

**EFFECT OF NOISE SPECTRUM ON CORTICAL AUDITORY EVOKED
POTENTIALS IN CHILDREN WITH AND WITHOUT DYSLEXIA**



Potdar Riddhi Suhas
Register No: 14AUD016

A Dissertation Submitted in Part Fulfilment for the degree of
Master of Science (Audiology),
University of Mysore, Mysuru

**ALL INDIA INSTITUTE OF SPEECH AND HEARING
MANASAGANGOTHRI
MYSURU-570006**

May, 2016

Certificate

This is to certify that this dissertation entitled “**Effect of Noise Spectrum on Cortical Auditory Evoked Potentials in Children With and Without Dyslexia**” is a bonafide work in part fulfillment for the Degree of Master of Science (Audiology) of the student (Registration No.14AUD016). This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysuru

May, 2016

Dr. S. R. Savithri

Director

All India Institute of Speech and Hearing
Manasagangothri, Mysuru- 570006.

Certificate

This is to certify that this dissertation entitled “**Effect of Noise Spectrum on Cortical Auditory Evoked Potentials in Children With and Without Dyslexia**” is a bonafide work of in part fulfillment for the degree of Master of Science (Audiology) of the student (Registration No. 14AUD016). This has been carried out under my guidance and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

Mysuru,

May, 2016

Dr. Animesh Barman

Guide

Professor of Audiology

Department of Audiology

All India Institute of Speech and Hearing

Manasagangothri, Mysuru-570006.

Declaration

This dissertation entitled “**Effect of Noise Spectrum on Cortical Auditory Evoked Potentials in Children With and Without Dyslexia**” is the result of my own study under the guidance of Dr. Animesh Barman, Professor of Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysuru, and has not been submitted earlier in any other University for the award of any Diploma or Degree.

Mysuru,

May, 2016

Register No. 14AUD016

ACKNOWLEDGEMENTS

First and foremost, I would like to express my sincere gratitude towards my guide Dr. Animesh Barman for his valuable guidance and patience throughout the time. I would also like to thank Sreekar sir for his constant support, motivation and patience with all three of us.

I would like to thank the Director Dr. S.R. Savithri for allowing me to carry out the study and the HOD Audiology, Dr Sandeep M. for making the instruments available even in non working hours of the institute.

Special thanks to Nike sir and Ganapathy sir for giving their time off from work and opening the department on weekends. A huge thanks to Vasanthlakshmi ma'am for helping me out with the statistical analysis. Ma'am, without your guidance the number game would have been a lot more difficult.

I thank my lovely family from the bottom of my heart for everything they have given me. Muma, Baba without your support and love this journey would have been impossible. Thank you for being my closest friends! Prathamesh and chaitral, thanks for being my pillars of support.

A very warm thanks to all the participants of the study. I appreciate the patience and effort put in by the parents' of all the participants during testing.

A huge thanks to my AOSTH family! Masterminds, you guys are the best batchmates I could have ever found. Special thanks to Dindhya, Suveetha, Radhi, Maithri, Swathi for all the late night working sessions in the past few days. Minions! I couldn't have asked for better classmates than you guys! Thank you for making me feel at home all the time. Thank you Preeti, Tina, Latika, Shalini for watching out for me always. A very special thanks to Sindhu and her Pleasure for making my printing visits easier! My lovely juniors, Rachi, Pratyasha and Setu,

thanks a lot for keeping the mood lively always! I thank all my seniors for their timely guidance.

A big thank you to all my BASLP classmates and seniors, especially Niki, Gopika and Pooja, for making the inaccessible articles available for me at any point of time. A hearty thank you to all my BASLP teachers for giving me strong basics of the subjects in the field.

I consider myself the luckiest to have found friends like my school buddies! Without you guys I cannot imagine how difficult my journey would be. I cannot thank you enough Sayali, Manali and Anya for being there for me NO MATTER WHAT through all these 16 years!

Last but not the least, I thank my dissertation partners Meghana and Madhu for sticking by my side through all the thick and thins. I had a great time working with you guys!

*Thank you to one and all who directly and indirectly helped
me complete the research.*

Abstract

Aim: The aim of the present study was to know whether different spectra of noise have a differential effect on different components of Auditory long latency responses (ALLRs) in children with and without Dyslexia and to investigate if any correlation exists between measures of cortical potentials and speech perception in noise scores.

Method: ALLRs were recorded for natural syllables /ba/ and /da/ presented in three conditions i.e. in quiet, in presence of high pass filtered noise having cut off frequency of 4000 Hz and in presence of low pass filtered noise having cut off frequency of 200 Hz in 10 children with dyslexia and 15 typically developing children. The age of all participants ranged from 8 to 14 years. Behavioural SPIN scores were obtained from all the children at 0 dB SNR using recorded material of monosyllabic words in English. Latencies and absolute amplitude of the peaks P1, N1 and P2 in all three stimulus conditions and SPIN scores were subjected to statistical analysis.

Results: Latency of peak P1 and N1 for /ba/ and /da/ both, was significantly prolonged in children with dyslexia as compared to their typically developing counterparts in quiet condition. Additionally latency of N1 was significantly prolonged in response to stimulus /ba/ in presence of low pass filtered noise. Presence of low pass noise affected the earlier peaks P1 and N1 in terms of prolonged latencies and reduced amplitudes in children with dyslexia as well as typically developing children. For /da/, a significant negative correlation between P1 latency in low pass noise and SPIN scores was seen in control group while a significant positive

correlation between P2 amplitude in high pass noise and SPIN scores was obtained in control group.

Conclusion: We can conclude from the study that low pass noise affected the latency and amplitude of earlier peaks P1 and N1 more than the later peaks in both the groups probably due to obligatory nature of these peaks. A positive correlation between SPIN scores and P2 amplitude in high pass noise in children with dyslexia may suggest that P2 may be sensitive to speech processing deficits in these children.

Table of contents

List of Tables.....	ii
List of Figures.....	iv
Chapter 1.....	1
Introduction	1
Chapter 2.....	8
Review of literature.....	8
Chapter 3.....	23
Methods.....	23
Chapter 4.....	35
Results.....	35
Chapter 5.....	55
Discussion	55
Chapter 6.....	66
Summary and Conclusions.....	66
References	70

List of Tables

Table 3.1. Stimulus and Acquisition parameters used for recording ALLR.....	32
Table 4.1. Mean, Median and Standard Deviation (SD) for P1, N1 and P2 latency and baseline to peak amplitude obtained at 70 dB SPL for group I for three conditions in response to syllable /ba/.....	38
Table 4.2. Mean, median and Standard Deviation (SD) for P1, N1 and P2 latency and baseline to peak amplitude obtained at 70 dB SPL for group II across conditions in response to syllable /ba/.....	39
Table 4.3. Man Whitney U test results for ALLR peaks obtained across group I and Group II in response to speech syllable /ba/.....	40
Table 4.4. Friedman's test results for latency and amplitude of P1 and N1 across the three stimulus conditions obtained for stimulus /ba/ within group.....	42
Table 4.5. Wilcoxon Signed Rank test for amplitude and latency of peaks P1 and P2 evoked in response to speech stimulus /ba/ across test conditions for control group.....	43
Table 4.6. Wilcoxon Signed Rank test for amplitude and latency of peaks P1, N1 and P2 evoked in response to speech stimulus /ba/ across test conditions for clinical group.....	44
Table 4.7. Mean, median and Standard Deviation (SD) for P1, N1 and P2 latency and baseline to peak amplitude obtained at 70 dB SPL for group I for three conditions in response to syllable /da/.....	45

Table 4.8. Mean, median and Standard Deviation (SD) for P1, N1 and P2 latency and baseline to peak amplitude obtained at 70 dB SPL for group II for three conditions in response to syllable /da/.....	46
Table 4.9. Man Whitney U test results for ALLRs obtained across group I and Group II in response to speech syllable /da/.....	47
Table 4.10. Friedman’s test results for latency and amplitude of P1 and N1 across the three stimulus conditions obtained for syllable /da/ within control and clinical group.....	48
Table 4.11. Wilcoxon Signed Rank test for amplitude and latency of peaks P1, N1 and P2 evoked in response to speech stimulus /da/ across test conditions for control group.....	49
Table 4.12. Wilcoxon Signed Rank test for amplitude and latency of peaks P1, N1 and P2 evoked in response to speech stimulus /da/ across test conditions for clinical group.....	50
Table 4.13. Correlation between latency and amplitude of peaks P1, N1 and P2 with behavioural SPIN scores for different conditions within group for stimulus /ba/.....	51
Table 4.14. Correlation between latency and amplitude of peaks P1, N1 and P2 with behavioural SPIN scores for different conditions within group for speech stimulus /da.....	52

List of figures

Figure 3.1: Syllable /ba/ of 100 ms.....	27
Figure 3.2: speech syllable /ba/ occurring at 300 ms of high pass filtered noise having a cut off 4000 Hz.....	28
Figure 3.3 speech syllable /ba/ occurring at 300 ms of low pass filtered noise having a cut off 200 Hz.....	28
Figure 3.4 Syllable /da/ of 100 ms.....	28
Figure 3.5 speech syllable /da/ occurring at 300 ms of high pass filtered noise having cut off 4000 Hz.....	29
Figure 3.6 speech syllable /da/ occurring at 300 ms of low pass filtered noise having a cut off 200 Hz.....	29

Chapter 1

Introduction

Speech perception is a phenomenon that begins at the auditory periphery and continues up to central nervous system. The perception of speech is difficult in the presence of noise for the normal hearing as well as different clinical population (Summers & Leek, 1998; Warrier, Johnson, Hayes, Nicol, & Kraus, 2004). This decrement in perception may be due to suppression of stimulus activity as a result of interference within the cochlea (Kidd, Best & Mason., 2008). However, new evidence suggests that basic neurophysiologic processes related to stimulus encoding and discrimination may be involved (Nagarajan et al., 1999; McAnally & Stein, 1997).

Central Auditory Processing (CAP) involves neural processes needed for auditory signal discrimination, recognition, ordering, grouping, and localization (American Speech-Language-Hearing Association 2005). The effects of background noise on CAP and attention have been extensively studied with behavioral response measures in various age. These studies showed that noise impairs episodic memory in school-age children (Hygge, Boman & Enmarker 2003; Boman 2004; Söderlund, Sikstrom, Loftesnes & Barke. 2010) as well as attention in school-age children (Hygge et al. 2003) and in adults (Enmarker 2004). Background noise also reduces the accuracy of word recognition both in preschool-age children and in adults (Stuart, Givens, Walker & Elangovan, 2006). Younger children are more vulnerable to noise than older ones (Wilson, Farmer, Gandhi, Shelburne & Weaver, 2010). School-age children with linguistic problems are even more sensitive to noise than typically developing children (Ziegler, Pech-Georgel, George & Lorenzi, 2009; Vance & Martindale, 2012). Children with dyslexia are found to be poorer in consonant

discrimination than typically developing children in presence of noise (Hazan, Messaoud & Rosen; 2013) and they show deficits in speech perception in a noisy environment (Ziegler et al., 2009).

Likewise it is also possible to see the effect of noise on Central Auditory Processing through electrophysiological measures. One way to evaluate what is happening in the cortex is to record the electrical fields generated in response to an acoustic stimulus. Cortical Auditory Evoked Potentials like Auditory Late Latency Response (ALLR) are thought to reflect the functional integrity of auditory pathways involved in processing of complex speech stimuli (Novak, Kurtzberg, Kreuzer & Vaughan, 1989). Event-related potentials (ERPs) are an objective tool and eligible method to investigate CAP even in very young children (Friedrich & Friedrich, 2010; Jansson-Verkasalo et al. 2010; Partanen, Kujala, Tervaniemi, Huotilainen, 2013) and in clinical groups (Jeste & Nelson, 2009; Naatanen et al., 2012). The obligatory ERPs, such as Auditory Late Latency Response in children, are generated at the thalamocortical pathway (Ponton, Eggermont, Kwong, & Don, 2000). ALLR are characterized by components occurring in the time domain of 50-500 ms (McPherson & Starr, 1993). Major components in the late latency auditory evoked potentials include a positive component at 60 ms (P1) followed by a negative components at about 100 ms (N1), another positive component at 160 ms (P2) and a negative component at 200 ms (N2) (McPherson & Starr, 1993). The robust positive P1 indexes the automatic encoding of acoustic sound features (Ceponienė, Rinne & Naatanen, 2002, 2005). P1 latency is also a biomarker of the maturation of the central auditory pathway (Sharma, Dorman & Spahr, 2002). N2 response reflects the forming of sensory representations (Anderson, Chandrasekaran, Yi, & Kraus, 2010; Choudhury & Benasich 2011). In infants and young children, CAEPs are dominated

by P1, which becomes earlier and smaller as N1 and P2 begin to emerge in the waveform at about 8 to 10 years of age (Sharma, Kraus, McGee & Nicol, 1997; Ponton et al., 2000). These also provide a tool to investigate neurophysiological processes that underlie our ability to perceive speech (Purdy et al., 2001; Tremblay, Piskosz & Souza, 2003).

ALLRs that are elicited to speech sounds correlate with the acoustic features of speech. Sharma and Dorman (1999) carried out a study by increasing the voice onset time of speech sounds from 0-30ms to 50-80ms. They observed that the LLR contained two negative peaks N1 and N1' for longer VOT stimuli instead of one N1. Tremblay et al., (2003) found distinctive cortical response patterns for syllables that differed in their initial phoneme. It has been shown that P1 encodes the acoustic features of sound i.e. frequency and timing and N2 synthesises these features into sensory representation (Shtyrov et al., 1998; Ceponine et al., 2005).

Martin and Stapels (1997) investigated the effects of decreased audibility in low frequency spectral regions, produced by high pass masking on cortical ERP's using speech sounds /ba/ and /da/. Low pass cutoff were 250Hz, 500Hz, 1000Hz, 2000Hz and 4000Hz. They showed that as the cutoff frequency of high pass noise was raised, ERP latencies increased and amplitudes decreased. N1 latencies showed significant changes when high pass masker was increased to 1000Hz while N2 and P3 did not change until high pass masker was raised to 2000Hz. They concluded that, decreased audibility from masking affects N1 in differential manner compared to N2 and P3 complex. Also, N1 indexes the presence of audible stimulus energy because N1 was present when the signal was heard irrespective of whether it was discriminable or not but N2 and P3 peaks were present only when the signals were discriminable, hence they index behavioral discrimination of speech sounds.

Martin and Stapels (1997, 2005) also investigated decreased audibility produced by low pass masking noise and found that as the cutoff frequency of low pass masker was reduced, latencies increased and amplitude decreased.

Kaplan, Henkin, Rabin and Muchnik (2006) conducted a study to characterize the effect of background noise on identification of syllables using behavioral and electrophysiological measures. The speech sounds /da/ and /ga/ were embedded in white noise with +15, +3, 0, -3 and -6 dB SNRs. It was seen that performance accuracy and reaction time were prolonged due to noise. N1 and P3 latencies were increased. /ga/ was better identified than /da/ in all noisy conditions, P3 latency was prolonged for /da/ in all conditions and N1 latency was prolonged for /ga/. They concluded that the effects of noise on speech recognition occur at both physical and perceptual processing levels of sounds. The studies by Whiting et al. (1998) and Kaplan et al. (2006) indicate the effect of SNR on the peaks of ALLR. They found that in spite of high behavioral recognition scores, there were significant changes in the peaks of AEP's in the presence of noise even in normal hearing individuals.

The population considered in this study, children with Dyslexia, is a specific learning disability that is neurological in origin. It is characterized by difficulties with accurate or fluent word recognition and by poor spelling and decoding abilities. Electrophysiological studies have shown physiological deficits in children with learning disorders (Purdy et al., 2002). Such deficits result in brain cognitive dysfunction related to selective attention, working memory or language processing. In general delayed values of the components in ALLR in dyslexic children's group are observed compared with children without dyslexia. The latency is delayed especially for N1 and P2 (Lippanen & Lyytinen, 1997), also N2 (Mazzotta & Gallai, 1991) complex in this population. The delayed N2 and reduced amplitude in students with

dyslexia reflects difficulties in passive and automatic auditory sensory processing responsible for auditory perception, attention and discrimination. Purdy, Kelly and Davis (2002) also studied LLR in children with learning disabilities and reported that the latency of P1 was earlier while that of P3 was prolonged. They also reported that the amplitudes of waves were lesser for children with learning impairments in comparison to typically developing children. The study concluded that the cortical processing of auditory signals is abnormal in children with learning impairment. A study conducted by Kumar and Gupta (2014) reveals that children with dyslexia exhibited prolonged latencies and reduced amplitudes of speech evoked auditory late latency response in comparison to normally developing children. Warriar et al (2004) reported that amplitude of P2N2 complex was found to be reduced in typically developing children as well as in children with Learning Problems when broadband noise was added to the speech stimulus. They also found significant latency shifts in the N2 region for the group of children having learning problems. Cunningham, Nicol, Zecker, Bradlow and Kraus (2001) observed two positive peaks P1 and P1' and two negative peaks N1 and N1' in response to a 40 msec syllable da. They also reported that in noise, both normal and children with learning problems showed a reduction in P1-to N1 and P1'-to-N1' amplitude to /da/. This difference between the groups was not noticed in quiet conditions.

1.1. Need for the study:

In India, the occurrence of dyslexia ranges from 3% to 7.5% of children (Ramaa, 2000). The prevalence estimate of this disability has been found to be 3 to 10% in western literature (Snowling, 2000). The learning disability negatively affects

a variety of behaviours, so early intervention is one of the most important steps in this regard. Thus the population has been considered in the study.

Majority of the studies have focused on recording of ALLR on click stimulus or more frequency specific tone bursts. But recording of ALLR using tone burst does not give much information about the processing or perception of speech. The P1-N1-P2 evoked neural response is heavily influenced by acoustic content of evoking signal. Hence it is important to know more about how the speech signal is processed in children with dyslexia (Kumar & Gupta, 2014). Studies have shown abnormal processing of speech stimuli and normal processing for tonal stimuli in children with Dyslexia (Serniclaes , Carre et al., 2001).

Studies have also shown that N1 in Cortical Event Related Potentials represents an obligatory response to audible stimuli and directly does not correspond to perception (Hillyard & Picton, 1987) whereas peaks N2 and P3 responses reflect an individual's ability to discriminate different stimuli and are present only when he is attending to it (Hillyard & Picton, 1987). Martin and Stapeles (1997, 2005) have reported that different types of noises have differential effects on different components of ALLR in normal individuals (age range 18-30). However there are very few studies that focus on the markers or peaks that mark the audibility and perception. Previously done studies have used a continuous noise. Presence of intermittent noise may have a different effect on the cortical evoked potentials and hence needs to be studied. Also, there is dearth of studies investigating what happens to the components of ALLR evoked in response to speech stimulus in presence of different spectra of noise for typically developing children as well as for children having Dyslexia and correlating these findings with the behavioural measures of speech perception.

1.2. Aim of the study:

The aim of the present study was to know whether different spectrum of noise has a differential effect on different components of ALLR in children with and without Dyslexia and which of those components best correlates with the behavioural speech perception ability of the same group.

1.3. Objectives of the study:

The objectives of the study were as mentioned below:

1. To see whether there is a difference in the ALLRS obtained in quiet between typically developing children and children with Dyslexia.
2. To compare the effect of different spectrum of noise on different components of ALLR between the two groups.
3. To see the effect of Low pass noise of cut off frequency 200 Hz and high pass noise of cut off frequency 4000 Hz on different components of ALLR in typically developing children.
4. To see the effect of Low pass noise of cut off frequency 200 Hz and high pass noise of cut off frequency 4000 Hz on different components of ALLR in children with dyslexia.
5. To find out the correlation between different components of ALLR and behavioural SPIN scores.

Chapter 2

Review of Literature

Learning disability (LD) is a broad term under which there are many subtypes. Learning disabilities is a general term that refers to a heterogeneous group of disorders manifested by significant difficulties in the acquisition & use of listening, speaking, reading, writing, reasoning, or mathematical abilities. These disorders are intrinsic to the individual, presumed to be due to central nervous system dysfunction (National Joint Committee on Learning Disabilities). Snowling (2000) reported the prevalence of LD to be 3 to 10% in western literature. It has been reported that dyslexia can occur along with Auditory Processing Deficits and percentage of persons with developmental Dyslexia and comorbid APD is substantial (King, Lombardino, Crandell & Leonard; 2003). Ramus (2003) reported the incidence of APD in children having dyslexia was 40%.

It has been previously reported that, at a behavioural level, some of the children with developmental dyslexia have primary disturbance in phonological processing (Adlard & Hazan,1998). In contrast, it has also been reported that children with dyslexia have poor speech discrimination ability which in turn leads to phonological processing deficits (Rosen & Manganari, 2001). In addition, the perception of speech is reported to worsens in presence of background noise for children with Dyslexia (Warrier, Johnson, Hayes, Nicol and Kraus,2004). .Reduced ability for understanding speech in noise is a primary symptom of (central) auditory processing disorders (Bamiou, Musiek & Luxon, 2001). APD is estimated to affect between 2% to 3% of children (Chermak & Musiek, 1997) and 22.6% of adults over 60 years of age (Cooper & Gates, 1991). It is also a characteristic of many individuals

with learning disabilities (King, Lombardino, Crandell & Leonard, 2003; Hugdahl et al., 1998). Much research investigating the neural bases of speech perception focuses on problems such as the perception of speech and non-speech sounds (Binder, Liebenthal & Possing; 2004) and the perception of consonants (Burton, Small, & Blumstein, 2000) as measured by electrophysiological measures. Auditory Late Latency Response obtained in children depends upon several factors.

2.1. Factors affecting ALLR:

2.1.1. Maturation and aging

The maturation starts from peripheral auditory system and moves towards the central auditory system. The generators of ALLR potential include Primary auditory cortex, frontal cortex, auditory association areas and also subcortical regions (Stapels, 2002). These areas mature at different rates and therefore cause changes in latency, amplitude and morphology of different components as the age increases (Cunningham, Nicol, Zecker & Kraus, 2000; Ponton, Eggermont, Kwong & Don, 2000). The ALLR latency decreases and amplitude increases as a function of age during childhood, upto 10 years of age (Ponton, Don, Eggermont, Waring & Masuda, 1996; Weitzman & Graziani, 1968). Previous research has also reported that latency and amplitude of ALLR components do not reach adult like values until 16 to 18 years of age (Ponton et al, 2000; Ptok, Blachnik & schonweiler, 2004). McIsaac and Polich (1992) observed that latency and amplitude values of N1, P1 and N2 were longer and smaller, respectively, for infants as compared to adults.

Developmentally, P1 emerges first and dominates the ALLR. Ponton et al (1996) reported that in children 8 years of age and younger, the AEPs are dominated

by positive peaks when faster stimulus rates like that of 1 Hz or more is used. However N1 peak is not consistently present until the age of 9 years or more.

Sussman et al. (2008) studied development of cortical auditory evoked potentials in normally developing children between age 8 years to 11 years, in adolescents (16 years) and in adults (22-40 years) with pure tone stimuli using faster stimulus rates. Their results showed that both age and stimulus rate produced a large change in CAEP morphology. The P1 and N2 components dominated ALLR response at all stimulus rate in the children of age 8 to 11 years. N1, which is a dominant component seen in adults was smaller and appeared as a bifurcation in a broad positive peak at early ages, and emerged as a separate peak only at adolescence. In the waveforms of adolescents, P1-N1-P2 complex was more adult like however the hallmark of child obligatory response, N2 was still identifiable. Faster stimulus rates resulted in presence of only P1 in the adults and adolescents, and both P1 and N2 in the younger children.

Kraus et al. (1993) recorded Event Related Potentials (ERPs) in response to synthetic speech stimuli /ga/ in children aged between 7-11 years and compared it to the response obtained in 10 adults. They reported that a well-defined N1-P2 complex as seen in adults was not found in children. Peaks P1 and N1 had longer latencies in children and amplitude of P2 was smaller as compared to adults. They concluded that the latencies of P1, N1 and P2 may provide a measure of maturation of central pathways.

Sharma et al. (1997) investigated the maturational changes in central auditory pathways by comparing ALLR obtained from children aged 6-15 years and that obtained from 10 normal hearing adults in response to speech stimulus /ba/. They

reported a decrease in latency of P1 and N1, and a decrease in the amplitude of P1 as the age increased. No age related changes in the amplitude of N1 was observed.

2.1.2. State of Arousal

Researchers have found that sleep has a significant effect on different components of ALLR. It affects different components in different ways. Campbell and Colrain (2002) observed that N1 amplitude progressively reduced as one moved from state of arousal to sleep. They also reported that during the transition to deep sleep amplitude of P2 increased. It has been reported in literature that during the recording of ALLR, P1 does not get affected by attention or wakefulness and sleep whereas N1 increases in amplitude by about 0.61 microvolt when the stimulus is being attended (Picton & Hillyard, 1974). The same study found that P2 also increased in amplitude by about 0.70 microvolt when stimulus was attended to. Latency and amplitude of N2 decreases in early childhood and again in adulthood until it becomes essentially absent in seniors. N2 latency remains stable during the school age years (Sharma et al. 1997).

2.1.3. Type of stimuli

ALLR can be elicited in response to different types of stimuli such as click, tone bursts, noise and various kinds of speech stimuli (Naatanen & Picton, 1987).

Generally tonal stimuli are used to elicit ALLR (Davis, Bowers & Hirsh, 1968). The optimal tone burst stimuli used to elicit ALLR have rise/fall and plateau time of greater than 10 ms (Onishi & Davis, 1968; Rothman, Davis & Hay, 1970).

Studies have reported that low frequency tones elicit significantly larger cortical response amplitude as compared to that elicited by high frequency tones (Alain, Woods & Covvarubias, 1997).

Researchers have used various kinds of speech stimuli to elicit ALLR which include natural or synthetic vowels, syllables and words (Ceponine et al., 2001; Martin & Boothroyd, 1999; Sharma, Marsh & Dorman, 2000; Tremblay et al., 2003). Tremblay et al. (2003) also reported that natural speech sounds elicited reliable ALLR components. Investigators have observed that synthetically produced voiced speech stimuli evoked ALLR with robust amplitudes than those evoked by voiceless speech stimuli. Similar pattern was found for natural speech stimuli also.

Speech evoked ALLR are frequently used to study the neural representation of speech in population who have impaired speech understanding. The underlying assumption is that the speech perception is dependent on the neural detection of rapid varying spectral and temporal cues present in the speech signal (Tremblay, Billings & Rohila, 2004).

2.2 ALLR evoked in presence of background noise

Martin, Sigal, Kurtzberg, and Stapells (1997) carried out the first systematic investigation of the effects of decreased audibility on N1, N2, and P3 elicited to speech sounds /ba/ and /da/. 11 normal hearing adults participated in the study. Responses to the speech sounds were obtained in quiet, in broadband noise and in 250, 500, 1000, 2000, and 4000 Hz high-pass noise presented ipsilaterally. The results revealed that N1 was clearly present in the quiet condition but was absent in the BBN condition. The amplitude of N1 decreased and its latency increased steadily as the

high-pass cutoff frequency for noise was lowered. This was seen for both the stimuli /ba/ and /da/. So the study provided evidence that N1 is related to the presence of audible speech energy. As more low frequency noise was allowed to pass through, audibility was more compromised probably due to upward spread of masking. N1 was present when speech sounds were audible, whether or not they were discriminable.

Martin and Stapells (2005) also studied the effect of reduced audibility due to masking by low pass noise of cut off frequencies 4000, 2000, 1000, 500, and 250 Hz. 10 normal hearing adults were chosen for the study. Cortical potentials were recorded at two intensity levels 65dB SPL and 80 dB SPL to represent normal conversational level speech and louder speech, respectively. The findings revealed N1 was clearly present in the responses to stimuli presented **at 65 dB SPL**, in the quiet, 250, 500, and 1000 Hz low-pass noise conditions. It was absent in the 4000 Hz and BBN conditions. The amplitude of N1 decreased and its latency increased as the low-pass cutoff frequency was raised. N2 amplitudes decreased and latencies increase as the lowpass noise cutoff was raised to 1000 Hz. When masker cut off was further raised in frequency N2 disappeared. At **80 dB SPL presentation level** N1 was present in quiet and in all of the noise masking conditions. Its amplitude decreased and latency increased as the low-pass noise cut-off was raised. N2 was present in the quiet, 250, 500, 1000, and 2000 Hz conditions. Its amplitude decreased and latency increased as the low-pass noise masker cutoff was raised to 2000 Hz. N2 was absent in the BBN condition. Based on the findings they concluded, decreased audibility results in decreased ERP amplitudes, and increased ERP latencies, the effects of the low-pass noise are maximum when the 1000 to 2000 Hz spectral region is masked.

Kaplan, Henkin, Rabin and Muchnik (2006) conducted a study to characterize the effect of background noise on identification of syllables using behavioral and

electrophysiological measures. 20 adult females with normal hearing sensitivity participated in the study. The stimulus used was speech sounds /da/ and /ga/ embedded in white noise with +15, +3, 0, -3 and -6 dB SNRs. It was seen that performance accuracy and reaction time were prolonged due to noise. N1 and P3 latencies were increased. /ga/ was better identified than /da/ in all noisy conditions, P3 latency was prolonged for /da/ in all conditions and N1 latency was prolonged for /ga/. They concluded that the effects of noise on speech recognition occur at both physical and perceptual processing levels of sounds.

Haapola, Haapala, Jansson-Verkasalo and Kujala (2015) examined the effect of background noise on P1 and N2 in a group of 18 normally developing toddlers in the age range of 22-26 months. Semisynthetic stimuli /ke/ and /pi/ were used to elicit the response. The results showed that the P1 amplitude was smaller and the N2 amplitude larger in the noisy conditions compared with the silent conditions. However, noise had no effect on P1 and N2.

Billings, Bennet, Molis and Leek (2011) recorded P1-N1-P2 complex in response to tones and speech stimulus /ba/ on 9 normal hearing adults. Stimuli were presented in 4 conditions i. e. in quiet, continuous noise, interrupted noise, and four-talker babble. The SNR was maintained at -3 dB. In terms of latency they found significant effect of noise on P1 and N1 peaks whereas for amplitude, only N1 was significantly affected. Peaks were generally earliest and largest for the interrupted noise and latest and smallest for the four-talker babble. The speech babble could have been a better masker probably because it contained both informational masking and energetic masking components.

2.3. ALLR in children with learning disability:

Ample amount of studies have been carried out using ALLR to understand the encoding of auditory stimulus in children with Dyslexia. A few of them have been discussed below.

Rohith (2010) recorded ALLR in children in the age range of 2-15 years using speech and non speech stimuli. He found that as age increased, P1 latency reduced and absolute amplitude of P1 reduced for speech stimulus /da/ as well as for 500 Hz tone burst. P1 amplitude obtained in older children in the age range of 10-15 years was significantly lower than obtained in the younger group of 2-5 years old children, for both speech and non speech stimuli. On comparison, P1 amplitude was found to be larger for speech evoked stimulus than for 500 Hz tone burst. He observed reduced N1 latency and diminished amplitudes for speech stimulus /da/ however such pattern for non speech stimulus of 500 Hz was not observed. He reported a significant difference between speech and non speech stimulus for N1 latency in younger children (2-4.11 years) and for older children (10-15 years). No significant difference between latency and amplitude for children in the age range of 5-9.11 years was observed. The study also reported that P2 latency decreased as age increased for both speech stimulus /da/ and non speech stimulus of 500 Hz tone burst. Within the same age group, mean latencies were longer for P2 evoked by speech stimulus than for the ones evoked by non speech stimulus.

Byring and Jaryilheto (1985) studied cortical auditory evoked potentials in 23 young adolescent poor spellers and 21 normally developing children. They found increased latency and reduced amplitude of peaks P1 and P3 in the group of poor

spellers. They also exhibited prolonged latencies for P2. These abnormal findings were attributed to the deficits in early filtering processes of attention and also to a brain maturational delay that presents as an attentional disorder.

Tonnquist-Uhlen, Borg, Persson and Spen (1996) compared the P1-N1-P2 complex between a group of 20 children having severe Developmental Language Disorder (DLD) and a group of 20 normally developing children having average age of 12 years. Stimulus used was a pure tone of 500 Hz with an inter-stimulus interval of 1 second. The latency of peak N1 was significantly prolonged in children with DLD as compared to normally developing children. They attributed these findings to slower processing of signal in central auditory pathways of children with DLD probably due to delayed maturation.

Pinkerton, Watson and McClelland (1989) recorded P1-N1-P2 complex evoked in response to a 2000 Hz tone bursts in a group of 14 boys labelled as poor readers and compared it with a group of 18 normal readers. The age range of the participants was 8-9 years. They found significant reduction in the amplitude of P1 and N1 in the group of poor readers which was not observed in the normal readers. They suggested that reduced amplitude of P1 could reflect reduced or disturbed early auditory input to the left hemisphere whereas reduction in amplitude of N1 could be related to the processes mediating the attention.

Purdy, Kelly and Davies (2002) used cortical auditory evoked potentials to evaluate auditory processing in 10 children aged 7 to 11 years who were diagnosed as learning disabled (LD) and compared it with the potentials obtained from an age and gender matched control group consisting of 10 children. An oddball paradigm was used for recording with the 1000-Hz frequent and the 2000-Hz deviant tones

presented on 80 percent and 20 percent of trials, respectively, at 1.1/sec. The findings suggested that the earlier peaks, P1 and N1, had shorter latencies and smaller amplitudes in the LD group. P3 was later and smaller in the LD children for both standard and deviant stimuli. P2-N2 relative amplitude for the deviant stimulus was larger in the LD group. The reason for the unusual finding of shorter P1 latency was not clear, however the researchers related it to the abnormal metabolism and/or neurotransmitter function in the thalamic reticular activating system as seen in autism, stuttering, Schizophrenia, and Alzheimer's disease, which causes earlier latency of MLR wave Pb, which is regarded as equivalent to P1. Arehole (1995) reported even though there was an increase in latency of N1 and P2 in children with LD, it was not statistically significantly different from the typically developing group of children.

Putter-Katz et al. (2005) found latencies of ALLR recorded in response to speech syllable /da/ and /ga/ were prolonged in Dyslexia group as compared to those children who did not have a learning problem. Also, the amplitude of N1 was larger and P3 amplitudes were smaller in the dyslectic group. They concluded that the latency and amplitude of Auditory Event Related Potentials (AERPs) are sensitive measures of complexity of phonological processing in children with and without a learning disability.

Kumar and Gupta (2014) reported that children with dyslexia in the age range of 10-12 years had prolonged latencies and diminished amplitudes for P1, P2 and N2 of ALLR evoked to speech stimulus /da/. N1 component did not show statistically significant difference between the two groups.

Satterfield et al (1984) reported that average latency of P1 was 88.72 ms in children with LD. Along with latency, studies have also reported reduced amplitude of

P1 in this population. It was found to be ranging from -3.0 to -4.9 microvolt (Leppanen & Lyytinen, 1997). Most of the studies done in children with LD have also reported an increase in latency of N1 (Arehole, 1995; Jirsa & Clontz, 1990, Leppanen & Lyytinen, 1997; Tonnquist-Uhlen, 1996). The mean latency value reported in literature for this group ranges from 113 ms- 153 ms (Arehole, 1995; Jirsa & Clontz, 1990, Leppanen & Lyytinen, 1997; Tonnquist-Uhlen, 1996). Previous research has reported reduced N1 amplitude in children with LD (Brunswick & Rippon, 1994; Jirsa & Clontz, 1990; Radhika, 1997) which ranged from -0.1 to 1.93 microvolt (Brunswick & Rippon, 1994; Radhika, 1997). Amplitude of N1 in typically developing children has been reported to be ranging from -3.7 to 3.9 microvolt (Jirsa, 1992). Previous studies have reported prolonged P2 latency in children with LD (Arehole, 1995; Jirsa & Clontz, 1990, Leppanen & Lyytinen, 1997; Tonnquist-Uhlen, 1996) which ranges from 160-188 ms. Few studies Along with prolonged latencies, reduced absolute amplitudes of P2 have also been reported by many researchers, for children with LD (Brunswick & Rippon, 1994; Leppanen & Lyytinen, 1997; Tonnquist-Ulhen, 1996). The amplitude of N2 has been found to be ranging from -4.5 to 5.6 microvolt in children with LD (Satterfield et al, 1984; Tonnquist-Ulhen, 1996). have reported the absence of N2 altogether in children with LD (Duncan et al., 1994; Jirsa & Clontz, 1990; Radhika, 1997) while the others have reported increased amplitude of N2 in this population (Byring & Jaryeiehto, 1985; Mason & Mellor, 1984).

Few studies have found that P2 was not identifiable in children with LD (Jirsa & Clontz, 1990; Pinkerton et al, 1989; Radhika, 1997). Satterfield et al. (1984) reported a reduction in P2-N2 relative amplitude. In contradiction to the above findings a study by Jirsa (1992) reported normal P2 amplitudes in children with LD.

2.4 Effect of noise on ALLR in children with dyslexia

As we know the speech perception is hampered in the presence of noise for the normal hearing population as well as in clinical population. As reported by the studies carried out in children with Dyslexia ALLR components show significant differences in terms of latency and amplitude when elicited in quiet. So it is important to know what changes happen to the cortical response when noise is added to the stimulus.

Warrier and Johnson (2004) carried out a study which included 80 children having Learning problems and 32 typically developing children. ALLR was recorded in response to speech syllable /da/ of 40 ms in quiet as well as in presence of broadband noise. The results revealed that the mean N2 latency of the group of children with learning problems was approximately 20 ms as compared to the group of typically developing children and this was evident when a stimulus was presented in noise as opposed to in quiet. Amplitude of P2-N2 complex was found to be reduced in both the groups in presence of noise which suggests that addition of noise to speech stimulus affected perception in typically developing children and in children with learning problems.

Cunningham, Nicol, Zecker and Kraus (2000) examined the effect of noise on speech syllable /da/ for 150 typically developing children and a group of 86 children having learning disability. They observed two positive peaks P1 and P1' and two negative peaks N1 and N1' in response to 40 ms syllable da (40 msec). They reported that in noise, both normal and children with learning problems showed a reduction in P1-to N1 and P1'-to-N1' amplitude. This difference between the groups was not

noticed in quiet conditions. Also, the latencies were not significantly different between the two groups even in the presence of noise.

2.5 Behavioural speech perception in noise in children with Dyslexia

Many studies reported deficits in speech perception among a large proportion of the individuals with learning disabilities (Tallal, 1980; Elliott et al., 1989; Kraus et al., 1999; Baran, 2002). These deficits are reported to be enhanced in the presence of background noise (Nabelek and Pickett, 1974; Bellis, 1996; Chermak and Musiek, 1997; Cunningham et al., 2001).

Ziegler, Pech-Georgel, George and Lorenzi (2009) studied speech perception in noise in 19 children having dyslexia. One set of 48 unprocessed Vowel Consonant-Vowel (VCV) stimuli was recorded. A gated speech-shaped noise masker was added to each utterance. It was either stationary or Amplitude Modulated (AM). The results revealed that mean comparisons showed there was a clear speech perception deficit in all noise conditions but not in silence. The deficit was slightly more in amplitude modulated noise than in stationary noise.

Putter- Katz, Banai and Ahissar (2005) studied speech perception in noise in the group of 31 individuals with mild learning difficulties. (13.2 ± 0.4 years) and 19 age matched controls. 50 female native Hebrew speakers were chosen to deliver the stimuli. Bisyllabic pseudowords were recorded by a female native Hebrew speaker. The stimuli were normalized in terms of intensity and duration. The masking noise was generated by summing tones at: 500,530,550,580,600,620,680 and 700Hz, and multiplying the sum with a sinusoidal envelope at 4Hz. The intensity of the pseudowords was adapted in a 3 down/1 up staircase procedure giving the criteria of 80%

correct while the noise level remained fixed. Identification threshold (JND) was calculated as the arithmetic mean of last 10 reversals. They reported that performance between the two groups was not significantly different in presence of noise. This finding is contradictory to the finding of Chermak et al., (1989) who reported word identification performance of learning disabled adults was depressed relative to controls performance in the presence of speech spectrum noise. This could be due the differences in stimuli used in both the studies. Chermak et al. used monosyllabic words whereas Putter–Katz et al. used bisyllabic pseudowords which might have caused similar scores in both the groups due to redundancy and not actually reflecting perception in presence of noise. Elliot et al. (1979) also found better identification of monosyllabic nouns in noise among children achieving normal school progress compared to children with learning problems. So, monosyllabic words seems to be more sensitive to reflect speech perception in presence of noise for children with dyslexia.

Anderson, Chandrasekaran, Yi and Kraus (2010) recorded cortical responses to the 170 ms speech syllable /da/ in quiet and multi-talker babble noise in 32 typically developing children. They also correlated the cortical response to behavioural speech in noise understanding measured using Hearing in Noise Test (HINT) sentences. They divided the participants into two groups based on the HINT score i.e. the top SIN group and bottom SIN group. They reported a negative correlation between N2 amplitude and HINT score i.e. for children in the bottom SIN group, who did poorly on behavioural speech perception task, N2 amplitude increase when speech babble was added to /da/. This finding of larger rather than smaller N2 amplitudes in the bottom SIN perceivers was thought to suggest that the top SIN perceivers may be

recruiting fewer neural resources due to greater neural efficiency and hence getting smaller N2 amplitudes.

In literature there is a dearth of studies which attempt to correlate the findings of electrophysiological measures with the behavioural perception of speech in noise for children with dyslexia. Also the type of noise used in previous studies was continuous. Intermittent noise could affect the components of ALLR in a differential manner, like how fluctuating noise affects the behavioural speech perception differently than a stationary noise (Ziegler et al., 2009).

Chapter 3

Method

The present study was taken up with the aim to know whether different spectrum of noise has a differential effect on different components of ALLR in children with Dyslexia and those without Dyslexia. An attempt was also made to correlate the behavioural speech perception scores with the recorded evoked potentials. To achieve the objectives of the study following method was used.

3.1 Participants:

The study included two groups of participants, Group I had 10 children having Dyslexia and Group II had 15 typically developing children.

Group I: Clinical group (Children with Dyslexia)

Participants were selected based on the following criteria:

1. 10 participants in the age range of 8 to 14 years diagnosed as having Dyslexia by Speech Language Pathologist and Psychologist at All India Institute of Speech and Hearing were considered. Skill level of participants was between II to V grade below their chronological age on the Early Reading Skills (Loomba, 1995).
2. The participants had normal hearing sensitivity i.e. thresholds better than 15 dB HL in the frequency range of 250 Hz to 8000 Hz.
3. The participants were studying in an English medium school however their mother tongue was Kannada. All of them had Speech identification scores of 90% or above at 40 dB SL.

4. The participants had “A” type tympanogram with normal acoustic reflexes to ensure the absence of conductive component.
5. Auditory Brainstem Response (ABR) was present at 80 dBnHL for all the participants.
6. SCAP (Screening Checklist for Auditory Processing) developed by Yathiraj and Mascarenhas (2004) was administered on all the children to rule out presence of Auditory Processing Disorder as Dyslexia and APD often coexist. Participants at risk for Auditory processing disorder, i.e. having a score of 6 or more out of 12 were also included in the study.
7. None of the participants reported of any neurological and otological disorder.

Group II: Control group (Typically developing children)

Participants were selected based on the following criteria:

1. All 15 participants were in the age range of 8 to 14 years
2. The participants had normal hearing sensitivity indicated by air conduction thresholds better than 15 dB HL in the frequency range of 250 Hz to 8000 Hz.
3. The participants were studying in an English medium school however they were native speakers of Kannada. Speech identification scores were 90% or above at 40 dB SL for all the children.
4. All the participants had “A” type tympanogram with normal acoustic reflexes to ensure the absence of conductive component.
5. ABR was present for all the participants at 80 dBnHL SCAP scores obtained for participants revealed no risk of having Auditory processing disorder.
6. None of the participants had any indication of any neurological and otological disorder.

3.2 Instrumentation:

- A two channel diagnostic audiometer, GSI-61 (Grason-Stadler Incorporation, USA) with Telephonics TDH 50 supra aural headphones and Radio ear B-71 bone vibrator calibrated as per ANSI (2004) was used for threshold estimation.
- A calibrated GSI- tymptstar (Grason-Stadler Incorporation, USA) clinical immittance meter, calibrated as per ANSI (1987) was used for tympanometry and reflexometry.
- Intelligent Hearing System (HIS smart EP windows USB version 4.3.02) with AgCl electrodes and ER-3A insert earphones was used to record Auditory Brainstem Response and Auditory Late Latency Responses.
- A HP laptop with Adobe Audition version 3.0 was used to normalize the recorded stimulus used for the study and also to carry out speech audiometry and Speech Perception In Noise test using the recorded material loaded in the system.

3.3 Procedure:

1. **Pure-tone thresholds** were obtained using modified version of Hughson and Westlake procedure (Carhart & Jerger, 1959) at octave frequencies between 250 Hz to 8000 Hz for air conduction and between 250 Hz to 4000 Hz for bone conduction.
2. Ascending method was used to determine **participant's Uncomfortable Loudness Level (UCL)** for both ears using speech stimuli that was presented through headphones (TDH 50).

3. **Speech Recognition Scores** were obtained using monosyllabic PB word list in English developed for children by Rout and Yathiraj (1996).
4. **Speech Perception In Noise (SPIN)** scores were obtained at 0 dB SNR monaurally at 40 dB SL (reference SRT). A PB word list consisting of 25 monosyllabic words in English developed for children by Yathiraj, Vanaja and Muthuselvi (2009) was used to carry out SPIN.
5. **SCAP** (Screening Checklist for Auditory Processing) developed by Yathiraj and Mascarenhas (2004) was administered on all the participants to rule out presence/absence of auditory processing disorder
6. **Immittance evaluation** was carried out with a probe tone frequency of 226 Hz. Ipsilateral and contralateral acoustic reflex thresholds were measured for 500, 1000, 2000, and 4000 Hz.
7. **Auditory Brainstem Response (ABR)** was carried out at the intensity of 80 dBnHL using click stimulus at the stimulation rate of 11.1/s. the standard protocol was followed for recording of ABR.

Phase I: Stimulus preparation

Stimuli selected for the current study consisted of 2 CV syllables in the context of the vowel /a/. Speech syllables /ba/ and /da/ were recorded digitally in a sound treated room by a native male Kannada speaker. Adobe Audition version 3.0 software was used for this purpose. The recording was done on a data acquisition system using 44100 Hz sampling frequency with a 32 bit analogue to digital converter. A high quality omni directional microphone was used to record the stimulus.

Each syllable (CV) was recorded five times and the middle three recordings were considered for rating.. The stimulus was given to 5 Audiologists to perceptually rate the best recording out of three recording for each CV syllable. It was rated based on a 3 point rating scale. The best rated one was chosen and later were mixed with noise of different types.

Signal processing:

The stimulus consisted of 500 ms of noise onto which, a 100 ms stop consonant was mixed at 300 ms. The speech stimulus was inserted after 300 ms in order to avoid the interference caused by the ALLR recorded in response to onset of noise. The post speech stimulus noise was added to avoid overlap of response generated due to the offset of stimulus. Different stimulus conditions used are shown in figures below.

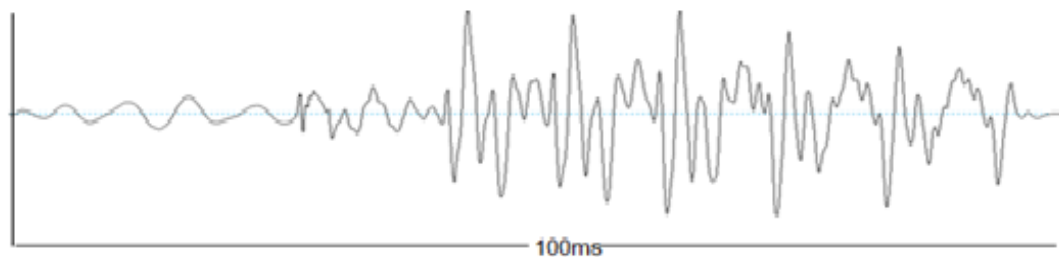


Figure 3.1.: Syllable /ba/ of 100 ms.

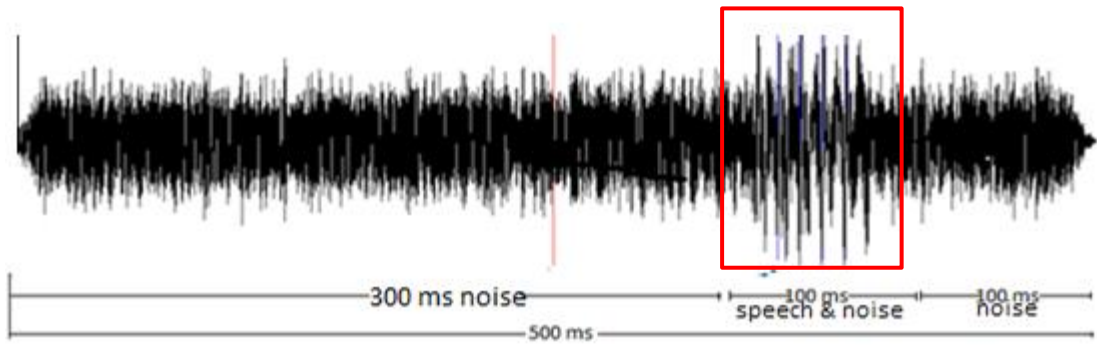


Figure 3.2.: speech syllable /ba/ occurring at 300 ms of high pass filtered noise having a cut off 4000 Hz.

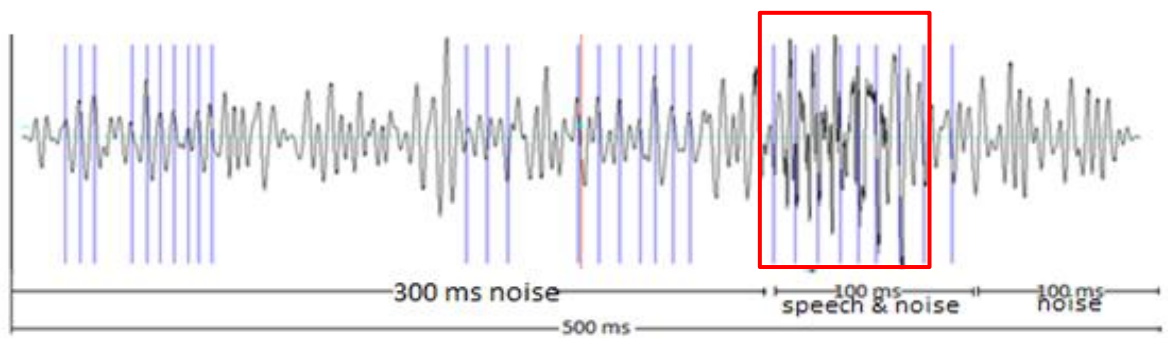


Figure 3.3.: speech syllable /ba/ occurring at 300 ms of low pass filtered noise having a cut off 200 Hz.

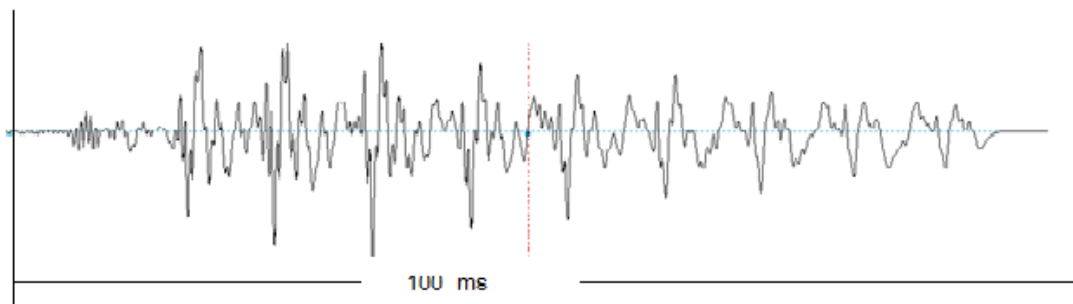


Figure 3.4.: Syllable /da/ of 100 ms.

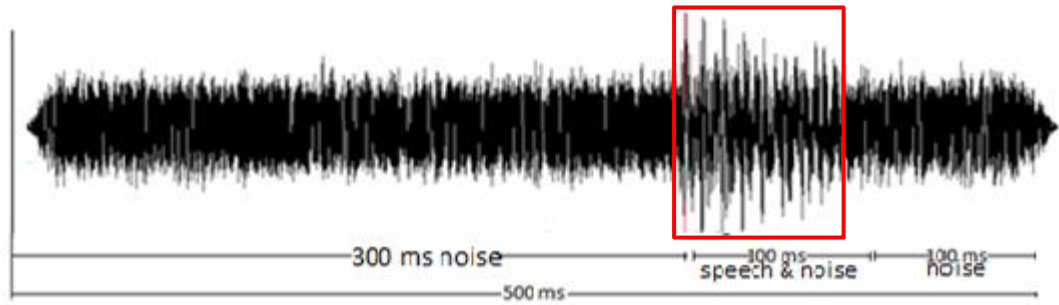


Figure 3.5.: speech syllable /da/ occurring at 300 ms of high pass filtered noise having cut off 4000 Hz.

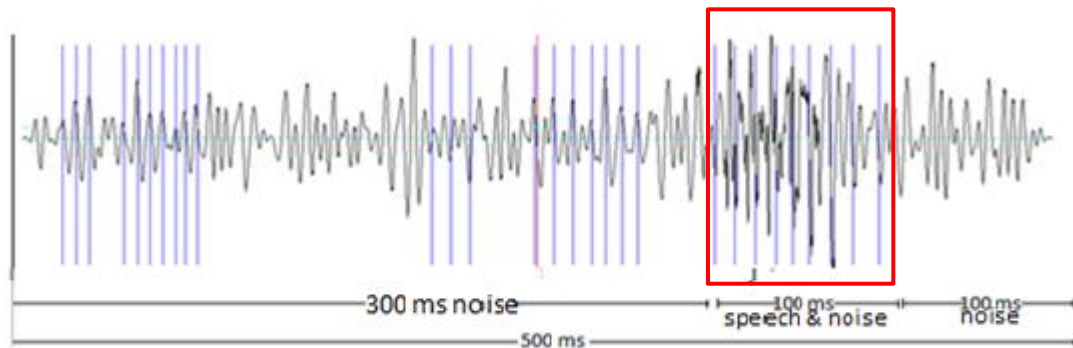


Figure 3.6.: speech syllable /da/ occurring at 300 ms of low pass filtered noise having a cut off 200 Hz.

Two types of noise were considered: Low pass noise (<200Hz) and High pass noise (>4000Hz). These two cut off frequencies were selected based on previous studies using similar cut off values (Martin & Stapells, 1997, 1999, 2005). Also, another intention was for the noise to not affect intelligibility much. These different types of noise having duration of 500 ms were generated with software Aux Viewer 1.37. Two stop consonants /ba/ and /da/ of 100 ms were taken as speech stimuli. RMS value of speech signal was found and noise RMS value was equated to it. Then both noise and speech stimuli were mixed to achieve 0 dB SNR. Noise was added in such a way that it started 300 ms before the speech stimulus and continued

for 100 ms after the speech stimulus ended. The initial and final 10 ms were ramped with a cosine window to ensure smooth onset and offset. All the above were carried out with the Adobe Audition software, version 3.0.

The specific speech stimulus was chosen because /ba/ and /da/ differ in terms of place of articulation and hence in the frequency domain (Martin et al. 1997). These speech syllables differ primarily in the second and third formant onset transition frequencies which are important for speech perception. Syllable /ba/ has spectral energy mainly in the lower frequency region whereas /da/ has a broader spectrum as compared to /ba/ with more spectral energy at higher frequencies (Vesco, Bon, Ryan & Polich, 1993). Also, previously done studies (Martin & Stapells 1997, 1997, 2005) have examined the effect of differential noise on ALLR using speech stimuli /ba/ and /da/ so it would be easier to discuss the findings of the current study with respect to their findings.

Phase II: Recording of evoked potentials

The participants were seated in an electrically and acoustically shielded room in a comfortable reclining chair. They were made to watch a movie of their choice with the soundtrack turned off. They were instructed not to pay attention to the stimulus and to avoid excessive eye blinking while the recording proceeded. A skin abrasive was used to clean the electrode sites in accordance with the 10-20 International system (Jasper, 1958). The disc electrodes dipped in a conduction paste were placed on the proposed sites and were secured with a surgical tape. The non-inverting electrode was positioned on vertex (Cz) and the inverting electrode on the mastoid of test ear. Ground electrode was placed on lower forehead. Ocular channel

was used to eliminate ocular artifacts. The Auditory Late Latency Response were obtained for the following stimulus conditions:

- 1) Baseline ALLR for speech stimulus /da/ having a duration of 100 ms.
- 2) ALLR for /da/ with high pass noise with cutoff frequency of 4000 Hz with a total duration of 500 ms.
- 3) ALLR for /da/ with low pass noise with cutoff frequency of 200 Hz with a total duration of 500 ms.
- 4) Baseline ALLR for speech stimulus /ba/ having duration of 100 ms.
- 5) ALLR for /ba/ with high pass noise with cutoff frequency of 4000 Hz. with a total duration of 500 ms.
- 6) ALLR for /ba/ with low pass noise with cutoff frequency of 200 Hz with a total duration of 500 ms.

The order of presentation was randomized to avoid order effect. The speech stimulus was presented in 5 sets of 30 sweeps in all 3 conditions. The recordings with less noise were considered. The following protocol was followed:

ALLR was recorded twice for each stimulus condition in order to replicate it. Waveforms were given to experienced audiologists to identify the peaks. Following protocol was used to record cortical potentials.

Table 3.1. *Stimulus and Acquisition parameters used for recording ALLR*

Stimulus parameters		Acquisition parameters	
Speech stimulus	/ba/, /da/	Analysis time	-50 ms to 1024 ms
Duration of stimulus	1. /ba/ and /da/ in quiet condition 100 ms 2. In presence of noise of 500 ms (300 ms of noise + 100 ms of speech stimulus and noise + 100 ms of noise post stimulus)	Band pass filter	1 Hz- 30 Hz.
Noise	Low pass noise (<200 Hz), High pass noise (>4000 Hz)	Number of channels used	2 channels Channel A: to record ALLR Channel B: ocular activity
Stimulus level	70 dB SPL	Sweeps	150
Polarity	Alternating	Electrode impedance	$\leq 5 \text{ k } \Omega$
Transducer	Insert earphones ER-3A	Inter electrode impedance	$\leq 2 \text{ k } \Omega$
Repetition rate	0.9/sec	Number of recordings	2 for reproducibility
Mode of presentation	Ipsilaterl presentation of speech stimulus in quiet and in presence of noise monaurally.	Notch filter	Off
		Artefact rejection	100 mV
		Gain	Channel A : 50000 (for ALLR) Channel B: 5000 (ocular activity)

3.4 Response Analysis:

In the stimulus initially 300 ms of continuous noise was present which lasted throughout. After 300 ms the speech stimulus was presented. An ALLR for speech stimulus was recorded. This ALLR waveform for speech stimulus was considered for analysis.

The latency and absolute amplitude of the P1, N1 and P2 were measured with respect to the baseline and subjected to analysis. Mean and Standard Deviation of amplitude and latency were calculated. The following parameters were considered for statistical analysis:

- Comparison of amplitude of P1, N1 and P2 for different stimulus conditions within group.
- Comparison of latency of P1, N1 and P2 for different stimulus conditions across groups.
- Comparison of latency of P1, N1 and P2 for different stimulus conditions within group (for typically developing children as well as for the children with dyslexia)
- Comparison of amplitude of P1, N1 and P2 for different stimulus conditions across groups.
- Correlation between behavioural speech identification and components of ALLR within group.

The ALLR components P1, N1 and P2 were considered for analysis because the previous studies have concluded that N1 marks the presence of audible energy (Martin & Stapeles 1997). Also early latencies and reduced amplitude of P1 were found in children with Learning Disability as compared to that of the typically

developing group (Purdy, Kelly & Davies, 2002). N2 indexes the presence of discrimination ability of the individual (Martin & Stapeles 1997). However it was not considered for the analysis as it could not be recorded even in most of the typically developing children.

Chapter 4

Results

The aim of the study was to know whether different spectrum of noise has a differential effect on different components of ALLR in children with Dyslexia and in typically developing children. To achieve this, the latencies and baseline to peak amplitude of P1, N1 and P2 were calculated. These parameters were compared across the two groups as well as across the three conditions within the same group i.e. P1, N1, and P2 obtained in quiet condition were compared with those obtained in high pass and low pass noise conditions. The other aim of the study was to find out which of those components best correlates with behavioural speech perception ability measured by Speech Perception In Noise (SPIN) scores in response to monosyllabic words at 0 dB SNR.

Data obtained from the two groups were subjected to statistical analysis using Statistical Package for the Social Sciences, SPSS, (version 20.0). Initially normality test was administered to see whether data obtained across different stimulus conditions in both the groups followed normal distribution. Data obtained from many of the participants from both clinical and control group was not normally distributed for many parameters as seen on Shapiro Wilk test. Also the sample size of the two groups was unequal i.e. there were 10 participants in the clinical group whereas control group consisted of 15 participants. So, Non-Parametric tests were administered for the data analysis. The following statistical analysis were carried out across groups and within the group across conditions for ALLR parameters for syllables /ba/ and /da/.

- **Descriptive statistics:** was administered to obtain the mean, median and standard deviation for latency and amplitude of P1, N1 and P2 across all the conditions elicited by both the stimuli in two groups. Mean and Standard Deviation was also obtained for behavioural SPIN scores for both the groups.
- **Man Whitney U test:** was carried out to see the significant difference between the groups for above mentioned parameters in all test conditions. Also the differences across groups were obtained for SPIN scores based on this test.
- **Friedman's test:** was administered to see the differences across conditions within the same group i.e. to see whether there is a significant main effect on ALLRs obtained in response to syllable /ba/ and /da/ in quiet, in presence of high pass noise and in presence of low pass noise for typically developing children and similarly for children with Dyslexia.
- **Wilcoxon Signed Rank test:** was administered when there was a statistically significant main effect of condition obtained on Friedman's test.
- **Spearman's rank-order correlation:** was administered to see whether there is a correlation between behavioural SPIN scores and latency and amplitude of different components of ALLR in three stimulus conditions for each stimulus (/ba/ and /da/) within the group.

Test results of the current study are discussed under the following headings:

- 4.1. Comparison of latency and amplitude of ALLR evoked in response to speech syllable /ba/ between typically developing children and children with Dyslexia
- 4.2. Comparison of latency and amplitude of ALLR in response to /ba/ across different test conditions within the group.

4.3. Comparison of latency and amplitude of ALLR evoked in response to speech syllable /da/ between typically developing children and children with Dyslexia

4.4. Comparison of latency and amplitude of ALLR in response to /da/ across different test conditions within the group.

4.5. Comparison of SPIN scores between the two groups.

4.6. Correlation between ALLR components and SPIN score within the group.

4.1. Comparison of latency and amplitude of ALLR evoked in response to speech syllable /ba/ between typically developing children and children with Dyslexia

Latency and absolute amplitude of the peaks P1, N1 and P2 were noted for speech stimulus /ba/ across the stimulus conditions in the group of children having Dyslexia. Descriptive statistical analysis was administered to obtain mean, median and standard deviation. This can be seen in Table 4.1.

Table 4.1. *Mean, Median and Standard Deviation (SD) for P1, N1 and P2 latency and baseline to peak amplitude obtained at 70 dB SPL for group I for three conditions in response to syllable /ba/*

Condition	Latency			Amplitude				
	Mean (ms)	Median (ms)	SD	Mean (μ V)	Median (μ V)	SD		
Quiet	P1	76.2	76	7.20	3.28	3.12	0.85	
	N1	130.6	131	7.42	-2.85	-2.14	1.64	
	P2	190.2	192	13.38	1.60	1.66	0.51	
Group I (N=10)	High pass noise	P1	84.4	85	5.79	2.61	2.52	0.93
		N1	137.8	139	13.21	-2.14	-1.59	1.68
	P2	190.0	190	24.05	1.10	1.17	1.73	
	Low pass noise	P1	92.00	90	6.18	3.28	2.96	1.83
		N1	156.2	151	16.95	-1.64	-1.60	0.58
		P2	203.3	210	15.14	1.10	0.18	1.73

As reported in Table 4.1, the latencies of all the peaks were prolonged in presence of high pass noise as well as in low pass noise as compared to quiet condition. But low pass noise condition caused more prolongation of latencies than high pass noise for P1, N1 as well as P2. For amplitude this kind of trend was not found. Even though amplitudes of all the parameters reduced in presence of noise, P1 showed more diminished amplitude in presence of high pass noise whereas N1 showed more reduction in amplitude in presence of low pass noise. P2 was affected similarly by both the noises.

Like for the clinical group, mean, median and standard deviation of the above mentioned ALLR components were also calculated for the group of typically developing children across different stimulus conditions for stimulus /ba/. The values are depicted in Table 4.2.

Table 4.2. *Mean, median and Standard Deviation (SD) for P1, N1 and P2 latency and baseline to peak amplitude obtained at 70 dB SPL for group II across conditions in response to syllable /ba/.*

Condition		Latency			Amplitude			
		Mean (ms)	Median (ms)	SD	Mean (μ V)	Median (μ V)	SD	
Group II (N=15)	Quiet	P1	60.66	58	11.25	1.46	1.55	.92
		N1	119.49	120	9.54	-3.25	-3.23	1.56
		P2	181.06	180	17.85	1.32	1.14	1.21
	High pass noise	P1	76.40	80	17.69	3.27	3.27	0.98
		N1	125.60	132	13.58	-2.15	-2.38	1.30
		P2	186.8	194	25.21	1.92	0.96	1.35
	Low pass noise	P1	82.26	86	15.65	3.89	2.36	2.94
		N1	124.66	124	20.80	-1.57	-1.25	1.09
		P2	184.54	192	29.03	1.49	1.00	1.26

It can be read from the Table 4.2 that latencies of P1, N1 and P2 are prolonged in noise conditions as compared to quiet condition. However a specific trend could not be obtained across the noise conditions i.e. latency of P1 is more in presence of low pass noise than in high pass noise whereas latencies of N1 and P2 are more prolonged in presence of high pass noise than in low pass noise.

In terms of mean amplitude, P1 and P2 showed increased absolute amplitude in presence of both high pass and low pass noise as compared to the quiet condition. In contrast, N1 demonstrated reduction in amplitude in both high pass and low pass noise conditions.

Mann Whitney U test was administered to see if there was a significant difference between the groups in terms of latency and amplitude of peaks P1, N1 and P2 for syllable /ba/. The results are shown in Table 4.3.

Table 4.3. *Mann Whitney U test results for ALLR peaks obtained across group I and Group II in response to speech syllable /ba/*

Conditions		Latency		Amplitude	
		z value	Sig. level (<i>p</i>)	z value	Sig. level (<i>p</i>)
Quiet	P1	3.25	0.001	3.69	0.000
	N1	2.64	0.008	0.80	0.421
	P2	1.61	0.107	0.71	0.474
High pass noise	P1	1.08	0.277	0.58	0.560
	N1	1.89	0.058	1.11	0.267
	P2	0.29	0.769	0.42	0.672
Low pass noise	P1	1.87	0.062	0.08	0.934
	N1	3.47	0.001	0.52	0.598
	P2	1.09	0.275	1.09	0.275

Bold indicates significant difference ($p < 0.05$).

Note. Sig. level = significant level

Significant difference between typically developing children and children with Dyslexia was observed for latency and amplitude of peak P1 in quiet condition.

Latency of peak N1 was also significantly different across the two groups for quiet condition as well as when low pass noise of cut off frequency of 200 Hz was mixed with speech syllable /ba/. However other parameters did not show any significant difference between the groups in any other conditions.

4.2. Comparison of latency and amplitude of ALLR evoked in response to speech stimulus /ba/ across different test conditions within the group

Friedman's test was carried out to see whether there was a significant main effect of conditions within the group. P2 latency and amplitude was not subjected to this test as few of the children in both groups had absent P2 in one or the other condition. So, peak P2 was subjected to Wilcoxon Signed Rank test directly for pairwise comparison.

Table 4.4. *Friedman's test results for latency and amplitude of P1 and N1 across the three stimulus conditions obtained for stimulus /ba/ within group*

Peak	Control group				Clinical group			
	Latency (ms)		Amplitude(μ V)		Latency (ms)		Amplitude (μ V)	
	χ^2	Sig.	χ^2	Sig.	χ^2	Sig.	χ^2	Sig.
	value	level(<i>p</i>)	value	level(<i>p</i>)	value	level(<i>p</i>)	value	level(<i>p</i>)
P1	12.93	0.002	6.53	0.038	14.82	0.001	3.20	0.20
N1	3.28	0.19	4.71	0.09	15.00	0.001	6.20	0.04

Bold Indicates significant difference

Note. Sig. level = significance level

It can be observed from Table 4.4. that condition had significant effect on P1 latency for both control and clinical group. Significant effect was seen in P1 amplitude for control group only whereas significant effect of condition in N1 latency was observed only for clinical group.

Wilcoxon Signed Rank test was administered for the parameters in which the significant difference was noted in Friedman's test. This was done especially to see in which two conditions ALLR components were significantly different from each other. i.e. whether significant difference was present between ALLR recorded in quiet condition versus that recorded in presence of high pass noise or whether it was between the response in quite versus that in presence of low pass noise or between the one recorded in presence of high pass noise versus in low pass noise. The results are shown in Table 4.5.

Table 4.5. *Wilcoxon Signed Rank test for amplitude and latency of peaks P1 and P2 evoked in response to speech stimulus /ba/ across test conditions for control group*

Peak	Conditions	Latency		Amplitude	
		z value	Sig. level (<i>p</i>)	z value	Sig. level (<i>p</i>)
P1	H – Q	2.72	0.006	2.89	0.004
	L – Q	2.98	0.003	2.38	0.017
	L – H	1.08	0.280	0.65	0.513
P2	H - Q	0.77	0.441	0.88	0.374
	L -Q	1.20	0.230	0.35	0.722
	L -H	0.50	0.611	1.35	0.176

Bold indicates significant difference

Note. Sig. level = significance level

Q = ALLR obtained in quiet condition

H = ALLR obtained in presence of high pass noise of 4000 Hz.

L = ALLR obtained in presence of high pass noise of 200 Hz.

(Same abbreviation is used for other tables.)

Above table shows that significant difference in the P1 latency and amplitude was observed between quiet condition and presence of high pass noise. The difference in P1 amplitude and latency was also significant between quiet condition and in presence of low pass noise. None of the other parameters reached the significance difference level between any two conditions for P1 and P2 peak.

Similar to control group, Wilcoxon Signed Rank test was also administered for clinical group. Results are shown in Table 4.6.

Table 4.6. *Wilcoxon Signed Rank test for amplitude and latency of peaks P1, N1 and P2 evoked in response to speech stimulus /ba/ across test conditions for clinical group*

Peaks	Conditions	Latency		Amplitude	
		z value	Sig. level (<i>p</i>)	z value	Sig. level (<i>p</i>)
P1	H – Q	2.57	0.010	-	-
	L – Q	2.67	0.007	-	-
	L – H	2.50	0.012	-	-
P2	H – Q	0.59	0.553	0.10	0.917
	L – Q	1.60	0.109	1.34	0.180
	L – H	1.34	0.180	0.44	0.655
N1	H – Q	1.22	0.219	1.88	0.059
	L – Q	2.80	0.005	1.88	0.049
	L – H	2.80	0.005	0.05	0.959

Bold indicates a significant difference.

Note. Sig. level = significance level

Latency of peak P1 was significantly different between each pair compared. Significant different was also obtained for latency of peak N1 when compared between quiet condition and in presence of low pass noise and also between low pass noise and high pass noise. N1 amplitude showed significant difference between quiet condition and low pass noise condition. No other two conditions showed any significant different for any other parameter. Amplitude of P1 was not subjected to this test as there was no significant effect of condition obtained in Friedman's test itself.

4.3. Comparison of latency and amplitude of ALLR evoked in response to speech syllable /da/ between typically developing children and children with Dyslexia

Latency and absolute amplitude of the peaks P1, N1 and P2 were noted for speech stimulus /da/ across the stimulus conditions in the group of children having Dyslexia. Descriptive statistical analysis was administered to obtain mean, median and standard deviation. This can be seen in Table 4.7.

Table 4.7. Mean, median and Standard Deviation (SD) for P1, N1 and P2 latency and baseline to peak amplitude obtained at 70 dB SPL for group I for three conditions in response to syllable /da/

condition			Latency (ms)			Amplitude (μ V)		
			Mean	Median	SD	Mean	Median	SD
Group I (N=10)	Quiet	P1	74.40	77	6.44	2.49	2.59	1.36
		N1	133.40	132	6.73	-2.69	-1.96	2.56
		P2	191.75	189	11.78	0.81	0.75	0.85
	High pass noise	P1	82.40	82	4.69	2.37	2.30	0.96
		N1	133.80	136	7.91	-1.41	-1.30	0.57
		P2	180.80	176	18.47	0.91	1.07	0.44
	Low pass noise	P1	78.40	78	8.31	2.45	2.19	1.12
		N1	127.20	127	14.94	-1.53	-1.46	1.08
		P2	180.00	184	18.47	1.68	1.21	1.58

As reported in table 4.7 latencies of peaks P1 and N1 were prolonged in presence of high pass noise as well as low pass noise when compared to quiet condition. However latency of P2 was earlier in both types of noises as compared to quiet condition. In terms of amplitude, peaks P1 and N1 showed reduction in presence high pass and low pass noise whereas P2 exhibited an increase in amplitude in presence of both types of noise.

Table 4.8. Mean, median and Standard Deviation (SD) for P1, N1 and P2 latency and baseline to peak amplitude obtained at 70 dB SPL for group II for three conditions in response to syllable /da/

conditions			Latency (ms)			Amplitude (μ V)		
			Mean	Median	SD	Mean	Median	SD
Group II (N=15)	Quiet	P1	66.26	66	11.56	2.14	2.26	0.56
		N1	121.06	122	13.68	-2.96	-3.28	2.56
		P2	177.84	180	17.23	1.83	1.53	1.28
	High pass noise	P1	75.33	78	11.20	2.64	2.94	1.45
		N1	127.06	130	9.52	-2.52	-2.31	1.92
		P2	179.45	182	15.18	1.70	1.72	0.98
	Low pass noise	P1	72.40	74	14.06	3.66	3.66	1.91
		N1	127.60	124	8.95	-2.04	-1.77	1.08
		P2	188.50	192	19.9	2.31	1.60	2.27

As it can be read from Table 4.8, latencies of all the components of ALLR increased in presence of high pass and low pass noise as compared to quiet condition. In terms of absolute amplitude, P1 showed an increase whereas N1 showed a decrease in presence of both types of noises. Peak P2 demonstrated an increase in amplitude in presence of low pass filtered noise whereas a decrease in amplitude was noted in presence of high pass filtered noise.

To see if there was a statistically significant difference between the two groups, in terms of latency and amplitude Mann Whitney U test was administered. The results are shown in table 4.9.

Table 4.9. *Mann Whitney U test results for ALLRs obtained across group I and Group II in response to speech syllable /da/*

Conditions		Latency		Amplitude	
		z Value	Sig. level (<i>p</i>)	z value	Sig. level (<i>p</i>)
Quiet	P1	2.01	0.044	0.94	0.346
	N1	2.65	0.008	1.61	0.107
	P2	1.59	0.110	2.32	0.020
High pass noise	P1	1.591	0.112	0.388	0.698
	N1	1.481	0.139	1.110	0.267
	P2	0.114	0.910	1.871	0.061
Low pass noise	P1	0.947	0.344	1.915	0.056
	N1	0.278	0.781	0.943	0.346
	P2	1.100	0.271	0.508	0.611

Bold indicates significant difference.

Note. Sig. level = significance level

Significant difference between the latency of peak P1 and N1 and amplitude of P2 was found between the two groups of participants, only in quiet condition. There was no significant difference between typically developing children and children with Dyslexia in terms of latency and amplitude of all the peaks in presence of high pass and low pass noise.

4.4. Comparison of latency and amplitude of ALLR evoked in response to speech syllable /da/ across different test conditions within the group

Friedman's test was carried out to see whether there was a significant effect of condition on P1 and N1 latency and amplitude within the group. The results are shown in Table 4.10. Peak P2 was directly subjected to Wilcoxin signed rank test as few of the children in both groups had absent P2 in one or the other condition.

Table 4.10. *Friedman's test results for latency and amplitude of P1 and N1 across the three stimulus conditions obtained for syllable /da/ within control and clinical group.*

Parameter	Control group				Clinical group			
	Latency		Amplitude		Latency		Amplitude	
	χ^2	Sig.	χ^2	Sig.	χ^2	Sig.	χ^2	Sig.
	value	level (<i>p</i>)	value	level (<i>p</i>)	value	level (<i>p</i>)	value	level (<i>p</i>)
P1	5.71	0.057	8.93	0.011	7.53	0.023	1.40	0.49
N1	3.34	0.18	7.42	0.024	4.15	0.12	2.40	0.30

Bold indicates a significant difference.

Note. Sig. level (*p*) = significance level

It can be observed from Table 4.10 that condition had a significant effect on P1 latency only for clinical group whereas significant effect was seen on amplitude of P1 in the control group. In terms of amplitude control group showed a significant effect of condition for P1 and N1 both while for the clinical group no condition effect was found for either P1 or N1.

Wilcoxon Signed Rank test was also administered for the parameters in which the significant difference was noted in Friedman’s test. Results obtained are shown in table 4.11.

Table 4.11. *Wilcoxon Signed Rank test for amplitude and latency of peaks P1, N1 and P2 evoked in response to speech stimulus /da/ across test conditions for control group*

Peaks	Conditions	Latency		Amplitude	
		z value	Sig. level (<i>p</i>)	z value	Sig. level (<i>p</i>)
P1	H – Q	-	-	1.194	0.233
	L – Q	-	-	2.954	0.003
	L – H	-	-	2.045	0.041
P2	H – Q	0.357	0.721	0.623	0.533
	L – Q	0.765	0.444	0.561	0.575
	L – H	0.677	0.498	0.561	0.575
N1	H – Q	-	-	1.601	0.109
	L – Q	-	-	2.954	0.003
	L – H	-	-	1.023	0.307

Bold indicates significant difference ($p < 0.05$)

Note. Sig. level = significance level

Significant difference was found between amplitude of peak P1 and N1 recorded in quiet and in presence of low pass noise. No significant difference was found for the peak P2 across conditions. Latency of P1 and N1 was not subjected to this test as there was no significant effect shown in Friedman’s test.

Table 4.12. *Wilcoxon Signed Rank test for amplitude and latency of peaks P1, N1 and P2 evoked in response to speech stimulus /da/ across test conditions for clinical group*

Peaks	Conditions	Latency		Amplitude	
		z	Sig. level (<i>p</i>)	z	Sig. level (<i>p</i>)
P1	H – Q	2.051	0.012	-	-
	L – Q	1.232	0.218	-	-
	L – H	1.643	0.100	-	-
P2	H – Q	0.000	1.00	1.826	0.068
	L – Q	0.000	1.00	0.135	0.893
	L – H	0.365	0.715	1.095	0.273

Bold indicates a significant difference.

Note. Sig. level = significance level

Latency of P1 was found to be significantly different between quiet condition and in presence of high pass noise whereas for peak P2 there was no significant difference between the conditions in terms of amplitude as well as latency.

4.5. Comparison of SPIN scores between the two groups.

For the group of typically developing children, mean SPIN score was 78.40 % with a Standard Deviation (SD) of 8.65 whereas for the group of children with dyslexia the mean score was 59.20 % with an SD of 17.66. A significant difference was found between the scores of the two groups when Man Whitney U test was administered ($p = 0.004$).

4.6. Correlation between ALLR components and SPIN score within the group for /ba/ and /da/ stimulus.

As one of the aims of the study was to correlate the findings of behavioural SPIN score with latency and amplitude of different components of ALLR in different stimulus conditions, Spearman’s rank-order correlation was administered. The results are depicted in the table below.

Table 4.13. *Correlation between latency and amplitude of peaks P1, N1 and P2 with behavioural SPIN scores for different conditions within group for stimulus /ba/*

Peaks	condition	SPIN							
		Control				Clinical			
		Latency		Amplitude		Latency		Amplitude	
		r	p	r	p	r	p	r	p
P1	Q	0.80	0.77	0.13	0.63	-0.53	0.11	0.10	0.77
	H	0.27	0.92	-0.13	0.62	0.04	0.89	-0.03	0.93
	L	-0.32	0.24	0.24	0.37	0.89	0.13	0.17	0.62
N1	Q	-0.19	0.47	-0.21	0.44	0.09	0.80	0.30	0.40
	H	-0.16	0.55	0.13	0.63	0.21	0.55	-0.01	0.96
	L	0.29	0.28	0.33	0.22	0.47	0.16	-0.43	0.20
P2	Q	-0.09	0.73	0.12	0.66	-0.60	0.06	0.33	0.37
	H	-0.06	0.85	0.10	0.78	0.32	0.48	-0.64	0.11
	L	0.22	0.50	0.20	0.50	0.86	0.33	-0.86	0.33

Note. r indicates correlation coefficient

As seen from the table, no significant correlation was found between the behavioural SPIN scores and parameters of ALLR considered in any of the stimulus conditions for typically developing children as well as for the children having Dyslexia. The minus sign in the table indicates negative correlation between the SPIN

scores and the parameter under concern. However, correlation between the two, for any parameter did not reach the significance value (p was > 0.05)

Similarly Spearman's rank-order correlation was also administered to see the correlation between ALLR components P1, N1 and P2 evoked in response to stimulus /da/ and SPIN scores. The results are depicted in Table 4.14.

Table 4.14. *Correlation between latency and amplitude of peaks P1, N1 and P2 with behavioural SPIN scores for different conditions within group for speech stimulus /da/*

Parameter	Condition	SPIN							
		Control				Clinical			
		Latency		Amplitude		Latency		Amplitude	
		r	P	R	p	r	p	r	p
P1	Q	-0.20	0.47	-0.09	0.73	-0.36	0.29	0.34	0.33
	H	-0.45	0.08	-0.13	0.63	0.46	0.17	-0.15	0.67
	L	-0.65	0.008	0.02	0.93	0.16	0.65	-0.18	0.61
N1	Q	-0.16	0.55	-0.44	0.09	-0.51	0.12	0.21	0.55
	H	-0.14	0.61	-0.007	0.97	0.55	0.09	-0.24	0.49
	L	-0.17	0.53	-0.34	0.21	0.15	0.67	-0.20	0.57
P2	Q	0.06	0.82	0.34	0.24	-0.32	0.49	-0.03	0.93
	H	0.10	0.76	0.43	0.18	0.90	0.03	-0.40	0.50
	L	-0.54	0.07	0.08	0.79	-0.18	0.96	-0.05	0.90

Bold indicates significant difference

Note. r indicates correlation coefficient

From Table 4.14 it can be seen that in the group of typically developing children a significant negative correlation was obtained between behavioural SPIN scores and latency of P1 in presence of low pass noise($r = -0.65$, $p = 0.008$). on the

other hand, for the group of children with Dyslexia a significant positive correlation was found between the behavioural SPIN scores and absolute amplitude of P2 in presence of high pass noise ($r = 0.90$, $p = 0.03$). For other parameters there was no significant correlation obtained.

So, major findings of the study can be summarized as follows:

- For stimulus /ba/, significant difference between typically developing children and children with Dyslexia was observed in the form of increased latency and increased amplitude of peak P1 in quiet condition. Latency of peak N1 was significantly increased for the clinical group for quiet condition as well as in presence of low pass noise. For stimulus /da/, significant increase in the latency of peak P1 and N1 as well as significant decrease in amplitude of P2 in this group when evoked in response to /da/ in quiet condition.
- Within control group significant condition effect was seen for latency and amplitude of P1 in response to stimulus /ba/. This effect was present between quiet condition and high pass noise condition as well as quiet condition and in low pass noise condition however low pass noise affected P1 more causing increase in latency and decrease in amplitude. For stimulus /da/, condition effect was seen for amplitude of P1 and N1 where low pass noise caused significant increase in P1 amplitude whereas N1 amplitude decreased significantly as compared to quiet condition.
- Within clinical group, latency of P1 and N1 was significantly prolonged in both noise conditions as compared to quiet in response to /ba/. However low pass noise caused more prolongation of latency of P1 and N1. Low pass noise also resulted in significant reduction in amplitude of N1. For stimulus /da/,

high pass noise caused a significant reduction in P1 amplitude when compared to quiet condition.

- No significant correlation was obtained between ALLR components considered for analysis in response to stimulus /ba/ and SPIN scores for typically developing children as well as for children with dyslexia.
- A significant negative correlation was obtained between SPIN scores and latency of P1 evoked in response to stimulus /da/ in presence of low pass noise for typically developing children. Also, a significant positive correlation was obtained between the SPIN scores and absolute amplitude of P2 in presence of high pass noise for the children with dyslexia.

Chapter 5

Discussion

The current study aimed to investigate whether different spectra of noise have a differential effect on components of ALLR evoked in response to speech stimuli /ba/ and /da/. Also, an attempt was made to see if there was a correlation between the behavioural SPIN scores and components of ALLR - P1, N1 and P2 evoked in quiet condition, in presence of high pass filtered noise and in presence of low pass filtered noise.

5.1.1. Effect of different spectra of noise on latency of P1, N1 and P2 evoked in response to speech stimulus /ba/ between the two groups.

Results of the current study showed that mean latencies of all the components of ALLR under investigation were prolonged in the group of children with dyslexia when compared to the group of typically developing children in quiet condition as well as in presence of high pass filtered noise and low pass filtered noise. However the difference was statistically significant only for the peaks P1 and N1. For the peak P1, latency was significantly prolonged in the children with Dyslexia as compared to their typically developing counterparts only in the quiet condition. This result is in line with the previous research finding (Byring & Jaryilhetto, 1985; Satterfield et al., 1984; Leppanen & Lyytinen, 1997). This could be because of problem in processing rapidly changing auditory stimulus and utilizing short duration cues. The mean P1 latency noted in the current study was 76.2 ms for the group of children with dyslexia which is somewhat shorter as compared to that reported in the literature i.e. 88.72 ms (Satterfield et al., 1984).

Latency of peak N1 was also significantly different across the two groups for quiet condition as well as in presence of low pass filtered noise. Previous studies have also reported prolonged latency of N1 in children having Learning Disability in quiet condition. (Arehole, 1995; Jirsa & Clontz, 1990, Leppanen & Lyytinen, 1997; Tonnquist-Uhlen, Borg, Persson and Spen,1996). The N1 peak of ALLR is known to reflect the processes preceded by sensory memory and other higher processes. So, the delay in N1 latency could indirectly reflect slower speed of sensory processing and higher level processing (Leppanen and Lyytinen, 1997). The delay in the Dyslexic group could also be due to abnormal neurological maturation as reported by Jirsa and Clontz (1990). The mean latency value reported in literature for this group ranges from 113 ms- 153 ms (Arehole, 1995; Jirsa & Clontz, 1990, Leppanen & Lyytinen, 1997; Tonnquist-Uhlen et al., 1996). *The mean latency of N1 in quiet condition was 130.6 ms for the group of children with dyslexia as found in the current study,* which falls in the range documented by previous studies. The study also showed significant difference between the two groups in terms of N1 latency in presence of low pass noise. Mean N1 latency in the group of dyslexic children was 156.2 ms whereas that for the group of typically developing children was 124.66 ms in presence of low pass filtered noise. This finding of prolonged latency in both groups could be attributed to reduced audibility of speech stimulus caused by masking due to low pass noise as mentioned by Martin and Stapells (2005) in their study done in normal hearing adults. As the latency of N1 is found to be prolonged in Dyslexic children even in quiet condition as compared to their typically developing counterparts probably due to slower speed of sensory processing (Arehole, 1995; Jirsa & Clontz, 1990, Leppanen & Lyytinen, 1997; Tonnquist-Uhlen et al., 1996), prolongation of latency in presence of low pass noise in this population could be attributed to a mixed effect of decreased

audibility and slower speed of sensory processing (Leppanen & Lyytinen, 1997). However, there have been no previous reports on the effect of any type of noise on N1 latency in this group.

Even though the mean latency of P2 in clinical group was prolonged, it was not significantly different from those exhibited by the group of typically developing children which is similar to the finding reported by Arehole (1995). This could have been so because the low pass and high pass cut off frequency of noise in the current study was out of the spectrum of speech stimulus /ba/, so perception of the stimulus was not hampered. This may indicate that P2 latency maybe less sensitive to changes in audibility.

5.1.2. Effect of differential noise on amplitude of different components of ALLR evoked in response to speech stimulus /ba/ between the two groups.

Significant difference between typically developing children and children with Dyslexia was observed for the amplitude of peak P1 in quiet condition. Increased absolute amplitude of P1 was observed in children with dyslexia as compared to typically developing children. Mean amplitude noted in the dyslexic children was 3.28 μ V whereas that in typically developing children was 1.46 μ V. This finding is in contradiction to results of previous studies which reported reduced amplitude of P1 in this population with mean amplitude ranging from -3.0 to -4.9 μ V (Pinkerton, Watson & McClelland, 1989; Leppanen & Lyytinen, 1997). This could be because of the differences in subsets of dyslexia chosen for the study. Or it could be attributed to a sampling error.

Even though absolute amplitude of N1 was reduced in all the stimulus conditions for the group of children with dyslexia when compared between the two

groups, it was not statistically significant. This finding is similar to that reported by Kumar and Gupta (2014). Other studies have reported significantly decreased N1 amplitude in children having Dyslexia (Brunswick & Rippon, 1994; Jirsa & Clontz, 1990; Radhika, 1997). This was thought to be due to reduced attention and cognitive processing of auditory stimulus in this specific population as reported by Radhika (1997) as she had asked the subjects to pay attention to the auditory stimuli and count them unlike the current study. Also the stimulus used was non speech as opposed to speech stimulus.

5.2. Effect of differential noise on latency and amplitude of different components of ALLR evoked in response to speech stimulus /ba/ within the group.

A. Typically developing children:

It was observed from the study that mean latency of P1, N1 and P2 was prolonged in both noise conditions as compared to the quiet condition. However statistically significant difference was found only for P1 latency when comparison was made between quiet condition and high pass noise as well as quiet condition and low pass noise. Low pass noise caused more prolongation of P1 latency (82.26 ms) than caused by the high pass noise (76.40 ms) when compared to quiet condition (60.66 ms). Previous research does not report of any effect of noise on P1 latency. However N1 latency delay due to presence of low pass noise has been reported by Martin and Stapells (2005). They attributed this to reduced audibility due to masking of speech stimulus /ba/ by noise which is simultaneously presented. As P1 and N1 are obligatory responses, this effect of reduced audibility could hold good for P1 as well.

Similar findings were reported in terms of absolute amplitude. P1 component of ALLR showed significant increase in absolute amplitude in presence of high pass noise as well as in presence of low pass noise when compared to quiet condition. The

increase in absolute amplitude was more in presence of low pass noise. Previous studies have reported reduction in P1 to N1 amplitude in presence of broadband noise (Cunningham, Nicol, Zecker & Kraus, 2000). However they have not reported anything about absolute amplitude of N1. Decreased P1 to N1 amplitude could be due to reduction in absolute amplitude of N1 which was also found in the current study and not necessarily due to reduction in P1 amplitude. In contrast to the findings of amplitude of P1, amplitude of N1 demonstrated significant reduction in presence of high pass and low pass noise compared to quiet condition and amplitude reduction was more prominent in presence of low pass noise. This finding correlates with previous research which has shown that, amplitude of N1 reduced as the cut of frequency of high pass noise lowered from 4000 Hz to 250 Hz and cut off frequency of low pass noise increased beyond 1000 Hz (Martin, Sigal, Kurtzberg & Stapells, 1997; Martin & Stapells, 2005). In their study, the effects of the low-pass noise were greatest beginning when the 1000- to 2000-Hz spectral region was masked as it was more of a discrimination study recorded in response to /ba/ to /da/ continuum. This is the frequency region containing the primary acoustic cues for differentiating /ba/ from /da/. However the latter study was done in normal hearing adults and not in children. For the present study low pass noise of 200 Hz cut off frequency could have resulted in more prominent reduction of amplitude due to partial masking of speech stimulus /ba/ and hence causing reduced N1 amplitude which could be due to reduced audibility.

B. Children with Dyslexia:

As observed in the control group, the mean latency of P1, N1 and P2 was prolonged in both noise conditions as compared to the quiet condition even in the group of children with Dyslexia. However the difference was statistically significant only for latency of P1 and N. In pairwise comparison all three pairs demonstrated significant difference in terms of P1 latencies but maximum prolongation was found between P1 latency in quiet condition (76.2 ms) and in presence of low pass filtered noise (92 ms). Similarly for N1 peak, significant difference for latency was found between quiet- low pass and quiet- high pass noise with prolongation being more pronounced in low pass noise. This gives an impression that low pass filtered noise affected the latency of P1 and N1 more than high pass filtered noise in this population. Martin and Stapells (2005) found similar results in their study done in adults. They reported prolongation of N1 latencies in presence of low pass noise of different cut off frequencies. In their study N1 latencies showed significant changes when the low-pass noise masker was raised to 1000 Hz. this could be attributed to decreased audibility of the speech sounds produced by low-pass noise masking which resulted in increased latencies. Thus, it can be concluded that prolonged latency of N1 was a combined effect of decreased audibility and slower speed of sensory processing (Arehole, 1995; Jirsa & Clontz, 1990, Leppanen & Lyytinen, 1997; Tonnquist-Uhlen et al., 1996) both together. As both P1 and N1 are obligatory or pre perceptual responses (Shtyrov et al., 1998; Ceponiene et al., 2005), they are affected by decreased audibility. The later peaks are mainly affected when the acoustic feature required to perceive the stimulus is masked. It maybe for this reason that a later peak like P2 was not affected.

In terms of amplitude, significant difference was obtained when N1 was compared across quiet condition and low pass noise condition. In presence of low pass noise the amplitude of N1 was significantly reduced (-1.64 μ V) as compared to in quiet condition (-2.85 μ V). This could again be attributed to reduced audibility due to partial masking of stimulus /ba/ in presence of low pass filtered noise.

5.3.1. Effect of differential noise on latency of different components of ALLR evoked in response to speech stimulus /da/ between the two groups.

Results of the current study showed that mean latencies of all the components of ALLR under investigation were prolonged in the group of children with dyslexia when compared to the group of typically developing children in quiet condition as well as in presence of high pass filtered noise. This difference in latency was statistically significant only for the peaks P1 and N1 when comparison was made in quiet condition between the two groups. Mean P1 latency in quiet condition was 66.26 ms in typically developing children whereas it was significantly prolonged (74.40 ms) for the group of children with Dyslexia. Similar finding was noted for N1 latency in quiet too with mean latencies being 121.06 ms and 133.40 ms for typically developing children and Dyslexic children respectively. Previous research has also reported prolonged latencies of P1 and N1 (Byring & Jaryilheto, 1985; Tonnquist-Uhlen et al.,1996). The finding could be due to slower speed of sensory processing in children having Dyslexia. However the finding of current study is in contradiction to the finding reported by Purdy, Kelly and Davies (2005). They found P1 and N1 had shorter latencies the LD group. One of the reasons for this difference could be the use of non-speech stimulus like 1000 Hz and 2000 Hz tones instead of speech stimulus.

5.3.2. Effect of differential noise on amplitude of different components of ALLR evoked in response to speech stimulus /da/ between the two groups.

Mean amplitudes of all the components of ALLR under investigation were reduced in the group of children with dyslexia when compared to the group of typically developing children in quiet condition as well as in presence of high pass filtered noise and low pass filtered noise. However this difference in amplitude between the groups was significant only for the peak P2 in quiet condition. This finding is in consensus with the results of previous research done in children with Learning Disability (Brunswick & Rippon, 1994; Leppanen & Lyytinen, 1997; Tonnquist-Ulhen, 1996).

5.4. Effect of differential noise on latency and amplitude of different components of ALLR evoked in response to speech stimulus /da/ within the group.

A. Typically developing children

It was observed from the study that mean latency of P1, N1 and P2 was prolonged in both noise conditions as compared to the quiet condition. However statistically significant difference was not obtained across any of the conditions in terms of latency of P1, N1 and P2. Significant difference in P1 and N1 latency was expected as seen for speech stimulus /ba/ in the present study. This could be attributed to the spectral difference between the two stimuli. More importantly difference in the rise times between the two stimuli (/ba/- 43 ms and /da/ -19 ms) which could lead to a differential neural excitation. Due to these spectral differences, probably noise did not have significant effect on P1 and N1 elicited by stimulus /da/.

In terms of amplitude significant difference was obtained for P1 and N1 across the three conditions. Pairwise comparison showed significant increase in

amplitude of P1 in presence of low pass noise (3.66 μ V) than in quiet condition (2.14 μ V). In contrast, amplitude of N1 showed significant reduction in presence of low pass noise (-2.04 μ V) as compared to quiet condition (-2.96 μ V) which is similar to the findings reported by Cunningham et al (2000). They have reported reduction in P1 to N1 amplitude in presence of broadband noise. However they have not reported anything about absolute amplitude of N1. Decreased P1 to N1 amplitude could be due to reduction in absolute amplitude of N1 and not necessarily due to reduction in P1 amplitude.

B. Children with Dyslexia:

It was observed from the study that there was a trend for mean latency of P1, N1 and P2 to be prolonged in high pass noise condition when compared to the quiet condition. Significant difference was seen only for P1 latency between quiet condition and high pass noise condition. Presence of high pass noise caused prolongation of P1 latency (82.40 ms) as compared to quiet condition (74.40 ms). Other parameters did not show significant latency differences. Similar to these findings, Billings et al., (2011) has reported prolonged latency of P1 evoked by stimulus /ba/ in presence of 4 speaker babble in adults. They attributed the findings to lowered rise time and informational masking. However this study was done in normal hearing adults.

Amplitude of P1 and N1 were diminished in presence of high pass and low pass noise as compared to quiet condition in this group whereas amplitude of P2 was more in presence of both types of noises when compared to amplitude in quiet. However this difference in amplitude was not statistically significant.

So the results reveal that in the group of children with dyslexia, latencies of ALLR components are more prolonged in all conditions for both stimuli /ba/ and /da/ possibly indicating slower speed of sensory processing. Earlier obligatory peaks are

more affected by noise indicating reduced audibility of the speech stimulus whereas later peaks are not significantly affected.

5.5. Correlation between ALLR components and SPIN scores within the group for stimulus /ba/ and /da/.

From the results of the current study there was no significant correlation found between behavioural SPIN scores parameters of ALLR considered in any of the stimulus conditions in response to speech stimulus /ba/. This was observed for typically developing children as well as for the children with Dyslexia.

For stimulus /da/, the group of typically developing children showed a significant negative correlation between behavioural SPIN scores and latency of P1 in presence of low pass noise i.e. as the SPIN scores decreased the latency of P1 increased. No significant correlation was obtained in terms of amplitude. Using a similar paradigm, Yashaswini (2013) reported no significant correlation between measures of cortical potentials for evoked by stimulus /da/ and behavioural speech in noise perception for bisyllabic words in normal hearing adults. This may indicate that P1 latency may be a sensitive measure only in children. Sharma et al. (2005) has also reported that latency of P1 can be used as an index of central auditory development in children. Cunningham et al. (2000) have reported a negative correlation between latency of N2 and tests of auditory functions like auditory processing, listening comprehension, sound patterning in children having dyslexia. However, no such correlation was found in our sample of children with dyslexia indicating a need for further investigation.

On the other hand, for the group of children with Dyslexia a significant positive correlation was found between the behavioural SPIN scores and absolute amplitude of P2 in presence of high pass noise stating that as the SPIN scores increased the amplitude of P2 also increased. This may suggest that P2 amplitude maybe sensitive to the degree of speech processing difficulty in individuals with dyslexia. Anderson, Chandrasekaran, Yi, & Kraus (2010) found a negative correlation between amplitude of N2 evoked in response to syllable /da/ in presence of speech babble as competing noise and HINT scores in typically developing children. Later peaks may hence yield information on perceptual deficits in these children.

Chapter 6

Summary and Conclusion

The aim of the present study was to know whether different spectra of noise have a differential effect on different components of Auditory long latency responses (ALLRs) in children with and without Dyslexia and to investigate if any correlation exists between measures of cortical potentials and speech perception in noise scores.

The objectives of the study were:

1. To investigate if there is a difference in the ALLRS evoked in different noise conditions between typically developing children and children with Dyslexia.
2. To see if there is a significant difference across the stimulus conditions within the group for typically developing children and children with dyslexia.
3. To investigate the correlation between different components of ALLR and behavioural SPIN scores.

10 children in the age range of 8-14 years diagnosed as having Dyslexia were chosen for the study. 15 typically developing children within the age range of 8-14 years were taken as a control group. ALLRs were recorded for natural syllables /ba/ and /da/ presented in three conditions i.e. in quiet, in presence of high pass filtered noise having cut off frequency of 4000 Hz and in presence of low pass filtered noise having cut off frequency of 200 Hz. SPIN scores were obtained from all the children at 0 dB SNR using monosyllabic words in English developed by Yathiraj, Vanaja and Muthuselvi (2009).

Results highlighted the following points:

- Latency of peak P1 and N1 for /ba/ and /da/ both, was significantly prolonged in children with dyslexia as compared to their typically developing counterparts in quiet condition. Additionally latency of N1 was significantly prolonged in response to stimulus /ba/ in presence of low pass filtered noise. This could be due to combined effect of slower speed of sensory processing in dyslexia and decreased audibility of speech signal cause by low pass filtered noise.
- In terms of absolute amplitude, P1 showed an unusual but significant increase in the group of children with dyslexia in quiet condition for stimulus /ba/ whereas P2 showed significant decrease in amplitude in this group when evoked in response to /da/ in quiet condition.
- A trend towards increase in latency with introduction of noise was observed for both /ba/ and /da/ in typically developing children as well as children with dyslexia.
- Statistically significant difference was found however, only for P1 latency prolongation for /ba/ in control group and P1 and N1 prolongation in the dyslectic group.
- Low pass noise condition was the most efficient in causing latency prolongation in both the groups. This could be attributed to reduced audibility of stimulus due to upward spread of masking.
- In the control group, introduction of noise tended to increase amplitude of P1 while it caused reduction in amplitude of P1 in the clinical group. N1 amplitude also tended to decrease in amplitude in presence of noise whereas P2 showed no specific trends.

- Statistically significant increase in amplitude was found for P1 in control group while decrease in amplitude for N1 was found in both the groups for stimulus /ba/.
- In general, no correlation was seen for ALLR elicited by /ba/ and SPIN scores in either of the two groups of participants.
- For /da/, a significant negative correlation between P1 latency in low pass noise and SPIN scores was seen in control group while a significant positive correlation between P2 amplitude for /da/ in high pass noise and SPIN scores was obtained in control group.

Thus, we can conclude from the study that low pass noise affected the latency and amplitude of earlier peaks more than the later peaks for in both the groups both /ba/ and /da/. This could be because the peaks P1 and N1 are obligatory and affected by reduced audibility of stimulus. As low pass noise causes upward spread of masking it masks the stimulus better than high pass noise and hence causes more reduction in audibility reflected by prolonged latencies and reduced amplitude. A positive correlation between SPIN scores and P2 amplitude evoked in response to /da/ in high pass noise in children with dyslexia may suggest that P2 may be sensitive to speech processing deficits in these children. A negative correlation was found between SPIN and P1 latencies for /da/ in low pass noise in typically developing children supports the possibility that P1 can be a biomarker of central auditory development in children as reported by Sharma et al. (2005).

Conclusion:

It can be concluded that one can expect prolonged ALLR latency in children with dyslexia as compared to typically developing children. We can conclude from the study that low pass noise affected the latency and amplitude of earlier peaks P1

and N1 more than the later peaks in both the groups probably due to obligatory nature of these peaks. A positive correlation between SPIN scores and P2 amplitude in high pass noise in children with dyslexia may suggest that P2 may be sensitive to speech processing deficits in these children.

However the study needs to be investigated further with a larger sample to confirm and generalize the findings to the population. Future studies could focus on using speech babble as a masker, recording with different electrode montages and subtyping of dyslexia.

Implications of the study:

- ALLRs evoked in response to speech stimuli in presence of different spectra of noise can be used as a clinical tool to study speech perception in typically developing children and clinical population.
- It could be used as an objective measure to monitor improvement in auditory performance in presence of noise post auditory training in children with dyslexia.

References

- Adlard, A., & Hazan, V. (1998). Speech perception abilities in children with developmental dyslexia. *Quarterly Journal of Experimental Psychology Section A: Human Experimental Psychology*, *51*, 153-177.
- Alain, C., Woods, D. L., & Covarrubias, D. (1997). Activation of duration-sensitive auditory cortical fields in humans. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, *104*(6), 531-539.
- American National Standards Institute, (1987), Specifications for instruments to measure aural acoustic impedance and admittance (aural acoustic immittance). *ANSI S3. 39-1987-R1996*. New York: American National Standards Institute.
- American National Standards Institute, (1991). Maximum permissible ambient noise levels for audiometric test rooms. *ANSI S3.1-1991*. New York: American National Standards Institute.
- American National Standards Institute. (2004), specification for audiometers. *ANSI S3.6-1987- R1996*. New York: American National Standards Institute.
- Anderson, S., Chandrasekaran, B., Yi, H. G., & Kraus, N. (2010). Cortical-evoked potentials reflect speech-in-noise perception in children. *European Journal of Neuroscience*, *32*(8), 1407-1413.
- American National Standards Institute (1996). Specifications for Audiometers (ANSI S3.6). Newyork.
- Arehole, S. (1995). A preliminary study of the relationship between long latency response and learning disorder. *British Journal of Audiology*, *29*(6), 295-298.
- Bamiou, D. E., Musiek, F. E., & Luxon, L. M. (2001). Aetiology and clinical presentations of auditory processing disorders—a review. *Archives of Disease in Childhood*, *85*(5), 361-365.

- Binder, J. R., Liebenthal, E., Possing, E. T., Medler, D. A., & Ward, B. D. (2004). Neural correlates of sensory and decision processes in auditory object identification. *Nature Neuroscience*, 7(3), 295-301.
- Billings, C. J., Bennett, K. O., Molis, M. R., & Leek, M. R. (2011). Cortical encoding of signals in noise: effects of stimulus type and recording paradigm. *Ear and Hearing*, 32(1), 53
- Brunswick, N., & Rippon, G. (1994). Auditory event-related potentials, dichotic listening performance and handedness as indices of lateralisation in dyslexic and normal readers. *International Journal of Psychophysiology*, 18(3), 265-275.
- Burton, M. W., Small, S. L., & Blumstein, S. E. (2000). The role of segmentation in phonological processing: an fMRI investigation. *Cognitive Neuroscience*, 12(4), 679-690.
- Byring, R., & Järvillehto, T. (1985). Auditory and visual evoked potentials of schoolboys with spelling disabilities. *Developmental Medicine & Child Neurology*, 27(2), 141-148.
- Carhart, R., & Jerger, J. J. (1959). Preferred method for clinical determination of pure-tone thresholds. *Journal of Speech and Hearing Disorders*, 24, 330-345
- Campbell, K. B., & Colrain, I. M. (2002). Event-related potential measures of the inhibition of information processing: II. The sleep onset period. *International Journal of Psychophysiology*, 46(3), 197-214.
- Čeponien, R., Rinne, T., & Näätänen, R. (2002). Maturation of cortical sound processing as indexed by event-related potentials. *Clinical Neurophysiology*, 113(6), 870-882.
- Ceponiene, R., Alku, P., Westerfield, M., Torki, M. & Townsend, J. (2005) ERPs differentiate syllable and nonphonetic sound processing in children and adults. *Psychophysiology*, 42, 391–406

- Chermak, G. D., and Musiek, F. E., 1997, Central auditory processing disorders: new perspectives. Singular, San Diego, CA. *Neurophysiology*, 113(6), 870-882.
- Choudhury, N., & Benasich, A. A. (2011). Maturation of auditory evoked potentials from 6 to 48 months: prediction to 3 and 4 year language and cognitive abilities. *Clinical Neurophysiology*, 122(2), 320-338.
- Cooper JC, Jr, Gates GA. (1991). Hearing in the elderly. The Framingham Cohort, 1983-1985: Part II . Prevalence of central auditory processing disorders . *Ear & Hearing* 12(5): 304-311 .
- Cunningham, J., Nicol, T., Zecker, S., & Kraus, N. (2000). Speech-evoked neurophysiologic responses in children with learning problems: development and behavioral correlates of perception. *Ear and Hearing*, 21(6), 554-568.
- Davis, H., Bowers, C., & Hirsh, S. K. (1968). Relations of the human vertex potential to acoustic input: Loudness and masking. *The Journal of the Acoustical Society of America*, 43(3), 431-438.
- Durlach, N. I., Mason, C. R., Kidd, G. Jr, et al. (2003). Note on informational masking
- Friedrich, M., & Friederici, A. D. (2010). Maturing brain mechanisms and developing behavioral language skills. *Brain and language*, 114(2), 66-71
- Hazan, V., Messaoud-Galusi, S., & Rosen, S. (2013). The effect of talker and intonation variability on speech perception in noise in children with dyslexia. *Journal of Speech, Language, and Hearing Research*, 56(1), 44-62
- Hillyard, S. A., & Picton, T. W. (1987). Electrophysiology of cognition. *Comprehensive Physiology*. 519-584
- Hugdahl, K., Heiervang, E., Nordby, H., Smievoll, A. I., Steinmetz, H., Stevenson, J., & Lund, A. (1998). Central auditory processing, MRI morphometry and brain laterality: applications to dyslexia. *Scandinavian Audiology*, 27(4), 26-34.

- Hygge, S., Boman, E., & Enmarker, I. (2003). The effects of road traffic noise and meaningful irrelevant speech on different memory systems. *Scandinavian Journal of Psychology, 44*(1), 13-21
- Jansson-Verkasalo, E., Ruusuvirta, T., Huotilainen, M., Alku, P., Kushnerenko, E., Suominen, K., Hallman, M. (2010). Atypical perceptual narrowing in prematurely born infants is associated with compromised language acquisition at 2 years of age. *BMC Neuroscience, 11*(1), 1.
- Jasper, H. H. (1958). The ten twenty electrode system of the international federation. *Electroencephalography & Clinical Neurophysiology, 10*, 371-375.
- Jeste, S. S., & Nelson III, C. A. (2009). Event related potentials in the understanding of autism spectrum disorders: an analytical review. *Journal of Autism and Developmental Disorders, 39*(3), 495-510.
- Jirsa, R. E., & Clontz, K. B. (1990). Long latency auditory event-related potentials from children with auditory processing disorders. *Ear and Hearing, 11*(3), 222-232.
- Kaplan-Neeman, R., Kishon-Rabin, L., Henkin, Y., & Muchnik, C. (2006). Identification of syllables in noise: electrophysiological and behavioral correlates. *The Journal of the Acoustical Society of America, 120*(2), 926–933.
- King, W. M., Lombardino, L. J., Crandell, C. C., & Leonard, C. M. (2003). Comorbid auditory processing disorder in developmental dyslexia. *Ear and Hearing, 24*(5), 448-456.
- Kumar, P., Gupta, R. (2014). Cortical Processing of Speech in Children with Dyslexia *International Journal of Health Sciences and Research, 4* (10), 221-228
- Kuruvilla-mathew, A., Purdy, S. C., & Welch, D. (2015). Cortical encoding of speech acoustics : *Effects of noise and amplifi cation*, 1–13.
- Leppänen, P. H. T., & Lyytinen, H. (1997). Auditory event-related potentials in the study of developmental language-related disorders. *Audiology and Neurotology, 2*(5), 308-340.

- Loomba, M. (1995). Descriptive analysis of the sequential progression of English reading skills among Indian children. *An Unpublished Master's dissertation*, University of Mysore, Mysore.
- Manuscript, A., & Level, A. S. (2010). *NIH Public Access*, 254, 15–24.
- Martin, B. A., & Boothroyd, A. (1999). Cortical, auditory, event-related potentials in response to periodic and aperiodic stimuli with the same spectral envelope. *Ear and hearing*, 20(1), 33-44.
- Martin, B. A., Sigal, A., Kurtzberg, D., & Stapells, D. R. (1997). The effects of decreased audibility produced by high-pass noise masking on cortical event-related potentials to speech sounds /ba/ and /da. *The Journal of the Acoustical Society of America*, 101(3), 1585-1599.
- Martin, B. a, & Stapells, D. R. (2005). Effects of low-pass noise masking on auditory event-related potentials to speech. *Ear and Hearing*, 26(2), 195–213.
- Mason, S. M., & Mellor, D. H. (1984). Brain-stem, middle latency and late cortical evoked potentials in children with speech and language disorders. *Electroencephalography and Clinical Neurophysiology/ Evoked Potentials Section*, 59(4), 297-309.
- McIsaac, H., & Polich, J. (1992). Comparison of infant and adult P300 from auditory stimuli. *Journal of Experimental Child Psychology*, 53(2), 115-128.
- McAnally, K. I., & Stein, J. F. (1997). Scalp potentials evoked by amplitude-modulated tones in dyslexia. *Journal of Speech, Language, and Hearing Research*, 40(4), 939-945.
- McPherson, D.L., & Starr,A. (1993). Auditory Evoked Potentials in the clinic. In Halliday, A.M. (Ed), *Evoked Potentials in Clinical Testing*. *Edinburgh*.
- McPherson, D. L., & Starr, A. (1993). Binaural interaction in auditory evoked potentials: brainstem, middle-and long-latency components. *Hearing Research*, 66(1), 91-98.

- Nagarajan, S., Mahncke, H., Salz, T., Tallal, P., Roberts, T., & Merzenich, M. M. (1999). Cortical auditory signal processing in poor readers. *Proceedings of the National Academy of Sciences*, *96*(11), 6483-6488.
- Näätänen, R. and Picton, T. (1987), The N1 Wave of the Human Electric and Magnetic Response to Sound: *A Review and an Analysis of the Component Structure*.
- Näätänen, R., Kujala, T., Escera, C., Baldeweg, T., Kreegipuu, K., Carlson, S., & Ponton, C. (2012). The mismatch negativity (MMN)—a unique window to disturbed central auditory processing in ageing and different clinical conditions. *Clinical Neurophysiology*, *123*(3), 424-458.
- Novak, G. P., Kurtzberg, D., Kreuzer, J. A., & Vaughan, H. G. Jr. (1989). Cortical responses to speech sounds and their formants in normal infants: maturational sequence and spatiotemporal analysis. *Electroencephalography and Clinical Neurophysiology* *73*(4), 295-305
- Niemitalo-Haapola, E., Haapala, S., Jansson-Verkasalo, E., & Kujala, T. (2015). Background noise degrades central auditory processing in toddlers. *Ear and Hearing*, *36*(6), e342-e351.
- Onishi, S., & Davis, H. (1968). Effects of duration and rise time of tone bursts on evoked V potentials. *The Journal of the Acoustical Society of America*, *44*(2), 582-591.
- Partanen, E., Kujala, T., Tervaniemi, M., & Huotilainen, M. (2013). Prenatal music exposure induces long-term neural effects. *PloS one*, *8*(10), e78946.
- Pinkerton, F., Watson, D. R., & McClelland, R. J. (1989). A neurophysiological study of children with reading, writing and spelling difficulties. *Developmental Medicine & Child Neurology*, *31*(5), 569-581.
- Ponton, C. W., Don, M., Eggermont, J. J., Waring, M. D., & Masuda, A. (1996). Maturation of human cortical auditory function: differences between normal-hearing children and children with cochlear implants. *Ear and hearing*, *17*(5), 430-437.

- Ponton, C. W., Eggermont, J. J., Kwong, B., & Don, M. (2000). Maturation of human central auditory system activity: evidence from multi-channel evoked potentials. *Clinical Neurophysiology*, *111*(2), 220-236.
- Ptok, M., Blachnik, P., & Schönweiler, R. (2004). [Late auditory potentials (NC-ERP) in children with symptoms of auditory processing and perception disorder. With and without attention deficit disorder]. *HNO*, *52*(1), 67-75.
- Putter-Katz, H., Kishon-Rabin, L., Sachartov, E., Shabtai, E. L., Sadeh, M., Weiz, R., & Pratt, H. (2005). Cortical activity of children with dyslexia during natural speech processing: evidence of auditory processing deficiency. *Journal of Basic and Clinical Physiology and Pharmacology*, *16*(2-3), 157-172.
- Putter-Katz, H., Banai, K., & Ahissar, M. (2005). Speech perception in noise among learning disabled teenagers. In *Plasticity and Signal Representation in the Auditory System* (pp. 251-257). Springer US.
- Purdy, S., Katsch, R., Storey, L., Sharma, M., Dillon, H., & Ching, T. (2001). Electrophysiological evaluation of hearing aids in infants. *National Acoustic Laboratories. Annual report*.
- Purdy, Suzzane C., Kelly, Davies, Merren G. (2002). Auditory Brainstem Response, Middle Latency Response, and Late Cortical Evoked Potentials in Children with Learning Disabilities. *Journal of American Academy of Audiology*, *13*(7), 367-382.
- Radhika, A. (1998). Auditory late latency potentials in learning disabled children. *Unpublished Master's Dissertation, University of Mysore, Mysore*.
- Ramaa, S. (2000). Two decades of research on learning disabilities in India. *Dyslexia*, *6*(4), 268-283.
- Ramus, F. (2003). Developmental dyslexia: specific phonological deficit or general sensorimotor dysfunction?. *Current Opinion in Neurobiology*, *13*(2), 212-218.

- Rohith, M. (2010). Cortical Auditory Evoked Potentials in Children Using Speech and Non Speech Stimuli. *Unpublished Master's Dissertation, University of Mysore, Mysore.*
- Rosen, S., & Manganari, E. (2001). Is there a relationship between speech and nonspeech auditory processing in children with dyslexia?. *Journal of Speech, Language, and Hearing Research, 44*(4), 720-736.
- Rothman, H. H., Davis, H., & Hay, I. S. (1970). Slow evoked cortical potentials and temporal features of stimulation. *Electroencephalography and Clinical Neurophysiology, 29*(3), 225-232.
- Rout, A. (1996). Perception of monosyllabic words in Indian children. *Master's Dissertation, University of Mysore, Mysore.*
- Satterfield, J. H., Schell, A. M., Backs, R. W., & Hidaka, K. C. (1984). A cross-sectional and longitudinal study of age effects of electrophysiological measures in hyperactive and normal children. *Biological Psychiatry.*
- Snowling, M. J. (2000). *Dyslexia*. Blackwell publishing. Malden, 461 pp
- Söderlund, G. B., Sikström, S., Loftesnes, J. M., & Sonuga-Barke, E. J. (2010). The effects of background white noise on memory performance in inattentive school children. *Behavioral and Brain Functions, 6*(1), 1.
- Sharma, A., Kraus, N., McGee, T. J., & Nicol, T. G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section, 104*(6), 540-545.
- Sharma, A., & Dorman, M. F. (1999). Cortical auditory evoked potential correlates of categorical perception of voice-onset time. *The Journal of the Acoustical Society of America, 106*(2), 1078-1083.
- Sharma, A., Marsh, C. M., & Dorman, M. F. (2000). Relationship between N1 evoked potential morphology and the perception of voicing. *The Journal of the Acoustical Society of America, 108*(6), 3030-3035.

- Sharma, A., Dorman, M. F., & Spahr, A. J. (2002). A sensitive period for the development of the central auditory system in children with cochlear implants: implications for age of implantation. *Ear and hearing, 23*(6), 532-539.
- Sharma, A., Martin, K., Roland, P., Bauer, P., Sweeney, M. H., Gilley, P., & Dorman, M. (2005). P1 latency as a biomarker for central auditory development in children with hearing impairment. *Journal of the American Academy of Audiology, 16*(8), 564-573.
- Shtyrov, Y., Kujala, T., Ahveninen, J., Tervaniemi, M., Alku, P., Ilmoniemi, R. J., & Näätänen, R. (1998). Background acoustic noise and the hemispheric lateralization of speech processing in the human brain: magnetic mismatch negativity study. *Neuroscience Letters, 251*(2), 141-144.
- Summers, V., & Leek, M. R. (1998). F0 processing and the separation of competing speech signals by listeners with normal hearing and with hearing loss. *Journal of Speech, Language, and Hearing Research, 41*(6), 1294-1306.
- Sussman, E., Steinschneider, M., Gumenyuk, V., Grushko, J., & Lawson, K. (2008). The maturation of human evoked brain potentials to sounds presented at different stimulus rates. *Hearing Research, 236*(1), 61-79.
- Stuart, A., Givens, G. D., Walker, L. J., & Elangovan, S. (2006). Auditory temporal resolution in normal-hearing preschool children revealed by word recognition in continuous and interrupted noise. *The Journal of the Acoustical Society of America, 119*(4), 1946-1949.
- Tampas, J. W., & Harkrider, A. W. (2006). Auditory evoked potentials in females with high and low acceptance of background noise when listening to speech. *The Journal of the Acoustical Society of America, 119*(3), 1548-1561.
- Tonnquist-Uhlen, I. (1996). Topography of auditory evoked long-latency potentials in children with severe language impairment: the P2 and N2 components. *Ear and hearing, 17*(4), 314-326.

- Tremblay, K. L., Billings, C., & Rohila, N. (2004). Speech evoked cortical potentials: effects of age and stimulus presentation rate. *Journal of the American Academy of Audiology*, *15*(3), 226-237
- Tremblay, K. L., Piskosz, M., & Souza, P.(2003). Effects of age and age related hearing loss on the neural representation of speech cues. *Clinical Neurophysiology*, *114*(7), 1332-1343.
- Vance, M., & Martindale, N. (2012). Assessing speech perception in children with language difficulties: Effects of background noise and phonetic contrast. *International Journal of Speech-Language Pathology*, *14*(1), 48-58.
- Vesco, K. K., Bone, R. C., Ryan, J. C., & Polich, J. (1993). P300 in young and elderly subjects: auditory frequency and intensity effects. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, *88*(4), 302-308.
- Warrier, C. M., Johnson, K. L., Hayes, E., Nicol, T., & Kraus, N. (2004). Learning impaired children exhibit timing deficits and training-related improvements in auditory cortical responses to speech in noise. *Experimental Brain Research. Experimentelle Hirnforschung. Experimentation Cerebrale*, *157*(4), 431-441.
- Weitzman, E.D. & Graziani, L.J.(1968). Maturation and topography of the auditory evoked response of the prematurely born infant. *Developmental Psychobiology*, *1*(2), 79-89.
- Whiting, K. A, Martin, B. A., & Stapells, D. R. (1998). The effects of broadband noise masking on cortical event-related potentials to speech sounds /ba/ and /da/. *Ear and Hearing*, *19*(3), 218-231.
- Wilson, R. H., Farmer, N. M., Gandhi, A., Shelburne, E., & Weaver, J. (2010). Normative data for the Words-in-Noise Test for 6-to 12-year-old children. *Journal of Speech, Language, and Hearing Research*, *53*(5), 1111-1121.
- Yashaswini, L. (2013).Cortical Encoding of Speech in Noise Older Adults. *Unpublished Master's Dissertation, University of Mysore, Mysore.*

- Yathiraj, A., & Mascarenhas, K. (2004). Audiological profile of children with suspected auditory processing disorder. *Journal of Indian Speech and Hearing Association*, 19, 5-14.57
- Yathiraj, A., Vanaja, C.S. & Muthuselvi. T. (2009). Maturation of Auditory Processes in Children aged 6 to 10 years. AIISH Research Fund project, *All India Institute of Speech and Hearing*.
- Yathiraj, A., & Maggu, A. R. (2013). Comparison of a screening test and screening checklist for auditory processing disorders. *International Journal of Pediatric Otorhinolaryngology*, 77(6), 990-995.
- Ziegler, J. C., Pech-Georgel, C., George, F., & Lorenzi, C. (2009). Speech-perception-in-noise deficits in dyslexia. *Developmental Science*, 12(5), 732-745.