



## Cortical auditory evoked potentials in children who stutter



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### ABSTRACT

**Introduction:** It has been hypothesized that impaired auditory processing influence the occurrence of stuttering. Also, it is suggested that speech perception in children who stutter differed from normal. Auditory processing should be investigated in children who stutter shortly after the onset of stuttering in order to evaluate the extent to which impaired auditory processing contributes to the development of stuttering. CAEPs provide the necessary temporal and spatial resolution to detect differences in auditory processing and the neural activity that is related or time-locked to the auditory stimulus. The primary goal of the present study was to determine the difference in latency and amplitude of P1-N2 complex between children who stutter and non-stuttering children in response to speech stimuli.

**Material & methods:** This case-control study was performed over 60 children, 30 were non-stuttering children (control group) and 30 were children who stutter (study group) ranging in severity from Bloodstien I to Bloodstien IV in the age range of 8–18 years.

**Results:** CAEPs of children who stutter with stuttering severity Bloodstien IV showed significant prolonged latencies and reduced amplitudes when blocks and IPDs were the most predominant core behaviors. P1 and N1 were prolonged in concomitant behaviors.

**Conclusion:** It could be speculated that speech processing was affected in children who stutter with stuttering severity Bloodstien IV at the level of early perceptual auditory cortex.

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### 1. Introduction

Persistent developmental stuttering is a subtype of speech fluency disorders characterized clinically by abnormal frequency or duration of interruption in the flow of speech, namely repetitions, prolongations, and/or blocks [9].

Stuttering presents in the form of overt and covert stuttering. Overt stuttering is characterized by repetitions, prolongations, blocks, and/or intraphonemic disruptions (IPDs). Covert stuttering is characterized by word substitutions and interjections which help to postpone, avoid, and hide the impact of stuttering [5]. The presence of associated concomitant problems like eye blinking, flaring nostrils, head nodding, and feet tapping indicates greater stuttering severity [4].

According to these symptoms [2], rated the severity of stuttering into four degrees; the 1st degree is when stuttering is episodic and consists only of repetitions of which the child is not aware, the 2nd

degree is when stuttering is chronic or habitual, other forms of interruptions start to appear, and the child is aware of his dysfluency, the 3rd degree is when stuttering occurs in feared situations and word substitution may be used to avoid feared word, and the 4th degree (which is the most advanced form), is when word fear and situation avoidance occur in addition to secondary reactions.

Although a variety of theories have been proposed to explain its etiology, the exact cause of stuttering is still unknown [9]. Theories of stuttering incorporate many factors like atypical auditory processing, genetics, personality, linguistic factors and atypical neurophysiology. According to [21], disturbed cerebral dominance in left handed individuals (either with right hemispheric shift or bilateral dominance with low left hemispheric activation) also contributed to the occurrence of stuttering.

The main focus in stuttering research has been on speech production, but a growing literature suggests that stuttering may also be characterized by atypical neural mechanisms underlying speech perception. Central speech sound processing is essential for speech acquisition, production and comprehension [14].

Cortical Auditory evoked potentials (CAEPs) are series of positive and negative peaks labeled P1-N1-P2-N2 occurring between

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50 and 500 ms after stimulus onset. They reflect obligatory neural events for speech representation in the central auditory system independently of the listener attention. The P1–N2 complex has been suggested to be a representation of the sensory encoding of auditory stimulus characteristics [27]. Peaks of CAEPs are generated from the auditory thalamo-cortical pathways involving both primary and association auditory cortices [19].

There were not many studies found in the literature to examine P1–N2 complex in children who stutter. Most of these studies have been done with adults using pure tone stimuli [13]. Speech auditory processing in children who stutter needs to be evaluated cautiously under variability found in neuroimaging. CAEPs proved to be effective measure for speech processing ability. Recent neurological studies have shown abnormal neural activity in the auditory areas in developmental stuttering. Magnetoencephalographic (MEG) studies have demonstrated that subjects with persistent developmental stuttering show functional and structural abnormalities in the central auditory nervous system including unusual activation patterns in the auditory areas [16].

### 1.1. Aim of work

The aim of the present study is to evaluate cortical auditory evoked potentials' (CAEPs) response in stuttering children, with variable severity of the presenting symptoms, and to compare them with non-stuttering children.

## 2. Materials and methods

This case-control study was performed in the Phoniatric and Audiology Units of AL Mansoura International Hospital, in the period from March 2014 to March 2015, with 60 children in the age range of 8–18 years. They were divided into two groups; the study group composed of 30 children who stutter, ranging in severity from Bloodstien I to Bloodstien IV, who were tested prior to any therapeutic intervention, and the control group which was composed of 30 children, all of them were non stuttering. The control group was selected from volunteers and relatives who visited the Audiology Unit and Otorhinolaryngology Clinics in the hospital.

Both groups had bilateral normal peripheral hearing sensitivity (hearing threshold level did not exceed 15 dB at any frequency of the range of 250–8000 Hz), normal middle ear function, normal general health condition and fully developed language. Any children having ear problems (as hearing impairment, otological diseases, ototoxic drug intake, ear surgery or head trauma), children complaining from systemic diseases (e.g. any endocrinal, vascular, renal, convulsions or neurological complaints) and children who received previous Phoniatric rehabilitation for stuttering or suffering from any other Phoniatric disorder were excluded.

The severity of stuttering among the study group was determined using [2] classification of severity of stuttering.

All children underwent complete phoniatric history and evaluation and otological examination. Hearing was tested using basic audiological evaluation such as pure tone audiometry, speech recognition threshold [23], speech discrimination [24], and immittanceometry. Cortical auditory evoked potentials (CAEPs) were done, using speech stimuli, by the Smart Evoked Potentials of Intelligent Hearing System.

The collected data were organized, tabulated and statistically analyzed using SPSS (Statistical Package for Social Science) version 16. Qualitative data were described using number and percent. Association between categorical variables was tested using Chi-square test. Chi-square test ( $\chi^2$ ) was used for comparison between more than two groups. Parametric analysis (student *t*-test)

was used for comparison between means of two groups only. Significance was adopted at  $p < 0.05$  for interpretation of results of tests of significance [28]. Post-hoc test was calculated for significant values after ANOVA to determine which comparisons contributed strongly to the significant values [12].

## 3. Results

Thirty children exhibited developmental stuttering were included in this study. They were classified according to the severity of stuttering into Bloodstien I (three children), Bloodstien II (four children), Bloodstien III (sixteen children) and Bloodstien IV (seven children). They were compared to thirty fluent children as a control group. Both groups are matched as regard age, gender and handedness.

Table 2 shows the frequency of occurrence of different symptoms among the study group. The predominant symptom among stutterers Bloodstien I was repetition, whereas stutterers Bloodstien II exhibited repetitions and prolongations in addition to secondary reactions. Stutterers Bloodstien III and IV displayed repetitions, prolongations, blocks, and intraphonemic disruptions in addition to secondary reactions and associated concomitant behaviors.

Children who stutter had prolonged latencies and smaller amplitudes in both ears than controls and in the right ear than the left according to absolute latencies and amplitudes of P1, N1, P2 and N2. But, none of these differences had reached a statistically significant level ( $p > 0.05$ ) as shown in Table 3 and Figs. 1–3. Because the differences between Bloodstien IV and the control group were close to reach a statistically significant level, further analysis using *t*-test was required because *t*-test is mainly designed to investigate the significance between 2 groups.

Bloodstien IV children who stutter had statistically significant prolonged latencies and smaller amplitudes of P1, N1, P2 and N2 than the control group as shown in Table 4 and Fig. 3.

Children who stutter with positive family history had prolonged latencies and smaller amplitudes in both ears than stutterers with negative family history according to absolute latencies and amplitudes of P1, N1, P2 and N2. None of these differences had reached a statistically significant level ( $p > 0.05$ ) as shown in Table 5.

Regarding absolute latencies and amplitudes of P1–N2 complex, Table 6 showed that both groups had prolonged latencies in the right ear (left hemisphere) than in the left ear (right hemisphere). Left handed stutterers had prolonged latencies and smaller amplitudes in both ears than right handed children who stutter. None of these differences had reached a statistically significant level ( $p > 0.05$ ).

Children who stutter had prolonged latencies in both ears than controls and in the right ear than in the left according to absolute latency of P1, N1, P2 and N2 when repetition and prolongation were the predominant core behaviors. Bloodstien IV had the most prolonged latency than other types. None of these differences had reached a statistically significant level ( $p > 0.05$ ).

Stutterers had statistically significant prolonged latencies ( $p < 0.05$ ) in both ears than controls and in the right ear than in the left according to absolute latency of P1, N1, P2 and N2 when blocks were the predominant core behaviors. Bloodstien IV had the most prolonged latency than other types.

Stutterers had statistically significant prolonged latencies ( $p < 0.05$ ) in both ears than controls and in the right ear than in the left according to absolute latency of P1, N1 and P2 when IPDs were the predominant core behaviors as shown in Table 7. Bloodstien IV had the most prolonged latency than other types.

Post-hoc test was done after ANOVA to determine if there was a statistically significant difference between Bloodstien IV children

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