

Auditory evoked potentials: predicting speech therapy outcomes in children with phonological disorders

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OBJECTIVES: This study investigated whether neurophysiologic responses (auditory evoked potentials) differ between typically developed children and children with phonological disorders and whether these responses are modified in children with phonological disorders after speech therapy.

METHODS: The participants included 24 typically developing children (Control Group, mean age: eight years and ten months) and 23 children clinically diagnosed with phonological disorders (Study Group, mean age: eight years and eleven months). Additionally, 12 study group children were enrolled in speech therapy (Study Group 1), and 11 were not enrolled in speech therapy (Study Group 2). The subjects were submitted to the following procedures: conventional audiological, auditory brainstem response, auditory middle-latency response, and P300 assessments. All participants presented with normal hearing thresholds. The study group 1 subjects were reassessed after 12 speech therapy sessions, and the study group 2 subjects were reassessed 3 months after the initial assessment. Electrophysiological results were compared between the groups.

RESULTS: Latency differences were observed between the groups (the control and study groups) regarding the auditory brainstem response and the P300 tests. Additionally, the P300 responses improved in the study group 1 children after speech therapy.

CONCLUSION: The findings suggest that children with phonological disorders have impaired auditory brainstem and cortical region pathways that may benefit from speech therapy.

KEYWORDS: Central Auditory Pathway Disorders; Auditory Brainstem Evoked Response; Auditory Evoked Potentials; P300 Event-Related Potentials; Articulation Disorders.

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INTRODUCTION

Phonological disorders are the most common speech and language impairments in children, with a prevalence of 8 to 9% (1). Auditory evoked potential (AEP) investigations in children with phonological disorders have shown that these children have altered auditory brainstem responses (ABR) (2) and P300 potentials (3), suggesting the involvement of the central auditory pathway.

Motivated by an interest in identifying auditory processing deficits in children with speech and language disorders

and in monitoring the rehabilitation process, AEPs have been used to assess the auditory pathway in children with language impairments. In fact, many authors have emphasized the importance of using AEPs to promote comprehension of the underlying language impairment neurophysiology and to identify the many changes that occur along the central auditory pathway, some of which are not clinically manifested (4,5).

The AEPs provide information about the neural activity and functional integrity of the auditory pathway in response to an acoustic signal. They are classified as early, middle, and late, according to response latencies (6).

The ABR test is a noninvasive objective test that registers the responses generated by the synchronous firing of structures in the nuclei along the auditory brainstem. This potential is composed of a set of seven waves that appear from 0 to 10 ms after acoustic stimulus presentation (7). The auditory middle-latency response (AMLR) test is composed of a set of positive and negative waves (Na, Pa, Nb, Pb, Nc,

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and Pc) that follow the ABR and are present until 80 ms after acoustic stimulus presentation (8). The generators of these waves include the auditory structures of the middle brainstem, reticular formation, sensory nuclei of the thalamus, and the primary auditory cortex (9). The P300 cognitive potential is an endogenous long-latency auditory evoked potential that appears approximately 300 ms after acoustic stimulus presentation (10). P300 generators are relatively diffuse across the brain and include the auditory cortex, centroparietal cortex, hippocampus, and the frontal cortex (11).

Studies have shown that speech sound perceptions can be modified with training (12-14). Additionally, considering that AEP changes occur before behavioral sound perception improvements, it is possible, through electrophysiologic recordings, to evaluate modifications that occur after speech therapy by practicing a skill or by frequent stimulus exposure due to neuronal plasticity.

Therefore, the aims of the present study were to investigate whether neurophysiologic responses (AEPs) differ between typically developing children and children with phonological disorders and whether these responses are modified in children with phonological disorders after speech therapy.

■ MATERIALS AND METHODS

Study design

This prospective research was developed in the Department of Communication Sciences and Disorders Auditory Evoked Potential and Phonology laboratories at the Universidade de São Paulo School of Medicine (FMUSP). The project was approved by the institutional Research Ethics Committee (CAPPesq - HC FMUSP) under protocol number 236/03.

Participants

The participants were 47 native Portuguese-speaking children (19 male and 28 female) aged between 8 and 11 years (mean age = eight years and 11 months) who had normal bilateral hearing (pure tone thresholds ≤ 15 dB HL for octaves 250-8000 Hz) and type A tympanometry curves (15). The parents or legal guardians of the children signed a free and informed consent form, which allowed participation in the study. The subjects were divided into control (CG) and study groups (SG). The CG comprised 24 typically developing children (five male and 19 female), with a mean age of eight years and 11 months. The SG comprised 23 children with phonological disorders (14 male and nine female), with a mean age of eight years and 10 months. This group was divided into two subgroups: SG1, which was composed of 12 children who were enrolled in speech therapy sessions, and SG2, which was composed of 11 children who were not enrolled in speech therapy but were on the Phonology Lab outpatient care waiting list. Subjects from both subgroups were given an initial electrophysiological evaluation and a second electrophysiological evaluation after three months. One child from SG1 abandoned the speech therapy and was not reassessed, and two children from SG2 did not return for their second evaluation.

The SG subjects were referred for electrophysiological assessments by the Phonology Lab and were diagnosed with phonological disorders prior to inclusion in the study. The CG subjects were referred by an elementary school.

Additionally, the CG subjects were selected based on the following criteria: no language and speech disorder complaints, no previous enrollment in a speech therapy session, adequate performance in phonology and fluency (16) tests, and adequate performance in reading and writing screening tests.

Stimuli and recording parameters

The children were tested in a soundproof booth. Five silver electrodes were used for the AEP recordings (impedance < 5 k Ω), which were placed according to the International 10-20 Electrode System (IES) (17). Trials in which artifacts were measured in excess of ± 45 μ V were rejected from the averaged response calculations. The stimulus was delivered by a PC-based stimulus delivery system (Traveler Express; Bio-logic Systems Corp., Mundelein, IL, USA) using supra-aural earphones (TDH-39).

For the ABR test, a 19.1 clicks per second rate with a 0.1 ms duration was used with a 12 dB/octave filter slope, 100-3000 Hz band pass filter, and 2000 sweeps. Stimuli were presented at 80 dBnHL. The ABR recordings were duplicated for reliability. The peaks were marked, and their latencies (waves I, III, and V) and interpeak latencies (I-III, III-V, and I-V) were analyzed.

AMLRs were elicited by click stimuli (rarefaction polarity with a 0.1 ms duration) presented at 70 dBnHL at a 9.9 clicks/s rate. Additionally, a 10-150 Hz band pass filter was used to collect 1000 sweeps. Peaks were marked, and their latencies (waves Na and Pa) and amplitudes (Na-Pa) were calculated.

The P300 testing was performed using tone burst stimuli (alternating polarity, plat 30.00, and a 10.0 rise/fall) at 75 dBnHL and a 1.1 stimuli per second rate. The frequent (80%) stimulus was a 1000 Hz tone, and the rare (20%) stimulus was a 1500 Hz tone. Frequent and rare stimuli were randomized so there was no predictable sequence during the test. A 1-30 Hz band pass filter was used to record 300 stimuli. Each subject was instructed to identify the rare stimuli by raising a hand every time it appeared. The subjects responses to frequent and rare events were time-locked, sorted, and averaged separately. The peak was selected, and its latency (P300) was calculated.

Therapeutic model

The therapeutic model used in the Phonology Lab was adapted from the cycles approach proposed by Hodson and Paden (1991) (18). This approach predicts that phonological acquisition in children with phonological disorders will be gradual, as in typically developing children. During the 12-week cycle, some phonological processes were selected, and minimal pairs to each target sound were presented in weekly sessions. The target phonological process selected was usually the one with the greatest occurrence. Additionally, target sound articulation training was performed at the beginning of each session, along with hearing training, which involved delivering the target sound through an earphone.

Data collection and analyses

Speech and language tests

All subjects were tested using the ABFW Child Language Test, which is a standardized speech and language performance measurement (16). The test was standardized



for Brazilian Portuguese and is commonly used for diagnostics in the Phonology Lab. For the CG, phonology and fluency subtests were used. For the SG, vocabulary, pragmatics, phonology, and fluency subtests were used. Additionally, other tests were used to analyze continuous speech, oral motor skills, and phonological processing. The speech samples collected were recorded and later transcribed and analyzed.

Electrophysiological evaluations

All electrophysiological evaluation procedures were performed on the same day to avoid interference. After the first electrophysiological evaluation, which included ABR, AMLR, and P300, the SG1 children were admitted to a speech therapy program, which consisted of 12 weekly 45-minute therapy sessions. The established session number represented the period of best progress with the least amount of absences and therefore involved a greater number of consecutive sessions. Children from this subgroup underwent a second electrophysiological evaluation after 12 speech therapy sessions (three months). The SG2 children were also given a second electrophysiological evaluation after a three-month period to ensure that any AEP result improvements observed in the SG1 children were due to neuronal plasticity rather than to maturational aspects.

One child from SG1 abandoned speech therapy and was not reassessed, and two children from SG2 did not return for their second evaluation.

Experienced evaluators manually marked the waves I, III, and V ABR latencies, the AMLR Na-Pa amplitude, and the P300 latency. The evaluators were blinded to the subjects' identities and diagnostic categories. The mean, median, and standard deviation (SD) values were obtained for the ABR, AMLR, and P300 parameters for both the CG and SG groups. The measures were compared between groups.

Statistical analysis

The ANOVA test was used for the latency and amplitude measurement statistical analyses. Differences between the group and subgroup responses were considered significant when $p \leq 0.05$.

■ RESULTS

Auditory brainstem response (ABR)

No significant differences were observed between the right and left ears in either group regarding the latency values for waves I, III, and V and interpeaks I-III, III-V, and I-V. Thus, the mean latency values (absolute and interpeak intervals) for each group were determined and compared between the groups.

The means, medians, and SD for each wave and the interpeak values for both groups are displayed in Table 1. Statistical analysis showed that the latency values for wave III ($p=0.022$) and interpeaks I-III and I-V ($p=0.003$ and $p=0.015$, respectively) obtained at the first electrophysiological evaluation were significantly longer in the SG compared with the CG.

The ABR latency values recorded in the first and second electrophysiological evaluations for subgroups SG1 and SG2 are displayed in Table 2. Statistical analyses demonstrated that the wave I latency in SG1 was significantly shorter in the first electrophysiological evaluation in comparison with the second evaluation. In SG2, no significant latency value differences were observed between the two evaluations (Table 2).

Auditory middle latency response (AMLR)

There were no group differences for AMLR regarding the Na-Pa amplitudes measured during the first electrophysiological evaluation (Table 3).

There were also no significant differences observed within the subgroups (SG1 and SG2) between the first and

Table 1 - Control (CG) and study (SG) group comparisons regarding the wave I, III, and V and I-III, III-V, and I-V interpeak ABR latencies.

ABR		Mean	Median	SD	Mini	Max	Size	IL	SL	p-value
I	CG	1.50	1.48	0.09	1.32	1.84	48	1.48	1.53	0.405
	SG	1.49	1.48	0.08	1.32	1.68	46	1.47	1.51	
III	CG	3.61	3.60	0.11	3.40	3.80	48	3.58	3.64	0.022*
	SG	3.68	3.64	0.16	3.44	4.48	46	3.63	3.72	
V	CG	5.59	5.60	0.12	5.40	5.8	48	5.55	5.62	0.091
	SG	5.64	5.60	0.19	5.28	6.48	46	5.59	5.70	
I - III	CG	2.11	2.12	0.12	1.84	2.32	48	2.07	2.14	0.003*
	SG	2.19	2.16	0.14	2.00	2.88	46	2.15	2.23	
III - V	CG	1.98	1.96	0.07	1.84	2.16	48	1.96	2.00	0.480
	SG	1.97	1.96	0.08	1.80	2.16	46	1.94	1.99	
I - V	CG	4.08	4.10	0.11	3.84	4.28	48	4.05	4.11	0.015*
	SG	4.15	4.16	0.18	3.88	4.88	46	4.10	4.20	

CG = Control Group.

SG = Study Group.

ABR = Auditory Brainstem Response.

*Statistically significant values ($p < 0.05$).

SD = Standard Deviation.

Mini = Minimum.

Max = Maximum.

IL = Inferior Limit.

SL = Superior Limit.

**Table 2** - The comparisons between the first and second evaluations regarding the wave I, III, and V and I-III, III-V, and I-V interpeak ABR latencies for subgroups SG1 and SG2.

ABR		SG1			SG2		
		Mean	Median	p-value	Mean	Median	p-value
I	1st Eval	1.49	1.48	0.018*	1.47	1.48	0.657
	2nd Eval	1.56	1.52		1.46	1.48	
III	1st Eval	3.70	3.64	0.973	3.64	3.64	0.739
	2nd Eval	3.70	3.64		3.66	3.66	
V	1st Eval	5.68	5.68	0.909	5.61	5.60	0.913
	2nd Eval	5.67	5.60		5.61	5.60	
I - III	1st Eval	2.21	2.18	0.361	2.17	2.18	0.507
	2nd Eval	2.16	2.16		2.20	2.20	
III - V	1st Eval	1.98	1.96	0.973	1.96	1.96	0.670
	2nd Eval	1.98	2.00		1.96	2.00	
I - V	1st Eval	4.19	4.18	0.432	4.14	4.16	0.646
	2nd Eval	4.13	4.12		4.15	4.16	

ABR = Auditory Brainstem Response.

*Statistically significant values ($p < 0.05$).

1st Eval = 1st evaluation.

2nd Eval = 2nd evaluation.

second electrophysiological recorded measurements (Table 4).

Cognitive potential (P300)

No significant differences were observed between the right and left ears in either group regarding the P300 wave latencies. The mean latency was therefore determined for each group for comparisons between groups.

The P300 latency mean, median, and SD values for both groups (CG and SG) are displayed in Table 5. Statistical analysis demonstrated that the wave latency recorded in the first electrophysiological evaluation was significantly longer in the SG than in the CG.

The comparison between P300 wave latency results measured in the first and second electrophysiological evaluations demonstrated significant differences only in SG1, whereby shorter latency values were observed after speech therapy. There was no significant difference between the first and second evaluations in SG2, however (Table 6).

DISCUSSION

Auditory brainstem response (ABR)

In the ABR latency measurements (wave III and I-III and I-V interpeaks), the SG children presented longer latencies compared with their controls, which suggested an encoding delay at the cochlear nucleus region for the SG (Table 1). These results corroborate previous literature findings that demonstrated that children with phonological disorders have longer ABR wave latencies (waves I, III, and V) compared with typically developing individuals (2). This observation supports our hypothesis that these subjects present abnormal neural synchrony at the brainstem level.

Some studies have demonstrated different results when comparing click ABR results between normal children and those diagnosed with language disorders (i.e., lower amplitudes for waves I, III, and V compared with the control group) (19), learning difficulties (i.e., shorter latencies for wave V and interpeak III-V than the control group) (20), and specific language impairments (i.e.,

Table 3 - The control (CG) and study (SG) group comparisons regarding the AMLR Na-Pa amplitudes in the C3/A1, C4/A1, C3/A2, and C4/A2 modalities.

	C3/A1		C4/A1		C3/A2		C4/A2	
	CG	SG	CG	SG	CG	SG	CG	SG
Mean	1.83	2.17	2.08	1.99	1.96	1.91	2.51	2.15
Median	1.59	1.65	1.77	1.75	1.19	1.25	1.54	1.76
Standard Deviation	1.01	2.07	1.44	1.36	2.36	1.90	3.19	2.14
Minimum	0.78	0.36	0.45	0.22	0.27	0.23	0.30	0.20
Maximum	4.78	10.69	6.60	6.76	10.11	8.63	13.46	11.06
Size	24	23	24	23	24	23	24	23
Inferior Limit	1.43	1.32	1.50	1.44	1.01	1.13	1.23	1.27
Superior Limit	2.24	3.01	2.66	2.55	2.90	2.69	3.78	3.02
p-value	0.480		0.832		0.938		0.656	

A1 = left mastoid.

A2 = right mastoid.

C3 = left temporo-parietal junction.

C4 = right temporo-parietal junction.

CG = Control Group.

SG = Study Group.

**Table 4** - The comparisons between the first and second evaluations regarding the AMLR Na-Pa amplitude for subgroups SG1 and SG2 in the C3/A1, C4/A1, C3/A2, and C4/A2 modalities.

		C3/A1		C4/A1		C3/A2		C4/A2	
		1st eval	2nd eval	1st eval	2nd eval	1st eval	2nd eval	1st eval	2nd eval
Mean	SG1	2.03	2.77	2.02	2.71	2.02	2.59	1.71	2.78
	SG2	2.63	1.82	2.35	1.72	1.96	2.13	2.63	1.77
Median	SG1	1.64	1.76	2.24	2.2	1.22	1.13	1.69	1.54
	SG2	1.94	1.8	2.09	1.6	1.59	1.14	1.31	1.74
Standard Deviation	SG1	1.11	3.50	0.68	3.48	2.27	3.87	0.76	4.23
	SG2	3.11	0.89	1.94	0.89	1.81	1.88	3.36	1.27
Minimum	SG1	0.91	0.46	1.05	0.33	0.66	0.25	0.2	0.78
	SG2	0.36	0.75	0.22	0.56	0.23	0.41	0.32	0.69
Maximum	SG1	4.84	12.98	3.45	12.96	8.63	13.62	2.72	15.39
	SG2	10.69	3.33	6.76	2.86	5.96	6.18	11.06	4.63
Size	SG1	11	11	11	11	11	11	11	11
	SG2	9	9	9	9	9	9	9	9
Inferior Limit	SG1	1.37	0.70	1.62	0.65	0.68	0.30	1.26	0.28
	SG2	0.60	1.24	1.09	1.13	0.77	0.90	0.43	0.94
Superior Limit	SG1	2.68	4.84	2.42	4.77	3.36	4.87	2.16	5.27
	SG2	4.66	2.40	3.62	2.30	3.14	3.36	4.83	2.61
p-value	SG1	0.357		0.554		0.351		0.476	
	SG2	0.360		0.215		0.517		0.289	

A1 = left mastoid.

A2 = right mastoid.

C3 = left temporo-parietal junction.

C4 = right temporo-parietal junction.

increased waves III and V latencies compared with the control group) (21). Among these findings, longer latencies and reduced wave amplitudes were more frequently observed. Such findings show that children with phonological disorders present lengthening of ABR waves' latencies more often than typically developing children, suggesting different brainstem auditory pathway functions. We emphasize that the wave III and the I-III and I-V interpeak latencies in children with phonological disorders are within the normal range.

The comparison between ABR waves and interpeak latencies obtained before and after the speech therapy sessions verified a significant difference only for the SG1 wave I latency. In contrast, no differences were observed in SG2 for the variables analyzed (Table 2). Notably, the wave I latency was within the normal limits in both assessments for all SG1 individuals. It is believed, therefore, that this ABR difference pre and post therapy in the SG1 can actually be considered a normal latency lengthening and should not be regarded as a change due to stimulation.

Table 5 - The control (CG) and study (SG) group comparisons regarding the P300 latencies in the first electrophysiological evaluation.

P300	Control Group	Study Group
Mean	341.29	373.20
Median	330.00	382.00
Standard Deviation	46.68	59.50
Minimum	282.00	266.00
Maximum	484.00	464.00
Size	48	45
Inferior Limit	328.09	355.82
Superior Limit	354.50	390.58
p-value	0.005*	

*Statistically significant values ($p < 0.05$).

P300 = Cognitive Potential.

Auditory middle latency response (AMLR)

There was no significant differences between groups regarding the obtained AMLR results. These results, however, do not corroborate the literature findings, which report that children with speech and language disorders or learning difficulties present altered AMLR amplitudes (19,22) and Pa latency values (23).

A potential limitation of the present study is that the subjects were not given auditory processing behavioral assessments. It is possible, therefore, that the CG children also presented with auditory processing disorders, which could have been responsible for the AMLR result alterations.

Considering the mean age of both groups, the altered AMLR results might have been due to maturation in the auditory pathway (24,25). AMLR alterations in children younger than 10 years old do not necessarily indicate abnormality because the Na and Pa waves are not fully formed until 10-12 years of age (25). As previously mentioned, the adult AMLR standard is reached between 8 and 10 years of age (24); therefore, findings obtained in younger children should be carefully interpreted. Thus, caution should be used in AMLR data evaluations for diagnostic purposes in children entering the maturation phase.

Cognitive potential (P300)

Several studies have employed different long-latency auditory evoked potential types to evaluate children with language disorders, often finding latency and amplitude alterations (8,19,22,26,27). Our results indicated that children with phonological disorders have abnormal auditory pathway neural coding. These results corroborate literature findings that reported longer latencies and decreased P300 amplitudes in children with phonological disorders (3). Another study observed that this population presented increased P300 latencies and decreased N2 component

**Table 6** - The comparisons between the first and second evaluations regarding the P300 latencies for subgroups SG1 and SG2.

P300	SG1		SG2	
	1st evaluations	2nd evaluations	1st evaluations	2nd evaluations
Mean	394.73	361.82	349.65	358.00
Median	407	363	330.00	336.00
Standard Deviation	54.24	37.66	60.68	54.94
Minimum	306	300	266.00	280.00
Maximum	464	430	442.00	472.00
Size	22	22	17	17
Inferior Limit	372.06	346.08	320.80	331.88
Superior Limit	417.39	377.56	378.49	384.12
p-value	0.024*		0.677	

*Statistically significant values ($p < 0.05$).

P300 = Cognitive Potential.

amplitudes, suggesting that neurophysiologic functioning in these children differs from that of typically developing children (27).

Nevertheless, other authors observed no significant Mismatch Negativity differences between children with and without phonological disorders. Notably, this evaluation also tests long latency auditory-evoked potentials (28). Additionally, another study reported altered results in children with learning difficulties (22). The authors of the latter study suggested that these alterations could be related to auditory discrimination difficulties that are due to a central auditory pathway deficit and therefore do not depend on the child's voluntary response or attention.

Another study indicated correlations between the temporal aspects of brainstem-evoked responses and cortical asymmetry for the processing of speech sounds (29). These results reinforced the idea that the timing deficits reflected by electrophysiological brainstem measurements could affect cortical acoustic information processing.

In the present research, as in the others studies, increased latency values were the most frequent P300 alteration observed in children with language disorders (8,30).

Cortical auditory processing may be modified by several stimuli types, and these modifications can be quantified by AEP measures (31). Our results indicated modifications in the cortical auditory region, as evidenced by decreased P300 values only in the children who received speech therapy. Likewise, a previous study reported P300 wave latency improvements in children with central auditory processing disorders that received therapeutic interventions (32).

The P300 modifications observed after speech therapy can also be attributed to the neuronal plasticity phenomenon (33). According to the literature, three plasticity types can occur in the auditory pathway, including developmental plasticity, compensatory plasticity (resulting from an injury to the auditory pathway), and learning-related plasticity (14). The shorter P300 latency observed only in the group given speech therapy suggests that the plasticity type evidenced in the present study was learning-related plasticity.

Our P300 findings corroborate other studies (12,34) that suggest that speech perception skills can be improved by training and that post-training improvements may be assessed by measuring auditory evoked potentials (12). Several perceptual learning studies have shown that auditory behavioral training improves auditory discrimination skills

due to the expansion of cortical representations to the stimuli used during training (34).

The present study results indicate that children with phonological disorders present longer P300 latencies compared with typically developing children. This suggests that the cortical auditory pathway functions differently in children with phonological disorders and that they can benefit from speech therapy to improve their auditory abilities.

The Auditory Brainstem Response (ABR) and P300 results in the present study suggest that children with phonological disorders have auditory pathway impairments in the brainstem and cortical regions. Furthermore, the P300 potentials may improve with speech therapy.

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■ AUTHOR CONTRIBUTIONS

Leite RA, Matas CG, and Magliaro FC participated in manuscript conception, design, data analysis and interpretation, drafting, and final approval of the published version. Leite RA, Matas CG, Magliaro FC, Gonçalves IC, and Wertzner HF participated in drafting the manuscript and critically revising it for important intellectual content. Leite RA participated in data acquisition. Additionally, we confirm that all authors sufficiently participated in the conception and design of the work, data analysis, and manuscript writing, and they take public responsibility for it.

■ REFERENCES

1. Law J, Boyle J, Harris F, Harkness A, Nye C. Prevalence and natural history of primary speech and language delay: findings from a systematic review of the literature. *Int J Lang Commun Disord*. 2000;35(2):165-88.
2. Gonçalves IC, Wertzner HF, Samelli AG, Matas CG. Speech and non-speech processing in children with phonological disorders: an electrophysiological study. *Clinics*. 2011;66(2):293-8. <http://dx.doi.org/10.1590/S1807-59322011000200019>.
3. Leite RA, Wertzner HF, Matas CG. Long latency auditory evoked potentials in children with phonological disorder. *Pró-Fono Revista de Atualização Científica*. 2010;22(4):561-6. <http://dx.doi.org/10.1590/S0104-56872010000400034>.
4. Wible B, Nicol T, Kraus N. Correlation between brainstem and cortical auditory processes in normal and language-impaired children. *Brain*. 2005;128(2):471-23.
5. Abrams D, Kraus N. Auditory pathway representation of speech sound in humans. In: Katz J, Hood L, Burkard R, Medwetsky L (Ed). *Handbook of Clinical Audiology*. Baltimore: Lippincott. Williams & Wilkins; 2009. p.611-26.



6. Picton TW, Hillyard AS, Kraus HI, Galambos R. Human auditory evoked potentials. *Electroencephalogr Clin Neurophysiol*. 1974;36(2):179-90, [http://dx.doi.org/10.1016/0013-4694\(74\)90155-2](http://dx.doi.org/10.1016/0013-4694(74)90155-2).
7. Durrant JD, Ferraro JA. Potenciais auditivos evocados de curta latência: eletrococleografia e audiometria de tronco encefálico. In: Musiek FE, Rintelmann WF. *Perspectivas atuais em avaliação auditiva*. São Paulo: Manole; 2001. p.193-238.
8. Musiek FE, Lee WW. Potenciais auditivos de média e longa latência. In: Musiek FE & Rintelmann WF. *Perspectivas Atuais em Avaliação Auditiva*. Barueri: Manole; 2001. p.239-67.
9. Kraus N, Kileny P, McGee T. Potenciais auditivos evocados de média latência (MLR). In: Katz J. *Tratado de Audiologia Clínica* 4ª ed. São Paulo: Manole; 1999. p.384-402.
10. Sutton S, Braren M, Zubin J, John ER. Evoked potential correlates of stimulus uncertainty. *Science*. 1965;150(3700):1187-8, <http://dx.doi.org/10.1126/science.150.3700.1187>.
11. McPherson DL. *Late potentials of the auditory system (evoked potentials)*. San Diego: Singular Press; 1996.
12. Kraus N. Speech sound perception, neurophysiology, and plasticity. *Int J Pediatr Otorhinolaryngol*. 1999;47(2):123-9, [http://dx.doi.org/10.1016/S0165-5876\(98\)00130-X](http://dx.doi.org/10.1016/S0165-5876(98)00130-X).
13. Kraus N. Auditory pathway encoding and neural plasticity in children with learning problems. *Audiol Neurotol*. 2001;6(4):221-7, <http://dx.doi.org/10.1159/000046837>.
14. Musiek FE, Shinn JMS, Hare CMA. Plasticity, auditory training, and auditory processing disorders. *Semin Hear*. 2002;23:265-75, <http://dx.doi.org/10.1055/s-2002-35862>.
15. Loyd LL, Kaplan H. *Audiometric interpretation: a manual of basic audiometry*. Baltimore: University Park Press; 1978.
16. Andrade CR, Béfi-Lopes DM, Fernandes FDM, Wertzner HF. ABFW teste de linguagem infantil nas áreas de fonologia, vocabulário, fluência e pragmática. Carapicuíba. Pró-Fono; 2000.
17. Jasper HH. The ten-twenty electrode system of the international federation. *Electroencephalogr Clin Neurophysiol*. 1958;10:371-5.
18. Hodson BW, Paden EP. Targeting intelligible speech: a phonological approach to remediation. 2ª ed. Texas: Austin; 1991. p.190.
19. Mason SM, Mellor DH. Brain-stem, middle latency and late cortical evoked potentials in children with speech and language disorders. *Electroencephalogr Clin Neurophysiol*. 1984;59:297-309, [http://dx.doi.org/10.1016/0168-5597\(84\)90047-9](http://dx.doi.org/10.1016/0168-5597(84)90047-9).
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;13(7):367-82.
21. Basu M, Krishnan A, Weber-Fox C. Brainstem correlates of temporal auditory processing in children with specific language impairment. *Developmental Science*. 2010;13(1):77-91, <http://dx.doi.org/10.1111/j.1467-7687.2009.00849.x>.
22. Kraus N, McGee TJ, Carrel TD, Zecker SG, Nicol TG, Koch DB. Auditory Neurophysiologic responses and discrimination deficits in children with learning problems. *Science*. 1996;273(5277):971-3, <http://dx.doi.org/10.1126/science.273.5277.971>.
23. Arehole S, Augustine L, Simadri R. Middle latency response in children with learning disabilities: preliminary findings. *J Commun Disord*. 1995;28(1):21-38, [http://dx.doi.org/10.1016/0021-9924\(94\)00004-J](http://dx.doi.org/10.1016/0021-9924(94)00004-J).
24. Hall III JW. *Handbook of auditory evoked potential*. Boston: Allyn and Bacon; 1992.
25. Musiek FE, Gollegly KM. Maturation considerations in the neuroaudiological evaluation of children. In: Bess H. Bess editor. *Hearing impairment in children*. Maryland: York Press; 1988. p.230-50.
26. Musiek FE, Bornstein S. Contemporary aspects of diagnostic audiology. *Am J Otolaryngol*. 1992;13(1):23-33.
27. 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.
28. Roggia SM. *O processamento temporal em crianças com distúrbio fonológico [tese]*. São Paulo: Faculdade de Medicina da Universidade de São Paulo; 2003.
29. Abrams DA, Nicol T, Zecker SG, Kraus N. Auditory brainstem timing predicts cerebral asymmetry for speech. *J Neurosci*. 2006;26(43):11131-7, <http://dx.doi.org/10.1523/JNEUROSCI.2744-06.2006>.
30. 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(4):314-26, <http://dx.doi.org/10.1097/00003446-199608000-00003>.
31. Gilbert CD. Learning and receptive field plasticity. *Proc Natl Acad Sci U S A*. 1996;93(20):10546-47, <http://dx.doi.org/10.1073/pnas.93.20.10546>.
32. Jirsa RE. The clinical utility of the P3 AERP in children with auditory processing disorders. *J Speech Hear Res*. 1992;35(4):903-12.
33. Grafman J. Conceptualizing functional neuroplasticity. *J Commun Disord*. 2000;33(4):345-55; quiz 355-6, [http://dx.doi.org/10.1016/S0021-9924\(00\)00030-7](http://dx.doi.org/10.1016/S0021-9924(00)00030-7).
34. King AJ. Auditory perception: does practice make perfect? *Curr Biol*. 1999;9(4):143-6, [http://dx.doi.org/10.1016/S0960-9822\(99\)80084-0](http://dx.doi.org/10.1016/S0960-9822(99)80084-0).