was observed that the subject could identify silence having a length of 15 to 20 ms for tones with 25 to 200 ms duration. When the duration was reduced below 25 ms, the gap detection threshold increased drastically to 80 ms for tones having a duration of 10, 15 and 20 ms. The GDT further increased to 200 ms for the 5 ms duration tone. In contrast, normal hearing individuals could identify a silence period of 2 ms for all these duration tones.

In 1996, Starr et al. extended their study to determine the gap detection threshold on two subjects having AD and described in detail the responses of one client. This client was not a part of the study conducted by their group in 1991. They presented three stimuli and the patient was asked to identify the stimulus which was different form the other two. Gradually, the difference between the stimuli was reduced and the monaural gap detection threshold was determined using a bracketing method. It was carried out for a 1000 Hz tone anchor stimulus of 750 ms duration with a silence at the centre of the tone with a rise and fall time of 5 ms. The test was carried out at 60 dB HL. The gap detection threshold in this client with AN was more, with it being 6 ms and 12 ms for the right and left ear respectively. However, the norm developed in their laboratory was 1-5 ms.

Zeng et al. (1999) studied the gap detection threshold in eight patients with auditory neuropathy, including one with unilateral neuropathy using broad band noise. They also obtained data from a control group including the healthy ear of a subject with unilateral neuropathy, one cochlear-impaired subject with a low-frequency hearing loss

and six normally hearing subjects. The psychophysical tests used a three-alternative, forced-choice procedure to measure the threshold that resulted in a 70.7% correct response. Detection of short silent intervals, or gaps, in acoustic signals was found to be uniformly impaired in the neuropathy patients. In the normal-hearing listeners, as well as the unilateral control, the gap detection thresholds improved from 20 to 30 ms at low sound levels, to 2-3 ms at high sound levels. In contrast, subjects with auditory neuropathy had large deficit at the highest sound level with their gap detection thresholds being 2-25 times greater than the threshold obtained in the normal hearing group.

Using ten subjects (5 females and 5 males) with auditory neuropathy, the gap detection threshold was studied by Zeng at al. (2001). Eight of these subjects had participated in their earlier study carried out in 1999 by Zeng et al. These subjects were aged 10 to 53 years with an average age of 28 years. In the gap detection, a silent interval was produced in the centre of the broad band noise. The procedure used was similar to that used in their earlier study conducted in 1999. The results indicated impairment of gap detection in all the subjects with AN, which can be seen in Figure 2.5.

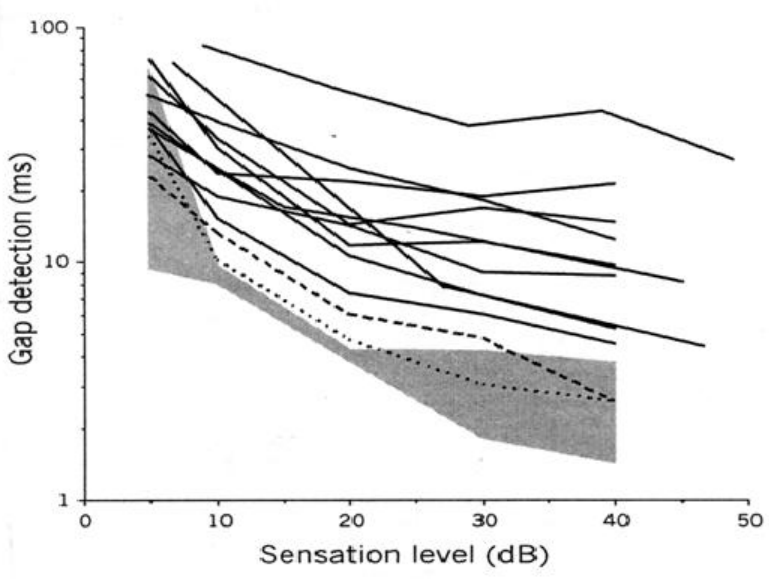

Figure. 2.5. Findings of Zeng et al. (2001) for gap detection thresholds. Normal control data are represented in the shaded area. Neuropathy data are represented by solid lines. The dashed line represents the cochlear impaired case, and the dotted line represents the healthy ear of the unilateral cases. (Figure reprinted with permission from Delmar Learning, a division of Thomson Learning).

Zeng et al. (2005) analyzed 20 previously diagnosed subjects with AD and measured their gap detection abilities. Eight of the subjects were those who participated in an earlier psychophysical study to assess temporal processing by Zeng et al. (1999). The subjects with AD had an age range from 6 to 53 years, with a mean age of 21 years. Their degree of hearing loss varied from near normal hearing (20 dB HL) to severe hearing loss (70 dB HL). They calculated pure-tone average using all the frequencies from 125 to 8,000 Hz. They also employed an adaptive, three-interval, three-alternative, forced-choice, two down and one-up procedure. The results suggested that the subjects

with AD had difficulty in gap detection even at comfortable loudness levels. The normal controls required about a 50 ms silent interval to detect a gap using a very soft signal (5 dB), but improved to 3 ms at high sensation levels (40 and 50 dB). The subjects with AD performed similar to the normal controls at low sensation levels (5 and 10 dB) but required significantly longer gaps (15–20 ms) than the normal-hearing subjects at higher sensation levels (Figure 2.6). The result suggested that the subjects with AD have difficulty in gap detection even at comfortable loudness levels.

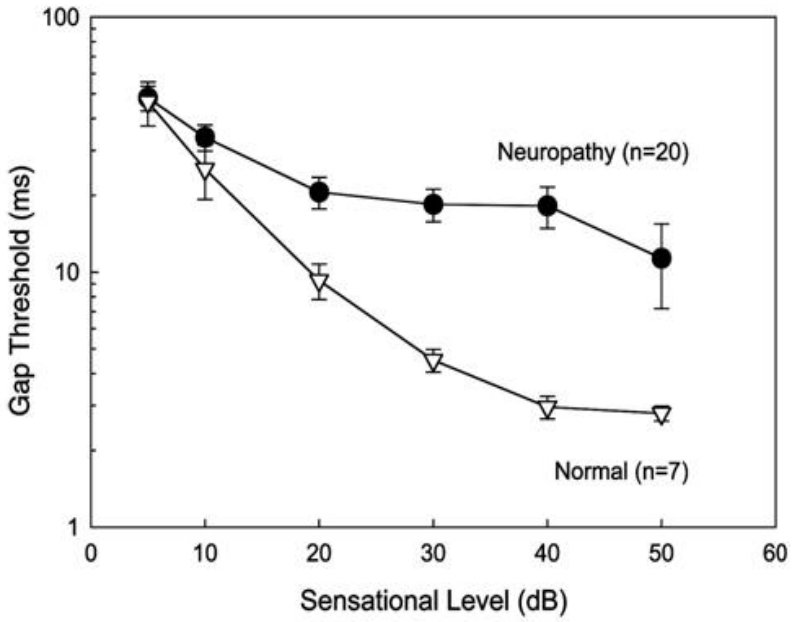

Figure. 2.6. Findings of Zeng et al. (2005) for gap detection in individuals with AD and normal-hearing subjects. (Figure reprinted with permission from The American Physiological Society).

Michalewski, Starr, Nguyen, Kong, and Zeng (2005) compared the psycho-acoustically and electro-physiologically measured gap detection threshold for 12 normal

hearing individuals and 12 subjects with AD. Psycho-acoustical gap detection threshold was measured using a 3-interval, 3-alternative forced chose (2-down,1-up) adaptive procedure. Psycho-acoustic gap detection threshold was found to be between 2 and 3 ms for the individuals with normal hearing. This was within the norms established at their laboratory, which was 2-4 ms. Psycho-acoustical gap detection threshold was much higher for subjects with AD, which ranged from 5 ms to 40 ms. However, a minimum gap of 5 ms could elicit N100/P200 component for normal where as it was still higher for patient with AD which ranged from 10 ms to 50 ms.

Based on their findings on gap detection threshold in individuals with AD, Zeng et al. (1999, 2001) presented the phenomenological model of the disrupted synchronous neural activity. This model assumed that the main effect of dys-synchronous activity was due to smeared temporal representation of the acoustic stimulus (Figure 2.7).

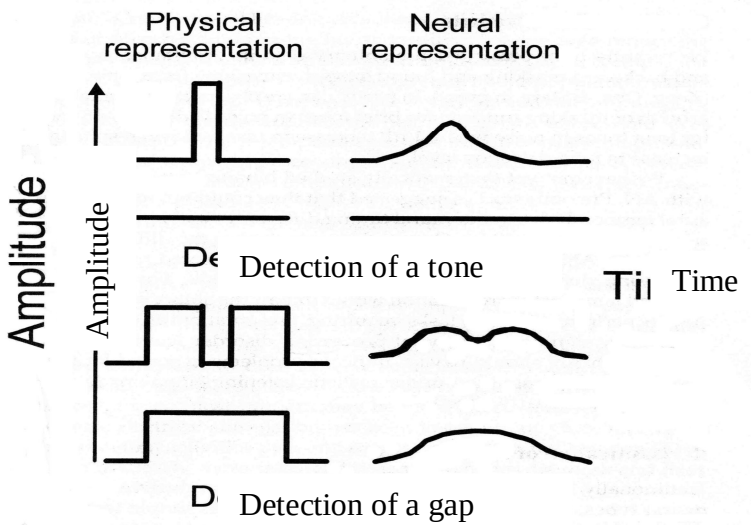

Figure. 2.7. A phenomenological model of auditory neuropathy proposed by Zeng et al. (2001) to explain impaired gap detection threshold. (Figure reprinted with permission from Delmar Learning, a division of Thomson Learning).

The model proposed that the sharp temporal changes in the physical representation of the stimulus were lost in the internal neural representation due to smearing of the waveform. It was noted that if the listening task was merely detection of the sound, then this smeared representation would not effect the perception. However, if the task was to discriminate between the two sounds, one with a gap and one without a gap, then smearing in the internal representation made the task more difficult and affected perception.

Later in 2005, Zeng et al. proposed another model to explain how demylinated neurons and reduce nerve conduction can lead to poor gap detection ability in individuals with auditory dys-synchrony. Figure 2.8*A* shows the normal auditory pathway with synchronized neural conduction in three auditory nerve fibres. The bottom trace represents the gap stimulus, while the 'average' trace represents the central neuron's output in response to the three auditory nerve fibres synchronized discharges (within 0.5 ms). The neural synchrony preserved the gap in terms of the temporal discharges relative to background spontaneous or random activity. Figure 2.8B shows the earlier AN model proposed by Zeng et al. (1999, 2001). The desynchronized nerve conduction in three demyelinated nerve fibres, which differentially delayed neural representations of the gap (~1.5 ms) and therefore produce a smeared central representation of the gap at the output are depicted. Figure 2.8C shows their second AN model based on reduced nerve conduction with only one nerve fibres able to transmit the gap information. Both desynchronized (Figure 2.8B) and reduced (Figure 2.8C) nerve conditions were consider to produce an averaged discharge pattern that is difficult to distinguish from the background spontaneous activity. In most cases of AN, it has been suggested that both desynchronized and reduced spikes may co-exist to exaggerate the perceptual consequences of neural synchrony.

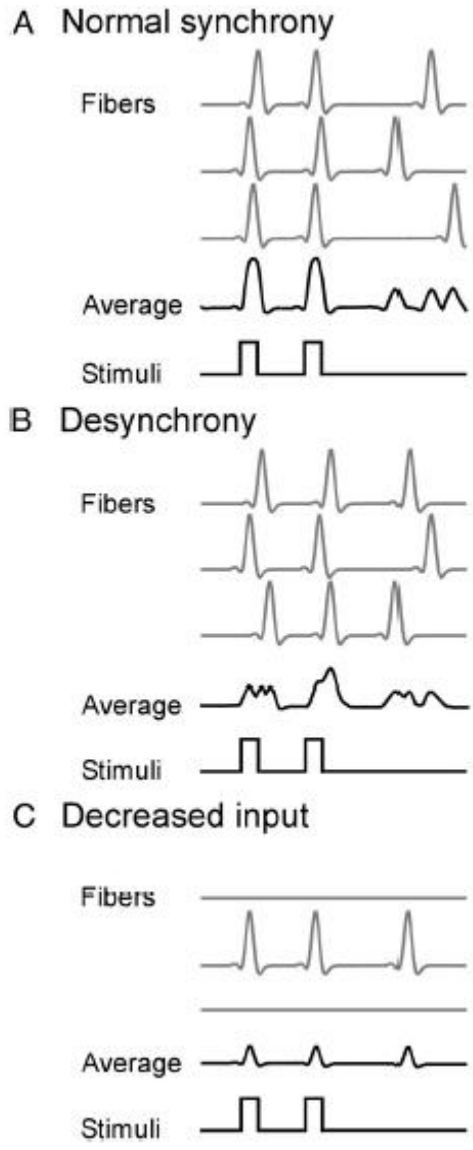

Figure.2.8. Phenomenological models of AN proposed by Zeng et al. (2005) to explain impaired gap detection threshold. A: Normal auditory pathway converting the 'gap' stimuli (*bottom trace*). B: AN model with desynchronized nerve conduction, in which the central representation of the gap is distorted due to different delays. C: Another AN model with reduced nerve conduction, in which the central representation of the gap is also difficult to detect because of its similarity to the background spontaneous activity. (Figure reprinted with permission from The American Physiological Society).

It is evident from the studies reported in literature that cases with auditory dys-synchrony exhibit higher gap detection threshold at least at high intensity levels. This has been demonstrated by using tonal stimuli at most comfortable levels as well as broad band noise at different levels. The gap detection threshold obtained in individuals with AD varied vastly from near normal to large abnormalities in almost all the studies. The variability across studies could be on account of the differences in stimuli used as well as inter subject variability. A study on a large population might help generalize findings made by earlier researchers.

2.5.3.3. Temporal Integration function

The absolute threshold of sounds has been found to depend upon the duration of the signal (Moore, 2003). It was observed that the threshold was almost independent of the duration of the signal if the tone burst duration exceeded about 500 ms. In contrast, sound intensity required to detect the presence of a signal increased if the signal duration reduced to below 200 ms. It was also found that the threshold in dB fell with a slope of -3 dB for every doubling of duration in normal hearing individuals (Moore, 2003). Thus, temporal integration has been considered responsible for a person being able to perceive longer signals (> 200 ms) at lower intensity compared to requiring higher intensities to perceive shorter duration signals (< 200 ms). Zwislocki (1960) and Penner (1972) opined that it was the neural activity which was responsible for integration of an auditory signal and not the peripheral structure of the auditory system.

Controversy over variation in temporal integration with frequency still exists. Plomp and Bouman (1959) reported that the change in detection threshold varied across frequencies for a fixed duration stimulus in normal hearing individuals. On the contrary, Olson and Carhart (1966) observed no variation in threshold with stimulus duration for frequencies of 250, 1000 and 4000 Hz. Floretine et al. (1987) also reported similar findings as Olson and Carhart.

In individuals with a cochlear hearing loss, Moore (1995b) found that the change in threshold intensity with signal duration was often smaller compared to normal hearing individuals. The slopes observed in those with cochlear hearing impairment were usually much less in than the absolute value of -3 dB/octave which was typically observed in the normal hearing population. Other researchers have also reported that such individuals usually had reduced temporal integration (Hall & Fernandes, 1984; Carlyon, Buus & Florentin, 1989).

Measurement of temporal integration in a client with AN was first attempted by Starr et al. (1991). They obtained monaural threshold as a function of signal duration using 1000 Hz tone bursts. The duration of the tone bursts were between 200 and 5 ms. The threshold was obtained using the method of limits with a step size of 2 dB. The average of 'T', the point of an ascending and descending signal series, was considered as the threshold. The threshold of the tone burst increased by 3 dB per halving of signal duration between 300 and 30 ms for both normal and the subject with AN. However, the threshold increased abruptly to 20 dB for a halving of signal duration, when the signal durations were shorter than 30 ms.

The temporal integration function in eight subjects with auditory neuropathy, including one with unilateral auditory neuropathy was measured by Zeng et al. (1999). As mentioned earlier, they also studied eight individuals with AN, including the individuals with unilateral neuropathy, one subject with cochlear impairment and six normally hearing subjects. A broad band white noise (20 to 14000 Hz) with a duration of 500 ms and 2.5 ms cosine-squared ramp was used to measure temporal integration function. Detection threshold for the normal hearing listeners decreased at a rate of about 3 dB per doubling of the signal duration for duration up to 100-200 ms. All subjects with auditory neuropathy showed near normal or normal temporal integration function.

Zeng et al. (2001), in a sequel to their earlier study, reported the temporal integration function of ten subjects with auditory neuropathy. This study included the data of eight subjects from their earlier study in 1999. One subject with AN had a steeper slope of -8 dB per doubling of the duration of the stimulus compare to the other subjects. The remaining nine subjects with AN had an average slope of -4 dB per doubling of duration of the signal which is much closer to the value obtained in normal hearing subjects (Figure 2.9).

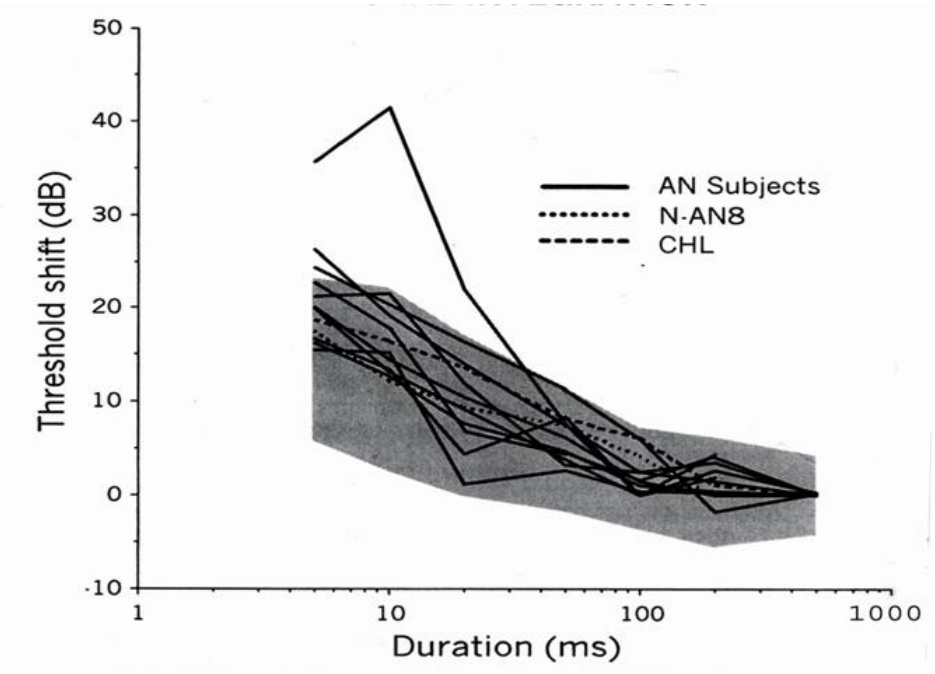

Figure. 2.9. Findings of Zeng et al. (2001) for temporal integration functions. Normal control data are represented as the shaded area. Neuropathy data are represented by solid lines. The dashed line represents the cochlear-impaired case, and the dotted line represents the healthy ear of the unilateral case. (Figure reprinted with permission from Delmar Learning, a division of Thomson Learning).

In line with their earlier studies, Zeng et al. (2005) carried out several psycho-acoustical tests, including temporal integration, on 16 individuals with auditory dys-synchrony and 4 normal hearing controls. They adopted an adaptive three-interval three-alternative forced choice, two-down, one-up procedure to track a 70.7% correct response criterion. In individuals with auditory dys-synchrony, like the normal hearing subjects, thresholds improved as the duration of the signal was increased. However, the slope of the integration function was slightly elevated in individuals with auditory dys-synchrony

(-9 dB per doubling of duration) compared to the normal hearing subjects (-3 dB per doubling of the duration), which is seen Figure 2.10.

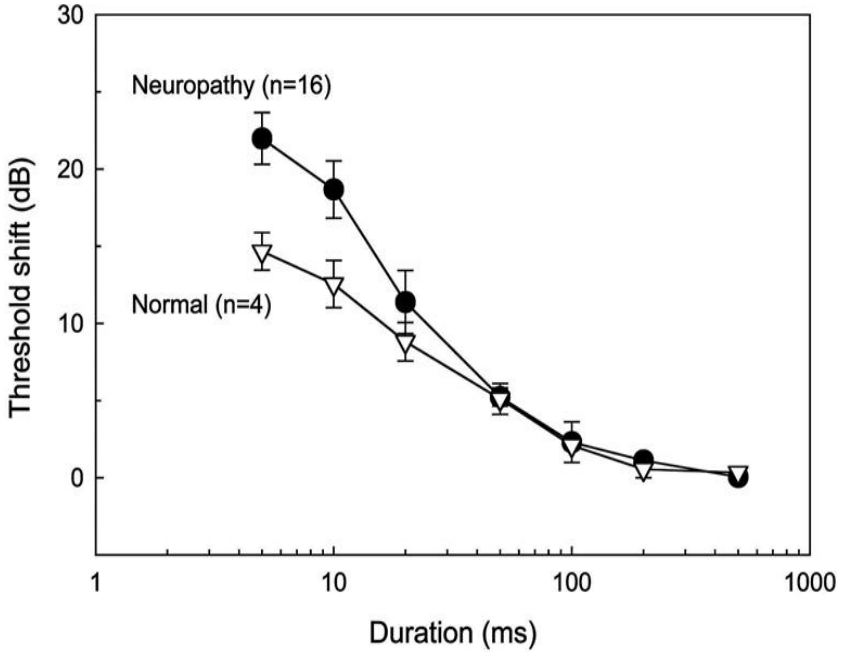

Figure 2.10. Findings of Zeng et al. (2005) for temporal integration in individuals with AD and normal-hearing subjects. Threshold shift (dB re: threshold with a 500 ms noise) is plotted as a function of stimulus duration (ms). (Figure reprinted with permission from The American Physiological Society).

Thus, the studies showed equivocal findings regarding temporal integration functioning individual with AN. Starr et al. (1991) and Zeng et al. (1999) found normal or near normal temporal integration function in individuals with AD. In contrast, Zeng et al. (2001) and Zeng et al. (2005) observed a steeper slope for temporal integration function, especially for short duration stimuli. Hence, a study on a large population might give a better idea about the slope of temporal integration function in individuals with AN.

2.5.3.4. Binaural Masking Level Differences (MLDs)

Binaural MLD has been referred to as the improvement in detection of a signal-in-noise that occurs when the signal and noise differ in interaural time or interaural intensity (Moore, 1989). The MLD has been reported to become larger as the spectrum level of the masking noise increases, especially when the noise is presented to both ears at the same level (Hirsh, 1948; Dolan & Robinson, 1967; McFadden, 1968). The largest MLDs were obtained when either the signal (SπNo) or the noise (SoNπ) was opposite in phase at the two ears (Gelfand, 1990). The firing pattern of the auditory nerve fibres has been found to be phase-locked especially at low frequencies. Thus, the large MLD reported to be associated with antiphasic conditions, might be related to this phase-locking mechanism in the neural coding of stimuli (Green & Henning, 1969). Since the degree of phase-locking is greater at low frequencies, the size of MLD has been reported to be related to the stimulus frequency. The MLD was reported to be about 15 dB for 250 Hz and decreased to 3 dB about 1500-2000 Hz (Gelfand, 1990). Durlach and Colburn (1978) reported that the MLD values could be as large as 15 dB at low frequencies (500 Hz) and decrease by 2-3 dB for frequencies above 1500 Hz, in normal hearing individuals.

Several other studies confirmed that MLDs are typically smaller in subjects with cochlear hearing loss than those with normal hearing (Hall, Tyler & Fernandes, 1983; Jerger, Brown & Smith, 1984; Staffel, Hall, Grose & Pillsbury, 1990). A trend observed in the studies was that smaller MLDs were obtained in subjects with higher absolute thresholds, although this association was not very strong. Further, it was reported that subjects with similar absolute thresholds could have very different MLDs. MLDs also

tended to decrease with increasing asymmetry of the loss, as reported by Jerger et al. (1984).

Starr et al. (1991) compared the MLD value of their single case with AN with the values obtained form five control subjects. The binaural MLD value obtained in individuals with AN was 0 dB at 300 Hz. Later, Starr et al. (1996) carried out the MLD test on six of their 10 subjects with AD. The test was carried out at 60 dB HL. They found monaural MLD by getting the difference in threshold for a monaural tone (Sm), presented in monaural noise (Nm) as well as binaural correlated noise (No) at the same intensity (NmSm versus NoSm comparison). None of their subjects showed any improvement in hearing sensitivity between the conditions tested.

Hood and Berlin (2001) also did not observe any difference in threshold between a homophasic and antiphasic condition in individuals with auditory neuropathy. Likewise, Hood et al., (2002) also reported a lack of improvement in detection of acoustic signals in the antiphasic condition in comparison to the homophasic condition. They concluded saying that the impaired MLD has been considered an indication of poor auditory neural functioning at the brain stem level.

Based on the studies reported in literature, it is evident that MLD is severely affected in individuals with AD. Such findings have been obtained for monaural MLD as well as binaural MLD.

2.5.3.5. Temporal masking

The temporal masking (forward masking and the backward masking) results have been reported to depend on the stimulus intensity, duration of the masker and the interval between the two stimuli. Moore and Glassberg (1983) reported that the effect of

backward masking is higher than the effect of forward masking, keeping all the factors constant. They also reported that the maximum masking effect could be approximately 30 dB in individuals with normal hearing. This maximum effect was seen when the duration between the stimuli reduced or masker duration or intensity of the masker was increased.

Kraus et al. (2000) reported about exaggerated masking effect in one patient with auditory dys-synchrony who had near normal hearing thresholds. Also, temporal masking and simultaneous masking paradigm results, in a study by Zeng et al. (2005), indicated that individuals with auditory dys-synchrony have difficulty in separating auditory signals from noise that occurred in succession or simultaneously. Those with auditory dys-synchrony showed 60% masking effect even when the signal and masker were separated by as much as 100 ms. However, normal controls showed only 15% masking with a signal delay of < 20 ms in a forward masking paradigm. The slope of the masking function varied between subjects, some having relatively normal slope, some with an abnormally steep slope and some with abnormally shallow slope. Excessive masking of about 20 dB was seen in simultaneous masking in cases with auditory dys-synchrony compared to their normal hearing counterparts. This excessive masking was found to be independent of threshold of frequency tested.

Zeng et al. (2005) also assessed the binaural processing of intra-aural time, intra-aural intensity, fusion and beats in individuals with auditory dys-synchrony. In the intra-aural intensity experiment, the group with auditory dys-synchrony performed similar to the normal control group. Participants with AD could effectively use intra-aural intensity cues to localize sound. On the contrary, in the intra-aural time experiment, performance

of group with auditory dys-synchrony was significantly poorer compared to the normal control group. The normal subjects localized sound to the ear with a leading phase, whereas subjects with auditory dys-synchrony could not use the intra-aural time cue to localize the sounds. Further, the subjects with auditory dys-synchrony performed similar to the normal control group on a monaural beat task but failed to perceive the sensation of beats on binaural presentation. They attributed this to the fact that perception of monaural beats required spike synchrony of 3 Hz modulations in the waveform envelope, whereas detection of binaural beats required spike synchrony to rapidly changing carrier frequencies. This result shows that individuals with auditory dys-synchrony can perceive slow temporal fluctuations but not fast. Similar findings were reported by Starr et al. (1991) in a subject who displayed symptoms of auditory dys-synchrony.

Thus, the above review suggests that in individuals with AD perform poorly on temporal masking task in comparison with normal hearing individuals. However, subjects with AD could use intensity disparity cues to localize sounds similar to what is observed in normal hearing individuals.

2.5.3.6. Temporal modulation transfer function

Several authors have reported on the attenuation slope of the temporal modulation transfer function in normal hearing individuals. Rodenburg (1977) reported a slope of nearly -6 dB per octave. Several other investigators have reported of a slope of about -3 dB per octave in individual with normal hearing (Veimeister, 1979; Bacon & Veimeister, 1985; Forrest & Green, 1987; Formby & Muir, 1988; Eddins, 1993).

Temporal modulation transfer function is another temporal process that is found

to be abnormal in individuals with auditory dys-synchrony is (Zeng et al., 1999; Rance et al., 2004; Zeng et al., 2005; Kumar & Jayaram, 2005). Zeng et al. (1999) reported that individuals with auditory dys-synchrony showed a high peak sensitivity of -8.7 dB compared to -19.9 dB observed in normal controls. They also had a lower cut off frequency of 17 Hz compared to 258.1 Hz obtained in normal controls. Kumar and Jayaram (2005) observed that the average modulation detection threshold in individuals with auditory dys-synchrony was three times higher than their normal hearing listeners. The difference between normal listeners and individuals with auditory dys-synchrony was more pronounced for higher modulation frequencies. The authors attributed this to the differential processing of higher and lower modulation frequencies in the auditory system.

Wang and Sachs (1993, 1994) and Frisina (2001) reported that synchronous responses for temporal coding were essential at the auditory nerve and brainstem level that codes high modulation frequencies. Less synchrony was considered necessary at higher centres which code low modulation frequencies. Hence, it can be expected that individuals with auditory dys-synchrony will have more problems in processing high rates of modulations which requires synchronous firing of the auditory nerve fibres. Furthermore, Kumar and Jayaram (2005) also demonstrated a strong correlation between peak sensitivity and speech perception scores. The poor performance of individuals with auditory dys-synchrony on a modulation detection task was also reported by other investigators. Rance et al. (2004) also observed significant differences in modulation detection thresholds in individuals with auditory dys-synchrony having good and poor speech perception scores. Subjects with auditory dys-synchrony having speech

identification scores less than 30% had poorer modulation detection thresholds compared to subjects with auditory dys-synchrony having speech identification scores more than 30%.

These psychophysical findings indicate that timing and synchronicity in firing of neuron in the auditory brainstem and auditory nerve are important for auditory perception. Patients with auditory dys-synchrony have difficulty in timing related perception but not intensity or frequency related perception. As discussed earlier they have difficulty in discriminating pitch at low frequencies, temporal integration, gap detection, modulation detection, detecting the beats when stimuli are presented binaurally and using intra-aural time cue in localizing sound. These deficits in patients with auditory dys-synchrony differ from deficits that arise from damage to cochlea that results in disruption of intensity and frequency related perception.

Though the review of literature does throw light on the psycho-acoustic deficits seen in individual with AD, the findings are equivocal. Variation observed across studies could be an account of inter-participant variability or due to the procedural variations. It could also be related to the varying degree of difficulty of task employed in these studies. Some of the task may not have been sensitive enough to detect the subtle deficit exhibited by this group. In order to get more comprehensive information regarding perceptual deficits in individuals with AD, it is necessary to carry out a study using a larger group. With this in mind, the present study has been designed. Parameters that have been noted in literature to differentiate individuals having AD as a group, have been considered while designing the present study.

3. METHOD

The objectives of the present study were to measure the fine-grained discrimination ability for frequency, intensity and duration; gap detection threshold (GDT); temporal integration; and masking level difference (MLD) in normal hearing individuals and individuals with auditory dys-synchrony. The experiment involved three phases. The first phase involved development of materials for the study. While the second phase involved participant selection, the third phase dealt with obtaining psycho-acoustical data from both normal hearing individuals and individuals with auditory dys-synchrony. A non-experimental, standard group comparison research design was adopted to achieve the objectives.

3.1 Participants

A total of 78 individuals participated in the study. They were classified into a clinical group and normal hearing group. In each group, there were 39 participants. The details of the participants of the two groups are discussed below.

3.1.1. Clinical group

The clinical group consisted of 39 participants, with confirmed diagnosis of auditory dys-synchrony. Their mean age was 19.46 years with the range being 14 to 28 years. This age range was selected as it has been reported that psycho-acoustical abilities reach a plateau in this age range (Lynne, Werner & Gray, 1998). Participants who met the following criteria were included in the study:

- Exhibited signs and symptoms of difficulty in understanding speech especially in a group or adverse listening condition,
- Showed no symptoms of external or middle ear problems,
- Had no history of ototoxic drug intake or exposure to continuous loud noise,
- Had not undergone any formal training in auditory learning activities, and
- Were fluent speaker of a Dravidian language used in southern India.

This information was elicited through a structural clinical interview and case history. Further, only participants who manifested the following audiological characteristics were included in the clinical group:

- Pure-tone hearing thresholds less than 55 dB HL at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz with an air-bone gap within 10 dB HL,
- Symmetrical hearing loss, where the difference in threshold between the two ears of a participant did not exceed 10 dB HL at any frequency,
- Disproportionately poor speech identification scores (SIS) in relation to pure-tone threshold in a quiet situation or poor speech identification scores (less than 10%) in a speech-in-noise test at 0 dB SNR, tested using the Common Speech Discrimination Tests Materials for Indians developed by Mayadevi (1978),
- 'A' type tympanogram with no ipsilateral and contralateral reflexes,
- Absent auditory brainstem responses (ABR) at 90 dBnHL,
- Transient evoked otoacoustic emissions (TEOAEs) with normal or robust amplitude,
- Absence of any external or middle ear problem, based on an otological evaluation, and

- Absence of any peripheral neuropathy or space occupying lesion, determined based on a neurological examination and CT or MRI evaluation interpreted by a qualified neurologist.

The demographic details and audiological findings of the clients having auditory dys-synchrony (AD) are discussed in Table 3.1 and Table 3.2. It can be observed that 23 individuals had mild hearing loss, while 14 of them had a moderate hearing loss. Two of the clients had hearing loss only in the low frequencies up to 1000 Hz, which did not exceed 40 dB HL at any frequency.

Table 3.1

Demographic details of the individuals with auditory dys-synchrony

Degree of hearing loss	**No. of subjects**	**Mean age in years**	**Age range**	**Gender**	
				Male	**Female**
Mild	23	19.78	14 – 28	06	17
Moderate	14	18.28	14 – 24	09	05
Low frequency hearing loss	02	24	20 – 28	02	00

Table 3.2

Audiological findings of individuals with auditory dys-synchrony with the mean value given within brackets

Degree of hearing loss	**SIS**		**Configuration of Audiogram**		
	Right ear	Left ear	Flat	Rising	Peak
Mild	0 – 85 (52.17)	0 – 85 (53.91)	03	08	12
Moderate	0 – 85 (48.21)	0 – 85 (49.64)	02	07	05
Low frequency hearing loss	80 – 85 (82.5)	75 – 80 (77.5)	–	02	–

SIS: Speech identification scores

3.1.2. Normal hearing group

The normal hearing group consisted of 39 participants with a mean age of 18.98 years and an age range of 16 to 26 years. It was ascertained from a structural interview that none of these participants had difficulty in understanding speech in daily listening conditions, and that they did not have any history of neurologic or otologic problems. The participants included in this group had the following audiological findings:

- Pure-tone hearing thresholds within 15 dB HL at octave frequencies between 250 Hz to 8000 Hz and 250 Hz to 4000 Hz for air conduction and bone conduction respectively,

- Speech identification scores of 95 to 100% at 40 dB SL (ref: average hearing thresholds at 500 Hz, 1000 Hz and 2000 Hz), determined using the Common Speech Discrimination Test Material for Indians developed by Mayadevi (1978),
- 'A' type tympanogram with ipsilateral and contralateral reflexes at normal hearing levels,
- Normal TEOAEs amplitude, and
- Speech identification scores above 80% in a speech-in-noise test, determined using the test material developed by Mayadevi (1978), presented at 40 dB SL in the presence of speech noise with a signal-to-noise ratio of 0 dB.

3.2. Instrumentation

- A Pentium IV personal computer, with software to generate sound developed by Yost (2000), was used for the generation of stimuli, used for the behavioural discrimination task, gap detection threshold and temporal integration function. The same computer was also used to present the generated signals.
- The Audio Lab software-version 2 (Voice and Speech system, India) was utilized to make the stimulus pair and also to normalize the stimuli.
- A calibrated audiometer (GSI 61) with TDH 50P headphones and B-71 bone vibrator was used for the estimation of pure-tone AC and BC thresholds. To obtain the just noticeable difference for intensity in a sound field condition, the signal was routed through an impedance matched speaker.

- A calibrated audiometer (MA 53) with an impedance matched loudspeaker (FF) AL-5 was used to obtained pure-tone threshold, and monitor the presentation level during psycho-acoustical tests in the sound field situation.
- An Interacoustic calibrated clinical audiometer (AC-40) with TDH-39 was made use of to obtain Masking Level Difference (MLD) values.
- Tympanogram and acoustic reflex thresholds were determined through a calibrated immittance (GSI Tympstar) instrument.
- ILO 292- DP Echoport was utilized to measure Otoacoustic emissions.
- The Intelligent Hearing Systems, Smart-EP (Version 2-12c) with an ER-3A insert earphone was used to elicit and record ABR.

3.3 Speech material

The standardized Common Speech Discrimination Test Material developed for Indians by Mayadevi (1978) was utilized to obtain speech identification scores and also for the speech-in-noise test. The test containing 20 nonsense CVs, was randomised to form four equivalent lists to avoid familiarity playing a role.

3.4. Test environment

A sound treated audiometric room was used for psycho-acoustical tests. The noise level in the room was as per the specification given by ANSI S3.1- (1996).

3.5. Procedure for subject selection

The participants were selected based on the following tests:

- A systematic detailed case history was obtained from all the participants which included information regarding demographic details, ability to understand speech in different situations, and symptoms related to general health and hearing.
- Pure-tone testing was done using a modified version of the Hughson-Westlake method (Carhart & Jerger, 1959). Air-conduction thresholds were obtained at octave frequencies from 250 Hz to 8000 Hz and bone conduction thresholds from 250 Hz to 4000 Hz, for each ear. The better ear was tested first for those who reported an ear to be better. In the remaining participants, half were initially tested in the right ear and other half in the left ear.
- Speech identification score was obtained using phonetically balanced monosyllables developed by Mayadevi (1978) at 40 dB SL under the earphones, for each ear independently. Half of the participants were initially tested in the right ear and other half in the left ear to avoid any ear effect.
- The speech-in-noise test was administered at 40 dB SL under the earphones using the speech material developed by Mayadevi (1978). All the participants in normal hearing group and the subjects with auditory dys-synchrony who had good speech identification scores in quiet underwent the speech-in-noise test. Speech identification scores were obtained at 0 dB SNR in the presence of speech noise. Half of the participants were tested in the right ear initially and rest in the left ear. The oral responses of the participants were noted and scored as correct or wrong.

- Immittance evaluation (tympanometry and reflexometry) were carried out using a 226 Hz probe tone frequency with a calibrated middle ear analyzer (GSI Tympstar). The tympanogram was obtained with the pressure varying from +200 dapa to -400 dapa. Stapedial acoustic reflexes were obtained for 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz tones. Only those who got normal findings were subjected to further evaluation.
- Auditory brainstem responses testing were carried out using Intelligent Hearing System evoked potential system. An identical protocol was adopted with all the participants as given in Table 3.3. Auditory brainstem responses were recorded at least twice in each participant to ensure wave reproducibility. The test was carried out to find out the presence or absence of responses.

Table 3.3

Stimulus and acquisition parameters used for ABR recording

Stimulus parameters		**Acquisition parameters**	
Stimulus:	Clicks	Filter:	30 – 3000 Hz
Polarity:	Rarefaction	Montage:	Cz-A1 and Cz-A2
Level:	90 dB nHL	Time window:	15 ms
Duration:	100 μs	Artefact rejection:	>50 μv
Transducer:	Electrically shielded head phones		-
Number of sweep:	1500		-
Rate:	11.1/s		-

- Transient evoked otoacoustic emissions (ILO 292) were measured in a sound treated room for non-linear clicks at 80 dB ± 5 dB peSPL. A total number of 256 non-linear clicks were presented. An emission was considered to be present if the waveform reproducibility was more than 50% and the overall signal-to-noise ratio was more than 6 dB SPL at least at two frequency bands. This physiological test (OAE) measurements was carried out to confirm outer hair cell functioning.

All the above test results were analyzed to confirm presence or absence of auditory dys-synchrony. A client was considered to have auditory dys-synchrony if speech identification scores in quiet or noise were poor and if auditory brainstem response was absent and TEOAEs were present. Only such clients were included in the clinical group for further research.

3.6. Parameters tested

The fine-grained behavioural discrimination abilities of the following acoustical parameters were evaluated for:

- Frequency at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz at 10 and 40 dB SL,
- Intensity at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz at 10 and 40 dB SL, and
- Duration at 1000 Hz for two anchor duration stimuli of 50 ms and 500 ms at 10 and 40 dB SL.

In addition, the gap detection threshold in white noise was also obtained at 10 and 40 dB SL. Temporal integration function using different stimulus duration and the MLD value were also determined.

3.7. Procedure for stimulus generation

For the measurement of behavioural just noticeable difference (JND) for frequency and duration and also gap detection threshold, stimuli were generated. Three programs to generate simple tones and complex sounds, developed by Yost (2000) were used to generate the signals with a sampling rate of 44.1 kHz and a resolution of 16 bits. The Audio Lab V.2 software was used to make paired stimuli for use in an AX design. 'A' was an anchor stimulus in a pair that did not vary with respect to frequency, intensity or duration for the frequency discrimination task, intensity discrimination task or duration discrimination task respectively. 'X' was the variable tone that was varied either in terms of frequency, intensity, or duration, depending on the task involved. The inter-stimulus interval within a pair was 500 ms. Each pair was then saved as independent wave files in a Pentium IV personal computer. The variable tone for each task differed along a continuum in order to determine the JND for frequency, intensity, or duration. To evaluate the gap detection threshold, the stimuli were generated in a similar manner, except that instead of a tone a 1000 ms duration white noise was used.

The details of how the stimuli were generated for the frequency discrimination, duration discrimination, gap detection and temporal integration tasks are further described below. Signals for the intensity discrimination task were not separately generated. These stimuli were directly presented from the GSI-61 audiometer.

3.7.1. Frequency discrimination task

For the measurement of JND for frequency, pure-tones of 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz were generated as anchor stimuli. For each anchor stimulus a continuum

of variable tones were generated which were higher in frequency than the anchor stimulus. The both anchor and variable tones for all the stimuli had a duration of 1000 ms with a rise and fall time of 10 ms. The difference between the anchor and variable tones ranged from 10% to 0%. The step size for the initial pairs was more. As the variable tone approached the anchor tone, the step size reduced to 0.2% for the 500 Hz and 0.1% for the 1000 Hz, 2000 Hz and 4000 Hz anchor stimuli. The final pair had a difference of 0 Hz (0%) for all frequencies. These variations in frequency were selected as Moore (1995) reported that the frequency discrimination threshold was approximately 0.1 to 0.2% for normal hearing individuals. Thus, several pairs of stimuli were generated, with one signal of the pair being the standard and other being the deviant for each frequency. The details of the stimulus pairs which were generated and used to obtain JND for different anchor tones are given in the Table 3.4.

3.7.2. Duration discrimination

Two different duration anchor tones, having a frequency of 1000 Hz, were generated. While one had a total duration of 50 ms the other had a duration of 500 ms. Both had a rise and fall time of 10 ms. The 1000 Hz variable tones also had a rise and fall time of 10 ms. For the 50 ms tone the difference between the anchor and variable tones ranged from 150 ms to 50 ms in decrements of 5 ms. Likewise, for the 500 ms tone this difference ranged from 1500 ms to 500 ms in decrements of 50 ms. A step size of 5 ms was used when the variable tones were less than 100 ms for the 50 ms anchor tone. Likewise the step size was 50 ms when the variable tones were less than 1000 ms for the

500 ms anchor tone. This procedure was similar to that adopted by Starr et al. (1991). Details of stimulus generation are outlined in the Table 3.5.

Table 3.4

Stimulus pairs used for the frequency discrimination task for each anchor tone

Anchor	**Variable tone**	**Anchor**	**Variable tone**	**Anchor**	**Variable tone**	**Anchor**	**Variable tone**
500 Hz	550Hz	1000 Hz	1100 Hz	2000 Hz	2200 Hz	4000 Hz	4400 Hz
500 Hz	540 Hz	1000 Hz	1075 Hz	2000 Hz	2175 Hz	4000 Hz	4300 Hz
500 Hz	530 Hz	1000 Hz	1060 Hz	2000 Hz	2150 Hz	4000 Hz	4275 Hz
500 Hz	525 Hz	1000 Hz	1050 Hz	2000 Hz	2125 Hz	4000 Hz	4250 Hz
500 Hz	520 Hz	1000 Hz	1040 Hz	2000 Hz	2100 Hz	4000 Hz	4225 Hz
500 Hz	515 Hz	1000 Hz	1030 Hz	2000 Hz	2075 Hz	4000 Hz	4200 Hz
500 Hz	512 Hz	1000 Hz	1025 Hz	2000 Hz	2060Hz	4000 Hz	4175 Hz
500 Hz	510 Hz	1000 Hz	1020 Hz	2000 Hz	2050 Hz	4000 Hz	4150 Hz
500 Hz	508 Hz	1000 Hz	1015 Hz	2000 Hz	2040 Hz	4000 Hz	4125 Hz
500 Hz	506 Hz	1000 Hz	1012 Hz	2000 Hz	2030 Hz	4000 Hz	4100 Hz
500 Hz	505 Hz	1000 Hz	1010 Hz	2000 Hz	2025 Hz	4000 Hz	4075 Hz
500 Hz	504 Hz	1000 Hz	1008 Hz	2000 Hz	2020 Hz	4000 Hz	4050 Hz
500 Hz	503 Hz	1000 Hz	1006 Hz	2000 Hz	2015 Hz	4000 Hz	4040 Hz
500 Hz	502 Hz	1000 Hz	1004 Hz	2000 Hz	2012 Hz	4000 Hz	4032 Hz
500 Hz	501 Hz	1000 Hz	1002 Hz	2000 Hz	2010 Hz	4000 Hz	4028 Hz
500 Hz	500 Hz	1000 Hz	1001 Hz	2000 Hz	2008 Hz	4000 Hz	4024 Hz
-	-	1000 Hz	1000 Hz	2000 Hz	2006 Hz	4000 Hz	4020 Hz
-	-	-	-	2000 Hz	2004 Hz	4000 Hz	4016 Hz
-	-	-	-	2000 Hz	2002 Hz	4000 Hz	4012 Hz
-	-	-	-	2000 Hz	2000 Hz	4000 Hz	4008 Hz
-	-	-	-	-	-	4000 Hz	4004 HZ
-	-	-	-	-	-	4000 Hz	4000 Hz

Table 3.5

Stimulus pairs used for the duration discrimination task for two different duration anchor stimuli

50 ms tone		**500 ms tone**	
Anchor	**Variable**	**Anchor**	**Variable**
50 ms	150 ms	500 ms	1500 ms
50 ms	125 ms	500 ms	1000 ms
50 ms	110 ms	500 ms	950 ms
50 ms	100 ms	500 ms	900 ms
50 ms	95 ms	500 ms	850 ms
50 ms	90 ms	500 ms	800 ms
50 ms	85 ms	500 ms	750 ms
50 ms	80 ms	500 ms	700 ms
50 ms	75 ms	500 ms	650 ms
50 ms	70 ms	500 ms	600 ms
50 ms	65 ms	500 ms	550 ms
50 ms	60 ms	500 ms	500 ms
50 ms	55 ms	-	-
50 ms	50 ms	-	-

3.7.3. Gap detection

For the gap detection test a 1000 ms duration white noise, with a rise and fall time of 10 ms, was generated. A continuum of signals, with varying durations of silence introduced within the centre of the white noise was developed. The duration of the silences was of 25 ms, 20 ms, 15 ms, 12 ms, 10 ms, 8 ms, 7 ms, 6 ms, 5 ms, 4 ms, 3 ms, 2 ms, 1 ms and 0 ms, without any rise or fall time at the gap. Thus, a total of 14 stimuli were generated with varying duration of silences to obtain the gap detection threshold.

The first stimulus had a silence of 25 ms and last stimulus had no gap. The stimulus duration of 1000 ms was selected since Forrest and Green (1987) reported that changes in the stimulus duration had little effect on gap-detection threshold.

White noise was selected instead of a pulse tone since it has been observed by Arlinger (1993) that when there was a sharp onset and offset in the pulse signal, broadening of the spectrum occurred. This broadening of spectrum resulted in an audible click being added to the beginning and end of the tone. This was found to cause an error in the response because a listener could detect the clicks without hearing the gap.

3.7.4. Temporal integration

Tone bursts at 1000 Hz were generated having durations of 20 ms, 50 ms, 100 ms, 200 ms, 300 ms and 400 ms, with a rise and fall time of 10 ms. These range of durations were selected which included stimuli which might not result in threshold variation (durations above 200 ms) as well as stimuli that might result in threshold variations (durations below 200 ms). It has been reported by Arlinger, (1993) that shorter duration tone pulses below 200 ms resulted in the hearing threshold level of normal hearing listeners decreasing by approximately 10 dB when the tone pulse duration was reduced by a factor of ten.

3.7.5. Intensity discrimination

To obtain the JND for intensity, a GSI- 61 diagnostic audiometer was used. The JND for intensity was obtained for 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz tones. Both the anchor stimulus and variable stimulus had a duration of 400 ms with a difference in

intensity. Each anchor was paired with several such variable tones with a gap of 400 ms. The anchor and variable tone differed by 20 dB to 0 dB. The initial test pair had a difference of 10 dB for the normal group and 20 dB for the group with AD. Thereafter it was varied in 1 dB steps in succession.

3.8. Procedure for psycho-acoustical tests

The stimuli generated for the frequency and duration discrimination tasks, gap detection threshold, and temporal integration test were saved in a Pentium 4 personal computer. These stimuli were store as wave files and played using Praat software from the computer. From the computer the signals were routed through the Maico MA-53 diagnostic audiometer. The out-put intensity of the signals were control by the audiometer and was presented through the single speaker placed at a 0° Azimuth at a distance of 1 meter and at the ear level for each participant. For the intensity discrimination task, the GSI-61 diagnostic audiometer was used. To obtained MLD value for both normal and clinical group, a standard MLD test was carried out using the Interacoustic AC-40 diagnostic audiometer with TDH-39 earphones. All the audiometers were calibrated objectively before the start of the data collection and every two months thereafter as per ANSI S3.6 - (1996). Subjective calibration was daily.

3.8.1. Stimuli presentation

Prior to the psycho-acoustical tests, sound field thresholds were obtained at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz for each of the participants through speakers. This was done to determine the level at which the signals were to be presented. The entire

psycho-acoustical study was carried out in two sittings with each session lasting for a duration of approximately 45 minutes to one hour. The JND for frequency, duration and temporal integration test was established in the first sitting and the JND for intensity, gap detection and MLD were obtained in the second sitting. Thus, a total of three sittings were required for each participant, the first being for audiological assessment for selecting the participants and the next two for the psycho-acoustical assessment. If the any participant showed any signs of fatigue or restlessness, further breaks were given within each test session.

3.8.2. Behavioural discrimination

Testing procedure for frequency discrimination

A fine-grained auditory discrimination for frequency was carried out using the developed material. This was done to determine the smallest difference that could be discriminated between the two frequencies that had differences as shown in Table 3.4. This task was carried out separately for the continua of 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz. Each participant was tested independently. The participants were comfortably seated in a sound treated room and the stimuli were presented through the loudspeaker. The participants were instructed to judge and indicate verbally whether the stimuli in a pair were same or different. Initially, each participant was familiarized with the task at least three to four times by presenting the first stimuli pair that had a maximum difference and the last pair that did not have any difference. Following this, the actual test items were presented. The initial pair of the test item for the normal group had a difference of 5% between the anchor and the variable tone, whereas for the group with

AD the difference was 10%. If a participant was able to differentiate the two, then the adjacent stimulus pair was skipped and the next pair was presented. This procedure continued till the subject failed to differentiate between the two stimuli in a pair. Once the subject was not able to identify the difference between the two stimuli then the earlier pair, with a larger difference, was presented. A two-down-one-up procedure was followed to trace the threshold. Near the threshold, catch trials having pairs with no difference were presented to eliminate false positive or negative responses. The order of the stimuli was counter balanced, wherein half of the participants were tested for the discrimination task from 500 Hz to 4000 Hz and other half was tested in the reverse order. The procedure was carried out at all the frequencies at 40 dB SL and 10 dB SL, with the higher intensity being presented first and then the lower. Stimuli were presented at equal sensation level to compensate for the audibility in each of the participants. The smallest discriminable difference between the anchor stimulus and variable tone with respect to frequency was noted for each frequency and each sensation level. A participant had to report that a stimulus pair was different on at least two out of the three/four trials, for a pair to be considered as the smallest perceptible difference. This smallest perceptible difference was considered to be the behavioural discrimination threshold for frequency.

Testing procedure for intensity discrimination

The fine-grained auditory intensity discrimination task was carried out using the GSI-61 diagnostic audiometer. The stimuli from the audiometer were presented through the impedance-matched loudspeaker kept at 0° Azimuth with a distance of 1 meter. The

audiometer was set such that pure-tones could be presented to the left speaker through both channels. Further, the special feature of the audiometer was set to enable the signals from the two channels be presented with an inter-stimulus interval of 400 ms through the same speaker. The intensity of the two stimuli was controlled using the attenuators of the two channels. The intensity of the anchor stimulus was kept constant and that of the variable tone altered.

As done with the other tests, initially each participant was familiarized with the task by presenting the stimuli pair that had a 20 dB difference and another pair that had a difference of 0 dB. Following this, the actual test was carried out. The initial pair of the test item that was presented to the normal group had a difference of 10 dB between the anchor and the variable tone, whereas for individuals with AD the difference was 20 dB. Half the participants were tested for the discrimination task from 500 Hz to 4000 Hz and other half from 4000 Hz to 500 Hz to avoid any order effect. The instruction and the procedure to obtain JND for intensity was similar to that were adopted for the frequency discrimination task. The procedure to obtain JND for intensity was done at all the frequencies at 40 dB SL first and then at 10 dB SL. The stimuli were presented at equal sensation levels to compensate for the difference in audibility among participants. A participant had to report that a stimulus pair was different on at least two out of three trials, for a pair to be considered as the smallest perceptible difference. This smallest perceptible difference was considered to be the behavioural discrimination threshold for intensity. The smallest discriminable difference between the anchor stimulus and variable tone with respect to intensity was noted for each frequency and each sensation level.

Testing procedure for duration discrimination

The fine-grained auditory duration discrimination task was carried out using the material developed in the present study (Table 3.5). This test was done to determine the smallest difference that could be discriminated between the two 1000 Hz tones which differed only in terms of duration. This task was carried out separately for the 50 ms and 500 ms anchor stimuli. The participants were instructed to judge and indicate verbally whether the stimuli in a pair were same or different. Initially each participant was familiarized with the task by presenting the stimulus pair that had the maximum difference and the last pair that did not have any difference. The first pair had a difference of 100 ms and 1000 ms between the anchor and variable stimulus for 50 ms and 500 ms tones respectively. The first test pair for the normal group had a difference of 50 ms for the 50ms anchor tone and 500 ms for the 500 ms anchor tone. However, the difference was 100 ms for the 50 ms anchor and 1000 ms for the 500 ms anchor tone for the group having AD. The instruction and the procedure followed to obtain JND for duration was the same that was adopted for the frequency discrimination task. The order of the stimuli was randomized, wherein half of the subjects were tested initially with the 50 ms anchor contrast and other half was tested with the 500 ms anchor contrast first. The procedure was done for both the anchor stimulus continua at 40 dB SL and then at 10 dB SL. The stimuli were presented at equal sensation level to compensate for the audibility differences across the participants. A participant had to report that a stimulus pair was different on at least two out of three trials, for a pair to be considered as the smallest perceptible difference. This smallest perceptible difference was considered to be the behavioural discrimination threshold for duration. The smallest discriminable

difference was noted for the two anchor stimuli and also the two presentation levels for each participant.

Testing procedure for gap detection

The generated material was used to establish the gap detection threshold for both the groups. This was done to determine the smallest silence that they could identify within a white noise. The participants were instructed to indicate verbally whether the noise was continuous or not. Initially, each participant was familiarized with the task by presenting the first stimuli pair that had the maximum difference and last pair that did not have any difference in order to enable them to perceive the contrast. These pairs were presented 3 to 4 times. The initial test pair used for normal hearing and group having AD had a silence of 25 ms. The procedure adopted to obtain gap detection threshold was similar to that used for determining the frequency discrimination task, i.e. a two-down-one-up procedure with catch trials near the threshold. The gap detection threshold was first obtained at 40 dB SL and then obtained at 10 dB SL. A participant had to identify the presence of a gap in the white noise at least two out of three trials for it to be considered as perceptible to him/her. The minimum perceptible silence in the white noise was considered as the gap detection threshold.

Testing procedure for temporal integration function

1000 Hz tone bursts having durations of 400 ms, 300 ms, 200 ms, 100 ms, 50 ms and 20 ms, that were stored in the Pentium IV computer, were presented. The stimuli were played, one at a time, using the Praat software. The out-put from the computer was

routed through an MA-53 diagnostic audiometer. This out-put was presented through the speaker which was kept at a distance of 1 meter and at 0° Azimuth from the participants. The intensity of the stimulus was monitor using the audiometer. The threshold was obtained for each of the durations (400 ms, 300 ms, 200 ms, 100 ms, 50 ms, & 20 ms) using the Modified Hughson-Westlake procedure (Carhart & Jerger, 1959). The test stimuli were varied in 1 dB steps to obtain the threshold. The behavioural threshold was always obtained first for the 400 ms duration stimulus followed by the 300 ms, 200 ms, 100 ms, 50 ms and 20 ms stimuli. The same procedure was adopted to obtain threshold for all duration stimuli. The minimum intensity, at which at least two out of three positive responses were observed in the ascending trials, was considered to be the behavioural threshold.

The threshold obtained at 400 ms was considered as the baseline threshold. The threshold obtained in subsequent duration stimuli i.e. 300 ms, 200 ms, 100 ms, 50 ms and 20 ms were subtracted from the threshold obtained at 400 ms. The difference was noted to know how much more intensity was required by each participants to detect the presence of the stimulus, at each stimulus duration.

Testing procedure for Masking Level Difference

MLD test was carried out using an Interacoustic AC-40 diagnostic audiometer with TDH-39 earphones. Each participant were presented binaurally with a narrow-band noise of 40 dB SL, centred around 500 Hz and 500 Hz pulsed tones with on and off time of 200 ms. The complete testing was done under headphones. The noise level was kept constant while obtaining the threshold for the pulsed tone in the presence of this noise,

using the Modified Hughson-Westlake procedure (Carhart & Jerger, 1959). The testing was done under the following conditions:

- Homophasic (NoSo), where both noise and signal were in phase in the two ears,
- Antiphasic (NoSπ), where the phase of the signal was reversed at the two ears, and
- Antiphasic (NπSo), where the phase of the noise was reversed at the two ears.

The intensity of the pulsed tone was varied in 1 dB steps to obtain the threshold. The pure-tone threshold in the homophasic and antiphasic conditions were noted. The threshold obtained in the homophasic condition was subtracted from that obtained in each of the antiphasic conditions. The antiphasic condition that was more deviant from the homophasic condition was utilized to calculate the MLD.

It was ensured that the instrumentation, test environment and procedure for all audiological tests were similar for both the groups of participants. Using the data collected from the two groups of participant, the following were determined:

- Just Noticeable Difference for frequency, intensity and duration in normal hearing individuals and individuals with AD, was obtained at different frequencies and at two sensation levels.
- Temporal integration function, gap detection threshold and MLD values obtained in normal hearing individuals and individuals with AD.

Appropriate statistical analysis using the Statistical Package for the Social Science-version 10 (SPSS) were carried out to get between group and within group comparison for all the psycho-acoustical tests results obtained across the parameters. Details of these analyses are discussed in the next chapter.

4. RESULTS AND DISCUSSION

The primary aim of study was to compare the performance of normal hearing individuals with individuals with auditory dys-synchrony on six different psycho-acoustical tests. The tests included fine-grained discrimination ability for frequency, intensity and duration; gap detection threshold; temporal integration; and masking level difference. The data from 78 participants (39 normal hearing & 39 with auditory dys-synchrony) were analyzed. Their psycho-acoustical responses were analyzed using the statistical package for social sciences (SPSS) software version 10.

The following analyses were carried out between groups:

- Descriptive statistics for all the parameters,
- Repeated measures mixed ANOVA was administered, for the comparision of data obtained from the psychophysical tests,
- Bonferroni's multiple comparison was done to test pairwise differences when the repeated measures ANOVA results were significant,
- Independent t-test was carried out to see group differences for all the parameters.

The within group analyses were done using the following statistical procedures:

- Separate one-way ANOVAs were done to see if a significant difference existed between the discrimination values obtained at different anchor frequencies for frequency and intensity discrimination. This was done for the two sensation levels for each of the acoustic parameters. It was also administered for the temporal integration function,
- Bonferroni's multiple comparison was carried out to check pairwise differences, if the repeated measure results were significant, and

- Paired t-test was carried out to determine whether a significant difference existed between the sensation levels for each acoustic parameter, within each group.

The results of the analyses are discussed under following headings:

4.1. Fine-grained behavioural discrimination of frequency

4.1.1. Fine-grained discrimination of frequency in the normal hearing group

4.1.2. Fine-grained discrimination of frequency in the clinical group

4.1.3. Comparison of difference limen for frequency (DLF) between the normal hearing and the clinical groups

4.2. Fine-grained behavioural discrimination of intensity

4.2.1. Fine-grained discrimination of intensity in the normal hearing group

4.2.2. Fine-grained discrimination of intensity in the clinical group

4.2.3. Comparison of difference limen for intensity (DLI) between the normal hearing and the clinical groups

4.3. Fine-grained behavioural discrimination of duration

4.3.1. Fine-grained discrimination of duration in the normal hearing group

4.3.2. Fine-grained discrimination of duration in the clinical group

4.3.3. Comparison of difference limen for duration (DLT) between the normal hearing and the clinical groups

4.4. Gap detection threshold

4.4.1. Gap detection threshold in the normal hearing group

4.4.2. Gap detection threshold in the clinical group

4.4.3. Comparison of gap detection threshold (GDT) between the normal hearing and the clinical groups

4.1. Fine-grained behavioural discrimination of frequency

The fine-grained behavioural discrimination responses for frequency (ΔF) and the ΔF/Fc% were calculated where 'ΔF' was the difference limen for frequency and 'Fc' the anchor frequency at which ΔF was obtained. These were analysed separately using descriptive statistics as well as ANOVA. This was done for both the normal hearing and

the clinical groups. Within each participant group, the data were analysed to check the influence of the four anchor frequencies and two presentation levels on ΔF/Fc%. Further, the behavioural discrimination responses of the clinical group were compared with the normal hearing group. The results of these analyses are presented in the following section.

A repeated measures mixed ANOVA (4 frequencies × 2 SLs × 2 groups) for ΔF/Fc% was administered to see the interaction between the variables. Frequency and sensation level served as the within subject variables and groups served as the between subject variable. The results revealed a highly significant main effect between the frequencies, [$F(3,228) = 204.94$, $p < 0.01$], frequencies and groups [$F(3,228) = 165.43$, $p < 0.01$], SLs [$F(1,76) = 986.43$, $p < 0.01$], SLs and groups [$F(1,76) = 138.47$, $p < 0.01$], frequencies and SLs [$F(3,228) = 14.63$, $p < 0.01$], frequencies, SLs and groups [$F(3,228) = 4.75$, $p < 0.01$] and between the groups [$F(1,76) = 373.98$, $p < 0.01$]. Bonferroni's pairwise comparison was done to check for any significant difference between the frequencies, irrespective of the groups. The results indicated that the ΔF/Fc% obtained at the four frequencies differed significantly from each other at the 0.01 level. As there was a significant difference between the groups, frequencies of the anchor stimuli and sensation levels, these findings were further analyzed and discussed separately.

4.1.1. Fine-grained discrimination of frequency in the normal hearing group

The mean and standard deviation of the fine-grained behavioural discrimination scores for the four frequencies and two sensation levels in individuals with normal

hearing are shown in Table 4.1. From Table 4.1 it can be noted that the mean just noticeable difference (JND) value increased with an increase in frequency. The JND value also increased as the level of presentation was reduced. Further, the participant-to-participant variation for fine-grained discrimination values were more at the high frequencies.

Tabe 4.1

Mean, standard deviation (SD), minimum and maximum ΔF in normal hearing individuals. Mean and SD of ΔF/Fc% are given within brackets

Frequency	Intensity	Mean	SD	Minimum	Maximum
500 Hz	40 dB SL	4.00 (0.8)	1.61 (0.32)	2.00	8.00
	10 dB SL	8.36 (1.67)	1.95 (0.39)	5.00	15.00
1000 Hz	40 dB SL	7.51 (0.75)	2.27 (0.23)	4.00	15.00
	10 dB SL	13.21 (1.32)	2.53 (0.25)	8.00	20.00
2000 Hz	40 dB SL	14.38 (0.72)	3.10 (0.16)	8.00	20.00
	10 dB SL	23.13 (1.16)	6.65 (0.33)	12.00	40.00
4000 Hz	40 dB SL	31.79 (0.79)	7.32 (0.18)	20.00	50.00
	10 dB SL	50.15 (1.25)	10.66 (0.27)	32.00	75.00

In addition, the increase in mean ΔF with increase in frequency, was evident at both sensation levels (Table 4.1). To check whether the frequency had any effect on ΔF/Fc% at each sensation level (40 dB SL & 10 dB SL), a one-way repeated measure ANOVA was carried out. The results indicated a significant difference across the frequencies at 40 dB SL [$F(3, 114) = 2.65$, $p < 0.05$] and at 10 dB SL [$F(3, 114) =$

49.56, $p < 0.01$]. Bonferroni's multiple comparison test was administered to see whether the mean difference was significant between the frequencies. The results indicated a significant difference only between 2000 Hz and 4000 Hz at the 0.05 level at 40 dB SL. No significant difference was observed between any other combinations of frequency at this presentation level (Table 4.2). On the other hand, mixed results were obtained at 10 dB SL. A significant difference at the 0.01 level was observed for all pairs of frequencies except between 1000 Hz and 4000 Hz as well as 2000 Hz and 4000 Hz (Table 4.3).

Table 4.2

Results of the Bonferroni's pairwise comparison of ΔF/Fc*% at 40 dB SL in the normal group*

Anchor frequency	**1000 Hz**	**2000 Hz**	**4000 Hz**
500 Hz	Not Significant $p > 0.05$	Not Significant $p > 0.05$	Not Significant $p > 0.05$
1000 Hz		Not Significant $p > 0.05$	Not Significant $p > 0.05$
2000 Hz			Significant $p < 0.05$

Table 4.3

Results of the Bonferroni's pairwise comparison of ΔF/Fc% *at 10 dB SL in the normal group*

Anchor frequency	**1000 Hz**	**2000 Hz**	**4000 Hz**
500 Hz	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
1000 Hz		Significant $p < 0.01$	Not Significant $p > 0.05$
2000 Hz			Not Significant $p > 0.05$

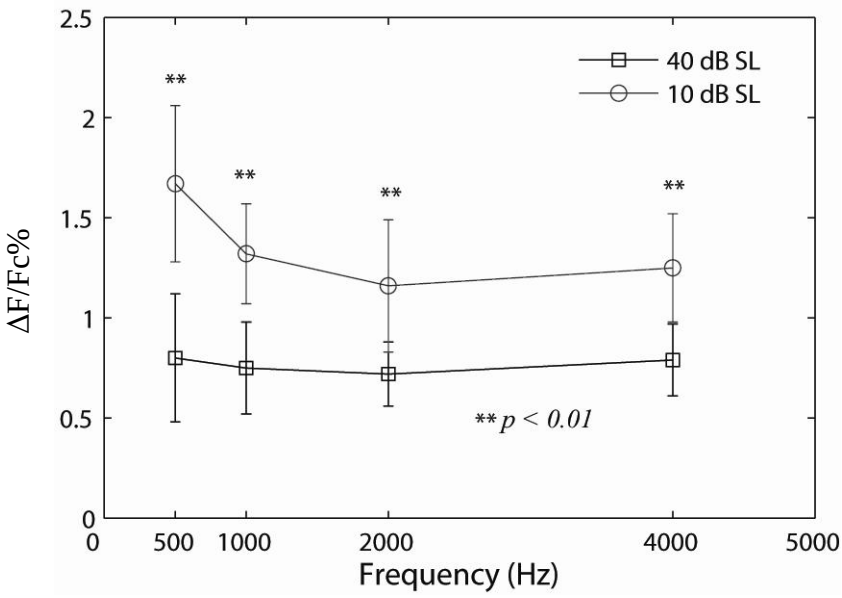

Figure 4.1. Mean and standard deviation of ΔF/Fc% in normal hearing participants, at 40 and 10 dB SL, across different anchor frequencies.

Further, the presentation level was found to influence ΔF/Fc%. With a decrease in presentation level, the ΔF/Fc% for frequency increased. The effect of sensation level was greatest at low frequencies and decreased at high frequencies. The ΔF/Fc% was almost a flat line when plotted as a function of frequency for the 40 dB SL, whereas a falling pattern was observed for the 10 dB SL (Figure 4.1).

To determine whether a significant difference in ΔF/Fc% values existed between the two sensation levels at a particular frequency, paired t-test was administered. The results indicated a significant difference at the 0.01 level for the ΔF/Fc% values obtained between the 40 dB SL and 10 dB SL at each of the frequencies in the normal hearing group. This is evident from Figure 4.1 and Table 4.4.

Table 4.4

t-value and significance level for ΔF/Fc% *obtained between the presentation levels in the normal hearing group*

Frequency	**Presentation level**	**t-value**
500 Hz	40 dB SL	28.38**
	10 dB SL	
1000 Hz	40 dB SL	27.33**
	10 dB SL	
2000 Hz	40 dB SL	10.89**
	10 dB SL	
4000 Hz	40 dB SL	23.04**
	10 dB SL	

** $p < 0.01$

4.1.2. Fine-grained discrimination of frequency in the clinical group

The mean and standard deviation of the fine-grained behavioural discrimination scores for frequency in individuals with auditory dys-synchrony were noted. This was done for all the frequencies (500 Hz, 1000 Hz, 2000 Hz & 4000 Hz) and also two intensity levels (40 dB SL & 10 dB SL). ΔF/Fc% values were also computed for which mean and standard deviation values were calculated. Table 4.5 depicts the results obtained from the clinical group.

Table 4.5

Mean, standard deviation (SD), minimum and maximum ΔF in the clinical group. Mean and SD of ΔF/Fc% are given within brackets

Frequency	Intensity	Mean	SD	Minimum	Maximum
500 Hz	40 dB SL	32.44 (6.49)	10.25 (2.05)	15.00	50.00
	10 dB SL	39.49 (7.9)	10.25 (2.05)	20.00	50.00
1000 Hz	40 dB SL	51.67 (5.17)	13.10 (1.31)	25.00	75.00
	10 dB SL	67.05 (6.71)	17.50 (1.75)	30.00	100.00
2000 Hz	40 dB SL	67.18 (3.36)	18.74 (0.94)	40.00	125.00
	10 dB SL	92.05 (4.6)	26.99 (1.35)	50.00	175.00
4000 Hz	40 dB SL	104.49 (2.61)	36.22 (0.91)	50.00	200.00
	10 dB SL	142.31 (3.56)	45.22 (1.13)	75.00	250.00

It can be seen from Table 4.5 that the mean JND values increased with an increase in the anchor frequency. The JND value also increased as the level of stimuli presentation was reduced. Further, the SD values were rather high across frequencies at

both presentation levels. Also, there was considerable overlap in the DLF values obtained at 40 dB SL and 10 dB SL between participants. This is evident from the minimum and maximum DLF values obtained at each presentation level, across anchor frequencies in Table 4.5.

In contrast, ΔF/Fc% showed a opposite trend. It reduced as the frequency increased at both sensation levels (40 dB SL and 10 dB SL). To check whether ΔF/Fc% values obtained at different frequencies differed significantly at 40 dB SL or 10 dB SL, one-way repeated measure ANOVA was done. The results indicated a significant difference in ΔF/Fc% values across the frequencies at 40 dB SL [$F(3, 114) = 168.68$, $p < 0.01$] and at 10 dB SL [$F(3, 114) = 167.19$, $p < 0.01$]. The significance of difference between frequencies was determined using Bonferroni's multiple comparison test. The results indicated a significant difference ($p < 0.01$) for the ΔF/Fc% values across the frequencies at both 40 dB and 10 dB SL presentation levels, in the clinical group. A falling pattern was noticed when ΔF/Fc% was plotted against frequency for both presentation levels (Figure 4.2).

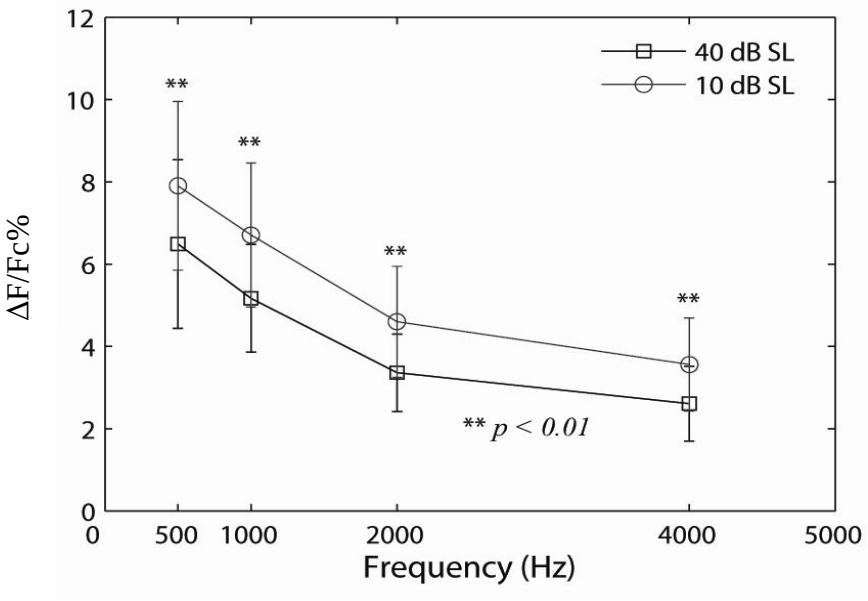

Figure 4.2. Mean and standard deviation of ΔF/Fc% in the clinical group obtained at 40 and 10 dB SL, across different anchor frequencies.

The presentation level also had an effect on the JND and ΔF/Fc% obtained at all four frequencies in the clinical group. The JND and ΔF/Fc% increased as the presentation level reduced. Paired t-test was administered to determine whether a significant difference occurred in the ΔF/Fc% values obtained at the two sensation levels at a particular frequency. The results indicated a significant difference ($p < 0.01$) in the ΔF/Fc% values obtained at 40 dB SL and 10 dB SL. This was observed at each of the frequencies, as shown in Figure 4.2 and Table 4.6.

Table 4.6.

t-value and significance level for DLF obtained between presentation levels in the clinical group

Frequency	Presentation level	t-value
500 Hz	40 dB SL	10.37**
	10 dB SL	
1000 Hz	40 dB SL	13.70**
	10 dB SL	
2000 Hz	40 dB SL	13.71**
	10 dB SL	
4000 Hz	40 dB SL	14.68**
	10 dB SL	

** $p < 0.01$

4.1.3. Comparison of DLF between the normal hearing and the clinical groups

It is evident from Tables 4.1 and 4.5 that the mean DLF value increased with an increase in frequency for both the normal hearing controls and the participants with AD. At each frequency, the mean DLF was also more for the 10 dB SL signal than that obtained for the 40 dB SL signal. Though both normal hearing individuals and individuals with AD followed a similar pattern, the latter group required much larger variations (in Hz) to perceive the difference in the two stimuli compared to the normal hearing individuals. This difference was more at the higher presentation level than at the lower presentation level. In comparison with those having normal hearing, the group with AD required almost 8.1, 6.9, 4.7 and 3.3 times more difference at the 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz anchor stimuli respectively, at 40 dB SL. This is evident from

the values shown in Tables 4.1 and 4.5. This difference between the two groups was lesser at the 10 dB SL. It was just 4.7, 5.1, 4 and 2.8 times more at the 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz anchor stimuli respectively, at 10 dB SL. When ΔF/Fc% were plotted against frequency, in individuals with AD, a similar pattern was observed at each sensation level (Figure 4.2). On the contrary, individuals with normal hearing showed almost a straight line at 40 dB SL and shallow sloping pattern at 10 dB SL (Figure 4.1). Further, this difference was more pronounced at the low frequencies, which can be seen in Figures 4.3 and 4.4.

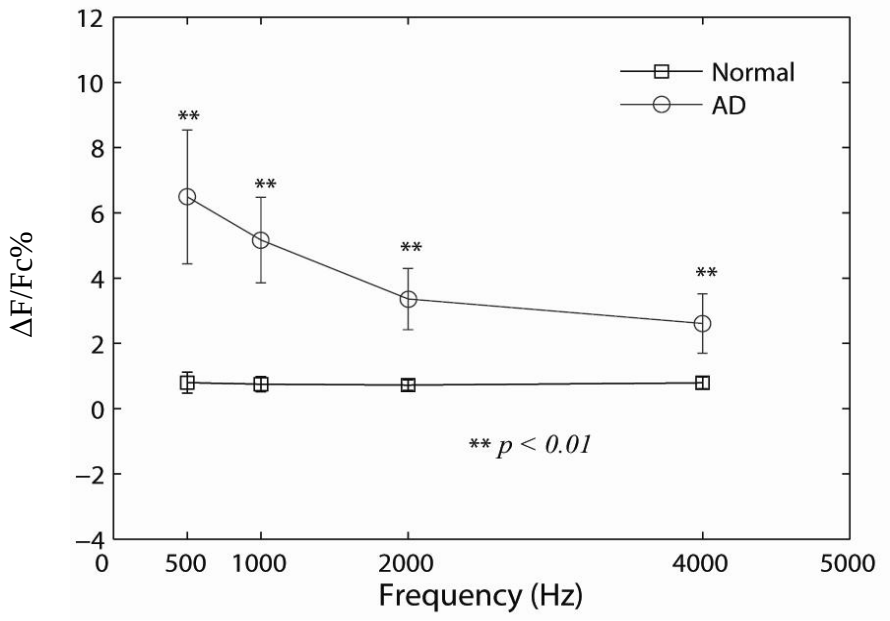

Figure 4.3. Mean, standard deviation and level of significance for ΔF/Fc% in the normal group and clinical group, at 40 dB SL, across different anchor frequencies.

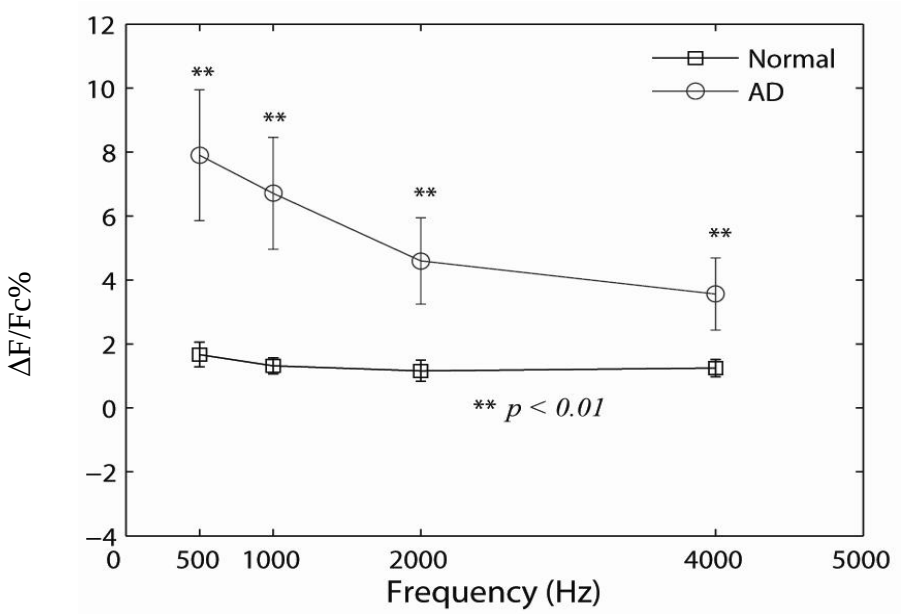

Figure 4.4. Mean, standard deviation and level of significance for ΔF/Fc% in the normal group and clinical group, at 10 dB SL, across different anchor frequencies.

To determine the significance of difference between the groups at each frequency and each sensation level separately, independent sample t-tests were done. A total of 8 t-tests were run. The independent t-tests indicated a statistically significant difference for ΔF/Fc% between the groups at the 0.01 level at both 10 dB and 40 dB SL (Table 4.7, Figures 4.3 & 4.4). This significant difference was seen for all the frequencies. The multiple t-tests did not increase type I error since a significant main effect was obtained at a 0.01 level on the repeated measure ANOVA.

Table 4.7

Mean, t-value and significance level for ΔF/Fc% *obtained between the participant groups*

Frequency	Participant group	Presentation level	Mean	t-value
500 Hz	Normal	40 dB SL	4.00	17.11**
	Clinical	40 dB SL	32.44	
	Normal	10 dB SL	8.36	18.64**
	Clinical	10 dB SL	39.49	
1000 Hz	Normal	40 dB SL	7.51	20.75**
	Clinical	40 dB SL	51.67	
	Normal	10 dB SL	13.21	19.02**
	Clinical	10 dB SL	67.05	
2000 Hz	Normal	40 dB SL	14.38	17.36**
	Clinical	40 dB SL	67.18	
	Normal	10 dB SL	23.13	15.48**
	Clinical	10 dB SL	92.05	
4000 Hz	Normal	40 dB SL	31.79	12.29**
	Clinical	40 dB SL	104.49	
	Normal	10 dB SL	50.15	12.39**
	Clinical	10 dB SL	142.31	

** $p < 0.01$

The results obtained in the *normal hearing* group in the present study is consistent with the results of the earlier findings reported in literature. Wier, Jesteadt, and Green

(1977) found that normal hearing individuals required just a few Hz difference in the low frequencies, that increased to several Hz in the higher frequencies. Sek and Moore (1995) also found that low frequency difference limens were significantly smaller. They observed that the JND was so small at low frequencies that it could not be explained by the place of excitation. Rather, they attributed this small JND mainly to the use of temporal information.

However, in the present study it was observed that when the difference across anchor frequencies was computed in terms of ΔF/Fc%, no significant difference was seen except between 2000 Hz and 4000 Hz at 40 dB SL in normal hearing individuals. Wier et al. (1977) also noted that the DLF as a function of frequency fell on a straight line when plotted as log (DLF) against √ (frequency).

The JND values obtained from the normal hearing group in the current study was also in close approximation with that reported by Starr, Picton, Sininger, Hood and Berlin (1996), Zeng, Oba, Garde, Sininger and Starr (2001) and Zeng, Kong, Michalewski & Starr (2005). However, Rance, McKey, Grayden (2004) got higher JND values than that obtained in the current study. This discrepancy in the findings could be mainly because of the difference in age groups evaluated in the two studies. While they studied children, the current study constituted of individuals above the age of 15 years. It has been reported by Lynne, Werner and Gray (1998) that the psycho-acoustical abilities reach a plateau at around 15 years of age.

Also, the JND obtained in the current study was slightly higher than that reported by Wier et al. (1977). While the mean JND in the present study varied from 4 Hz to 32 Hz for anchor stimuli of 500 Hz to 4000 Hz at 40 dB SL, Wier et al. got JNDs of

approximately 1 Hz to 18 Hz. This could be because the subjects who participated in their study had undergone training prior to the experiment. Unlike their study, none of the participants in the current study had undergone any formal training. In addition, the number of subjects taken in the current study was large, which resulted in a wider range due to inter-subject variability which is typically seen in a behavioural discrimination task. The difference could also be due to the difference in procedure adopted. They used a continuous pedestal method whereas in the current study a gated pedestal method was adopted to obtain DLF. Turner, Zwislocki and Filion (1989) reported better DLs for continuous pedestal method than the gated pedestal method. Despite this, most of the researchers prefer using a gated pedestal method (Starr et al., 1991; Starr et al., 1996; Zeng et al., 2001; Rance et al., 2004; Zeng et al., 2005).

In the current study, sensation level was found to affect ΔF/Fc% at all the anchor frequencies. The ΔF/Fc% obtained at the higher sensation level was lower than that obtained at the lower sensation level. The ΔF/Fc% values seemed to have a greater effect at the low freqencies than the higher frequencies. Wier et al. (1977) also observed a similar finding. In contrast, Kamath (1989) did not observe any significant difference in frequency modulated difference limen (FMDL) from 20 dB SL to 80 dB SL. This could be due to the use of modulated signals by her as opposed to gated signals in the present study.

A comparison of the findings in the *normal hearing individuals versus those with AD* in the present study, revealed that ΔF/Fc% was affected more at lower frequencies than that observed in the higher frequencies (Figure 4.3 & Figure 4.4) in indivduals with AD. Also, those with AD had significantly higher ΔF/Fc% when compared to normal

hearing individuals. This result is in close agreement with that reported in literature. Starr et al. (1991) reported in their single case with AD required almost 4 to 15 fold increase in frequency compared to normal hearing individuals. Starr et al. (1996) also reported that individuals with AD required much higher differences in frequency than that required by normal hearing individuals.

Like the present study, Zeng et al. (2001) and Zeng et al. (2005) also found that while the DLF in normal hearing individuals increased monotonically as the frequency increased, it was non-monotonic in individuals with AD. They too noted that those with AD were found to perform considerably poorer than individuals with normal hearing at frequencies below 2000 Hz. In the current study, it was observed that participants with AD got 8 times poorer scores than normal individuals, in a low frequency (500 Hz). This difference decreased to 3.3 times at a high frequency (4000 Hz). Rance et al. (2004) also found similar results. They too noted that the difference between normal hearing individuals and those with AD was more at 500 Hz (11 times poorer) and less at 4000 Hz (4.5 times poorer). Zeng et al. (2005) reported that this result pattern may reflect a disruption of the low frequency temporal discrimination processes in those with auditory dys-synchrony.

In the normal hearing individuals, in the present study, a significant difference across anchor frequencies was observed only between a few frequencies. This occurred at both sensation levels. In contrast, a significant difference across anchor frequencies was noticed on $\Delta F/Fc\%$ at both sensation levels in the individuals with AD. A similar pattern, was observed by Rance et al. (2004) in their subjects with normal hearing and the individuals with AD.

The difference in DLF obtained between normal hearing individuals and those with AD could be explained on the basis of differential mechanisms of frequency coding at high and low frequencies. For frequencies above 4 kHz, frequency discrimination is thought to be dependent on spatial changes in the excitation pattern along the basilar membrane (Sek & Moore, 1995). In contrast, discrimination of frequencies at or below 4 kHz is considered to be enhanced by the use of neural phase locking cues (Blackburn & Sachs, 1989; Goldberg & Brownell, 1973; Winter & Palmer, 1990). Also, Moore (2003) reported that the VIII nerve's phase locking mechanism for frequency decreases at about 1000 Hz to 2000 Hz and is absent above 4000 Hz to 5000 Hz. Possibly individuals with auditory dys-synchrony cannot use the phase locking cues to the same extent as normally hearing individuals. Hence, their performance in the higher frequencies that requires a lesser phase locking mechanism, is relatively less affected. Thus, it can be inferred that the low frequency discrimination problem observed in individuals with AD could be due to a temporal processing problem due to a disrruption in phase locking rather than a frequency coding problem.

Another reason for the increase in DLF in individuals with AD could be due to a leakage of signal to neighbouring fibers. Partial or complete loss of myelin has been noted to have profound effects on the generation and propagation of action potentials within the auditory nerve fibers. Demyelination has been found to result in an increase in membrane capacitance and a decrease in membrane resistance, leading to delayed excitation, a reduction in the velocity of action potential propagation and an increase in conduction vulnerability (McDonald & Sears, 1970; Rasminsky & Sears, 1972; Pender & Sears, 1984). This has been found to result in ephaptic transmission (cross-talk) between

fibers, with one active fiber setting off discharges in adjacent fibers (Starr, Picton & Kim, 2001). If this occurs in the auditory nerve fibers, there would be severe distortion in the coding of frequency.

In general, it can be observed that the findings of the present study are in agreement with that reported by earlier researchers. The results of the present study indicated that presentation level has a significant effect on DLF in the normal hearing individuals as well as individuals with AD. However, the effect was less for individuals with AD compared to normal hearing individuals as the latter group had an almost uniformly poor performance at both levels. The results also showed that ΔF/Fc% values were almost same across the frequency in the normal hearing individuals at 40 dB SL and there was a gradual improvement from the low to high freqencies at 10 dB SL. In contrast, in individuals with AD the improvement from low to high frequencies was more steep for both sensation levels. Hence, ΔF/Fc% if calculated across frequency at 40 dB SL would provide better information regarding frequency processing in individuals with AD. Thus, the findings of the present study suggests that individuals with AD have more problem at lower frequencies that at higher frequencies. This could be attributed to them having a problem in the phase locking mechanism, which is essential for frequency discrimination at low frequencies.

4.2. Fine-grained behavioural discrimination of intensity

The influence of various parameters on fine-grained behavioural discrimination of intensity were analysed separately. This was done for both the normal hearing and clinical groups independently. Within each subject group the data were analysed to check the influence of four frequencies and two intensities on JND for intensity. Further, the behavioural discrimination responses of the clinical group were compared with that of the normal hearing group. The results of these analyses are presented in this section.

Repeated measures mixed ANOVA for JND for intensity was administered, with frequency and intensity as within subject factors and groups as between subject factors. These 4 frequencies × 2 SLs × groups ANOVA revealed a highly significant main effect between the frequencies [$F(3,228) = 4.79$, $p < 0.01$], SLs [$F(1,76) = 1320.21$, $p < 0.01$], SLs and groups [$F(1,76) = 53.61$, $p < 0.01$], frequencies and SLs [$F(3,228) = 4.69$, $p < 0.01$], frequencies, SLs and groups [$F(3,228) = 6.35$, $p < 0.01$] and between the groups [$F(1,76) = 294.01$, $p < 0.01$]. However, no significant main effect was noticed between the frequencies and groups [$F(3,228) = 2.31$, $p > 0.05$]. Bonferroni's pairwise comparison was done to see the significance of difference between the frequencies irrespective of group. The results indicated a significant difference only between the DLI obtained at 500 Hz and 4000 Hz at the 0.05 level and between 2000 Hz and 4000 Hz at the 0.01 level. None other combinations of frequency showed a significant difference, which can be seen in Table 4.8.

Table 4.8

Results of the Bonferroni's pairwise comparison of JND for intensity

Anchor frequency	**1000 Hz**	**2000 Hz**	**4000 Hz**
500 Hz	Not Significant $p > 0.05$	Not Significant $p > 0.05$	Significant $p < 0.05$
1000 Hz		Not Significant $p > 0.05$	Not Significant $p > 0.05$
2000 Hz			Significant $p < 0.01$

4.2.1. Fine-grained discrimination of intensity in the normal group

The mean and standard deviation of the fine-grained behavioural discrimination scores for intensity in individuals with normal hearing were computed. This was done across four frequencies (500 Hz, 1000 Hz, 2000 Hz & 4000 Hz) and two presentation levels (40 dB SL & 10 dB SL), which can be seen in Table 4.9.

Table 4.9

Mean, standard deviation, minimum and maximum ΔI across four frequencies at two SLs in the normal hearing group

Anchor Frequency	Presentation level	Mean	Standard Deviation	Minimum	Maximum
500 Hz	40 dB SL	4.03	0.90	2.00	6.00
	10 dB SL	6.03	1.01	4.00	8.00
1000 Hz	40 dB SL	3.97	0.81	3.00	6.00
	10 dB SL	5.97	0.90	5.00	8.00
2000 Hz	40 dB SL	4.00	0.79	3.00	6.00
	10 dB SL	6.05	0.79	5.00	8.00
4000 Hz	40 dB SL	4.15	0.63	3.00	5.00
	10 dB SL	6.23	0.71	5.00	8.00

From Table 4.9 it can be noted that the mean JND value for intensity changed only marginally with an increase in frequency. The JND values increased slightly as the level of presentation was reduced. The DLI values fell almost on a straight line when ploted against frequency at both presentation levels, which can be seen in Figure 4.5. The participant-to-participant variation in fine-grained discrimination values were more at the high frequencies.

To check whether the JND values obtained at different frequencies differed significantly, a one-way repeated measure ANOVA was used at each presentation level. The results of the one-way ANOVA did not indicate any significant difference between the DLI obtained at different frequencies at 40 dB SL [$F(3, 114) = 0.66$, $p > 0.05$] and at

10 dB SL [F (3, 114) = 1.19, p > 0.05]. Since a significant difference was not obtained in the one-way ANOVA, Bonferroni's multiple comparision test was not administered.

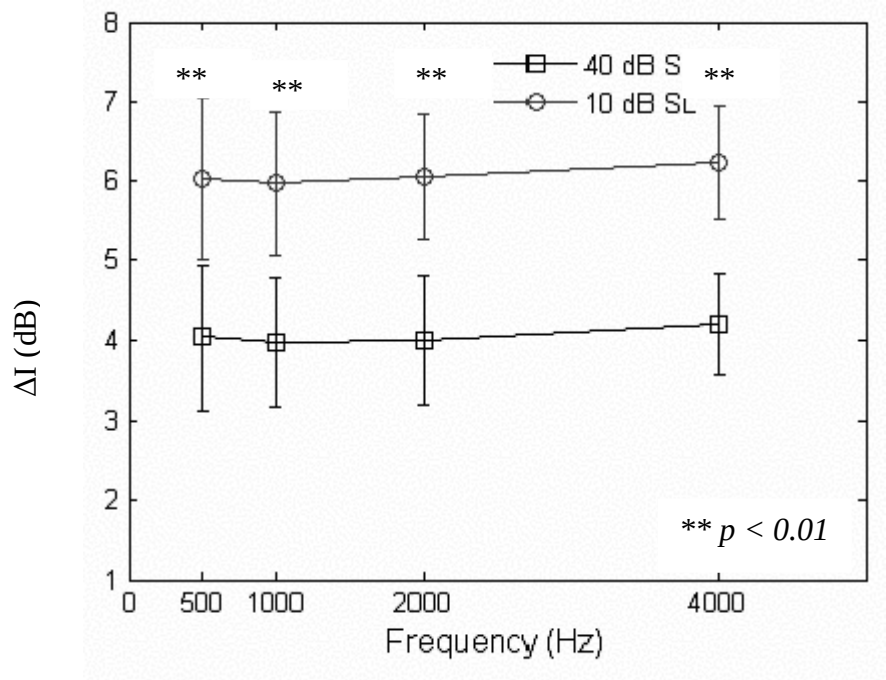

Figure 4.5. Mean and standard deviation of DLI in normal-hearing participants at 40 and 10 dB SL across different anchor frequencies.

Paired t-test was administered to know whether the DLI values obtained between the two sensation levels at a particular frequency differ significantly or not. The results indicated a significant difference at the 0.01 level in DLI values obtained at 40 dB SL and 10 dB SL at each frequency, within the normal hearing group (Table 4.10 & Figure 4.5).

Table 4.10

t-value along with significance level for DLI obtained between the presentation levels in normal hearing individuals

Anchor Frequency	Presentation level	t-value
500 Hz	40 dB SL	22.23**
	10 dB SL	
1000 Hz	40 dB SL	19.25**
	10 dB SL	
2000 Hz	40 dB SL	21.18**
	10 dB SL	
4000 Hz	40 dB SL	36.61**
	10 dB SL	

** $p < 0.01$

4.2.2. Fine-grained discrimination of intensity in the clinical group

The mean and standard deviation of the fine-grained behavioural discrimination scores for intensity in individuals with AD were derived. This descriptive statistics was done for all four frequencies and two intensity levels at which stimuli were presented. Table 4.11 depicts the results obtained from this group.

Table 4.11

Mean, standard deviation, minimum and maximum ΔI across four frequencies at two SLs in the clinical group

Anchor Frequency	Presentation level	Mean	Standard Deviation	Minimum	Maximum
500 Hz	40 dB SL	6.82	1.59	4.00	11.00
	10 dB SL	10.26	1.63	8.00	15.00
1000 Hz	40 dB SL	7.10	1.73	3.00	12.00
	10 dB SL	10.59	2.22	5.00	15.00
2000 Hz	40 dB SL	7.36	2.47	3.00	13.00
	10 dB SL	9.95	2.42	6.00	15.00
4000 Hz	40 dB SL	8.18	2.83	3.00	15.00
	10 dB SL	10.9	2.98	3.00	15.00

It can be observed from Table 4.11 and Figure 4.6 that the mean JND value for intensity increased slightly with an increase in anchor frequency in the clinical group. Also, the JND value increased marginally as the level of stimuli presentation was reduced. However, there was a lot of overlap in the DLI values obtained at 40 dB SL and 10 dB SL between one subject to another. The variation in fine-grained discrimination values were more for the high frequencies than for the low frequencies.

To check whether the JND values obtained at different frequencies differed significantly at each sensation level, one-way repeated measure ANOVA was carried out. The results indicated that behavioural discrimination for intensity differed significantly across frequencies at 40 dB SL [$F(3, 114) = 6.34$, $p < 0.01$] and also at 10 dB SL [F (3,

114) = 2.63, $p < 0.05$]. As the one-way ANOVA depicted a main effect for frequencies, Bonferroni's multiple comparison test was administered. The results of the Bonferroni's tests is seen in Table 4.12 and 4.13 for signals presented at 40 dB SL and 10 dB SL respectively.

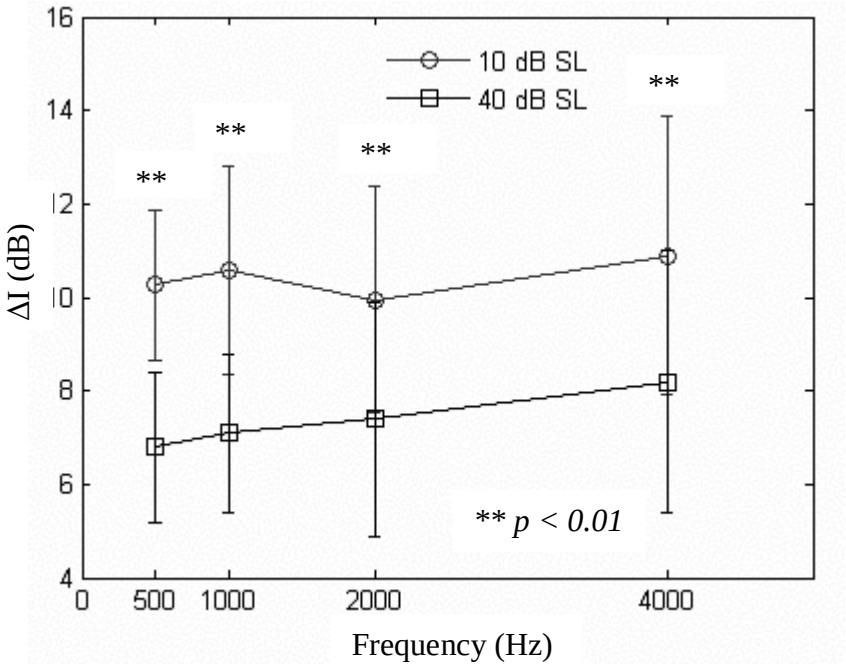

Figure 4.6. Mean and standard deviation of DLI in individuals with AD at 40 and 10 dB SL across different anchor frequencies.

Table 4.12

Results of the Bonferroni's pairwise comparison of JND for frequency at 40 dB SL in the clinical group

Anchor frequency	**1000 Hz**	**2000 Hz**	**4000 Hz**
500 Hz	Not Significant $p > 0.05$	Not Significant $p > 0.05$	Significant $p < 0.01$
1000 Hz		Not Significant $p > 0.05$	Significant $p < 0.05$
2000 Hz			Not Significant $p > 0.01$

Table 4.13

Results of the Bonferroni's pair wise comparison of JND for frequency at 10 dB SL in the clinical group

Anchor frequency	**1000 Hz**	**2000 Hz**	**4000 Hz**
500 Hz	Not Significant $p > 0.05$	Not Significant $p > 0.05$	Not Significant $p > 0.05$
1000 Hz		Not Significant $p > 0.05$	Not Significant $p > 0.05$
2000 Hz			Significant $p < 0.05$

Paired t-test was administered to check the significance of difference in DLI values obtained between the two sensation levels, at each frequency. The results indicated significant differences at the 0.01 level in DLI values obtained between the 40

dB SL and 10 dB SL at each of the frequencies in the clinical group (Table 4.14 & Figure 4.6).

Table 4.14

t-value along with significance level for DLI obtained between the sensation levels for the clinical group

Anchor frequency	Presentation level	t-value
500 Hz	40 dB SL	24.32**
	10 dB SL	
1000 Hz	40 dB SL	17.37**
	10 dB SL	
2000 Hz	40 dB SL	10.79**
	10 dB SL	
4000 Hz	40 dB SL	12.17**
	10 dB SL	

** $p < 0.01$

4.2.3. Comparison of behavioural discrimination for intensity between the normal hearing and the clinical groups

It is evident from Table 4.9 and 4.11 that the mean DLI increased slightly with an increase in frequency for the normal hearing controls and in individuals with AD. At each frequency, the mean DLI was also more at the 10 dB SL than that obtained at 40 dB SL. Though both normal hearing individuals and individuals with AD followed almost the similar pattern, the latter group required larger intensity differences to distinguish the

two stimuli compared to the former group. The difference in DLI obtained between the groups remained almost constant across the frequencies with just a marginal increase at the higher frequencies. This was observed for the signals presented at 40 dB SL (Figures 4.7) and the signals presented at 10 dB SL (Figures 4.8).

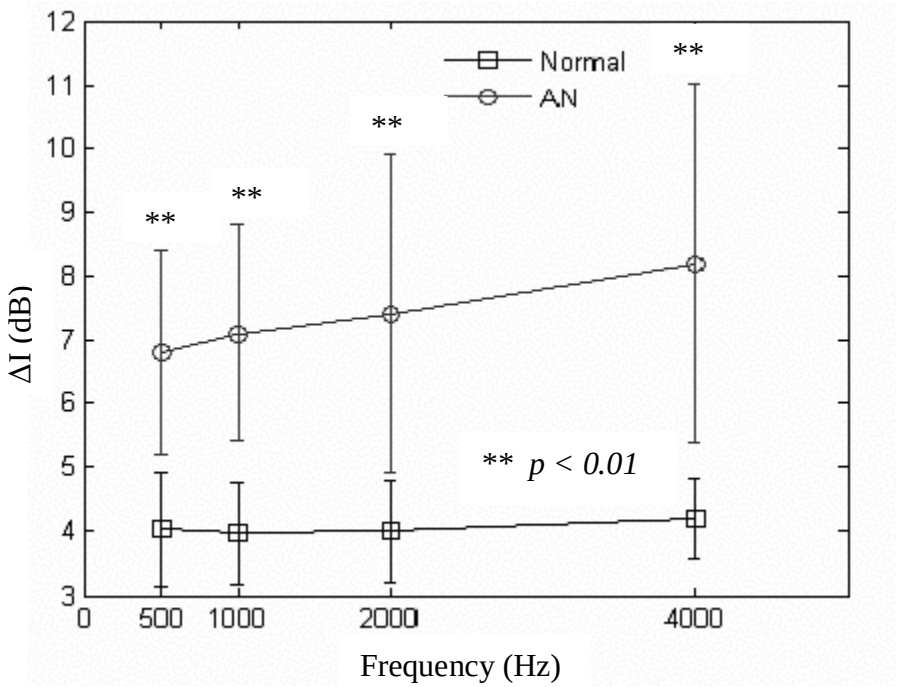

Figure 4.7. Mean and standard deviation for DLI in normal-hearing participants and individuals with AD at 40 dB SL across different anchor frequencies.

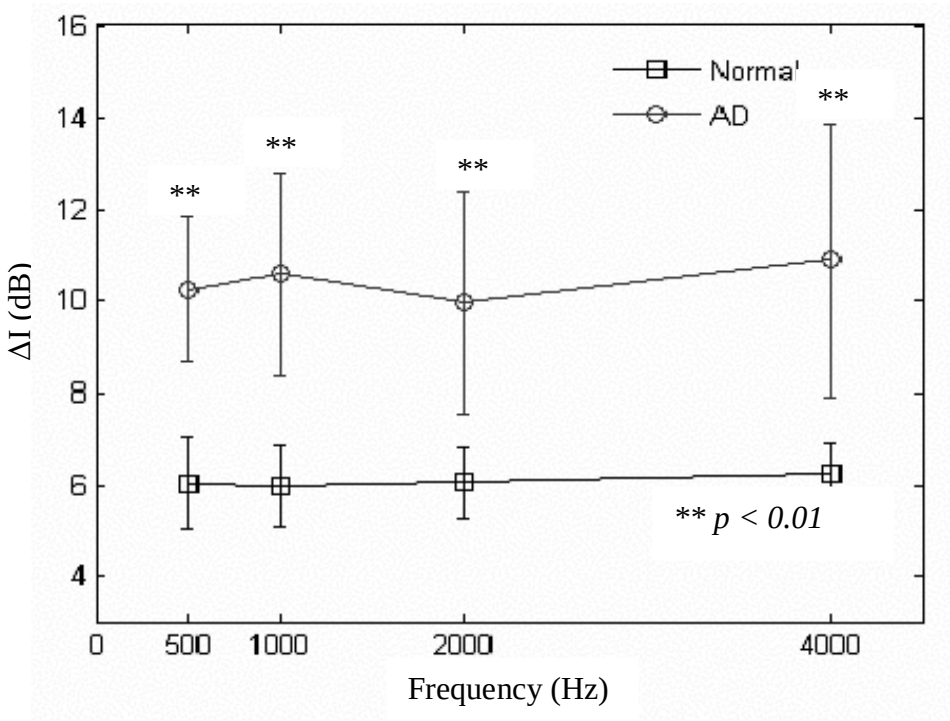

Figure 4.8. Mean and standard deviation for DLI in normal-hearing participants and individuals with AD at 10 dB SL across different anchor frequencies.

Independent sample t-tests were done to check for the significance of difference between the mean JNDs for intensity among the participant groups. This was done separately for each frequency and each sensation level. The independent t-tests revealed a statistically significant difference in DLI between the groups at the 0.01 level. This was evident at both sensation levels (10 dB and 40 dB) and all four anchor frequencies (Table 4.15). The multiple t-tests did not result in a type I error because a significant main effect at the 0.01 level was established in the repeated measure ANOVA for DLI.

Table 4.15

Mean and t-value along with significance level for DLI obtained between the participant groups

Anchor frequency	Participant group	Presentation level	Mean	t-value
500 Hz	Normal	40 dB SL	4.03	9.56**
	Clinical	40 dB SL	6.82	
	Normal	10 dB SL	6.03	13.75**
	Clinical	10 dB SL	10.26	
1000 Hz	Normal	40 dB SL	3.97	10.23**
	Clinical	40 dB SL	7.10	
	Normal	10 dB SL	5.97	12.02**
	Clinical	10 dB SL	10.59	
2000 Hz	Normal	40 dB SL	4.0	8.10**
	Clinical	40 dB SL	7.36	
	Normal	10 dB SL	6.05	9.57**
	Clinical	10 dB SL	9.95	
4000 Hz	Normal	40 dB SL	4.15	8.68**
	Clinical	40 dB SL	8.18	
	Normal	10 dB SL	6.23	9.52**
	Clinical	10 dB SL	10.90	

** $p < 0.01$

The results of the present study, obtained in the *normal hearing population* is in agreement with the results of studies reported in the literature. Several authors have found that normal hearing individuals required just a few dB difference to discriminate between two stimuli, irrespective of the frequency of the stimulus (Starr et al., 1991; Zeng et al., 2001; Zeng et al., 2005). Starr et al. (1991) reported that normal hearing individuals required almost 4 dB difference to differentitate between two 1000 Hz stimuli. Zeng et al. (2001) and Zeng et al. (2005) also obtained similar values. Like the present study, they also observed slight increase in ΔI values at a low sensation level in comparison to a high sensation level.

However, the JND obtained in the current study was slightly higher than that reported by Jesteadt, Wier, and Green (1977) and Starr et al. (1996). These researchers reported that normal hearing individuals had a DLI of less than 1 dB. This difference in finding could be attributed to the training they had provided to their participants. The variation in findings could also be due to the difference in procedure adopted. While they used a continuous pedestal method to obtain DLI, in the current study a gated pedestal method was adopted. Turner et al. (1989) compared the continuous pedestal method and gated methods and reported that the former method yeilded smaller DLIs. Despite the gated method resulting in larger DLIs, it is most commonly used method to obtain DLs. Starr et al. (1991), Zeng et al. (2001), Rance et al. (2004) and Zeng et al. (2005) adopted this technique to obtain DLs either for frequency or intensity.

The results obtained in *individuals with AD* showed higher DLI compared to normal hearing individuals, at both presentation level. These results obtained in the current study are in consonance with that reported in literature. Starr et al. (1991)

reported that the clients with AD in their study required almost 10 dB increment to differentiate between the two stimuli. Later Starr et al. (1996) also reported that their participants with AD required 3 dB and 6 dB increment for the right and left ear respectively to differentiate between two stimuli. However, in contrast Zeng et al. (2001) noticed that five of their subjects had normal DLI at higher sensation levels, whereas three of them required slightly higher ΔI for them to perceive the difference at low intensity levels.

In the current study, the intensity difference required by individuals with AD was higher at low sensation level than at high sensation levels. A similar observation was also noticed by Zeng et al. (2001) and Zeng et al. (2005) in their group of normal hearing individuals and individuals with AD.

The difference in DLI obtained between normal hearing individuals and those with AD can be explained based on the variations in physiology for intensity discrimination tasks. The increase in DLI in individuals with AD could be due to a lack of synchrony in firing of their nerve fibers. Partial or complete loss of myelin has been found to lead to the generation and propagation of action potentials within the auditory nerve fibers due to a process similar to what was described in the section on DLF (McDonald & Sears, 1970; Rasminsky & Sears, 1972; Pender & Sears, 1984). It has also been found that fibers that are demyelinated to differing degrees conducted neural signals at different speeds, thus resulting in loss of neural discharge synchrony. These phenomena have been reported to result in reduced amplitude and broadening of the compound action potentials (Starr et al. 2001; Rance, 2005). Hence, to have noticeable

changes in compound action potentials in individuals with AD, the intensity would have to be increased much more than that required for normal hearing individuals.

Further, it has been speculated by Starr et al. (2001) that individuals with AD could have axonal loss. This has been found to have different effect on compound action potentials than what was observed in cases with demyelinated disorder. Starr et al. (2001) and Rance (2005) reported that loss of axon would reduce the whole nerve action potential, without broadening of the compound action potentials. Thus, there may not be sufficient increase in compound action potential with an increase in intensity in clients with axonal loss. This lack of availability of the axons required to increase the whole nerve potential could result in the perception of low intensity sounds. Due to this, individuals with AD who have axonal loss would require more intensity to perceive the difference between two stimuli and also restrict their ability to improve their intensity discrimination at higher presentation levels.

Thus, it can be concluded from the findings of the present study on DLI, that individuals with AD required higher intensity differences to distinguish between two tones having the same frequency. This effect was seen at all frequencies and both the sensation level tested. However, the effect of frequency on DLI was negligible for both the groups as significant difference of DLI was noticed only between a few frequencies in the clinical group. These findings in the current study are in agreement with that reported in literature.

4.3. Fine-grained behavioural discrimination of duration

The fine-grained behavioural discrimination responses for duration in normal hearing and clinical group were computed separately using descriptive statistics. The difference limen for duration values obtained within each participant group were analysed to check the influence of duration of the stimuli (50 ms & 500 ms) and presentation levels (40 dB SL & 10 dB SL). The behavioural discrimination responses of the clinical group were also compared with the normal hearing group. The results obtained from the different statistical analyses are discussed in this section.

To determine the effect of the various parameters on JND for duration, a repeated measures mixed ANOVA (2 durations × 2 SLs × 2 groups) was done. The duration of the anchor stimuli and intensity served as within subject factors and the groups served as the between subject factors. The ANOVA results indicated a highly significant main effect between the durations, [$F(1,76) = 823.39$, $p < 0.01$], durations and groups [$F(1,76) = 49.06$, $p < 0.01$], SLs [$F(1,76) = 222.29$, $p < 0.01$], SLs and groups [$F(1,76) = 8.52$, $p < 0.01$], durations and SLs [$F(1,76) = 84.89$, $p < 0.01$], and between the groups [$F(1,76) = 74.09$, $p < 0.01$]. There was no significant main effect noticed between durations, SLs and groups [$F(1,76) = 1.34$, $p > 0.05$]. Since the repeated measures ANOVA indicated a significant main effect between durations, Bonferroni's pairwise comparison was done to determine the significance of difference in DL for duration between the two anchor duration stimuli. The result indicated that ΔT obtained for the two anchor stimuli that varied in duration were significantly different at the 0.01 level.

4.3.1. Fine-grained discrimination of duration in the normal hearing group

The mean and standard deviation of the fine-grained behavioural discrimination values for the two anchor stimuli that varied in duration were noted from those with normal hearing. This was done at two presentation levels. The results are depicted in Table 4.16.

Tabe 4.16

Mean, standard deviation, minimum and maximum ΔT across two anchor stimuli at two SLs in the normal hearing group

Anchor duration	Presnetation level	Mean	SD	Minimum	Maximum
500 ms	40 dB SL	133.33	36.87	100.00	200.00
	10 dB SL	174.35	41.15	100.00	250.00
50 ms	40 dB SL	26.79	4.36	20.00	35.00
	10 dB SL	34.74	5.72	25.00	45.00

It can be seen in the Table 4.16 that the mean JND values increased with an increase in duration of the anchor stimulus. Also, the JND value increased as the level of presentation was reduced. It was observed that there was approximtely a 1.3 fold increase in the ΔT when the presentation level was decreased from 40 dB SL to 10 dB SL. This was observed for the 50 ms and 500 ms anchor stimuli.

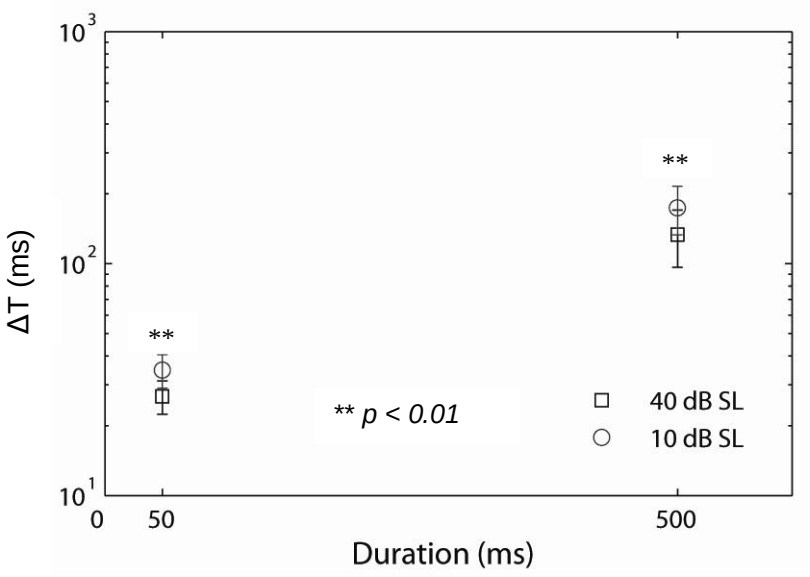

Figure 4.9. Mean and standard deviation of ΔT in normal hearing participants at 40 and 10 dB SL across two different durations of anchor stimuli.

To check whether the JND values obtained for the two different duration stimuli differed significantly from one another within and between presentation levels, paired t-tests were administered. The paired t-test result showed a significant difference between the ΔT values obtained for the 50 ms and 500 ms anchor stimuli at 40 dB SL [$t(38) = 18.52$, $p < 0.01$] and at 10 dB SL [$t(38) = 21.74$, $p < 0.01$]. The results also indicated a significant difference at the 0.01 level between the DL for duration values obtained at 40 dB SL and 10 dB SL. This was observed for both the anchor stimuli. Figure 4.9 and Table 4.17 provide these results.

Table 4.17

t-value along with significance level for DLI obtained between the levels in the normal hearing group

Anchor duration	Presentation level	t-value
500 ms	40 dB SL	9.21**
	10 dB SL	
50 ms	40 dB SL	15.57**
	10 dB SL	

** $p < 0.01$

4.3.2. Fine-grained discrimination of duration in the clinical group

From the data obtained from individuals with AD, the mean and standard deviation for the fine-grained behavioural discrimination scores for duration were calculated. This was done for both the anchor stimuli and also both intensity levels. Table 4.18 shows the results obtained from this group.

Table 4.18

Mean, standard deviation, minimum and maximum ΔT for the two anchor stimuli at two SLs in the clinical group

Anchor duration	Presentation level	Mean	SD	Minimum	Maximum
500 ms	40 dB SL	229.48	74.08	100.00	350.00
	10 dB SL	287.17	77.55	150.00	450.00
50 ms	40 dB SL	48.2	13.69	20.00	75.00
	10 dB SL	63.33	18.96	35.00	100.00

From Table 4.18 it can be observed that the mean ΔT values increased with an increase in duration of the anchor stimulus at which JND was obtained. The JND value also increased as the level of presentation reduced. There was an overlap between the ΔT values obtained at 40 dB SL and 10 dB SL among the participants, within each anchor stimulus. Further, the increase in ΔT values for the 50 ms anchor stimulus was approximately 1.32 times more at10 dB SL than that obtained at 40 dB SL. For the 500 ms anchor stimulus, it was marginally lower. The increase was approximately 1.25 times more at 10 dB SL than at 40 dB SL.

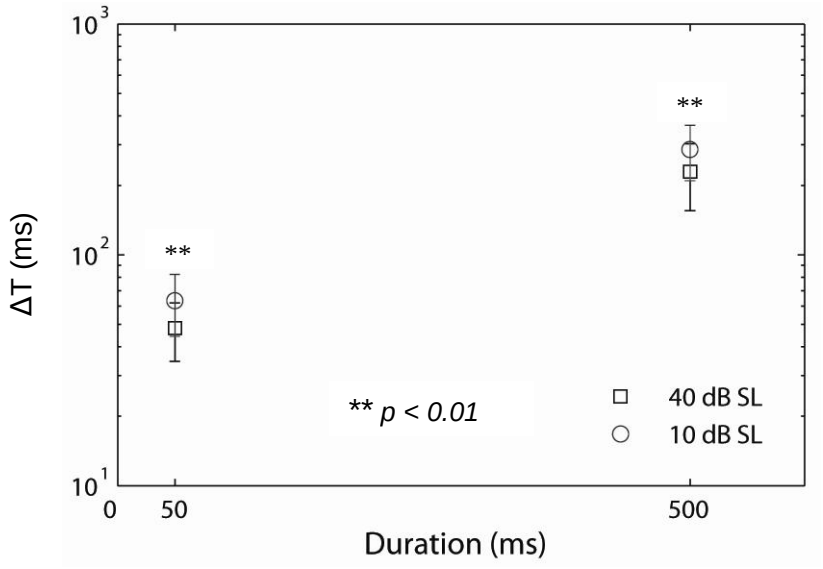

Figure 4.10. Mean and standard deviation of ΔT in individuals with AD at 40 and 10 dB SL across different anchor durations.

Paired t-test was done to check whether ΔT values obtained for the two anchor stimuli within and between the two presentation levels for the same duration anchor was significant. The results indicated a significant difference at the 0.01 level in ΔT values obtained between the two anchor stimuli at 40 dB SL [$t(38) = 17.39$, $p < 0.01$] and at 10 dB SL [$t(38) = 21.51$, $p < 0.01$], which can be seen in Figure 4.10. The results also showed a significant difference at the 0.01 level for the ΔT values obtained between the two presentation levels, for each stimulus duration, [$t(38) = 8.53$, $p < 0.01$] for the 500 ms anchor and [$t(38) = 13.11$, $p < 0.01$] for the 50 ms anchor stimuli.

4.3.3. Comparison of difference limen for duration between the normal hearing and the clinical groups

The ΔT values obtained in both groups followed a similar pattern. It can be observed in Tables 4.16 and 4.18 that the mean ΔT increased with an increase in duration of the anchor stimulus for both the normal hearing controls and also in individuals with AD. Also, in both groups the mean ΔT was more at 10 dB SL than that obtained at 40 dB SL, for each anchor stimulus. Further, both groups had an almost uniform increase in ΔT when the presentation level reduced from 40 dB SL to 10 dB SL. This increase was seen for both anchor stimuli durations. Though both normal hearing individuals and individuals with AD followed the similar pattern, the latter group required slightly larger differences (in ms) to perceive the difference in duration between the two stimuli. The relative difference reduces as the duration of the anchor stimulus at which ΔT was obtained increased. In comparison with the normal hearing group, the individuals with AD required almost 1.8 times more difference for the 50 ms anchor stimulus and 1.7 times more for the 500 ms anchor stimulus at 40 dB SL (Tables 4.16 & 4.18; Figure 4.11). With a reduction in presentation level to 10 dB SL, the difference did not change for the 50 ms anchor stimulus, but did change marginally for the 500 ms stimulus. It was approximately 1.8 times more for the 50 ms anchor stimulus and 1.6 times more for the 500 ms anchor stimulus at 10 dB SL (Table 4.16 & 4.18; Figure 4.12). This indicates that individuals with AD require greater duration differences between stimuli to differentitate them, if the duration of anchor stimulus is reduced.

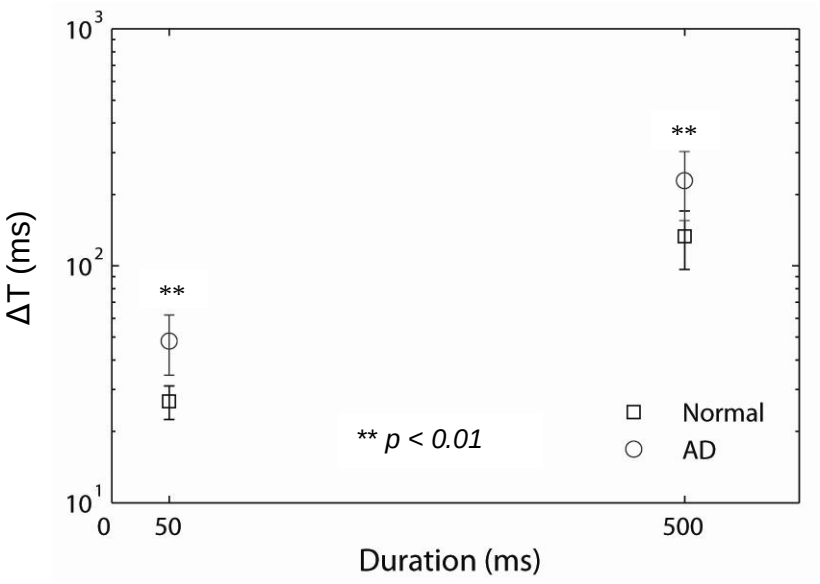

Figure 4.11. Mean and standard deviation of ΔT in normal hearing participants and individuals with AD at 40 dB SL across different anchor durations.

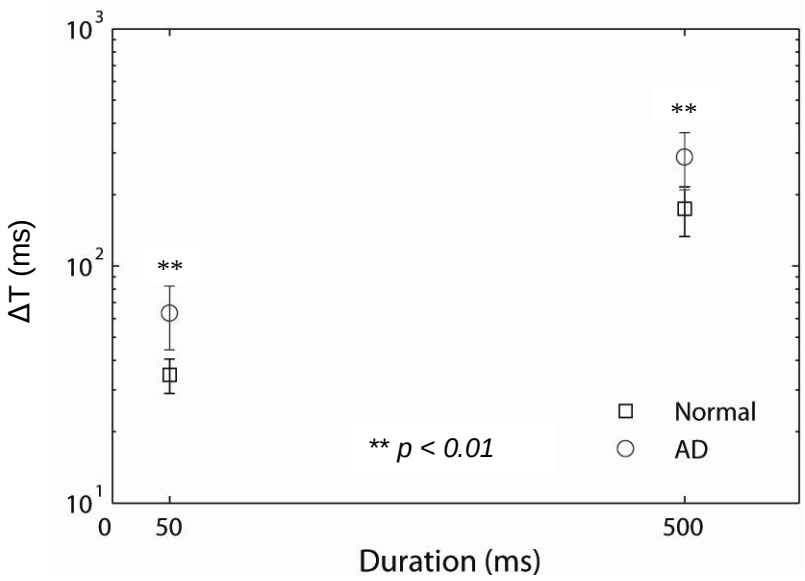

Figure 4.12. Mean and standard deviation of ΔT in normal hearing participants and individuals with AD at 10 dB SL across different anchor durations.

Independent sample t-test was done to know whether the JND for duration obtained for each anchor stimulus (50 ms & 500 ms) was significantly different between normal hearing and individuals with AD. This was done at both sensation levels. There was a statistcally significant difference between both participant groups at each anchor duration as well as each presentation level (Table 4.19). Since the repeated measure ANOVA for ΔT showed significant main effect at the 0.01 level, the multiple t-tests did not increase type I error.

Table 4.19

Mean and t-value along with significance level for ΔT obtained between the two participant groups

Anchor duration	**Participant group**	**Presentation level**	**Mean**	**t-value**
500 ms	Normal	40 dB SL	133.33	7.26**
	Clinical	40 dB SL	229.48	
	Normal	10 dB SL	174.35	8.02**
	Clinical	10 dB SL	287.17	
50 ms	Normal	40 dB SL	26.79	9.30**
	Clinical	40 dB SL	48.20	
	Normal	10 dB SL	34.74	9.01**
	Clinical	10 dB SL	63.33	

** $p < 0.01$

The results obtained in the current study showed that the ΔT increased with the increase in baseline duration of the anchor stimulus in *normal hearing individuals.* This

finding is in agreement with Creelman (1962), Abel (1972) and Starr et al. (1991). They also reported that the smallest detectable changes in duration between two stimuli increased with increase in baseline duration of the stimuli.

In the present study, the mean ΔT was 26.79 ms for the 50 ms tone and 133.33 ms for the 500 ms anchor at 40 dB SL. These values are similar to the data reported by Starr et al. (1991). They too observed that for a 50 ms duration tone, a mean increment of 20 ms was required by normal hearing individuals, while for a 500 ms duration tone they required a mean duration increment of 140 ms. Shylaja (2005) also reported that normal hearing individuals could differentiate 15 ms to 25 ms differences in duration for a 50 ms anchor duration at 40 dB SL. The values obtained by Shylaja were similar to the range got in the present study.

In contrast, the results obtained in the normal hearing group in the present study was slightly higher than what has been reported by Abel (1972). Abel found that for stimuli having baseline durations of 10 ms, 100 ms and 1000 ms stimuli, ΔT was about 4 ms, 15 ms and 60 ms respectively. The differences in values could be due to the method and stimuli used. Abel used noise bursts and unfiltered gated sinusoids of random phase at 1000 Hz. The participants were also provided with audible spectral cues from very short pulsed sinusoids. This probably resulted in a decreased ΔT.

Starr et al. (1996) also reported that for a 1000 Hz anchor stimulus of 750 ms duration with a rise and fall time of 5 ms, normal hearing individuals required 25 to 50 ms difference to identify the difference. Procedural variation could have contributed to the difference in ΔT, observed in their study.

Further, the present study showed an increase in ΔT with a decrease in presentation level. Moore (2003) also reported that ΔT increased at low sound levels. This indicates that temporal processing gets affected at lower presentation levels, in normal hearing individuals.

In addition, it was found in the present study that *individuals with auditory dys-synchrony* required higher duration difference for longer duration anchor stimuli. Also their ΔT value at the lower sensation level was more. The trend seen in the individuals with AD was similar to that displayed by the normal hearing individuals. Though the trend was similar, the individuals with AD required more duration differences to discriminate between the two stimuli compared to the normal hearing controls. This could be due to the lack of synchrony in the auditory nervous system. Reports in literature have highlighted that due to the dysynchronous firing, broadening of the compound action potentials occurs (Starr et al., 2001; Rance, 2005). This has been thought to result in a sensation of persistance, which might lead to feeling of an acoustic stimulus being present even after its cesation. This might have lead the clients with AD to perceive the stimulus longer than their actual duration. In turn, this would have lead them to require more differences between the anchor and variable tones. Such an effect was probably more for short duration stimuli, as slight variation in perception might have gone unnoticed in them. Thus, those with an AD required larger differences in duration for two signals to be perceptually different, compared to normal hearing individuals.

Hence, it can be concluded that ΔT increases with the increase in anchor duration in individuals with normal hearing as well as individuals with AD. However, the ΔT was significantly higher for individuals with AD for both duration anchor stimuli. Also, the

difference between normal hearing and those with AD was relatively less for the long duration anchor stimulus (500 ms) compared to the short duration anchor (50 ms). These findings are on par with the reports available in literature. The ΔT also reduced with the increase in presentation level for both participants group. However, no such effect has been reported in the literature in individuals with AD.

4.4. Gap detection threshold

The gap detection threshold (GDT) obtained from the normal hearing and clinical group was analysed using descriptive statistics. The effect of presentation level on gap detection threshold in each participant group, was also analysed. Out of the 39 individuals with AD, six of them could not identify a maximum gap of 25 ms in white noise at 40 dB SL and twelve of them could not identify it at 10 dB SL. While calculating mean and standard deviation, the scores obtained by those who could not identify the gap even at the maximum level were eliminated.

Following the analysis of data from each participant group, a comparision was made between them. In those who could not identify the maximum silence duration (25 ms) both at 40 dB and 10 dB SL, their GDT value was considered as 25 ms. This was done while determining the main effect or significance of difference of various parameters. To determine whether there was a statistically significant difference between GDT obtained at different SLs and from different participant group, a repeated measure ANOVA was carried out. This (2 SLs × 2 participant group) ANOVA was carried out, with the sensation levels being the within subject factor and participant group being the between group factor. The ANOVA results revealed a highly significant main effect between the SLs [$F(1,76) = 454.62$, $p < 0.01$], SLs and groups [$F(1,76) = 15.82$, $p < 0.01$], and between the groups [$F(1,76) = 204.55$, $p < 0.01$]. On account of the significant main effect, the data obtained were further analysed. This is discussed below.

4.4.1. Gap detection threshold in the normal hearing group

The mean and standard deviation of the gap detection threshold in individuals with normal hearing were computed. In the normal hearing group a mean GDT of 3.51 ms was obtained at 40 dB SL. The standard deviation was 0.72. While the minimum that could be detected by the normal hearing individuals at 40 dB SL was 2 ms, the maximum was 5 ms. At 10 dB SL the mean GDT increased sharply to 9.38 ms with a standard deviation of 1.58. The range of GDT at this sensation level was 7 ms to 12 ms. The same can be seen in Figure 4.13.

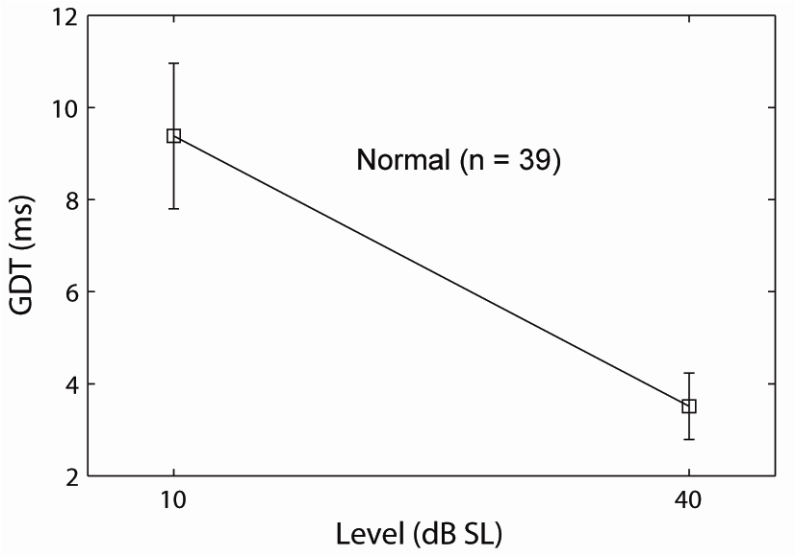

Figure 4.13. Mean and SD of gap detection threshold at 10 and 40 dB SL in normal hearing individuals.

From Figure 4.13, it can be noted that the mean GDT value increased sharply with a decrease in presentation level. The GDT increased approximately 3 times when the sensation level was dropped from 40 dB SL to 10 dB SL. To determine whether the

variation in GDT was statistically significant, a paired t-test was administered. The results indicated a significant difference at the 0.01 level [t (38)= 30.11, $p < 0.01$] between the GDTvalues obtained at 40 dB SL and 10 dB SL.

4.4.2. Gap detection threshold in the clinical group

The mean gap detection threshold calculated for 33 individuals with AD at 40 dB SL was 14.66 with a standard deviation of 4.55, while at 10 dB SL it was 18.18 with a standard deviation of 4.28 for 27 individuals. A minimum GDT that could be detected by the clinical group was 8 ms and 10 ms at 40 dB SL and 10 dB SL respectively. However, the maximum gap of 25 ms could not be detected by six individuals at 40 dB SL and by twelve individuals at 10 dB SL. Figure 4.14 depicts the results obtained from this group.

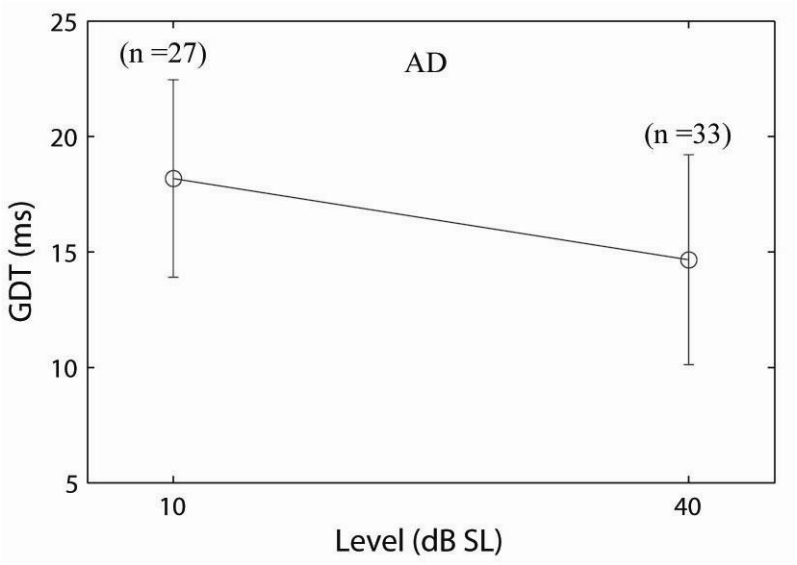

Figure 4.14. Mean and SD of gap detection threshold at 10 and 40 dB SL of the clinical group.

It can be seen in the Figure 4.14 that the mean GDT value increased gradually when the presentation level was reduced. There was an overlap in GDT values obtained at 40 dB SL and 10 dB SL across participants. The increase in GDT value was approximately 1.24 times more at 10 dB SL than at 40 dB SL. This indicates that the individuals with AD did not get much benefit in perceiving temporal gap detection by increasing the presentation level. To check whether GDT values obtained at the two presentation levels differed significantly, paired t-test was adminstered. For the purpose of this calculation, those who could not detect a gap even at the maximum gap, were assigned a score of 25 ms. The results indicated a significant difference at the 0.01 level [$t(38) = 9.56$, $p < 0.01$] between the GDT values obtained at 40 dB SL and 10 dB SL.

4.4.3. Comparison of gap detection threshold between normal hearing and the clinical groups

The mean GDT increased with a decrease in presentation level in both the normal hearing controls and also the clinical population. Despite the similar trend followed by the two groups, individuals with AD required larger silence (in ms) to perceive the presence of a gap in white noise than that was required by the normal hearing individuals. The slope which can be seen in Figure 4.15 was shallower for the individuals with AD compared to the individuals with normal hearing. The individuals with AD required almost 4.2 times more silence to perceive as gap at 40 dB SL. In contrast, at 10 dB SL, individuals with AD required approximately 1.9 times longer gap than the normal hearing individuals. This suggests that a high presentation level did not improve the detection of silence in individuals with AD whereas, it did in individuals with normal hearing.

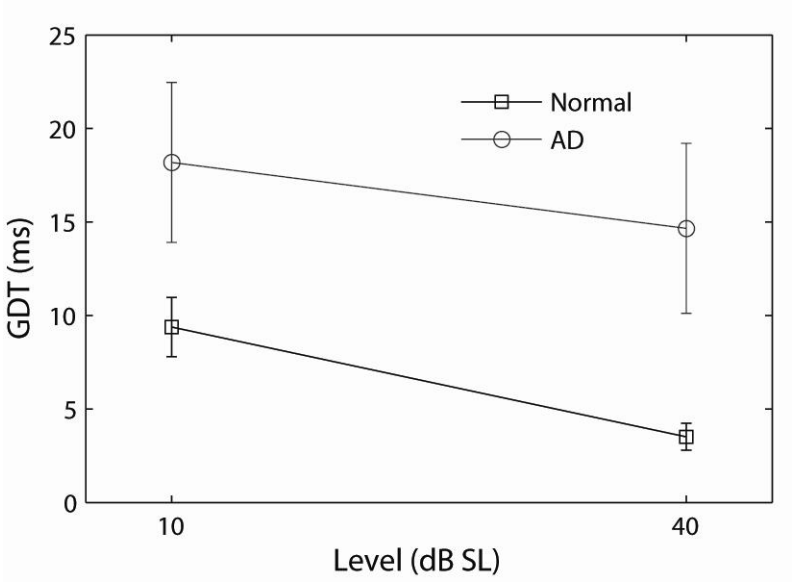

Figure 4.15. Mean and SD of gap detection threshold at two SLs for the normal hearing and clinical groups.

Independent sample t-tests were run to establish whether the mean GDT between the groups was significantly different or not. This was done at both sensation levels. The independent t-tests indicated statistically significant differences for GDT obtained between the groups at the 0.01 level for both 40 dB SL [$t(76) = 14.02$, $p < 0.01$] and 10 dB SL [$t(76) = 13.55$, $p < 0.01$].

Findings, similar to that obtained in the present study in *normal hearing individuals* have been earlier reported by Plomp (1964) and Penner (1977). They obtained a gap detection threshold of 2 to 3 ms at high SLs. This value was similar even for moderate to high levels. Later, Starr et al. (1991), Starr et al. (1996), Zeng et al. (1999, 2001) and Zeng et al. (2005) also reported of similar values for normal hearing

individuals. The findings of the present study are also in agreement with results of a study carried out on Indians by Shivaprakash (2003).

Further, in the present study, the gap detection threshold increased with a decrease in presentation level. Like the current study, Zeng et al. (1999, 2001) and Zeng et al. (2005) observed an improvement in GDT with an increase in presentation level. Moore (2003) also reported that the gap detection thresholds increased at low sound levels.

The comparison of *individuals having AD* with normal hearing individuals indicated that the clinical group exhibited higher gap detection thresholds both at 10 dB SL and 40 dB SL. Starr et al. (1991), Starr et al. (1996), Zeng et al. (1999, 2001), Zeng et al. (2005), and Michalewski, Starr, Nguyen, Kong, and Zeng (2005) also reported similar findings. They too reported that individuals with AD had higher GDT than normal hearing individuals. Starr et al. (1991) observed that individuals with auditory neuropathy could identify a silence of 15 to 20 ms for tones of 25 to 200 ms duration. Starr et al. (1996) also found that the gap detection threshold for a patient with AN was 6 ms and 12 ms for the right and left ear respectively.

A wide range of GDT value was observed in the present study. Michalewski et al. (2005) also reported of psycho-acoustical gap detection thresholds ranging from 5 ms to 40 ms in individuals with AD. Similarly, Zeng et al. (1999, 2001) and Zeng et al. (2005) also observed a wide range of GDT values. Zeng et al. (1999, 2001) reported that detection thresholds were 2-25 times greater for individuals with AD than the threshold obtained in normal hearing individuals. Zeng et al. (2005) also found similar differences between the groups, especially at higher sensation levels.

In the present study, an effect of presentation level on GDT was noticed in individuals with AD. Though the individuals with AD showed a significant increase in GDT with a change in sensation level, the difference in GDT was much smaller, unlike the normal hearing group. The results obtained in the current study concur with that reported in literature (Zeng et al. 1999, 2001; Zeng, 2005). The studies by Zeng and his group also noted that the GDT values were more at low sensation levels in individuals with AD. The slope of the GDT values they noticed in individuals with AD was much shallower than that observed in their normal hearing individuals.

One of the possible explanations for poor GDT in individuals with AD could be due to physiological changes that takes place in demyelinated fibres resulting in no synchronous firing. Such a phenomena in demyelinated neurons was also reported by McDonald and Sears (1970), Rasminsky and Sears (1972), Pender and Sears, (1984). Starr et al. (2001) also highlighted that variable slowing of action potential of each nerve fibre would result in reduced amplitude and broadening of the compound action potential. Thus, this might have resulted in persistence of the response at the neural level even during the silence period, leading to a larger GDT. This might be a reason why individuals with AD require larger gaps when compared to normal hearing individuals.

Another reason for poor GDT in clients with AD could be that with an increase in presentation level there may not have been sufficient increase in the average neural response which can be observed in normal hearing individuals (Rance, 2005). This could be due to variable slowing of each nerve conduction velocity (Starr et al., 2001). Further, axonal loss might result in insufficient increase in compound action potential with an increase in intensity, as the number of nerve fibres available to increase the compound

action potential may be less. Starr et al. (2001) and Rance (2005) also reported that in case of axonal loss the amplitude of compound action potential reduces. Thus, with the increase in intensity, individuals with AD might not perceive equally intense signals as normal hearing individuals do. This might have resulted in higher GDT in those with AD.

Yet another reason for the increased GDT in individuals with AD could be explained by the phenomenological model put forth by Zeng et al. (1999, 2001) and Zeng et al. (2005). They explained the increased gap detection threshold in those with AD based on desynchronized nerve conduction. The central representation of the gap is distorted, according to them, due to different delays and with reduced nerve conduction. Thus, the central representation of the gap would be difficult to detect because of its similarity to the background spontaneous activity.

From the above findings, it can be concluded that GDT increases with decrease in sound level both in normal hearing as well as in individuals with AD. The rate at which the GDT increases in individuals with AD is much shallower than that observed in normal hearing individuals. However, at any sound level presentation, individuals with AD show higher GDT than normal hearing individuals. Also, the gap required by individuals with AD is more at high sensation levels. The abnormal GDT obtained in individuals with AD could be attributed to physiological changes due to a demyelinated condition or axonal loss.

4.5. Temporal integration function

The differences between the baseline threshold, got for the 400 ms duration 1000 Hz tone, and the other five short duration tones (20 ms, 50 ms, 100 ms, 200 ms, 300 ms & 400 ms) were analysed. These six differences in threshold provided information regarding the temporal integration function of an individual. The six differences, which included the difference from the baseline (0 dB), were analysed separately using descriptive statistics as well as ANOVA for the normal hearing and clinical groups. Thus, the influence of duration of the stimulus on behavioural threshold on each participant group was obtained. In addition, a comparison was made between the responses got for each participant group. The results of these analyses are discussed further.

Initially a repeated measures ANOVA, for temporal integration function (6 stimuli duration × 2 participant groups) was administed. The duration of stimuli served as the within subject factors and the two participant groups served as the between subject factors. It revealed a highly significant main effect between the threshold shift noticed at different durations, [$F(5,380) = 789.41$, $p < 0.01$], durations and groups [$F(5,380) = 18.44$, $p < 0.01$], and between the groups [$F(1,76) = 11.57$, $p < 0.01$]. Bonferroni's pairwise comparison was done to determine the significance of difference between the threshold shift obtained for different duration stimuli, irrespective of group. The results indicated that threshold shift obtained as a function of duration of the stimulus differed significantly from each other except for the threshold shift obtained at 300 ms and 400 ms duration tones (Table 4.20).

Table 4.20

Results of the Bonferroni's pairwise comparison for temporal integration obtained for different duration tones for the normal and clinical groups

	50 ms	**100 ms**	**200 ms**	**300 ms**	**400 ms**
20 ms	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
50 ms		Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
100 ms			Significant $p < 0.01$	Significant $P < 0.01$	Significant $p < 0.01$
200 ms				Significant $p < 0.01$	Significant $p < 0.01$
300 ms					Not Significant $p > 0.05$

4.5.1. Temporal integration function in the normal hearing group

The mean and standard deviation of the threshold shift for different stimulus durations with reference to the threshold obtained for a 400 ms tone were computed. The results of the descriptive analysis is provided in Table 4.21. It is evident from the table that the 39 normal hearing individuals had marginal variations in thresholds for stimuli durations of 400 ms, 300 ms and 200 ms. A more noticeable shift in behavioural threshold was noticed when the duration of the tone reduced below 200 ms. The slope of the temporal integration function was approximately -3.0 dB per doubling of duration of the stimulus, i.e. the threshold increased by 3 dB when the stimulus duration reduced by half.

Table 4.21

Mean and standard deviation of the temporal integration function across the stimulus duration in normal hearing individuals

Duration of the stimulus	Mean	SD	Minimum	Maximum
20 ms	9.77	2.11	6.00	15.00
50 ms	6.36	2.24	2.00	12.00
100 ms	3.31	1.72	0.00	8.00
200 ms	0.79	1.20	-2.00	4.00
300 ms	0.08	0.77	-2.00	2.00
400 ms	0.0	0.00	0.00	0.00

To check the effect of stimulus duration on threshold shift, one-way ANOVA was used. The results indicated a significant difference between the threshold shift observed as a function of stimulus duration [$F(5,190) = 500.81$, $p < 0.01$]. Further, Bonferroni's multiple comparison test was administered to see whether the mean threshold shift difference was significant between two duration stimuli. The results indicated a significant difference at the 0.01 level in behavioural threshold obtained between all pairs of stimuli except for the threshold obtained for the 300 ms and 400 ms duration tones (Table 4.22).

Table 4.22

Results of the Bonferroni's pairwise comparison of the temporal integration obtained for different duration tones for the normal hearing group

	50 ms	**100 ms**	**200 ms**	**300 ms**	**400 ms**
20 ms	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
50 ms		Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
100 ms			Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
200 ms				Significant $p < 0.01$	Significant $p < 0.01$
300 ms					Not Significant $p > 0.05$

4.5.2. Temporal integration function in the clinical group

From the difference in behavioural thresholds obtained between the 400 ms tone burst and each of the five other tone bursts, the mean and SD were calculated. Table 4.23 depicts this information obtained from the participants with AD.

Table 4.23

Mean and standard deviation of the temporal integration function across stimulus durations in individuals with AD

Duration of the stimulus	Mean	SD	Minimum	Maximum
20 ms	13.51	3.52	6.00	21.00
50 ms	8.0	2.91	2.00	14.00
100 ms	3.92	2.39	0.00	10.00
200 ms	0.97	1.78	-2.00	6.00
300 ms	0.44	1.10	-2.00	3.00
400 ms	0.00	0.00	0.00	0.00

It can be seen from Table 4.23 that the mean behavioural threshold shift increased with a decrease in stimulus duration. The increase in threshold shift was lesser when the stimulus duration was reduced from 400 ms to 200 ms. This was in comparison with the shorter durations (200 ms to 100 ms, 100 ms to 50 ms, 50 ms to 20 ms). The slope of the temporal integration function was not uniform for every doubling of duration of the stimulus. The slope was -3 dB for reduction of the stimulus duration from 200 ms to 100 ms. Whereas, it increased to approximately -4 dB and -5.5 dB for reduction in stimulus duration from 100 ms to 50 ms and 50 ms to 20 ms respectively. This suggests that the individuals with AD have more problem in perceiving short duration stimuli.

To determine the significance of difference between the temporal integration for the 1000 Hz tones that varied in duration, one-way repeated measures ANOVA were

done. The results indicated a significant difference in the shift in thresholds obtained at different stimulus durations [F (5,190) = 365.05, $p < 0.01$]. On administering Bonferroni's multiple comparison test it was observed that the difference in mean threshold shift was statistically significant at the 0.01 level between pairs of stimuli with varying duration. This was observed between all but three pairs. The thresholds shift obtained at 200 ms and 400 ms were significantly different only at the 0.05 level. The threshold difference obtained between 200 ms and 300 ms as well as 300 ms and 400 ms duration tone did not differ significantly even at the 0.05 level (Table 4.24).

Table 4.24

Results of the Bonferroni's pairwise comparison for temporal integration obtained for different duration tones for the clinical group

	50 ms	**100 ms**	**200 ms**	**300 ms**	**400 ms**
20 ms	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
50 ms		Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
100 ms			Significant $p < 0.01$	Significant $p < 0.01$	Significant $p < 0.01$
200 ms				Not Significant $p > 0.05$	Significant $p < 0.05$
300 ms					Not Significant $p > 0.05$

4.5.3. Comparison of temporal integration function between the normal hearing and the clinical groups

A similar mean threshold shift with reference to the threshold obtained for the 400 ms tone burst was observed in both participant groups. In both groups, it increased with a decrease in stimulus duration. However, the individuals with AD required slightly higher intensity (in dB) to perceive the presence of a 1000 Hz tone, especially for tones less than 200 ms. While the normal hearing group demonstrated an almost linear increase in threshold (-3 dB per doubling of duration of the signal) for tone varying in duration from 20 ms to 200 ms, it was not so for individuals with AD. The clinical group required differential increase in intensity as the duration of tone reduced beyond 200 ms. As can be seen in Figure 4.16, the slope of the threshold shift curve as a function of stimulus duration was steeper in the clinical group for durations less than 200 ms.

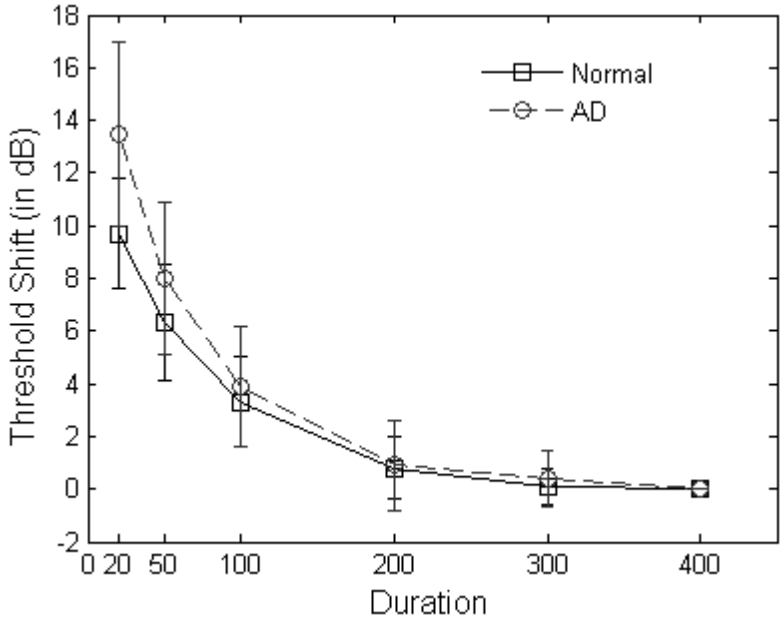

Figure 4.16. Temporal integration functions of individuals with the normal hearing and AD.

Independent sample t-tests were run to determine the significance of difference in mean behavioural threshold shift between participant groups at each duration of tone. A total of five t-tests were run. The results indicated a statistically significant difference ($p < 0.01$) between the groups for the 20 ms and 50 ms duration tone bursts. On the contrary, no significant difference was obtained between the groups for the other duration tone bursts (Table 4.25).

Table: 4.25

Mean and t-values along with level of significance between the participant groups for different duration tones

Participant group	**Duration of 1000 Hz tone**	**Mean**	**t-value**
Normal	20 ms	9.77	5.69**
Clinical	20 ms	13.51	
Normal	50 ms	6.36	2.79**
Clinical	50 ms	8.0	
Normal	100 ms	3.31	1.31
Clinical	100 ms	3.92	
Normal	200 ms	0.79	0.52
Clinical	200 ms	0.97	
Normal	300 ms	0.08	1.67
Clinical	300 ms	0.44	

*** p < 0.01*

The results obtained in the *normal hearing group* is in agreement with the results of the earlier findings in normal individuals, reported in the literature. Several authors found that normal hearing individuals required just a few dB increase in intensity to perceive the presence of a tone as the duration of stimulus was reduced. Moore (2003) reported of a slope of -3 dB per doubling of duration in normal hearing individuals. Likewise, Starr et al. (1991) noted that the threshold of a tone burst increased by 3 dB per halving of signal duration, between 300 and 30 ms, in a normal hearing group. Similar results were also observed by Zeng et al. (1999) and Zeng et al. (2005) for durations up to 100-200 ms. In the present study, the normal hearing participants also showed a decrease in threshold by -3 dB per doubling of duration of the tone burst. This effect was seen till the duration of tone increased up to 200 ms. Once the duration of the tone burst increased beyond 200 ms there was almost no change noticed in the threshold. Thus, the findings of the present study, with reference to normal hearing individuals, are similar to that noted in earlier published studies.

The temporal integration slope observed in *individuals with AD* in the current study differed from that observed in normal hearing individuals. Their slope of temporal integration function increased as the duration of the stimulus was reduced from 200 ms to 100 ms, 100 ms to 50 ms and 50 ms to 20 ms. Similar findings were also reported by Starr et al. (1991), Zeng et al. (1999, 2001), and Zeng et al. (2005). In contrast, Starr et al. (1991) observed that the slope of the temporal integration function was almost the same for both individuals with AD and normal hearing as stimuli decreased in duration till 30 ms. However, it increased sharply by 20 dB for individuals with AD once the duration of the stimulus was reduced below 30 ms. Zeng et al. (2001) reported that nine

of their subjects with AD had a -4 dB slope per doubling of duration of the signal. Later Zeng et al. (2005) reported a slope of -9 dB per doubling of duration in individuals with AD. In contrast, Zeng et al. (1999) reported near normal temporal integration function slope for individuals with AD. Thus, the findings of the present study concur with the majority of studies reported in literature.

The abnormal temporal integration function observed in participants with AD could be due to altered neurophysiology. Normal hearing individuals are able to summate energy over time which would result in increase in loudness. In contrast, individuals with AD might have failed to summate energy over time due to their pathology in the nervous system, leading to reduced growth of loudness. Demyelination has been noted to result in an increase in membrane capacitance and a decrease in membrane resistance (McDonald & Sears, 1970; Rasminsky & Sears, 1972; Pender & Sears, 1984) which might lead to leakage of signal. This might also lead to a conduction block of some of the nerve fibres and a reduction in the number of excitatory potentials at the next higher neuron. Thus, to be excited, the next higher-level neuron might require higher intensity stimuli to perceive the presence of a signal. This effect might be more pronounced for shorter duration stimuli. This probably led to the steeper slope for temporal integration function in individuals with AD, observed in the present study.

It is evident from the reports in literature, that there are diverse findings regarding the slope of temporal integration function. However, the findings of the present study, regarding temporal integration function, are in consonance with the majority of studies reported in literature. Most of the studies, like the present one, observed that individuals with AD do have temporal integration function that are poorer than noted in normal

hearing individuals. This effect is more pronounced as the duration of tone is reduced. Thus, this suggests that the individuals with AD are likely to have problem in processing short duration signals.

4.6. Masking level difference

The MLD values, obtained by subtracting the threshold obtained between homophasic and antiphasic condition in both normal hearing and in individuals with AD, were analyzed. These values were analysed using descriptive statistics, for both normal hearing and clinical groups. Further, the MLD values of the clinical group were compared with the values obtained in the normal hearing group. The results of these analyses are discussed below.

4.6.1. Masking level difference in the normal hearing group

The mean and standard deviation of the MLD values obtained in individuals with normal hearing were computed. The mean MLD value obtained in the normal hearing group was 12.23 with a standard deviation of 1.53. The minimum and maximum MLD value obtained in this group was 10 dB and 17 dB respectively (Table 4.26). It was also noticed that in 26 of the 39 normal hearing participants, the SπNo condition resulted in more deviation. Thirteen normal hearing subjects showed no or negligible difference in threshold shift between the SπNo and SoNπ condition.

4.6.2. Masking level difference in the clinical group

The mean and standard deviation of MLD values obtained in individuals with AD were also computed. These individuals had a mean MLD value of 1 dB with a standard deviation of 1.21. The minimum and maximum MLD values obtained in this group was 0 dB and 5 dB respectively (Table 4.26). Out of 39 individuals with AD, eighteen of them had MLD value of 0 dB and only one participant had an MLD value of 5 dB. Those

who had MLD ranging from 1 dB to 5 dB did not show any differences in scores in the SπNo and SoNπ conditions.

4.6.3. Comparison of masking level difference between the normal hearing and clinical group

Individuals with normal hearing had much higher MLD values, whereas, they were much lower in individuals with AD (Table 4.26). Independent sample t-test was done to see the significance of difference in MLD values obtained between the two groups. The results revealed the presence of a statistically significant difference in MLD values between the two groups [$t(76) = 35.91$, $p < 0.01$].

Table 4.26

Mean, standard deviation, minimum and maximum MLD values obtained in individuals with normal hearing and AD

Participants	**Mean**	**SD**	**Minimum**	**Maximum**
Normal group	12.23	1.53	10	17
Clinical group	1.0	1.21	0	5

The MLDs obtained in the *normal hearing group* in the current study is similar to that reported in literature. The MLD was found to be about 15 dB for 250 Hz and decreased to 3 dB at 1500 to 2000 Hz by Gelfand (1990). Durlach and Colburn (1978) also reported that the MLD values could be as large as 15 dB at low frequencies (500 Hz) and decreased by 2-3 dB for frequencies above 1500 Hz, in normal hearing individuals.

The SπNo antiphasic condition was shown to yeild better MLDs for most of the individuals with normal hearing, in the current study. Green and Henning (1969) also reported higher MLD values for the SπNo condition than for the SoNπ condition, in individuals with normal hearing. The large MLD, reported to be associated with the antiphasic conditions, has been considered to be related to phase-locking that occurs during the neural coding of stimuli (Green & Henning, 1969). This phase-locking is a phenomenon observed in normal hearing individuals. The presence of a large MLD value indicates the presence of a normal phase-locking process in individuals with no hearing problem.

In contrast, in *individuals with AD* the MLD values were almost 0 dB for many of the clients in the present study. This significantly lowered MLD found in this group, has also been observed by Starr et al. (1991), Starr et al. (1996), Hood and Berlin (2001) and Hood et al. (2002). It was observed in these studies also that individuals with AD did not demonstrate any difference in threshold between a homophasic and antiphasic condition.

It can be inferred from the lower MLD found in the present study as well as from studies reported in literature, that individuals with AD lack phase locking responses. Rance et al. (2004) also reported that individuals with AD cannot use the phase locking cues to the same extent as normally hearing subjects.

Thus, it can be concluded that individuals with an AD would have a lower score on an MLD test. This low score can be attributed to the lack of a phase locking phenomena in their auditory nervous system.

4.7. Comparison across psycho-acoustical test results

The six psycho-acoustical tests were compared with reference to the mean and significance of difference between various parameters within each test. ANOVA could not be administered to assess the significance of difference across the tests, as the parameters across the tests were different having different measuring units. Also, each parameter assessed different aspect of auditory processing. Hence, the comparison was done with the information obtained within each test. The comparison was initially done with the normal hearing population (Table 4.27), then within the group with AD (Table 4.28), followed by a comparison of two participant groups (Table 4.29).

4.7.1. *Comparison of the psycho-acoustical test results obtained from the normal hearing group*

The mean values obtained for the different parameters of six psycho-acoustical tests, in the normal hearing group, were tabulated. The mean values along with the significance level for psycho-acoustical test results (DLF, DLI, DLT, GDT, TIF and MLD) are shown in Table 4.27.

Table 4.27

Performance of the normal hearing group on DLF, DLI, DLT, GDT, TIF and MLD

Test →	**DLF (ΔF/Fc%)**								**DLI**							
	500 Hz		**1000 Hz**		**2000 Hz**		**4000 Hz**		**500 Hz**		**1000 Hz**		**2000 Hz**		**4000 Hz**	
dB SL	40	10	40	10	40	10	40	10	40	10	40	10	40	10	40	10
500 Hz			NS	**	NS	**	NS	**			NS	NS	NS	NS	NS	NS
1000 Hz					NS	**	NS	NS					NS	NS	NS	NS
2000 Hz							*	NS							NS	NS
Mean	4	8.36	7.51	13.21	14.38	23.13	31.79	50.15	4.03	6.02	3.97	5.97	4.0	6.05	4.15	6.23
Sign.	**		**		**		**		**		**		**		**	

Test →	**DLT**				**GDT**		**TIF**							**MLD**
	50 ms		**500 ms**				**ms ↓**	**20**	**50**	**100**	**200**	**300**	**400**	(in dB)
dB SL	40	10	40	10	40	10	**20**		**	**	**	**	**	1.0
50 ms			**	**			**50**			**	**	**	**	
Mean	26.79	34.74	133.33	174.35	3.51	9.38	**100**				**	**	**	
Sign.	**		**		**		**200**					**	**	
							300						NS	
							Mean	9.77	6.36	3.3	0.79	0.08	0	

*** p < 0.01, * p < 0.05, NS: Not significant (p > 0.05)*

The *effect of intensity* was compared in four conditions (DLF, DLI, DLT & GDT) where it was considered as one of the independent variables. It can be seen in Table 4.27 that the intensity had a significant effect on differential threshold in the normal hearing individuals for all four psycho-acoustical tests. In all four tests, the performance was better for the higher presentation level. The effect was almost two folds for the frequency discrimination threshold and 1.5 folds for the intensity discrimination threshold across the frequencies. For the duration discrimination threshold the effect was almost 1.3 folds for anchor stimuli. The effect was maximum for the gap detection threshold which was almost 3 folds.

The *impact of frequency* of the anchor stimuli on DLF and DLI were compared. The frequency of the anchor stimulus showed a weak significant effect ($p < 0.05$) only between 2000 Hz and 4000 Hz on $\Delta F/Fc\%$ at 40 dB SL. On the contrary, a significant effect ($p < 0.01$) was noticed between 500 Hz versus the other three frequencies and 1000 Hz and 2000 Hz at 10 dB SL in the individuals with normal hearing. However, the frequency of the anchor stimulus had no effect on DLI at both SLs. As can be seen from Table 4.27, the mean performance across all frequencies was similar.

The *perception of temporal information* in the normal hearing group was obtained from DLT, GDT, TIF and MLD. For the duration discrimination task, the duration of the anchor stimuli had a significant effect on DLT. The DLT was more for the longer duration anchor stimulus. The mean GDT value in the normal hearing individuals was 3.51 ms and 9.38 at 40 dB SL and 10 dB SL respectively. The temporal integration function test revealed a significant shift in threshold when the duration of the stimuli was below 200 ms. The slope of the temporal integration function was almost 3 dB per

halving of the duration of the stimulus. In addition, the mean MLD value obtained in the present study was approximately 12 dB at 500 Hz.

4.7.2. Comparison of the psycho-acoustical test results obtanied from the clinical group

Table 4.28 shows the summery of the six psycho-acoustical test results obtained in individuals with auditory dys-synchrony. The *impact of intensity* was determined from the results of the DLF, DLI, DLT and GDT tests. A significant difference was obtained between the two presentation levels (40 dB SL & 10 dB SL) for all four tests in the clinical group. Thus, it is evident that presentation level does have a significant effect on all the psycho-acoustical tests carried out to obtained differential threshold in individuals with AD (Table 4.28). The effect of sensation level on discrimination threshold was almost uniform across the parameters tested. There was approximately a 1.21 to 1.36 fold increase in DLF and a 1.34 to 1.5 fold increase in DLI across the frequencies between the mean value obtained at 40 dB SL and 10 dB SL. The increase in differential threshold was 1.25 to 1.3 folds for DLT for the two anchor duration stimuli and 1.24 folds for GDT between the two presentation levels.

The *perception across frequency* was got for ΔF/Fc% and DLI. The effect of frequency on ΔF/Fc% was significant at both the presentation level. In contrast, the effect of frequency was not significant on DLI except between 2000 Hz and 4000 Hz.

Table 4.28

Performance of the clinical group on DLF, DLI, DLT, GDT, TIF and MLD

Test →	**DLF (ΔF/Fc%)**								**DLI**							
	500 Hz		**1000 Hz**		**2000 Hz**		**4000 Hz**		**500 Hz**		**1000 Hz**		**2000 Hz**		**4000 Hz**	
dB SL	40	10	40	10	40	10	40	10	40	10	40	10	40	10	40	10
500 Hz			**	**	**	**	**	**			NS	NS	NS	NS	**	NS
1000 Hz					**	**	**	**					NS	NS	*	NS
2000 Hz							**	**							NS	*
Mean	32.44	39.49	51.67	67.05	67.18	92.05	104.49	142.31	6.82	10.26	7.1	10.59	7.36	9.95	8.18	10.9
Sign.	**		**		**		**		**		**		**		**	

Test →	**DLT**				**GDT**		**TIF**							**MLD**
	50 ms		**500 ms**				**ms ↓**	**20**	**50**	**100**	**200**	**300**	**400**	(in dB)
dB SL	40	10	40	10	40	10	**20**		**	**	**	**	**	1.0
50 ms			**	**			**50**			**	**	**	**	
Mean	48.2	63.33	229.48	287.17	14.66	18.18	**100**				**	**	**	
Sign.	**		**		**		**200**					NS	*	
							300						NS	
							Mean	13.51	8.0	3.92	0.97	0.44	0	

*** p < 0.01, * p < 0.05, NS: Not significant (p > 0.05)*

Temporal perception was determined from DLT, GDT, TIF and MLD. A significant difference was observed for DLT obtained between the two anchor stimuli that varied in duration. The GDT threshold was 14.66 ms at 40 dB SL and 18.18 ms at 10 dB SL. In the temporal integration function test, stimuli having a duration of less than 200 ms resulted in a significant shift in threshold. The slope of the temporal integration function was almost 3 dB per halving of the duration of the stimulus, for stimulus durations of 200 ms to 100 ms. However, the slope increased to 5 dB per halving of duration for signals that was less than 50 ms. The fourth temporal based test, MLD, had a mean value of 1 dB at 500 Hz in individuals with AD in the present study.

4.7.3. Comparision of the psycho-acoustical test results between the normal hearing and clinical groups

It can be seen from Tables 4.27 and 4.28 that the *presentation level* had a significant effect on discrimination threshold in individuals with normal hearing as well as individuals with AD. The discrimination threshold increased significantly as the presentation level was reduced. However, this effect was more evident in normal hearing individuals than in individuals with AD.

Compared to normal hearing individuals, the ΔF/Fc% value in individuals with AD were almost 8.1, 6.9, 4.7 and 3.3 times higher at 40 dB SL and 4.7, 5.1, 4 and 2.8 times higher at 10 dB SL for 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz anchor stimuli respectively. The GDT values were higher by more than 4.2 times at 40 dB SL and approximately 1.9 fold at 10 dB SL. In contrast, DLI values were approximately 1.7 times to 1.9 times higher for individulas with AD across frequencies at both sensation

levels. The DLT values were 1.6 times to 1.8 times more for both anchor stimuli and sensation levels. The temporal intengration function was affected only for the shoter duration stimuli in individuals with AD. The MLD values were almost 12 times greater in individuals with AD in compare to normal hearing individuals.

A highly significant *effect of frequency* on ΔF/Fc% was observed for the clinical group. In contrast, the effect of frequency on DLI was much weaker or not significant in both the groups.

The four *temporal perception* tests (DLT, GDT, TIF & MLD) indicated that there was a significant difference between the two groups for most test parameters. The baseline duration of the anchor stimulus of the DLT test had a significant effect on the performance in both the groups. However, DLT and GDT values were higher for individuals with AD at both sensation levels. Individuals with AD showed a steeper temporal integration function. In contrast, the slope was shallower for the normal hearing group. Further, the normal hearing individuals had a much greater MLD value of approximately 12 dB at 500 Hz, while the individuals with AD had almost no MLD. The summery of the comparison between the two groups can be seen in Table 4.29.

Thus, the present study indicated a significant effect of presentation level on discrimination threshold for all the parameters tested in *normal hearing individuals.* Similar results were also reported by Wier et al., (1977), Zeng et al. (2001) and Zeng et al. (2005) for DLF; Zeng et al. (2001) and Zeng et al. (2005) for DLI; Moore (2003) for DLT; Zeng et al. (1999), Zeng et al. (2001) and Zeng et al. (2005) for gap detection threshold.

Table 4.29

Summery of the significance of difference between the individuals with normal hearing and auditory dys-synchrony for six the psycho-acoustical tests. The number of times the test values increased in those with AD compared to the normal group is given in brackets

	Normal hearing versus individuals with AD																			
Stimulus →	**500 Hz**		**1000 Hz**		**2000 Hz**		**4000 Hz**		**50 ms**		**500 ms**		**WBN**		**20 ms**	**50 ms**	**100 ms**	**200 ms**	**300 ms**	**400 ms**
dB SL → **Test ↓**	40	10	40	10	40	10	40	10	40	10	40	10	40	10						
DLF/ (ΔF/Fc%)	** (8.1)	** (4.7)	** (6.9)	** (5.1)	** (4.7)	** (4.0)	** (3.3)	** (2.8)												
DLI	** (1.7)	** (1.7)	** (1.8)	** (1.8)	** (1.8)	** (1.6)	** (1.9)	** (1.7)												
DLT									** (1.8)	** (1.8)	** (1.7)	** (1.6)								
GDT													** (4.2)	** (1.9)						
TIF															** (1.4)	** (1.3)	NS	NS	NS	NS
MLD	** (12)																			

*** p < 0.01, NS: Not significant (p > 0.05)*

The present study also indicated that ΔF increased with increase in frequency in normal hearing individuals. Wier et al. (1977), Zeng et al. (2001) and Zeng et al. (2005) also reported that ΔF increased with the increase in frequency. However, there was no significant difference in the ΔF/Fc% obtained across most anchor frequencies in the present study. Similarly, no significant difference between anchor frequencies on DLI was noticed in the present study. Jesteadt et al. (1977) also did not find anchor frequency to have an effect on DLI.

In the present study a slope of -3 dB per doubling of duration of the stimulus in temporal integration function was obtained. Moore (2003) also reported a similar slope in normal hearing individuals. Further, the MLD value obtained in the current study is in agreement with the findings of Durlach and Colburn (1978).

The fine-grained psycho-acoustical discrimination threshold obtained in the present study, *in individuals with AD* were poorer than that obtained in individuals with normal hearing at both presentation levels. Several researchers also reported of individuals with AD having a significantly poorer DLF value for frequency (Starr et al., 1991; Starr et al., 1996; Zeng et al., 2001 and Zeng et al., 2005), for intensity (Zeng et al., 2001; Zeng et al., 2005), for duration (Starr et al., 1991; Starr et al., 1996) and for gap detection threshold (Zeng et al., 1999; Zeng et al., 2001; Rance et al., 2004 & Zeng et al., 2005). It can be observed from their data that ΔF/Fc% values reduced as the frequency increased in individuals with AD. Unlike the findings obtained for ΔF/Fc% in the present study, frequency had a negligible effect on DLI in individuals with AD. However, the effect of frequency on DLI in individuals with AD has not been reported in the literature.

Further, the data obtained in the present study in individuals with AD also revealed that the presentation level had a significant effect on psycho-acoustic discrimination threshold. This finding concurs with reports given in literature by Zeng et al. (2001) and Zeng et al. (2005) for DLI and by Zeng et al. (1999, 2001) and Zeng et al. (2005) for GDT. However, the effect of presentation level on DLF and DLT in individuals with AD has not been reported in literature.

In the present study, individuals with AD showed a steeper slope than the normal hearing individuals in temporal integration function especially for shorter duration stimuli. Individuals with normal hearing had a slope of -3 dB per doubling of duration of the stimulus, whereas individuals with AD had a slope of -5 to -3 dB per doubling of stimulus from 20 ms to 200 ms. This finding is in consonance with the results of Zeng et al. (1999, 2001) and Zeng et al. (2005). A negligible MLD value was obtained in individuals with AD in the present study. Likewise, Starr et al. (1991) and Hood and Berlin (2001) reported no MLD in individuals with AD.

Thus, it can be concluded that the individuals with AD perform poorly in all parameters tested in the present study in comparison with normal hearing individuals. From the results of the present study it can be observed that the hierarchy of tests that resulted in the maximum to minimum difference between the two participant groups were: MLD, $\Delta F/Fc\%$ at 500 Hz, gap detection threshold, DLI, DLT and TIF.

This suggests that individuals with AD have more problems in processing signals, which requires neural phase locking mechanism. The overall results indicate that individuals with AD have more problem in processing temporal information in comparison to frequency or intensity information.

5. SUMPMARY AND CONCLUSIONS

The impact of hearing loss has been found to vary depending on the type of hearing loss. Individuals with sensorineural hearing loss, along with the reduced hearing sensitivity, often have difficulty in understanding speech, especially in noisy environments. The particular difficulties experienced by the person depend on which part of the system is affected (Moore, 2003). Lesions affecting the auditory nerve or the cochlear nucleus are generally associated more with loss of sensitivity than lesions in more rostral areas of the central auditory nervous system (CANS). Difficulty in understanding speech in the presence of noise is associated with CANS disorders (Musiek, Baran & Pinheiro, 1994).

Auditory neuropathy, more recently referred to as auditory dys-synchrony, by Berlin, Hood and Ross (2001) is known to be a retro-outer-hair-cell disorder, where the patient displays characteristics consistent with normal outer hair cell function and abnormal function at the level of the VIII nerve (Starr, Picton, Sininger, Hood & Berlin, 1996; Berlin et al., 2001). The speech understanding deficits of individuals with auditory dys-synchrony have been found to be disproportionate to their degree of hearing loss unlike those with cochlear hearing loss (Starr et al., 1996; Li, Wang, Chen & Liang, 2005). van Wieringen and Pols (2006) reported that it is difficult to isolate or manipulate specific properties of speech signals. Under such conditions, it is necessary to make use of non-speech analogs such as tone stimuli. Hence, there is a need for a series of psycho-acoustical tests to determine the processing deficit of any or all the three acoustical parameters (frequency, intensity and temporal) of a sound.

The primary objectives of the present study were to measure the fine-grained discrimination ability for frequency, intensity and duration; gap detection threshold; temporal integration; and masking level difference in normal hearing individuals and individuals with auditory dys-synchrony. The experiment involved three phases. The first phase involved development of material for the study. While the second phase involved participant selection, the third phase dealt with obtaining psycho-acoustical data from 39 normal hearing individual with an age range of 16 to 26 years and 39 individuals with auditory dys-synchrony, having an age range of 14 to 28 years. A non-experimental, standard group comparison research design was adopted to achieve the objectives.

Three programs to generate simple tones and complex sounds, developed by Yost (2000) were used to generate the signals with a sampling rate of 44.1 kHz and a resolution of 16 bits. Signals were generated to evaluate DLF, DLT, GDT and TIF. Signals for the intensity discrimination task were not separately generated. These stimuli were directly presented from the GSI-61 audiometer. An AX design was used for DLF, DLI, DLT and GDT, where 'A' was the anchor stimulus and 'X' the variable signals.

All the tests were carried out in a sound treated room. While the fine-grained discrimination thresholds, GDT and TIF were assessed in a sound field condition, MLD was carried out under earphones.

A two-down-one-up procedure was followed to trace threshold. Near the threshold, catch trials having pairs with no difference were presented to eliminate false positive or negative responses. It was adopted for all the discrimination task, and GDT.

For the temporal integration function and MLD, the threshold was obtained using the Modified Hughson-Westlake procedure (Carhart & Jerger, 1959).

The data obtained from both the participant groups were analysed independently and then compared. This was done for all six psycho-acoustical measures, which included DLF, DLI, DLT, GDT, TIF and MLD. The major findings and conclusions of the present study are as follows:

Fine-grained behavioural discrimination of frequency in the normal group

A significant effect of frequency on ΔF/Fc% was seen only between 2000 Hz and 4000 Hz at 40 dB SL and between most frequencies at 10 dB SL. The presentation levels (40 dB SL & 10 dB SL) showed a significant effect on ΔF/Fc% at the 0.01 level.

Fine-grained behavioural discrimination of frequency in the clinical group

The anchor frequency and presentation level had a significant effect ($p < 0.01$) on ΔF/Fc%. It increased with the decrease in frequency and decreased with increase in presentation level.

Comparison of the fine-grained behavioural discrimination of frequency between the normal and clinical groups

The DLF or ΔF/Fc% was higher in individuals with AD in comparison with the normal hearing group. A significant difference was observed between the groups at both sensation levels across all four anchor frequencies at the 0.01 level. These

differences could be attributed to the lack of phase locking mechanism in individuals with AD.

Fine-grained behavioural discrimination of intensity in the normal group

Frequency of the anchor stimuli did not have a significant effect ($p > 0.05$) on DLI. However, a significant increase ($p < 0.01$) in DLI was observed with decrease in presentation level.

Fine-grained behavioural discrimination of intensity in the clinical group

No significant difference between anchor frequencies was observed for the DLI scores at each sensation level except between a few frequencies. Nevertheless, the effect of presentation level was significant ($p < 0.01$) on DLI at each of the anchor frequencies.

Comparison of the fine-grained behavioural discrimination of intensity between the normal and clinical groups

The DLI obtained in individuals with AD were significantly higher ($p < 0.01$) than that obtained in the normal hearing group. This effect was noticed at all the anchor frequencies and both sensation levels. The increased DLI values in individuals with AD may be due to the reduced compound action potentials as a result of demyelination of the auditory nerve.

Fine-grained behavioural discrimination of duration in the normal group

An increase in ΔT was noticed with an increase in baseline duration of the anchor stimulus. This increase was significant ($p < 0.01$) between the anchor durations as well as between presentation levels.

Fine-grained behavioural discrimination of duration in the clinical group

Fine-grained behavioural discrimination scores for duration increased with increase in baseline duration of the stimuli. This effect was significant at the 0.01 level. The presentation levels also had a significant effect on ΔT for both anchor stimuli.

Comparison of the fine-grained behavioural discrimination of duration between the normal and clinical groups

Fine-grained discrimination threshold for duration was significantly higher ($p < 0.01$) in individuals with AD. This was observed for both anchor signals (50 ms & 500 ms) as well as at both sensation levels (40 dB SL & 10 dB SL). Broadening of compound action potentials due to the demyelination could have resulted in increased DLT values in individuals with AD.

Gap detection threshold in the normal group

The sensation level was observed to have a significant effect on GDT at the 0.01 level. It increased sharply as the presentation level was reduced.

Gap detection threshold in the clinical group

The GDT values increased with decrease in presentation levels. This increase in GDT value was significant at the 0.01 level.

Comparison of gap detection threshold between the normal and clinical groups

The GDT value obtained in individuals with AD was significantly higher than ($p < 0.01$) the normal hearing group. This difference was significant at both intensity levels. However, the increase in GDT value with decrease in presentation level was higher for normal hearing individuals than that observed in individuals with AD. The higher GDT value could be attributed to the reduced amplitude and broadening of compound action potentials due to axonal loss or demyelination of auditory nerve in individuals with AD.

Temporal integration in the normal group

A reduction in threshold shift was noticed for tones having a duration of 200 ms and less. These threshold shifts were significant between any two duration stimuli when the duration of the signals was less than 300 ms. A slope of -3 dB per doubling of duration was observed till 200 ms for the temporal integration function.

Temporal integration in the clinical group

The temporal integration function showed a significant improvement in threshold with an increase in stimulus duration till 200 ms. The slope of the temporal integration function was steeper for the stimuli below 100 ms.

Comparison of temporal integration between the normal and clinical groups

The slope of the temporal integration function was steeper for individuals with AD compared to the normal hearing group. A significant shift ($p < 0.01$) between the normal and clinical groups was noticed only for shorter duration stimuli (20 ms & 50 ms). The inability of the auditory nerve to integrate signals or a conduction block due to demyelination in individuals with AD could have resulted in such variations.

Masking level difference in the normal group

Individuals with normal hearing had a mean MLD value of 12.23 dB at 500 Hz. This could be attributed to the intact neural phase locking mechanism in them.

Masking level difference in the clinical group

Negligible MLD values (1 dB) were obtained in individuals with AD. This indicates impaired phase locking mechanism in individuals with AD.

Comparison of masking level difference between the normal and clinical groups

Individuals with AD had a negligible MLD value compared to normal hearing individuals. This was significantly lower ($p < 0.01$) in individuals with AD than the normal hearing group.

Comparison across psycho-acoustical tests in the normal group

The significant effect of frequency on $\Delta F/Fc\%$ were not seen between the most of frequency combinations at 40 dB SL and seen in a few combinations at 10 dB SL. In

contrast, no significant effect across frequencies was noticed for DLI at both sensation levels.

The duration of the anchor stimuli had a significant effect on ΔT values. The ΔT values increased with increase in baseline duration of the stimulus.

Further, the presentation level was seen to have a significant effect on all the psycho-acoustical tests where it was evaluated (DLF, DLI, DLT and GDT). The scores increased with a reduction in presentation level. This effect was maximum for GDT and minimum for DLI and DLT. Presentation level had an intermediate effect on DLF.

Temporal integration function reduced by 3 dB per doubling of duration of the stimulus from 20 ms to 200 ms. In addition, MLD values obtained in this group were high, as typically observed in normal hearing individuals.

Comparison of psycho-acoustical tests in the clinical group

A significant effect of frequency was noticed on ΔF/Fc% in the clinical group. This effect was noticed only between a few frequency combinations for DLI. Also, the duration of the stimuli had a significant effect on DLT.

The presentation level had a significant effect ($p < 0.01$) on ΔF/Fc%, DLI, DLT and GDT. The scores obtained from all these tests increased with decrease in presentation level.

The temporal integration function showed a sharper slope for the shorter duration stimuli. It was approximately -5 dB per doubling of duration from 20 ms to 100 ms. Further, MLD values obtained in this group were negligible.

Comparison of psycho-acoustical tests between the normal and clinical groups

The data obtained for the psycho-acoustical tests in individuals with AD showed significantly higher scores in DLF, DLI, DLT and GDT at both sensation levels in comparison to the normal hearing group. The temporal integration function was significantly higher in the clinical group only for short duration stimuli. Likewise, the MLD values were severely affected in individuals with AD in comparison to normal hearing group.

Thus, it can be concluded that to assess perceptual deficits in individuals with AD a series of psycho-acoustical tests can be carried out. Based on the findings of present study, the below mentioned recommendations can be made to assess the perceptual deficits of the individuals with AD. The recommendations are given in a hierarchical order. The tests that differentiate the two groups more are listed earlier.

- MLD values should be obtained at 500 Hz,
- ΔF/Fc% should be assessed for a 500 Hz anchor frequency at 40 dB SL,
- Gap detection threshold should be obtained at 40 dB SL,
- Duration discrimination ability could be assessed at any one anchor stimulus duration either at 40 dB SL or 10 dB SL,
- Intensity discrimination task could be carried out at any frequency (500 Hz, 1000 Hz, 2000 Hz or 4000 Hz) at 40 dB SL or 10 dB SL,
- Temporal integration function curved should be obtained only for shorter duration stimuli (20 ms, 50 ms and 100 ms). These thresholds should be compared with the threshold obtained for a 400 ms tone.

The results of this study indicate that the psycho-acoustical tests, which assess the temporal processing, are affected in individuals with AD. Thus, the temporal based psycho-acoustical tests could be used to indentify individuals with AD. These results reaffirm the findings of previous researchers that temporal processing is severely affected in individuals with AD.

The implications of the present study are as follows:

- The study has provided data for the six psycho-acoustical tests (DLF, DLI, DLT, GDT, TIF & MLD) in normal hearing Indians. This information could be used as a reference against which individuals with a suspected auditory perceptual deficit could be compared to make a decision regarding their auditory abilities.
- The present study has highlighted the presence of behavioural auditory processing deficits in individuals with AD using different psycho-acoustical tests. It substantiates that the perceptual deficits are primarily due to temporal processing problems.
- Information about the specific deficit of acoustical parameters which differentiate normal hearing individuals from those with an AD has also been provided. This information, given in hierarchy, could guide the clinicians during the assessment of individuals with AD.
- The findings of this study have also reaffirmed previous research finding that the responses of psycho-acoustical tests reflect the auditory perceptual deficits in individuals with AD. Therefore, the utility of the psycho-acoustical tests to assess the auditory processing deficit has been reiterated.

- Appropriate behavioural training programs can be designed for a particular clients having AD, based on the various test results.
- Progress following training programs could be monitored behaviourally using the fine-grained discrimination paradigm.
- Finally, it can be inferred from the findings of the psycho-acoustic tests that the perceptual problems in individuals with AD could be due to the demyelinated or axonal loss of the auditory nerve.

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