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Posterior parietal cortex and developmental dyslexia

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Review

Abstract. Dyslexia is defined as a specific reading disorder despite normal intelligence and conventional teaching. One of the most influential theories attempting to explain problems suffered by dyslexics assumes that dyslexia is caused by deficits of the magnocellular system. This system, generally responsible for processing fast sensory information, projects mostly to the parietal cortex. Consistent with this theory, dyslexics should have problems with tasks which specifically involve parietal cortex. In the article, we review data and show that, indeed, dyslexics have problems with fast attention shifts, show some symptoms of mild unilateral neglect syndrome and have abnormal saccadic and pursuit eye movements. Little is known about visuo-motor coordination and mental rotation, the tasks in which the parietal cortex is thought to play important roles.

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INTRODUCTION

Developmental dyslexia is a disorder found in 5–15% of school-aged children. It is traditionally defined as specific difficulties in reading and writing which are not consequences of mental handicap, damage to sense organs (vision, hearing), emotional and behavioral disorders, or socio-cultural failure. Despite the existence of serious problems in understanding the etiology of developmental dyslexia, researchers agree on its basic symptoms. The following specific difficulties of dyslexics are most often mentioned in the literature: very slow speed of reading and writing, specific difficulties in decoding and recognizing single words, in phonological features of spoken and written language, in maintenance of reading and writing direction from left to right and in understanding and memorization of the content of written text. Moreover, dyslexics often omit or change the place of letters and syllables within words during reading and writing, alter unknown and difficult words into sensible or absurd ones, make spelling errors in spite of their knowledge of orthographical and grammatical rules (Willows 1998, Willows and Terepocki 1993).

Besides these symptoms, dyslexics show also a myriad of other non-linguistic difficulties including problems with co-ordination of eye movements, motor control and early sensory processing.

The number of accounts of developmental dyslexia is almost as high as the number of symptoms described in the literature. However, besides the most influential theory of phonological skills deficits (Bradley and Bryant 1978, Liberman 1983, Liberman et al. 1971, Snowling 1998, 2001, Vellutino et al. 2004) only two other, in our view, should be mentioned as theories which have been attracting remarkable scientific attention. These are the magnocellular (Stein and Walsh 1997, Stein et al. 2000, 2001) and the cerebellar deficit theory (Fawcett et al. 1996, Nicolson and Fawcett 1999, Nicolson et al. 2001). They will be described in more detail later in this paper. The main concern of this paper will, however, be problems of dyslexics viewed as the parietal lobe dysfunctions.

Traditionally the posterior parietal cortex (PPC) was believed to be an "association area", a higher-level sensory structure responsible for associating different sensory modalities. Indeed, PPC seems to be the "crossroads of the brain" (Critchley 1953): it receives information from three important sensory inputs – visual, auditory and tactile – and projects to many cortical and subcortical structures. Currently, PPC is considered to play much more complex roles. It is generally accepted that PPC is responsible for sensorimotor integration. Indeed, within this structure there are centers controlling the processing of spatial relations and visually guided movements (e.g., Goodale and Milner 2004, Milner and Goodale 1995, Pisella et al. 2000), spatial attention (e.g., Bisley and Goldberg 2003a, Corbetta and Shulman 2002, LaBerge 2002, Stein and Glickstein 1992) and eye movement (Andersen 1989, Bisley and Goldberg 2003b). It remains unclear, however, where exactly "the area sits along this sensorimotor continuum" (Andersen and Buneo 2003, p. 162).

PPC consists of Broadmann's area 5, 7a and 7b. These fields were recognized on cytoarchitectural criteria. Further neuroanatomical, clinical and physiological investigations revealed that cells in PPC form small (ca. 0.25 cm^2) subregions of different connectivity and response properties (Andersen and Buneo 2003).

Lesions of PPC lead to many different deficits. Of importance for the subject of this paper, many problems of dyslexics can be viewed as mild symptoms of the parietal lobe dysfunctions. Bearing in mind the predominance of magnocellular input into PPC and magnocellular deficits in dyslexics this seems not to be surprising. In the following, we briefly review evidence indicating PPC deficits in dyslexia, especially concerning attention, eye movements and mental rotation. We will also suggest some possible practical consequences and new lines of investigations. Due to our own scientific interests, the review will be restricted to visual problems only.

MAGNOCELLULAR DEFICITS IN DYSLEXIA

In an attempt to describe interconnections between the different anatomical structures involved in visual processing, Ungerleider and Mishkin (1982) suggested that two anatomical pathways originate in occipital cortex (V1 and V2). The dorsal path projects to PPC. The ventral path projects to inferior-temporal cortex.

The functional role of the ventral system did not cause trouble. As early as in 1940s, it was known that impairment of the temporal lobes results in problems recognizing structures and shapes (Klüver and Bucy 1938). More recent findings suggest that the ventral route plays a crucial role in building representations of objects. Investigations of primate brains have shown that in inferior parts of the temporal lobes (PIT, CIT and

AIT areas) there are cells responding to specific shapes as stars or squares irrespective of where in the visual field the objects were located. Area V4 consists of neurons sensitive to color and orientation. Moreover, in the temporal cortex some neuronal assemblies were found which reacted specifically to as complex stimuli such as faces, buildings (Haxby et al. 1999, Ishai et al. 1999, 2000, Puce et al. 1996), letters (Polk and Farah 1998), animals and tools (Chao et al. 1999), hands (Puce et al. 1996), and chairs (Ishai et al. 1999). These findings were interpreted as evidence for modular organization of temporal cortex, with modules being specific for given categories of objects. Ishai and coauthors (1999) showed recently that the topology of the modules is very similar for different persons.

The investigations of primates revealed that impairments of the dorsal system led to relatively mild symptoms unless the tasks to be done involved reaching, pointing or shifting gaze to an object in the visual field (Ungerleider and Mishkin 1982). There is a bulk of behavioral, anatomical and physiological evidence showing that visual areas of parietal lobes are responsible for visual guidance of limb, eye movements and visual attention (Glickstein 2000, Glickstein et al. 1985, Goodale and Milner 2004, Milner and Goodale 1995). According to Milner and Goodale's very influential theory (Milner and Goodale 1995) the dorsal system extracts from the visual stream this information, which serves to perform visually guided actions. For example, patients with temporal lobe impairments (unlike those with PPC lesions) lose visual awareness of form and dimensions of objects in the environment, but preserve their ability to make visually guided reaching and grasping movements, i.e., they can scale their hands to the size, shape and orientation of a goal object (Milner and Goodale 1995). Milner and Goodale (1995) argue that these visuo-motor abilities are intact in such patients because they are mediated by the dorsal route.

The dorsal and ventral pathways are, in a sense, the prolongation of a neuronal division which starts already in the retina. Ten percent of retinal ganglion cells are bigger, their axons are thicker myelinated and their receptive fields are larger than the rest of the ganglion cells. This division is also visible in the lateral geniculate body. The characteristic of these two types of cells, magno (large) and parvo (small), differ significantly. Parvo cells respond relatively slowly but are sensitive to colors and due to their small receptive fields contribute to vision of high spatial resolution. Conversely, magno cells are sensitive to fast changing stimuli and moving objects and are insensitive to colors (e.g., Palmer 1999). It has been shown that magno cells dominate the dorsal-system projection from the primary visual cortex and further to attentional and eye movement control regions of the parietal lobe (Livingstone and Hubel 1988).

In 1980s, for the first time an impairment of the magnocellular system was demonstrated in dyslexics (Lovegrove et al. 1980a, 1980b, 1982). From this time, a lot of further evidence was collected showing impaired performance of dyslexics in tasks assumed to be mediated by magnocellular system, like motion perception (Cornelissen and Hansen 1998, Cornelissen et al. 1995, 1998, Eden et al. 1996, Wilmer et al. 2004) and contrast sensitivity (Lovegrove et al. 1980a,b, 1982). Neuroimaging studies have consistently shown lower activity of magnocellular pathways in dyslexics than in normals (Demb et al. 1998, Eden et al. 1996), electrophysiological studies have shown longer latencies and smaller amplitudes evoked by fast changing or moving stimuli (Kubová et al. 1995, Lehmkuhle et al. 1992, Livingstone et al. 1991) and neuroanatomical methods have revealed abnormalities in the magno layers of the lateral geniculate bodies in dyslexics (Livingstone et al. 1991).

It should be noted, however, that the issue is not uncontroversial in spite of the number of studies devoted to these deficits (Bednarek and Grabowska 2002, Evans et al. 1994, Keen and Lovegrove 2000, Sperling et al. 2003, Walther-Müller 1995). For example, Evans and coauthors (1994) administered several tasks in which magnocellular system is thought to be involved. They found some deficits in flicker detection at 10 Hz. Other tests very weakly correlated with reading abilities. Sperling and coauthors (2003) showed recently that the magnocellular deficit of dyslexics is negatively correlated with orthographic rather than with reading abilities and positively correlated with their phonological awareness. In the balance, however, much more evidence has been collected to support than to reject this hypothesis.

ATTENTIONAL DEFICITS IN DYSLEXIA

Attention and parietal cortex

Attention is a brain mechanism that enhances information processing at the attended location. Thus, atten-

tion operates as a filter removing irrelevant information from sensory-input streams. Two mechanisms of attention are distinguished (for a recent review see Corbetta and Shulman 2002) which were recognized already by James (1890). The stimulus-driven mechanism operates in bottom-up fashion and its role is to capture an intrinsic property of the stimulus, provided that it is sufficiently salient to divert attention from the current focus. Therefore, it enables the processing of novel, unexpected events. The second, goal-driven, mechanism operates in top-down fashion and consists in shifting or focusing attention according to ones will. (Note however that there is controversy surrounding the problem of how voluntary this "voluntary" attentional mechanism is (Lambert et al. 1999)).

Although some early neuropsychological studies provided evidence for the hypothesis that the brain structures controlling attention are situated in PPC (e.g., Posner and Cohen 1984), systematic research employing neuroimaging methods has only recently begun to elucidate increasingly fine-grained distinctions in the parietal cortex and their functional correlates (for reviews see Behrmann et al. 2004, Corbetta and Shulman 2002). Recent findings have shown that both mechanisms of attention are located in the parietal cortex. It has been shown that the tasks engaging the stimulus-driven attention activated the temporo-parietal junction (TPJ) (Downar et al. 2001). Two other structures of the parietal cortex are involved in the voluntary control of attention. These are superior parietal lobule (SPL) and precuncus (PC) (Giesbrecht et al. 2003, Yantis et al. 2002).

Problems with attention shift and visual search in dyslexics

The "parietal" hypothesis predicts attentional problems in dyslexics. Indeed, there are a remarkable number of studies showing some attentional deficits in dyslexics.

Brannan and Williams (1987) were the first to demonstrate that poor readers had problems with shifting attention from one target to another. Casco and coauthors (1998) classified their subjects into four categories according to their accuracy in a letter cancellation task involving selective attention. The task involved searching for a target letter in a set of background letters and accuracy was measured as a function of set size. They found that children with the lowest performance in this task read significantly slower than those with the highest performance.

These findings demonstrate a link between attention and reading achievements but do not necessarily provide an argument in favor of the attentional hypothesis of dyslexia. However, such a deficit was demonstrated by Facoetti and coauthors (Facoetti and Molteni 2001, Facoetti and Turatto 2000, Facoetti et al. 2001, 2000, 2003a,b,c, 2004). They showed several abnormalities in dyslexic children at attentional control. First, dyslexics seem to have more diffused spatial attention. In one study (Facoetti et al. 2000) the distribution of attention was measured by reaction time to detect a white dot target projected at different eccentricities from the fovea. Normally reading children showed a normal gradient of detection speed (the larger eccentricity, the longer reaction times). In contrast, the eccentricity had no effect on reaction times of dyslexic children. Furthermore, dyslexics showed sluggishness of their automatic attention. To demonstrate this, Facoetti and coauthors (2003a) used so-called cue-size procedure. The target which had to be identified was presented inside either a large circle (= cue) or a small circle. If a circle is displayed briefly before the target, attention is automatically focused inside the circle. Because of this, identification time is longer when the target is presented inside a large circle than when the circle is small. For dyslexic children, no effect of the circle size was found for the short (100 ms) cue-target interval while it was present at a longer cue-target delay (500 ms), in contrast to normally reading children who showed a cue-size effect for both intervals. These results were interpreted as a manifestation of the sluggishness of automatic focusing of visual attention in dyslexics.

A task which is typically attributed to attention function is visual search. In this task a subject is asked to locate a target stimulus embedded in an array of non-target elements. If the target can be distinguished from non-targets by a unique feature, search time is independent of the array size. Such a result is traditionally interpreted as resulting from pre-attentive parallel processing. If search time increases with array size, it is assumed that attention has to be directed to each item until the target is located (for a review see Quinlan 2003). Iles and coauthors (2000) and Vidyasagar and Pammer (1999) showed that the search time–size function increased more steeply for dyslexics than for normals in a serial search task. Even more important is that Iles and coauthors (2000) found clear correlation between

magnocellular deficit and search performance supporting the idea that magnocellular deficit extended to PPC functions.

Unfortunately, the findings on search performance in dyslexics are inconsistent. For example, Facoetti and coauthors (2000) found flatter serial search function in dyslexics than in normals, while Casco and Prunetti (1996) and Hayduk and coauthors (1996) found no differences.

Minineglect syndrome

The most intriguing dyslexic dysfunction described by Facoetti and coauthors (Facoetti and Molteni 2001, Facoetti and Turatto 2000) is the left-right asymmetry of attentional resources. For example, in one study (Facoetti et al. 2001) they used the Posner paradigm (Posner 1980) with peripheral and central cues which validly or invalidly cued the target location. The subjects' task was to react to the target side. To test stimulus-driven attention, the cue consisted of a peripheral abrupt onset. For normal readers, the typical cue effect was found, i.e. performance superiority of validly cued target over invalidly cued target and no visual field asymmetry. In contrast, dyslexics showed no cue effect when the target was presented in the right visual field. Surprisingly, when the target was presented in the left visual field the cue effect was even greater in dyslexics than in normally reading children. Similar (although much smaller) asymmetry of cue effect was found when central cues were used. This last finding indicates that this attentional asymmetry also concerns the goal-driven attention.

A very similar left-right asymmetry was observed by Hari and coauthors (2001) in the temporal order judgments (TOJ) task. Two visual stimuli were presented, one to the left and one to the right of the fixation point. The subjects' task was to indicate their order. Hari and coauthors (2001) found that dyslexics performed worse if the left stimulus was displayed first. Moreover, a similar left-to-right deficit was demonstrated for the so-called line-motion phenomenon. The line-motion phenomenon is an illusion in which a continuous horizontal line presented on the screen is perceived as an unrolling line if it is preceded by a peripheral cue (Shimojo et al. 1997)[1].

On the basis of these results, Hari and coauthors (2001) hypothesized that dyslexics showed a left-side minineglect syndrome, i.e., a disadvantage of the left visual hemifield in selecting and processing visual information. Unilateral neglect occurs usually after a posterior brain damage. Patients with such damage may fail to orient towards or report information that appears on the side of space opposite the lesion. For example, they may copy or draw only features on the ipsilesional side. Of importance for our considerations is the finding that neglect occurs more often and with greater severity after right than left hemisphere lesions in humans (Barttolomeo and Chokron 2004, Driver and Vuilleumier 2001)

Two further empirical facts corroborate minineglect as a possible cause of dyslexia. First, Rorden and coathors (1997) demonstrated that neglect patients show a remarkable left-right asymmetry in the TOJ task indicating that the similar asymmetry in dyslexics might have the same cause. Second, it is well known that neglect patients have severe reading problems. It must be emphasized, however, that the errors made by neglect patients are similar but not identical to those seen in dyslexics. Usually two types of errors have been reported: omissions of the most leftward letters (e.g., Behrmann et al. 1990) or the most rightward letters for people reading from right-to-left (Friedmann and Nachman-Katz 2004) and substitution (e.g., Làdavas et al. 1997). Typical errors for dyslexia (i.e., changing of letter order or confusing the letter of the same shape but different spatial orientation like "b" and "p") were not described in the literature devoted to unilateral dyslexia. However, many aspects of reading problems in the unilateral neglect patients are still unclear and controversial (Behrmann et al. 2004).

According to Hari and coauthors (2001), the minineglect syndrome is caused by magnocellular deficit. Indeed, as the magnocellular system projects mostly to the parietal cortex and the circuits controlling attention are located in the dorsal system, a diffuse functional disruption of the magnocellular pathway could weaken the input to this cortex. Moreover, the unilateral neglect syndrome usually stems from an impairment of the right rather than the left parietal cortex. Therefore, it seems reasonable to assume that generally weakened magnocellular input should result in a left-side disadvantage.

[1]Unfortunately, we failed to replicate the Hari and coauthors (2001) study (Jaśkowski and Rusiak, unpublished results). Under very similar experimental conditions we found general deficit in TOJs independent of whether the left or right stimulus was presented first.

Similarly, Facoetti and coauthors (Facoetti and Turatto 2000, Facoetti et al. 2003a) interpreted their results of attentional deficits in dyslexia keeping in mind the role that the magnocellular system is thought to play in the attention control (for recent evidence supporting this idea see Buchholz and McKone 2004, Kinsey et al. 2004).

To summarize, although this hypothesis seems to be very promising, it needs further tests. In particular, it would be important to check how dyslexics perform on other tests which are typically used in unilateral neglect. Line bisection is such a task (Ferber and Karnath 2001, Misonou et al. 2004): when asked to divide a horizontal interval into two equal parts, unineglect patients have a tendency to put a division mark on the right side of the interval rather than in the middle. The same tendency one may expect in dyslexics. However, Polikoff and coauthors (1995) found that dyslexics seemed to have rather a right-side neglect, if any: they tended to put marker slightly more to the left than normals. To the best of our knowledge, cancellation tests (Ferber and Karnath 2001, Wojciulik et al. 2004) have not been performed in dyslexics, yet.

The minineglect syndrome hypothesis also provides a causal explanation of reading problems. Indeed, as mentioned, patients with unilateral neglect have serious problems with reading. In this case, however, it remains an open question why the errors made by dyslexics in reading are much more diverse than those made by neglect patients.

To summarize, although this hypothesis seems to be very promising, it needs further tests. In particular, it would be important to check how dyslexics perform on other tests which are typically used in unilateral neglect diagnosis, "normal" cancellation test (Ferber and Karnath 2001) or so-called invisible cancellation test (Wojciulik et al. 2004). To the best of our knowledge such tests have not been performed yet.

It would also be interesting to compare the eye movement patterns showed by neglect and dyslexic patients especially in search for left-right asymmetry. There is a vast literature on eye movements in dyslexia and unilateral neglect. Unfortunately few studies are directly comparable.

Prism adaptation

The minineglect hypothesis also promises a very efficient way of ameliorating reading problems, namely, prism adaptation. Indeed, it has been shown recently that wearing binoculars with Frensel prisms which shift the whole visual field to the right can drastically reduce the problems of neglect patients (Rode et al. 2001, Rossetti et al. 1998). It was shown that after a short adaptation prisms improved performance on many typical tasks used in neglect diagnostics, like line bisection (Pisella et al. 2002, Rossetti et al. 1998). Furthermore, Berberovic and coauthors (2004) found that prism adaptation significantly reduces attentional bias in TOJ suggesting that this treatment helps to rebalance the distribution of spatial attention. Some improvements were reported in addition to those connected with distortions of visual space representation (Dijkerman et al. 2003, Girardi et al. 2004).

The durability of dyslexia amelioration after prism adaptation would be a crucial factor in the practical application of such a treatment. Two recent studies showed that the effect of prism adaptation can persist as long as several weeks after treatment (Dijkerman et al. 2004, Frassinetti et al. 2002). Frassinetti and coauthors' (2002) patients were treated with prismatic lenses twice a day over a period of 2 weeks. Improvements revealed by a battery of behavioral and ecological visuospatial tests were maintained during the 5-week period after treatment. This result suggests that the prismatic treatment, provided that it is effective in case of dyslexia, would be rather "painless".

Attentional deficit as an extension of magnocellular dysfunction

The attentional hypothesis (especially minineglect syndrome) seems to be very attractive also because it provides a plausible solution to the long-standing controversy concerning the problem why relatively small deficits in contrast sensitivity and motion perception could be responsible for readings problems (Stein et al. 2000). Indeed, the magnocellular system is sensitive to fast changing stimuli and to relatively low spatial frequencies. These features seem to be rather irrelevant to reading. On the contrary, reading needs intact perception of details (high spatial frequencies) and processing of stationary stimuli (during fixation). Some hypotheses were formulated to explain this apparent contradiction (e.g., Cornelissen and Hansen 1998, Cornelissen et al. 1998, Stein 2001). Stein (2003) emphasized the unstable fixation of dyslexics which leads to the unsteady appearance of letters. Cornelissen and Hansen (1998) speculated that what makes reading difficult is the impaired coding of

letters' positions, as the dorsal system responds to the question "where" (Ungerleider and Mishkin 1982). The plausibility of these accounts is, however, controversial and they are not commonly accepted.

Since it is quite obvious that a dysfunction of the attentional system has to lead to reading problems, the attentional problems found in dyslexics provides a plausible link between their magnocellular deficits and reading problems. Omtzigt and coauthors (2002) directly addressed this question. In their experiment, the subjects had to name a letter flanked by two other letters. If the letters where written with a magnodisadvantageous color contrast, naming accuracy was significantly lower than when the letters were written in parvo- disadvantageous weak luminance contrast. The authors claimed that this finding supports the contribution of the magno system to the allocation of attention and thus focus on the importance of attention in reading difficulties.

Magnocellular deficits as caused by attentional problems

The researchers concerned with attentional deficits in dyslexia usually accept a causal link between magnocellular and attentional deficits (e.g., Facoetti et al. 2000, Hari et al. 2001, Stein 2003). Besides neuroanatomical data, there is some evidence directly supporting this view. Some of it was reviewed above. There is however, some evidence in contrary. Roach and Hogben (2004) recently compared performance of dyslexics on a visual search task with performance on some tasks usually used to check functioning of the magnocellular system. While all (n=5) dyslexics showed clear problems with visual search task, the "magnocellular" tasks were performed perfectly. This result can be viewed as evidence supporting an opposite causal link between magnocellular and attentional deficits. In other words, magnocellular deficits are due to the problems dyslectics have with attention concentration during task performance. It was shown that the results obtained by dyslexics in psychophysical tasks could be easily simulated by assuming some random responses caused by a number of subtle non-sensory task difficulties like lapses of concentration (Davis et al. 2001, Peli and Garcia-Pérez 1997, Roach et al. 2004, Stuart et al. 2001). Roach and coauthors (2004) proposed some tests which can help to distinguish between sensory and non-sensory contributions to poor performance by dyslexics. For example, given magnocellular deficits, there should be strict correlation among dyslexics between performances on different magnocellular tests. In other words, a dyslexic who performs poor on one test should perform also poor on another test. Roach and coauthors (2004) noted that "while multitask studies are becoming increasingly common, sufficient information to allow this comparison is seldom provided" (p. 825).

EYE MOVEMENTS AND DYSLEXIA

Saccadic and smooth pursuit eye movements

Eye movements and attention are closely related. The shift of attention from one object to another usually precedes a saccade, i.e., a fast jump of the gaze aiming to foveate the new object of interest. Both an attention shift and the following saccade are parts of the orienting response.

Besides saccades there are three other types of eye movements – smooth pursuit, the vestibular ocular reflex and convergence. Saccades and smooth pursuit are triggered and controlled by cortical structures: the posterior part of frontal lobe and PPC (Glimcher 1999, Pierrot-Deseilligny et al. 1997). Particularly important to our considerations are parietal regions controlling eye movements. So-called parietal eye field (PEF) is located in the intraparietal sulcus. Two other nearby structures involved in eye-movement control are located in TPJ, at the border between the temporal and parietal lobes. These are the middle temporal area (area MT) and the medial superior temporal area (MST) (Pierrot-Deseilligny et al. 1997). Lesion of PEF leads to elongation of the latency and reduction of accuracy of reflexive saccades (i.e., saccades which are triggered by a visual target suddenly appearing in the visual field) (Pierrot-Deseilligny et al. 1991a,b).

Bearing in mind the involvement of area MT in motion perception, its role in eye movements is of special interest for us. Lesions of MT affect the initial part of smooth pursuit (smoothly tracking a slowly moving object in the visual field) when the target is moving in the visual field contralateral to the lesion. This deficit is rather difficult to detect. It is much easier to observe disturbances of eye movements occurring after impairment of MST. After such impairment, pursuit velocity becomes too low and the eye movement becomes saccadic, i.e., some saccades are superimposed on smooth tracking movements (Pierrot-Deseilligny et al. 1997).

Eye movements in dyslexia

Even though the problem of eye movements in dyslexia attracts a lot of scientific attention, their contribution to reading problems remains unclear (Adler-Grinber and Stark 1978, Biscaldi et al. 1998, Black et al. 1984a, Brown et al. 1983, Crawford and Higham 2001, De Luca et al. 1999, 2002, Fischer and Weber 1990, Fischer et al. 1993, Hutzler and Wimmer 2004, Hyönä and Olson 1994, Mackeben et al. 2004, Martos and Vila 1990, Olson et al. 1983, Pavlidis 1981, Rayner 1998, Stanley et al. 1983). On the one hand, there are studies showing a quite normal pattern of eye movements in different linguistic and non-linguistic tasks. On the other hand, a remarkable number of studies have been published demonstrating significant differences in eye-movement control in dyslectics and normal readers.

We will restrict our review only to those studies in which saccades and smooth pursuits were investigated in non-linguistic tasks. In several early studies no differences between dyslexics and normal readers were found in a simple saccadic task (re-fixation) (Black et al. 1984a,c). However, more recent studies show different patterns of results for both groups (Biscaldi et al. 1998, 2000, Fischer and Weber 1990, Fischer et al. 1993). For example, Biscaldi and coauthors (1998) measured saccadic eye movements in a single target (re-fixation) and in a sequential-target task (target jumped from one position to another). Their results indicated that dyslexics, relative to normal readers, had much more scattered saccadic reaction times, i.e., many express saccades (i.e., saccades with latencies <135 ms) and late saccades. The authors suggest that the attentional problems of the people suffering from dyslexia are responsible for their worse saccadic control. In particular, they assume that deficits in selective attention might result in deficits in fixation disengagements, and consequently in increased generation of late saccade and irregular saccade triggering.

There are few studies devoted to smooth pursuit in dyslexics. Black and coauthors (1984b) reported an increased number of saccades superimposed on smooth pursuit movements in dyslexics relative to normal readers. In a more recent study, Eden and coauthors (1994) found poor smooth pursuit in the dyslexic group, particularly when pursuing a target moving from left to right. Both these deficits might be interpreted as symptoms of mild MST/MT dysfunctions. Eden and coauthors (1994) proposed a slightly different account of their results, suggesting that eye-movement abnormalities might be due to the insufficient inhibition of parvocellular activity from magnocellular activity. Both these accounts are consistent with the parietal deficit hypothesis.

It should be also noted that left-right asymmetry reported by Eden and coauthors. (1994) fits very well with the minineglect hypothesis. Indeed, similar asymmetry in pursuit eye movement was found in neglect patients (Thurston et al. 1988).

VISUO-MOTOR CONTROL OF LIMB MOVEMENTS

The parietal hypothesis suggests some further possible dysfunctions in dyslexics which, to our knowledge, have never been tested.

Neurological patients with damage to the dorsal system (optic ataxia) often show impaired grasping movements although their perception seems to be intact (Milner et al. 2001). They are also unable to reach in the correct direction to objects located on the contralesional side. In contrast, patients with form agnosia are able to reach and to prepare their grip according to shape of an object to be picked up but their awareness of the goal object seems to be absent. Hence one can speak about two different kinds of vision: vision-for-perception and vision-for-action (Goodale et al. 2004). Vision-for-action is thought to extract from the visual stream the information which is necessary to immediate use in fast motor actions and to rely on computations made mainly in the dorsal system. Therefore, one would predict that certain dysfunctions of parietal lobe can lead to poorer performance on tasks requiring visuomotor control. For example, in a recent study devoted to vision-for-action, Schindler and coauthors (2004) asked their subjects to reach between two vertical cylinders whose location varied from trial to trial. They found that normal subjects' movement trajectories ran through the midpoint of the line connecting the cylinders as if they tried to avoid the obstacles (i.e., cylinders). A patient with optic ataxia, however, apparently ignored the obstacles, reaching every time along the same path independently of the cylinders' positions. The parietal hypothesis of dyslexia predicts that dyslexics would perform this task similarly to patients with ataxia.

MENTAL ROTATION

Correct side discrimination is important for daily navigation in the real world as well as for learning to

read and write. Side discrimination helps to recognize and discriminate letters, especially those of similar shapes but of different meaning (p – d – b). The letter "d" rotated 180° becomes the letter "p". The letter "d" can also be treated as a mirror-image of the letter "b". It is a well known fact that one of the most famous and most often described symptoms of dyslexia are reversal errors, that is, dyslexics tend to confuse letters having the same shapes but different spatial orientation (e.g., p/q; b/d; p/d) (Willows and Terepocki 1993).

This is why we will focus on mental rotation, a task which requires visuo-spatial processing of images. In this task, participants are usually asked to decide whether or not two items, presented either simultaneously or in succession, are same or different regardless of their orientation. The first study on mental rotation was published by Shepard and Metzler (1971). They showed that response time (RT) increased linearly with angle of rotation of visually presented 3-dimensional objects. Shepard and Metzler (1971) suggested that the image of the object was rotated in the subjects' minds just as if they would have physically manipulated the object. Their result has been replicated many times with different types of stimuli and in different tasks (e.g., Cohen et al. 1996, Harris et al. 2000, Jordan et al. 2001, Kosslyn et al. 1998)

The neuronal mechanisms underlying mental rotation are still under investigation. Most neuroimaging, electrophysiological and trans-cranial magnetic stimulation (TMS) studies indicate that the parietal lobes play a major role in mental rotation (Alivisatos and Petrides 1997, Bestmann et al. 2002, Cohen et al. 1996, Harris et al. 2000, Jordan et al. 2001, Kosslyn et al. 1998, Milivojevic et al. 2003, Podzebenko et al. 2002, Richter et al. 1997, 2000, Windischberger et al. 2003, Yoshino et al. 2000). What remains unclear is which part or neural networks of parietal cortex are involved in mental rotation processing, and which hemisphere plays a role in the transformational operations performed on images of objects. Most authors (Bestmann et al. 2002, Cohen et al. 1996, Jordan et al. 2001, Kosslyn et al. 1998, Richter et al. 1997, 2000, Windischberger et al. 2003) have reported bilateral activation of the parietal lobe during the manipulation of object representations in the mind. Others found an increase of activation only in the right intraparietal sulcus (Harris et al. 2000) or bilateral with right dominance (Podzebenko et al. 2002). However, some authors have claimed that exact neural circuit activated during mental rotation depends on task difficulty (Milivojevic et al. 2003) or on the category of objects to be rotated (Kosslyn et al. 1998). This last claim is consistent with results by Alivisatos and Petrides (1997) who reported activation of the left inferior area of the parietal lobe when using alphanumeric symbols (however, see Jordan et al. 2001).

Mental rotation and dyslexia

Although mental rotation seems to be a "parietal" task, relatively little is known about mental rotation in dyslexics.

Corballis and coauthors (1985b) found no group differences in a task in which participants had to name disoriented letters (mental rotation was not explicitly required). In another study (Corballis et al. 1985a), the subjects were asked to discriminate between letters b and d presented in varying angular orientations. Again, no between-group differences were found.

More recently, Brendler and Lachmann (2001) showed that children with specific difficulties in reading and writing could correctly differentiate shapes of stimuli whereas differentiation of objects of the same form but of different orientation caused them some trouble. The results of their experiment also revealed that dyslexics had problems with processing spatial information for letters exclusively.

We recently tested how well people suffering from dyslexia perform on Shepard and Metzler's tasks (Rusiak, Lachmann, Jaśkowski, van Leeuwen, submitted). The stimuli were letters and their exact mirror-images. The participants were instructed to decide whether the displayed figure was a normal or mirrored letter regardless of its orientation. Dyslexics were found to be remarkably slower than normal readers (1 085 ms vs. 770 ms) but their response times increased with angle of rotation identically in both groups. This indicates that dyslexics performed the mental rotation task itself as efficiently as normal readers and that their slowness was due to elongation of other processes involved. We replicated this experiment with both letters and non-letter figures. Significant differences between normal and disabled readers were obtained exclusively for letters. These results show that dyslexics have problems with processing special (overlearned) characters, especially if they have to extract information from long-term memory. They performed, however, on the mental rotation test as good as normal readers. This finding seems to provide no support for the parietal hypothesis. Rather it

is consistent with Lachmann's (2002) recent hypothesis concerning the cause of dyslexia. He proposed that letter-reversal errors (e.g., confusing p/d or b/d) are due to a failure in suppressing so-called symmetry generalization in dyslexics. Efficient recognition of the objects around us relies on shape generalization, i.e., ability to recognize objects as same if they have the same shape but different orientation. This generalization, however, would disturb reading as some different letters have the same shape but different orientation (e.g., p, d, b). Therefore, to read properly one has to suppress this symmetry generalization in case of letters. Lachmann (2002) hypothesized that in dyslexics this suppression does not develop properly.

POSTURAL INSTABILITIES IN DYSLEXIA

Cerebellar deficits in dyslexia

Nicolson and coauthors (2001) presented a new theory which assumes that dyslexia is caused by some cerebellar deficits. Traditionally the cerebellum was considered as responsible for the control of coordinated movements. However, more recent studies have revealed its role in cognitive processes (Molinari et al. 1997). Cerebellar dysfunctions turn out to seriously impair linguistic processing, abstract thinking, and acquisition and automatization of new cognitive procedures (Ito 1984, 1990, Krupa et al. 1993). Such a wide range of cerebellar functions results most likely from many reciprocal projections between the cerebellum and different structures of the brain, for example, to Broca's area in the frontal lobe or to the parietal lobe (Ackerman and Cianciolo 2000, Fabbro 2001, Silveri et al. 2000)

Nicolson and Fawcett demonstrated some structural and functional deficits in the cerebellum in dyslexics. Relative to normal readers, dyslexics were shown to have problems with postural stability especially if they had to perform an additional cognitive task like counting or pressing a button (Fawcett and Nicolson 1999, Fawcett et al. 1996). Moreover, dyslexics suffer problems with automatization of some skills (Nicolson and Fawcett 1990, Overy et al. 2003), time estimation (Nicolson et al. 1995), speeded performance (Nicolson and Fawcett 1994) and eye blink conditioning (Nicolson et al. 2002). All these disabilities might be considered to be symptoms of cerebellar dysfunction. Moreover, a neuroimaging study revealed that dyslectics showed anatomical anomalies within the cerebellum (Finch et al. 2002). Rae and others (Ito 1984, 1990, Krupa et al. 1993, Rae et al. 2002) found atypical symmetry of both hemispheres of the cerebellum in individuals with specific difficulties in reading and writing. In normals there is a structural asymmetry of the cerebellum, the right cerebellar hemisphere being larger than the left. They claimed that left-side cortical dominance corresponds to right-side superiority of cerebellum in normal readers.

Cerebellum vs. magnocellular theory

An important question is how these cerebellar dysfunctions relate to the "parietal" deficits? Are there two different types of dyslexia – parietal and cerebellar? Or are the cerebellar and parietal deficits two sides of the same coin, i.e., different manifestations of the same general failure?

Nicolson and coauthors (2001) argued that the cerebellar deficit theory can account for the full range of difficulties established for dyslexics and that it provides a causal explanation for this disorder. The hypothetical causal chain starts with cerebellar problems. These in turn lead to impaired acquisition of articulatory skills and further to phonological problems. This causal chain unfortunately does not provide an account for early sensory (magnocellular) deficits. They write that "cerebellar deficit is an alternative, or perhaps parallel, mechanism to magnocellular abnormality" (Nicolson et al. 2001, p. 510). Stein (2001) suggested rather that cerebellar deficits in dyslexics are due to the fact that the cerebellum is a very important element of the whole cerebral magnocellular system. Another possibility is that magnocellular deficits are simply artefacts as proposed by some authors (see above).

Some of the problems of dyslexics found by Nicolson and Fawcett might appear to be consistent with the parietal hypothesis. As mentioned, they showed postural instabilities which were particularly severe if subjects had to perform an additional cognitive task. Bearing in mind, however, attentional deficits demonstrated by dyslexics, one can wonder if their balance instability is not due to inability to switch attention quickly between two different tasks, like stance stabilization and a cognitive task. Indeed, some data suggest that the inability to allocate sufficient attention to postural control under multitask conditions may be a contributing factor to imbalance (Marsh and Geel 2000, Redfern et al. 2001,

Shumway-Cook and Woollacott 2000, Teasdale and Simoneau 2001).

There is insufficient space here to further discuss the possible relations between these two biological theories of dyslexia. Certainly, more research is needed. Let us mention three important points concerning the relationships between the theories.

(1) Little is known about co-occurrence of cerebellar and parietal symptoms. Are these symptoms independent of each other or do they usually occur simultaneously? Co-occurrence of the leading symptoms of parietal deficits (orienting of attention) with the leading symptoms of the cerebellar deficits would support Stein's position, i.e., that there is one biological factor (for example the magnocellular system) which affects both PPC and the cerebellum as well as (perhaps) other brain structures. Independence of these symptoms would suggest that different biological factors can lead to similar macrolevel symptoms like phonological awareness and problems with reading and writing.

(2) The question, what is meant by "leading symptom" is as important as it is difficult. This is because some of the non-linguistic symptoms of dyslexia might be explained by both theories. For example, both PPC and the cerebellum contribute to pursuit eye movement control (see reviews Krauzlis 2004, Pierrot-Deseilligny et al. 1997). Therefore, such a deficit can be interpreted as evidence in favor of both theories, especially since symptoms of dyslexia are generally very mild. By the same token, problems with postural stabilization might be treated as a leading cerebellar symptom. However, bearing in mind that the postural problems are particularly outstanding when undertaking a second task, skeptics might argue that balance deficits are caused by poor control of attention as argued above. Even structural and metabolic abnormalities in a structure cannot be taken for granted as the evidence for a causal role of this structure in dyslexia (Nicolson et al. 2001), as small anatomical changes have been found in many other cerebral structures (Zeffiro and Eden 2000, 2001)

(3)When reading the exposition of a theory, one often gains impression that its authors try to gather as many confirmations as possible, leaving out skeptics and commentators who have defined weaknesses and shortcomings. This leads to a situation when some theories evolve without (or with very weak) interactions with concurrent theories. Such a situation seems to take place in dyslexia research. Certainly, more "across the board" research is needed to pin down the problem.

CONCLUSIONS

We reviewed some arguments in favor the parietal deficits as a cause of developmental dyslexia. Dyslexics were shown to perform worse on tasks which are thought to be mediated by the posterior parietal cortex. For example, they have problems with attention focusing, pursuit and saccadic eye movements and show some symptoms similar to those shown by people suffering from unilateral neglect. A plausible reason for these parietal deficits is impairment of the magnocellular system. Although this hypothesis looks very promising, we pointed out that several questions remain which should be addressed in future research. These questions concern other tasks mediated by the parietal cortex, like mental rotation or visuo-motor guidance and the relations to other theories of dyslexia, especially to the cerebellar deficit theory.

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