Original Article

https://doi.org/10.1590/2317-6431-2024-2982en

Audiology Communication Research
ISSN 2317-6431

Cortical auditory evoked potentials in children with speech sound disorder: characterization and reference intervals

Potenciais evocados auditivos corticais em crianças com transtorno dos sons da fala: caracterização e intervalos de referência

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ABSTRACT

Purpose: To characterize cortical auditory evoked potentials in children with speech sound disorders, to determine whether there are differences between the results of children with speech sound disorders and those with typical development, and to present reference ranges for the observed values. **Methods:** This is a cross-sectional, observational, and analytical study involving 40 assessments (20 in the Control Group and 20 in the Study Group). The latencies and amplitudes of the P1N1P2N2 complex were analyzed in both groups. Comparisons of these values between groups were made, and reference intervals were established. **Results:** No statistically significant differences were found in the P1N1P2N2 latency and amplitude values between the study and control groups. However, children with speech sound disorders exhibited increased latencies in all analyzed components. **Conclusion:** The analysis of cortical auditory evoked potentials in children with speech sound disorders and typical development showed adequate morphology of the P1, N1, P2, and N2 waves, allowing for the establishment of reference values for both groups. Although no statistically significant differences were observed between the children with speech sound disorders and typically developing children, the former group exhibited increased latencies across all components of the cortical potential.

Keywords: Child; Evoked potentials; Auditory; Speech sound disorder; Electrophysiology; Brain stem

RESUMO

Objetivo: caracterizar os potenciais evocados auditivos corticais em crianças com transtorno dos sons da fala, verificar se existe diferença entre os resultados de crianças com transtorno dos sons da fala e com desenvolvimento típico, bem como apresentar os intervalos de referência dos valores encontrados. **Método**: trata-se de um estudo transversal, observacional e analítico, no qual foram realizadas 40 avaliações (20 no grupo-controle e 20 no grupo de estudo). Foram analisadas as latências e amplitudes do complexo P1 N1 P2 N2 para os dois grupos, realizadas comparações dos valores entre os grupos e estabelecidos os intervalos de referência. **Resultados**: não foi encontrada diferença estatística nos valores de latência e amplitude de P1 N1 P2 N2 entre os grupos. Porém, as crianças com transtorno dos sons da fala apresentaram latências aumentadas em todos os componentes analisados. **Conclusão**: a análise dos potenciais evocados auditivos corticais em crianças com transtorno dos sons da fala e com desenvolvimento típico mostrou ondas P1 N1 P2 e N2 com morfologia adequada, o que possibilitou o estabelecimento de valores de referência para ambos os grupos. As crianças com transtorno dos sons da fala, embora não tenham apresentado diferenças estatisticamente significativas das crianças típicas, apresentaram aumento das latências em todos os componentes do potencial cortical.

Palavras-chave: Criança; Potenciais evocados auditivos; Transtorno fonológico; Eletrofisiologia; Tronco encefálico

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Conflict of interests: No.

Authors' contribution: LGGS: lead researcher, research design, schedule development, literature survey, data collection and analysis, article writing, submission and procedures; TNUS and RCCC: co-supervisors, research design, schedule development, data analysis, and article writing review; KVTC: collaborator, article writing review; IRSC: collaborator, literature survey; KCLA: collaborator, literature survey, article writing review; PLM: collaborator, article writing review; NLBS: collaborator, literature survey, data collection and analysis; ATLC: supervisor, research design, schedule development, data analysis, article writing review, approval of the final version.

Funding: Fundação de Amparo e Pesquisa de Alagoas (FAPEAL), process number E:01700.0000002734/2021.

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Received: September 05, 2024; **Accepted:** March 07, 2025

INTRODUCTION

Speech sound disorder (SSD) is the most common communication disorder in children. Consequently, it is responsible for the greatest demand for speech therapy services. Children with SSD may present impairments in different neurodevelopmental substrates, such as auditory and somatosensory representation and transcoding, involving planning, programming, and motor execution[1].

SSD may therefore be associated with difficulties in motor production or in the perception and phonological representation of sounds and speech segments[2]. The most common type of SSD does not present impairment in the motor plan of speech, but rather changes in the auditory-perceptual and cognitive-linguistic levels, compromising auditory and somatosensory representation and particularly impairing the phonological component[1,3].

Thus, the integrity of auditory structures and functions is an important predictor for oral language acquisition. Auditory changes regarding sound reception or processing can impair speech development[4].

The peripheral auditory system receives, conducts, and amplifies the sound signal, which is sent to the central auditory pathways to be encoded. Then the auditory system integrates with the language system in the auditory cortex to make auditory stimulation functional for the individual[5].

The central auditory pathways can be assessed with behavioral and electrophysiological examinations. The latter offers a distinct advantage in that they do not rely on the patient's active response. Thus, responses are elicited independently of the person's will, facilitating the process for young children[6,7].

The cortical auditory evoked potential (CAEP), which occurs between 50 and 300 ms, is an electrophysiological test with positive and negative polarity peaks, forming the P1, N1, P2, and N2 complex[8,9]. Its generating sites have a complex location and encompass thalamic regions and the auditory cortex[10].

Studies have investigated CAEP in children with SSD due to the importance of auditory perception in forming and organizing sound representation for its recognition[2-11], although with conflicting results. Therefore, it is still necessary to carry out research characterizing CAEP in children with SSD and establishing reference intervals to understand how cortical sound processing occurs in this population.

Therefore, this study aimed to characterize CAEP in children with SSD to verify whether their results differ from those of typically developing children and to present the resulting reference ranges of values.

METHODS

This research was submitted for evaluation by the Research Ethics Committee of the State University of Health Sciences of Alagoas (CEP – UNCISAL) and approved under number 3.472.675. The study was developed at the institution's Hearing and Technology Laboratory (LATEC).

The sample was defined by convenience, with children treated at UNCISAL's Specialized Rehabilitation Center. They were selected based on the inclusion and exclusion criteria, allocating children with SSD in the study group (SG) and same-age children with typical speech and language development, able to perform the research procedures, in the control group (CG). They were recruited by contacting the parents/guardians, who signed an informed consent form, stating their consent, and ensuring the children's voluntary participation. The participating children received clear and accessible explanations about the research procedures and objectives and signed an informed assent form.

The sample size was calculated by comparing the means, considering the following statistical parameters: 0.05 significance level (α), 90% test power (1-β), 0.5 standard deviation, and 0.8 minimum detectable difference between groups. Hence, 20 participants were defined for each group, totaling 40 children.

The SG comprised 20 children diagnosed with phonological SSD, and the CG had 20 children with typical speech and language development. The groups were matched by age and sex to ensure comparability. The groups' makeup aimed to minimize biases, providing greater robustness to the statistical analyses.

The inclusion criteria for SG and CG individuals were as follows: pure-tone hearing thresholds within the normal range (up to 20 dB HL in octaves from 250 to 8000 Hz)[12]; normal visual inspection of the external auditory meatus; type "A" tympanograms[13]; contralateral and ipsilateral acoustic reflexes for all frequencies evaluated (500, 1000, 2000, and 4000 Hz); absolute and interpeak latencies for waves I, III, and V within normal range bilaterally[14]; diagnosis of SSD for the SG, and absence of speech changes for the CG.

The exclusion criteria were any ear changes, history of ear surgeries, more than three ear infections in the previous year, diagnosis of auditory neuropathy, cochleovestibular changes, and reports of possible cognitive or behavioral changes in the medical history survey.

Sampling was carried out for convenience, considering the inclusion and exclusion criteria. The selected children and their parents/guardians were duly informed about the research procedures.

All participating children underwent speech-language-hearing therapy with the same frequency and therapeutic approach, ensuring uniform intervention. However, the number of sessions performed up to the time of the evaluation varied among participants, due to interruptions and adjustments in routines caused by the COVID-19 pandemic. This variability was considered during the analysis of the results, being identified and documented as a relevant factor in the research, especially due to the potential influence of health restrictions and social distancing on individual therapeutic progress.

Procedures

The parents/guardians were interviewed during the study to collect information about the children's general development. The children also underwent phonological assessment, analyzing phonological processes with the Child Language Test – ABFW – Part A: Phonology[15] to identify phonological SSD in the SG and the absence of speech changes in the CG. The speech samples were recorded on video, using a digital camera integrated into the iPhone 12 Pro, positioned 20 centimeters from the child.

Next, the following were performed: inspection of the external auditory meatus with a Heine Mini 3000 otoscope; basic audiological assessment with pure-tone audiometry using an Interacoustics AD629b audiometer; and immittance measurements using an Interacoustics AT235 immittance meter.

The ipsilateral and contralateral reflex thresholds were investigated with eliciting stimuli of 500, 1000, 2000, and 4000 Hz.

The electrophysiological assessment used a two-channel Navigator Pro – Bio-logic® device with four electrodes. Participants were comfortably positioned in a reclining chair for adequate muscle relaxation. Disc-type electrodes were applied to the skin after preparation with abrasive paste (NUPREP®), in the following locations: M1 and M2 (references), Cz (active), and Fz (ground), according to the SI 10-20 system[16]. The assessments were performed with EAR-Phones 3A insert earphones and started when the impedance of the electrodes connected to the skin was below 3 kΩ and the difference between the electrodes was below 1.5 kΩ.

Brainstem auditory evoked potential (BAEP) was performed to assess the neural integrity of the auditory pathway. CAEPs were then obtained by presenting 750 monaural stimuli (right side), consisting of 150 infrequent and 600 frequent sounds, at 70 dB HL, within a 533 ms window, including 100 ms of pre-stimulation. CAEPs were assessed at 70 dB because the literature widely uses and describes this intensity, ensuring robust and consistent auditory responses. This level optimizes the signal-to-noise ratio and minimizes individual variations in auditory sensitivity, providing greater reproducibility and validity of the findings.

The stimulus rate was maintained at 1.7 stimuli per second, with filters from 0.1 to 30 Hz and alternating polarity. An oddball paradigm was used with pairs of natural speech verbal stimuli (/ga/ - frequent, /da/ - rare), developed by the researchers and presented with an 80/20 probability of occurrence per stimulus. The stimulus spectral characteristics are detailed in Chart 1. An overview of the parameters used to capture CAEPs is presented in Chart 2.

CAEPs were analyzed using the latencies and amplitudes of the P1, N1, P2, and N2 peaks. Two experienced researchers in the field analyzed all tracings independently to ensure the reliability of the results.

Chart 1. Frequency and time analysis of the stimuli used

Parameters		Stimuli	
		/ga/	/da/
Duration		156.71 ms	116.33 ms
VOT (Voice Onset Time)		-118.677 ms	-96.588 ms
F0	Mean	162.78 Hz	170.77 Hz
	Minimum	149.36 Hz	161.05 Hz
	Maximum	175.73 Hz	183.46 Hz
	Median	163.15 Hz	169.20 Hz
	Standard deviation	9.45 Hz	8.31 Hz
Formants	F1	477.83 Hz	821.44 Hz
	F2	2049.08 Hz	1519.26 Hz
	F3	2588.44 Hz	2686.89 Hz
	F4	3826.87 Hz	3663.64 Hz

Subtitle: ms (milliseconds); Hz (Hertz); F0 (fundamental frequency)
Source: The authors (2022)

Chart 2. Parameters used to capture cortical auditory evoked potentials

Transducer	Insert	
Polarity	Alternating	
Intensity	70 dB HL	
Presentation	Monaural	
Stimuli	Verbal stimuli (/ga/ x /da/) developed by the researchers, presented casually, with an 80/20 probability of occurrence per stimulus, according to the oddball paradigm.	
Replicability	Twice	
Electrode positioning	Reference	M1 and M2
	Active	Cz
	Ground	Fz
Impedance of the electrodes	≤ 3 kΩ	
Impedance between electrodes	≤ 1.5 kΩ	
Band-pass filter	High pass	0.1 Hz
	Low pass	30 Hz
Capture window	533 ms	
Number of mediations	750 stimuli (150 rare and 600 frequent)	
Stimulation rate	1.7 stimuli/second	
Artifact rejection threshold	10% of the total recorded	
Gain	50000 µV	

Subtitle: ms (milliseconds); kΩ (Kiloohm); Hz (Hertz); µV (microvolts); ≤ = less than or equal to
Source: The authors (2022)

Data statistics analysis

The t-test for independent samples compared continuous variables after confirming the homogeneity of the residual variances with the Levene test. The 95% confidence interval (CI) of the means and the values associated with the 5th and 95th percentiles of the distribution were calculated to determine a possible reference interval for the values. All analyses used an alpha value equal to 5% and the statistical package SPSS v23.0 (IBM Inc., Chicago, IL).

RESULTS

The sample consisted of 40 participants, comprising the GC (n = 20) and SG (n = 20).

The results were assessed and analyzed exclusively in the right ear, totaling 40 records. Table 1 details the characteristics of the sample.

Altogether, 65% of the sample were males, and 50% were 5 years old. The most frequent severity was moderate-mild (60%), and the phonological processes with the highest occurrence were simplification of consonant clusters, simplification of liquids, and simplification of final consonants.

The intergroup comparison of P1, N1, P2, and N2 latencies and amplitudes showed the presence of the N1, P2, and N2 waves in the tracing of all exams, with similar intrasubject morphology in both scans. However, two SG participants did not present P1. The groups' responses did not differ statistically significantly, although the SG latencies were higher in all CAEP components (Table 2).

The data found for P1, N1, P2, and N2 were used to determine reference values for both groups (Table 3).

DISCUSSION

This research aimed to characterize CAEPs in children with SSD, compare the results with typically developing children, and establish the reference ranges of values.

The P1, N1, P2, and N2 components were observed with adequate morphology. The two groups were not statistically significantly different regarding the latency and amplitude of the recordings. However, the SG had greater latencies in all CAEP components than the CG. These findings may indicate a delay in the auditory information processing speed and a lack of auditory integration and attention to verbal stimuli[2].

Authors reported higher values for long latency potentials in children with SSD when assessed with verbal stimuli[11]. These findings may indicate that neural coding in these children with SSD differs from that of typically developing children and that, if stimulated, these latency and amplitude recordings may improve. This indicates that such tests may be useful indicators for monitoring neural plasticity in this population[17]. Research with long-latency auditory evoked potentials (LLAEP) observed the ability to monitor neural plasticity in this population, including cortical and cognitive analysis in children with SSD. After 3 months of intervention, the study found a trend of improvement in the LLAEP results in children with SSD[11].

However, not all studies found increased latency in children with SSD – one observed the opposite result, with a higher latency in the CG[18]. This research was conducted with pure tone, and the authors found that the P2 latency was higher in the CG than in the group with SSD. They reported that this may have occurred due to the variety of risk factors possibly related to SSD[18].

Other researchers reinforce this finding[19]. They performed a similar assessment with 20 normal hearing children (10 with

Table 1. Characterization of the study group regarding sex, age, severity of the phonological disorder, and phonological processes

Variables		Study Group	
		N	%
SEX	Males	13	65
	Females	7	35
AGE	4 years	4	20
	5 years	10	50
	6 years	4	20
	7 years	2	10
	Mean	5.25	
SEVERITY OF THE PHONOLOGICAL DISORDER	Mild	5	25
	Mild-Moderate	12	60
	Moderate-Severe	2	10
	Severe	1	5
PHONOLOGICAL PROCESSES	Consonant Cluster Reduction	20	100
	Liquid Reduction	16	80
	Final Consonant Simplification	12	60
	Velar Fronting	5	25
	Palatal Fronting	4	20
	Fricative Stopping	4	20
	Fricative Devoicing	4	20
	Plosive Devoicing	3	15
	Palatal Backing	1	5

Subtitle: N = number of participants; % = percentage

Table 2. Intergroup comparison of cortical auditory evoked potentials in the time domain

Variables	Groups						p-value*
	Control Group (n = 20)			Study group (n = 20)			
Latencies	**N**	**Mean**	**SD**	**N**	**Mean**	**SD**	
P1	20	68.84	15.99	18	74.43	11.58	0.22
N1	20	87.93	22.83	20	101.80	26.78	0.08
P2	20	122.53	19.00	20	128.70	23.89	0.37
N2	20	237.32	35.40	20	253.14	26.98	0.12
Amplitudes							
P1	20	2.35	1.88	18	3.02	2.37	0.33
N1	20	1.48	1.73	20	1.46	2.02	0.97
P2	20	3.79	1.36	20	3.41	1.80	0.46
N2	20	-4.26	1.22	20	-4.40	1.81	0.77

*Student's t-test
Subtitle: N = number of participants; % = percentage; SD = standard deviation

Table 3. Reference intervals for analysis of cortical auditory evoked potentials in the time domain for the P1, N1, P2, and N2 peaks regarding latencies and amplitudes

Variables		Intervals	
		Control Group (n = 20)	
Latency	**Mean**	**95% CI**	**p5; p95**
P1	68.84	61.35; 76.32	50.05; 97.31
N1	87.93	77.25; 98.61	59.77; 149.40
P2	122.53	113.63; 131.42	98.06; 177.45
N2	237.32	220.75; 253.88	165.67; 287.40
Amplitude			
P1	2.34	1.46; 3.22	-0.74; 5.80
N1	1.48	0.66; 2.28	-1.49; 3.98
P2	3.79	3.15; 4.42	1.29; 6.21
N2	4.26	-4.82; -3.68	-6.58; -2.39
		Study Group (n = 20)	
Latency	**Mean**	**95% CI**	**p5; p95**
P1	74.43	68.67; 80.19	56.08; 95.57
N1	101.80	92.16; 118.08	65.31; 153.56
P2	128.70	118.59; 142.67	100.69; 175.60
N2	253.14	244.64; 268.69	192.94; 293.88
Amplitude			
P1	3.01	1.83; 4.19	-1.25; 7.02
N1	1.48	0.47; 2.49	-3.09; 4.27
P2	3.23	2.43; 4.03	0.83; 7.38
N2	4.40	-5.19; -3.46	-7.80; -1.28

Subtitle: n = number of participants; P = percentile; CI = confidence interval

SSD and 10 in the CG), aged 7 to 14 years, of both sexes. They used pure-tone stimuli, and the children were matched by age and sex. The authors found higher N2 latencies in CG and reported that these data may indicate that children with SSD have different neurophysiological behavior than typically developing children. Moreover, the study's sample size did not allow generalizations to the population[19].

These results may be explained by the fact that children with SSD have different temporal processing patterns from those of typically developing children. They may have a poorer perception of intervals between sounds and be less likely to provide correct answers in tests that depend on good temporal sequencing[20].

Similar data were found when analyzing these potentials with pure tone in typically developing children of both sexes, aged 4 to 14 years, who underwent 42 evaluations. The latencies were within the study's confidence interval in the age range of 4 to 7 years[21].

The different results between studies may be due to cortical auditory responses being highly dependent on stimulation characteristics. Thus, studies may have different findings due to the diversity of protocols for capturing cortical potentials since latency time and amplitude depend on the type of stimulus and its intrinsic characteristics[22].

When investigating the differences in stimuli in long-latency potentials in children with SSD, it was found that the combination of stimuli can interfere with the latency and amplitude of the potentials. This may be justified in that the central auditory system may discriminate the processing of some phonological contrasts more easily than others[22,23].

This reinforces the idea that the stimulus type and characteristics can directly reflect the evocation of these potentials. It is estimated that vowels and consonants are processed in different nervous regions, depending on the degree of contrast of the consonants and vowels used. Thus, the latencies and amplitudes of these components can vary considerably, leading to differences in the findings between studies[24].

Shorter stimuli produce potentials with higher amplitudes and lower latencies. When analyzing stimuli lasting 100 and 500 ms, it was observed that the shorter ones produced P1 and N2 with higher amplitudes and lower latencies. However, stimulus duration is not the only issue – high-frequency stimuli generate potentials with lower amplitudes and higher latencies. This is due to the tonotopic neural organization of the auditory cortex, in which more superficial neuronal regions respond better to low-frequency sounds, which are closer to the electrodes that capture the response[25].

It is believed that the stimuli used in this study had lower frequency and shorter duration, leading to better latency and amplitude results, except for N1, which had a positive peak in both groups. This finding may have occurred because N1 is sensitive to changes in duration, as different neurons in the auditory cortex respond differently to temporal changes in the stimulus. Thus, some nerve regions have different refractoriness times and may interfere with the action potential generated[25]. Another explanation is that 750 prolonged mediations and evoked stimuli were performed, which may change the baseline, and the results may appear more positive or more negative than expected[26].

The N1 component, normally negative, may present a positive peak due to technical, physiological, neurophysiological, or artifactual factors. Among the technical aspects, the configuration of the reference electrode, recorded polarity, inadequate filters, or extensive averaging may alter the expected polarity[25]. Physiologically, individual variations, P1 interference by temporal overlaps, and nonspecific neural activity can modify the shape of the N1. Neurophysiologically, low-frequency or short-duration stimuli can cause atypical responses. Artifacts such as muscle movements and baseline fluctuations also interfere with recordings[26].

Limitations and further research

The limitations of the research include the challenges in controlling the time that the children were exposed to therapy. As some children had more severe SSD than others, they consequently required longer therapy that included auditory stimulation, which may have influenced the auditory cortical results.

Hence, two main strategies were used to minimize bias related to variability in the children's number of therapy sessions: 1) documenting in detail the number of sessions completed by each participant up to the time of assessment, including factors that could influence this frequency (e.g., difficulties in attending healthcare during the social isolation imposed by the COVID-19 pandemic). This approach contextualized the data and considered such variations in subsequent analyses; 2) ensuring the greatest possible homogeneity in the sample, selecting children whose number of sessions was as similar as possible to reduce initial variability and ensure greater comparability between the groups. These measures strengthened the reliability of the results and mitigated potential interferences resulting from differences in therapeutic exposure.

The study could not analyze the association between the severity of phonological disorder and CAEP findings due to the limitations imposed by the COVID-19 pandemic, which directly impacted participant recruitment. The insufficient sample size compromised the statistical robustness needed to explore this relationship, highlighting the need for further studies with greater sample representation to clarify this issue.

CONCLUSION

CAEP analysis in children with SSD and typically developing ones showed P1, N1, P2, and N2 waves with adequate morphology and similar latencies, establishing reference intervals for both groups. Children with SSD had CAEP latencies and amplitudes similar to those of typically developing children.

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