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Antisaccades in Dyslexic Children: Evidence for Immaturity of Oculomotor Cortical Structures

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1. Introduction

The antisaccade task has been introduced for the first time by Hallet (1978) in order to explore the ability of the brain to control behaviour flexibly. Antisaccades are voluntary saccades during which subjects have to inhibit the movement towards a peripheral visual target. Usually subjects fixate a central fixation point, which is then extinguished and the peripheral target is presented. Subjects are instructed to generate a saccade of the same amplitude to the opposite direction, as quickly and accurately as possible. It is generally assumed that the sudden appearance of the target in an antisaccade task automatically triggers a motor program for a prosaccade in this direction, and that errors occur when certain endogenous processes fail to inhibit or cancel this program (Everling & Fischer, 1998). It is argued that correct antisaccade latencies are increased compared to prosaccade latencies because the application of the inhibitory processes is time consuming (Olk & Kingstone, 2003). Everling and Fischer (1998) argued that antisaccade performance requires two intact subprocesses: 1) the ability to suppress a reflexive saccade towards the target; 2) the ability to generate a voluntary saccade in the opposite direction. In clinical research, increased antisaccade error rates are often interpreted as reflecting failures in inhibitory processing (Crawford, Bennett, Lekwuwa, Shaunak, & Deakin, 2002; Hutton et al., 2008).

Neuropsychological studies have shown an important role of the frontal cortices during performing antisaccades. For instance, Everling and Munoz (2000), and Funahashi et al. (1993) revealed that several frontal structures (frontal eye field, dorsolateral cortex and supplementary eye field) are more activated during antisaccade tasks than during prosaccades (a saccade made towards the peripheral target). Furthermore, Matsuda et al. (2004) reported increased activity in the inferior parietal cortex during antisaccade tasks compared to prosaccades. Interestingly, Ettinger et al. (2008) showed activity in such area during a period preceeding the antisaccade generation, suggesting an inhibitory role of this region. Other studies found out that the parietal cortex (some regions in the intraparietal

sulcus) is responsible for the vector inversion required to generate an antisaccade to the correct location (Clementz et al., 2007; Zhang & Barash, 2000).

Several researchers have focused on the development of the ability to perform antisaccades. For example, as suggested in Luna's exhaustive review (Luna et al., 2008) exhaustive review, the maturity of the cortical structures devoted to eye movement performances is reached at 14-15 years. Consequently, the improvements in antisaccade performance continue during adolescence even though the ability to successfully inhibit a saccade toward a new target is already present at 8 years old (Johnson, 1995).

Moreover, the antisaccade task has also been used as important clinical tool for investigating dysfunction in various neurological and psychiatric disorders (Leigh and Kennard, 2004). Patients with discrete lesions of the dorsolateral cortex and in the frontal eye field have difficulty in performing correctly the antisaccade task (Guitton et al., 1985; Walker et al., 1998; Gaymard et al., 1999; Davidson et al., 1999).

The antisaccade task has been extensively studied in dyslexic children by the Fischer's group. Indeed, Biscaldi et al. (2000) and Fischer & Hartnegg (2000a) compared the performance in an antisaccade task between dyslexic children and non-dyslexic children of similar age. These authors reported an increased number of directional errors and several saccades being missed in dyslexic children. Furthermore, Fischer and Hartnegg (2000b) showed that this poorer performance in dyslexic children could be improved by training, leading to obtain a performance similar to that reported in non-dyslexic children. Therefore, although some evidence exists suggesting impaired inhibitory processing in dyslexic children, such a deficit can be overcome by training.

Based on all these findings we aimed to explore whether the poor antisaccade performance reported in dyslexic children could be a consequence of immaturity of cortical structures responsible of triggering and execution of saccadic eye movements rather than a congenital deficit of these areas. Indeed, the fact that dyslexic children are able to improve antisaccade performance with training as shown by Fischer and Hartnegg (2000b) is in line with the hypothesis of a delayed maturation of the oculomotor system in such type of subjects (Bucci et al., 2008).

In the present study we compared antisaccade performance in three different groups of children: (i) dyslexic children; (ii) age-matched non-dyslexic children; (iii) reading age-matched non-dyslexic children.

2. Materials and methods

2.1 Participants

Twenty-one dyslexic children were recruited from the pediatric hospital where they were referred for a complete evaluation of their dyslexia state with an extensive examination including neurological/psychological and phonological capabilities. For each child the time required to read a text, its comprehension, and the capacity of reading word/pseudowords was evaluated by using the L2MA battery (Chevrie-Muller et al., 1997). This is a standard test developed by the Applied Psychology Centre of Paris (Centre de Psychologie Appliquée de Paris), and is used everywhere in France. Inclusion criteria for dyslexic were: scores on

this test below 2 standard deviations of normalized values; and a normal mean intelligence quotient, between 85 and 115 (IQ, evaluated with WISC IV). The mean age of the dyslexic children was 11.19 ± 0.2 years, the mean IQ was 100 ± 6 and the mean reading age was 8 ± 1 years. A carefully selected age-matched (29 children, mean age 11.6 ± 0.17) and reading age-matched (24 children, mean age 7.8 ± 0.19) groups of non-dyslexic children were selected. These children had to satisfy the following criteria: no known neurological or psychiatric abnormalities, no history of reading difficulty, no visual impairment or difficulty with near vision. For the two groups of non-dyslexic children reading capabilities were in normal range. Both the similitude test of the WISC IV assessing the verbal capability, and the matrix test of the WISC IV assessing the logic capability were performed. Normal range for both tests is 10 ± 3 (Wechsler intelligence scale for children—fourth edition, 2004). The selected reading age-matched group was normal for verbal (11.78 ± 0.8) and for logic (9.97 ± 0.6) capabilities. The selected age-matched group was also normal (10.36 ± 0.4 for verbal and 11.89 ± 0.5 for logic).

Both non-dyslexic and dyslexic children underwent an ophthalmologic and orthoptic examination in order to evaluate their visual function (median values shown in Table 1). All children had normal binocular vision (60 sec of arc or better), which was evaluated with the TNO random dot test. Visual acuity was normal (≥20/20) for all children, dyslexic as well as non dyslexic. The near point of convergence was normal for all three groups of children tested (≤ 5 cm). Moreover, an orthoptic evaluation of vergence fusion capability using prisms and Maddox rod was carried out at far and at near distance. At far distance, the divergence and convergence amplitudes were similar in the three groups of children examined. In contrast, at near distance, the divergence and convergence amplitudes were significantly different in the dyslexic group with respect to the other two groups of non dyslexic children. ANOVA showed significant main effects of group, $F_{(2,71)} = 6.36$, $p < 0.003$ and of the divergence and convergence amplitudes, $F_{(2,71)} = 3.18$, $p < 0.04$., respectively). The LSD test showed that the dyslexic group had significantly smaller value of divergence and convergence amplitudes with respect to the two groups of non-dyslexic children (younger and older).

Finally, phoria (i.e. latent deviation of one eye when the other eye is covered, using the cover-uncover test) was normal for all three groups of children tested.

|	TNO	NPC	Phoria Far	Phoria Near	Div Far	Div Near	Conv Far	Conv Near
D 10-13	63	3	0	Exo 1	4	10	15	32
ND 7-9	45	2	0	Exo 2	4	14*	16	40*
ND 10-13	40	2	0	Exo 2	6	13*	17	40*

Note: dyslexic children, D 10-13; non-dyslexic children chronological age matched, ND 10-13; and non-dyslexic children reading age matched, ND 7-9. Median values of: binocular vision (Stereoacuity test, TNO measured in seconds of arc); near point of convergence, NPC measured in cm; Heterophoria at far and near distance, measured in prism diopters; Exo = exophoria; Vergence fusional amplitudes (divergence and convergence) at far and at near distance, measured in prism diopters. Asterisks indicate that value is significantly different with respect to the group of dyslexic children (p<0.01).

Table 1. Clinical characteristic of the three groups of children examined

The investigation adhered to the principles of the Declaration of Helsinki and was approved by our Institutional Human Experimentation Committee. Informed consent was obtained from the children's parents after explaining the procedure for the experiment to them.

2.2 Oculomotor paradigm

Stimuli were presented on a PC screen of 22″, its resolution was 1920×1080 and the refresh rate was 60 Hz. The stimulus consisted in a red filled circle subtending a visual angle of 0.5 deg. The trial consisted of a target positioned at the center of the screen for a variable delay between 2000 and 3500 ms. The central target disappeared and after a period of 200 ms (= gap period), a lateral target (green filled circle) appeared at 22.8 degrees, randomly to the left or to the right of the center, and stayed on for 1000 ms. After this duration, the central fixation target appeared again, signalling the beginning of the next trial as shown in Figure 1. The lateral target appeared randomly to the left or right and each direction was presented an equal number of times (i.e., 15 each). Children were instructed to look at the central fixation point, then to trigger a saccade as soon as possible in the opposite direction and symmetrically to the lateral target. Thus, when the target moved to the right, the child had to look at the same distance to the left side. When the target returned to the center, the child was instructed to follow it back to the center. An initial training block of trials was given to ensure that the instructions were understood.

1000 ms

200 ms

2000-3500 ms   Time = 3200-4700 ms
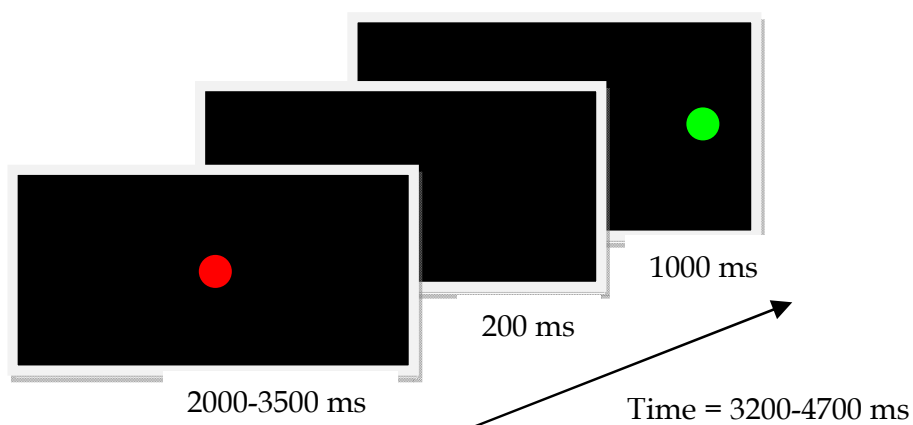
Note: When the green target appears, the child has to make a saccade to it mirror position as quickly as possible. The duration of each trial was between 3200 and 4700 ms.

Fig. 1. Schematic trial of the antisaccade task.

2.3 Eye movements recording

Eye movements were recorded with the Mobile Eyebrain Tracker (Mobile EBT®, e(ye)BRAIN, www.eye-brain.com), an eye-tracking device CE marked for medical purpose (see Figure 2). The Mobile EBT® benefits from cameras that capture the movements of each eye independently. Recording frequency was set up to 300 Hz.

2.4 Procedure

Children were seated in a chair in a dark room with the head leaning on a forehead and chin support; viewing was binocular; the viewing distance was 58 cm. Calibration was carried

out at the beginning of eye movements recordings. During the calibration procedure, children were asked to fixate a grid of 13 points (diameter 0.5 deg) mapping the screen. Each calibration point required a fixation of 250 ms to be validated. A polynomial function with five parameters was used to fit the calibration data and to determine the visual angles. After the calibration procedure, the antisaccade task was presented to the child. Duration of the task was kept short (lasting a couple of minutes) allowing an accurate evaluation of eye movement recordings.
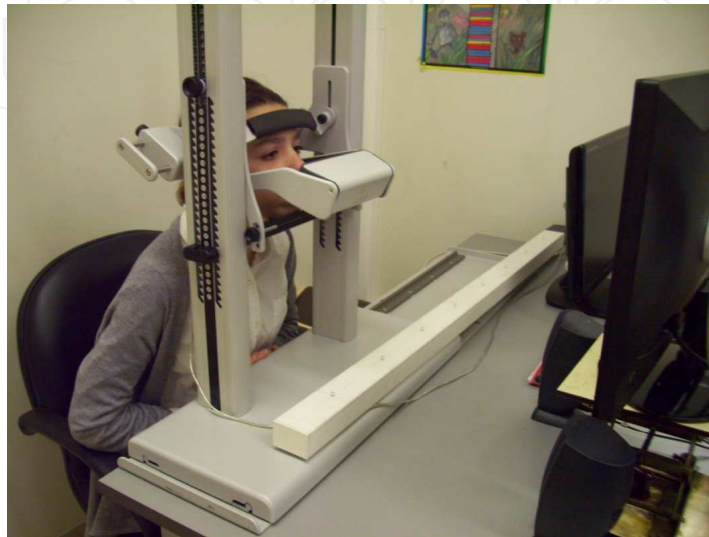
Fig. 2. Mobile Eyebrain Tracker (Mobile EBT®) used to record eye movements from both eyes in children.

2.5 Data analysis

The software MeyeAnalysis (provided with the eye tracker) was used to extract saccadic eye movements from the data. It determines automatically the onset and the end of each saccade. All detected saccades were verified afterwards by the investigator and corrected/discarded if necessary.

The latency and the gain (saccade amplitude/mirror target amplitude) of correct responses and of wrong responses, as well as the percentage of correct antisaccade responses were analyzed in the three different groups of children. Saccades with latencies inferior to 100 ms were counted but not included in the analysis.

Statistical analysis was performed by a three-way ANOVAs using the three groups of children (dyslexics and non-dyslexics, chronological and reading-age matched) as inter-subject factor.

3. Results

The ANOVA showed a main effect of age ($F_{(2,71)}=130.9$, $p<0.001$). Post hoc comparisons showed that reading age matched non-dyslexic children (ND 7-9) were significantly younger than the two other groups ($p<0.001$). There was no age difference between the group of dyslexic children (D 10-13) and the group of chronological age-matched non dyslexic children (ND 10-13) ($p=0.22$).

Figure 3 shows the mean latency of antisaccades for each group of children examined (dyslexic children 10-13 years (D 10-13), non dyslexic children, 7-9 (ND 7-9), and 10-13 years old (ND 10-13) respectively).

The mean latency value for correct antisaccades was 337 ± 14.7 ms for the group of dyslexic children and 353 ± 14.0 ms and 282 ± 12.5 ms for the group of younger and older non dyslexic children respectively.
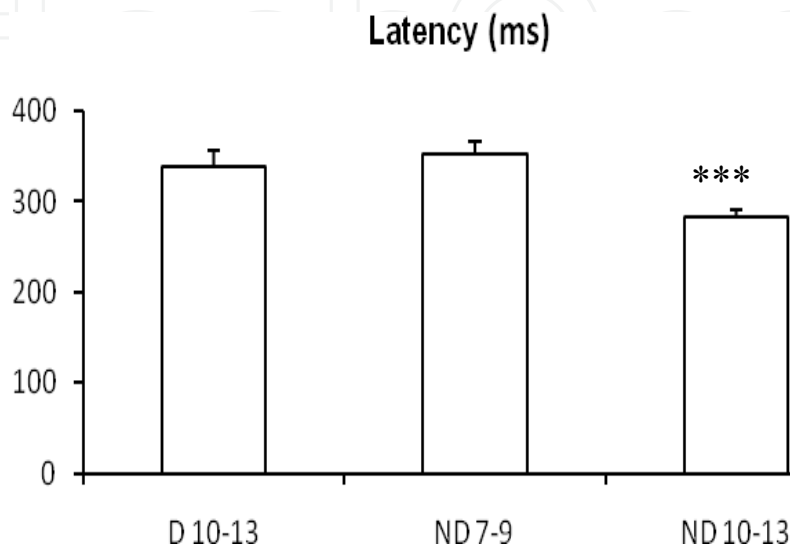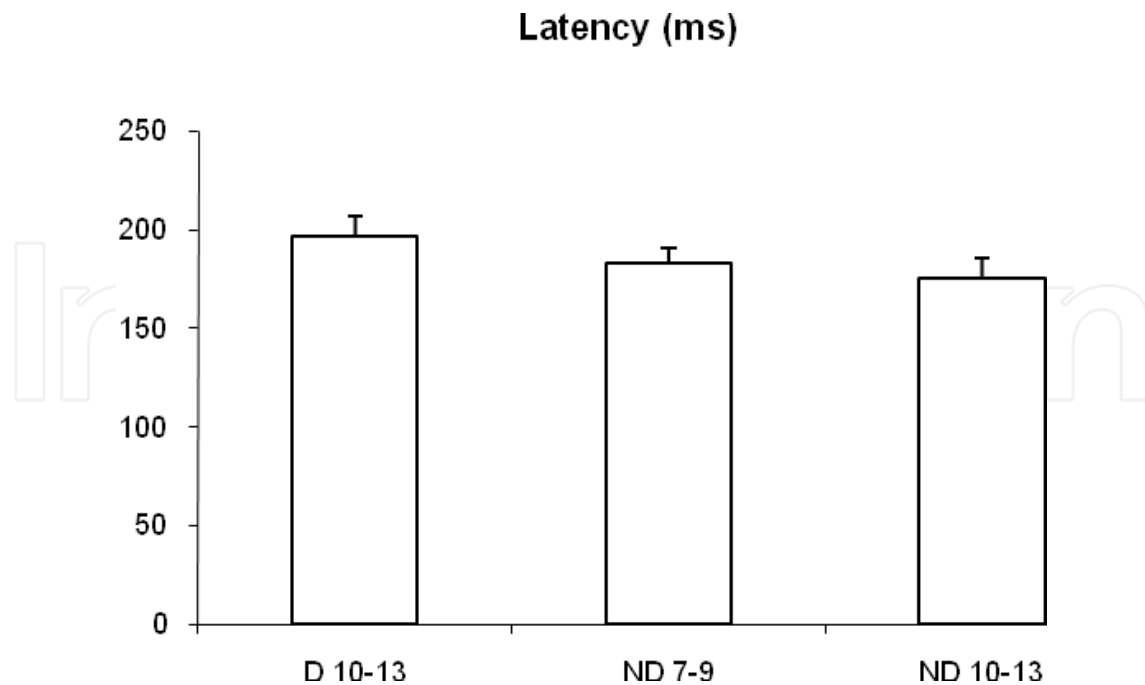

Note: Vertical lines indicate standard error. *** = p<0.01.

Fig. 3. Mean latency of antisaccades for dyslexic children 10-13 years old (D 10-13) and non dyslexic children 7-9 years old (ND 7-9) and 10-13 years old (ND 10-13), respectively.

The ANOVA showed a significant main effect of group, $F_{(2,71)}$ = 8.18, p<0.0006 on the latency of antisaccades. Post hoc comparison showed that the latency of antisaccades of the older group of non-dyslexic children was significant shorter with respect to the group of dyslexic children (p<0.01) and to the younger group of non dyslexics (p<0.0001). The latency of dyslexics was similar to that of non-dyslexic reading age matched children (ND 7-9) (p=0.73).

The mean latency value measured for saccades in the wrong direction (prosaccades towards the target) is showed in Figure 4. The mean value was 196 ± 10.2 ms for the group of dyslexic children and 182 ± 9.5 ms and 175 ± 8.8 ms for the group of younger and older non dyslexic children. The ANOVA showed no significant main effect of group ($F_{(2,71)}$ = 1.18, p=0.31).

For each group of children tested we counted also the frequency of anticipatory saccades (latency < 100 ms). The ANOVA did not show group effect ($F_{(2,71)}$=1.60, p=0.20). Dyslexic children (D 10-13) made 5.7 ± 1.4 % of anticipatory saccades; while reading age matched (ND 7-9) and chronological age matched non-dyslexic children (ND 10-13) made 3.8 ± 1.3 and 2.4 ± 1.2 % of anticipatory saccades, respectively.

In Figure 5 the gain of correct and wrong antisaccade trials are shown for the different groups of children. The ANOVA revealed that a main effect of group was approaching significant for the gain of the antisaccades ($F_{(2,71)}$ = 2.97, p<0.057) but this was not significant for the wrong prosaccades ($F_{(2,86)}$ = 0.72, p=0.48).

Latency (ms)

Note: Vertical lines indicate standard error.

Fig. 4. Mean latency of wrong prosaccades (towards the target) for dyslexic children 10-13 years old (D 10-13) and non dyslexic children 7-9 years (ND 7-9) and 10-13 years old (ND 10-13).
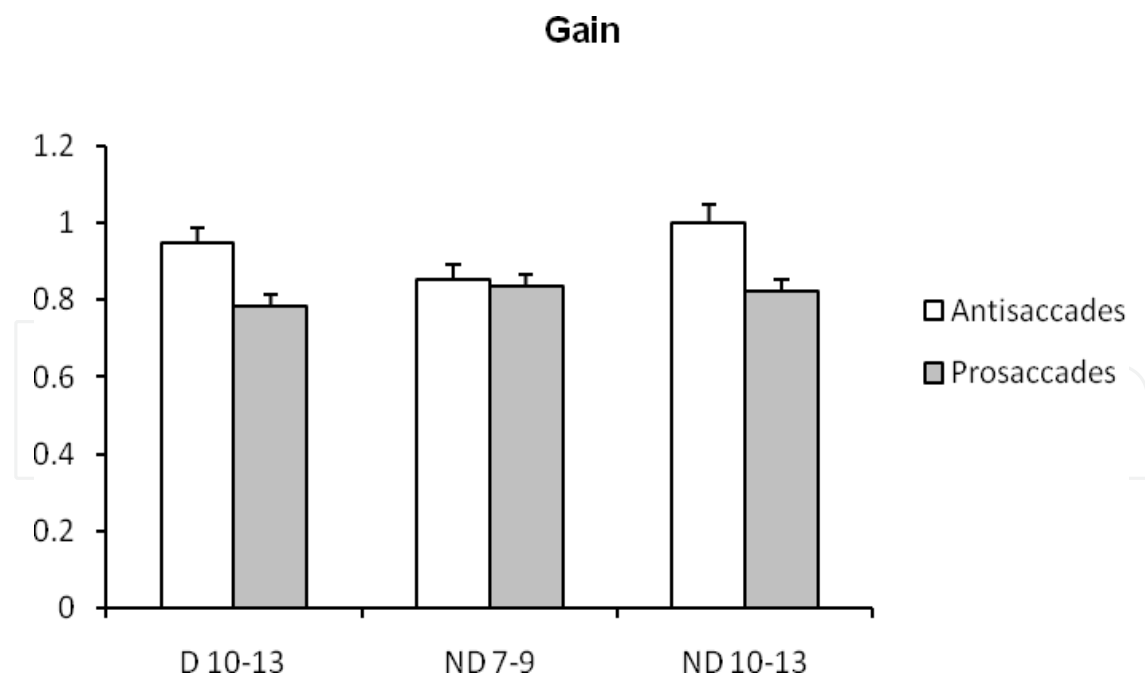
Gain

Note: Vertical lines indicate standard error.

Fig. 5. Gain (amplitude of eye movements/amplitude of the attended position target) for antisaccades and wrong prosaccades for dyslexic children 10-13 years old (D 10-13) and non dyslexic children (younger and older, 7-9 (ND 7-9) and 10-13 years old (ND 10-13), respectively).

The mean error rate was also examined (see Figure 6). The mean error rate was 50.8 ± 4.4 % for the group of dyslexic children and 63.3 ± 4.2 % and 30.3 ± 3.8% respectively for the group of younger and older non dyslexic children.
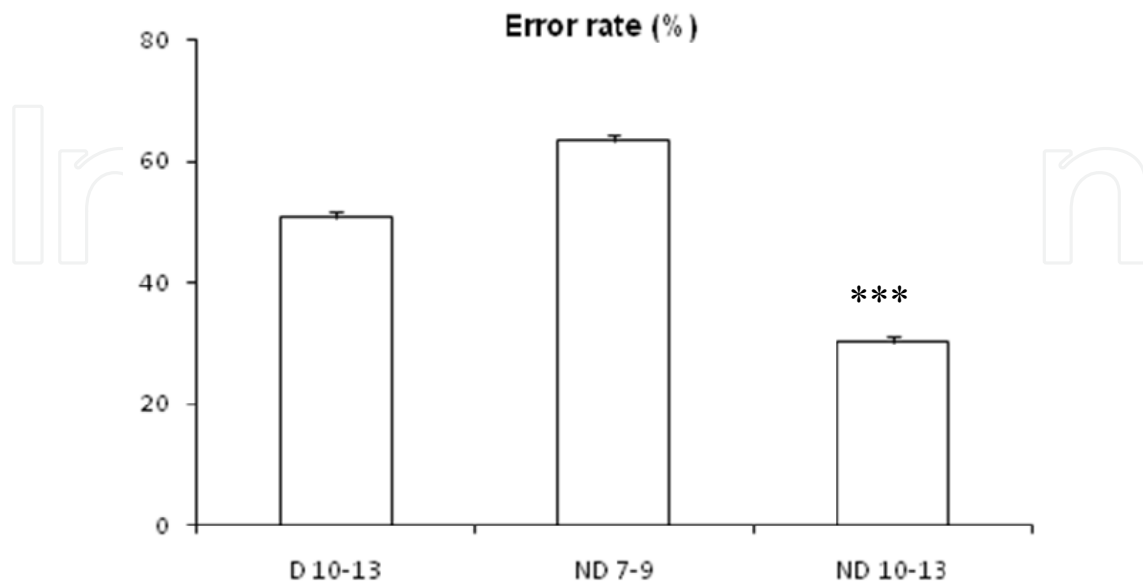
Note: Vertical lines indicate standard error. *** = p<.0003.

Fig. 6. Mean error rate in antisaccades for dyslexic children 10-13 years old (D 10-13) and non dyslexic children (younger and older, 7-9 (ND 7-9) and 10-13 years old (ND 10-13), respectively).

The ANOVA on error rate showed a significant main effect of group, $F_{(2,86)}$ = 17.88, p<0.0001. Post hoc comparison showed that the error rate for the older group of non-dyslexic children was significantly lower with respect to the other groups of children: p<0.003 for the dyslexics and p<0.0001 for the younger non dyslexic children. There was no difference between the non-dyslexic younger group and the dyslexic group (p=0.10).

4. Discussion

The present study showed first that dyslexic children performed the antisaccade task differently to chronological age matched non-dyslexic children: the latency values of correct antisaccades were longer; furthermore the error rate for dyslexic children was significantly higher compared to that of non dyslexic children of similar age. Secondly, this study showed that in non-dyslexic children the performance in the antisaccade task improved with age.

Both results lend support to the previous studies conducted by Fischer's group with dyslexic children (Biscaldi et al., 2000; Fischer & Hartnegg, 2000a) and also other studies with normal children conducted by Fukushima et al. (2000) and by Irving et al. (2009). Note also that in this study the mean latency values of wrong prosaccades were similar in all three groups of children tested. This finding is only apparently in contrast with developmental evidences showing that latency of saccades is age dependent (see Leigh & Zee, 2006 for review). Indeed, in all developmental studies exploring latency of saccades, children had to saccades as quickly as possible to the target (by making a prosaccade) and it

is well known, particularly in the case of children, that latency value depends on the subject's attention and motivation (Clark, 1999). In the present study child had to perform an antisaccade task and the latency here reported for prosaccades is due to a wrong response. Note that a similar finding has been also reported from the study of Munoz et al. (1998).

The new important finding of the present study comes from the comparison between dyslexic children with reading age matched non-dyslexic children. Indeed, the oculomotor behavior of the group of dyslexic children 10-13 years old was similar to that observed in the group of reading age matched non-dyslexic children (7-9 years old). Both the latency values of correct antisaccades and the error rate in the antisaccade task of dyslexic children 10-13 years old were similar to those found in reading age matched non dyslexic children (7-9 years old).

During saccade latency, it is assumed that several processes occur such as the shift of the visual attention to the new stimulus, the disengagement of oculomotor fixation, and the computation of the new parameters (Fischer & Ramsperger, 1984; Findlay & Walker, 1999). These processes involve different cortical and sub-cortical areas (see Leigh & Zee, 2006 for a full review). The longer saccade latency has frequently been attributed to an underdeveloped related cortex, and some investigations have also suggested that increased latency of saccades is related to difficulty in controlling visual fixation (Munoz et al., 1998).

To perform an antisaccade it is necessary to first inhibit the reflexive response towards the stimulus, and then to prepare a voluntary saccade in the opposite direction (antisaccade). Klein (2001), and Klein and Foerster (2001) reported that the capability to inhibit this type of saccade as well as the circuitry controlling cognitive processes is present as early as at 6 years old. They suggested that what is immature in young children is the capability to use such cognitive facilities, leading to a partially correct antisaccade response but to an overall impaired general performance for this task.

Malone and Iacono (2002) hypothesized that although young children have adequate working memory capability to perform correctly on the antisaccade task, they might not be capable of maintaining these instructions continuously throughout the course of the experiment. This may explain why young children in the current study showed long latencies and a high error rate in the antisaccade task.

On the other hand, it is also well known that the parietal cortex, the frontal eye field, the supplementary eye fields and the prefrontal cortex play important roles in antisaccade performance (Luna et al., 2008; McDowell et al., 2008). Further, the inferior parietal cortex has been suggested to be important for the inhibitory period preceding an antisaccade movement (Ettinger et al., 2008) and regions in the intraparietal sulcus (within parietal cortex) could be responsible for generating a correct antisaccade response (Clementz et al., 2007; Nyffeler et al., 2007).

Based on all the available evidences, we postulate the hypothesis that in dyslexic children the delayed maturation of all these structures could lead to longer latencies and increased error rate in the antisaccade task, similar to those reported in reading age matched non-dyslexic children.

Furthermore, it should be noted that the limited fusional amplitude in divergence and convergence capabilities reported in dyslexic children with respect to the two groups of non dyslexic children found with the orthoptic tests is also in favour of a general immaturity of the cortical structures controlling the oculomotor system. Indeed, fusional vergence capabilities are age dependent (Scheiman et al., 1989; von Norrden and Campos, 2006) and at the cortical level some studies showed evidence of vergence control. For instance, the study of Gamlin & Yoon (2000) identified an area close to frontal eye field containing cells that discharge before and during vergence movements. More recently, Quinlan and Culham (2007) with an fMRI study showed an activation of parietal and occipital cortex while humans performed convergence. Thus, in the light of the existing physiological evidence for cortical control of vergence both in monkeys and in humans, the results of the clinical tests presented here in dyslexics suggest immaturity of the neuro-physiological circuitry responsible for generating vergence movements that are closer to the structures for generating saccades.

Finally, it should be noted that orthoptic training is widely used by clinicians for improving vergence capabilities (e.g., von Noorden & Campos, 2006). van Leeuven et al. (1999) and Bucci et al. (2004) reported objective studies on eye movements recordings in children, showing an improvement of vergence eye movements performance after orthoptic training. Consequently, orthoptic training could be applied also for dyslexic population.

5. Conclusion and future directions

The deficits in oculomotor behavior reported in dyslexic children seem to be due to the immaturity of their adaptive mechanism. We believe that visual attentional training along with oculomotor training could help dyslexic children to override such deficiencies allowing an appropriate control of the triggering and execution of saccadic eye movements. We hope to develop new training techniques resulting from this principle to help dyslexic children.

6. Acknowledgments

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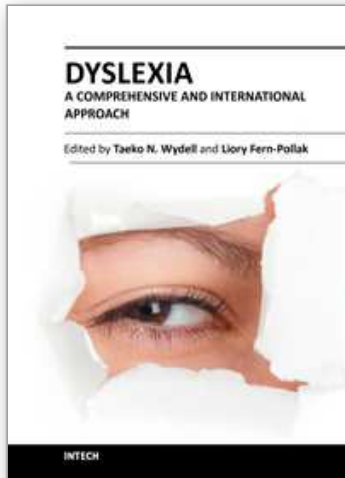
Dyslexia - A Comprehensive and International Approach
Edited by Prof. Taeko Wydell

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