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The gradient of visual attention in developmental dyslexia

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Abstract

This study investigated the gradient of visual attention in 21 children, 11 children with specific reading disorder (SRD) or dyslexia and 10 children with normal reading skills. We recorded reaction times (RTs) at the onset of a small point along the horizontal axis in the two visual fields. In 70% of the cases the target appeared inside a circle acting as focusing cue and in 30% of the cases it appeared outside, allowing us to study the distribution of attentional resources outside the selected area. Normally reading children showed a normal symmetric distribution of attention. Indeed, RTs were directly proportional to the eccentricity of the target, and no visual field effect was observable. In contrast, children with SRD showed an anomalous and asymmetric distribution. The effect of the target eccentricity influenced RTs only when the stimulus was projected in the left visual field, whereas no effect was observable when the stimulus was projected in the right visual field. Findings allowed us to discuss the relation between this anomalous spatial distribution of visual attentional resources and dyslexia. To interpret the visual perceptual difficulties of children with SRD the hypothesis was made of a selective disorder of spatial attention (left inattention and right over-distractibility) related to a right parietal cortex dysfunction.

Keywords: Dyslexia; Specific reading disorder; Spatial attention; Attentional gradient; Right parietal cortex; Reaction time

1. Introduction

Although several studies have provided evidence for a phonological deficit in developmental dyslexia (e.g. [3]), many dyslexic children also show visual perceptual deficits (characteristic missequencing, omission and mislocating errors) when they attempt to read. These deficits may be attributed to defective visual information processing. Recent studies have revealed a deficit of the magnocellular (M) or transient visual system — responsible for processing information about the position of visual stimuli (e.g. [2,26]) — in some children with specific reading disorder (SRD) or dyslexia (for a review, cf. [38]). However, the specific mechanism through which the M-pathway deficit might cause dyslexia has not been described in full detail yet. The information processed by the M system ends in the parietal posterior cortex (PPC), which is an important supramodal selective spatial attention area [40].

The complex process of reading presupposes as an intrinsic mechanism the capacity to select a particular area of the visual field so as to process relevant information and filter irrelevant and distracting information [25]. This mechanism, also known as spatial attention, is generally defined as the operation that facilitates processing in a particular area of the visual field. Theoretically, spatial attention acts as filter to enhance the information from a target object (facilitation) or suppress the information from the distractor objects (inhibition), or operations that do both (for a review, cf. [8]).

Empirical evidence showed the difficulty of subjects with dyslexia to perform serial visual search tasks [5,23,41,44]. Furthermore, Casco et al. [6] found reading skills to be related to selective visual attention. Previous research indicated that poor readers have considerable difficulty attending to local details when such details are embedded in a more global structure [43].

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Brannan and Williams [4] showed that poor readers were unable to orient their attention using a peripheral cue. Rayner et al. [30] reported the case of a subject with developmental dyslexia who performed better than controls in reporting letters shown in parafoveal vision. The authors attributed that performance to a deficit of selective spatial attention present in dyslexics so that letters from words viewed parafoveally would interfere with the processing of concurrently fixated words. Also, Geiger and coworkers [20,21] reported that dyslexics showed a disability to suppress information from the periphery of the visual field, which would cause a deficit in foveal reading. Subjects with learning disabilities (LD) made more errors than controls on a selective attention task when distractor letters were adjacent to the target letter, thus suggesting that LD children are less able to narrow the focus of attention [32]. Facoetti et al. [15] proved that children with dyslexia have difficulty in sustaining attentional focusing which is necessary for effective processing of visual information. Lastly, in a recent study Facoetti et al. [13] suggested that the M pathway deficit might influence reading, thus hampering inhibition of lateral information. The authors used two experiments to show that children with dyslexia exploit a diffuse-distributed attention mode. More specifically, results of the first experiment proved that reaction times (RTs) of children with dyslexia vs. normally reading children are not influenced by eccentricity of the target. These data suggest that visual perceptual disorders, often associated with dyslexia, might be determined by a deficit of spatial attention, that is, a deficit of the mechanisms inhibiting laterally distracting information.

Nevertheless, much converging evidence indicated an asymmetric distribution of attention between the two visual fields in dyslexics. Hari and Koivikko [22] suggested that, as compared to the right visual field (RVF), dyslexics suffer from 'mini-neglect' in the left visual field (LVF). This left-side deficit appears to be linked to a right-side enhancement in the processing of visual information, as demonstrated by an increased ability of dyslexics in letter recognition in the RVF [20,21]. Indeed, dyslexics exhibited a reduced interference effect in the LVF (left inattention), concomitant with a strong interference effect in the RVF (right over-distractibility) [16].

Therefore, the present study was aimed at further exploring a possible attentional visual field asymmetry in dyslexia. The objective was to determine whether the visual spatial attention deficit might be specific to a visual field or involve both visual fields. Indeed, the visual field factor and thus hemisphere factor had not been studied previously [13]. To this end, we investigated the gradient of visual attention in a group of normally reading children and in a group of children with SRD or dyslexia. The attentional gradient is defined as the increase in RTs with increasing eccentricity of the target from the attentional focus (e.g. [25,36]). The gradient that generates the shape of the RTs V curve is assumed to be represented in the location expectation domain [25], whose function may be instantiated by cells of the posterior parietal cortex that are responsive to the visuotopic location of objects (e.g. [28,29]). RTs recording at the onset of a white dot projected at different eccentricities from the fixation point allowed us to study the distribution of attentional resources inside the visual field. A focusing cue indicated the most probable location where the target would appear. As shown by many studies, the spatial cue-size allows one to regulate the dimension of the attentional focus (e.g. [7,12,14,39]). Another area of investigation was, therefore, the processing of visual information outside the attentional focus. Indeed, in some trials (30%) the target appeared outside the focusing cue at 6 or 9° from the fixation point along the horizontal axis.

2. Method

2.1. Subjects

We tested 21 children (16 males and 5 females). Inclusion criteria were: (1) full scale IQ > 85 as measured by the Wechsler Intelligence Scale for Children-Revised [42]; (2) no known gross behavioral or emotional problems; (3) normal or corrected-to-normal vision and hearing; (4) absence of drug therapy; (5) normal visual field; and (6) absence of attention deficit disorder with hyperactivity (ADHD) [1]. Eleven children (9 males and 2 females), mean age 12.1 years, were classified as dyslexic as their performance in oral reading of text, words and non-words was 2 S.D. below the norm on age-standardized Italian tests. The remaining ten children (7 males and 3 females), mean age 11.4 years, were normal readers. Children of the two groups were matched for age and IQ. Table 1 shows descriptive data of the two groups.

Table 1
Details (means and S.D.) of age, full IQ and reading abilities (z scores) of the two groups participating in the study

	Normal readers	Dyslexics	P
Age (years)	11.4 (2.1)	12.1 (1.7)	>0.05
Full IQ (WISC-R)	108 (8.5)	102 (12.2)	>0.05
Text errors	0.3 (0.5)	−3.1 (1.5)	<0.05
Text time	0.4 (0.3)	−3.6 (1.8)	<0.05
Words errors	0.6 (0.5)	−2.8 (2.1)	<0.05
Words time	0.1 (0.7)	−3.4 (1.9)	<0.05
Non-words errors	−0.2 (0.4)	−2.6 (2.1)	<0.05
Non-words time	0.4 (0.7)	−3.2 (1.8)	<0.05

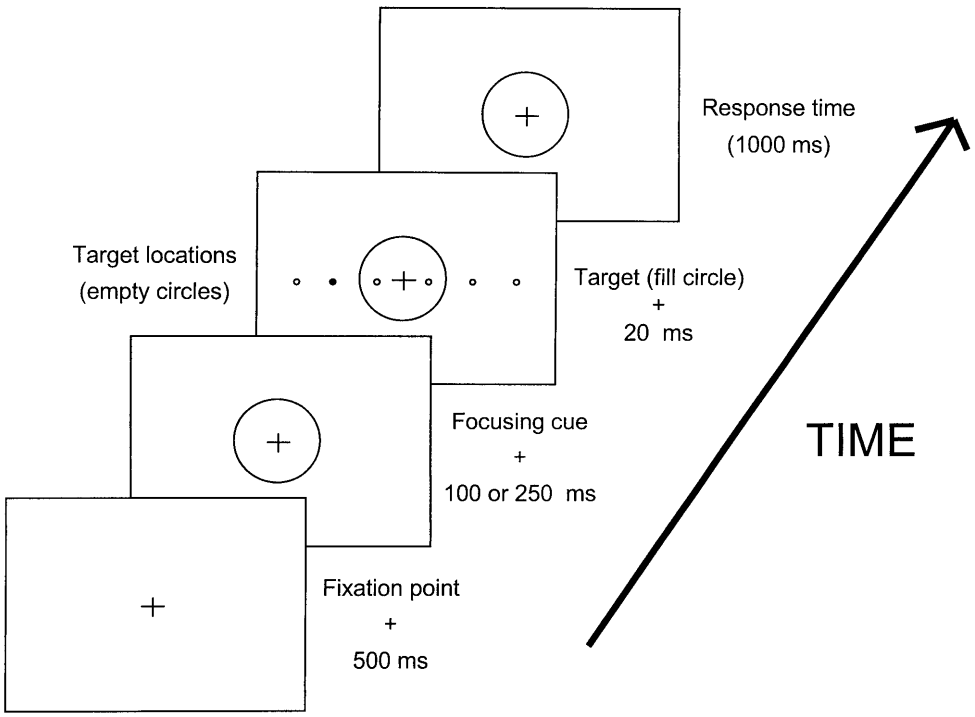

Fig. 1. Schematic representation of the display used in the experiment.

2.2. Apparatus and procedure

Testing was carried out in a dimly lit room with an ambient luminance of 1.5 cd/m^2. Subjects sat in front of a monitor screen (15 inch and with a luminance 0.5 cd/m^2) with their head positioned on a headrest so that the eyes-screen distance was 40 cm. Fixation point was a cross with a visual angle of 0.5° presented in the middle of the screen. One circle with a visual angle of 4.5° was presented concentrically to the fixation point and served as focusing cue. The target was a white dot of 0.5° which could appear at three different distances along the horizontal axis from the fixation point: 3, 6 and 9° visual angle. At the first distance (3°) the target fell inside the focusing cue (circle), whereas at the second (6°) and third distance (9°) it fell outside. There were two possible sites in which the target could appear: left and right visual field. Stimuli luminance was 24 cd/m^2. Subjects were instructed to keep their eyes on the fixation point throughout the duration of the trial. Eye movements were monitored by means of a system composed of infrared-ray spectacles connected with an amplifier, an analog-digital converter and a computer. Any eye movement larger than 1° was detected by the system and in such cases the trial was automatically suppressed. Each trial started with the onset of the fixation point accompanied by a 1000 Hz tone. After 500 ms, one focusing cue was projected. Afterwards, the target was shown for 20 ms; time intervals between cue and target or stimulus onset asynchrony (SOA) were variable (100–250 ms) in order to modify the warning set but were not considered in subsequent analyses (Fig. 1).

Subjects were instructed to press the space-bar on the keyboard as fast as possible at the onset of the target and their reaction times (RTs) were recorded by the computer. The maximum time allowed to respond was 1 s. A certain number of catch trials in which the stimulus was not presented and the subject did not have to respond were intermingled with normal trials. The experimental session consisted of 180 trials divided into two blocks of 90 trials. Trials were distributed as follows: 80 trials, 56 at 3° eccentricity (28 for right and 28 for left), 12 at 6° eccentricity (6 for right and 6 for left), 12 at 9° eccentricity (6 for right and 6 for left) and 10 catch trials.

3. Results

Errors, that is responses on catch trials and missed responses, were less than 4% and were not analyzed. Analysis of variance (ANOVA) was performed on mean correct RTs, with eccentricity of target (3, 6 and 9°) and visual field (left and right) as within-subject factors, and group (dyslexic and normally reading children) as between-subject factor.

The group main effect was not significant ($F(1,19) = 1.09$, $P = 0.31$). The main effect of eccentricity of the target was significant, $F(2,38) = 19.64$, $P < 0.001$; RTs

were 343 ms at 3°, 347 ms at 6° and 365 ms at 9° eccentricity. The main effect of visual field was significant, $F(1,19) = 5.77$, $P < 0.05$. Mean RTs were 348 ms in the RVF and 355 ms in the LVF. The visual field × eccentricity interaction was significant, $F(2,38) = 5.91$, $P < 0.005$; in the RVF mean RTs were 343, 346 and 356 ms at 3, 6 and 9° eccentricity, whereas in the LVF they were 343, 348 and 375 ms at 3, 6 and 9° eccentricity. Also, the group × visual field interaction was significant, $F(1,19) = 7.55$, $P < 0.02$; for dyslexics, mean RTs were 353 ms in the RVF and 367 ms in the LVF; for normal readers, they were 344 ms in the RVF and 343 ms in the LVF. Planned comparisons (Newman–Keuls post-hoc test) showed that in children with dyslexia the difference in RTs between the RVF and the LVF was significant ($P < 0.01$), whereas it was not in normally reading children ($P = 0.98$). Furthermore, no significant difference was found between the two groups with regard to the RVF ($P = 0.16$) as compared to the LVF ($P < 0.001$).

The eccentricity × visual field x group interaction was significant, $F(2,38) = 3.36$, $P < 0.05$. Planned comparisons (Newman–Keuls post-hoc test) denoted that children with dyslexia showed no significant differences between LVF and RVF at 3° eccentricity (RVF = 354 and LVF = 353, difference = 1 ms, $P = 0.94$) and at 6° eccentricity (RVF = 346 and LVF = 358, difference = 12 ms, $P = 0.37$). In contrast, the difference between the two visual fields at 9° eccentricity was significant (RVF = 358 and LVF = 389, difference = 31 ms, $P < 0.001$). No significant differences were found in the group of normal readers between LVF and RVF at 3, 6 and 9° eccentricity. In addition, in normally reading children the eccentricity effect of the target (RTs difference between 3° inside the attended area and 9° outside the attended area) was significant both in the RVF (3° = 331 ms and 9° = 354 ms, difference 23 ms, $P < 0.02$) and in the LVF (3° = 333 ms and 9° = 359 ms, difference 26 ms, $P < 0.01$). In contrast, in children with dyslexia the eccentricity effect of the target was significant only in the LVF (3° = 353 ms and 9° = 389 ms, difference 36 ms, $P < 0.001$). Indeed, the eccentricity of the target did not have an effect on the RVF (3° = 354 ms and 9° = 358 ms, difference 4 ms, $P = 0.59$). Finally, group comparisons showed that in the LVF dyslexic children were slower than normal readers at all eccentricities (3° eccentricity: dyslexics = 353 ms vs. normal readers = 333, $P < 0.05$, 6° eccentricity: dyslexics = 358 ms vs. normal readers = 336, $P < 0.02$ and 9° eccentricity: dyslexics = 389 ms vs. normal readers = 359, $P < 0.001$) whereas, in the RVF dyslexics were slower only at 3° eccentricity (inside the attentional focus) (dyslexics = 345 ms vs. normal readers = 331, $P < 0.05$). Fig. 2 shows the effect of eccentricity × visual field in normal readers and children with dyslexia.

4. Discussion

Recently, the hypothesis of a phonological temporal processing deficit has become of central interest in the study of dyslexia etiology [18]. However, a temporal processing deficit appears to affect information processing in different sensory modalities [38]. Accordingly, it has been suggested that the major problem of dyslexic children would be a general impairment in the processing of rapid streams of information, regardless of the stimulated modality. The magnocellular-transient (M) system, which would also include the auditory system, is thought to be the neural basis underlying such deficit [27]. An impairment of this pathway would lead to a reduced ability to focus attention in the stimulated modality.

The major finding of the present research is an asymmetrical spatial distribution of visual attention in the sample of children with SRD or dyslexia. Indeed, slower RTs at the target onset in the left versus the right visual field seem to suggest an attentional deficit in the right parietal cortex. Many studies proved that a lesion to the right parietal cortex elicits slower target detection speed in the LVF and/or higher target detection speed in the RVF (e.g. [28,29,36]). It has recently been suggested that two-thirds of subjects with dyslexia show a deficit of the right posterior parietal lobe resulting in poor oculomotor control [10,37]. These findings point to a possible reduced right parietal lobe functioning in dyslexic children during visual information processing and are in agreement with other studies of patients with lesions to the right hemisphere (e.g. [19,24,34]).

These results give further support to the hypothesized right parietal impairment [16,22] as assumed by the magnocellular (M) theory of dyslexia [38]. Indeed, the parietal cortex is dominated by M pathway inputs and

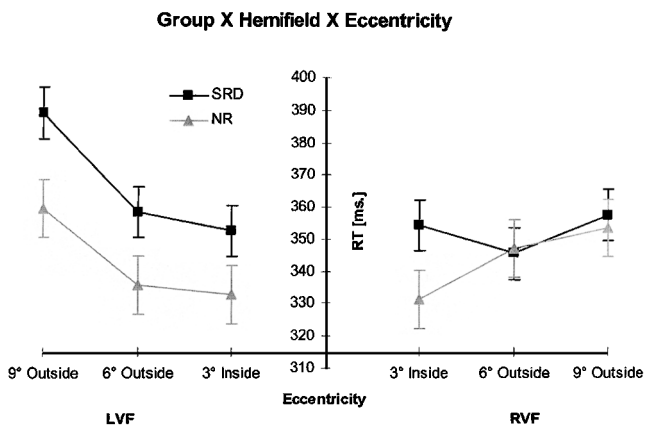

Fig. 2. Mean reaction times (RTs) and standard errors as a function of groups CSRD = dyslexics and NR = normal readers, eccentricity (3, 6 and 9°) and visual field of the target, CLVF = visual field and RVF = right visual field.

brain imaging, transcranial magnetic stimulation (TMS) and brain-damaged patients studies strongly support the notion of a specific and critical involvement of the right parietal cortex in selective spatial attention (for a recent review see [9]).

In addition, our results also seem to point to the presence of a visual attentional gradient disorder in children with SRD during selection of relevant information projected in the RVF (right over-distractibility). In children with dyslexia, when the target stimulus appeared in the RVF, RTs were not influenced by eccentricity as if there were a specific difficulty in inhibiting visual stimuli appearing to the right of the attentional focus, whereas when the target stimulus appeared in the LVF, RTs were abnormally influenced by eccentricity (left inattention). Correct decoding of a written word relies upon detailed processing which is made possible by attentional focusing. Therefore, during reading, which necessarily requires minimization of the effect of laterally distracting information, processing can be shifted from the diffused to the focused modality by restricting the focus of attention [14,25]. These specific deficits of the gradient of visual attention, too, may be explained in terms of an alteration of spatial attentional functions associated with the right PPC [33,36].

Skottun [35] recently reviewed some data (contrast sensitivity) on magnocellular deficits in developmental dyslexia and suggested that, although there are studies that are consistent with the M deficit, they are outnumbered both by the studies that have found no deficit and by studies that are incompatible with an M impairment. Skottun [35] also suggested that the visual deficits found in dyslexics using other psychophysical tests lend themselves to alternative explanations. For instance, to investigate the pathophysiology of dyslexia, Eden et al. [11] used functional magnetic resonance imaging (fMRI) to study visual motion processing in normal and dyslexic male adults. In all dyslexics, presentation of moving stimuli failed to produce the same task-related functional activation in area V5/MT. Recent studies of human perception measuring visual motion indicated that simple aspects of motion processing may be strongly affected by the attentional processes of the perceiver (for a review see [31]).

In conclusion, our results suggest that the deficit of the mechanism subserving selection of stimuli (spatial attention) in the right visual field might determine some visual perceptual disorders that are frequently found in subjects with SRD or dyslexia. Slower detection speed of stimuli projected in the left visual field seems to confirm the hypothesis of an alteration of attentional functions in the right parietal cortex [16,17,22]. A right parietal cortex dysfunction is thought to be the neurological basis underlying the dyslexics' attentional deficit.

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