Forward masking in cortical auditory evoked potentials with speech stimuli in children with attention deficit hyperactivity disorder

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INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD)is a neurodevelopmental disorder characterized by difficulties in self-control, including problems with attention, impulse control, and physical and mental activity levels[1]. Historically seen as a childhood condition, ADHD is now recognized as a disorder that persists into adulthood. Prevalence rates vary widely, ranging from 6% to over 30%[2]. Children with ADHD often experience executive function impairments, such as response inhibition and behavior self-regulation[3].

Diagnosing ADHD in adults is a complex issue with ongoing debate regarding its prevalence from childhood. Some researchers suggest that hyperactivity symptoms decrease in adulthood, while inattention remains more persistent[4]. Experts believe that ADHD is underdiagnosed in adults[5], contributing to the complexities of understanding its full impact across the lifespan.

Recent research into ADHD's etiology suggests widespread anomalies in multiple brain structures, including the prefrontal cortex, basal ganglia, and cerebellum[6]. Studies utilizing brain imaging techniques have revealed decreased neural activity in the frontal cortex, anterior cingulate cortex, and basal ganglia in individuals with ADHD[7]. Given the close relationship between auditory processing abilities and ADHD, objective and behavioral auditory assessments are increasingly being used to understand the neural underpinnings of this disorder and its impact on communication and learning[8].

The aim of this study was to analyze the effect of post-masking on components P1, N1 and P2 of cortical auditory evoked potential responses with speech stimulus from the condition without noise and with noise in children diagnosed with attention deficit hyperactivity disorder.

METHODS

Inclusion and Exclusion Criteria

This study included 32 children aged 7 to 11 years, divided into a Control Group (CG) of typically developing children and a Study Group (SG) of children diagnosed with ADHD. The CG participants had normal auditory thresholds, type A tympanograms, and no complaints of central auditory processing disorder (CAPD). The SG included children with ADHD and academic difficulties, some with CAPD symptoms. Exclusion criteria for both groups included ear disorders, noise exposure, ear surgery, cognitive deficits, and use of ototoxic medications.

Main Pre-Exams

Pre-exams included cognitive assessment with the Raven's Colored Progressive Matrices and audiological exams such as otoscopy, pure-tone audiometry, tympanometry, stapedial reflex testing, and auditory brainstem response (ABR).

Forward masking **protocol in cortical auditory evoked potentials (CAEP)**

Test Condition	Stimulus	Stimulus Features	Presentation Rate	Recording Window	Electrodes
Without masking	Syllable /ba/	70 dB HL, 40 ms duration	0.7 stimuli/sec	799.5 ms	Ref.: M2, Active: Cz, Ground: Fpz
With masking	Syllable /ba/	/ba/ (70 dB) + noise	3 ms after noise onset	799.5 ms	Same as above
Number of stimuli per test	140	Alternating polarity			
Noise Characteristics	Speech Shaped Noise (SSN)			70 dB HL, 250 ms duration	

RESULTS

Participants

The participants' ages ranged from 7 to 11 years, average of 8.52 (±1.672) years. Among the ADHD participants, 81.25% (13/16) exhibited a combined ADHD subtype, while 18.75% (3/16) presented with predominantly inattentive ADHD. Raven test scores showed that 68.75% of CG participants scored above average, compared to 43.75% in the SG. Auditory skill assessments in the SG revealed deficits in several areas: sound localization (12.5%), sequential verbal memory (12.5%), sequential non-verbal memory (31.2%), figure-ground association (37.5%), figure-ground processing (62.5%), temporal ordering (43.7%), and temporal resolution (68.7%).

CAEP non-masking and masking conditions

The intragroup analysis revealed significant differences in latencies between the two test conditions (presentation of the syllable /ba/ without preceding masking and presentation of the syllable /ba/ 3 ms after the masking noise at the same intensity) for all evaluated components (P1, N1, P2) in both groups. The p-values for the control group (CG) were (p= 0.001; p= 0.000; p= 0.000) and for the study group (SG) (p=0.042; p=0.002; p= 0.004), respectively. In both groups, latencies were longer when the noise preceded the speech stimulus. Amplitude differences were significant for P1 only in the CG (p=0.002) when noise preceded the stimulus.

Control group x Study group (CAEP)

Non-masking condition

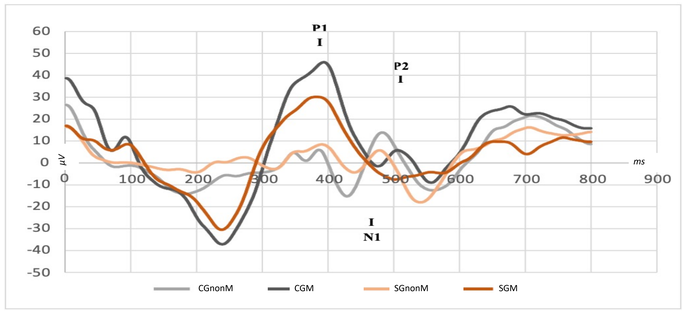
		CONTROL GROUP (N=16)		STUDY GROUP (N=16)		
		Mean	SD	Mean	SD	P-value
Latency (ms)	P1	386.2	21.8	391.8	26.9	0.501[1]
	N1	439.3	23.5	450.3	30.2	0.303[2]
	P2	487.4	32.1	503.1	40.6	0.438[2]
Amplitude (µv)	P1	2.45	2.37	1.64	2.27	0,.72[2]
	N1	-3.39	3.03	-1.58	4.04	0.049*
	P2	2.63	3.40	1.41	1.82	0.215[1]

Masking condition

		CONTROL GROUP (N=16)		STUDY GROUP (N=16)		
		Mean	SD	Mean	SD	P-value
Latency (ms)	P1	415.1	16.5	407.5.	18.4	0.347[2]
	N1	490.2	33.0	487.0	30.2	0.751[2]
	P2	561.1	52.3	548.1	35.5	0.469[1]
Amplitude (µv)	P1	6.08	3.02	4.21	2.03	0.053[2]
	N1	-1.62	3.12	-2.01	1.75	0.438[1]
	P2	2.84	2.33	0.44	1.34	0.005[2]

Descriptive analysis (mean and standard deviation) and statistical analysis of the latency and amplitude values of the P1, N1, and P2 waves for the control and study groups.

Figure 1 - Grand average of the recordings from the 32 participants, divided into control and study groups for the two test conditions.



CGnonM - control group non-masking; GCM - control group with masking; SGnonM - study group non-masking; SGM - study group with masking.

Statistical differences

Amplitude

N1: CGnonM (-3.39±3.03) x SGnonM (-1.58±4.04)[1]

P2: CGM (2.84±2.33) x SGM (0.44±1.34)[2]

1. p = 0,049; 2. p= 0,005

DISCUSSION

The sample in this study consisted of 22 boys and 10 girls, with boys being the majority. This reflects the higher prevalence of ADHD in boys, as previously reported in the literature. The intragroup analysis demonstrated significant differences in latency for the P1, N1, and P2 components between the control and study groups. These findings suggest that, despite the neurobiological alterations in children with ADHD, the forward masking effect observed indicates that this phenomenon is independent of ADHD characteristics. Longer latencies suggest delayed neural response to auditory stimuli in both groups, particularly after the masking noise was presented[9].

Regarding amplitudes, the intragroup analysis showed that only the P1 component presented a significant increase when the masking noise preceded the speech stimulus in the control group. P1 is considered a biomarker of auditory cortex activity, primarily related to sound detection. The variability in protocols for recording P1, such as stimulus intensity and type, may influence these results. However, as noise interferes with sound detection and discrimination, post-masking typically leads to a reduction in amplitude, contrary to the findings of this study[10].

Intergroup analysis revealed significant differences in the amplitudes of the N1 and P2 waves for the non-masking and masking conditions, respectively. The study group (ADHD) exhibited lower amplitudes than the control group, suggesting reduced synaptic activity in auditory processing. The N1 component, generated by the primary auditory cortex, is a key marker for auditory decoding and discrimination. Reduced N1 amplitudes, as observed in the ADHD group, indicate potential difficulties in these auditory processes[11].

Finally, reduced P2 amplitudes in the study group are associated with impaired auditory discrimination, especially when noise precedes the target stimulus. In similar studies, the simultaneous presentation of noise and speech stimuli resulted in decreased P2 amplitudes. The findings of this study provide important insights into auditory cortical processing in children with ADHD, particularly those with figure-ground difficulties, and contribute to the development of more tailored auditory rehabilitation protocols for this population.

KEY REFERENCES

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ACKNOWLEDGEMENTS

Foundation for the Support of Research of the State of Alagoas (FAPEAL) and National Council for Scientific and Technological Development (CNPq).