Exp Brain Res (2001) 138:46–53
DOI 10.1007/s002210100700

RESEARCH ARTICLE

Andrea Facoetti · Massimo Turatto · Maria Luisa Lorusso · Gian Gastone Mascetti

Orienting of visual attention in dyslexia: evidence for asymmetric hemispheric control of attention

Received: 11 July 2000 / Accepted: 30 January 2001 / Published online: 10 March 2001

Abstract The control of attentional orienting was studied in children with specific reading disorder (SRD) or dyslexia, and it was compared with that of normal readers. We used the covert orienting paradigm to measure subjects' reaction times for target detection both in valid and invalid cue conditions, either in the left or in the right visual fields. In experiment 1, we investigated exogenous orienting. The cue consisted of a peripheral abrupt onset and the cue-target delay was 350 ms. As compared with normal readers, in dyslexics the cue effect was absent in the right visual field, whereas in the left visual field a greater cue effect was observed. No visual field asymmetry was found in normal readers. In experiment 2, we investigated endogenous orienting. The cue was shown centrally and the cue-target delay was 750 ms. In dyslexics and normal readers, orienting of attention was present in both visual fields. However, in the invalid condition, dyslexic children showed significantly slower reaction times in the left visual field than in the right visual field. These results were interpreted as being due to an asymmetric control of visual spatial attention, possibly related with a posterior attention mechanism deficit in the right parietal cortex and/or an interhemispheric dysfunction and/or an impairment of cerebellar functions.

Keywords Dyslexia · Spatial attention · Orienting of attention · Visual asymmetry · Reaction time

A. Facoetti (✉) · M. Turatto · G.G. Mascetti
General Psychology Department, University of Padova,
Via Venezia 8, 35131 Padova, Italy
e-mail: andreaf@psico.unipd.it
Tel.: +39-049-8276672, Fax: +39-049-8276600

A. Facoetti
Neuropsychiatric Unit, General Hospital of Bergamo, Bergamo, Italy

M.L. Lorusso
Neuropsychology Unit,
Scientific Institute "E. Medea" of Bosisio P., Lecco, Italy

Introduction

Learning to read requires appropriate visual and phonological skills. It is well accepted that there are two routes, lexical and nonlexical, which can be used for reading (Coltheart et al. 1993). To read an unknown word, a sequence of visual symbols must be recognized and transformed into a sequence of sounds via the nonlexical route. At the same time, to be read correctly, a familiar word must be isolated from the others in the text (lexical route). In both cases, however, what is crucial is the ability to select the relevant information while excluding the irrelevant one. This filter mechanism is generally defined as the operation which facilitates processing in a particular area of the visual field. Although several studies have provided evidence for a phonological deficit in developmental dyslexia (e.g., Bradley and Bryant 1983), many dyslexic children also show visuoperceptual deficits: they tend to displace letters within a word and invert them, causing words to appear distorted, overlapping, and moving. These deficits may be attributed to defective visual information processing.

In addition, other studies have shown an involvement of visual attention in reading. For example, the crowding effect may impair letter and word recognition (Atkinson 1991). Serial visual search and perceptual grouping are related to reading performance (Casco et al. 1998; Williams and Bologna 1985). There is evidence suggesting that the analysis of strings of letters or words requires sustained focused attention (LaBerge and Brown 1989) and fast and precise control of visual orienting (Inhoff et al. 1989). Finally, reading presupposes an accurate planning and control of ocular saccades and fixations (Morris and Rayner 1991; Pavlidis 1981). In a recent study, Facoetti et al. (2000a) suggested that visual 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 (attentional focusing deficit). This empirical evidence suggests a crucial involvement of visual spatial attention in dyslexia.

Other evidence suggests that the magnocellular (M) system, which plays a crucial role in shifting of attention (Steinman et al. 1996), is defective in such a reading disorder (for a review, see Stein and Walsh 1997). The M system, which processes information about location and movement of visual stimuli, may affect reading by hampering focusing of attention (which requires precise coding of stimulus location). Therefore, there are reasons to suspect that orienting of attention can be compromised in dyslexic children (Vidyasagar 1999).

Orienting of attention is typically investigated by means of the covert orienting paradigm, in which attention is shifted from one point to another without movements of the eyes (Posner 1980; Posner et al. 1980). The method consists of presenting a spatial cue followed by the target. The cue can be valid (when the target appears in the cued position) or invalid (when the target appears in an uncued position). In the valid condition, reaction times (RTs) are generally faster than in the invalid condition. This is called the *cue effect*. It has been pointed out that abnormal patterns of orienting response across the different cueing conditions may highlight specific neurocognitive deficits. Specifically, neuropsychological studies have shown that unilateral damage of the posterior parietal cortex selectively affects contralateral target detection in the invalid cue condition (Petersen et al. 1989; Posner et al. 1984, 1987). Orienting is thought to occur either in an exogenous or endogenous fashion, given three main features of the cue: position of the cue (peripheral or central), cue validity, and cue-target delay. A peripheral cue, with a short cue-target delay (about 100 ms), would elicit an automatic shift of attention regardless of its validity. By contrast, an informative-central cue and a longer cue-target delay (for example, longer than 500 ms) allow voluntary control of orienting (Jonides 1981; Müller and Rabbitt 1989; Warner et al. 1990).

Brannan and Williams (1987) demonstrated that, compared with normally reading subjects, poor readers were not able to use the information provided by a peripheral cue. Jonkman et al. (1992) investigated voluntary shifting of attention in dyslexic and normally reading children using a paradigm with a central spatial cue, but they did not detect any difference between the two groups of subjects. Lastly, Facoetti et al. (2000b) showed that the deficit in spatial orienting found in dyslexic children seems to selectively involve the automatic exogenous control, but not the voluntary endogenous one.

Nevertheless, other studies have found visual-field asymmetric performances in dyslexic subjects in visual search tasks (Eden et al. 1993; Fowler et al. 1991). Hari and Koivikko (1999) suggested that, compared with 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 (Geiger et al. 1994). Indeed, dyslexics exhibited a reduced interference effect in the LVF (mild left inattention), concomitant with a strong interference effect in the RVF (right over-distractibility) (Facoetti and Turatto 2000). Lastly, Facoetti and Molteni (2001) investigated the gradient of visual attention in children with dyslexia and with normal reading skills. 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 dyslexia showed an anomalous and asymmetric distribution. The effect of target eccentricity influenced RTs only when the stimulus was projected in the LVF, whereas no effect was observable when the stimulus was projected in the RVF. The hypothesis was made of a selective disorder of spatial attention (left inattention and right over-distractibility).

Therefore, because dyslexic children seem to exhibit a visual-field asymmetry in the gradient of spatial attention (Facoetti and Molteni 2001; Facoetti and Turatto 2000), the aim of the present study was to verify whether children with specific reading disorder also show an asymmetric hemispheric control of attentional orienting. In particular, we were interested in covert orienting because its underlying neural mechanisms are involved both in the selection of competing visual stimuli and in related activities such as reading (Inhoff et al. 1989; Posner and Rafal 1987).

In the present study, we carried out two experiments designed to assess whether dyslexics show a visual-field asymmetry in the control of covert orienting when attention is either directed voluntarily or summoned by a peripheral abrupt visual onset. In both experiments, two different groups of children (normal readers and dyslexics) matched for age, sex, and IQ were selected.

Given that in dyslexics greater attentional resources are available in the right visual field than in the left visual field (Facoetti and Turatto 2000; Hari and Koivikko 1999) and that such resources are concentrated (narrow focus) in the left visual field (mild left inattention), whereas they are excessively diffuse (wide focus) in the right visual field (right over-distractibility) (Facoetti and Molteni 2001), it could be assumed that also orienting of the attentional focus is asymmetrically controlled in the two visual fields. Specifically, it could be hypothesized that the invalid cue has a greater effect when the target is projected in the LVF, whereas this effect is reduced or absent when the target is presented in the RVF. Further, if the deficit in orienting of visual attention exclusively involves automatic control (Brannan and Williams 1987) and not voluntary control (Facoetti et al. 2000b), this attentional asymmetry should be reduced when the endogenous modality is investigated (experiment 2) vs. the exogenous modality (experiment 1).

Experiment 1

In this experiment, exogenous orienting of attention was studied in dyslexic and normally reading children. The cue was presented peripherally and consisted in an abrupt visual onset. The cue-target delay was 350 ms, and the cue-set validity was 80%. The choice

Table 1 Descriptive data on the two groups in experiment 1

	Age (years)		Full IQ (WISC-R)		Reading test			
					Accuracy errors/ 200 syllables		Speed time for syllable %	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Normal readers	11.4	8–15	108	95–118	2.8	1–7	23	16–35
Dyslexics	12.1	8–15	102	91–114	12.7	8–21	115	75–193

of this stimulus-onset asynchrony (SOA) was motivated by the fact that, in a previous study with dyslexic children, Facoetti et al. (2000b) found that with shorter SOAs (150 and 250 ms) the cue was not able to produce a shift of attention. It follows that, because we used a long cue-target delay (350-ms), we also had to adopt an informative cue to avoid a possible inhibition of return (IOR; Berlucchi et al. 1989).

Therefore, given this experimental setting, a caveat on automatic orienting here investigated should be considered. In fact, in the present conditions, we were able to elicit an involuntary shift of attention towards the cue, but subjects maintained their attention voluntarily on the cued position. Therefore, we studied possible orienting deficits in dyslexics when attention is triggered by peripheral onsets rather than the automatic orienting process per se, as these events have a peculiarity in summoning attention involuntarily (Yantis and Jonides 1984).

Materials and methods

Participants

Participants were 20 children (14 males and 6 females) selected by: (1) absence of a spoken language impairment (for crucial implications, see McArthur et al. 2000); (2) a full scale IQ >85 as measured by the Wechsler Intelligence Scale for Children-Revised (Wechsler 1986); (3) no known gross behavioral or emotional problems; (4) normal or corrected-to-normal vision and hearing; (5) normal visual field; (6) absence of Attention Deficit Disorder with Hyperactivity (ADHD) (American Psychiatric Association 1994); and (7) right manual preference (Briggs and Nebes 1975). Ten children (7 males and 3 females), mean age 12.1 years (SD=1.74), were classified as dyslexic as their performances in oral reading of a text, words, and nonwords were 2 SDs below the norm on age-standardized Italian tests. Variables considered were speed and accuracy. The group of children with dyslexia showed both "visual" and "phonological" symptoms (mixed-type dyslexia). The remaining ten children (7 males and 3 females), mean age 11.4 years (SD=1.76), were normal readers. Children of the two groups were individually matched for age, sex, and IQ. Table 1 shows descriptive data of the two groups.

Apparatus and procedure

Tests were carried out in a dimly lit room (luminance of 1.5 cd/m^2). Participants sat in front of a monitor screen (15 inches and with a background luminance of 0.5 cd/m^2), with their head positioned on a headrest so that the eye-screen distance was 40 cm. The fixation point consisted of a cross (1.4° of visual angle) appearing at the center of the screen. Two circles (3°) were presented peripherally (10° of eccentricity), one to the left and one to the right of the fixation point. A vertical arrow (1.5°) shown above the circles was used as cue. A dot (0.5°) in the center of one of the two circles was the target stimulus. Stimuli were white and had a luminance of 24 cd/m^2. Participants were instructed to keep their eyes fixed 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 the corresponding trial was discarded but not replaced.

Each trial started with the onset of the fixation point accompanied by a 1000 Hz warning signal tone. After 500 ms, the two circles were displayed peripherally, and 500 ms later the cue was shown for 100 ms. Then, after the 350-ms SOA, the target appeared for 100 ms inside one of the two circles. On valid trials, the target was presented inside the circle indicated by the cue, whereas on invalid trials the target appeared in the circle on the side opposite to that indicated by the cue. At the target onset, participants were instructed to react as quickly as possible by pressing the spacebar on the computer keyboard, and RTs were recorded by the computer. The maximum time allowed to respond was 1s. The inter-trial interval was 1 s. Catch trials, in which the target was not presented and participants did not have to respond, were intermingled with normal trials. The experimental session consisted of 104 trials divided into two blocks of 52 trials each. Trials were distributed as follows: 32 valid trials (16 for each side), 8 invalid trials (4 for each side), and 12 catch trials.

Results

Errors, that is responses on catch trials and missed responses, were less than 1.8% and were not analyzed. In both experiments, outliers were excluded from the data sets before the analyses were carried out. Outliers were defined as RTs faster than 150 ms or more than 2.5 standard deviations above the mean. In the present experiment, this resulted in the removal of approximately 2% of all observations. Eye movements were about 3% of total trials. Mean correct RTs were analyzed with a three-way analysis of variance (ANOVA), in which the two within-subjects factors were cue condition (valid and invalid) and target position (right visual field and left visual field), and the between-subjects factor was group (dyslexic and normally reading children).

The group main effect was close to significance: $F(1,18)=4.261$, $P=0.054$; RTs were faster in normal readers (430 ms) than in dyslexics (533 ms). The main effect of cue condition was significant: $F(1,18)=28.745$, $P<0.001$; RTs were faster in valid trials (443 ms) than in invalid ones (520 ms). The main effect of visual field was also significant: $F(1,18)=5.189$, $P<0.05$. RTs were faster in the RVF (462 ms) than in the LVF (501 ms).

The cue condition × visual field interaction was significant: $F(1,18)=7.946$, $P<0.02$. The cue effect was greater in the LVF (115 ms) than in the RVF (41 ms). The cue condition × group interaction was not significant ($P>0.8$). In contrast, the group × visual field interaction was significant: $F(1,18)=6.715$, $P<0.02$, indicating that RTs varied across groups according to the visual fields. In normal readers, the RT difference between LVF (427 ms) and RVF (433 ms) was 6 ms; in dyslexics, the RT difference between LVF (574 ms) and RVF (492 ms) was 82 ms. However, these findings should be interpreted in light of the three-way cue condition × group × visual field interaction, which was also significant: $F(1,18)=5.451$, $P<0.05$. Planned comparisons showed that on invalid trials normally reading children showed slower RTs than on valid ones in both visual fields (RVF 69 ms, LVF 81 ms, all *P*s

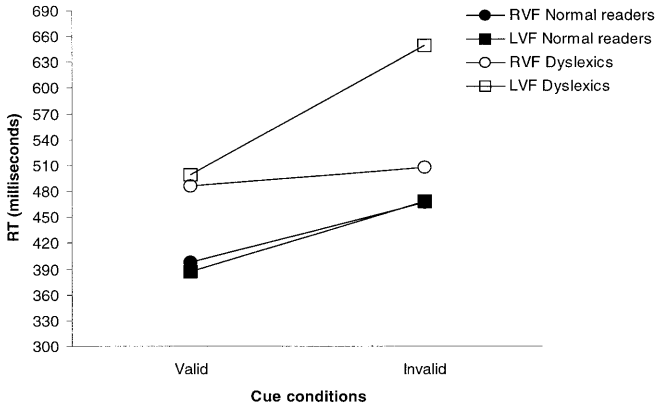

Fig. 1 The cue effect for dyslexics and normal readers in experiment 1. *RVF* (right visual field) and *LVF* (left visual field) refer to the target position

<0.05). Also, in the two conditions, RTs were similar across the visual fields (all *P*s >0.6). Dyslexic children showed a different RT pattern, in that in the RVF the cue effect was absent (*P*>0.5), whereas in the LVF it was highly reliable (149 ms, *P*<0.001). Figure 1 shows the effect of the cue condition for target detection in both visual fields in dyslexic and normally reading children.

Discussion

Our results indicate that the peripheral cue was generally able to elicit an exogenous orienting of attention (e.g., Posner 1980). In addition, compared with normal readers, dyslexic children were generally slower in responding to target onset (e.g., Jonkman et al. 1992). Dyslexics also showed an asymmetry between the two visual fields, with faster RTs in the RVF than in the LVF. Normal readers did not show such difference. The cue effect in the visual fields was different in the two groups. In dyslexics, the cue effect was present in the LVF, but it was absent in the RVF, whereas in normal readers the cue effect was present in both the LVF and the RVF.

Spatial attention is generally defined as the cognitive operation that allows the selection of a particular area where information processing is facilitated. It has also been suggested that the act of shifting attention to one side of the visual field facilitates the selection and detection of information on that side, meanwhile causing inhibition of information processing in the contralateral visual field (Facoetti 2001; Posner and Rafal 1987). A similar conclusion was also reached by Cohen et al. (1994), who stated that cueing of a spatial position results in the activation of the corresponding visual field and in the active inhibition of the contralateral visual field.

The pattern found in dyslexics might be interpreted as follows. Valid trials were similar in both fields because when the cue provided correct information, the facilitation mechanism was activated for the corresponding visual field, either to the right or to the left. On the other hand, the inhibition mechanism of the contralateral visual field occurred only in the LVF. There was no inhibition in the RVF because invalid trials did not differ from valid ones. Therefore, responses seemed to be facilitated in the RVF.

In conclusion, it appeared that in dyslexic children suppression of information in the RVF was absent when a cue was presented in the opposite visual field. In addition, planned comparisons revealed a close to significance (*P*=0.08) differential cue effect between dyslexics and normally reading children in the LVF (see Fig. 1). This seems to indicate that, in the invalid cue condition, not only dyslexics show a lack of inhibition in the RVF, but they also exhibit a stronger inhibition in the LVF. The next experiment was aimed at investigating whether such differences were present in the endogenous attention mechanism too. However, given previous evidence of normal functioning of voluntary orienting in children with dyslexia (Facoetti et al. 2000b; Jonkman et al. 1992), it is assumed that, in the next experiment, asymmetric hemispheric control of visual attention is absent or reduced.

Experiment 2

The aim of the present experiment was to explore orienting in dyslexic and normally reading children when attention is controlled endogenously. Given the results of experiment 1, our aim was to verify whether, given a cognitive cue, the two groups of subjects differed in the ability to orient their attention. This experiment differed from experiment 1 under two main aspects. First, the cue was presented on the center of the visual field; second, a 750-ms SOA was used. It is known that voluntary control of attention seems to produce a peak of facilitation after 500 ms from cue presentation (Müller and Rabbitt 1989; Warner et al. 1990).

Materials and methods

Participants

Participants were 23 children (16 males and 7 females). Inclusion criteria were the same as in experiment 1. Ten children (7 males and 3 females, mean age 11.5 years (SD=1.87), were classified as dyslexic as their performances in oral reading of a text, words, and nonwords were 2 SDs below the norm on age-standardized Italian tests. The group of children with dyslexia showed both “visual” and “phonological” symptoms (mixed-type dyslexia). The other 13 children (9 males and 4 females, mean age 11.1 years (SD=1.6), were classified as normal readers. Children of the two groups were individually matched for age and IQ. Table 2 reports descriptive data of the two groups. None of the subjects had participated in experiment 1.

Apparatus and procedure

They were the same as in experiment 1, but on valid and invalid trials the cue was a unidirectional arrow presented centrally (above the fixation point), pointing to the right or to the left side. A 750-ms SOA was used.

Table 2 Descriptive data on the two groups in experiment 2

	Age (years)		Full IQ (WISC-R)		Reading test			
					Accuracy errors/ 200 syllables		Speed time for syllable %	
	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Normal readers	11.1	8–15	110	94–120	3.1	1–6	24	16–35
Dyslexics	11.5	8–15	104	86–112	13.4	8–23	109	75–193

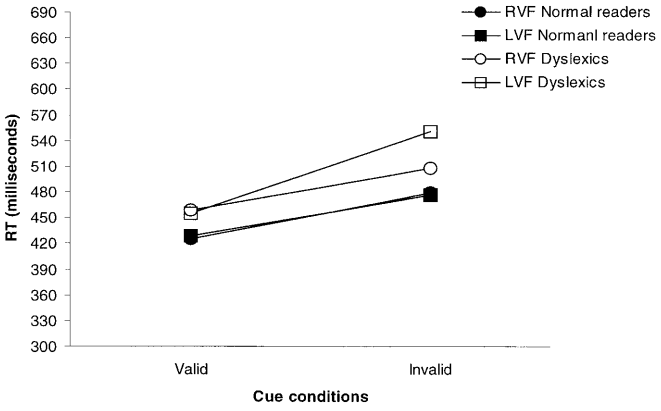

Fig. 2 The cue effect for dyslexics and normal readers in experiment 2. *RVF* (right visual field) and *LVF* (left visual field) refer to the target position

Results

Errors, that is responses on catch trials and missed responses, were less than 2.6% and were not analyzed. The outliers-latency criterion removed less than 1.5% of the data. Eye movements were about 2% of total trials. Mean correct RTs were analyzed with a three-way ANOVA, in which the between-subjects factor was group (dyslexic children and normally reading children), and the within-subjects factors were cue condition (valid and invalid) and target visual field (RVF and LVF).

The group main effect was not significant: $F(1,21)=1.774$, $P>0.20$. The main effect of cue-condition was significant, $F(1,21)=71.318$, $P<0.001$. RTs were faster on valid trials (440 ms) and slower on invalid ones (500 ms). The main effect of visual field was not significant: $F(1,21)=1.907$, $P>0.1$. The following interactions, cue condition × visual field [$F(1,21)=2.867$, $P>0.1$], cue condition × group [$F(2,21)=2.382$, $P>0.1$], and group × visual field [$F(1,21)=2.117$, $P>0.1$], were not significant. However, the cue condition × group × visual field interaction was significant: $F(2,21)=8.376$, $P<0.01$. Data were further explored by planned comparisons. As shown in Fig. 2, the cue effect was present in both groups in both visual fields (all *P*s <0.01). However, whereas in normally reading children valid and invalid trials were similar in the two visual fields (all *P*s >0.6), dyslexics showed a different RT pattern depending on the cue condition and the visual field. There was no difference between the LVF and the RVF on valid trials ($P>0.6$), whereas on invalid trials RTs were slower in the LVF than in the RVF (RT difference 42 ms, $P<0.001$). In addition, planned comparisons showed that, in the dyslexic children, the cue effect was significantly greater in the LVF (95 ms) than in the RVF (49 ms) ($P<0.01$).

Discussion

As already found in previous studies (e.g., Posner 1980), a central cognitive cue was effective in eliciting correct orienting of attention. The effect of the cue in dyslexic children was different in the two visual fields, whereas normal readers did not show any difference between the two fields. On invalid trials dyslexics showed slower RTs in the LVF (550 ms) than in the RVF (508 ms). As suggested in the discussion of experiment 1, this might indicate a stronger inhibitory effect in the LVF than in the RVF in the invalid-cue condition. In accordance with that, planned comparisons showed that, in the LVF, the cue effect was significantly greater for dyslexics (95 ms) than for normal readers (46 ms) ($P<0.01$). However, unlike experiment 1, in the RVF endogenous orienting seemed to be effective in dyslexics, as indicated by the presence of the cue effect. Like in experiment 1, normally reading children showed a reliable cue effect in both visual fields, thus providing evidence for a correct endogenous orienting of attention and no visual field asymmetry.

General discussion

In the present study, the behavioral correlates (simple RTs) of the ability to shift attention to both visual fields were investigated in dyslexic and normally reading children. Subjects with normal reading skills showed a symmetric visual field ability to orient the focus of attention, either exogenously or endogenously. On the other hand, dyslexic children shaved an asymmetric visual-field control of orienting, which would impair exogenous capture to a greater extent than endogenous orienting.

An interesting finding is the lack of the cue effect in the RVF when attention is triggered by a peripheral cue (experiment 1). We speculated that, when a stimulus was presented in the LVF, the right brain hemisphere did not inhibit stimulus processing in the left hemisphere, namely the processing of stimuli presented to the RVF. In fact, we found no RT differences between valid and invalid trials in the RVF. By contrast, when the left hemisphere processed visual information coming from the RVF, it sent an inhibitory signal to the right hemisphere, as hypothesized on the basis of the cue effect observed in the LVF. It should be noted that the size of the cue effect in the LVF of dyslexics was greater than that of normal readers. This may suggest that dyslexics suppressed information coming from the LVF to a greater extent than normal readers. In addition, this increased suppression could be related to lack of inhibition in the RVF. Usually, the two brain hemispheres compete by mutual inhibition to take control of visual information processing in the visual field (Cohen et al. 1994; Nakamura and Gazzaniga 1978; Seyal et al. 1995). However, a parietal cortex deficit may lead to a decreased inhibitory influence on the contralateral hemisphere (Ro et al. 1998; Smania et al. 1998). Therefore, when the cue was presented in the LVF, the left hemisphere was not suppressed by the right one, causing lack of inhibition for the following target stimuli presented in the RVF. Likewise, this lack of inhibition from the right hemisphere would fail to compensate the inhibition exerted by the left hemisphere. It follows that, when the cue was shown in the RVF, the left hemisphere suppressed the right hemisphere, determining slower RTs for the stimuli presented in the LVF.

The results of experiment 2 confirmed the hypothesized right posterior parietal cortex (PPC) dysfunction, showing slower RTs on invalid trials in the LVF than in the RVF (Posner et al. 1984, 1987). On the other hand, the difference between the two visual fields was reduced when orienting was driven endogenously, as indicated by the fact that the cue effect was present in the RVF, and it was comparable to that of normal readers (see Fig. 2).

The main reading problem of dyslexic children appears to be caused by poor phonological skills (Bradley and Bryant 1983), but also by frequent visual-perceptual problems. A promising line of research revealed the

presence of a selective deficit in the processing of visual information caused by a dysfunction of the M-system (Stein and Walsh 1997), which, as suggested by Livingstone and Hubel (1988), encodes the visual information about location and movement of stimuli. Recent studies on visual attention pointed out that visual-spatial attention is dominated by M-system inputs (e.g., Steinman et al. 1996), which culminate on the PPC (Ungerleider and Haxby 1994). The PPC is thought to play an important role in the reading process, since it seems to regulate the normal control of ocular movements and fixations (Anderson et al. 1994), the perception of spatial position, the movement of stimuli (Sagi and Julesz 1985), and the distribution of visual-spatial attention (Townsend and Courchesne 1994). Accordingly, damage to this region determines an acquired reading disorder (Kinsbourne and Warrington 1962; Shallice and Warrington 1977). In addition, deficits due to an anomalous functioning of the PPC are similar to those observed in dyslexia: incorrect recognition of letter position, reversal of letters, "cocktail party" problems, as well as impairment in both visuo-motor coordination and visuo-verbal association (Stein and Walsh 1997). Electroencephalographic and regional cerebral blood flow studies revealed an unusual pattern of activation in the PPC, which is consistent with the abnormal activity observed in the parietal cortex of dyslexic subjects (Duffy et al. 1980; Wood et al. 1991). To investigate the pathophysiology of dyslexia, Eden et al. (1996) used functional magnetic resonance imaging (fMRI) to study visual motion processing in normal and dyslexic male subjects. In all dyslexics, presentation of moving stimuli failed to produce the same task-related functional activation in area V5/MT, which is part of the M-system localizable to the parietal lobe. In addition to evidence showing the role of PPC in reading disorders, a selective attention deficit has long been known to be associated with reading-disabled individuals. A visual search task requires a rapid and precise control of attention to single out a target among many distracters (Treisman and Souther 1985), and in subjects with dyslexia searching for relevant stimuli among distracters it seems to be particularly defective (Ruddock 1991; Vidyasagar and Pammer 1999; Williams et al. 1987). A possible explanation for this deficit might be that dyslexics present an altered functioning of the focused attention modality (Facoetti et al. 2000a; Geiger et al. 1994; Williams and Bologna 1985).

The task used in the present experiments is a typical task engaging orienting functions, which are known to be mainly controlled by the dorsal stream or M-system, whose end station is the PPC (Vidyasagar 1999). Specifically, recent studies have shown that the right parietal lobe is dominant for selective spatial attention (e.g., Chelazzi 1999; Kim et al. 1999). The present results and our speculations about a right PPC dysfunction are consistent with recent psychophysical evidence suggesting that dyslexics would suffer from a left-side "mini-neglect" (Hari and Koivikko 1999). Fowler et al. (1991) and Eden et al. (1993) demonstrated that dyslexic children show poorer visual search performances in the LVF than in the RVF. A weakness of the right parietal lobe in dyslexics has also been suggested by Hari et al. (1999), who showed a prolonged attentional dwell time in dyslexic subjects. Dyslexic children showed an asymmetrical distribution of visual spatial attention using both interference tasks of irrelevant lateral stimuli (Facoetti and Turatto 2000) and detection stimuli that appeared outside the attentional focus (Facoetti and Molteni 2001). Also, Eden et al. (1994) recently proposed that the presence of oculomotor control abnormalities in dyslexic children could be due to a dysfunction of the right parietal cortex. A strong inhibition in the LVF could also hamper rapid and exact planning of regression saccades (backward movements from right to left), that is deemed as fundamental for fluent and correct reading and which is known to be altered in children with dyslexia (Morris and Rayner 1991). Lastly, a study of Schulte-Körne et al. (1999; also see Mazzotta and Gallai 1992), with visual evoked potentials, revealed a visual information processing deficit in the right brain hemisphere (posterior region).

It is worth noting, however, that experiment 2 showed a reduction in the orienting deficit, which could be attributed to two main factors. First, in experiment 2 attention was allocated using a "central" cue and, secondly, in comparison with experiment 1, subjects had more time for orienting (350 vs. 750 ms cue-target delays). Although both factors (cue location and SOA) might have produced different RT patterns between experiments 1 and 2, it should also be mentioned that there is evidence for different neuroanatomical structures underlying exogenous and endogenous mechanisms. The former seems to be mainly controlled by the parietal lobe, whereas the latter seems to be mainly controlled by the frontal lobe (Posner and Petersen 1990). However, a recent study by Corbetta et al. (2000) suggests that the intra-parietal sulcus (IPS) was activated before presentation of the target stimulus. In fact, IPS was the only region to show sustained activation after the cue presentation, presumably forcing the subject to attend to the cued location. On the other hand, it was shown that the temporo-parietal junction (TPJ) showed no activation during the cue presentation, whilst activation of the right TPJ was greater during the target presentation in invalid than during valid trials.

It is interesting to note that findings of the present study are, however, in line with two other etiological hypotheses of developmental dyslexia. The former reflects a deficit in interhemispheric processing that bears a causal connection to the reading disorder. Therefore, the present results provide converging evidence for the interhemispheric dysfunction hypothesis of dyslexia that was first advanced by Hynd et al. (1979). A magnetic resonance imaging (MRI) study which revealed a significantly smaller corpus callosum in a group of dyslexic children is consistent with this conclusion (Hynd et al. 1995). In addition, the RTs of the callosotomized subject showed a left-right gradient for both cue and target locations, being longest for the leftmost location and shortest for the right

locations. The rightward bias could be attributed to the callosal interhemispheric disconnection rather than to the right parietal dysfunction (Berlucchi et al. 1997).

In contrast, the other hypothesis refers to the cerebellar deficit hypothesis, which is now held to be one of the main causal theories of developmental dyslexia. The cerebellum may be specially designed for accurate timing of events, concerning not only movements, but also certain cognitive tasks. Recent evidence suggests that the cerebellum is involved in learning of the automaticity, not only in motor skills, but also in language and cognitive skills via the rich interconnections in the brain (for a review, see Nicolson and Fawcett 1999). Indeed, Townsend et al. (1999) present evidence of slowed covert orienting of visual spatial attention in patients with developmental and acquired cerebellar abnormality. Patients with cerebellar dysfunction showed little evidence of having oriented the attentional focus within 100 ms, but did show the effects of attention orienting after 800–1200 ms. These data suggest that damage to the cerebellum disrupts the spatial encoding of a location for an attentional shift. Consistent with the cerebellar deficit hypothesis, Facoetti et al. (2000b) showed that dyslexic children have a specific disability in the shifting of visual attention caused by a spatial cue at shorter cue-target delays. In addition, the cerebellum is biochemically asymmetric in dyslexic men, indicating altered development of this organ (Rae et al. 1998). Other results provided direct evidence that, in dyslexic adults, the behavioral signs of cerebellar abnormality reflect underlying abnormalities most in right cerebellar activation (Nicolson et al. 1999).

What is the relation between our findings and developmental dyslexia?

Detection of a letter within a word or a word within a text seems to require a precise control of the size of the attentional focus to exclude irrelevant information (LaBerge and Brown 1989). An excessive inhibition of LVF stimuli (left inattention), concomitant with a lack of inhibition of RVF stimuli (right over-distractibility) may influence the decoding process of words either by an anomalous suppression of letters in the left side of a string, or by a difficulty in the inhibition of distracting peripheral stimuli coming from the RVF, which corresponds to the direction of reading. The present study might suggest some explanations for the frequent visuoperceptual problems found in developmental dyslexia like, for instance, anticipation of letters, frequent errors in word endings, wrong position of letters within a word, hesitations, pauses, and slowness during reading. An increased distractibility in the RVF together with mild inattention for stimuli in the LVF may indirectly explain, via the oculomotor control system, movement, deformation, and overlapping phenomena of letters and/or words reported by children with dyslexia. The involvement of visual spatial attention in reading disorders has been clearly pointed out by Stein and Walsh (1997).

Acknowledgements The authors wish to thank Barbara Alberti for translation, Caterina Sala, Pierluigi Paganoni, and Massimo Molteni for their help in conducting this study. Thanks are also due to two anonymous reviewers for their helpful comments on the manuscript.

References

American Psychiatric Association (1994) Diagnostic and statistical manual of mental disorders, 4th edn. DC author, Washington

Anderson TJ, Jenkins IH, Brooks DJ, Hawken MB, Frackowiak RSJ, Kennard C (1994) Cortical control of saccades and fixation in man. A PET study. Brain 117:1073–1084

Atkinson J (1991) Review of human visual development: crowding and dyslexia. In: Stein JF (ed) Vision and visual dysfunction, Vol 13. MacMillian Press, London, pp 44–45

Berlucchi G, Tassinari G, Marzi CA, Di Stefano M (1989) Spatial distribution of the inhibitory effect of peripheral non-informative cues on simple reaction time to non-fixated visual targets. Neuropsychologia 27:201–221

Berlucchi G, Aglioti S, Tassinari G (1997) Rightward attentional bias and left hemisphere dominance in a cue-target light detection task in a callosotomy patient. Neuropsychologia 35:941–952

Bradley L, Bryant (1983) Categorizing sounds and learning to read: a causal connection. Nature 301:419–421

Brannan J, Williams M (1987) Allocation of visual attention in good and poor readers. Percept Psychophys 41:23–28

Briggs GG, Nebes RD (1975) Patterns of hand preference in a student population. Cortex 11:230–238

Casco C, Tressoldi P, Dellantonio A (1998) Visual selective attention and reading efficiency are related in children. Cortex 34:531–546

Chelazzi L (1999) Serial attention mechanisms in visual search: a critical look at the evidence. Psychol Res 62:195–219

Cohen JD, Romero RD, Servan-Schreiber D, Farah MJ (1994) Mechanisms of spatial attention: the relation of macrostructure to microstructure in parietal neglect. J Cogn Neurosci 6:377–387

Coltheart M, Curtis B, Aktins P, Haller M (1993) Models of reading aloud dual-route and parallel-distributed processing approaches. Psychol Rev 100:589–608

Corbetta M, Kincade JM, Ollinger JM, McAvoy MP, Shulman GL (2000) Voluntary orienting is dissociated from target detection in human posterior parietal cortex. Nat Neurosci 3:292–297

Duffy FH, Denckla MB, Bartels PH, Sandini B (1980) Dyslexia: regional differences in brain electrical activity by topographical mapping. Ann Neurol 7:412–420

Eden GF, Stein JF, Wood FB (1993) Visuospatial ability and language processing in reading disabled and normal children. In: Wright SF, Groner R (ed) Studies in visual information processing: facets of dyslexia and its remediation. North-Holland, Amsterdam, pp 321–335

Eden GF, Stein JF, Wood MH, Wood FB (1994) Differences in eye movements and reading problems in dyslexic and normal children. Vision Res 34:1345–1358

Eden GF, VanMeter JW, Rumsey JM, Malsog JM, Woods RP, Zeffiro TA (1996) Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. Nature 382:66–69

Facoetti A (2001) Facilitation and inhibition mechanisms of human visuospatial attention in a non-search task. Neurosci Lett 298:45–48

Facoetti A, Molteni M (2001) The gradient of visual attention in developmental dyslexia. Neuropsychologia 39:352–357

Facoetti A, Turatto M (2000) Asymmetrical visual fields distribution of attention in dyslexic children: a neuropsychological study. Neurosci Lett 290:216–218

Facoetti A, Paganoni P, Lorusso ML (2000a) The spatial distribution of visual attention in developmental dyslexia. Exp Brain Res 132:531–538

Facoetti A, Paganoni P, Turatto M, Marzola V, Mascetti GG (2000b) Visuospatial attention in developmental dyslexia. Cortex 36:109–123
Fowler MS, Richardson AJ, Stein JF (1991) Orthoptic investigation of neurological patients undergoing rehabilitation. Br Orthoptic J 48:2–27
Geiger G, Lettvin JY, Fahle M (1994) Dyslexic children learn a new strategy for reading: a controlled experiment. Vision Res 34:1223–1233
Hari R, Koivikko H (1999) Left-side mini-neglect and attentional sluggishness in dyslexic adult. Soc Neurosci Abstr 25:1634
Hari R, Valta M, Uutela K (1999) Prolonged attentional dwell time in dyslexic adult. Neurosci Lett 271:202–204
Hynd G, Obrzut J, Weed W, Hynd C (1979) Developmen of cerebral dominance: dichotic listening asymmetry in normal and learning-disabled children. J Exp Child Psycol 28:445–454
Hynd G, Hall J, Novey E, Eliopulos D, Black K, Gonzales JJ, Edmonds JE, Riccio C, Cohen M (1995) Dyslexia and corpus callosum morphology. Arch Neurol 52:32–38
Inhoff AW, Pollatsek A, Posner MI, Rayner K (1989) Covert attention and eye movements during reading. Q J Exp Psychol 41:63–89
Jonides J (1981) Voluntary versus automatic control over the mind's eye's movement. In: Long JB, Baddeley AD (ed) Attention and performance. Erlbaum, Hillsdale, pp 187–203
Jonkman I, Licht R, Bakker DJ, Van den Broek-Sandmann TM (1992) Shifting of attention in subtyped dyslexics children: an event-related potential study. Dev Neuropsych 8:243–259
Kim YH, Gitelman D, Nobre A, Parrish T, LaBar K, Mesulam MM (1999) The large-scale neural network for spatial attention displays multifunctional overlap but differential asymmetry. Neuroimage 9:269–277
Kinsbourne M, Warrington EK (1962) A variety of reading disability associated with right hemisphere lesions. J Neurol Neurosurg Psychiatry 25:339–344
LaBerge D, Brown V (1989) Theory of attentional operations in shape identification. Psychol Rev 96:101–124
Livingstone MS, Hubel DH (1988) Segregation of form, color movement, and depth: anatomy, physiology and perception. Science 240:740–749
Mazzotta G, Gallai V (1992) Study of the P300 event-related potential through brain mapping in phonological dyslexics. Acta Neurol 14:173–186
McArthur GM, Hogben JH, Edwards VT, Heath SM, Mengler ED (2000) On the "specifics" of specific reading disability and specific language impairment. J Child Psychol Psychiatry 41:869–874
Morris RK, Rayner K (1991) Eye movements in skilled reading: implications for developmental dyslexia. In: Stein JF (ed) Vision and visual dyslexia. MacMillan Press, London, pp 233–242
Müller HJ, Rabbitt PMA (1989) Reflexive and voluntary orienting of visual attention: time course of activation and resistance to interruption. J Exp Psychol Hum Percept Perform 15:315–330
Nakamura RK, Gazzaniga MS (1978) Hemispherectomy vs. commisurotomy in the monkey: one hemisphere can be better than two. Exp Neurol 59:202–208
Nicolson RI, Fawcett AJ (1999) Developmental dyslexia: the role of the cerebellum. Dyslexia 5:155–177
Nicolson RI, Fawcett AJ, Berry EL, Jenkins H, Dean P, Brooks DJ (1999) Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. Lancet 353:1662–1667
Pavlidis G (1981) Do eye movements hold the key to dyslexia? Neuropsychologia 19:57–64
Petersen SE, Robinson DL, Currie JN (1989) Influences of lesions of parietal cortex on visual spatial attention in humans. Exp Brain Res 76:267–280
Posner MI (1980) Orienting of attention. Q J Exp Psychol 32A:2–25
Posner MI, Petersen SE (1990) The attentional system of the human brain. Annu Rev Neurosci 13:25–42
Posner MI, Rafal RD (1987) Cognitive theories of attention and the rehabilitation of attentional deficits. In: Meier MJ, Benton AL (eds) Neuropsychological rehabilitation. Guilford Press, New York, pp 182–201
Posner MI, Snyder CRR, Davidson BJ (1980) Attention and the detection of signals. J Exp Psychol Gen 109:160–174
Posner MI, Walker JA, Friedrich AJ, Rafal RD (1984) Effect of parietal lobe injury on cover orienting of visual attention. J Neurosci 4:1863–1874
Posner MI, Walker JA, Friedrich FA, Rafal RD (1987) How do the parietal lobes direct covert attention? Neuropsychologia 25:135–145
Rae C, Lee MA, Dixon RM, Blamire AM, Thompson CH, Styles P, Talcott J, Richardson A, Stein JF (1998) Metabolic abnormalities in developmental dyslexia detected by ^{1}H magnetic resonance spectroscopy. Lancet 351:1849–1852
Ro T, Cohen A, Ivry RB, Rafal RD (1998) Response channel activation and the tempoparietal junction. Brain Cogn 37:461–476
Ruddock KH (1991) Visual search and dyslexia. In: Stein JF (ed) Vision and visual dyslexia. MacMillan Press, London, pp 58–79
Sagi D, Julesz B (1985) "Where" and "what" in vision. Science 228:1217–1219
Schulte-Körne G, Bartling J, Deimel W, Remschmidt H (1999) Attenuated hemispheric lateralization in dyslexia: evidence of a visual processing deficit. Neuroreport 10:3697–3701
Seyal M, Ro T, Rafal R (1995) Increased sensitivity to ipsilateral cutaneous stimuli following transcranial magnetic stimulation of the parietal lobe. Ann Neurol 38:264–267
Shallice T, Warrington EK (1977) The possible role of selective attention in acquired dyslexia. Neuropsychologia 15:31–41
Smania N, Martini MC, Gambina G, Tomelleri G, Palamara A, Natale E, Marzi CA (1998) The spatial distribution of visual attention in hemineglect and extinction patients. Brain 121: 1759–1770
Stein J, Walsh V (1997) To see but not to read; the magnocellular theory of dyslexia. Trends Neurosci 20:147–152
Steinman BA, Steinman SB, Lehmkuhle S (1996) Transient visual attention is dominated by the magnocellular stream. Vision Res 36:589–599
Townsend J, Courchesne E (1994) Parietal damage and narrow "spotlight" spatial attention. J Cogn Neurosci 6:220–232
Townsend J, Courchesne E, Covington J, Westerfield M, Singer Harris N, Lyden P, Lowry TP, Press GA (1999) Spatial attention deficits in patients with acquired or developmental cerebellar abnormality. J Neurosci 19:5632–5643
Treisman A, Souther J (1985) Search asymmetry: a diagnostic for preattentive processing of separable feature. J Exp Psychol Gen 114:285–310
Ungerleider LG, Haxby JV (1994) "What" and "where" in the human brain. Curr Opin Neurobiol 4:157–165
Vidyasagar TR (1999) A neural model of attentional spotlight: parietal guiding the temporal. Brain Res Rev 30:66–76
Vidyasagar TR, Pammer K (1999) Impaired visual search in dyslexia relates to the role of the magnocellular pathway in attention. Neuroreport 10:1283–1287
Warner CB, Juola JF, Koshino H (1990) Voluntary allocation versus automatic capture of visual attention. Percept Psychophys 48:243–251
Wechsler D (1986) Scala di Intelligenza Wechsler per Bambini-Riveduta. Organizzazioni Speciali Firenze
Williams MC, Bologna NB (1985) Perceptual grouping in good and poor readers. Percept Psychophys 38:367–374
Williams MC, Brannan JR, Latirgue EK (1987) Visual search in good and poor readers. Clin Vision Sci 1:367–371
Wood F, Felton R, Flowers L, Naylor C (1991) Neurobehavioural definition of dyslexia. In: Dwayne D, Grey D (eds) The reading brain. Parkton Press, New York, pp 1–25
Yantis S, Jonides J (1984) Abrupt visual onset and selective attention: evidence from visual search. J Exp Psychol Hum Percept Perform 10:601–621