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Cortical Evoked Potentials in Children with Learning Disorder

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Abstract
The purpose of the study was to investigate MLR and LLR response from children with LD. Auditory cortical potential were recorded in 30 subjects with LD (experimental group) and 30 age-gender matched children (control group) aged 10 -14 years with means age 11.2years. The responses were recorded using the click stimulus. MLR (Pa, Na) & P1, N1, P2, N2 latency and amplitude were analyzed. Result of study indicates that both the group had statistically significant difference in latency. Pa, Na , P1 and N1 amplitude had significant different between two test group. The present study finding reports that click evoked auditory late latency response is traceable in all children with LD and typically developing children. However, children with LD exhibited prolonged latency with reduced amplitude with inconsistent wave morphology responses. The present study, inferred that the abnormalities in processing from the thalamus, auditory cortical area results in altered auditory cortical recording. However further, research is required to use MLR, Long latency response as a tool that clinically differentiates between individuals with and without LD.
Key Words: *Evoked potential, LLR, MLR, Normal Hearing, Learning disorder.*

Introduction

Learning disorders is characterized by difficulty in Reading, Mathematics, written expression and learning disorder not otherwise specified (NOS) (American Psychiatric Association, 1994). Children with learning disorders subjects are characterized by impairment in a particular or several areas of brain functioning. Children with Learning Disability (CWLD) show distinct gap between a person's level of expected achievement and their performance usually attributed to their

inattentiveness and laziness. In the eastern countries, earlier LD was misconception that is seen only in English speaking children (i.e. due to phonological awareness skills). Incidence of LD reported in India is low could be due to relative lack of concern, awareness and sensitivity about LD among educators. By considering Indian population most of the classroom are overcrowded i.e. 60- 70 (Karanth, 2002). Epidemiology and Biostatistics, National Institute of Mental Health and Neuro-sciences, Bangalore (2006) reported that prevalence rate of LD in 4-16 year old children in urban middle class, slum and rural areas was 12%. The prevalence of LD in Mumbai city reported by the L.T.M.G. Hospital, Sion (2006), that of the total number of 2,225 children visiting the hospital for certification of any kind of disability, out of that 640 were diagnosed as LD. For LD certification required at the Lokamanya Tilak M.G. Hospital, Sion, Mumbai, neurological assessment, vision and hearing tests, analysis of school progress report, I.Q. test, educational assessment, psychiatric assessment were required. With growing audiological field, Auditory evoked potential plays significant role in hearing assessment, as AEP are reliably recorded from different sites such as brain stem , auditory cortex etc. Hall (2006); Hood (1998), Jacobson (1985); McPherson et al (1996).

The Middle Latency Auditory Evoked Potentials (MLAEP) is a series of waveform which is observed in 10 to 80 millisecond interval following an auditory stimulus. The MLAEP site of generation is still an area under research but it appears to have multiple generators, with greater participation of thalamic-cortical pathways and a lesser contribution from the inferior colliculus and the reticular formation (midbrain) Hall (2006). MLAEP indicates functioning of cortical activity involved in primary auditory abilities (recognition, discrimination and figure-background) and non-primary auditory abilities (selective attention, auditory sequence and auditory/visual integration) Hall (2006). MALP peaks are denoted as Po, Na, Pa, Nb. various research studies reported that Na and Pa component have higher amplitude than other components. Hence they are widely used to identify auditory disorders and for the behavioral auditory threshold estimation in children and adults.

LLR (P_1, N_1, P_2, N_2, P_3):

The late evoked potentials are complex signals of the neural processing in the auditory cortex therefore, called as cortical potential. Hall (2006); Hood (1998), Jacobson (1985); Edgemont (1999). LLR waveforms are the cortical responses that occur within 50 – 300 ms after the acoustic stimulus is presented to the ears. The peak potentials in the wave forms are denoted as N_1, P_2, N_2 and P_3 [Hall (2006); Hood (1998); Jacobson (1985); Eggermont (1999) McPherson (1996)]. These peaks generations sites are in the auditory cortex mainly from structures of the thalamocortical and cortico-cortical auditory pathways, primary auditory cortex and associated cortical areas [Picton et al (2000); Kraus et al. (1993); Näätänen(1994); Sharma et al(2009); Picton et al (2002); Vaughan et al(1970); Hall(

2006);Hood(1998),Jacobson(1985)]. LLR Peaks also reflect the auditory neural activity even of the dendrites are involved in the skills of attention, discrimination, memory, integration and decision making. The amplitude and latency changes in the P1 N1 P2 N2 wave form indicates that response is being presented structurally and physiologically in the auditory cortex (Picton 2006). Various research studies supports that it is possible to capture the MLR & LLAEP reliably, even in young children [Purdy et al (2002); Sharma et al (2009)]. In Recent years auditory cortical potential has provided unique dynamic spatiotemporal scope to study brain processes underlying auditory processing and perception (Rugg and Coles 1995; Steinschneider et el. 1992). Due to clear representation of P1 N1 P2 it has been investigated for analyzing various groups of neurological dysfunction such as language disorders, auditory processing disorders and auditory neuropathy. Martin et al 2007; cone Wesson and wundberlich 2003; Leppanen and Lyyinen 1997; Picton 1992; McEvoy et al., 1990; Picton 1991; Nerville and Bavelier 2002; Steinschneider et. al. 1992). Previous research finding in children with LD shows auditory perceptual dysfunction that affects the ability to learn to use phonics skills adequately (Tallai 1980). Reed et. al. (1989) reported that auditory perceptual deficits were found in children with LD, which interferes with the processing of phonological information. John et al (1981) found LD children have difference in the pattern identification and discrimination than control and significant relationship was obtained between reading level and speech discrimination. Marc. et. al. 2000 research finding indicate that LD children and Language impairment group showed clear speech perception deficits.

Reviews of literature indicate that children with LD may also have central auditory dysfunction. Therefore to get a clearer picture present research study has been taken up. As MRL & LLR test provide most suitable information about thelemo-cortical area of cortical auditory system functioning therefore the present research study has been conducted on children with LD. This research will help us to better understand similarities and difference in central neuro-auditory functioning in individuals with LD and control subjects.

Aim and Objectives: To compare similarities and differences in auditory cortex functioning between age gender matched children with learning disability & normal children.

Method:

Subjects: 30 subjects enrolled in the study that had LD certification from LD certification board Maharashtra. 30 age- gender match subject were taken as control group. Subjects participated in the present study were in age range of 10 to 14 years with mean age of 11.8 years, with hearing sensitivity of < 25dBHL on pure tone across audiometric octave band frequencies. All subjects had 'A' type tympanogarm with presence of reflexes at normal sensation levels. All the subjects were screened with TEOAE and ABR for any underlying auditory synchrony/ neuropathy.

Instrumentation: The Interacoustic AC 40 dual channel clinical audiometer (Version 2) was used for pure tone testing and speech audiometry. The GSI Tympstar middle ear analyzer was used for tympanometry and acoustic reflex measurement and recording. GSI Audio Screener was used to screen with TEOAE and AABR. The study was conducted on IHS Smart EP version 3.56. It was ensured that all the equipments were in calibrated condition. (ANSI S 3.6- 2003)

Materials: click was used which provided by the manufacturer IHS to record the AEPs.

Test Procedure: On the day of tests, subjects were evaluated using the tools noted above, and otoscopy performed on all subjects to ensure that no visible external or middle ear abnormalities were present on the day of the test. Pure tone thresholds were acquired from 250 to 8000 Hz via air conduction, and when clinically appropriate, bone conduction thresholds were also acquired from 250 to 4000 Hz, using modified Hughson and Westlake procedure. As indicated above, tympanometry and acoustic reflexes were recorded to rule out middle ear pathology. Tympanometry test was carried out using 226 Hz probe tone at 85 dBSPL , reflexometry, acoustic reflex test were done at tone of 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz ipsilaterally and contralaterally TEOAE was also conducted to rule out for any underlying auditory synchrony/ neuropathy. Transient evoked otoacoustic emissions (TEOAE) were measured using click stimuli at 85 dBSPL in both ear. All the testing was performed in recommended test environment and with standardized test protocol. Subjects were seated in a reclining chair in an electrically shielded and acoustically treated room. Silver chloride electrodes (AgCl) were placed at the recording sites, after cleaning those sites with an abrasive gel. Electroencephalography (EEG) paste and surgical adhesive tape was used to hold the electrodes firmly in place. In essence, standard and well accepted evoked potential protocols were used throughout all evoked potential acquisitions.

For the MLR, LLR measurements, the electrodes were inserted for recording of auditory evoked potentials occurring on channel A and the recording of eye movements and blinking on the B channel . On channel A, the active electrode was placed at Cz connected to the input (+) of the pre-amplifier, and the reference electrode placed on the mastoid of the stimulated ear and connected to the input (-). The ground electrode was placed on Fpz connected to the ground position in junction box. [Kraus et al (1993); Sharma et al (2009)].On channel B, the active electrode was placed on the supraorbital position contralateral to the ear stimulated connected to the input (+) of the pre-amplifier and the reference electrode on the infraorbital position on the same side connected to the (-) input. With this arrangement of electrodes, we sought to establish the amplitude of the eye movement and eye blink were recorded from channel B. Also to minimize the artifacts from channel A, the recorded channel B were subtracted data to overcome the eye blink artifact. With this procedure, the interference of the eye movement artifacts were minimized.

Analysis of evoked potentials: Having identified the auditory evoked potential , amplitude was established as the difference between the 0.0 uV point and the maximum positive value , in this case the P1 and P2 components , and the negative value, specifically for N1 component are measured in uV . P1 N1 P2 accounts the maximum amplitude points. Testing was done in an acoustically and electrically treated room & subjects were seated comfortably in a reclining seat. P1 was marked as the relative positivity occurring within the range of approximately 50 to 100 msec. N1 was marked the earlier negativity between 110 to 160 msec seen in all the subjects. Further waveform printouts were given to two examiners to mark the potentials both examiners had clinically experiences more than 5 years in the field of evoked potential measurement.

Following protocol were used for MLR ,LLR [Hall (2006); Hood (1998)]

Table 1. Showing Test protocol for MLR & LLR

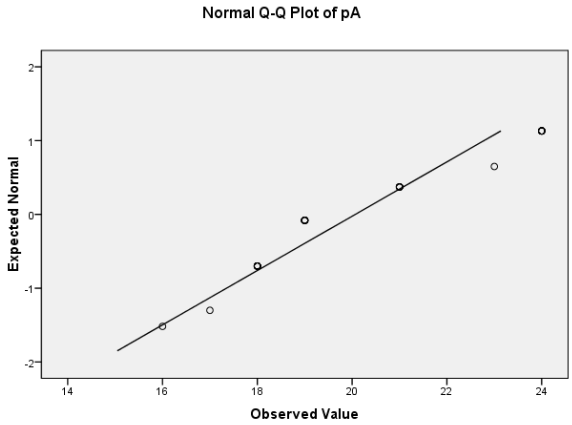
Stimulus	MLR Click	LLR
Rate	7.1	1.1
Polarity	Alternate	Alternate
Transducer	Insert earphone	Insert earphone
Intensity	70 dB nHL	70dB nHL
Filters	10-1500Hz	1-30Hz
Amplification	100K	100K
Runs	2	2
Analysis window	Overall 100ms	Overall 500ms
Sweeps	1000	250

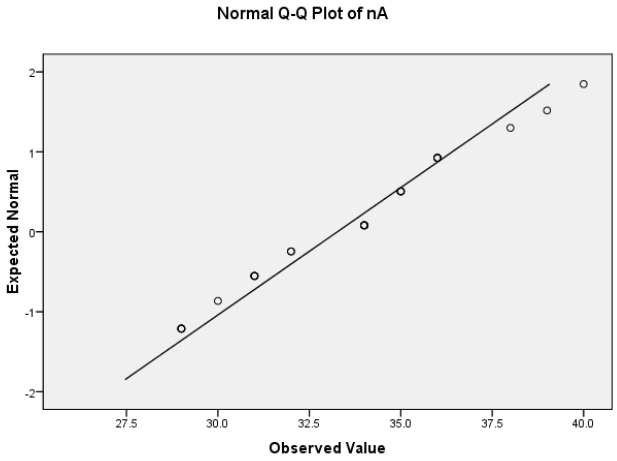
Result and Discussion

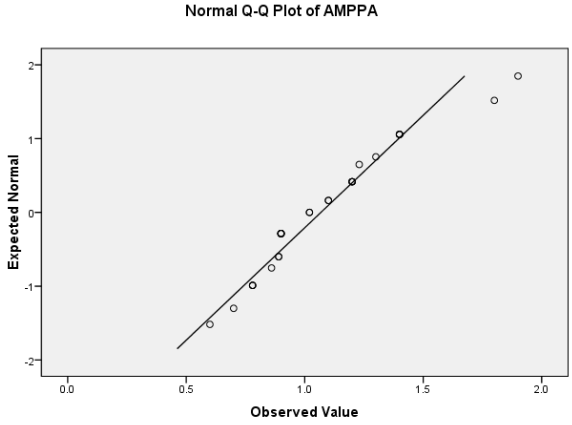
Statistical Analysis: Descriptive statistical analysis of the scores in terms of mean, standard deviation and parametric tests using independent 't' tail test was performed using Statistical package Social Science (SPSS 16.0) software for different parameters of evoked AMLR, ALLR. The results obtained are presented and discussed in the subsequent section.

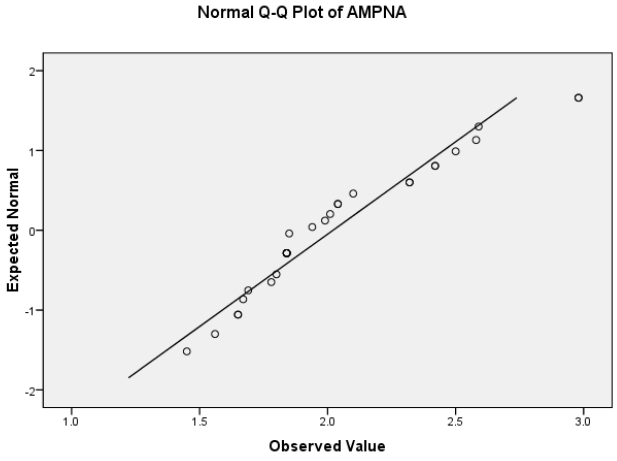
Table 2 Showing result of Kolmogorov-Smirnov & Shapiro-Wilk test (MLR data)

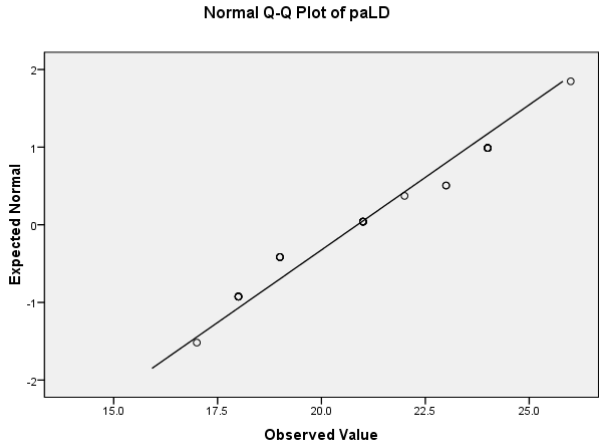
Tests of Normality						
	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	Df	Sig.	Statistic	df	Sig.
Pa	.219	30	.201	.886	30	.104
Na	.159	30	.501	.955	30	.223
Amplitude PA	.163	30	.052	.955	30	.226
Amplitude NA	.154	30	.068	.951	30	.184
Na in LD	.135	30	.168	.941	30	.195
Pa Amplitude LD	.190	30	.107	.933	30	.057
Na Amplitude LD	.100	30	.200*	.961	30	.328
Pa in LD	.191	30	.107	.920	30	.127

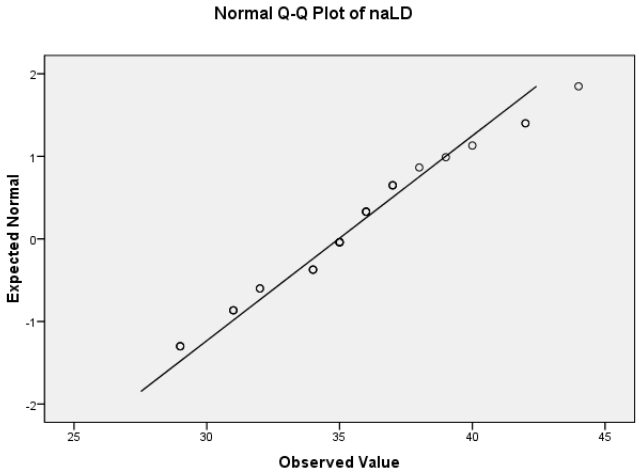

Normal Q-Q Plot of pA
Expected Normal
Observed Value
14
16
18
20
22
24
2
1
0
-1
-2

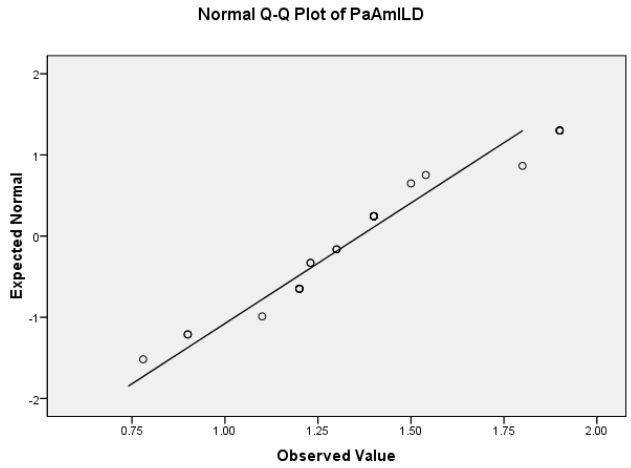

Normal Q-Q Plot of nA
Expected Normal
Observed Value
27.5
30.0
32.5
35.0
37.5
40.0
2
1
0
-1
-2

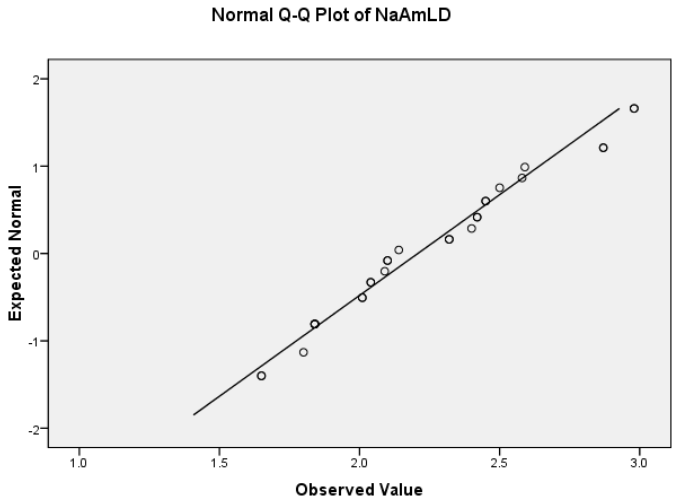

Normal Q-Q Plot of AMPPA
Expected Normal
Observed Value
0.0
0.5
1.0
1.5
2.0
2
1
0
-1
-2


Normal Q-Q Plot of AMPNA
Expected Normal
Observed Value
1.0
1.5
2.0
2.5
3.0
2
1
0
-1
-2


Normal Q-Q Plot of paLD
Expected Normal
Observed Value
15.0
17.5
20.0
22.5
25.0
2
1
0
-1
-2


Normal Q-Q Plot of naLD
Expected Normal
Observed Value
25
30
35
40
45
2
1
0
-1
-2





Descriptive statistics presented data in Table, Q plots ,Box Plots Indicate that mean for LD group evoked potential and control group evoked potential which are within normal distribution. The difference of means between the two groups is quite big in the context of their standard deviation & positive skewness in the distribution for both group. The Kolmogorov – Smirnov Z value are not statically significant ($p>0.05$). Thus the small skewness in the two distributions is not major concern and the two distributions met the assumption of normality therefore further analysis was done by using parametric test.

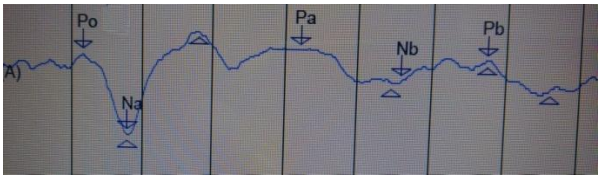


Figure 1 Normal subject MLR waveform

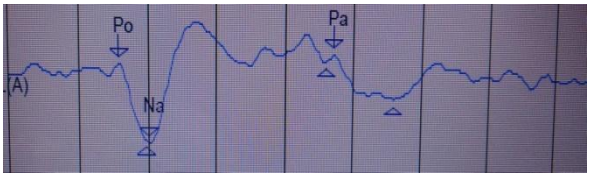


Figure 2 prolonged subject LD MLR waveform

Table 3: Descriptive table of MLR means value of Pa, Na , Amplitude Pa , Amplitude Na

Group Statistics					
	group	N	Mean	Std. Deviation	Std. Error Mean
Pa	normal	30	20.0667	2.71564	.49581
	LD	30	22.4667	4.62924	.84518
Na	normal	30	33.2667	3.13966	.57322
	LD	30	36.4333	4.96667	.90679
Amplitude PA	normal	30	1.0683	.32814	.05991
	LD	30	0.4627	.43763	.07990
Amplitude NA	normal	30	2.0210	.43198	.07887
	LD	30	1.6823	.50447	.09210

Table 4: Showing Two independent sample't' tail test results of MLR in LD and Control subjects.

	t-test for Equality of Means						
	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
						Lower	Upper
Pa	-2.449	58	.017	-2.40000	.97987	-4.36143	-.43857
Na	-2.952	58	.005	-3.16667	1.07277	-5.31406	-1.01928
Amplitude PA	-3.949	58	.000	-.39433	.09987	-.59424	-.19443
Amplitude NA	-2.980	58	.004	-.36133	.12126	-.60406	-.11861

After comparing means it can be seen that both the subject had statistically significant difference between them at significance level 0.05.

LLR test

Table 5 Showing result of Kolmogorov-Smirnov & Shapiro-Wilk test (LLR data)

LLR data were subjected for Tests of Normality							
Peaks	Group	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
		Statistic	Df	Sig.	Statistic	Df	Sig.
p1	LD	.077	30	.200*	.985	30	.658
	NORMAL	.072	30	.200*	.982	30	.496
n1	LD	.142	30	.064	.932	30	.202
	NORMAL	.123	30	.025	.958	30	.339
p2	LD	.100	30	.200*	.973	30	.207
	NORMAL	.202	30	.24	.917	30	.201
n2	LD	.198	30	.100	.903	30	.200
	NORMAL	.107	30	.083	.972	30	.176
p3	LD	.142	30	.104	.924	30	.201
	NORMAL	.104	30	.171	.963	30	.068
n3	LD	.203	30	.100	.799	30	.200
	NORMAL	.115	30	.147	.972	30	.174
Amplitude p1	LD	.117	30	.140	.965	30	.080
	NORMAL	.119	30	.133	.962	30	.057
Amplitude n1	LD	.113	30	.155	.975	30	.246
	NORMAL	.123	30	.125	.945	30	.009
Amplitude p2	LD	.112	30	.058	.916	30	.501
	NORMAL	.112	30	.159	.933	30	.003
Amplitude n2	LD	.102	30	.187	.977	30	.312
	NORMAL	.164	30	.0510	.958	30	.039
Amplitude p3	LD	.120	30	.050	.959	30	.042
	NORMAL	.129	30	.315	.934	30	.203
Amplitude n3	LD	.063	30	.200*	.990	30	.916
	NORMAL	.092	30	.200*	.979	30	.403

LLR data were subjected for Tests of Normality							
Peaks	Group	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
		Statistic	Df	Sig.	Statistic	Df	Sig.
p1	LD	.077	30	.200*	.985	30	.658
	NORMAL	.072	30	.200*	.982	30	.496
n1	LD	.142	30	.064	.932	30	.202
	NORMAL	.123	30	.025	.958	30	.339
p2	LD	.100	30	.200*	.973	30	.207
	NORMAL	.202	30	.24	.917	30	.201
n2	LD	.198	30	.100	.903	30	.200
	NORMAL	.107	30	.083	.972	30	.176
p3	LD	.142	30	.104	.924	30	.201
	NORMAL	.104	30	.171	.963	30	.068
n3	LD	.203	30	.100	.799	30	.200
	NORMAL	.115	30	.147	.972	30	.174
Amplitude p1	LD	.117	30	.140	.965	30	.080
	NORMAL	.119	30	.133	.962	30	.057
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	NORMAL	.164	30	.0510	.958	30	.039
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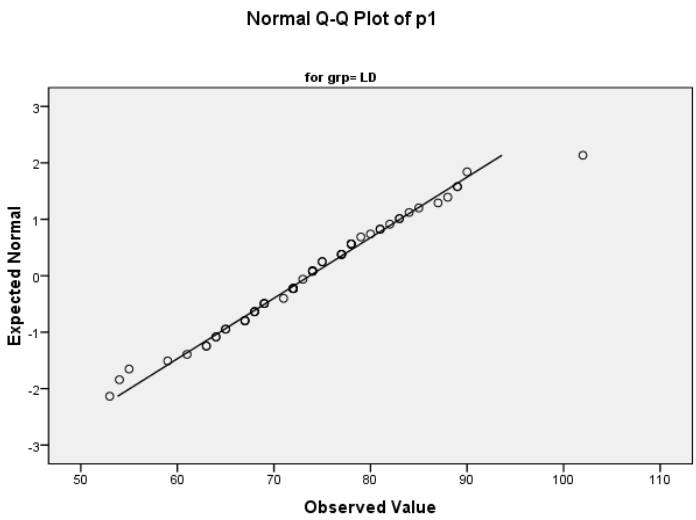
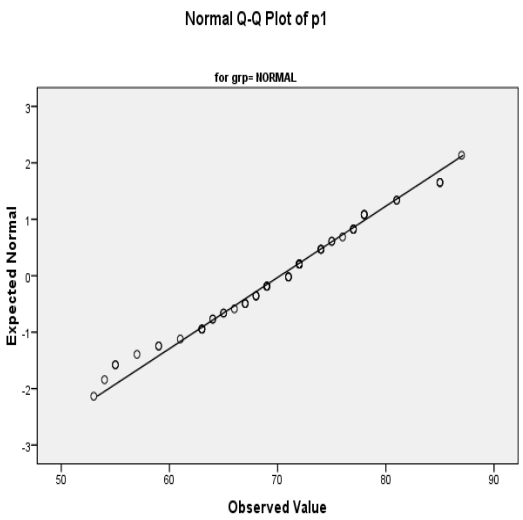
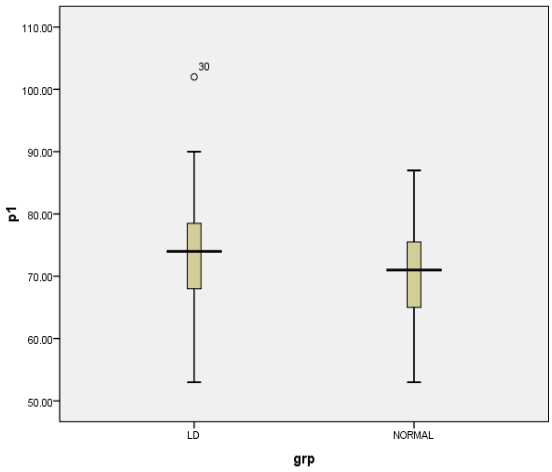
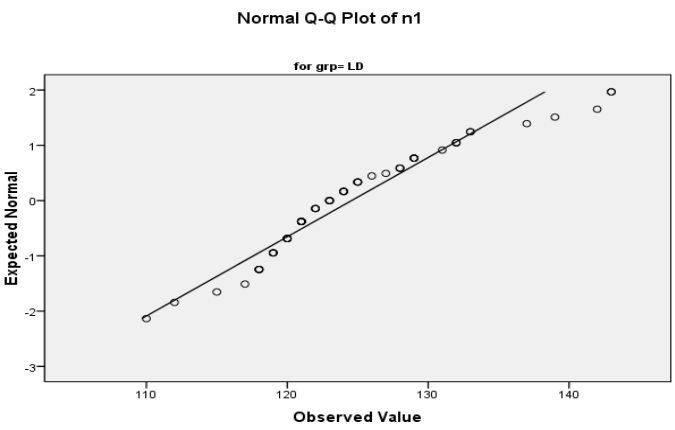
Amplitude n3	LD	.063	30	.200*	.990	30	.916
	NORMAL	.092	30	.200*	.979	30	.403

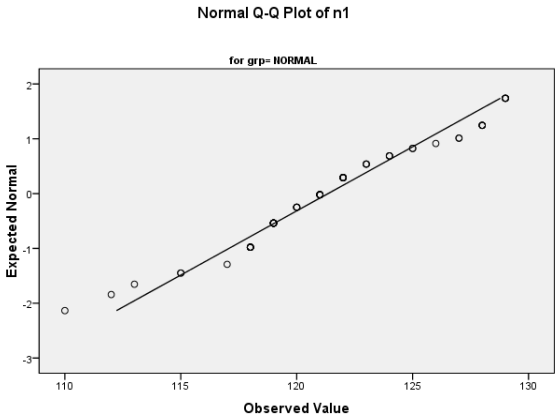
a. Lilliefors Significance Correction
*. This is a lower bound of the true significance.

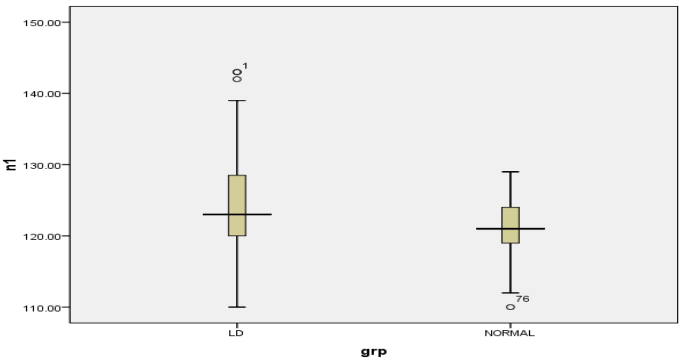

Normal Q-Q Plot of p1
for grp= LD
Expected Normal
Observed Value

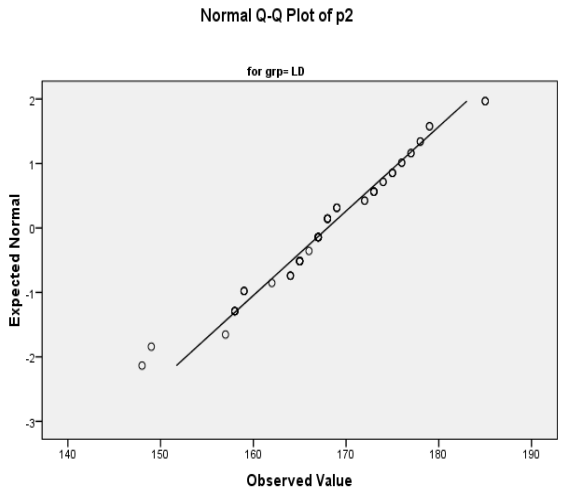

Normal Q-Q Plot of p1
for grp= NORMAL
Expected Normal
Observed Value

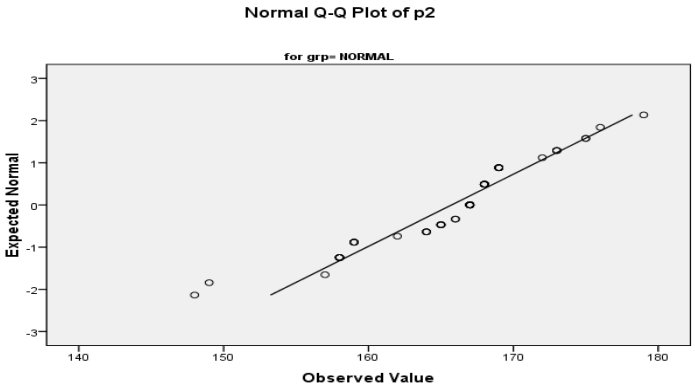

p1
grp
LD
NORMAL

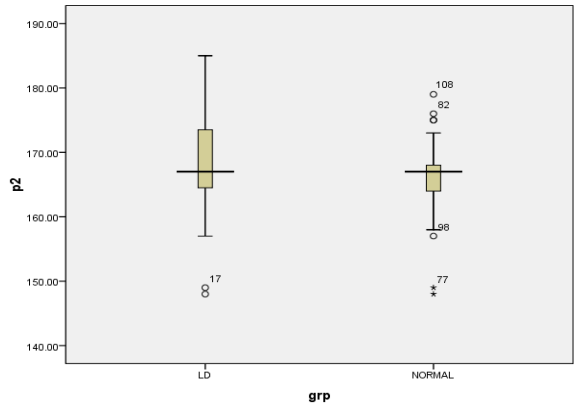

Normal Q-Q Plot of n1
for grp= LD
Expected Normal
Observed Value


Normal Q-Q Plot of n1
for grp= NORMAL
Expected Normal
Observed Value


n1
grp
LD
NORMAL







Descriptive statistics presented data in Table, Q plots ,Box Plots Indicate that mean for LD group evoked potential and control group evoked potential which are within normal distribution. The difference of means between the two groups is quite big in the context of their standard deviation. Positive skewness in the distribution for both group. The Kolmogorov – Smirnov Z value are not statically significant ($p>0.05$). Thus the small skewness in the two distributions is not major concern and the two distributions met the assumption of normality therefore further analysis done by using parametric test.

Group Statistics

Table 6: Indicative Descriptive value of different auditory evoked potential mean latency and amplitude.

	Group	N	Mean	Std. Deviation	Std. Error Mean
p1	LD	30	73.7167	9.32118	1.20336
	NORMAL	30	70.2333	7.92415	1.02300
n1	LD	30	1.2457E2	6.97542	.90052
	NORMAL	30	1.2135E2	4.27379	.55174
p2	LD	30	1.6800E2	7.63578	.98577
	NORMAL	30	1.6573E2	5.85088	.75535

n2	LD	30	2.2562E2	12.14055	1.56734
	NORMAL	30	2.1850E2	4.90417	.63313
Amplitude p1	LD	30	4.9938	.75564	.09755
	NORMAL	30	4.5295	.78804	.10174
Amplitude n1	LD	30	5.3288	.99947	.12903
	NORMAL	30	5.3465	1.14994	.14846
Amplitude p2	LD	30	4.1247	.85969	.11099
	NORMAL	30	3.9117	.79654	.10283
Amplitude n 2	LD	30	2.4210	.58072	.07497
	NORMAL	30	2.3618	.56963	.07354

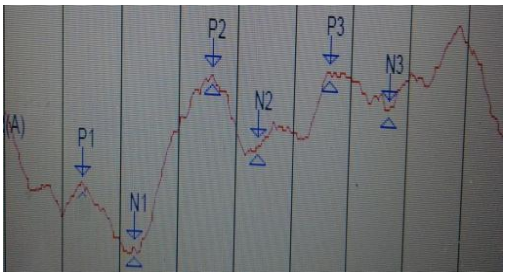


Figure 3: LLR normal waveform

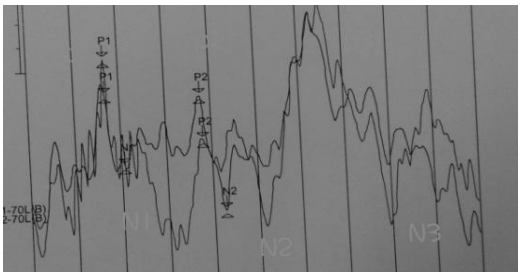


figure 4: LLR waveform of LD

Two independent 't' tail test results

Table 7: Showing Two independent sample't' tail test results of LLR in LD and Control subjects .

Peaks	T	Df	Sig(2 tailed)	Std. Error. Difference	Level of significance at 0.05	
					Lower	Upper
p1	2.205	58	.001	1.57943	.35563	6.61104
p2	1.825	58	.001	1.24189	-.19262	4.72595
n2	4.210	58	.000	1.69038	3.76925	10.46409
Amplitude p1	3.294	58	.0002	.14095	.18522	.74345
Amplitude n1	-.090	58	.001	.19669	-.40717	.37184
Amplitude p2	1.408	58	.162	.15130	-.08662	.51262
Amplitude n2	.563	58	.574	.10502	-.14880	.26713

Latencies:

Mean score and standard deviation were calculated for both the groups. Independent 't' tail test was used to check for any statistically significant difference between the two groups. Results obtained indicate the prolonged latencies and reduction in the amplitude of MLR potential and P1, N1, P2 in CWLD as compared to normal hearing children. Prolonged latencies were observed in CWLD which had a statistically

significant difference in comparison to the control group. Also, wave morphology was inconsistent and poor in CWLD. The present study findings reveal that CWLD have significant difference in central auditory system processing when compared to the normal hearing children. These cortical abnormalities indicate that children with LD have some difficulty in perception of auditory stimulus. Similar result finding were reported by Pinkerton et al (1981) ;Picton et al 2001 they study late auditory evoked potentials in children with reading, writing and spelling difficulties and their results revealed a prolonged latency and reduced amplitudes of responses and inferred that the abnormalities in the auditory cortical area results in altered auditory cortical recording. They further argued that these differences in responses may also reflect a disturbance in selective attention which may in turn affect the reading and writing skills. Purdy et al 2001 also studied LLR in children with learning disabilities and reported that the latency of P1 was earlier whereas P3 latency was prolonged compared to control group.

Amplitude:

In the current study only MLR potential and P1and N1 amplitude showed statically significant difference whereas other peak (i.e. P2, N2, P3, N3) amplitudes did not shown any difference with control subject. CWLD group showed MLR and P1, N1 P2 had smaller amplitude compared to control group. In a study by Satterfield et al. (1987), reported that click-evoked P1 amplitude, P2 amplitude, P1/N1 amplitude and P2/N2 amplitude in children with attention difficulties had no significant difference in 34 control group. Similarly, Byring and Jaryilehto 1985 studied the late latency auditory evoked potentials in individuals who exhibits high rate of spelling errors. They also reported that prolonged latency and reduced amplitude of the peaks of late latency response. Similar results indicated by Purdy et al 2005 found prolonged MLR latencies and shorter amplitude in children with learning disorders.

Discussion

Many school-age children have difficulty in demonstrating basic proficiency in academic areas and are eventually diagnosed with learning and/or attention problems. There is growing evidence to suggest that in some children the root cause of these learning problems may lie in auditory perceptual deficits specifically related to the processing of signals. (Elliott et. al. 1988; Kraus, et. al. 1996; Nittrouer, 1999). Present study results indicates that difference between normally developing children and children with learning problems using cortical evoked potentials that reflect different and more elementary levels of sensory encoding. MLR and P1/N1/N2 response complex has been described for decades (Davis, 1939) that characterized as a series of positive and negative waves of robust nature and easily identifiable in human and exhibit normal hearing sensitivity but due to time consumption, the test has got limited clinical value. Present research supports that this higher cortical response audiometry test gives more specific window to understand functioning of the central auditory system. Reduced amplitude, prolong latencies,

and inconsistent wave morphology in experimental group has been seen i.e. children with learning disorders. LD children having issue in structure like thalamus, auditory cortex which mainly involve in generation of MLR response and LLR response from the scalp. Defects in functioning of these structures can cause issues in temporal integration, filtering out unwanted signal, and categorical auditory perception etc.

Conclusion

The current research study explores the neurobiological bases of learning disorders. The present study contributes to our knowledge of electrophysiological measures (MLR,LLR), auditory cortex functioning, and emphasizes that the latencies of waves P1, P2and N1 were prolonged in children with learning disorders. MLR LLR demonstrates the diversity of these measures, which could be explained by the heterogeneity of the functional processes in children with learning disability. It can be concluded from the present study that click evoked auditory late latency response is easily traceable in all normal hearing children and children with LD. However, children with LD exhibited prolonged latency and reduced amplitude responses, indicative of neurological processing difference in auditory system. This reduced central auditory system activities seen in the children with learning disorder. Hence auditory evoked potential MLR, LLR may be used as a diagnosis protocol for children with LD. Therefore, this research study recommends that CWLD should be subjected to assess higher central auditory tests such as P300, MLR, and LLR. These are non invasive and objective procedures with significant clinical efficacy and will be helpful in underpinning the physiological processes involved in higher auditory function in normal as well as clinical population. Further research is required in this area to use MLR and LLR as a tool to clinically differentiate between individuals with and without LD.

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