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The role of the efferent auditory system in developmental dyslexia

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ABSTRACT

Objective: To assess the role of the efferent auditory system by inhibition of contralateral otoacoustic emission in dyslexic children with auditory processing disorders.
Methods: The study sample was 34 children: 17 with dyslexia and 17 age-matched controls. Sensitive speech tests (low-pass filtered, time-compressed, distorted and dichotic) were performed to assess coexisting auditory processing disorder. Distortion-product otoacoustic emission (DPOAE) values were measured in basal condition and with contralateral broadband noise signal delivered via an earphone transducer at 60 dB SPL.
Results: The lower scores at sensitive speech testing confirmed the association of an auditory processing disorder in the dyslexic children. DPOAE values were significantly attenuated by contralateral inhibition only in the control group ($p = 0.001$; dyslexics, $p = 0.19$); attenuation was not significant at any frequency in the dyslexic group.
Conclusions: The differences in DPOAE attenuation between the groups, although not statistically significant, suggest alterations in the auditory efferent system in the dyslexic population. These alterations may affect language perception. If confirmed in further studies with larger samples, these results could provide insight into a possible pathophysiological background of dyslexia.

1. Introduction

Developmental dyslexia, defined as learning difficulty with reading and spelling despite adequate intelligence and educational opportunity, is one of the most common childhood learning disabilities and affects about 5–10% of schoolchildren worldwide [1]. Although a genome involvement has been robustly demonstrated in developmental dyslexia, the link between specific DNA regions and the variability of manifestations and reading abilities/inabilities is still unknown [2]. Genome peculiarities in dyslexics may influence cell migration and organization. This biological basis for dyslexia is consistent with findings that some thalamus neurons are smaller than expected in dyslexics [3]. Such structural abnormalities may be related to the auditory temporal processing disorders (APD) found in language-impaired children [4] and constitute neural evidence for the auditory temporal processing hypothesis of dyslexia. Tallal's seminal study showed that children with dyslexia, as compared to normal-hearing children, had poorer performance when asked to determine the order of two non-speech tones presented at short inter-stimulus intervals [5]. In particular, she compared 20 reading-disabled children to 12 normal children on a repetition test requiring same/different and temporal order judgments of two 75-ms non-verbal complex tones differing only in fundamental frequency. According to this theory, dyslexic subjects have difficulty in perceiving short or rapidly varying sounds. This is particularly the case of spoken language, in which rapid spectral changes occur over brief time intervals. Coherent with this line of thinking is the hypothesis that if the early auditory pathway is altered, the quality of the phonemic representations extracted from its signals will be degraded, thus disrupting normal reading development. This would occur because of the close interrelationship between auditory temporal processing, speech perception, and reading acquisition.

Audition provides the input to the information processing stages that, in turn, generate the phonological information necessary for speech perception and the development of reading [6]. It has been suggested, however, that the auditory deficit observed in dyslexics has only a minor influence on the development of phonology and reading and that less than 39% of children with dyslexia present with auditory deficits [7].

Although it is widely accepted that the processing of phonological information is impaired in dyslexics, the origin of the difficulties associated with dyslexia is controversial [8] because separating one function from another when investigating such a

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complex central processing system is highly problematic. Among the numerous neural connections of the auditory pathway, its efferent system has never been studied in dyslexics. Theoretically, this may have some relevance, as it has in children with auditory processing disorders [9], because the olivocochlear complex and the medial olivocolchlear tract fibers, in addition to their protective action on the inner ear, seem to play a role also in sound localization and in acoustic signal detection in noise, as well as in improving auditory sensitivity and attention [10]. To date, there are no reports about the role of the efferent auditory system in subjects with dyslexia.

The efferent auditory pathways may be activated by acoustic stimuli; in humans, this activation can be easily studied by the suppression of otoacoustic emission (OAE) when the medial olivocochlear tract fibers – by outer hair synapse action – attenuate OAE responses in the presence of controlateral noise [11]. The aim of this study was to evaluate the efferent system in dyslexics with difficulties in auditory information processing.

2. Materials and methods

The study sample was 34 children (18 males and 16 females); 17 were dyslexic (mean age, 10.2 ± 2.25 years [±SD]) and 17 age-matched children (mean age, 11.3 ± 3.12 years) with no reported history of learning disorders.

Inclusion criteria were normal otoscopy and normal hearing sensitivity (≤20 dB HL at all octave frequencies from 0.5 to 4 kHz) and middle ear function. Dyslexia was diagnosed according to the Italian Consensus Conference criteria on specific learning disabilities [12]. All the dyslexics showed developmental phonological dyslexia, as described by Marshall [13]. Their reading performance was less than 2 SD on word and non-word speed reading tests and below the fifth percentile in reading accuracy as compared to age-matched normal controls. Only pure dyslexics without associated learning disorders (e.g. dyscalculia, dysgraphia) were included in the study.

After verbal and written explanation of the study purpose and design, the children and their legal guardians gave informed consent to participate. The local Ethics Committee approved the study protocol.

Assessment included the administration of sensitive speech tests to evaluate central auditory processing and recording of distortion-product otoacoustic-emissions (DPOAEs) with and without contralateral acoustic stimuli. Behavioral measures of central auditory processing included low-pass filtered word test, time-compressed word test, distorted speech test, and dichotic speech test according to the American Speech-Language-Hearing Association [14]. All measurements were performed in a sound-attenuated booth where the subjects listened to and then repeated 3 series of 10 words for each test; the percentage of correctly repeated words was recorded.

The words were monaurally presented at 40 dB HL for the low-pass filtered test with a cut-off filter set at 1000 Hz, monaurally at 50 dB HL for the quick speech test, and at 40 dB HL for the distorted speech test. Dichotic speech testing was administered by the simultaneous presentation of two different disyllabic words to each ear at 50 dB HL.

We recorded DPOAEs by means of an ILO 92 Distortion Product Analyzer (ILO 92 Otodynamic Analyzer, version 1.35; otodynamics). For DPOAE generation two primary tones (L_1 = 60 dB, L_2 = 50 dB), at a frequency ratio of f_2/f_1 = 1.22, were used: the distortion product of $2f1 - f2$ was selected to measure DPOAE amplitude (expressed in dB SPL). The cochlear regions were stimulated at five frequencies (1, 2, 3, 4, and 6 kHz). Both ears were tested separately in basal condition and with contralateral broadband noise signal delivered via an earphone transducer at 60 dB SPL. The recordings were considered significant when the DPOAE value was 3 dB higher than background noise.

Data elaboration and statistical analysis were carried out by means of Microsoft Excel and R software. The level of significance was set at $p < 0.05$. The Shapiro–Wilk test was used to verify data normal distribution and Student's t test was used in such cases. The Wilcoxon–Mann–Whitney test was used when data did not distribute normally.

3. Results

Speech test scores were lower for the dyslexic than for the control group; the differences were statistically significant on the time-compressed word, the distorted speech, and the dichotic tests (Fig. 1).

The mean DPOAE value for all frequencies tested at basal condition was 8.81 ± 7.71 dB SPL for the control group and 7.46 ± 8.61 dB SPL for the dyslexic group. As expected, there was a reduction in DPOAE amplitude on the contralateral inhibition test: mean DPOAE 7.44 ± 7.88 dB SPL and 6.43 ± 8.21 dB SPL for the control group and the dyslexic group, respectively. Between-group differences were not significant in either basal conditions (p = 0.66 at Wilcoxon–Mann–Whitney test) or with contralateral noise (p = 0.3 at Student's t test). The within-group differences were statistically significant at Student's t test only in the control group (p = 0.0001 at Student's t test; p = 0.19 at the Wilcoxon–Mann–Whitney test in the dyslexics).

Tables 1 and 2 report the DPOAE values for each frequency tested in basal condition and with contralateral stimulus.

4. Discussion

Learning disorders are frequently associated with auditory processing disorders (APD) [15,16] but how they relate to each other and why they coexist are currently unknown. One hypothesis is based on auditory temporal processing deficiency. Auditory temporal processing difficulty can be described as a limited ability to process acoustic elements of short duration, such as consonants with rapid formant transition. Because dyslexics seem to have difficulty perceiving and distinguishing these sounds properly within the speech spectrum, they are unable to associate letters with their specific sounds. This inability explains their learning disorder [17].

There is some evidence suggesting that the auditory medial efferent system enhances frequency-resolving capacity [18,19]

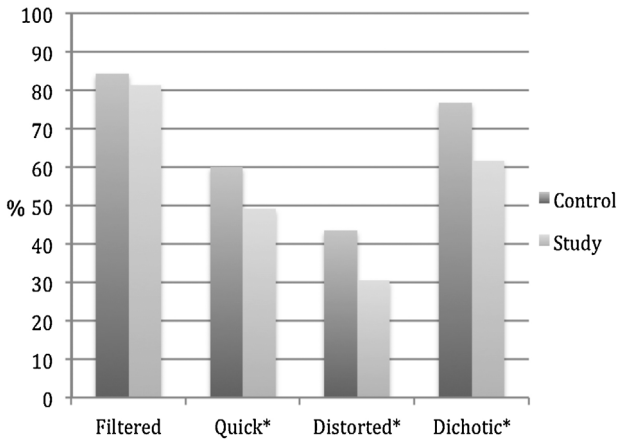

Fig. 1. Speech tests results in dyslexics and normal children. Significant between-groups differences are marked with *. p values were 0.24, 0.02, 0.003, and 0.0001, respectively.