Literature Review

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Cortical auditory evoked potentials in individuals who stutter: a scoping review

Potencial evocado auditivo cortical em indivíduos com gagueira: uma revisão de escopo

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ABSTRACT

Purpose: To map and describe the findings regarding the performance of individuals who stutter on cortical auditory evoked potentials in the speech-language pathology clinical setting. **Research strategy**: This scoping review was conducted according to the PRISMA-ScR guidelines and registered with the OSF (https://doi.org/10.17605/OSF.IO/KGHXV). The search was conducted in PubMed, LILACS, Embase, Cochrane Library, Web of Science, Science Direct, SpeechBITE, OpenGrey.eu, and DissOnline, with no language or period restrictions. **Selection criteria**: Primary studies comparing cortical auditory evoked potentials between individuals with and without stuttering, with normal peripheral hearing, and without comorbidities were included. **Results:** A total of 1,516 studies were identified. After exclusions and screening, seven articles were included. The total sample consisted of 151 adults (18 to 46 years) and 202 children and adolescents (4 to 18 years). The findings suggest that individuals who stutter, especially those with more severe symptoms, tend to have higher latencies and lower amplitudes in the P1-N1-P2-N2 components, especially in the right ear. More marked changes were observed in children, indicating greater sensitivity of cortical auditory evoked potentials in this age group. **Conclusion**: The data suggest that cortical auditory evoked potentials may reflect alterations in central auditory processing in individuals who stutter, especially in the most severe cases.

Keywords: Electrophysiology; Auditory evoked potentials; Stuttering; Childhood-onset fluency disorder; Adults

RESUMO

Objetivo: mapear e descrever os achados a respeito do desempenho de indivíduos com gagueira, por meio dos potenciais evocados auditivos corticais, no contexto clínico fonoaudiológico. **Estratégia de pesquisa:** trata-se de uma revisão de escopo conduzida segundo as diretrizes do *Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews* (PRISMA-ScR) e registrada no *Open Science Framework* (OSF) (https://doi.org/10.17605/OSF.IO/KGHXV). A busca foi realizada nas bases PubMed, LILACS, Embase, Cochrane Library, Web of Science, Science Direct, SpeechBITE, OpenGrey.eu e DissOnline, sem restrição de idioma ou período. **Critérios de seleção:** foram incluídos estudos primários que compararam o potencial evocado auditivo cortical entre indivíduos com e sem gagueira, com audição periférica normal e sem comorbidades. **Resultados:** foram identificados 1.516 estudos. Após exclusões e triagens, sete artigos foram incluídos. A amostra total foi composta por 151 adultos (18 anos a 46 anos) e 202 crianças e adolescentes (4 anos a 18 anos). Os achados sugerem que indivíduos com gagueira, especialmente com quadros mais severos, tendem a apresentar maiores latências e menores amplitudes do complexo P1-N1-P2-N2, sobretudo na orelha direita. Alterações mais marcantes foram observadas em crianças, indicando maior sensibilidade do potencial evocado auditivo cortical nessa faixa etária. **Conclusão:** Os dados sugerem que os potenciais evocados auditivos corticais podem refletir alterações no processamento auditivo central em indivíduos com gagueira, especialmente nos casos mais severos.

Palavras-chave: Eletrofisiologia; Potencial evocado auditivo; Gagueira; Transtorno da fluência com início na infância; Adultos

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INTRODUCTION

Stuttering, a fluency disorder, is characterized by repetitions of sounds, syllables, or whole words, along with prolongations, pauses, and blocks in speech production. It typically begins in early childhood, during a critical stage of language development, and may persist into adulthood. It can also involve physical manifestations and is believed to have a genetic and neurofunctional basis[1].

The exact cause of stuttering is not fully understood, but it is believed to be associated with atypical brain activation in a wide network of premotor, motor and sensory regions, and to result from a combination of these neurological factors with genetic and environmental factors[2,3].

Some of the main theories about stuttering propose that the disorder may be caused by deficits in specific sensorimotor integration processes, essential both for early speech motor learning and for mature speech motor control[4-7]. It is noteworthy that stuttering is not caused by anxiety or nervousness, although these factors may exacerbate the symptoms[3].

Studies have shown that individuals who stutter may exhibit differences in auditory processing abilities compared to fluent speakers. For instance, they may have difficulty discriminating subtle sound differences or processing rapid auditory stimuli. These findings have led to the hypothesis that deficits in auditory processing could contribute to the development or persistence of stuttering[8,9].

Furthermore, a study that investigated pre-speech auditory modulation using cortical auditory evoked potential (CAEP) in adults who stutter and adults with fluent speech observed that slower motor preparation in individuals with stuttering may cause the modulating neural signals to reach the sensory cortex at a later point in time, closer to the onset of speech[4].

Auditory information processing is associated with the perception of temporal, rhythmic, and melodic aspects of sound, which may be challenging for individuals who stutter. These difficulties in processing auditory information have been suggested as immediate contributing factors to stuttering, particularly in more severe cases. This is because the accurate perception of speech sound timing depends on the efficient processing of auditory information. Furthermore, auditory processing is closely associated with oral language processing and therefore constitutes an important component of the overall communication process[10,11].

One possibility to objectively assess auditory processing is through CAEP. These potentials provide valuable information on how the auditory system processes sound and can be used in various clinical and research settings, complementing behavioral assessments of auditory processing[12,13].

Due to its excellent ability to measure time intervals accurately, CAEP serves as an objective method for studying how speech perception develops over time. Its high temporal resolution makes it particularly well suited for investigating the acoustic-phonetic cues of speech, providing a reliable measure for exploring the neural aspects of speech processing[14].

CAEP is composed of peaks that form the P1-N1-P2-N2 complex, which represent cortical activities related to auditory discrimination skills[15,16]. The P1 component originates from thalamic projections and the primary auditory cortex and is associated with auditory coding[17]. The N1 component, in turn, is generated from the activation of the primary auditory cortex, specifically in the lateral region of the temporal gyrus, and is also influenced by both the lateral temporal lobe and the frontal motor and premotor cortex, sensitive to initial sound changes, frequency modulations, and intensity of a complex signal or sustained tone. This component is associated with auditory attention and decoding processes[18]. The P2 component, related to auditory discrimination, results from the joint activation of the primary auditory cortex and higher cortical areas, such as the supratemporal, frontal, and parietal regions. This integrated activation enables the analysis of the acoustic and temporal characteristics of sound stimuli[17].

OBJECTIVES

This research aimed to map and describe the findings regarding the performance of individuals with stuttering, using cortical auditory evoked potentials, in the speech-language pathology clinical context, as well as to analyze the latencies and amplitudes of the P1-N1-P2-N2 complex.

RESEARCH STRATEGY

This is a scoping review of the literature and follows the recommendations of the *Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews* (PRISMA-ScR) checklist[19]. The protocol was registered with the *Open Science Framework* (OSF) under the domain https://doi.org/10.17605/OSF.IO/KGHXV .

The bibliographic research guiding the production of this review aimed to compose a diffuse mapping of the literature to establish the existing relationships between the themes of stuttering and cortical auditory evoked potentials, starting from the question: "Are there differences in CAEP latencies within the P1-N1-P2-N2 complex between individuals with and without stuttering?".

Following the recommendations in the *Joanna Briggs Institute Reviewer's Manual*[20], the search strategy was developed following three steps. The first step consisted of an initial search, shown in Chart 1, carried out in the Virtual Health Library (VHL) and PubMed databases, aiming at identifying keywords and indexing terms relevant to the investigated topic.

Following this initial assessment, a strategy was devised based on combinations of the descriptors/keywords found in the first stage and the Boolean operators.

Subsequently, in the second stage, using the combinations described in the search strategy, refined searches were performed in the MEDLINE database (PubMed), LILACS, Embase via Elsevier, Cochrane Library, SpeechBITE, Web of Science, Science Direct, and grey literature OpenGrey.eu and DissOnline. There were no restrictions regarding the publication period or language. The last search was performed on June 2nd, 2025.

In the third stage, additional searches were conducted in the references of the publications included in the review, in addition to the websites of each journal.

Chart 1. Search strategy developed for the first stage

DATABASES	STRATEGIES	RESULT
LILACS (VHL)	#1 mh:gagueira OR (Stuttering) OR (Tartamudeo) OR (Bégaiement) OR (Paralalia Literal) OR (Pselismo) OR (Tartamudez) OR mh:C10.597.606.150.500.800.750$ OR mh:C23.888.592.604.150.500.800.750$	244
	#2 mh:"potenciais evocados" OR (potenciais evocados) OR (Evoked Potentials) OR (Potenciales Evocados) OR (Potentiels évoqués) OR (Onda N1) OR (Onda N2) OR (Onda N3) OR (Onda N4) OR (Onda P2) OR (Onda P50) OR (Potenciais Evento-Relacionados) OR (Potenciais Evocados N100) OR (Potenciais Evocados N200) OR (Potenciais Evocados N300) OR (Potenciais Evocados N400) OR (Potenciais Evocados P200) OR (Potenciais Evocados P50) OR (Potenciais Evocados P600) OR (Potencial Evento-Relacionado) OR (Potencial Evocado) OR (Potencial Evocado N100) OR (Potencial Evocado P50) OR (Potencial Relacionado ao Evento) OR (Event Related Potential) OR (Event Related Potentials) OR (Event-Related Potential) OR (Event-Related Potentials) OR (Evoked Potential) OR (Evoked Potential, N100) OR (Evoked Potential, N200) OR (Evoked Potential, N300) OR (Evoked Potential, N400) OR (Evoked Potential, P200) OR (Evoked Potential, P50) OR (Evoked Potential, P600) OR (Evoked Potentials, N100) OR (Evoked Potentials, N200) OR (Evoked Potentials, N300) OR (Evoked Potentials, N400) OR (Evoked Potentials, P200) OR (Evoked Potentials, P50) OR (Evoked Potentials, P600) OR (N1 Wave) OR (N1 Waves) OR (N100 Evoked Potential) OR (N100 Evoked Potentials) OR (N2 Wave) OR (N2 Waves) OR (N200 Evoked Potential) OR (N200 Evoked Potentials) OR (N3 Wave) OR (N3 Waves) OR (N300 Evoked) OR (Potential) OR (N300 Evoked Potentials) OR (N4 Wave) OR (N4 Waves) OR (N400 Evoked Potential) OR (N400 Evoked Potentials) OR (P2 Wave) OR (P2 Waves) OR (P200 Evoked Potential) OR (P200 Evoked Potentials) OR (P50 Evoked Potential) OR (P50 Evoked Potentials) OR (P50 Wave) OR (P50 Waves) OR (P600 Evoked Potential) OR (P600 Evoked Potentials) OR (Potential, Event Related) OR (Potential, Event-Related) OR (Potential, Evoked) OR (Potentials, Event Related) OR (Potentials, Event-Related) OR (Potentials, Evoked) OR (Potentials, N400 Evoked) OR (Related Potential, Event) OR (Related Potentials, Event) OR (Wave, N1) OR (Wave, N2) OR (Wave, N3) OR (Wave, N4) OR (Wave, P2) OR (Wave, P50) OR (Waves, N1) OR (Waves, N2) OR (Waves, N3) OR (Waves, N4) OR (Waves, P2) OR(Waves, P50) OR mh:G07.265.216.500$ OR mh:G11.561.200.500$	38,977
	#3 mh:eletrofisiologia OR (Electrophysiology) OR (Electrofisiología) OR (Électrophysiologie) OR mh:H01.158.344.528$ OR mh:H01.158.782.236$	1,135
	#4 #1 AND (#2 OR #3)	237
MEDLINE (PubMed)	#1 "Stuttering"[Mesh] OR (Stuttering, Acquired) OR (Acquired Stuttering) OR (Stammering) OR (Stuttering, Childhood) OR (Childhood Stuttering) OR (Stuttering, Developmental) OR (Developmental Stuttering) OR (Stuttering, Familial Persistent 1) OR (Stuttering, Adult) OR (Adult Stuttering)	7,113
	#2 "Evoked Potentials"[Mesh] OR (Evoked Potential) OR (Potential, Evoked) OR (Potentials, Evoked) OR (Potentials, Event-Related) OR (Event-Related Potential) OR (Potential, Event-Related) OR (Event-Related Potentials) OR (Event Related Potential) OR (Event Related Potentials) OR (Potential, Event Related) OR (Potentials, Event Related) OR (Related Potential, Event) OR (Related Potentials, Event) OR (P50 Evoked Potentials) OR (P50 Evoked Potential) OR (Evoked Potential, P50) OR (Evoked Potentials, P50) OR (P50 Wave) OR (P50 Waves) OR (Wave, P50) OR (Waves, P50) OR (N100 Evoked Potentials) OR (N100 Evoked Potential) OR (Evoked Potential, N100) OR (Evoked Potentials, N100) OR (N1 Wave) OR (N1 Waves) OR (Wave, N1) OR (Waves, N1) OR (P200 Evoked Potentials) OR (Evoked Potential, P200) OR (Evoked Potentials, P200) OR (P200 Evoked Potential) OR (P2 Wave) OR (P2 Waves) OR (Wave, P2) OR (Waves, P2) OR (N200 Evoked Potentials) OR (Evoked Potential, N200) OR (Evoked Potentials, N200) OR (N200 Evoked Potential) OR (N2 Wave) OR (N2 Waves) OR (Wave, N2) OR (Waves, N2) OR (N300 Evoked Potentials) OR (Evoked Potential, N300) OR (Evoked Potentials, N300) OR (N300 Evoked Potential) OR (N3 Wave) OR (N3 Waves) OR (Wave, N3) OR (Waves, N3) OR (N400 Evoked Potentials) OR (Evoked Potential, N400) OR (Evoked Potentials, N400) OR (N400 Evoked Potential) OR (Potentials, N400 Evoked) OR (N4 Wave) OR (N4 Waves) OR (Wave, N4) OR (Waves, N4) OR (P600 Evoked Potentials) OR (Evoked Potential, P600) OR (Evoked Potentials, P600) OR (P600 Evoked Potential)	206,034
	#3 "Electrophysiology"[Mesh]	81,614
	#1 AND (#2 OR #3)	119

Caption: VHL = Virtual Health Library

SELECTION CRITERIA

To gather information on the topic, specifically regarding the search and selection of publications, the strategy using the acronym PCC (P – population, C – concept, C – context) was employed.

- Population: individuals with stuttering
- Concept: to understand CAEP findings
- Context: Scientific literature (national and international journals in the area of health)

The study types included: mapping national and international publications, encompassing case studies or reports, case-control studies, cross-sectional studies, ecological studies, cohort studies, experimental or quasi-experimental studies, observational studies (descriptive and analytical), published in databases and/or virtual libraries. Regarding the type of publication, articles from primary studies and extended abstracts were included.

Articles published in full in scientific journals, originating from original studies, that evaluated CAEP in individuals with stuttering, with bilateral peripheral hearing within normal limits and without other comorbidities, in comparison with individuals with typical speech development, were included.

Other reviews, theses and dissertations, studies with content irrelevant to the correlation between the theme, and unavailable full texts whose abstracts lacked relevant data for the analysis were excluded.

Subsequently to the survey, the identified articles were exported from the databases to the Rayyan software, developed by the Qatar Computing Research Institute (QCRI), which is a platform for exploring and filtering research studies eligible for review[21].

In this stage, the study selection followed the eligibility criteria, carried out independently by two reviewers, both for the screening stage and for the full-text reading. In case of disagreements regarding the study selection, a consensus meeting was held and, in case disagreement persisted, a third reviewer was asked to arbitrate.

The selection process was carried out in three stages: reading all the titles; reading the abstracts; and reading the articles in full.

The information from the selected studies was extracted using a form developed for this review, in accordance with Appendix 11.1 JBI template source of evidence details, characteristics and results extraction instrument[20], and based on reading a sample of articles, as a pilot test.

The extraction sheet contained the following data: principal author, year of publication and country; number of participants and age; examination protocol data (stimulus type, presentation and quantity, intensity, paradigm, electrode position); mean and standard deviation values of CAEP latency and amplitude of the P1-N1-P2-N2 complex.

Given that the literature mapping process is dynamic and interactive, the form was adjusted by consensus among the authors during the pilot test conducted with some of the selected studies to avoid omitting information relevant to the construction of the results and discussion.

It is noteworthy that, in some studies, extraction of all the information mentioned above was impossible.

Data analysis

The extracted data were subjected to descriptive analysis, using tables and figures, summarizing the steps followed, central information from the studies, interpreting and comparing the productions, and describing the available evidence regarding the correlation between the proposed themes corresponding to the guiding question and objectives of this review.

RESULTS

The complete study selection process is described in Figure 1.

The data search resulted in 1,516 studies initially included for analysis; 83 duplicates were identified and excluded. Subsequently, 1,433 studies proceeded to title and abstract reading, and 1,421 were excluded. Thus, 12 studies were selected for full-text reading, and 5 were eliminated due to methodological issues. Finally, 7 studies were used for the review.

Chart 2 presents the general characteristics of the studies, including country of origin, year of publication, main author, and data regarding the protocol used.

Three studies were conducted in Egypt[22-24], 2 in Brazil[25,26] and 2 in the United States[27,28]. Participants' age ranged from 4 to 46 years, distributed into study groups (SG), consisting of individuals with stuttering, and control groups (CG), individuals without alterations. Three studies conducted tests with adult participants[22,25,28], 3 studies with children and adolescent participants[23,24,27] and 1 with adults and children[26], totaling 151 adult individuals and 202 children and adolescents.

Regarding the used protocol, only 1 study[24] used speech stimulus. The majority (85.71%) used non-verbal stimuli, with 2 using pure tone[27,28] and the remaining 4 using tone burst stimulus[22,23,25,26]. Four studies comprising this review used the oddball paradigm (80/20)[25-28] with frequent stimulus of 1 kHz and rare stimulus of 2 kHz[25,27,28] and frequent stimulus of 750 Hz and rare stimulus of 1 kHz[26]. The other 3 studies did not inform these parameters[22-24].

To define the degree of stuttering severity, most studies (57%) used the *Stuttering Severity Instrument* (SSI-3)[24-26,28], 1 study used the *Bloodstein Scale*[22], 1 did not report[23] and 1 used an 8-point severity scale marked by parents or a speech-language pathologist[27]. The degree of severity in the stuttering group ranged from very mild to severe.

The studies investigated CAEP component latencies and amplitudes in individuals with stuttering, with partially convergent findings. Regarding latencies, 2 studies identified a significant delay in N1 and P2 latencies in the stuttering group[22,23], although these latencies did not show a significant correlation with stuttering severity.

Conversely, 1 study[24] reported a tendency towards longer latencies and lower amplitudes of the P1-N1-P2-N2 complex in children with stuttering, especially in the right ear, with a statistically significant difference only in the group classified as *Bloodstein* severity IV. In the same study, behaviors such as blocking and repetitions were also associated with increased latency and reduced amplitude.

A study[26], which assessed children and adults, observed that, in children, there was an increase in P2 and N2 latency in the right ear and N2 latency in the left ear, compared to the control group. There was also a reduction in N2 amplitude in the experimental group. In adults, there was no significant difference in N1-P2-N2 latencies, but an increase in N2 latencies was observed in the right ear, suggesting that the alterations are more evident in children than in adults.

Some studies[25,27,28] did not identify significant differences in latency between the groups for N1 and P2. However, one study[25] found a difference between ears - the right ear showed lower latency for N1 compared to the left ear, in both groups. Another study[28] highlighted that, although not significant, N1 and P2 amplitudes were lower and more variable in the stuttering group, which may indicate a tendency towards instability in these individuals' auditory cortical response. The remaining studies analyzing amplitude[22,23] did not find statistically significant differences between the groups.

DISCUSSION

CAEP analysis in individuals with stuttering has gained prominence as a promising tool in investigating the neural

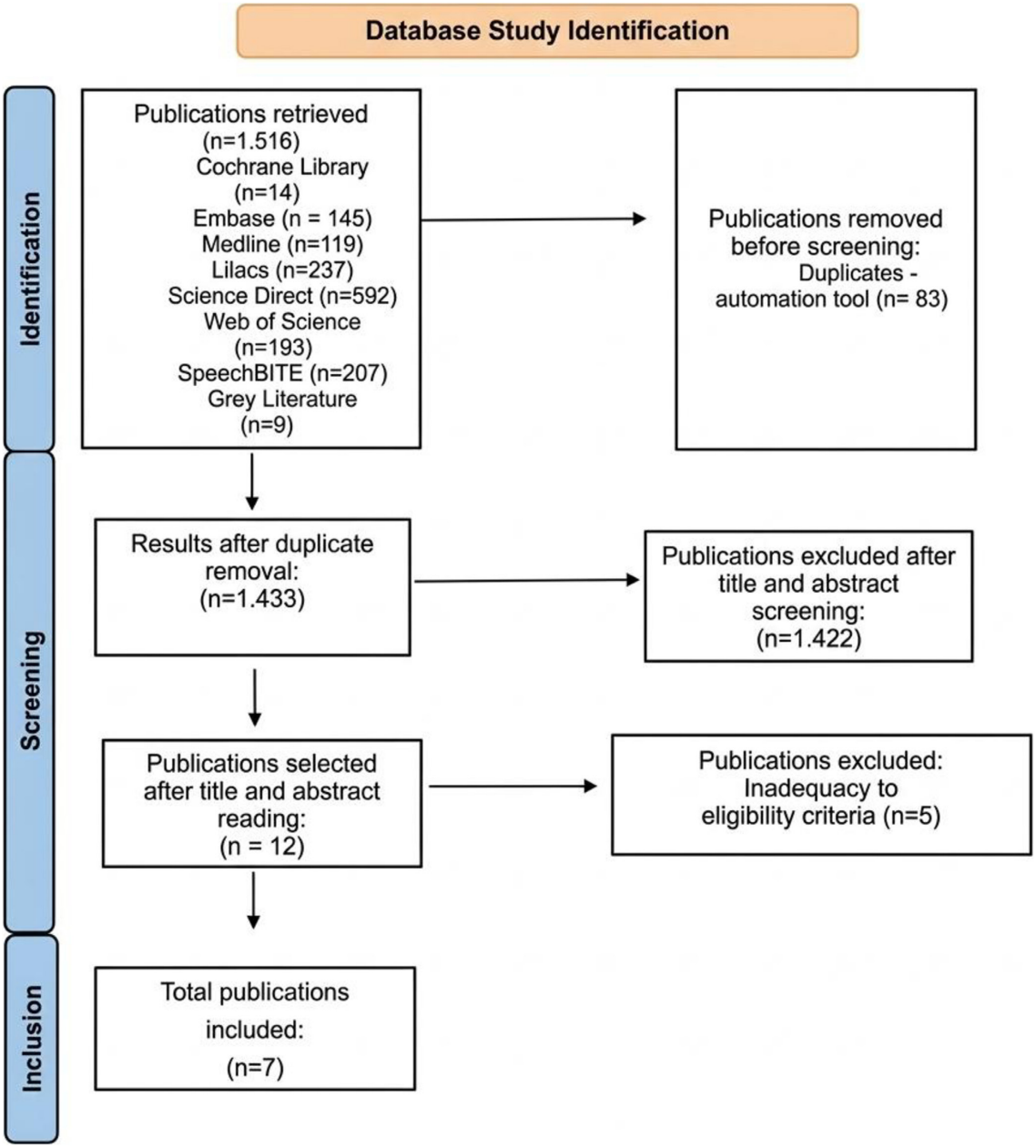

Figure 1. Preferred Diagram Reporting Items for Systematic Reviews and Meta- Analyses extension for Scoping Reviews (PRISMA) with the selection of studies

mechanisms underlying this fluency disorder. However, the results found in the literature are still heterogeneous, reflecting the multifactorial complexity of stuttering and the diversity of methods employed in research. Understanding the possible neurophysiological alterations associated with speech fluency requires considering technical and clinical aspects that directly influence the findings, such as the type of auditory stimulus used, the intensity of the presentation, participants' age, and the severity of stuttering.

Regarding the criteria for conducting the CAEP, the protocols used in the studies in this review demonstrated varied parameters. Although verbal and nonverbal stimuli can be applied, the most commonly used include clicks, tone bursts, vowels and syllables[13].

The choice of stimulus type directly influences how waves are generated, since discriminating verbal stimuli tends to be more challenging than discriminating nonverbal stimuli. This difference is reflected in the prolonged latencies and amplitudes, generally longer with speech stimuli, when compared to click-stimuli, for instance, attributed to the greater complexity of the sounds processed in the central auditory nervous system[13].

Some authors advocate the use of verbal stimuli as they provide more relevant information about speech detection and discrimination, allowing for more refined investigations into the neurological mechanisms associated with auditory perception[13-15].

Nonverbal stimuli tend to be more easily perceived when compared with verbal stimuli. Therefore, it is important to

Chart 2. General characteristics of the included studies

Author, year, country	Sample	Age, sex	Severity of stuttering	Electrode placement	Stimulus	Paradigm	Components
Khaled et al. (2023) Egypt	80 (40 CG; 40 SG)	SG 18-45 years old average 27 years 7 months 28 male participants 12 female participants CG 18-55 years old average 29 years 8 months 22 male participants 18 female participants	SSI-3: Very mild (n = 10) Mild (n = 19) Moderate (n = 11)	NI	*Tone Burst* 20 dB above the threshold Total 500 sweeps V - 1.1 per second	NI	N1, P2
Elhakeem et al. (2023) Egypt	80 (40 - CG 40 - SG)	SG 6-12 years average 8 years 17 months 29 male participants 11 female participants CG 6-12 years average 8 years 07 months 28 male participants 12 female participants	NI	Fpz; Cz Reference - tip of the nose	*Tone Burst* 60 dB Window - 630 ms	NI	P1, N1, P2, N2
Prestes et al. (2017) Brazil	41 (21 CG; 20 SG)	18-46 years old ± 30 years	SSI-3: mild to moderate	Fpz; Cz; A1 and A2	*Tone Burst* 70 dB 1 kHz (frequent), 2 kHz (rare) Total 3,000 sweeps V - 1.1 per second Window – 600 ms	*Oddball* 80/20	N1, P2, N2
Ismail et al. (2017) Egypt	60 (30 - CG 30 – SG)	8-18 years average 12 years 8 months 23 male participants 7 female participants (in both groups)	*Bloodstein* Scale: *Bloodstein I* (n = 3) *Bloodstein II* (n = 4) *Bloodstein III* (n = 16) *Bloodstein IV* (n = 7)	NI	Speech 70 dB	NI	P1, N1, P2, N2
Regaçone et al. (2015) Brazil	34 -13 CG Children and 13 SG; 4 adults CG and 4 adults SG	7–31 years	SSI-3 Very Mild	Fpz; Cz; A1 and A2	*Tone Burst* 750 Hz (frequent), 1 kHz (rare); V- 1.1 per second Window – 500 ms	*Oddball* 80/20	N1, P2, N2
Kaganovich ; Hampton; Weber-Fox (2010) USA	36 (18 CG; 18 SG)	4-5: average 4 years 10 months SG 13 male participants 5 female participants CG 12 male participants 6 female participants	The severity of the stuttering was rated as 2 or higher on an eight-point severity scale by their parents or a speech-language pathologist	32 electrodes: FZ, FCZ, CZ, CPZ, PZ, OZ; medial lateral sites FP1/FP2, F3/F4, FC3/FC4, C3/C4, CP3/CP4, P3/P4, O1/O2; and lateral sites F7/F8, FT7/FT8, T7/T8, TP7/TP8, P7/P8	Pure tone 1 kHz (frequent), and 2 kHz (rare); Total 608 sweeps Window - 1100 ms	*Oddball* 80/20	P1, N1
Hampton; Weber-Fox (2008) USA	22 (11 CG; 11 SG)	25 and 45 years old 16 male participants 6 female participants	NI	FZ, FCZ, CZ, CPZ, PZ, OZ, medial lateral sites FP1/ FP2, F3/F4, FC3/FC4, C3/ C4, CP3/CP4, P3/P4, O1/ O2 and lateral sites F7/F8, FT7/FT8, T7/ T8, TP7/TP8, P7/P8	Pure tone 1 kHz (frequent), and 2 kHz (rare) rare Total 900 sweeps	Oddball 80/20	N1, P2

Caption: CG = control group; SG = study group; SSI-3 = Stuttering Severity Instrument; A1 = left earlobe; A2 = right earlobe; V = velocity; kHz = kilohertz; dB = decibels; s = seconds; ms = milliseconds; NI = Not informed; USA = United States of America.

conduct experiments with CAEP using speech stimuli to explore the neurological mechanisms involved in speech perception[15].

Although most of the stimuli used in the studies analyzed in this review were nonverbal, there were differences in the frequencies chosen for frequent and rare stimuli. The study that used more distant frequencies[22] demonstrated the shortest latency values for N1 and P2 components in the group of individuals with stuttering, which confirms the expected notion that CAEP measures with more distant stimuli would have shorter latencies, as they are easier to be perceived as different[17].

Regarding stimuli intensity, studies have shown much variation. Only one study omitted this information[24], while others revealed intensities ranging from 20 dB above the participants' hearing threshold[22] to 90 dB. CAEP components, along with auditory potentials in general, are frequently influenced by stimulus intensity[18].

As intensity increases, latency decreases. This relationship between intensity and latency is attributed to the fact that more intense stimuli activate the auditory pathways more rapidly, resulting in a faster transmission of the signal to the auditory cortex. However, excessive intensity can also lead to a saturation of the response, where the effect of decreasing latency stabilizes or even increases[12,18].

Participants' age in the studies included in this review was heterogeneous and, despite the difference, latency values in the studies were very similar. Studies indicate that increasing age promotes more defined patterns, in which P1-N1-P2 latency values tend to decrease and show less variability[13].

Maturation of the central auditory nervous system is different across individuals, leading to variations in latency and amplitude values of the P1-N1-P2-N2 complex, which can be influenced by the type of stimulation and the stage of development. During childhood, these components' amplitude and latency values show variability. Among children, P1 and N2 components are the most commonly observed and described, while N1 and P2 begin to become visible and measurable as the maturation process progresses[13,17,29].

Studies report that N2 is a composite potential with attributes associated with exogenous and endogenous responses, important for processing and comprehension of physical and acoustic auditory information from the stimulus, and that it can be altered by intrinsic factors such as attention and sleep. This component is influenced by discrimination task, representing the activity of the supratemporal auditory cortex[12,13,18].

A difference was observed between the ears for the CAEP components[24-26], in which the right ear presented shorter latencies and higher amplitudes when compared to the left ear. Auditory processing occurs predominantly in the auditory cortex contralateral to the stimulated ear, i.e., the right ear has its main auditory processing pathway in the left hemisphere and the left ear in the right hemisphere[30]. However, the left hemisphere, responsible for complex linguistic and auditory functions, can process signals more efficiently, which would explain the observed difference in latencies and amplitudes between the ears[12].

The findings of the studies suggest that, especially in individuals with more severe stuttering, prolonged latencies and reduced amplitude of the CAEP P1-N1-P2-N2 complex occur. These findings may indicate that the early stages of auditory processing may be impaired, pointing to less neuronal synchronization and less auditory cortical activation[22].

This dysfunction can hinder accurate auditory feedback, essential for fine adjustments in speech production, thus contributing to motor instability and the typical symptoms of severe stuttering[24]. Similarly, studies conducted on adults who stutter show that they exhibit longer latencies in the N2 components, reinforcing the idea of a deficit in temporal processing and updating of sensory information[25].

This evidence supports the idea that, in more severe stuttering, the central auditory system, especially the cortical regions involved in rapid exogenous responses to stimuli, shows signs of atypical functioning[28].

These changes do not manifest uniformly in all individuals with stuttering, but appear to correlate with specific neurophysiological profiles and higher degrees of severity, suggesting that these electrophysiological markers may be indicative of subgroups with greater sensorimotor impairment within the stuttering spectrum. In general, the findings reinforce the heterogeneity of stuttering and that the more severe the condition, the more pronounced these changes in wave components tend to be[23]. Studying this wave complex provides important information about the neural processes responsible for speech perception.

The absence of statistical differences in some studies may be related to methodological variability, such as the type of auditory stimulus used, participants' age range, and the method of classifying stuttering. Even so, trends such as greater variability in amplitudes and asymmetries between ears suggest a possible instability in the auditory processing of individuals with stuttering, even when the results do not reach statistical significance. Thus, the data reinforce the importance of considering individual and clinical factors, such as the severity of stuttering, in the CAEP examination, and point to the need for further investigations with more standardized protocols and samples stratified by age and severity, in order to deepen the understanding of the neurophysiological correlations of stuttering.

Furthermore, a broader understanding of the hearing impairments of this population contributes to better therapeutic planning, which may include acoustically controlled or informal auditory training, so that improvements in auditory skills can be reflected in improved speech fluency.

Therefore, this examination represents an important tool for objectively measuring central auditory function and can serve as a marker of neuroplasticity in response to intervention. Conducting research with speech stimuli can provide important information about this population.

One limitation of this review is the lack of uniformity in research protocols and studies conducted with a heterogeneous sample, regarding stuttering severity and age, which may have hindered combined analysis.

CONCLUSION

This study has enabled CAEP mapping, characterization, and description in individuals with stuttering. Based on the publications and information presented, reported, and discussed here, it has been observed that individuals with stuttering, especially those with clinical severity, tend to present alterations in CAEP components, albeit in a heterogeneous manner. Most findings point to delayed latencies and reduced amplitudes in the P1-N1-P2-N2 complex, especially when considering factors such as age, laterality, and degree of stuttering severity.

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