

Evidence for Ocular Motor Deficits in Developmental Dyslexia: Application of the Double-Step Paradigm

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PURPOSE. Dyslexia is a language-based learning disability characterized by difficulties with reading, spelling, and writing. Persons with dyslexia often have deficits in processing rapid temporal sensory information. There is also evidence of sensorimotor deficits in persons with dyslexia. Whether these deficits include ocular motor problems is still an open question. Some previous studies have shown an increased saccadic latency in dyslexics, whereas others have not reproduced this finding. The purpose of the present study was to investigate saccadic latency in young adults with dyslexia during the double-step paradigm, a task that requires rapid sequential visual information processing and saccade generation. The study hypothesis was that dyslexics have a longer saccadic latency in the second orthogonal saccade, a task that nondyslexics parallel process and perform rapidly.

METHODS. Eight students with dyslexia and eight age-matched control subjects participated in the study. Their eye movements were monitored with the scleral search coil technique in simple saccade trials and in the double-step paradigm. The second saccade was either orthogonal or colinear to the first. Intersaccadic interval and latency were calculated for the second saccade.

RESULTS. No difference in saccadic latency was found for colinear second saccades; however, dyslexics had significantly longer latencies for orthogonal second saccades. This included a subset of subjects who had longer latencies for orthogonal than for colinear saccades.

CONCLUSIONS. The findings indicate that under certain conditions, when the demand for rapid visual information processing is high and a rapid saccade sequence is required, some persons with dyslexia show ocular motor deficits manifested by longer saccadic latencies. (*Invest Ophthalmol Vis Sci.* 2006; 47:4401–4409) DOI:10.1167/iovs.05-1657

The etiology of developmental dyslexia has been under investigation for over a century. Because reading is a spatiotemporal process that begins with decoding serial visual

information, a great deal of research has focused on the different levels and skills of visual information processing and responses to nonorthographic tasks. An important finding was that persons with dyslexia often have deficits in processing rapid temporal sensory (visual and auditory) information when compared with normally achieving readers.¹ Lovegrove and Brown² presented evidence that at each age, reading-disabled children have a significantly longer visual information storage time and a significantly slower rate of visual information transfer than do age-matched normally achieving readers, and that this deficit apparently persists into adulthood.³ It has also been demonstrated that both children and adults with dyslexia are significantly worse than normally achieving readers in tasks that require fast, sequential visual processing as opposed to tasks in which the visual information is displayed all at once.⁴ It is intuitive that deficits in the perception of rapid visual information will impact on a person's reaction time, and indeed there is evidence for longer reaction times for eye movements,^{5,6} hand movements, and vocal responses⁷ to rapid visual changes in persons with dyslexia compared with normally achieving readers.

Various hypotheses have been suggested to explain the presence of sensorimotor disorders in persons with dyslexia. One such theory, the "magnocellular theory," postulates a deficit in the magnocellular neuroanatomy and neurophysiology.⁸ The magno cells project via two different pathways to the superior colliculus (SC) and to area V5, also known as MT. The SC seems to be specialized for rapid responses and is more sensitive to sudden changes in illumination than to stationary stimuli. Area V5 appears to specialize in the detection of the speed and direction of motion.⁹ With a variety of techniques (e.g., psychophysics, fMRI, lesions, postmortem surgery) several studies have shown, both in human and nonhuman primates, that dysfunction of the magnocellular system leads to deficits in fixation and eye movements.^{5,10,11}

The "fast temporal deficit hypothesis" focuses on the limited ability of persons with dyslexia to process rapid sequential auditory and visual stimuli.^{12–16} This may be related to the magnocellular theory, because magno cells are particularly sensitive to high temporal frequencies and to visual change.¹¹ Alternatively, others suggest that persons with dyslexia have a basic sequential sensory processing deficit, unrelated to dysfunction of the magno cells, that influences the subsequent levels of processing, resulting in their having a limited ability to retain and compare perceptual traces across brief intervals.¹⁷ The mechanism of this deficit is not clear and may be related to an underlying problem in the ascending sensory pathways, short-term memory, or attention-shifting ability.¹⁸

A relatively new theory connects the more wide-ranging difficulties in dyslexia and explains them by the cerebellar-deficit hypothesis. This hypothesis suggests that many deficits associated with dyslexia, including the core phonological difficulties^{19,20} and the poor general motor control—such as automatization,²¹ time estimation,²² and speeded performance²³—are caused by abnormalities within the lateral parts of the posterior lobe of the cerebellum.²⁴

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Whether persons with dyslexia have ocular motor deficits in addition to their deficits in rapid visual information processing is still controversial. Some researchers have found differences in mean saccadic reaction time, stability of fixation, and number of regression movements between persons with and without dyslexia on nonorthographic tasks^{3,6,25} whereas others did not find these differences.^{26–28} Because of the inconsistent findings, sequenced saccadic eye movements are not considered diagnostically useful for early detection of dyslexia.²⁹ We speculate that only under certain conditions, such as those that require a very rapid sequence of visual information processing, will the two groups consistently differ in the reaction time of their eye movements.

For many years, eye-movement studies of dyslexia recorded only the horizontal component of each eye movement and concentrated on tasks not representative of those required during reading. Elterman et al.³⁰ were the first to establish the necessity of recording both horizontal and vertical eye movements to interpret the eye-movement patterns properly during reading paragraphs of different difficulties. Without vertical eye position, it is impossible to determine the true positions of the eyes. In addition, they isolated the ocular motor aspect of the reading process by using a symbol-driven task that mimicked the normal reading pattern. Their results showed that a variety of disturbances underlie so-called pure dyslexia, in that some dyslexic children demonstrated an ocular motor disability during reading but not on the symbol pattern, some had abnormal patterns on the symbol task and during actual reading, and some had no ocular motor abnormality. In the years since, eye movement studies in dyslexia have built on their results but have also come full circle and again are looking at basic tasks that do not directly reflect reading patterns, and many of the studies continue to produce conflicting results (see Rayner,³¹ for a summary). Although reading is self-paced and uses a staircase paradigm, our study involves a target-driven, double-step paradigm. Reading is a complex task in which the visual system has to process and integrate multiple stimuli. Previous studies³² have shown the utility of the double-step task in studying parallel processing. Our motivation was not necessarily to use movements necessary for normal reading, but rather to use a task that probes for a deficit in the parallel processing of saccades, which can indicate the degree with which the subject can cope with fast visual information processing. The speed of processing of the visual-orthographic system is known to be important in the reading process.³³

In the present study, we investigated the temporal performance of the saccadic system in persons with developmental reading disabilities (PRD) compared with normally achieving readers (NAR). In the experimental task, the ocular motor system performed either: one simple saccade, a slow sequence of two saccades, or a rapid sequence of two saccades in the “double-step paradigm.” This paradigm is a tool that has been used for decades to probe how the ocular motor system processes visual information for multiple saccadic sequences to nonlinguistic targets.³⁴ It has been demonstrated that the preparatory process of two sequential saccades may overlap in time³² and that this occurs more efficiently when the two saccades are orthogonal and not colinear, resulting in shorter latency second orthogonal saccades.³⁵

It should be noted that the term “double-step saccade task” is used in the literature in two different ways. Heide et al.,³⁶ used a double-step task in which the target rapidly stepped twice and then disappeared before the first saccade was executed. The utility of that paradigm is that it allows a separation of the second target’s retinal vector from its saccadic motor vector. The paradigm we used was described in the classic paper by Becker and Jurgens,³² and in it the target steps to its

second location before any reaction of the eye, and then remains visible at that final position. The usefulness of this paradigm is its ability to probe for parallel processing in the programming of sequential saccades. It is this ability of dyslexics that we were interested in, and it is thus this version of the paradigm that we used.

The double-step task forces the saccadic system to process visual stimuli rapidly in parallel and to program two saccades in rapid succession, thus placing a significantly heavier demand on the ocular motor system than required for a single visually guided saccade. In light of the debate regarding the ability of PRDs to generate rapid sequences of saccades, it is thus of interest to explore how these persons cope with this task. The double-step paradigm is also directly relevant to the study of reading disabilities because during reading, the ocular motor system is required to generate a series of rapid and accurate saccades. It should be noted, however, that the double-step paradigm requires only two sequential saccades and not multiple sequential saccades as might be required during reading. Our hypothesis was that the PRD group would have longer saccadic latencies for the second orthogonal saccade in the double-step paradigm when compared with the NAR group and that they would not show a significant difference between second orthogonal and second colinear saccades.

Some of these findings have been presented in abstract form.³⁷

METHODS

Subjects

Eight PRD (all men; mean age 28 ± 2.7) and eight age-matched NAR (5 women, 3 men; mean age 27 ± 2.9) subjects participated in the study. They were recruited by notices placed on a university campus and by direct mailing using a database from the university center for assisting students diagnosed with learning disabilities. All PRD participants had psychoeducational diagnoses of a developmental reading disability. All subjects had a minimum of several years of university education. The criterion for inclusion in the PRD group was a score on the speed of pseudoword reading test (described later) that was at least 1 SD above the average score for the control group. All subjects performed within the normal range on the matrices subtest of the Wechsler Adult Intelligence Scale (WAIS-III).³⁸ Performance on other subtests was not a basis for participants’ exclusion. All were tested on the Snellen visual acuity test. Monocular and/or binocular visual acuity was within normal range. All participants were native Hebrew speakers and naive to the purpose of the study.

Psychometric Tests

The following psychometric tests provided the aptitude and achievement profiles of the subjects.

Cognitive Measures. The tests used were all from WAIS-III.³⁸ An estimation of intelligence abilities was derived from the matrices subtest of the WAIS-III that resembles the Raven’s Advance Progressive Matrices (APM) which is very highly g-loaded.³⁹ The digit span (verbal short term memory) and digit-symbol coding (visual motor coordination and short-term visual memory) of the WAIS-III test were used to screen participants for distractibility.⁴⁰

Reading Measures. The reading measures used were speed and accuracy of reading lists of single words (measured in words per minute, WPM),⁴¹ pseudowords per minute (PWPM),⁴² and reading rate of an academic level passage (PASS speed).⁴³

Orthographic Skill. A test of the written spelling ability of each participant was administered. The score was based on the number of spelling errors.⁴⁴

Phonological Awareness. Hebrew translations of the spoonerism⁴⁵ and Pig Latin²⁸ tasks were used. The score was based on the number of errors.⁴⁶

Eye Movement Monitoring and Analysis

The experimental paradigm and data collection were under the control of “virtual instruments” written in commercial software (LabView; National Instruments, Austin, TX). Horizontal and vertical gaze of one eye were measured by the magnetic search coil technique⁴⁷ with 6-ft field coils (CNC Engineering, Seattle, WA) and a scleral search coil embedded in a silastic ring (Skalar, Delft, The Netherlands). The coil was placed on the eye after application of one or two drops of local anesthetic (benoxinate HCl, Localin, Fischer Laboratories, Tel Aviv, Israel). Coil signals were filtered (bandwidth, 0–200 Hz) before digitization at a 1000-Hz sampling rate, with 12-bit resolution. The visual stimulus was a dim (3 mW with a 100× neutral density filter) red laser spot (1.5-mm diameter) rear-projected onto a semitranslucent screen situated 1.1 m in front of the subject. The position of the laser spot was determined by an X-Y galvanometer (General Scanning, Billerica, MA). The fly-time of the mirrors is 2 to 3 ms, and they settle to their final location within 5 to 6 ms. The subject sat in front of the semitranslucent screen in a dark room and was given time to adjust to the dark.

Each experimental session of 30 to 40 minutes consisted of between 15 and 30 blocks of trials, with each block containing 10 to 25 trials. Each trial began with a red laser spot in the center of the display (1 in Fig. 1), on which the subject was instructed to fixate. The subjects were instructed to track the target as accurately and as rapidly as possible whenever it moved. After 1500 to 3000 ms the target made a purely horizontal jump of 12° (2 in Fig. 1) randomly to the right or left. In the single-saccade catch trials (e.g., Fig. 2D), the target remained at that location for 1500 ms and then returned to its starting position. In the double-step saccade trials, the target remained at this position (2 in Fig. 1) for one of three possible interstimulus intervals (ISIs) and then jumped 5 deg either colinear (Fig. 2A) or orthogonal (Figs. 2B, 2C) to the first target jump (Fig. 1, positions 3). The three ISIs were 150, 180 (both are “medium” ISI) and 250 ms (“long” ISI). It remained at the final location approximately 1300 ms and then jumped back to the origin. Each subject performed a minimum of 25 saccades of each type. The presence and direction of the second saccade as well as the ISI were random.

Analysis of the data was performed using with another commercial program (MatLab; The MathWorks, Natick, NA). Saccade detection was performed automatically and then verified by the investigator manually. A rough estimate of saccade location was obtained from the raw data by using a Haar wavelet transform. The advantage of using a wavelet is that at the appropriate scale the saccade signal is enhanced and the fixation noise is “filtered” out, thus causing the saccade location to be easily identifiable. Then, the beginning and the end of the saccade were precisely determined from the absolute velocity. Starting from the location of the velocity peak, the saccade’s beginning

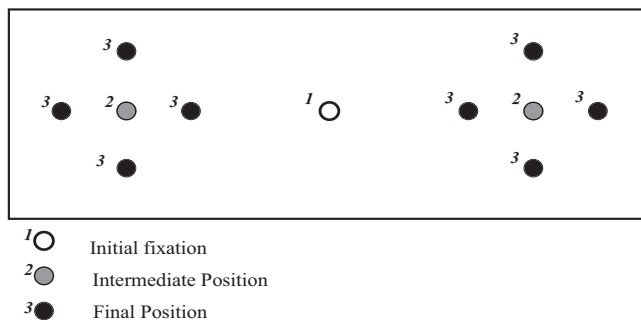


FIGURE 1. Diagrammatic representation of the spatial events during the individual trials. Each trial started with a target at the central location, marked 1. The target then jumped to one of the two positions labeled 2, where it either remained for the duration of the trial, or next jumped to one of the four surrounding positions labeled 3. This figure is for those trials in which the initial saccade was horizontal. For those in which the initial saccade was vertical the entire figure should be rotated through 90°.

and end were taken as the points where the velocity’s magnitude exceeded and decreased below noise level, respectively. The noise level was calculated from the peaks of the velocity magnitude’s distribution of all sampling points.

Our primary interest was to measure the saccadic latency of the second saccade in a double-step saccade. For this second saccade, latency was defined by two measures. This was necessary because for medium ISIs most subjects began the first saccade after the target had already completed its second step (as in Figs. 2A, 2B). In the first method, “reaction time” (RT), latency is defined as the time from the second target step to the beginning of the second saccade (*a* in Fig. 2A). In the alternate approach, “intersaccadic interval” (ISI), latency is defined as the time from the end of the first saccade to the initiation of the second saccade (*b* in Fig. 2A).

Magnocellular tasks

Stimuli and Procedure. The following two tasks were designed to examine magnocellular performance. A Gabor patch stimulus in two directions (45° and 135°) was used in a two-alternative, forced-choice (2AFC) paradigm to assess contrast detection thresholds. Contrast detection was varied in a two-down/one-up adaptive staircase procedure, converging on 71% correct.⁴⁸ Contrast was increased by 1 dB after an incorrect response, and decreased by 1 dB after two consecutive correct responses. The stimulus contrast was defined as $(L_{\max} - L_{\min}) / (L_{\max} + L_{\min})$, where L_{\max} and L_{\min} are the maximum and minimum luminances, respectively (Michelson contrast). All tasks included “catch trials” in which the Gabor patch had a permanent high contrast of 50%, and all subjects performed close to 100% on the catch trials. The viewing distance was 90 cm. The interval between a response and the next trial varied randomly between 1.0 and 1.5 seconds.

M-Selective Task. Contrast-detection thresholds of same-different judgment were measured for a 0.5-cyc/deg Gabor patch ($\sigma = \lambda = 2^\circ$). The stimuli were presented on the center of the screen with a low mean luminance of 5.7 cd/m². The subjects were asked to indicate via a button push the orientation of the lower part of the Gabor patch. The Gabor patch was displayed for 500 ms, with a flicker frequency of 10 Hz.

M-Selective Saccade Task. To further explore whether there is truly a magnocellular deficit, we added a second component that is known to be part of the magnocellular functions (i.e., saccadic eye movements).¹ Contrast-detection thresholds of same-different judgment were measured for a Gabor patch that appeared to jump from side to side. This procedure was repeated twice with different types of temporal variations: flicker, which has been demonstrated to reveal processing impairments in PRD,⁴⁹ and rotation, which is known to be processed by medial superior temporal (MST) area in the extrastriate cortex,⁵⁰ a region that receives input mainly from magno cells.⁵¹ The three serial Gabor patches in this experiment were not displayed on the center of the screen, but 2° to the left or to the right of the center randomly and it then jumped twice from side to side (right-left-right or left-right-left). The subjects were asked to indicate by means of a button push whether the three displays had the same or different orientations. Each Gabor patch was displayed for 500 ms with a low mean luminance of 5.7 cd/m² and had a spatial frequency of 0.5cyc/deg ($\sigma = \lambda = 2^\circ$). The flicker frequency was 10 Hz, and the rotational frequency was 10 Hz, with an amplitude of 45°, with the center at either 45° or 135°.

Apparatus

The psychophysical tasks were administered in a dark room, and the subjects were given several minutes in which to dark adapt. We used the VSG2\5 system (Cambridge Research Systems Ltd., Rochester, UK) for generating the stimuli that were displayed on a 21-in. video monitor with a frame rate of 170 Hz (model GDM-F520; Sony, Tokyo, Japan). The experiments were controlled by and the data analyzed using commercial software (MatLab ver. 7.0; The MathWorks). A colorimeter

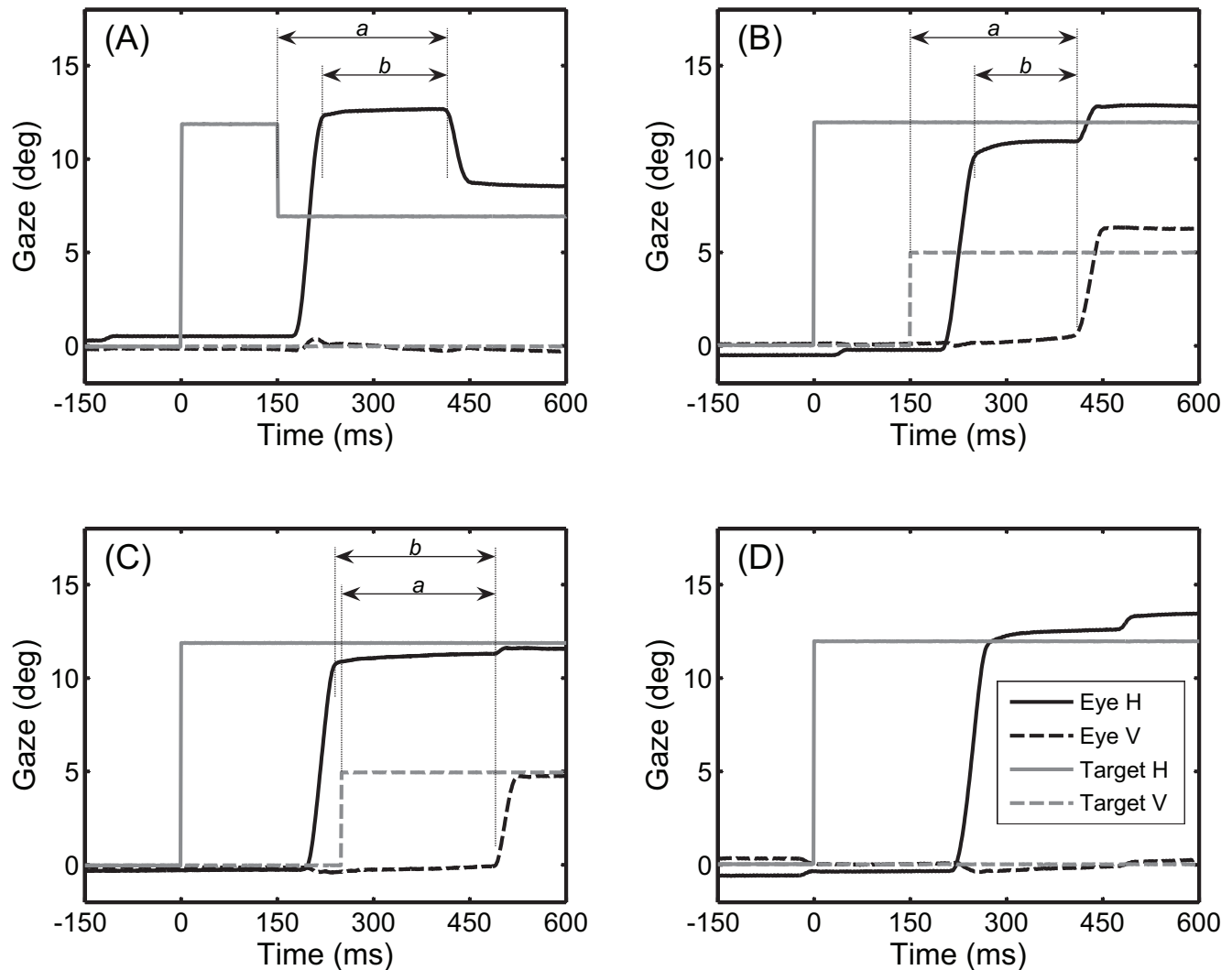


FIGURE 2. Sample data illustrating the various trial types. Positive is rightward-upward motion. (A) Saccade of an NAR participant. The target initially stepped horizontally (rightward) 12°, followed 150 ms later by a backward (leftward) step of 5°. Two latency measures for the second gaze shift are shown. The first, a , is the time from the second target step to the second saccade initiation (i.e., the reaction time). The second, b , is the time from the end of the first saccade to the start of the second (i.e., intersaccadic interval). (B) Saccade of a PRD participant; the initial target step was horizontal (rightward) followed 150 ms later by a vertical (upward) step of 5°. (C) Saccade of an NAR participant; The initial target step was horizontal (rightward) followed 250 ms later by a vertical (upward) step of 5°. Note that, unlike in A and B, the initial saccade occurs before the second target step. (D) Saccade of an NAR participant. A catch trial consisting of a single horizontal target step.

was used to calibrate the screen (ColorCAL; Cambridge Research Systems, Ltd.).

The local Helsinki Committee for human experimentation approved the eye movement measurement technique used in the study, and all subjects gave written informed consent before participation in accordance with the Declaration of Helsinki.

RESULTS

Assessment of Reading and Psychometric Skills

Table 1 summarizes the performance of the PRD and the NAR on the cognitive and reading-related tests along with the statistical significance. PRD and NAR participants did not differ in their mean scaled score on the matrices subtest ($P > 0.05$), typically used to match groups for cognitive abilities. However, NARs performed significantly better than PRDs on the screening factor for distractibility (digit-symbol coding and digit span combined; mean, 11.0 vs. 8.18; $P < 0.01$). As can be seen in Table 1, the PRDs scored significantly lower on the digit-

symbol coding ($P < 0.01$), but not on the digit span ($P > 0.05$). We attribute this to the high level of functioning of the students with dyslexia who participated in this experiment. On all the reading-related tests the PRDs were significantly impaired compared with the NARs. This finding is consistent with previous results.^{52,53}

Psychophysical Measures

Performance on Magnocellular Tasks. As shown in Table 2, the PRD and NAR groups did not differ as on the M-selective task. This means that despite using stimuli that were designed to stimulate magno cells, the two groups showed no difference in their contrast thresholds on this task.

The inability of some experiments to find magnocellular deficits in PRDs has been attributed to the fact that these deficits are subtle and that the magnocellular system is not "all or nothing."⁵⁴ The suggestion is thus that to search for magnocellular impairments in dyslexia one incorporates in the stimulus as many facets as possible that are unique to the

TABLE 1. Results of Psychometrics Tests

	NAR	PRD	<i>P</i> <i>t</i> -Test
Age	28 (2.7)	27 (2.9)	n.s.
Cognitive measures			
Matrices +	14.5 (1.4)	12.8 (3.4)	n.s.
Digit span +	10.2 (2.7)	9.2 (2.9)	n.s.
Digit symbol coding +	11.7 (2.4)	7.1 (1.9)	0.001
Reading measures			
WPM speed	123.7 (17)	75 (22.8)	0.000
PWPM speed	75.7 (12.1)	41 (15.5)	0.000
Paragraph	73.9 (7.8)	119.9 (31.2)	0.001
RAN letter	17.5 (7.1)	26.4 (3.9)	0.008
RAN symbols	32.3 (6.2)	51.9 (11.6)	0.001
RAS	21.1 (2.0)	29.7 (6.4)	0.003
Orthographic			
Spelling	2.9 (2.7)	32.4 (16.2)	0.000
Phonological awareness			
Spoonerism	5.75 (0.7)	3.6 (1.9)	0.011
Pig Latin	5.7 (0.4)	4.0 (2.4)	0.031

Data are expressed as the mean (SD). +, scaled score; NAR, normally achieving readers; PRD, persons with reading disabilities; WPM, words per minute; PWPM, pseudowords per minute; RAN, rapid automatized naming; RAS, rapid alternating stimuli.

magno system. We added to the second task jumping targets that would engage the saccadic eye movement system, which receives its input and some control from the magnocellular system.⁵⁵

ANOVA for repeated measures conducted on contrast-detection thresholds on the M-selective saccade task with group (PRD/NAR) as a between-subject variable and type of temporal frequencies (flicker/rotation) as a within-subject variable, revealed a significant effect only for type of temporal frequencies, $F(1,14) = 4.80$, $P < 0.04$ ($M = 2.37$ and $M = 2.07$ for flicker and rotation, respectively). No significant effects were found for both group, $F(1,14) = 2.05$, $P > 0.10$ and Group \times Type of Temporal Frequencies interaction, $F(1,14) = 0.45$, $P > 0.50$, indicating that PRD's and NAR's thresholds did not differ significantly, despite using various conditions similar to previous studies that revealed magnocellular impairments in persons with reading disabilities.

Ocular Motor Results. Representative raw data of the various trial types can be seen in Figure 2.

ANOVA for repeated measures was conducted on latency of the second saccade on trials with ISIs of 150 and 180 ms with group (PRD/NAR) and direction (colinear, orthogonal) as the between- and within-subjects effects, respectively. The ANOVA showed that the effect of group was not significant $F(1,14) = 0.46$, $P > 0.5$. However, the direction effect was significant $F(1,14) = 25.2$, $P < 0.001$. In addition, there was a significant group \times direction interaction $F(1,14) = 5.2$, $P < 0.05$. Pair-wise comparisons indicated that whereas for a colinear (horizontal) second saccade, PRD and NAR performed

equally well using both latency measures (RT and ISaI), for orthogonal (vertical) second saccades, PRD were significantly slower than NAR for both latency measures (RT and ISaI). The results are summarized in Table 3.

We then asked whether the latency patterns of the eye movements' response to DS target are the same in the PRD group in the NAR group. The results of a paired *t*-test, summarized in Table 4, revealed a significant effect between latencies for orthogonal (vertical) second saccades compared with colinear (horizontal) second saccades for each group and each method of measuring latency (RT and ISaI). The results for the NARs showed highly significant latency differences between horizontal and vertical second saccades, a finding that is consistent with previously reported results.³³ The PRD showed a smaller effect, which will be elaborated on later.

Because there are different subtypes of dyslexia, it is important to analyze each subject's eye movements independently in addition to the group analysis. The results of horizontal and vertical differential latency and the results of paired *t*-test for each subject are summarized in Figure 3. In the NAR group, seven of eight subjects had a significantly shorter reaction time for the orthogonal compared with colinear saccades, and six of eight had a significantly shorter intersaccadic interval for the orthogonal compared with colinear saccades. However, in the PRD group only three of eight subjects had significantly shorter reaction times and intersaccadic intervals between the two directions of the second saccade. These results indicate that almost all the NAR participants (87%) had significantly different reaction times between orthogonal and colinear saccades,

TABLE 2. Performance on Psychophysical Tasks

Visual Tasks	NAR	PRD	<i>P</i>
M-selective task			
M-selective	2.2 (0.1)	2.3 (0.5)	0.500*
M-selective and saccades task			
Flicker	2.0 (0.6)	2.6 (0.8)	
Rotation	1.8 (0.3)	2.2 (0.9)	0.173†

NAR, normally achieving readers; PRD, persons with reading disabilities.

* Two-sample *t*-tests.

† Group effect in a repeated measure ANOVA for group (NAR vs. PRD) by condition.

TABLE 3. Summary of the Results Comparing the Latency of a Second Colinear (Horizontal) Saccade to a Second Orthogonal (Vertical) Saccade between the Two Groups

	Latency
RT-H	
PRD	306 ± 11
NAR	307 ± 19
ISaI-H	
PRD	200 ± 11
NAR	222 ± 13
RT-V*	
PRD	277 ± 9
NAR	240 ± 7
ISaI-V†	
PRD	180 ± 10
NAR	159 ± 7

Data are expressed as mean milliseconds ± SE. RT, reaction time; PRD, persons with reading disabilities; NAR, normally achieving readers; H, horizontal; V, vertical; ISaI, intersaccadic interval.

* $P < 0.01$.

† $P < 0.05$.

whereas in the PRD group, significantly different reaction times was found in less than half of the group (37%).

For ISIs of 250 ms, in most trials both PRD and NAR subjects completed the initial saccade before the second target jump. In other words, the trials appear to be similar to two ordinary back-to-back saccades rather than a rapid double-step sequence. There is thus no parallel processing taking place and there were thus no significant differences between coplanar and orthogonal second saccade latencies in all but one of the subjects.

Often the distribution of saccade latencies has more information than the mere reaction times. To look for such patterns, the time the target spent in a retinal location that required updating for the programming of the second saccade was used as an independent variable to look for its effect on latency. As noted, all analyzed saccades had the second target step before the first saccade occurred. Thus, the period between the second step and the first eye movement is the "update-requiring location" (URL) and correlations between the time spent at the URL and the saccadic latency (both measures: RT and ISaI) were looked for. Because $RT = URL + ISaI$, there will be interdependencies. Some of the subjects showed weak correlations between several of the variables, but there was no consistent pattern between those findings and performance on the double-step task.

DISCUSSION

Summary of the Results

The results support our hypothesis that in specific conditions of very rapid sequential visual information processing, such as required in double-step orthogonal saccades, the PRD participants had longer latencies for the second saccade than did the NAR participants.

Whereas the saccadic system of seven of the eight NAR participants had the capability to generate a faster orthogonal second saccade using parallel processing, only three of eight subjects in the PRD group had this capability. For the other five PRDs no significant difference was found between the second rapid horizontal saccade and the second rapid vertical saccade. In three of those five subjects, the latency for the vertical saccade was actually longer than that for the horizontal saccade. This finding may indicate that under certain conditions, some persons with dyslexia have a slower saccadic reaction.

When Do Dyslexics Exhibit Saccadic Dysfunction?

Our findings support the assertion that there is a deficit in the saccadic eye movements in at least a subgroup of persons diagnosed with developmental dyslexia.^{56,57} Some previous studies examined saccadic eye movements with very long ISIs and found no significant difference between NAR and PRD, and concluded that defective ocular motor control is not associated with nor is it a causal factor in dyslexia and that abnormal eye movements in subjects with dyslexia during reading must thus be related to differences in higher cognitive processes.^{27,29,58-60} The procedure used in the present study (i.e., a very rapid double-step saccadic movement) succeeded in eliciting differences in eye movements between the NAR and PRD groups. Our explanation for why this procedure reveals differences in eye movements between PRD and NAR is that these differences are manifest only when the demands on the saccadic system are high and the system is required to function under relatively stressful conditions. Thus, while the saccadic system of the NAR has the flexibility to deal with those conditions (i.e., sequential saccades and short enough ISI so that the second target step occurs before the ocular motor system has finished producing the initial saccade), the saccadic system of the PRD does not. In this experiment, we did not monitor eye movements during reading, but these results may give us some indication that persons with dyslexia have a limited capability to cope with the stressful conditions of rapid visual sequential information processing and with the generation of saccades compared with normally achieving readers. Normal reading requires rapid processing of visual information and the generation of a sequence of saccades.

Possible Mechanisms for Explaining Longer Saccadic Reaction Times in Persons with Reading Disabilities

There are various possible explanations for the longer saccadic latencies observed in persons with dyslexia in the present study. It is possible that the increased latency is a result of an increased processing time during the perceptual stage, or alternatively, participants with dyslexia may take longer to program and execute the saccadic movement, or it could be a combination of the two. Several theories may provide explanations for the ocular motor deficit in persons with dyslexia when a sequence of saccades is required in response to rapidly presented visual information.

TABLE 4. Summary of the Results Comparing the Latency for Each Direction between PRD and NAR

	Latency
PRD†	
RT-H	306 ± 11
RT-V	277 ± 9
PRD*	
ISaI-H	200 ± 11
ISaI-V	180 ± 10
NAR†	
RT-H	307 ± 19
RT-V	240 ± 7
NAR†	
ISaI-H	222 ± 13
ISaI-V	159 ± 7

Data are expressed as mean milliseconds ± SE. Abbreviations are as defined in Table 3.

* $P < 0.05$.

† $P < 0.01$.

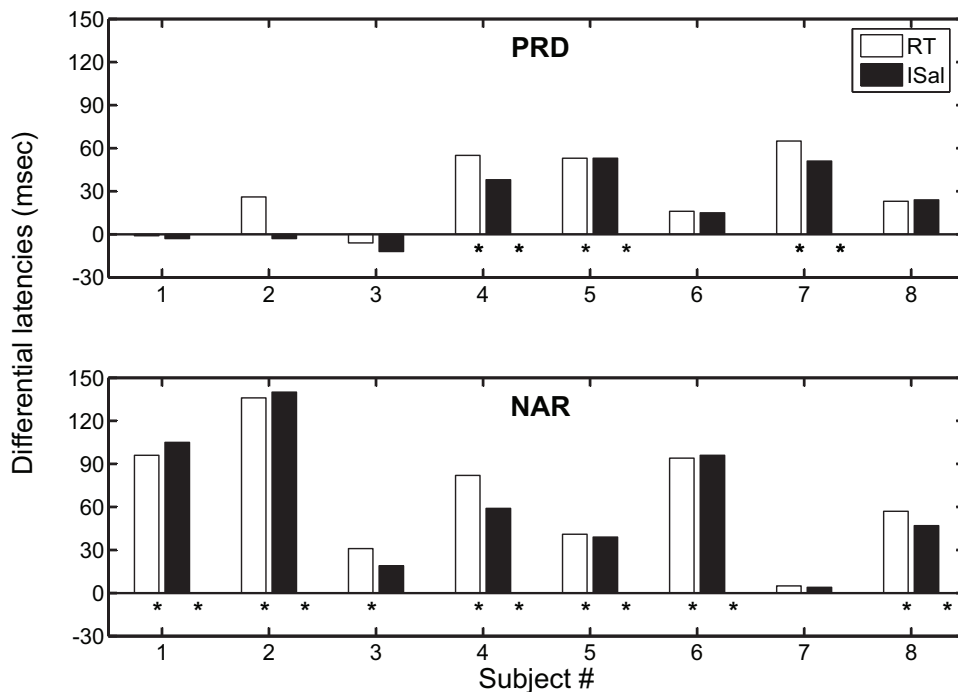


FIGURE 3. Summary of horizontal and vertical differential latency (horizontal minus vertical latency) for each subject. RT, reaction time; ISal, intersaccadic interval. * $P < 0.01$.

The magnocellular theory may explain the differences in the saccadic latencies between the two groups. Deficits of the magno cells which project to area V5 may result in a dysfunction in motion processing.^{61,62} Magno cells also project to the SC. It has been shown that the SC has both sensory and motor maps that lie in register.^{63,64} Based on this observation, it is now known that a major function of the superior colliculus is to generate saccadic eye movements.^{65,66} Thus, a dysfunction of the magno cells may lead to a disruption in the functioning of the SC, an area where a deficit may reduce the ability to perceive motion and/or to program and execute saccadic movements. The magnocells also play a prominent role in the bottom-up direction of attention.⁶⁷ It has been demonstrated that due to a weaker magnocellular system, the speed with which deficient readers can disengage and reengage attention on new targets is reduced compared with that of normally achieving readers.⁶⁸ All those dysfunctions of magno cells could result in a longer saccadic latency under conditions that require a very rapid eye movement response. However, it should be noted that we did not find a difference between the groups on the small number of magnocellular tests that we performed. Although several of the PRD differed significantly from the NAR group, they were not all part of the same subgroup as regards the saccadic findings. Of note is that the subject who most differed from the NAR group showed a saccadic effect opposite that seen in the NARs. This does not rule out a role for the magnocellular system in contributing to dyslexia or to our saccadic findings and it is possible that with additional tests the magnocellular deficit would reveal itself.

Another relevant theory is the "fast temporal deficit hypothesis" that postulates a deficit in temporal processing which is believed to be one of the prime factors affecting reading ability. Previous studies have shown that the reading disabled population has a longer visual persistence, which means that they need longer stimulus separations due to ongoing neural activity^{2,69} and that the greater difficulty in visual and auditory frequency discrimination is related to the interstimulus interval rather than to intrastimulus variability.¹⁷ Cumulative evidence suggests that a reduced capacity to process rapidly presented stimuli in a number of sensory modalities interferes with the reading process.^{16,70}

An alternative explanation for our findings could be the "cerebellar deficit hypothesis." Recent studies demonstrate the importance of the cerebellum for language processing.^{71,72} It has been shown that cerebellar damage can cause deficits in attention and working memory⁷³ and that it can cause dyslexia-like symptoms during reading.⁷⁴ It has further been demonstrated that the cerebellum is an indispensable component of the saccadic system that participates in the programming and execution of saccadic eye movements.⁷⁵ Because of its critical role in the generation of saccades, it may be suggested that dysfunctions of the cerebellum leads to a longer saccadic latency.

CONCLUSIONS

We suggest that persons with dyslexia may have abnormal saccadic eye movements when the saccadic system is required to deal with highly demanding tasks, such as a rapid sequential double-step movements and as perhaps is required during reading. The question of the underlying mechanism awaits further research, and in searching for the underlying mechanism, it must be kept in mind that all dyslexics are not the same. We found these deficits in a group of adults with higher education. We can speculate that those deficits are more prominent in average adults with dyslexia and in children with dyslexia. This research demonstrated that under specific stressful conditions, some adults with dyslexia have deficits in their ocular motor response to rapid visual information.

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References

1. Stein J, Talcott J. Impaired neuronal timing in developmental dyslexia: the magnocellular hypothesis. *Dyslexia*. 1999;5:59-77.
2. Lovegrove W, Brown B. Developmental of information processing in normal and disabled readers. *J Percept Mot skills*. 1978;46:1047-1054.
3. Breznitz Z, Meyler A. Speed of lower-level auditory and visual processing as a basic factor in dyslexia: electrophysiological evidence. *Brain Lang*. 2003;85:166-184.
4. Ben Yehudah G, Sackett E, Malchi Ginzberg L, Ahissar M. Impaired temporal contrast sensitivity in dyslexics is specific to return and compare paradigms. *Brain*. 2001;124:1381-1395.
5. Eden G, Stein J, Wood M, Wood F. Differences in eye movements and reading problems in dyslexic and normal children. *Vision Res*. 1994;34:1345-1358.
6. Biscaldi M, Stefan G, Volker S. Poor saccadic control correlates with dyslexia. *Neuropsychologia*. 1998;36:1189-1202.
7. Sigmundsson H. Do visual processing deficits cause problem on response time task for dyslexics? *Brain Cogn*. 2005;58:213-216.
8. Galaburda A, Livingstone M. Evidence for a magnocellular defect in developmental dyslexia. *Ann N Y Acad Sci*. 1993;682:70-82.
9. Coren S, Ward ML, Enns TJ. *Sensation and Perception*. 5th ed. Orlando, FL: Harcourt Publishers; 1999:65-83.
10. Keen A, Lovegrove W. Transient deficit hypothesis and dyslexia: examination of whole-parts relationship, retinal sensitivity, and spatial and temporal frequencies. *Vision Res*. 2000;40:705-715.
11. Stein J. The magnocellular theory of developmental dyslexia. *Dyslexia*. 2001;7:12-36.
12. Boden C, Brodeur D. Visual processing of verbal and nonverbal stimuli in adolescents with reading disabilities. *J Learn Disabil*. 1999;32:58-71.
13. Lovegrove W, Garzia R, Nicholson S. Experimental evidence for a transient system deficit in specific reading disability. *J Am Optom Assoc*. 1990;61:137-46.
14. Williams M, Lecluyse K. Perceptual consequences of a temporal processing deficit in reading disabled children. *J Am Optom Assoc*. 1990;61:111-121.
15. Tallal P. Auditory temporal perception, phonics, and reading disabilities in children. *Brain Lang*. 1980;9:182-198.
16. Tallal P, Miller S, Fitch R. Neurobiological basis of speech: a case for the preeminence of temporal processing. *Ann N Y Acad Sci*. 1993;682:27-47.
17. Amitay S, Ben-Yehudah G, Banai K, Ahissar M. Disabled readers suffer from visual and auditory impairments but not from a specific magnocellular deficit. *Brain*. 2002;125:2272-2285.
18. Ben-Yehudah G, Ahissar M. Sequential spatial frequency discrimination is consistently impaired among adult dyslexics. *Vision Res*. 2004;44:1047-1063.
19. Faust M, Sharfstein-Friedman S. Naming difficulties in adolescents with dyslexia: application of the tip-of-the-tongue paradigm. *Brain Cogn*. 2003;53:211-217.
20. Faust M, Dimitrovsky L, Shacht T. Naming difficulties in children with dyslexia: application of the tip-of-the-tongue paradigm. *J Learn Disabil*. 2003;36:203-215.
21. van der Leij A, van Daal VH. Automatization aspects of dyslexia: speed limitations in word identification, sensitivity to increasing task demands, and orthographic compensation. *J Learn Disabil*. 1999;32:417-428.
22. Nicolson RI, Fawcett AJ, Dean P. Time estimation deficits in developmental dyslexia: evidence of cerebellar involvement. *Proc Biol Sci*. 1995;259:43-47.
23. Compton DL, DeFries JC, Olson RK. Are RAN- and phonological awareness-deficits additive in children with reading disabilities? *Dyslexia*. 2001;7:125-49.
24. Fawcett A, Nicolson R. *Dyslexia: the Role of the Cerebellum*. London: Whurr; 2001.
25. Pavlidis GT. Eye movements in dyslexia, their diagnostic significance. *J Learn Disabil*. 1985;18:42-50.
26. Stanley G, Smith GA, Howell EA. Eye-movements and sequential tracking in dyslexic and control children. *Br J Psychol*. 1983;74:181-187.
27. Brown B, Haegerstrom-Portnoy G, Adams A, et al. Predictive eye movements do not discriminate between dyslexic and control children. *Neuropsychologia*. 1983;21:121-128.
28. Olson RK, Wise BW, Conners F, Rack JP, Fulker D. Specific deficits in component reading and language skills: genetic and environmental influences. *J Read Disabil* 1989;22:339-348.
29. Black J, Collins D, De Roach J, Zubrick S. A detailed study of sequential saccadic eye movements for normal- and poor-reading children. *Percept Mot Skills*. 1984;59:423-434.
30. Elterman RD, Abel LA, Daroff RB, Dell'Osso LF, Bornstein JL. Eye Movement patterns in dyslexic children. *J Learn Disabil*. 1980;13:11-16.
31. Rayner K. Eye movements in reading and information processing: 20 years of research. *Psychol Bull*. 1998;124:372-422.
32. Becker W, Jurgens R. An analysis of the saccadic system by means of double step stimuli. *Vision Res*. 1979;19:967-983.
33. Breznitz Z, Misra M. Speed of processing of the visual-orthographic and auditory-phonological systems in adult dyslexics: the contribution of "asynchrony" to word recognition deficits. *Brain Lang*. 2003;85:486-502.
34. Westheimer G. Eye movement response to a horizontally moving visual stimulus. *Arch Ophthalmol*. 1954;52:932-941.
35. Ram-Tsur R, Caspi A, Gordon C, Zivotofsky AZ. The Saccadic system more readily co-processes orthogonal than co-linear saccades. *Exp Brain Res*. 2005;160:398-403.
36. Heide W, Blankenburg M, Zimmermann E, Kompf D. Cortical control of double-step saccades: implications for spatial orientation. *Ann Neurol*. 1995;38:739-748.
37. Ram-Tsur R., Faust M., Caspi A., Zivotofsky AZ. Evidence for visuomotor deficits in developmental dyslexia: Application of the Double-Step Paradigm. Paper presented at the annual meeting of Theoretical and Experimental Neuropsychology (TENNET), Montreal, Quebec, Canada; 2005.
38. Wechsler D. *WAIS-III: Administration and Scoring Manual: Wechsler adult intelligence scale*. 3rd ed. San Antonio, TX: Psychological Corp; 1997.
39. Raven J, Raven J, Court J. *Raven's Progressive Matrices and Vocabulary Scales*. Oxford, UK: Oxford Psychologists Press; 1998.
40. Anastopoulos A, Spisto M, Maher M. WISC-III Freedom from Distractibility factor: its utility in identifying children with attention deficit hyperactivity disorder. *Psychol Assess*. 1994;6:368-371.
41. Shatil E. One-Minute Test for Words. Unpublished test. Haifa: University of Haifa; 1995.
42. Shatil E. One-Minute Test for Pseudowords. Unpublished test. Haifa: University of Haifa; 1995.
43. Shatil E. Passage Reading. Unpublished test. Haifa: Haifa University; 1997.
44. Shatil E. Spelling Test. Unpublished test. Haifa: Haifa University; 1997.
45. Perin D. Perin spoonerism task. *Br J Psychol*. 1983;74:129-144.
46. Shatil E. Phonological Processing. Unpublished test. Haifa: Haifa University; 1997.
47. Robinson DA. A method of measuring eye movement using a scleral search coil in a magnetic field. *Trans Biomed Eng*. 1963;10:137-145.
48. Levitt H. Transformed up-down methods in psychoacoustics. *J Acoust Soc Am*. 1971;49:467-477.
49. Martin F, Lovegrove W. Flicker contrast sensitivity in normal and specifically disabled readers. *Perception*. 1987;16:215-221.
50. Tanaka K, Saito H. Analysis of motion of the visual field by direction, expansion/contraction, and rotation cells clustered in the dorsal part of the medial superior temporal area of the macaque monkey. *J Neurophysiol*. 1989;62:626-641.
51. Merigan W, Maunsell J. How parallel are the primate visual pathways? *Annu Rev Neurosci*. 1993;16:369-402.
52. Ransby M, Swanson H. Reading comprehension skills of young adults with childhood diagnoses of dyslexia. *J Learn Disabil*. 2003;36:538-555.
53. Pennington B, Van Orden G, Smith S, Green P, Haith M. Phonological processing skills and deficits in adult dyslexics. *Child Dev*. 1990;61:1753-1778.

54. Stein J, Walsh V. To see but not to read; the magnocellular theory of dyslexia. *Trends Neurosci.* 1997;20:147-152.
55. Schiller P, Lee K. The effects of lateral geniculate nucleus, area V4, and middle temporal (MT) lesions on visually guided eye movements. *Vis Neurosci.* 1994;11:229-241.
56. Fischer B, Hartnegg K, Mokler A. Dynamic visual perception of dyslexic children. *Perception.* 2000;29:523-530.
57. Biscaldi M, Fischer B, Hartnegg K. Voluntary saccadic control in dyslexia. *Perception.* 2000;29:509-521.
58. Black J, Collins D, De Roach J, Zubrick S. Dyslexia: saccadic eye movements. *Percept Mot Skills.* 1984;58:903-910.
59. De Luca M DPE, Judica A, Spinell D, Zoccolotti P. Eye movement patterns in linguistic and non-linguistic tasks in developmental surface dyslexia. *Neuropsychologia.* 1999;37:1407-1420.
60. Leisman G, Schwartz J. Aetiological factors in dyslexia: I saccadic eye movement control. *Percept Mot Skills.* 1978;47:403-407.
61. Cornelissen P, Stein J. Contrast sensitivity and coherent motion in dyslexics. *Vision Res.* 1995;35:1483-1494.
62. Stein J. Visual motion sensitivity and reading. *Neuropsychologia.* 2003;41:1785-1793.
63. Purves D, Augustine G, Fitzpatrick D, et al. *Eye Movements and Sensory Motor Integration.* Sunderland, MA: Sinauer Associates; 2001.
64. Apter J. Eye movements following strychninization of the superior colliculus of cats. *J Neurophysiol.* 1946;9:73-86.
65. Optican LM, Quaia C. Distributed model of collicular and cerebellar function during saccades. *Ann N Y Acad Sci.* 2002;956:164-177.
66. Pitts W, McCulloch W. How we know universals: the perception of auditory and visual forms. *Bull Math Biol.* 1947;9:127-147.
67. Vidyasagar T. A neuronal model of attentional spotlight: parietal guiding the temporal. *Brain Res.* 1999;30:66-76.
68. Brannan J, Williams M. Allocation of visual attention in good and poor readers. *Percept Psychophys.* 1987;41:23-28.
69. Eden G, Stein J, Wood H, Wood F. Temporal and spatial processing in reading disabled and normal children. *Cortex.* 1995;31:451-468.
70. Meyler A, Breznitz Z. Visual, auditory and cross-modal processing of linguistic and nonlinguistic temporal patterns among adult dyslexic readers. *Dyslexia.* 2005;11:93-115.
71. Fabbro F, Moretti R, Bava A. Language impairments in patients with cerebellar lesions. *J Neurolinguist.* 2000;13:173-188.
72. Silveri M, Misciagna S. Language, memory, and the cerebellum. *J Neurolinguist.* 2000;13:129-143.
73. Malm J, Kristensen B, Karlsson T, Carlberg B, Fagerlund M, Olsson T. Cognitive impairment in young adults with infratentorial infarcts. *Neurology.* 1998;51:433-440.
74. Moretti R, Bava A, Torre P, Antonello R, Cazzato G. Reading errors in patients with cerebellar vermis lesions. *J Neurol.* 2002;249:461-468.
75. Quaia C, Lefevre P, Optican LM. Model of the control of saccades by superior colliculus and cerebellum. *J Neurophysiol.* 1999;82:999-1018.