

Potenciais evocados auditivos de longa latência em crianças com transtorno fonológico****

Long latency auditory evoked potentials in children with phonological disorder

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Abstract

Background: auditory evoked potentials in children with phonological disorder. **Aim:** to characterize the long latency auditory evoked potentials (LLAEP) results N1, P2, N2 and P300 of children with phonological disorder and to verify the improvement of such potentials with speech therapy. **Method:** 25 children without phonological disorder (control group) and 41 with phonological disorder (study group) underwent a basic audiological evaluation and LLAEP. The study group was divided into two subgroups: subgroup A composed by 22 children, underwent 12 sessions of speech therapy and were submitted to audiological retesting after this period, and subgroup B composed by 19 children, who were also reassessed three months after the initial testing. **Results:** statistically significant differences between groups for the P2 and P300 latencies and P300 amplitude were observed. Comparison between the first and the second audiological assessments indicated no significant statistical differences between both subgroups regarding wave latencies. However, a significant statistical difference was verified for the P300 (study subgroup A) and P2/N2 (study subgroup B) wave amplitudes. The study group presented higher percentage of altered results in the P300; wave latency increase was the most frequent type of alteration. After speech therapy, the results of all components improved, however, there was no association between the improvement of LLAEP results with the background of otitis, as well as with the Percentage of Consonants Correct-Revised. **Conclusion:** children with phonological disorder present altered P300 suggesting involvement of the central auditory pathway, probably due to alterations in the auditory processing, presenting improvement in all components of LLAEP results after speech therapy.

Key Words: Auditory Evoked Potentials; Articulation Disorders; Language Therapy; Neuronal Plasticity.

Resumo

Tema: potenciais evocados auditivos em crianças com transtorno fonológico. **Objetivo:** caracterizar os resultados dos Potenciais Evocados Auditivos de Longa Latência (PEALL) N1, P2, N2 e P300 obtidos em crianças com transtorno fonológico, e verificar a evolução dos resultados destes potenciais frente à terapia fonoaudiológica. **Método:** foram avaliadas, por meio da avaliação audiológica básica e dos PEALL, 25 crianças sem transtorno fonológico (grupo controle) e 41 com transtorno fonológico (grupo estudo), estas divididas em dois subgrupos: 22 formaram o subgrupo estudo A, que foram submetidas a 12 sessões de terapia fonoaudiológica e reavaliadas audiológicamente após este período e 19 o subgrupo estudo B, que foram reavaliadas após três meses da avaliação inicial. **Resultados:** observaram-se diferenças estatisticamente significantes entre os grupos controle e estudo para as latências de P2 e P300 e amplitude do P300. Na comparação entre as duas avaliações audiológicas, não foram observadas diferenças significantes para as latências em ambos os subgrupos, e verificou-se diferença significativa para as amplitudes do P300 (subgrupo estudo A) e do P2/N2 (subgrupo estudo B). O P300 apresentou maior porcentagem de resultados alterados no grupo estudo, com predomínio do aumento de latência. Após terapia, observou-se melhora nos resultados para todos os componentes. Não existiu associação entre a evolução dos resultados dos PEALL e o histórico de otite, bem como correlação com o *Percentage of Consonants Correct-Revised*. **Conclusão:** crianças com transtorno fonológico apresentam alterações no P300, sugerindo alteração no processamento auditivo, apresentando melhora nos resultados de todos os componentes dos PEALL frente à terapia fonoaudiológica.

Palavras-Chave: Potenciais Evocados Auditivos; Transtornos da Articulação; Terapia da Linguagem; Plasticidade Neuronal.

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Introduction

The phonological disorder is a difficulty in speech characterized by inappropriate use of sounds and different degrees of severity and speech intelligibility (1).

In the literature, the investigation of long-latency auditory evoked potentials (LLAEP) in children with phonological disorder shows that these children exhibit no alteration in the Mismatch Negativity (MMN); however, they do exhibit changes in behavioral tests, suggesting the existence of deficits in temporal or non-auditory processing such as attention (2). It is noteworthy that these children may exhibit alterations in the values of latency and amplitude of the N2 component (3), as well as the latency values of Brainstem Auditory Evoked Potential (BAEP) and P300 - with a higher occurrence of alterations being reported for the P300. It is also emphasized that the Speech therapy can improve the outcome of these potentials (4).

Therefore, the objective of this study was to characterize the results of LLAEP obtained in children with phonological disorder and to investigate the development of the potential after Speech therapy as well as to correlate the development of potentials and the history of otitis and severity of the phonological disorder.

Method

This research was approved by Cappesq HC FMUSP with protocol number 1360/06.

Sixty-six children between eight and 11 years of age participated in this study. Twenty-five children with no phonological disorder comprised the control group (CG) - mean age = 8 years and 11 months, SD = 11 months. Forty-one children with phonological disorder comprised the study group (SG) - mean age = 9 years, SD = 1 year and 1 month. Children from the SG were divided into two subgroups: 22 were submitted to Speech therapy (study subgroup A - SEA); and 19 children were not submitted to speech therapy (study subgroup B - SEB). Children from SEB were on the waiting list for the treatment.

The ABFW Child Language Test (5) was used for the selection of children. The severity of this disorder was determined by the Percentage of Consonants Correct-Revised (PCC-R) (6).

The following procedures were performed to ensure hearing thresholds between zero and 15 dB HL at all tested frequencies: acoustic immittance measures - middle ear analyzer GSI-33 from Grason-Stadler Inc., Milford, NH, USA; pure tone audiometry,

performed at frequencies from 250 to 8000 Hz and speech audiometry - Grason-Stadler GSI-68 Audiometer and supra-aural ear phones model TDH 50 from Telephonics Corp., Farmingdale, NY, USA.

The LLAEP (N1, P2, N2 and P300) were recorded through the program EP317 of the 2-channel Portable equipment model Traveler Express from Bio-logic Systems Corp., Mundelein, IL, USA. The electrodes were placed at left and right mastoid (M2 and M1), vertex (Cz), and forehead (Fpz). The electrode fixed on mastoid of the tested ear was considered the active electrode. The electrode fixed at the vertex was considered the reference electrode and the electrode fixed on the forehead was considered the ground electrode. The acoustic stimulus used was a tone burst at 75 dB HL at frequencies of 1000 Hz (frequent stimulus) and 1500 Hz (rare stimulus). The acoustic stimuli was presented monaurally and on a random order by the computer with display speed of 1.1 stimuli per second, analysis window of 512 ms, high-pass filters of 30.00 Hz, low-pass of 1.00 Hz, and a gain of 15000. The rare stimulus represented 20% of the total of 300 stimuli. The child was oriented to pay attention and to identify the rare stimuli by counting aloud the number of times the rare event occurred.

LLAEP component analysis was performed by the first author and by a judge at different moments in order to ensure the reliability of data.

For the qualitative analysis, reference latency values proposed in the literature (7) - in milliseconds (ms) - were used for the analysis of latencies: N1= 83-135; P2= 137-194; N2= 200-280; P300= 241-396. The results were classified as normal and altered for each individual. Results were considered altered when at least one ear was compromised. Alterations were classified as increased latency, no response, and both (increased latency and no response occurred concomitantly in the same subject).

For the quantitative analysis, the maximum latency value obtained for this parameter in the total sample plus 25% of the variation was considered the latency value for children with no response for the components (8). Thus, the stipulated latency values (in ms) were: N1 = 195 and P2 = 270. The N2 and P300 were present in all children tested. With respect to amplitude, the minimum value of amplitude zero μ V (8) was adopted. It was not possible to classify the results as normal and altered in cases of absence of the component because there are no normative values for the amplitude of the components analyzed.

The control group children were subjected to only one audiological assessment (conventional and electrophysiological hearing), while those from the SG were submitted to assessment and reassessment.

After the first audiological assessment, the SEA initiated weekly Speech therapy with 45 minutes of total duration. Children from the SEA returned for audiological reassessment after 12 sessions (this range was established for inducing fewer absences in therapy). The therapeutic model used was adapted from the model of cycles, as proposed in the literature (9). Children of SEB underwent audiological reassessment three months after the initial assessment. This was to ensure that possible improvements in the LLAEP of children from SEA were not related to the aspect of maturity.

It should be emphasized that the audiological reassessment of SEA and SEB maintained all the same testing conditions established for the first assessment such as day period, procedures sequence and parameters.

The differences of latency and amplitude between the first and second audiological assessments were calculated for both groups. From this, the development of the results of the SEA LLAEP was classified as improved and not improved. The mean differences in latencies and amplitudes of the LLAEP components of SEB were used as a parameter for normal range (reference). The criteria adopted for this classification are described below:

. Improved: when the difference obtained for the SEA was higher than the average difference in the results obtained in the SEB, in at least one ear;

. Not improved: when the difference obtained for the SEA was equal to or less than the average difference obtained in the SEB, in both ears.

These results were also used to study the association between the LLAEP development and history of otitis as well as to analyze the correlation between the LLAEP development and PCC-R in children from the SEA.

The following tests were used on the statistical analysis: Mann-Whitney, Wilcoxon, Equality of Two Proportions, Chi-Square for Independence, and Spearman correlation. The significance level adopted was of $p < 0.05$ (5%) for all tests.

Results

In the between groups comparison of LLAEP latencies and amplitudes (quantitative analysis), there were statistically significant differences between the groups for latency of components P2 and P300 and P300 amplitude (Table 1).

The increased latency was the most frequent alteration observed in both groups for all components studied.

There were no statistically significant differences in the latency of the components studied when comparing the results between audiological assessment and reassessment of SEA. A statistically significant difference was observed only for the P300 amplitude ($p = 0.039$), which average value was lower in the second audiological evaluation (1st assessment = 17.97 μV and 2nd assessment = 13.83 μV). As for the SEB, there was statistically significant difference only for the P2/N2 amplitude ($p = 0.008$), which average value was higher in the second audiological assessment (1st assessment = 6.90 μV and 2nd assessment = 8.13 μV).

In the analysis of LLAEP components development of SEA, statistically significant differences between the results classified as improved and not improved were observed for both latency and amplitude, with higher percentage of improvement observed for all components studied (Table 3).

Statistically significant difference was observed only for the latency of the N1 component ($p = 0.040$) in the comparison of LLAEP components of children with phonological disorders with and without history of otitis.

There was no association between the development of the N1, P2, N2 and P300 components (amplitude and latency) of children of SEA and history of otitis, as well as a no correlation between the development of the LLAEP components (latency and amplitude) and PCC-R of children from SEA.

TABLE 1. Comparison of latencies and amplitudes of N1, P2, N2 and P300 components between children with and without phonological disorders (control and study groups).

LLAEP		Mean	Median	SD	Q1	Q3	N	CI	p-value	
Latency (ms)	N1	CG	119,7	105	32,7	96	144	50	9,1	0,955
		SG	113,5	109	24,3	100	122	82	5,3	
	P2	CG	175,4	176	36,9	159	194	50	10,2	0,012*
		SG	159,5	156	27,3	144	178	82	5,9	
	N2	CG	244,3	244	26,7	225	264	50	7,4	0,071
		SG	233,2	239	30,3	220	252	82	6,6	
	P300	CG	326,8	326	40,1	309	340	50	11,1	0,008*
		SG	353,2	344	58,6	311	392	82	12,7	
Amplitude (µV)	N1/P2	CG	4,94	4,47	3,47	2,09	7,02	50	0,96	0,148
		SG	4,07	3,40	3,03	1,60	5,67	82	0,66	
	P2/N2	CG	6,32	5,77	3,41	3,87	8,92	50	0,94	0,063
		SG	7,82	7,37	4,18	4,62	10,76	82	0,90	
	P300	CG	16,66	16,28	7,98	10,55	21,69	50	2,21	0,027
		SG	13,48	12,91	5,58	9,48	17,34	82	1,21	

Note: CG – control group; SG – study group; SD – standard deviation; Q1 – first quartile; Q3 – third quartile; N – number of ears tested; CI – confidence interval * p-value – considered statistically significant

TABLE 2. Distribution of the occurrence of normal and abnormal results for the N1, P2, N2 and P300 component in the control and study groups

	Control Group		Study Group		p-value	
	N	%	N	%		
N1	normal	15	60	31	75,6	0,181
	abnormal	10	40	10	24,4	
	p-value	0,157		<0,001*		
P2	normal	16	64	35	85,4	0,045*
	abnormal	9	36	6	14,6	
	p-value	0,048*		<0,001*		
N2	normal	21	84	37	90,2	0,451
	abnormal	4	16	4	9,8	
	p-value	<0,001*		<0,001*		
P300	normal	22	88	27	65,9	0,046*
	abnormal	3	12	14	34,1	
	p-value	<0,001*		0,004*		

Note: N – number of children tested; * p-value – considered statistically significant

TABLE 3. Analysis of the development of latency and amplitude of components N1, P2, N2 and P300 in children with phonological disorder submitted to Speech therapy (study subgroup A)

Development (SEA)		Improved		Not improved		p-value
		N	%	N	%	
Latency	N1	20	90,9%	2	9,1%	<0,001*
	P2	19	86,4%	3	13,6%	<0,001*
	N2	19	86,4%	3	13,6%	<0,001*
	P300	15	68,2%	7	31,8%	0,016*
Amplitude	N1/P2	15	68,2%	7	31,8%	0,016*
	P2/N2	20	90,9%	2	9,1%	<0,001*
	P300	17	77,3%	5	22,7%	<0,001*

Note: SEA – subgroup A of study; N – number of children tested

* p-value – considered statistically significant

Discussion

Although the literature reports the presence of changes in the N1, P2 and N2 LLAEP components in children with language disorders (10,11) and N2 in children with phonological disorders (3), such results were not observed in the current study (Table 1).

Some authors reported that children with specific language impairment (SLI) exhibit a modification on the morphology of components N1, P2 and N2, indicating an immaturity of the auditory cortex (12). In this study we found a higher percentage of normal results for the latency of these components in the SG, suggesting that this measurement parameter is not

the most suitable for such analysis. Future studies should investigate the morphology of these components in this population. Furthermore, these findings may have occurred due to the fact that phonological disorder has several correlated causes (6).

The P300 findings (Tables 1 and 2) corroborate those of other studies reporting that children with phonological disorders have increased mean latency, higher percentage of abnormal results and increased latency as the type of alteration most frequently observed (4). No studies that have analyzed the

amplitude of this potential in this population was found in the literature.

The higher incidence of LLAEP improvement in SEA (Table 3) allows one to raise the hypothesis that changes have occurred in the structural organization and/or functioning of the central nervous system after Speech intervention in children with phonological disorders. Some authors have shown that children attending Speech therapy exhibit improvement in parameters of the P300 component (4,13). Other studies also showed improvement in several long latency potentials after some type of auditory training (14,15,16). Finally, the observed changes in the LLAEP components after Speech therapy suggest that the practice of certain skills or frequent exposure to a stimulus during the therapeutic process favors the occurrence of neuronal plasticity (17).

Studies in the literature report that otitis can cause changes in central auditory pathways (18,19,20). However, evidence of this relationship was not observed in the current study (the comparison of LLAEP of children with phonological disorders with and without history of otitis showed no significance). This finding may be related to the manner which the history of otitis was obtained in the current study: parent reporting instead of specific audiological evaluation (19,21).

This fact may also have contributed to the lack of association between the development of LLAEP components and history of otitis, besides the hypothesis that the central auditory pathway undergoes modifications facing the auditory stimulation regardless of the presence or absence of a history of otitis.

Likewise, there were no significant correlations between development in latency and amplitude of LLAEP components and the PCC-R of children with

phonological disorder who underwent Speech therapy. However, it was observed that the development of P300 latency and the PCC-R were inversely proportional. This is an important finding because the decrease in P300 latency indicates a better response of the auditory pathway (22) and a higher percentage in the PCC-R indicates a better phonological system performance (6). Regarding amplitude, the results showed that the development of the N1/P2 amplitude and the PCC-R are directly proportional. The increase in the value of this parameter indicates a better response of the auditory pathway (22) and increased percentage of PCC-R indicates a better phonological system performance (6).

The findings of this study suggest that central auditory pathways suffered a structural reorganization with Speech therapy which directly influenced the processing of acoustic information. Thus, one can infer that the decrease in P300 latency indicates that the stimulus was decoded faster due to the responsiveness of neurons and that the increase in N1/P2 amplitude occurred due to activation of a greater number of neuronal fibers.

Conclusion

This study found that children with phonological disorder present altered P300 suggesting involvement of the central auditory pathway probably due to alterations in the auditory processing, presenting improvement in all components of LLAEP results after speech therapy. There was no association between development of LLAEP components and history of otitis, as well as no correlation between the development of LLAEP components and PCC-R.

References

1. Wertzner HF. Fonologia: Desenvolvimento e alterações. In: Ferreira LP, Béfi-Lopes DM, Limongi SCO. Tratado de Fonoaudiologia. São Paulo: Roca; 2004. p. 772-86.
2. Roggia SM. O processamento temporal em crianças com distúrbio fonológico [tese]. São Paulo: Faculdade de Medicina da Universidade de São Paulo; 2003.
3. Advíncula KP, Frizzo ACF, Costa EG, Santos PAG, Griz S. Estudo dos potenciais evocados auditivos de longa latência em crianças com desvios fonológicos. In: 20º Encontro Internacional de Audiologia; 2005; São Paulo. Anais. São Paulo; 2005.
4. Leite RA. Avaliação eletrofisiológica da audição em crianças com distúrbio fonológico pré e pós terapia fonoaudiológica [dissertação]. São Paulo: Faculdade de Medicina, Universidade de São Paulo; 2006.
5. Andrade CRF, Béfi-Lopes DM, Fernandes FDM, Wertzner HF. ABFW teste de linguagem infantil nas áreas de fonologia, vocabulário, fluência e pragmática. Carapicuíba: Pró-Fono; 2000.
6. Shriberg LD, Austin D, Lewis BA et al. The percentage of consonants corrects (PCC) metric: extensions and reliability data. *J Speech Lang Hear Res.* 1997;40:708-22.
7. McPherson DL. Late potentials of the auditory system (evoked potentials). San Diego: Singular Press; 1996.
8. Alonso R, Schochat E. A eficácia do treinamento auditivo formal em crianças com transtorno de processamento auditivo (central): avaliação comportamental e eletrofisiológica. *Braz J Otorhinolaryngol.* 2009;75(5):726-32.

9. Hodson BW, Paden EP. Targeting intelligible speech: a phonological approach to remediation. 2ª ed. Texas: Austin; 1991. p. 190.
10. Tonnquist-Uhlen I. Topography of auditory evoked long-latency potentials in children with severe language impairment: the P2 and N2 components. *Ear Hear.* 1996;17:314-26.
11. Bishop DVM, McArthur GM. Individual differences in auditory processing in specific language impairment: a follow-up study using event-related potentials and behavioural thresholds. *Cortex.* 2005;41:327-41.
12. McArthur GM, Bishop DVM. Which people with Specific Language Impairment have auditory processing deficits. *Cogn Neuropsychol.* 2004;21(1):79-94.
13. Jirsa RE. The clinical utility of the P3 AERP in children with auditory processing disorders. *J Speech Hear Res.* 1992;35:903-12.
14. Tremblay K, Kraus N, Carrell TD, McGee T. Central auditory system plasticity: generalization to novel stimuli following listening training. *J Acoust Soc Am.* 1997;102:3762-73.
15. Tremblay K, Kraus N, McGee T, Ponton C, Otis B. Central auditory plasticity: changes in the N1-P2 complex after speech-sound training. *Ear Hear.* 2001;22:79-90.
16. Nicol T, Kraus N. How can neural encoding and perception of speech be improved?. In: Syka J, Merzenich MM (eds), *Plasticity and signal representation in the auditory system.* Kluwer Plenum, New York; 2005. p. 259-70.
17. Grafman J. Conceptualizing functional neuroplasticity. *J Commun Disord.* 2000;33:346-56.
18. Hall JW, Grose JH, Buss E, Dev MB, Drake AF, Pillsbury. The effect of otitis media with effusion on perceptual masking. *Arch Otolaryngol Head Neck Surg.* 2003;129:1056-62.
19. Gravel JS, Roberts JE, Roush J, Grose J, Besing J, Burchinal M, et al. Early otitis media with effusion, hearing loss, and auditory processes at school age. *Ear Hear.* 2006;27:353-68.
20. Moore DR. Auditory processing disorders: acquisition and treatment. *J Commun Disord.* 2007;40:295-304.
21. Shriberg LD, Friel-Patti S, Flipsen Jr P, Brown RL. Otitis media, fluctuant hearing loss, and speech-language outcomes: a preliminary structural equation model. *J Speech Lang Hear Res.* 2000;43:100-20.
22. Hall III JW. Overview of auditory neurophysiology past, present, and future. In: Hall III JW. *New handbook of auditory evoked responses;* 2007. Pearson. Boston. p. 1-34.