Auditory brainstem response and speech mismatch negativity in children with phonological disorders

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Objective
This study was designed to explore the processing of auditory information through auditory brainstem and higher cortical regions in a sample of children with phonological errors compared to age-matched normal controls using AEPs.

Subjects and Methods
We recorded click and speech auditory brain-stem response and mismatch negativity in 30 children (15 children who were diagnosed clinically with phonological disorder, their ages ranged between 3.5–5.5 years, 15 children age-matched, sex matched and education matched with the study group, normal fluent speaker with no history of speech or language impairment).

Results
Absolute and inter-peak latency values of cABR demonstrated no statistically significant difference between the control and study groups. Moreover, All children had well identifiable and repeatable sABR and MMN but with delayed latencies in transient, transitional and sustained portions of speech-evoked ABR and MMN in study group when compared to control group. On the other hand, there were non significant difference as regard amplitude in the two groups as regard sABR and MMN.

Conclusion
Phonological disorder may affect the communication and language processes causing degradation of linguistic and para-linguistic information, also it can affect the quality of life and social interaction.

Keywords:
click ABR, mismatch negativity, phonological disorder, speech ABR

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Introduction

Phonology is the study of the sound system of a language, and the systemic sound changes that affect the class of sounds or sound sequences are defined as phonological processes [1].

Phonological disorder is one of the most prevalent types of communication impairment among children as it compromises the largest group of children receiving speech-language services [2]. These children make multiple errors in the articulation of specific sounds and its pattern in addition to that their speech is often unintelligible affecting their communication with their peers and teachers [3].

The speech sound disorders are classified into articulation disorders and phonological disorders. There is a big difference between articulation disorders, which pertain to the disorders that affect phonetic level, which takes care of the motor act of producing the vowel and consonant, and the phonological processes disorders, which affect the phonemic level. The phonemic level is in charge of the brainwork that goes into organizing the speech sound contrasts. These contrasts make sounds distinct from one another [4].

Language development needs normal auditory physiological processing for oral language skills, and the presence of any abnormality in the auditory brainstem responses (ABRs) may result in phonological disorders [5].

Multiple tools are present for measuring the acoustic stimuli at different levels of the auditory neural such as auditory-evoked potentials, the ABR, and mismatch negativity (MMN). The auditory-evoked potentials are widely used as convenient measures of brainstem and cortical auditory functions and pathway [6].

Processing of acoustic stimuli at different levels of the auditory-neural pathway could be examined in normal and clinical populations using an objective, noninvasive, and reliable tool, namely, the ABR, which represents synchronized neural response to brief acoustic signals from a large number of neurons through the auditory nerve and brainstem [7]. ABRs can be evoked using click as well as more complex stimuli.

Recently, speech-evoked auditory brainstem response (sABR) has been used a biological marker for brainstem neural asynchrony in children with phonological errors, as sABRs measures have been introduced as a method to study the brainstem encoding of speech sounds [6,7].

The ABR with speech stimuli can be divided into transient and sustained portions, onset response components (stimulus onset), and frequency-following response.

The onset responses are transient processes, similar to the click, with tenths of milliseconds precision. They primarily represent the response to discrete stimulus events, as during the initiation, and the successive modulations caused by the vibration of the vocal folds. The components of sustained response continue during the reproduction of a periodic stimulus and reflect the overall integrity of the response in relation to the stimulus [8].

On the contrary, MMN is an electrophysiological test that is generated bilaterally in the supra-temporal part of the auditory cortex and in the inferior frontal cortices [8].

It represents preattentive detection of a change from the active sensory memory trace of the standard stimulation. It can be elicited to a variety of the acoustic features of speech that underlie speech perception. Hence, MMN is thought to index preattentive speech discrimination [9].

This study was designed to explore the processing of auditory information through auditory brainstem and higher cortical regions in a sample of children with phonological errors compared with age-matched normal controls using AEPs. This might represent a step further in improving the clinical diagnosis and treatment of these disorders.

Patients and methods

Participants in this study were divided into two groups

(1) Study group: it consisted of 15 children, and their ages ranged between 3.5 and 5.5 years. This group was diagnosed with the phonological error.
(2) The control group: it consisted of 15 age-matched, sex-matched, and education-matched normally fluent speaking children with no history of speech and language impairments. All participants in our study had no history of neurological disorder.

This work was done in Phoniatric and Audiology Unit, ENT Department, Faculty of Medicine, Zagazig University, from April 2016 to October 2016. All participants gave their written consent before participation.

All participants in the current study were subjected to the following:

(1) Full history taking.
(2) Otological examination.
(3) Phoniatric assessment in form of the following:
    (a) Phonological analysis by speech sample of connected speech for analysis of segmental and suprasegmental level of phonology.
    (b) Standardized Arabic articulation test to identify the existing errors, the patterns, and consistencies in these errors [10].
(4) Basic audiological evaluation:
    (a) Pure tone audiometry: air and bone conduction audiometry.
    (b) Speech audiometry (two-channel audiometer Madsen Model Orbiter 922, GM Otomtrix, Denmark).
    (c) Immattancemetry including tympanogram and acoustic reflex thresholds (acoustic immittance meter Interacoustics Model AZ7, Amplifon, Italy).
(5) Auditory-evoked potentials tests.

ABR and MMN were examined in all participants using an auditory-evoked potential audiometer (model Smart EP, version 2.39; Intelligent Hearing Systems, Miami, Florida, USA).

Auditory brainstem response

Rarefaction acoustic click with a duration of $100 \, \mu s$ was used to evoke the click-evoked ABR. It was presented at an intensity of 90 dBnHL and at a rate of 19.3/s. A total of 1024 sweeps were obtained from the stimulated ear. Recordings were made with a band-pass filter of 100–1500 Hz in a time window of 10 ms.

Speech auditory brainstem response

The sABR was elicited using /ba/ stimulus. The stimulus duration was 114.875 ms, which is the default of the Smart EP. The /ba/ stimulus is characterized by having voicing onset at 10 ms and F0 (100 Hz). The formant transition duration is 50 ms and includes linearly rising and flat portions. The linearly rising portion comprises F1 (400–720 Hz), F2 (900–1240 Hz), and F3 (2400–2500 Hz), whereas the flat one includes F4 (3300 Hz), F5 (3750 Hz), and F6 (4900 Hz). Ten milliseconds of initial frication are centered at frequencies of around F4 and F5.

The /ba/ stimulus was presented in an alternating polarity at a rate of 8.42/s with an interstimulus

interval of 3.83 ms and intensity of 70 dBnHL. One thousand sweeps were collected from the right and left ears separately using a filter of 30–3000 Hz and digitized at 20 kHz. An artifact criterion of ±35 µV was applied to reject epochs that contained myogenic artifacts. Data were plotted in a time window of 10 ms before stimulus onset to 70 ms after stimulus onset.

The sABR is formed of transient, transitional, and sustained portions. The onset response of the transient portion has been analyzed for wave V and A latencies and V–A complex measures [interpeak amplitude, duration, and slope (interpeak amplitude/duration)], whereas the offset response has been analyzed for wave O latency and amplitude. The sustained portion represents the frequency following response. It was identified as negative troughs (D, E, and F) occurring every 10 ms and measured for their latency and amplitude. Wave C is a transitional negative wave between the two portions of sABR. Its amplitude and latency were also measured [11].

Mismatch negativity response
Stimuli presentation and recording speech stimuli were /wa/ as the standard and /ba/ as a deviant and were presented in an odd-ball paradigm with a probability of 80% for the standard and 20% for the deviant stimuli. A total of 250 stimuli were presented at alternating polarity, at an intensity of 80 dBnHL and a rate of 1.1/ s. Fifty sweeps from each ear were averaged apart. The analysis period was 500 ms with 50 ms prestimulus recording. Recordings were made with an amplification of 50, artifact rejection of 100 µV, and band-pass filter of 0.8–30 Hz.

Table 1 Comparison of wave latencies of click-evoked auditory brainstem response between the study (30 ears) and control group (30 ears)

Latencies (ms)	Control group	Study group	t	P
I	1.64±0.03	1.66±0.05	1.878	0.065
III	3.86±0.05	3.89±0.12	1.264	0.211
V	5.63±0.07	5.65±0.09	0.960	0.340

There were no statistically significant differences of click-evoked auditory brainstem response wave latencies in between study and control group.

Table 2 Comparison of wave interpeak latencies of click-evoked auditory brainstem response between the study (30 ears) and control group (30 ears)

Latencies (ms)	Control group	Study group	t	P
I–III	2.10±0.05	2.09±0.02	1.017	0.313
III–V	2.01±0.12	2.02±0.15	0.285	0.776
I–V	4.12±0.03	4.11±0.04	1.095	0.277

There were no statistically significant differences of wave interpeak latencies of click-evoked auditory brainstem response in between study and control group.

Statistical analysis
Independent *t*-test was used to compare quantitative variable in the two groups. Significant difference was considered when *P*=0.05 to more than 0.01, highly significant difference was considered when *P*=0.01 to more than 0.001 and very highly significant difference was considered when *P* value up to 0.001.

Results
All participant in our study had average hearing threshold not exceeding 20 dBHL in the frequency range of 250–8000 Hz, normal middle ear function, and normal speech discrimination. Statistical analysis performed on the absolute and interpeak latency values of click-evoked ABR demonstrated no statistically significant difference between the control and study groups in any of these measures (Tables 1 and 2). Moreover, all children had well-identifiable and repeatable sABR and MMN but with delayed latencies in transient, transitional, and sustained portions of sABR and MMN in the study group when compared with control group (Tables 3–5 and Figs 1–7).

Table 3 Comparison of wave latencies of transient portion of speech-evoked auditory brainstem response between the study (30 ears) and control group (30 ears)

| Latencies (ms)	Control group	Study group	t	P
V	6.55±2.72	11.33±2.82	6.682	0.000***
A	10.51±2.53	15.42±3.71	5.988	0.000***
O	60.77±3.67	64.80±1.66	5.480	0.000***

There were statistically significant differences of latencies in between study and control group with delayed latencies in study group. ***Very highly significant difference *P*=0.001.

Table 4 Comparison of wave latencies of transitional and sustained portions of speech-evoked auditory brainstem response in study (30 ears) group versus control group (30 ears)

Latencies (ms)	Control group	Study group	t	P
C	20.35±3.42	25.42±3.50	5.674	0.000***
D	28.22±4.11	35.63±4.34	6.790	0.000***
E	38.72±3.99	43.52±5.09	4.065	0.000***
F	50.98±4.82	54.63±3.79	3.260	0.001**

There were statistically significant differences of latencies in between study and control group with delayed latencies in study group. **Highly significant difference *P*=0.01. ***Very highly significant difference *P*=0.001.

Table 5 Comparison of mismatch negativity measures between the control and study groups

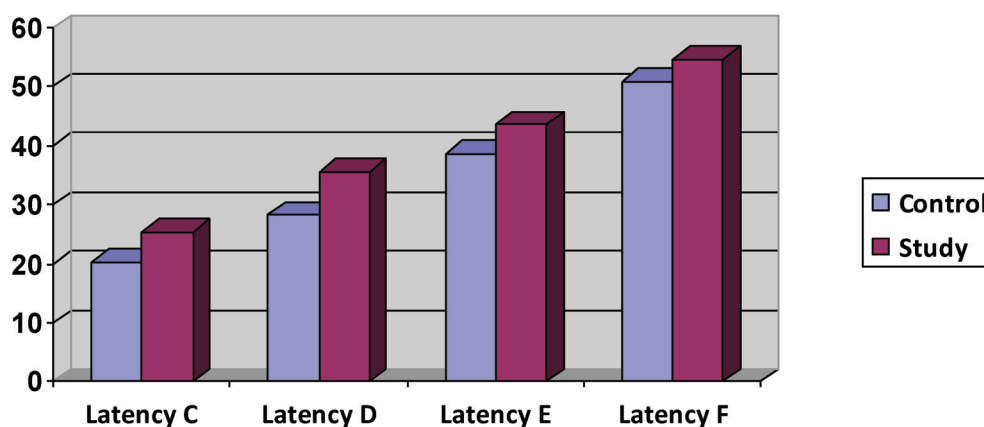|	Control group	Study group	t	P
Latency (ms)	228.33±13.2	288.76±35.3	8.782	0.000***
Amplitude (µV)	4.7±1.2	3.8±2.8	1.618	0.111
Duration (ms)	110±18	100±25.3	1.764	0.083
Area (ms×µV)	508±12.20	502±20.2	1.392	0.169

There were significant differences in latency between study and control group; on the contrary, there were no significant differences regarding amplitude, duration, and area. ***Very highly significant difference *P*=0.001.

Figure 1
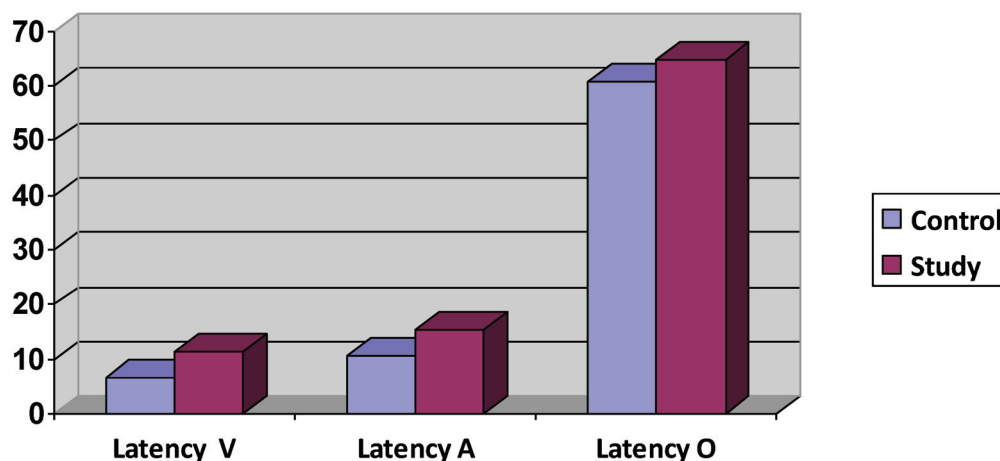
Wave latencies of transient portion of speech-evoked auditory brainstem response (ABR) in control and study group.

Figure 2

Wave latencies of transitional and sustained portions of speech-evoked auditory brainstem response in study and control group.

On the contrary, there was a nonsignificant difference regarding amplitude in the two groups concerning sABR and MMN (Tables 5–7 and Figs 4 and 5).

Discussion

The aim of this study was to determine whether neural encoding of speech features at the level of brainstem and cortex is changed in children complaining of phonological disorder or not. In the present study, click ABR, sABR and MMN were measured to assess the integrity of neurophysiological responses in those children.

The phonological disorder was confirmed by phonological analysis by speech sample of connected speech of those children who complain of multiple phonological errors in their speech in form of syllable-structure processes, substitution processes, and also, assimilation processes.

In the present study, all participants (control and children with phonological disorders) showed well-identifiable and repeatable ABR waves with normal latencies for click stimuli table [1]. In other studies, the click ABR results were varied in normally developing children and impaired population. Some studies proved that click ABR is similar in both groups (normally developing children and impaired population) [12,13]. On the contrary, other studies showed differences in absolute latencies in impaired children when compared with the normal children [14].

In our study, well-identifiable and repeatable sABR waves but with delayed latencies in transient, transitional, and sustained portions of sABR were detected in all children with phonological disorders when compared with the control group (Tables 3 and 4 and Figs 1 and 2). Sanfins et al. [15] results were matched with our findings, as they observed abnormalities in absolute latency of V and A waves, and also abnormality in the VA slope; these

Figure 3
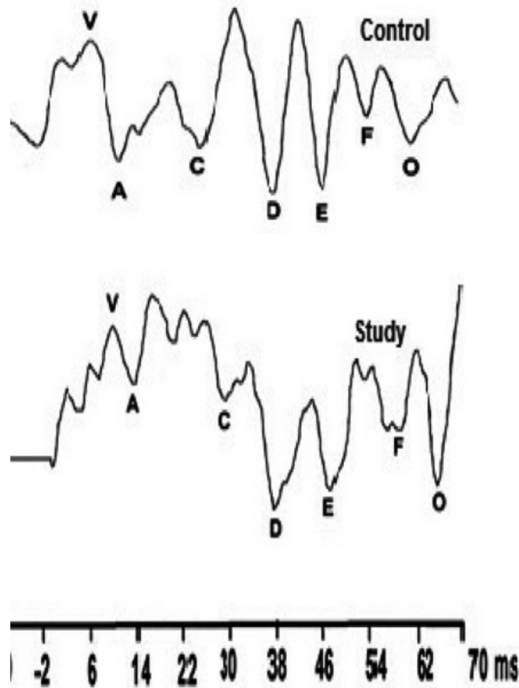
Speech-evoked auditory brainstem response in normal children (upper trace) and children with phonological error (lower trace) that show delayed latency in the lower trace.

Table 6 Comparison of V–A complex and amplitude of wave O of transient portion of speech-evoked auditory brainstem response between the study (30 ears) and control group (30 ears)

	Control group	Study group	*t*	*P*
V–A complex				
Duration (ms)	4.22±2.55	4.10±2.11	0.198	0.843
Amplitude (µV)	0.31±0.14	0.34±0.13	0.860	0.393
Slope	0.12±0.10	0.09±0.05	1.469	0.147
Amplitude of wave O	0.28±0.19	0.32±0.11	0.997	0.322

There were no statistical significant differences between study and control group regarding V–A complex and amplitude of wave O.

Table 7 Comparison of waves amplitude of transitional and sustained portions of speech-evoked auditory brainstem response in study (30 ears) group versus control group (30 ears)

Amplitude (µV)	Control group	Study group	*t*	*P*
C	0.21±0.05	0.24±0.09	1.596	0.115
D	0.33±0.14	0.38±0.15	1.334	0.187
E	0.35±0.18	0.40±0.13	1.233	0.222
F	0.32±0.10	0.36±0.08	1.710	0.092

There were no statistical significant differences regarding amplitude of transitional and sustained portions in between study and control group.

abnormalities were observed in children with phonological disorders when compared with children with normal development.

Moreover, Ghannoum *et al.* [16] studied the sABR in learning disabled children, and they found that sABR responses were affected in those children, suggesting abnormalities in brainstem encoding of auditory signals.

Those diverse findings of the speech and click-evoked results could be due to the difference in the acoustic character between click and speech stimuli. Click stimuli is characterized by rapid onset, brief duration, and flat broadband spectral components; however, speech stimuli is a complex signal with gradual onset and longer duration than click stimuli. Moreover, there is a difference in mechanism of encoding signal at the level of brainstem. Because of backward masking effect (effect of a vowel on brief consonant), the speech stimuli may be considered more challenging to the central auditory system [17,18].

On the contrary, in our study, assessment of MMN was performed in children with the phonological disorder, and our results showed that there were delayed MMN latencies in response to speech stimuli (Table 5) when compared with normal children. The same result was obtained by Ibraheem and Quriba [14] that reported

delayed MMN latency peak in 81.8% of the study group. The bottom-up influence or top-down feedback mechanism can clarify the combination of both cortical and brainstem abnormalities.

Bottom-up influence theory proposes that subtle deficit in the brainstem timing can lead to an impaired cortical ability to process acoustic stimuli under stress situation like noise or small differences between stimuli. This was supported by improvement of the cortical response in the patient with abnormal brainstem timing who undergo habituation training [19].

Top-down influences: as the auditory brainstem receives efferent inputs from the cortex, so the abnormal cortical function results in impaired cortical feedback to the brainstem, that affects the brainstem timing [20]. Moreover, the descending pathway has a role in gating the sensory information to the cortex by its influence on selective attention [21].

Accordingly, even without proven neurobiological abnormality in the study group of our work, this study supports the presence of a functional impairment in speech processing in the brainstem region and alteration in speech physiological mechanisms. Wible *et al.* [22] concluded that the physiological mechanism
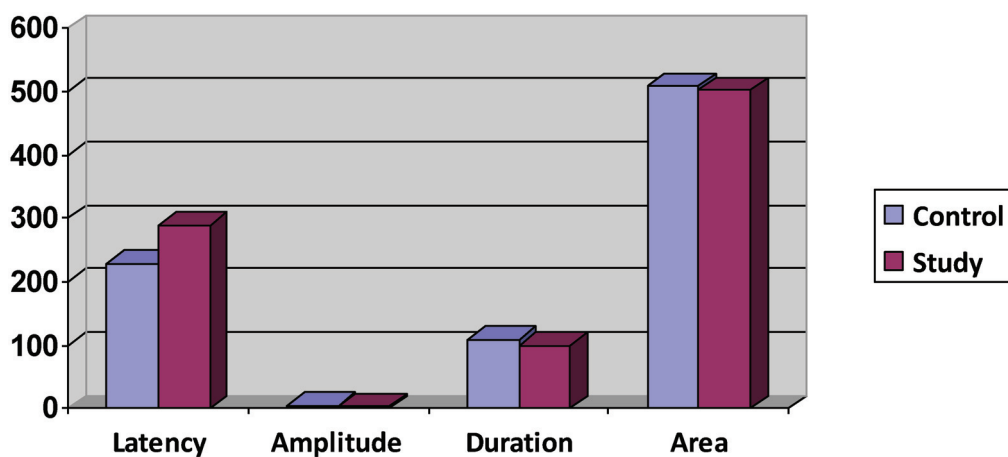
Figure 4
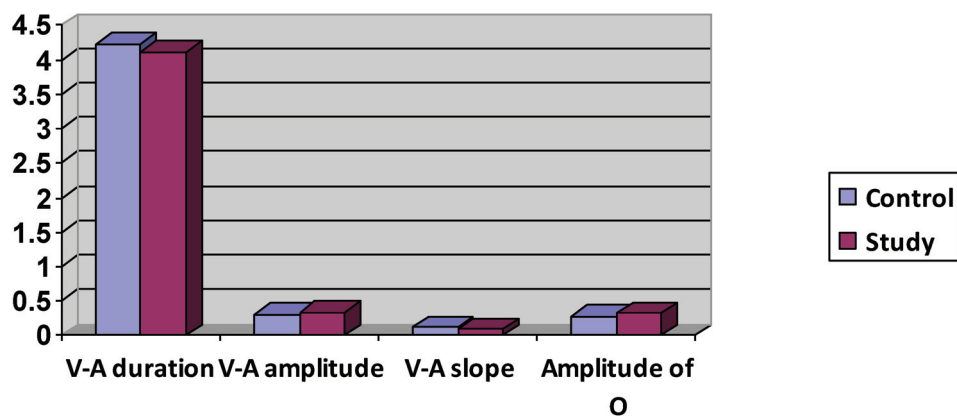
V–A complex and amplitude of wave O of transient portion of speech-evoked auditory brainstem response in control and study group.
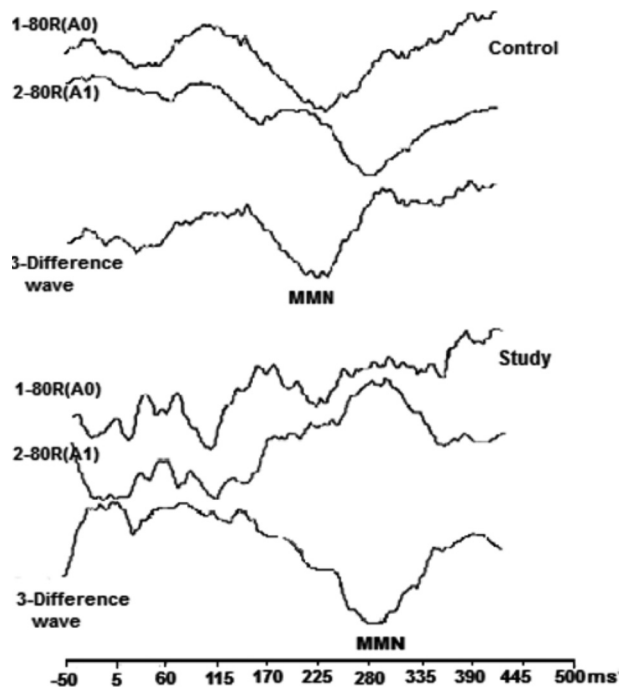
Figure 5
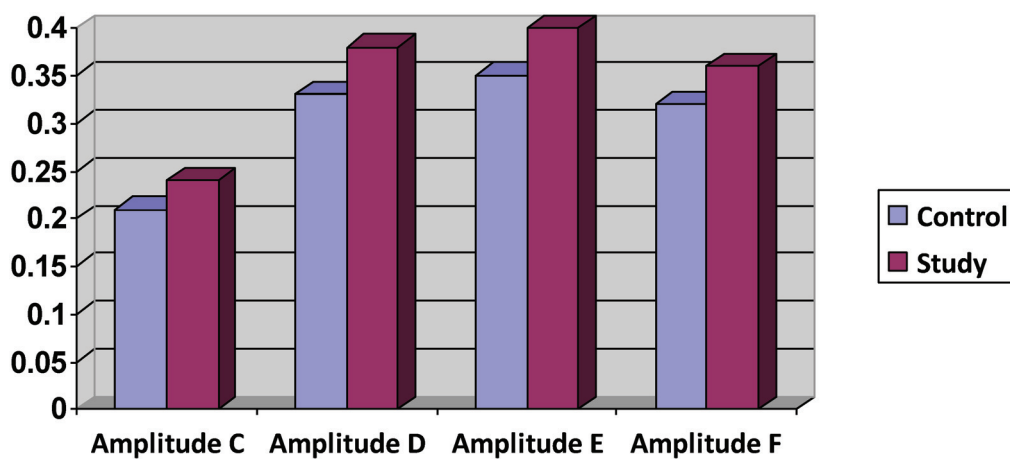
Waves amplitude of transitional and sustained portions of speech-evoked auditory brainstem response in study and control group.

Figure 6

Mismatch negativity measures in control and study groups.

Figure 7

Speech-evoked mismatch negativity in control and study group with delayed latency in study group.

alterations that cause an abnormal perception of speech and language abilities could be considered as an indicator for impairment of speech processing at the brainstem and the cortical level.

Finally, from these data, we concluded that the cortical auditory processing in children with phonological disorders could be evaluated by ABR and MMN. As abnormal brainstem timing and cortical dysfunction could present in the subset of patients, the deficit in the children with phonological disorders could be attributed to brainstem and cortical auditory processing abnormalities.

As this deficit in speech perception may affect the communication and language processes causing degradation of linguistic and paralinguistic information, it can also affect the quality of life and social interaction. There is a need for future research that aims to study the effect of auditory training on auditory processing capabilities of those children and whether the ABR and MMN could be used as tools to monitor the efficacy of auditory training program.

Conclusion

The phonological disorder may affect the communication and language processes causing degradation of linguistic and paralinguistic information, and also it can affect the quality of life and social interaction.

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