

**Original Article:****Auditory Long Latency Responses in Children with Learning Disabilities****Authors:**

**Puneet Sharma**, Student, Speech and Hearing Unit, Department of Otolaryngology,  
**Sanjay Kumar Munjal**, Additional Professor & In-charge (Speech and Hearing Unit), Department of Otolaryngology,  
**Anuradha Sharma**, Lecturer (Speech and Hearing Unit), Department of Otolaryngology,  
**Naresh K. Panda**, Professor and Head, Department of Otolaryngology,  
**Post Graduate Institute of Medical Education and Research (PGIMER), Chandigarh.**

**Address for Correspondence****Dr. Anuradha Sharma,**

Dept of Otolaryngology

PGIMER

Sector 12

Chandigarh 160012

India.

**E-mail:** anuradha2ks@yahoo.com.**Citation**

Sharma P, Munjal SK, Sharma A, Panda NK. Auditory Long Latency Responses in Children with Learning Disabilities. *Online J Health Allied Scs.* 2021;20(3):10. Available at URL: <https://www.ojhas.org/issue79/2021-3-10.html>

Submitted: Jul 6, 2021; Accepted: Oct 4, 2021; Published: Oct 31, 2021

**Abstract: Objective of the study:** To measure and compare the latencies of P1, N1 & P2 of ALLR in children with learning disability. **Methodology:** For 20 children diagnosed with learning disability (LD), Pure Tone Audiometry, Speech Audiometry, Impedance Audiometry, and ALLR were obtained, and the results were compared to a control group. **Important Findings:** Between children with LD and the normal control group, there were highly statistically significant variations in the amplitudes of the waves P1, N1, and P2. The latencies of the ALLR waves P1, N1, P2, and N2 did show differences, though the results did not reach statistical significance. These results indicate that children with LD may have problems in auditory sensory processing. **Conclusions:** Auditory Long Latency Responses can be used for the early detection of children with learning disabilities and provides the opportunity to plan the treatment for improvement of auditory skills.

**Key Words:** Auditory evoked potentials, Learning Disability, Central auditory processing disorder, Pure tone audiometry

**Introduction:**

The term learning disability (LD) describes a neurobiological disorder amid which a human brain is structured or worked differently. Children suffering from Learning Disabilities find difficulties in talking, listening, reading, writing, spelling, reasoning, recalling, organizing information, and in doing arithmetic. Other than these problems children with LD exhibit problems in attention, social-emotional problems, nonverbal learning disorders, motivational problem, attribution problems, and perceptual deficits [1]. The prevalence of learning disabilities is subject to much dispute thanks to the dearth of an agreed-upon definition of LD and objective diagnostic criteria[2-4]. The DSM-V reports prevalence estimates of 5-15 % for children with Learning Disabilities[5]. Children acquire language by the auditory mode; thus, it's crucial for learning that a child's auditory system should function properly. Children with LD can have problems in

auditory function e.g., they may have a problem in hearing loud sounds and can be very sensitive to soft sounds, and these children may also have Central Auditory Processing Disorders (CAPD), or disorders of auditory attention[6-9].

The Auditory Evoked Potentials (AEPs) gives us information about the functioning of the auditory system. The auditory P1-N1-P2 complex is constituted of three peaks. These responses are described by means of amplitude and latency. Children's LLRs are dominated by the P1 and N2 peaks, while the LLR of adults are prevailed by the P1-N1-P2 complex. In preschool-age children (1-4 years), the P1 is the most predominant peak[10-11], while in school going children (3-6 years) the wave N2 becomes progressively robust. After that, wave N2 dominates LLR potentials until adolescence[11,12]. From about age 3, with slow stimulation rate, the N1 can be recorded in addition to the N2 peak[13,14].

Auditory Long Latency Responses (ALLR) is a crucial way to evaluate auditory information processing at the central level. The integrity of primary auditory cortex and association cortex is an indicator that the auditory acuity can be assessed by using ALLR. Therefore, the objective of the current study is to assess the auditory sensory process in cortical areas by using auditory long latency responses, by comparing the latencies of P1, N1 & P2 in children with learning disability and normal control group.

**Methods**

The study was conducted at the Speech & Hearing Unit, Department of Otorhinolaryngology, PGIMER, Chandigarh. A total of 20 children in the age range of 7 – 12 years, diagnosed in the spectrum of learning disability by a specialist in clinical psychology were referred from the Out Patient Department of Psychiatry, PGIMER, Chandigarh. In order to avoid across-group variability during comparisons of ALLR parameters in children, age matched control was also enrolled. Twenty age and sex-matched otologically normal subjects as controls were taken. The children who have any history of

middle ear infection, family history of deafness, history of ototoxic drug usage and those children who have IQ < 70 were excluded from the study. An informed consent form was signed by all the subjects and their parents before participating in the study. The ethics committee of the institute approved the study before the commencement of the study.

#### **Instruments and Procedure:**

A detailed case history of all the subjects was taken which was followed by otological examination. All the patients were subjected to audiological test battery comprising of Pure Tone Audiometry (PTA), Speech Audiometry, Immittance Audiometry and Auditory Long Latency Responses (ALR). Each ear was tested separately.

#### **Pure Tone Audiometry**

Air conduction (AC) and bone conduction (BC) thresholds were measured for pure tone stimuli in a sound treated room at 250, 500, 1000, 2000, 4000 and 8000 Hz using Madsen Orbiter 922 diagnostic audiometer. The dual channel audiometer was used to measure the pure tone thresholds, speech discrimination score and speech reception thresholds of all the individuals. The instrument was calibrated as per American National Standards Institute (ANSI) standards. Telephonics TDH 39 supra aural earphones with noise excluding headsets were used for pure tone audiometry.

#### **Speech Audiometry**

**Speech Reception Threshold (SRT):** Madsen Orbiter 922 diagnostic audiometer and TDH 39 earphones were used to measure Speech Reception Threshold. Spondee words in Hindi (developed at AIIMS, New Delhi) and Punjabi (developed by Dr. Sanjay Munjal, PGIMER, Chandigarh) were presented to the subjects and they were asked to repeat them. SRT was taken as the minimum intensity, at which 50% of the spondee words were repeated correctly.

**Speech Discrimination Scores (SDS):** A 50-word phonetically balanced (PB) Hindi/Punjabi list was presented to each patient's ear at 40 decibels above the SRT. The number of words that were accurately repeated was recorded, and a speech discrimination score was calculated.

#### **Impedance Audiometry**

Immittance audiometer Maico MI 34 calibrated according to the "ANSI" standards, was used to assess the function of middle ear and to measure acoustic stapedial reflex of the subjects. The pressure range was varied from -400 to +200 daPa with air pump speed of 400 daPa/sec. The frequency of the probe tone used to measure the ear canal volume was 226 Hz, as required by CEI/IEC 1027: 1991 and ANSI S3.39-1987 standards.

#### **Auditory Late Latency Responses (ALR)**

ALLR was measured with a NeuroAudio auditory evoked potential diagnostic system EEG electrodes were used to obtain all the recordings. The electrode impedance was maintained below 5 K ohm. Responses were differentially recorded from Cz to ipsilateral mastoid with forehead as ground. **The following parameters were used for acquiring ALR responses**

**Stimulus:** 40ms Tone Burst

**Frequency:** 1000 Hz

**Rate:** 1.1/sec

**Polarity:** Alternating

**Transducers:** Insert Earphones

**Intensity** – 90 dBnHL

**Filters:** 1-30 Hz

**Notch Filter:** Off

**Amplification:** 50x

**Runs:** 2

**Analysis time window:** Overall 600ms

**Sweeps:** 250

**Electrode Montage:** Ipsilateral Array

**Statistical Analysis:** The statistical analysis was carried out with the SPSS 16 software package (statistical package for

social sciences). Initially the data was screened for normality. Descriptive statistics was carried out using chi-square test and student 't' test was applied to compare the study and control groups. The mean and standard deviation were reported and a p value less than 0.05 was considered statistically significant.

## **Results**

### **Characteristics of patients**

The children included for the study were all between the ages of 7 and 12, with an average age of 8.95 years (SD = 2.11) and 9.00 years (SD = 2.25) for control and study group respectively; 75 % (N=15) of children were males and 25 % (N=5) were females in the control group while in the study group, 80 % (N=16) of the subjects were males and 20 % (N=4) were females.

### **Audiological findings**

The mean pure tone average (average of 500 Hz, 1kHz & 2 kHz) for the right ear in the control group, was 17.50 dB HL (SD=1.91) while it was 17.42 dB HL (SD= 1.95) for the study group. The mean pure tone average for the left ear in the control group, was 17.45 dB HL (SD= 1.32) while it was 17.42 dB HL (SD=1.17) for the study group. The t value of 0.1278 (p>0.05) and 0.0724 (p>0.05) respectively for right and left ear depicted no significant differences between the two groups.

The SRT in the right ear of the normal group was 21.60 dB HL (SD=2.04) while it was 22.21 dB HL (SD=2.46) in the study group. While the SRT in the left ear of the normal group was 23.00 dB HL (SD=1.52) and 23.21 dB HL (SD=1.55) in the study group. The t value of 0.8455 (p = 0.4032) and 0.4282 (p= 0.6710) for the right and left ear respectively depicted no significant differences between the two groups.

SDS was 94.40 % (SD= 2.01) for the right ear in the control group, it was 94.32 % (SD= 2.03) in the study group. In the case of the left ear, it was 94.40 % (SD= 2.01) in the control group and 94.53 % (SD= 1.98) in the study group. The t values of t= 0.1302 (p= 0.8971) and t=0.1975 (p= 0.8846) for the right ear and left ear respectively, suggests no significant differences between the two groups.

Impedance Audiometry depicted type "A" tympanogram in both the ears of all the children in the study group and control group. Both Ipsilateral and Contralateral reflexes were present in both the ears of all the children in the study and control group.

The mean latency was 63.31 msec (SD=6.874) and 66.38 msec (SD=5.854) for wave P1. The t value of 1.4972 (p=0.1428). For wave N1, the mean latency was 97.09 msec (SD=2.457) and 97.37 msec (SD=3.709) for the control and study group respectively, t = 0.2777 (p=0.7828). For wave P2, the mean latency was 143.52 msec (SD=10.991) and 142.01 msec (SD=11.297) for the control and study group respectively, t = 0.423 (p=0.6748). For wave N2, the mean latency was 195.07 msec (SD=16.479) and 198.88 msec (SD=22.507) for the control and study group respectively, t = 0.6069 (p=0.5476). All these findings depicted no statistically significant differences between the groups.

In the left ear for wave P1, the mean latency was 70.86 msec (SD=7.27) and 71.64 msec (SD=6.22) for the control and study group respectively, t= 0.1759 (p=0.8613). While for wave N1 the mean latency was 99.07 msec (SD=6.986) and 104.14 msec (SD=9.588) for the control and study group respectively, t = 1.8971 (p=0.0656). The mean latency of wave P2 was 148.02 msec (SD=12.37) and 151.28 msec (SD=9.837) for the control and study group respectively, t = 0.9091 (p=0.3692). While for wave N2 the mean latency was 194.58 msec (SD=14.356) and 199.37 msec (SD=20.555) for the control and study group respectively, 't' value of 0.8479 (p=0.4019). All these findings show no statistically significant differences between the groups.

For wave P1, the mean amplitude was 5.24  $\mu\text{V}$  (SD=1.231) and 6.36  $\mu\text{V}$  (SD=1.837) for the control and study group respectively,  $t = 2.2540$  ( $p=0.0302$ ). For wave N1, the mean amplitude was 7.04  $\mu\text{V}$  (SD=2.243) and 5.26  $\mu\text{V}$  (SD=1.446) for the control and study group respectively,  $t = 2.9644$  ( $p=0.0053$ ). For wave P2, the mean amplitude was 4.53  $\mu\text{V}$  (SD=1.029) and 6.1  $\mu\text{V}$  (SD=1.216) for the control and study group respectively,  $t = 4.3583$  ( $p=0.0001$ ). These findings show highly statistically significant differences between the two groups.

In the left ear, the mean amplitude for wave P1 was 4.60  $\mu\text{V}$  (SD=1.280) and 6.51  $\mu\text{V}$  (SD=1.066) for the control and study group respectively,  $t = 5.0658$  ( $p=0.0001$ ). For wave N1 the mean amplitude was 6.53  $\mu\text{V}$  (SD=1.821) and 4.78  $\mu\text{V}$  (SD=1.417) for the control and study group respectively,  $t = 3.352$  ( $p=0.0019$ ). And for wave P2 the mean amplitude was 4.39  $\mu\text{V}$  (SD=1.669) and 5.69  $\mu\text{V}$  (SD=1.349) for the control and study group respectively,  $t = 2.6659$  ( $p=0.0113$ ). All these findings show statistically significant differences between the groups.

### Discussion

In the current study, significant differences in the AC thresholds, SRT, and SDS between the control and study group were not found. The results of the air conduction threshold, SRT, and SDS in the present study are in consonance with the findings of Murphy C F B & Schochat E done in 2009. They analyzed 30 dyslexic and 27 normal controls children. In their findings, all the children have hearing within normal limits in their audiometric evaluation [15]. However, in a study done in 1980 by Bennett F C et al in which 38% of children with LD and 16% of children in the control group had hearing problems on PTA test [16]. There was no observable difference in the tympanogram and stapedial reflex finding between the two groups. However, Freeman and Parkins in 1979 study 50 LD children. The clinical examination revealed that 20 percent of the LD children had evidence of OME [17] and as reported by Thomas WG et al in 1985, 32% of the children with LD have abnormal Acoustic Reflex Thresholds (ARTs) [18].

The results of LLR indicates that the latency of wave P1 is delayed but could not reach the level of significance. Similar findings were found by Farah Khaliq et al in 2010 [19]. However, Purdy et al in 2002 conducted a study on 10 individuals with LD and a normal control group in the age range of 7-11 years and found that P1 latencies were shorter in children with LD [20]. The amplitude of wave P1 is enhanced significantly in the study group as compared to the normal control group. In a study by Satterfield et al. (1987), the authors reported that click-evoked amplitude of waves P1, P2, P1/N1 and P2/N2 in children with attention difficulties were not significantly different in control group. On the contrary, Byring and Jarylehto (1985) reported the reduced amplitude of the peaks of late latency auditory evoked potentials in individuals who exhibited a high rate of spelling errors.

The values for wave N1 latencies were higher in children with learning disabilities though not statistically significant, and these findings are suggestive of the changes in the auditory processing of children with Learning Disabilities. Bernal et al [21] also did not find any significant differences in the latency of N1 between normal and poor readers. But Hämäläinen JA et al [22] in 2007 found that children with a reading disability had a statistically significant larger N1 response than control children. However, Purdy et al in 2002 reported that wave N1 was earlier in children with learning disability as compared to the normal control group. It was suggested that delayed latency of wave N1 component is associated with auditory processing onset failure, but specifically, it is related to deficits in auditory cortical information synchronization associated with auditory attention factors [20]. And, the amplitude of the wave N1 is reduced significantly in children of the study group

as compared to the normal control group. The reduction in amplitude of wave N1 in the children of the study group in our study is consistent with other studies [28,18]. Jutras B et al in 2001 also reported a reduction in amplitude of the N1 wave in an individual presenting verbal disfluency and learning difficulties [29]. However, Purdy S.C et al in 2002 found that for standard stimuli, N1 amplitude was smaller and P2 was earlier. And for deviant stimuli, N1 was earlier and P2-N2 was smaller [20]. The drop in amplitude of wave N1 could be due to a reduction in electrical activity in the supratemporal auditory cortex's primary and secondary areas, which is involved in processing [30].

The P2 wave is associated with auditory and temporal characteristics of the sound. In children with LD, the wave P2 has a late onset and they showed problems in encoding and depiction of the stimulus which is received by the central auditory pathway [23]. In the present study, the latency of wave P2 is also delayed but not statistically significant in children with LD. Analogous results have been observed by Lubar J F et al in 1992; however, they found that wave P2 latency is statistically significantly delayed. However, Cynthia King et al in 2001 did not find any latency or amplitude differences in children with learning impairment (LP) and those with normal hearing but delayed brainstem onset latencies to stimulus /da/ [24]. Prolongation of P2 wave latency in our study indicates a deficiency in stimulus discrimination in children with LD. P2 amplitude was enhanced statistically significantly in LD children as compared to the control group in the current study.

The N2 wave is associated with the physical discrimination of the stimuli and the N2 wave is also associated to sensory auditory processing, which is responsible for perception, recognition, attention, and discrimination of sounds [25]. The latency of the N2 wave in the present study was also delayed in children with LD when compared with the normal control group but statistically insignificant difference was seen. The findings of the current study are in agreement with the results of Farah Khaliq et al in 2010 [19]. The passive and pre-attentive habitual responses produced by the discrimination of a nonstandard stimulus in the midst of standard stimuli during LLR recording were weak in children with learning difficulties in the present study. This weakness in children with learning disabilities suggests that these children's discrimination and attention were distorted [26,27] and it also suggests that these children may have problems in auditory sensory processing which is accountable for discrimination of sounds, auditory perception, and attention.

The deviations in the values of waves P1, N1 & P2 between the study group and normal control group could also be attributed to the differences in the anatomical structure in the medial geniculate nucleus in the thalamus and corpus callosum as suggested by different studies [19,22,31]. The authors suggested that anatomical differences in these areas could be the reason for changed long latency responses in children with Learning Disabilities [32,33]. And some authors also suggested that there is a difference in the quantification of blood flow in children with Learning Disabilities mainly in the right hemisphere as compared to normal children [34]. The findings of the current study reveal that children with learning disabilities may have anatomical and functional problems. Therefore, LLR can be used to diagnose learning problems in children at an early age. This test also allows you to organise your treatment for improving your auditory skills using the auditory-linguistic training method. Because these auditory skills are required for the development of reading and writing abilities.

### Conclusion

The present study tried to assess the auditory sensory process in cortical areas by using auditory long latency responses. The

electrophysiological investigations using ALLR revealed differences in findings between the control group and children with LD. The deviations in the values of waves P1, N1 & P2 between the study group and normal control group could be attributed to the differences in the anatomical structure in the medial geniculate nucleus in the thalamus and corpus callosum. And it is also associated with auditory processing onset failure, but specifically, related to deficits in auditory cortical information synchronization associated with auditory attention factors. Therefore, LLR can be used for the early detection of children with learning disabilities and provides the opportunity to plan the treatment for improvement of auditory skills.

**Acknowledgments:** We would like to thank all the subjects who participated in the present research work.

## References

1. Lyon GR. IQ Is Irrelevant to the Definition of Learning Disabilities. *Journal of Learning Disabilities*. 1989;22(8):504–.
2. Lyon GR. Better understanding learning disabilities: new views from research and their implications for education and public policies. Baltimore, MD: Paul H. Brookes Pub. Co.; 1993.
3. Lyon GR. Toward a definition of dyslexia. *Annals of Dyslexia*. 1995;45(1):1–27.
4. Shaywitz SE, Fletcher JM, Shaywitz BA. Issues in the definition and classification of attention deficit disorder. *Topics in Language Disorders*. 1994;14(4):1–25.
5. Diagnostic and statistical manual of mental disorders: DSM-5. Arlington, VA: American Psychiatric Association; 2013.
6. Delacato CH. The ultimate stranger: the autistic child: Novato, CA: Arena Pr.; 1984.
7. Greenspan, S.I. Infancy and early childhood: The practise of clinical assessment and intervention with emotional and developmental changes. Madison, CT: International Universities Press (1992).
8. Powers MD. Early intervention for children with autism. In D.E. Berkell (Ed.), *Autism: Identification, education, and treatment*. Hillsdale, NJ: Erlbaum.; 1992. p. 225–252.
9. Rapin I. Autistic children: diagnosis and clinical features. *Pediatrics*. 1991 May;87(5 Pt 2):751–60.
10. Kushnerenko E, Ceponiene R, Balan P, Fellman V, Huottilainen M, Näätänen R. Maturation of the auditory event-related potentials during the first year of life. *Neuroreport*. 2002;13(1):47–51.
11. Pang E, Taylor M. Tracking the development of the N1 from age 3 to adulthood: an examination of speech and non-speech stimuli. *Clinical Neurophysiology*. 2000;111(3):388–97.
12. Ponton CW, Eggermont JJ, Kwong B, Don M. Maturation of human central auditory system activity: evidence from multi-channel evoked potentials. *Clinical Neurophysiology*. 2000;111(2):220–36.
13. Paetau R, Ahonen A, Salonen O, Sams M. Auditory Evoked Magnetic Fields to Tones and Pseudowords in Healthy Children and Adults. *Journal of Clinical Neurophysiology*. 1995;12(2):177–85.
14. Sharma A, Kraus N, Mcgee TJ, Nicol TG. Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalography and Clinical Neurophysiology/ Evoked Potentials Section*. 1997;104(6):540–5.
15. Murphy C, Schochat E. How auditory temporal processing deficits relate to dyslexia. *Brazilian Journal of Medical and Biological Research*. 2009;42(7):647–54.
16. Bennett FC, Ruuska SH, Sherman R. Middle ear function in learning-disabled children. *Pediatrics*. 1980 Aug;66(2):254–60.
17. Freeman BA, Parkins C. The Prevalence of Middle Ear Disease among Learning Impaired Children. *Clinical Pediatrics*. 1979;18(4):205–12.
18. Thomas WG, Mcmurry G, Pillsbury HC. Acoustic Reflex Abnormalities In Behaviorally Disturbed And Language Delayed Children. *The Laryngoscope*. 1985;95(7).
19. Khaliq F, Alam KK, Vaney N, Singh TB. Sensory, cognitive and motor assessment of children with poor academic performance: an auditory evoked potential study. *Indian J Physiol Pharmacol*. 2010 Jul-Sep;54(3):255–64.
20. Purdy SC, Kelly AS, Davies MG. Auditory brainstem response, middle latency response, and late cortical evoked potentials in children with learning disabilities. *J Am Acad Audiol*. 2002 Jul-Aug;13(7):367–82.
21. Bernal J, Harmony T, Rodríguez M, et al. Auditory event-related potentials in poor readers. *International Journal of Psychophysiology : Official Journal of the International Organization of Psychophysiology*. 2000 Apr;36(1):11–23.
22. Hämäläinen J, Leppänen P, Guttorm T, Lyytinen H. N1 and P2 components of auditory event-related potentials in children with and without reading disabilities. *Clinical Neurophysiology*. 2007;118(10):2263–75.
23. Hall JW. New handbook of auditory evoked responses. Boston: Pearson; 2007.
24. King C, Warrier CM, Hayes E, Kraus N. Deficits in auditory brainstem pathway encoding of speech sounds in children with learning problems. *Neuroscience Letters*. 2002;319(2):111–5.
25. Leppänen PHT, Lyytinen H. Auditory Event-Related Potentials in the Study of Developmental Language-Related Disorders. *Audiology and Neurotology*. 1997;2(5):308–40.
26. Näätänen R. Attention and brain function. Abingdon: Routledge, 2018. 494 p. (Psychology library edition. Neuropsychology). <https://doi.org/10.4324/9780429487354>
27. Ceponiene R, Rinne T, Näätänen R. Maturation of cortical sound processing as indexed by event-related potentials. *Clinical Neurophysiology*. 2002;113(6):870–82.
28. Tonnquist-Uhlen I. Topography of Auditory Evoked Long-Latency Potentials in Children with Severe Language Impairment: the T Complex. *Acta Oto-Laryngologica*. 1996;116(5):680–9.
29. Jutras B, Lagacé J, Lavigne A, Boissonneault A, Lavoie C. Auditory processing disorders, verbal disfluency, and learning difficulties: A case study. *International Journal of Audiology*. 2007;46(1):31–8.
30. Sauer L, Pereira LD, Ciasca SM, Pestun M, Guerreiro MM. Processamento auditivo e SPECT em crianças com dislexia. *Arquivos de Neuro-Psiquiatria*. 2006;64(1):108–11.
31. Gilley PM, Sharma A, Dorman M, Martin K. Abnormalities in central auditory maturation in children with language-based learning problems. *Clinical Neurophysiology*. 2006;117(9):1949–56.
32. Arehole S. A preliminary study of the relationship between long latency response and learning disorder. *British Journal of Audiology*. 1995;29(6):295–8.
33. Picton T, Alain C, Woods D, John M, Scherg M, Valdes-Sosa P, et al. Intracerebral Sources of Human Auditory-

- Evoked Potentials. *Audiology and Neuro-Otology*. 1999;4(2):64–79.
34. Calanchini PR, Trout SS. Neurologia dos distúrbios de aprendizagem. In: Tamopol L. Crianças com distúrbios de aprendizagem: diagnóstico, medicação, educação. São Paulo: Edart; 1980.