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Contralateral suppression of transient evoked otoacoustic emissions in children with phonological disorder

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Background: Perception of acoustic details in the speech signal is important for speech sound development. The medial olivocochlear pathway, a part of the auditory efferent system, plays a role in stimulus-related control of the cochlea. One clinical tool to evaluate the medial olivocochlear activity, which is thought to improve speech perception in noise, is the suppression of otoacoustic emissions.

Aims: This study investigated the suppression of transient evoked otoacoustic emissions in children with phonological disorder in comparison with that in typically developing controls.

Study Design: Case-control study.

Methods: A total of 23 children with phonological disorder (aged 5-10 years) and 21 age- and sex-matched controls ($P > 0.05$) participated in the study. Participants had pure-tone thresholds ≤ 15 dB hearing loss

and normal middle ear functions. Transient evoked otoacoustic emissions with and without contralateral acoustic stimulation were measured.

Results: Although the mean transient evoked otoacoustic emissions suppressions were lower in the group with phonological disorder than in the controls, these differences were not statistically significant ($P > 0.05$). No left/right ear asymmetry of transient evoked otoacoustic emissions suppression was detected in either of the groups ($P > 0.05$).

Conclusion: Children with phonological disorder did not show alterations in medial olivocochlear functioning in the medial olivocochlear activity as measured by the contralateral suppression of transient evoked otoacoustic emissions.

Speech sound disorder (SSD) has been defined as a developmental problem characterized by articulatory and/or phonological difficulties that impact a child's speech intelligibility.¹ Although nearly all children make speech sound errors when learning to speak, an SSD can be said to occur when these errors do not disappear at expected ages. To acquire the speech sound system of a language, the child must be able to hear sounds embedded in words and analyze them according to their acoustic and articulatory characteristics.² Hearing serves a dual role in speech sound acquisition, allowing children to access spoken language models and monitor the accuracy of their own productions.^{3,4} Perception of acoustic detail is important for speech sound development because it is eventually mapped to phonological and motoric representations governing speech production.^{2,5}

Although the full extent of its role in hearing is not well understood, there is growing evidence that the medial olivocochlear (MOC) efferent system could play functional roles in hearing.^{6,7} Activation of the MOC system by sound has an inhibitory effect on outer hair cell motility, suppressing the gain of the cochlear

amplifier.⁸ The effect of MOC activation on cochlear function can be monitored using otoacoustic emissions (OAEs). OAEs are sounds of cochlear origin generated by the motion of the outer hair cells. Acoustic stimulation of the MOC system results in a reduction in the amplitude of OAEs, which is called OAE suppression. The MOC efferent system improves the auditory signal to noise by reducing the response to a noisy background.⁹ Many studies have shown that MOC activity measured by contralateral suppression of OAEs is associated with the ability of understanding speech in noise.¹⁰⁻¹² However, there are also studies that have found no significant correlation between the magnitude of contralateral suppression and speech perception performance in noise.^{13,14}

It has been suggested that some children with SSD may have auditory processing problems that impact their ability to perceive phonetic details in the speech.^{15,16} Some studies reported evidence of MOC functioning alterations in individuals with auditory processing disorders (APDs)¹⁷⁻¹⁹ and specific language impairment.²⁰ However, a study of eight children with phono-

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logical disorders found no differences between the study and control groups with regard to transient evoked OAE (TEOAE) suppression effect.²¹

Everyday experiences with spoken language frequently occur in complex environments where there are background noises and/or multiple speakers. Some studies reported that children with language impairment had a greater difficulty in perceiving speech in noise than not only typically developing peers but also language-matched younger children to whom they showed similar speech perception performance in quiet conditions.^{22,23} Ziegler et al.²³ have suggested that speech-perception-in-noise deficits may have tremendous consequences for phonological development. The MOC system has been thought to be linked to the abilities of segregating the signal of interest from background sounds and selective auditory attention.⁷ Given the importance of these abilities in speech perception²⁴ and the fact that speech perception plays a role in speech-language abilities, it seems reasonable to investigate the MOC function in children with SSD. We hypothesized that children with phonological disorder will have reduced MOC activity, which possibly might have contributed to the emergence of phonological difficulties. We thought that reduced MOC inhibitory function may have had a negative impact on speech sound development, making it difficult for these children to notice the differences among speech sounds during the early years of life when the acquisition of language abilities is facilitated. Thus, the aim of this study was to evaluate the MOC efferent activity by the suppression of TEOAEs in children with phonological disorder in comparison with that in age- and sex-matched controls.

MATERIAL AND METHODS

Participants

A total of 44 children aged between 5 and 10 years (mean age = 7.6 ± 1.4 years, range: 5.2-9.9 years) participated in the study: 23 children with phonological disorder (9 females, 14 males) and 21 age- and sex-matched control subjects (9 female, 12 male). Sample size was calculated as 21 participants for each group on the basis of a high effect size in TEOAE suppression level between the groups with an alpha level of 5% and with a power of 80%. All participants were monolingual native speakers of Turkish. Inclusion criteria for all subjects were (1) having pure-tone thresholds ≤ 15 dB hearing loss (HL) for frequencies from 0.25 to 8 kHz, (2) showing a type A tympanogram graph, (3) presence of acoustic reflexes in 70 to 100 dB HL range, (4) presence of TEOAEs, (5) having no history of neurologic disorder or developmental impairment, and (6) being right handed.

Subjects in the study group were children who had previously been diagnosed in the institution where the study was conducted and were advised to receive therapy for the remediation of their phonological disorder. Before the definition of their problem by a speech pathologist as well as evaluations by otolaryngologist and audiologist, they had been assessed by a pediatrician, child psychiatrist, and if needed, child neurologist. Inclusion criteria for this group were as follows: (1) presence of phonologically based SSD of unknown origin, (2) having multiple speech sound

errors with a score below the tenth percentile on the standardized articulation test, (3) having a normal oral motor function, and (4) age-appropriate performance in other aspects of oral language. Children whose SSD was due to other secondary causes, such as HL, and those with other concomitant diagnoses, such as language impairment, autism spectrum disorder, learning disorder, attention deficit hyperactivity disorder, were excluded. Children who had articulation errors only or features consistent with childhood apraxia of speech were also excluded. The absence/presence of childhood apraxia was evaluated using a sign checklist. According to the evaluation, none of the participants had features suggesting childhood apraxia, such as vowel errors/distortions, difficulty with nonspeech movements, articulatory searching behaviors, slow rate of speech, and inappropriate prosody.²⁵ Thus, the study group comprised children who exhibit phonologically based speech sound errors (mean age: 7.5 ± 1.3 years, range: 5.2-9.2 years).

The control group consisted of 21 typically developing children without any speech or language difficulties (mean age: 7.7 ± 1.5 years, range: 5.2-9.9 years). Information about the control children's developmental and health history was obtained through parent interviews, and no further medical examination was requested. Participants in both groups had no family history of speech and/or language disorder, and all were right-handed according to parental reports and direct observation of their hand preferences during some tasks.

Procedure

This study was conducted in accordance with the principles of the Helsinki Declaration and approved by the relevant Institutional Research Ethics Committee (protocol no: 2014-54). After obtaining informed consent from their parents, children in the two groups were evaluated using the following measurements:

1. Evaluation of speech sound production: Although the study group comprised children who had previously been diagnosed, to see their current condition, all the children participated in a picture-naming task (Ankara Articulation Test) that was designed to assess speech sound development in children aged 2-12 years.²⁶ The test was conducted by an experienced speech-language pathologist to evaluate the child's ability to produce consonants in different positions of the syllables and words. The investigation of speech sound production in the study group showed that the mean raw score obtained by counting the number of speech sound mistakes was 11.35 ± 4.12 , and the mean standard score was 62.70 ± 10.45 , indicating a below-average performance for their sex and age. In addition, systematic sound changes (e.g., final consonant deletion, weak syllable deletion, assimilation) were observed in all children.
2. Evaluation of hearing: All participants underwent an audiological assessment, including otoscopy, impedance audiometry (tympanometry and acoustic reflexes), and pure-tone audiometry. Pure-tone air conduction hearing thresholds for the frequencies from 0.25 kHz to 8 kHz were measured in a sound-treated room using AC 40 (Interacoustics, Middel-

fart, Denmark) clinical audiometer and Telephonics TDH-39 headphones (Telephonics, NY, USA). The tympanometry was performed with a 226 Hz probe tone. Type A tympanogram, with peak pressure ranging from 50 daPa to 100 daPa, compliance between 0.3 and 1.5 cm³, and tympanometric width < 200 daPa, was considered normal. Acoustic reflex thresholds were measured with pure-tone signals in the frequency range of 0.5-2 kHz.

- TEOAE recordings and evaluation of MOC function: TEOAE measurements were binaurally performed using an ILO 292 Echoport USB II and ILO V6 Clinical OAE software (Otodynamics, London, United Kingdom). The testing was conducted in a sound-treated room. To check the presence of TEOAEs in all the participants, initial measurements with nonlinear click stimuli at 80 dB peak equivalent sound pressure level (peSPL) were done. TEOAEs were considered present when the reproducibility was ≥ 70%, stimulus stability was ≥ 80%, and the signal-to-noise ratio was ≥ 3 dB. For the suppression procedure, TEOAEs with and without contralateral acoustic stimulation (CAS) were measured with linear 80-μs click stimuli presented at 60 dB peSPL at a rate of 50 clicks per second. The CAS consisted of continuous white noise at 60 dB sound pressure level delivered through channel B of the OAE analyzer. The measurement time was 3.5-20 ms, and each recording was the average of 260 clicks. Measurements were made at 1, 1.4, 2, 2.8, and 4 kHz one-half octave frequency bands. Contralateral suppression was calculated by subtracting the value of OAEs with CAS from the value of OAEs without CAS.

Data Analysis

Statistical analysis of the data was carried out using Statistical Package for the Social Sciences 17.0 (SPSS Inc., Chicago, IL, USA). The normality of the data was investigated using the Shapiro-Wilks test. An independent samples *t*-test (for normal distribution)

or Mann-Whitney U test (for non-normal distribution) was used to compare differences between the SSD and control groups. A paired samples *t*-test or Wilcoxon test was used for within-subject comparisons of the left and right ears. Chi-square test was used for group comparisons including nominal data. A *P*-value <0.05 was considered statistically significant.

RESULTS

The two groups were similar with respect to age (independent samples *t*-test, *P* > 0.05) and sex (Chi-square test, *P* > 0.05). No significant differences were found between the groups or between the left and right ears in hearing thresholds at any of the examined frequencies and at the values of static compliance, tympanometric peak pressure, and acoustic reflex thresholds (*P* > 0.05).

There were no significant differences between the left and right ears of the subjects with regard to TEOAE amplitudes with and without CAS (paired samples *t*-test, Wilcoxon test, *P* > 0.05). When CAS was added, a decrease in the overall TEOAE response intensity was observed in all but one control subject and in all but three study group participants. The mean TEOAE amplitudes for each frequency before and after CAS are given in Table 1. No significant differences in TEOAE amplitudes at any of the frequencies were present between the study and control groups for either before CAS or after CAS measurements (Student's *t*-test, Mann-Whitney U test, *P* > 0.05).

No significant differences were found between the left and right ears in TEOAE suppression levels (Wilcoxon test, *P* > 0.05). Although we observed significant changes in emission amplitudes of both groups when CAS was added (paired samples *t*-test, Wilcoxon test, *P* < 0.05), the mean suppression values of the study group were lower than those of the control group at all frequencies (Table 2). However, these differences were not statistically significant (Mann-Whitney U test, *P* > 0.05).

TABLE 1. Mean TEOAE amplitudes without and with CAS in study and control groups

Frequency (kHz)	Ear	TEOAE without CAS (dB SPL) Mean (SD)		<i>P</i>	TEOAE with CAS (dB SPL) Mean (SD)		<i>P</i>
		Study group n = 23	Control group n = 21		Study group n = 23	Control group n = 21	
1	L	9.50 (4.97)	9.33 (7.50)	0.93	7.93 (5.58)	7.59 (7.39)	0.86
	R	9.43 (5.10)	8.58 (7.22)	0.65	8.27 (4.78)	7.36 (7.15)	0.62
1.4	L	11.33 (4.67)	11.28 (4.79)	0.97	10.11 (4.52)	9.44 (4.14)	0.65
	R	12.32 (5.85)	12.27 (5.93)	0.98	11.02 (5.56)	10.45 (6.22)	0.75
2	L	11.34 (5.69)	10.24 (5.18)	0.51	10.32 (5.58)	9.06 (5.10)	0.44
	R	11.70 (4.83)	10.92 (6.07)	0.64	10.49 (4.68)	9.44 (6.10)	0.52
2.8	L	10.18 (4.83)	9.68 (6.19)	0.77	9.05 (4.50)	8.33 (6.06)	0.67
	R	10.53 (6.35)	10.74 (5.52)	0.91	9.46 (5.89)	9.19 (6.00)	0.88
4	L	10.12 (5.25)	9.84 (6.45)	0.88	8.80 (5.07)	8.50 (6.45)	0.86
	R	8.90 (4.50)	9.56 (7.46)	0.73	7.56 (4.73)	8.04 (7.94)	0.81
Overall	L	18.47 (4.42)	18.24 (5.17)	0.88	17.11 (4.54)	16.71 (5.49)	0.79
	R	18.67 (4.49)	18.96 (5.30)	0.84	17.33 (4.39)	17.46 (5.56)	0.93

Analyses were conducted with independent samples *t*-test and Mann-Whitney U test.

CAS, contralateral acoustic stimulation; L, left; R, right; SD, standard deviation; SPL, sound pressure level; TEOAE, transient evoked otoacoustic emission.

TABLE 2. TEOAE suppression values (dB SPL) in the study and control groups

Frequency (kHz)	Ear	Study group n = 23		Control group n = 21		P
		Mean (SD)	Median (min–max)	Mean (SD)	Median (min–max)	
1	L	1.56 (1.49)	1.60 (–2.8 to –4.8)	1.74 (1.63)	1.70 (–1.6 to –5.6)	0.88
	R	1.17 (1.43)	1.00 (–2.1 to –4.7)	1.22 (1.43)	1.00 (–1.0 to –4.8)	0.93
1.4	L	1.21 (1.23)	1.30 (–1.0 to –3.4)	1.83 (1.10)	1.60 (0.1 to –4.1)	0.21
	R	1.30 (1.14)	1.30 (–1.4 to –4.1)	1.81 (1.14)	1.60 (–0.2 to –4.2)	0.19
2	L	1.02 (0.77)	1.20 (–0.5 to –2.6)	1.18 (0.49)	1.20 (0.2 to –2.3)	0.55
	R	1.20 (0.67)	1.20 (0.1 to –2.6)	1.48 (0.68)	1.40 (0.3 to –2.9)	0.18
2.8	L	1.11 (0.88)	1.20 (–1.7 to –2.7)	1.36 (0.63)	1.30 (–0.8 to –2.9)	0.23
	R	1.06 (0.76)	1.20 (–0.8 to –2.3)	1.56 (1.15)	1.20 (0.5 to –6.2)	0.25
4	L	1.32 (1.31)	1.20 (–0.3 to –6.9)	1.35 (0.33)	1.30 (0.7 to –2.0)	0.10
	R	1.34 (0.70)	1.20 (0.0 to –3.7)	1.51 (0.87)	1.40 (0.8 to –5.0)	0.69
Overall	L	1.35 (0.86)	1.20 (–0.2 to –3.0)	1.53 (0.70)	1.40 (–0.1 to –2.9)	0.47
	R	1.34 (0.65)	1.30 (–0.3 to –2.8)	1.50 (0.60)	1.40 (0.8 to –2.9)	0.49

Analyses were conducted with Mann–Whitney U test.

L, left; max, maximum; min, minimum; R, right; SD, standard deviation; SPL, sound pressure level; TEOAE, transient evoked otoacoustic emission.

DISCUSSION

In this study, we investigated whether children with phonological disorder have reduced MOC efferent activity compared with control subjects, as measured by the contralateral suppression of TEOAEs. Despite lower suppression values of the study group at all frequencies, statistical analysis indicated no significant difference between the groups with regard to TEOAE suppression values. The decrease in OAE levels with CAS reflects the inhibitory function of the MOC system on the outer hair cells.²⁷ Therefore, the fact that there was no significant difference between the TEOAE suppressions of children with and that of children without phonological disorder suggests no alteration in MOC efferent activity in our participants with phonological disorder.

Our findings are consistent with those reported by Didone et al.²¹ who investigated contralateral suppression of TEOAEs in 8 children with phonological disorders and 11 controls. Their results suggest that children with phonological disorders have no alterations in the MOC efferent system. In another study, Clarke et al.²⁸ studied the TEOAE suppression effect in children with specific language impairments and suggested that children with specific language impairment do not have auditory processing problems at the MOC system level. Contrary to Clarke et al.'s²⁸ findings, Rocha-Muniz et al.²⁰ determined that children with specific language impairment and poor speech-in-noise performance had reduced TEOAE suppression compared with the typical development group.

Language difficulties, including speech sound problems, are present in a large proportion of children with APD.²⁹ Although it has been suggested that reduced MOC activity is more common in children with APD^{17–19}, the results of other studies did not show a statistically significant evidence of alterations in the MOC system.^{6,30} Yalçinkaya et al.³¹ found lower suppression values in children with

listening problems. However, the results of a study by Mattson et al.⁶ did not support the hypothesized link between reduced MOC activity and listening difficulties in children with APD. Despite the evidence in favor of alterations in auditory processing in children with language problems,³² considering conflicting results obtained from previous studies regarding the suppression effect in APD, it seems difficult to establish a clear link between speech sound difficulties in these children and MOC system alterations. On the basis of the similarities between TEOAE suppression levels of the groups, we can conclude that the findings of our study did not provide any evidence that phonological difficulties of our participants may be related to the MOC system-level auditory processing problems.

In this study, we found no statistically significant difference in the values of TEOAE suppression between the left and the right ears. Higher values of suppression in the right ear were reported by several authors and were attributed to possible asymmetry in MOC activity in right-handed individuals favoring the right ear.^{33,34} However, studies including children aged 4–7 years,²¹ 7.5–12 years²⁸, and 8–14 years⁶ reported no significant ear difference in the suppression values. The absence of right/left ear suppression asymmetry in our subjects is consistent with views of Clarke et al.'s²⁸ and Mattsson et al.'s⁶ views suggesting that the suppression asymmetry between the ears is a situation that may develop with age.

This study has some limitations that have to be pointed out. The small sample size limiting the generalizability of the findings was one of them. In addition, children with phonologically based SSD constitute a heterogeneous group, with subgroups that differ in error patterns and the developmental trajectory. Dodd's classification system proposes three subtypes of phonological disorders: phonological delay, consistent atypical phonological disorder, and inconsistent phonological disorder.³⁵ In

our study, we tried to obtain an isolated phonological disorder group as much as possible by excluding children with concomitant conditions and any other difficulties. However, we did not attempt to describe our participants by classifying them into homogeneous groups according to error patterns (for example, those with typical/atypical error patterns) when selecting our participants. For this reason, there is a possibility that our sample may have included more children in any of the pattern-based subgroups of SSD. This situation, which can be considered as one of the limitations of the study, should be taken into consideration when interpreting the results.

In this study, although the mean TEOAE suppression values were lower in the group with phonological disorder than in the controls, these differences were not statistically significant. The findings of this study do not support our hypothesis that children with phonological disorder would have alterations in the MOC activity as measured by the contralateral suppression of TEOAEs. However, because the study sample is small, further research on the MOC function in this population is warranted.

Ethics Committee Approval: Ethics committee approval for the study was obtained from the Institutional Research Ethics Committee (protocol no: 2014-54).

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