Left minineglect in dyslexic adults

Riitta Hari, Hanna Renvall and Topi Tanskanen

Brain Research Unit, Low Temperature Laboratory, Helsinki University of Technology, Espoo, Finland

Summary

We searched for a core mechanism underlying the diverse behavioural and sensorimotor deficits in dyslexic subjects. In psychophysical temporal order judgement and line motion illusion tasks, adult dyslexics processed stimuli in the left visual hemifield significantly (~15 ms) more slowly than normal readers, indicating a left-sided 'minineglect'. Furthermore, abrupt stimuli captured attention in both Correspondence to: Riitta Hari, Brain Research Unit, Low Temperature Laboratory, Helsinki University of Technology, PO Box 2200, FIN-02015 HUT, Espoo, Finland E-mail: hari@neuro.hut.fi

visual hemifields less effectively in dyslexics than in normal readers. These abnormalities could reflect right parietal lobe hypofunction, a consequence of a general magnocellular deficit demonstrated previously. Based on these and previous data, we propose a causal chain which could result in several sensory and cognitive deficits observed in dyslexic subjects.

Keywords: dyslexia; neglect; visual processing; parietal lobe; magnocellular system

Introduction

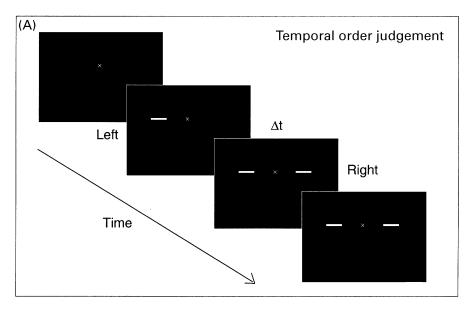
Dyslexia is a common neurocognitive disorder running in families: ~4-10% of the population have great difficulties in learning to read despite adequate training, opportunity and intelligence. Somewhat surprisingly, the problems of dyslexics extend beyond the skills directly needed for reading. For example, many dyslexic adults, as well as languagelearning impaired children (Tallal et al., 1998), are slow in processing rapid sound sequences (Hari and Kiesilä, 1996; Helenius et al., 1999); here the time scales of interest are of the order of hundreds of milliseconds. Dyslexics also display various visual abnormalities (Stein and Walsh, 1997; Demb et al., 1998) and may have trouble in balance and motor control (Nicolson et al., 1995) as well as in processing rapid sequences of tactile stimuli (Laasonen et al., 2000). Because the cognitive phenotype varies so widely across subjects, one is tempted to search for a core mechanism uniting this diversity of disorders. One such proposal is the magnocellular deficit hypothesis (Lovegrove et al., 1980; Galaburda and Livingstone, 1993; Stein and Walsh, 1997), suggesting that the basic disorder is a neurodevelopmental abnormality of the magnocellular system.

The magnocellular pathways, studied most extensively in vision, consist of large and fast-conducting neurons that respond effectively to stimulus transients. Although the magnocellular hypothesis is still under debate (Skottun, 2000; Stein *et al.*, 2000), several behavioural, histological and electrophysiological studies (Lovegrove *et al.*, 1980; Livingstone *et al.*, 1991; Galaburda and Livingstone, 1993; Stein and Walsh, 1997; Demb *et al.*, 1998; Iles *et al.*, 2000) have demonstrated deficits in the magnocellular pathways of dyslexic subjects.

Activation of the magnocellular system seems important for efficient capturing of automatic attention (Steinman *et al.*, 1997). Accordingly, dyslexic subjects, supposed to have a deficient magnocellular system, often suffer from minor attentional problems (Klein and Farmer, 1995; Asbjornsen and Bryden, 1998; Casco *et al.*, 1998; Facoetti *et al.*, 2000). We recently showed that the dwell time of visual attention is 30% longer in dyslexic adults than in normal-reading control subjects (Hari *et al.*, 1999b). The 'attentional blink' task used in our studies was based on the finding that the subject, after identifying a target, is 'blind' to other targets within the next 400–600 ms because the previous target ties up the attentional resources (Duncan *et al.*, 1994).

In a similar task, patients with left visuospatial neglect after right hemisphere lesions can have up to four times longer attentional blink than healthy controls (Husain *et al.*, 1997). Given the qualitative similarities between neglect patients and dyslexics in this respect, we wondered whether dyslexic subjects would also resemble neglect patients in other aspects, and whether such similarities would illuminate neuronal mechanisms underlying dyslexia.

We therefore tested adult dyslexics in a temporal order judgement task (Fig. 1A) in which neglect patients demonstrate prominent slowing of processing in the left visual hemifield (Robertson *et al.*, 1998). We also applied a line motion illusion task (Fig. 1B) (Hikosaka *et al.*, 1993) to quantify the strength of automatic attention capture by visual cues (Steinman *et al.*, 1997). Our results demonstrate that dyslexic adults suffer from a left-sided 'minineglect'. A preliminary report of this study has been presented in abstract form by Hari and Koivikko (Hari and Koivikko, 1999).



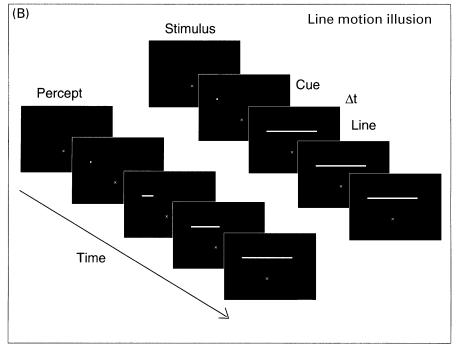


Fig. 1 (A) Set up for the temporal order judgement task. (B) Set up for the line motion illusion task. The stimuli are shown on the right and the subject's typical percept on the left.

Methods

Subjects

We studied nine dyslexic adults [age 32.0 ± 1.8 (mean \pm standard error of the mean) years; six females, three males; all right-handed] and 14 control subjects (31.4 ± 2.0 years; six females, eight males; one left-handed and one ambidextrous). All subjects gave informed consent. The dyslexics had a diagnosis of developmental dyslexia (eight since school age) and seven of them had participated in special tutoring during education. Subjects with any other anamnestic or observed neurological deficits were excluded.

At the time of our tests, the dyslexics were significantly slower than the control subjects in reading (100.6 ± 6.4 versus 159.1 ± 4.6 words/min; P < 0.001; simple Finnish story) and in word recognition (832 ± 60 versus 544 ± 21 ms; P < 0.002). In the latter task, the subject had to decide, as quickly as possible, whether a word presented on a computer screen was a real Finnish word or an orthographically legal pseudoword; the correctly recognized words were used for calculating the word recognition speed. The highest level of education completed by the dyslexics was 12.9 ± 0.9 years; all except the youngest one had finished professional education successfully, two had a university degree and two were studying for an academic-level professional degree. Most control subjects were members of the laboratory personnel; nine of them had a university degree and two had finished other professional education.

Stimuli and tasks

All subjects participated in two psychophysical experiments: a temporal order judgement task (Robertson et al., 1998) and a line motion illusion task (Hikosaka et al., 1993; Steinman et al., 1998). They viewed a screen controlled by a Macintosh Quadra 840AV computer from an ~65 cm distance. The stimuli were grey (7.9 cd/m²) on a black background (2.0 cd/m²). The main stimulus parameters were chosen on the basis of the original description of the tasks and adapted for local viewing conditions; the low luminance of the stimuli was selected to increase the proportion of magnocellular versus parvocellular visual processing (Purpura et al., 1988; Steinman et al., 1997; Demb et al., 1998). A two-alternative forced choice method was applied in both tasks, with no time pressure for the responses. The experiments were preceded by a short training period of five to seven trials on each task to ensure that the subject had understood the instructions fully.

In the temporal order judgement task, the subjects decided whether a visual bar in the left hemifield preceded or followed a similar bar on the right (Fig. 1A). The central fixation cross 'X' was on all the time, and horizontal bars, 1.4° in width and 0.1° in height, appeared at symmetrical locations in the left and right visual fields and at the same height as the fixation cross. The outer edges of the bars were 4.2° from the fixation cross. Randomly, either the left or the right bar appeared first, followed by the other bar after a delay that varied randomly from 0 to 210 ms in 15 ms steps. Both bars were removed 450 ms after the appearance of the second bar. The subjects indicated verbally, in a two-alternative forced choice manner, whether the left or the right bar had appeared first. The new trial was initiated 1.8 s after the experimenter's key press which coded to the computer the subject's vocal response.

The line motion illusion task (Hikosaka *et al.*, 1993) (Fig. 1B) was selected to obtain quantitative information on the strength and speed of automatic attention capture. In this task, the subject perceives a line growing from a site where a cue stimulus has been presented slightly earlier. The illusion is interpreted to reflect faster processing of stimuli falling into the attended locations (Hikosaka *et al.*, 1993).

The subject fixated on an 'X' situated 4.4° beneath the stimulus level. The cue (a $0.2 \times 0.2^{\circ}$ box) appeared to either the left or right visual field at an eccentricity of 3.7° , and was followed after a random 0-210 ms interval (in 15 ms steps) by a line which connected the two possible cue locations. However, the subjects perceived illusorily that the line 'grew' from the cue site towards the other end of the line. The new trials were initiated from the experimenter's

key press as in the above experiment. The subjects indicated verbally, in a two-alternative forced choice manner, whether the line had appeared to move from left to right or from right to left.

In both tasks, each subject viewed 250 stimulus presentations; 40% of the stimuli were scattered randomly across the whole interstimulus interval and 60% concentrated, again randomly, in the middle half of the interval to increase the number of data points and response reliability at the most important cue–target delays; the presentation order and cue–target delays were randomized online before each stimulus presentation.

Data processing and statistical analysis

The individual frequencies of 'left first'/'right first' (temporal order task) and 'from left'/'from right' (line motion task) responses were converted to probabilities of 'right first' (or 'from right') responses at each delay. A cumulative normal distribution was then fitted to the data of each subject using the least-squares criterion. The subject's bias to left- or right-sided responses is reflected as a deviation of the response distribution's mean from zero. The widths of the distributions were quantified by calculating the difference between the 75% and 25% points of the cumulative normal distributions; large values would indicate increased 'simultaneity windows' within which temporal order judgements are vague. An increase in the simultaneity window would also serve as an indicator of sluggishness of temporal processing. Two-tailed *t* tests were used in statistical comparison of the results.

Results

Figure 2A illustrates the results of the temporal order judgement task, and Fig. 3A shows the corresponding mean values of the response distributions across the subjects. The control subjects performed symmetrically for both stimulus orders (the mean of the distribution did not differ from zero). The 'simultaneity window', derived from the 75-25% width of the distribution, was 64 ms (i.e. from -26 to +38 ms). In dyslexics, the corresponding window was 92 ms (from -62 to +30 ms), which was statistically significantly (P < 0.04) prolonged compared with the controls, indicating increased sluggishness of temporal processing. In dyslexics, the response distributions centred towards the left from zero (P < 0.05), indicating preference for the right visual field. For dyslexics, the left-sided stimuli had to precede the right-sided ones on average by 16 ms to be perceived as simultaneous. The centres of the distributions clearly differed between the subject groups (P = 0.015).

Figures 2B and 3B illustrate the corresponding results for the line motion illusion task. The simultaneity window was 48 ms (from -22 to +26 ms) in controls and 56 ms (from -43 to +13 ms) in dyslexics. The normal-reading subjects showed a symmetric behaviour, and the mean of the response distributions did not differ from zero. In the dyslexics, the

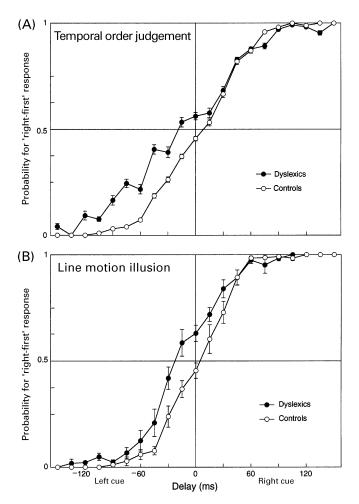


Fig. 2 (A) The mean (\pm standard error of the mean) responses of nine dyslexic and 14 normal-reading subjects in the temporal order judgement task of Fig. 1. The perceptual judgements, given as the probability of answering 'right first', presented as a function of the time delay between the left- and right-sided bars; the 0.5 level of the perceptual judgement axis refers to equal numbers of 'left first' and 'right first' responses. The negative and positive time delays refer to left- and right-sided stimulus precedence, respectively. (B) The corresponding data from nine dyslexic and 14 normal reading subjects in the line motion illusion task.

distribution was again centred significantly on the negative delays (P < 0.007), indicating preference for right-sided cues; the distribution centres differed statistically significantly between the two subject groups (P = 0.003). The dyslexics needed on average a 15 ms longer cue–target delay in the left than in the right visual field to perceive the illusion in a similar way. Although the mean widths of the response distribution were slightly larger in dyslexics than in controls, this difference did not reach statistical significance.

Figure 4 illustrates the response preferences in the two subject groups at different delay times. The traces illustrate the group means for differences between 'right' versus 'left' response probabilities as a function of the delay time; if there were no response bias, the curves should run along the zero line as they do for the control subjects. Dyslexics show a

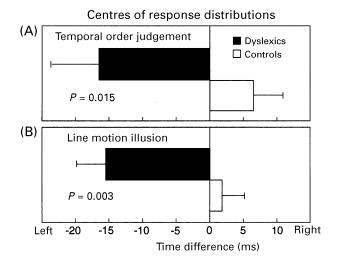


Fig. 3 The mean (\pm standard error of the mean) values of response distributions, determined by fitting a cumulative normal distribution to the individual distributions. The *P* values refer to differences between the two groups of subjects.

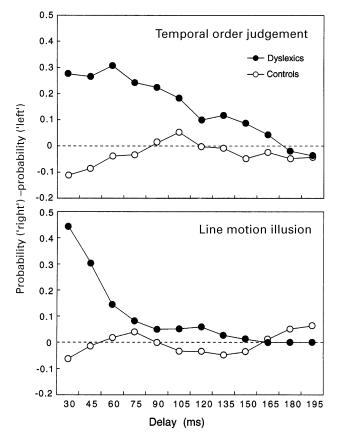


Fig. 4 The mean values across the two subject groups for the probability differences for responding 'right first' minus 'left first', and the corresponding responses to the line motion. The values are sliding averages of three time delays.

clear preference in both tasks for right-sided responses at short delays.

Discussion

Dyslexic and normal readers differed consistently both in judging the temporal order of visual stimuli presented to the two hemifields and in perceiving a line growing from left to right, or vice versa, in the line motion illusion task. The present experiments did not include any reaction time measurements and thus possible motor slowness did not contribute to the results.

Most interestingly, dyslexics showed a statistically significant right visual field advantage in both tasks, indicating that their temporal processing is asymmetric and impaired in the left relative to the right hemifield. This result supports our hypothesis of a left-sided 'minineglect' (Hari and Koivikko, 1999), i.e. a right-sided spatial bias in selecting and processing visual information. Moreover, the wider response distributions of the dyslexics (statistically significant only for the temporal order judgement task) suggest sluggish attention capture associated with slowed temporal processing in both visual hemifields.

In previous literature, left-sided deficits of visual processing have been observed in some dyslexic subjects (Stein and Walsh, 1997). For example, children with reading difficulties and unstable control of vergent eye movements made more errors in locating targets in the left than in the right visual hemifield (Stein *et al.*, 1989; Riddell *et al.*, 1990). Recent support for our minineglect hypothesis comes from dyslexic children who displayed left inattention in a visual flanker task (Facoetti and Turatto, 2000) as well as in a reaction time task (Facoetti and Molteni, 2001), associated with impaired suppression of distractor information in the right visual field.

We would like to emphasize the mildness of the observed minineglect, so that its direct consequences on the subject's reading and other performance may be minor. However, the existence of the minineglect is in line with the magnocellular deficit hypothesis, and it supports our view that deficits in processing rapidly presented stimulus sequences (Hari and Kiesilä, 1996; Helenius *et al.*, 1999) could be caused by sluggish attentional capture and shifting, as discussed in more detail below.

Dyslexia, sensory processing and the magnocellular deficit hypothesis

Dyslexia is often considered primarily a language disorder, resulting from a failure to acquire adequate phonological skills that are needed when written letters are translated into sounds and words. However, dyslexic subjects suffer from a multitude of minor symptoms that derive from diverse neural systems and occur in variable patterns across individuals. Dyslexia's clinical signs vary widely, including problems in spelling, writing, verbal memory, sensory processing and phonological awareness (Eden and Zeffiro, 1998). Any proper theory of dyslexia should try to account for these deficits, as well as for the poor reading performance. The magnocellular deficit hypothesis of dyslexia, providing one attempt at such a unifying mechanism, has explained visual abnormalities successfully (Stein and Walsh, 1997) and could also account for sensory deficits in other modalities because magnocellular divisions appear to exist in the auditory, somatosensory and motor systems (Livingstone *et al.*, 1991; Galaburda *et al.*, 1994). However, it has remained unclear how magnocellular deficits could cause reading disorders.

We propose that the causal link from the magnocellular deficit to reading and phonological problems involves regulation of automatic attention. Dyslexics have various sensory problems, many of which seem to be related to impaired processing of stimuli that are presented in rapid succession (Tallal and Piercy, 1973; Hari and Kiesilä, 1996; Stein and Walsh, 1997; Tallal et al., 1998; Helenius et al., 1999; Laasonen et al., 2000). Such problems, on a time scale of up to half a second, are unlikely to be accounted for by deficient phase locking of neuronal firing (Hari et al., 1999a), or by prolonged conduction times. The primary cause rather could be sluggish attention shifting, as suggested by our previous finding that dyslexics are slower than normal readers in disengaging their attention from the previous target (Hari et al., 1999b). Such a control of automatic attention is attributed commonly to the posterior parietal lobe (Posner and Raichle, 1994).

Parietal lobe and attention

The left-sided minineglect, i.e. a spatially asymmetric distribution of visual attention, seems to be a mild version of the left visuospatial neglect after right hemisphere lesion (Husain et al., 1997). Thus it would be logical to assume a minor right parietal lobe dysfunction in dyslexia. The magnocellular input is important for parietal lobe function (Merigan and Maunsell, 1993) and, due to the general magnocellular deficit, the dorsal visual stream of dyslexic subjects could receive weekend magnocellular input as was previously shown for the MT/V5 part of the dorsal pathway (Eden et al., 1996). It is clinically well known that lesions of the right parietal lobe often result in contralesional neglect, whereas corresponding disorders are rare after left parietal lobe lesions. Thus one may envisage that a diffuse functional disruption of the magnocellular pathways would first be seen as a right visual field advantage. Thus a left-sided minineglect could emerge as a result of decreased magnocellular input to the parietal cortex.

The importance of the right parietal lobe dysfunction for the deficits encountered in dyslexia is also emphasized by a recent functional MRI study in which the right intraparietal sulcus was activated consistently in an attentional blink task (Marois *et al.*, 2000), similar to that in which dyslexics have shown prolonged attentional dwell time (Hari *et al.*, 1999b). The right intraparietal sulcus thus seems important for capacity-limited attentional processing of visual information (Marois *et al.*, 2000).

Hypoarousal of the right parietal lobe has been suggested recently to underlie attentional deficit hyperactivity disorder: untreated children showed right-biased line bisection (suggesting a left neglect) but their behaviour was normalized by application of a stimulant drug; the reason for the improvement was assumed to be correction of the right hemisphere hypoarousal (Sheppard *et al.*, 1999). Interestingly, the spatial imbalance in the time course of visual awareness in neglect patients, observed in the temporal order judgement task, can be corrected transiently by a warning sound presented just before the visual stimuli; thus phasic alerting via subcortical projections can affect the speed of perceptual processing even when the right parietal function is disrupted to a certain extent (Robertson *et al.*, 1998).

Parietal lobe and reading

Written language has existed for a relatively short time, and it is thus not reasonable to assume innate brain mechanisms for reading; therefore, learning-related effects play a crucial role. The magnocellular visual deficits of dyslexic subjects typically are mild and cannot directly explain the reading and language problems. For example, the contrast sensitivity problems appear at low-luminance conditions that are not encountered during normal reading situations (Cornelissen *et al.*, 1995). Thus the link between the cellular level deficit and reading disorders has to be sought elsewhere.

The parietal lobe supports several functions that are important for reading. For example, covert attention and saccade control involve activation of common areas in the parietal, frontal and temporal lobes (Corbetta *et al.*, 1998), and these two functions are closely interrelated: one first has to shift attention to the target location before a saccade can be made towards it (McPeek *et al.*, 1999). Learning to read involves training of rapid attentional shifts, associated with eye movements, along the sequential letters and words along a line (Vidyasagar, 1999), in this process the integrity of the parietal lobe seems essential.

In line with the parietal lobe's importance for readingrelated skills, lesions of the posterior parietal lobe may produce 'acquired dyslexia' (Brunn and Farah, 1991). Moreover, right hemisphere injection of sodium amobarbital (Schwartz *et al.*, 1997) results in reading errors resembling those in dyslexia. Thus data from robust right parietal dysfunctions are concordant with the present suggestion of 'minineglect', a mild form of such a dysfunction in dyslexic subjects.

How one may become dyslexic

We now summarize our hypothetical causal chain from magnocellular deficit to various disorders in dyslexia.

Due to genetic predisposition (Gilger et al., 1991), some subjects may be more vulnerable to neurodevelopmental disorders, such as immunological attacks affecting the magnocellular neurones, which share a common surface antigen (Galaburda and Livingstone, 1993; Stein and Richardson, 1999). The resulting magnocellular deficit would have inevitable consequences in several neural systems, and could then both directly impair purely sensory functions, such as those underlying contrast sensitivity and identification of formant transitions, and, importantly for our hypothesis, also impair processing of stimulus sequences as a result of sluggish attention shifting (Hari et al., 1999b). The minor hypofunction of the right parietal lobe, for which we have now provided indirect quantitative evidence in a group of adult dyslexics, is in line with this reasoning; the hypofunction can be related to sluggishness of attentional capture and shifting, as well as to modified spatial distribution of attention (Merzenich et al., 1993; Facoetti et al., 2000; Facoetti and Molteni, 2001).

The problems of temporal processing, manifested at time scales up to a few hundred milliseconds, could themselves play an important role in the genesis of the reading disorder, for example by preventing the genesis of stable and invariant phonetic representations, thereby affecting phonological awareness that is crucial for development of reading disabilities (Merzenich *et al.*, 1996; Tallal *et al.*, 1998; Ahissar *et al.*, 2000).

Within this framework, the pattern of the subjects' functional deficits would be determined by the neural systems in which the magnocellular deficits are the most prominent. The subject's vulnerability could depend both on genetic factors and on the time of exposure to external noxious agents. Thereby, clear subgroups of dyslexic subjects would emerge.

Acknowledgements

We thank K. Uutela for advice in constructing the stimuli and P. Helenius for providing the reading tasks. This work was supported by the Academy of Finland.

References

Ahissar M, Protopapas A, Reid M, Merzenich MM. Auditory processing parallels reading abilities in adults. Proc Natl Acad Sci USA 2000; 97: 6832–7.

Asbjornsen AE, Bryden MP. Auditory attentional shifts in readingdisabled students: quantification of attentional effectiveness by the Attentional Shift Index. Neuropsychologia 1998; 36: 143–8.

Brunn JL, Farah MJ. The relation between spatial attention and reading: evidence from the neglect syndrome. Cogn Neuropsychol 1991; 8: 59–75.

Casco C, Tessoldi PE, Dellantonio A. Visual selective attention and reading efficiency are related in children. Cortex 1998; 34: 531–46.

Corbetta M, Akbudak E, Conturo TE, Snyder AZ, Ollinger JM, Drury HA, et al. A common network of functional areas for attention and eye movements. Neuron 1998; 21: 761–73.

Cornelissen P, Richardson A, Mason A, Fowler S, Stein J. Contrast sensitivity and coherent motion detection measured at photopic luminance levels in dyslexics and controls. Vision Res 1995; 35: 1483–94.

Demb JB, Boynton GM, Heeger DJ. Functional magnetic resonance imaging of early visual pathways in dyslexia. J Neurosci 1998; 18: 6939–51.

Duncan J, Ward R, Shapiro K. Direct measurement of attentional dwell time in human vision. Nature 1994; 369: 313–5.

Eden GF, Zeffiro TA. Neural systems affected in developmental dyslexia revealed by functional neuroimaging. [Review]. Neuron 1998; 21: 279–82.

Eden GF, VanMeter JW, Rumsey JM, Maisog JM, Woods RP, Zeffiro TA. Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. Nature 1996; 382: 66–9.

Facoetti A, Molteni M. The gradient of visual attention in developmental dyslexia. Neuropsychologia 2001; 39: 352–7.

Facoetti A, Turatto M. Asymmetrical visual fields distribution of attention in dyslexic children: a neuropsychological study. Neurosci Lett 2000; 290: 216–8.

Facoetti A, Paganoni P, Lorusso ML. The spatial distribution of visual attention in developmental dyslexia. Exp Brain Res 2000; 132: 531–8.

Galaburda A, Livingstone M. Evidence for a magnocellular defect in developmental dyslexia. [Review]. Ann N Y Acad Sci 1993; 682: 70–82.

Galaburda AM, Menard MT, Rosen GD. Evidence for aberrant auditory anatomy in developmental dyslexia. Proc Natl Acad Sci USA 1994; 91: 8010–3.

Gilger J, Pennington B, Defries J. Risk of reading disability as a function of parental history in three family studies. Reading Writing: Interdiscipl J 1991; 3: 205–17.

Hari R, Kiesilä P. Deficit of temporal auditory processing in dyslexic adults. Neurosci Lett 1996; 205: 138–40.

Hari R, Koivikko H. Left-sided minineglect and attentional sluggishness in dyslexic adults [abstract]. Soc Neurosci Abstr 1999; 25: 1634.

Hari R, Sääskilahti A, Helenius P, Uutela K. Non-impaired auditory phase locking in dyslexic adults. Neuroreport 1999a; 10: 2347–8.

Hari R, Valta M, Uutela K. Prolonged attentional dwell time in dyslexic adults. Neurosci Lett 1999b; 271: 202–4.

Helenius P, Uutela K, Hari R. Auditory stream segregation in dyslexic adults. Brain 1999; 122: 907–13.

Hikosaka O, Miyauchi S, Shimojo S. Visual attention revealed by an illusion of motion. [Review]. Neurosci Res 1993; 18: 11–8.

Husain M, Shapiro K, Martin J, Kennard C. Abnormal temporal dynamics of visual attention in spatial neglect patients. Nature 1997; 385: 154–6.

Iles J, Walsh V, Richardson A. Visual search performance in dyslexia. Dyslexia 2000; 6: 163–77.

Klein RM, Farmer ME. Dyslexia and a temporal processing deficit: a reply to the commentaries. Psychon Bull Rev 1995; 2: 515–26.

Laasonen M, Tomma-Halme J, Lahti-Nuuttila P, Service E, Virsu V. Rate of information segregation in developmentally dyslexic children. Brain Lang 2000; 75: 66–81.

Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. Proc Natl Acad Sci USA 1991; 88: 7943–7.

Lovegrove WJ, Bowling A, Badcock D, Blackwood M. Specific reading disability: differences in contrast sensitivity as a function of spatial frequency. Science 1980; 210: 439–40.

Marois R, Chun MM, Gore JC. Neural correlates of the attentional blink. Neuron 2000; 28: 299–308.

McPeek RM, Maljkovic V, Nakayama K. Saccades require focal attention and are facilitated by a short-term memory system. Vision Res 1999; 39: 1555–66.

Merigan WH, Maunsell JH. How parallel are the primate visual pathways? [Review]. Annu Rev Neurosci 1993; 16: 369–402.

Merzenich MM, Schreiner C, Jenkins W, Wang X. Neural mechanisms underlying temporal integration, segmentation, and input sequence representation: some implications for the origin of learning disabilities. [Review]. Ann NY Acad Sci 1993; 682: 1–22.

Merzenich MM, Jenkins WM, Johnston P, Schreiner C, Miller SL, Tallal P. Temporal processing deficits of language-learning impaired children ameliorated by training. Science 1996; 271: 77–81.

Nicolson RI, Fawcett AJ, Dean P. Time estimation deficits in developmental dyslexia: evidence of cerebellar involvement. Proc R Soc Lond B Biol Sci 1995; 259: 43–7.

Posner MI, Raichle ME. Images of mind. New York: Scientific American Library; 1994.

Purpura K, Kaplan E, Shapley RM. Background light and the contrast gain of primate P and M retinal ganglion cells. Proc Natl Acad Sci USA 1988; 85: 4534–7.

Riddell PM, Fowler MS, Stein JF. Spatial discrimination in children with poor vergence control. Percept Mot Skills 1990; 70: 707–18.

Robertson IH, Mattingley JB, Rorden C, Driver J. Phasic alerting of neglect patients overcomes their spatial deficit in visual awareness. Nature 1998; 395: 169–72.

Schwartz T, Ojemann G, Dodrill C. Reading errors following right hemisphere injection of sodium amobarbital. Brain Lang 1997; 58: 70–91.

Sheppard DM, Bradshaw JL, Mattingley JB, Lee P. Effects of stimulant medication on the lateralisation of line bisection judgements of children with attention deficit hyperactivity disorder. J Neurol Neurosurg Psychiatry 1999; 66: 57–63.

Skottun BC. On the conflicting support for the magnocellular-deficit theory of dyslexia. Response to Stein, Talcott and Walsh (2000). Trends Cogn Sci 2000; 4: 211–2.

1380 R. Hari et al.

Stein J, Richardson A. Cognitive disorders: a question of misattribution. Curr Biol 1999; 9: R374–6.

Stein J, Walsh V. To see but not to read; the magnocellular theory of dyslexia. Trends Neurosci 1997; 20: 147–52.

Stein J, Riddell P, Fowler S. Disordered right hemisphere function in developmental dyslexia. In: von Euler C, Lundberg I, Lennerstrand G, editors. Brain and reading. Basingstoke (UK): Macmillan Press; 1989. p. 139–57.

Stein J, Talcott J, Walsh V. Controversy about the visual magnocellular deficit in developmental dyslexics. Trends Cogn Sci 2000; 4: 209–11.

Steinman BA, Steinman SB, Lehmkuhle S. Transient visual attention is dominated by the magnocellular stream. Vision Res 1997; 37: 17–23.

Steinman SB, Steinman BA, Garzia RP. Vision and attention. II: Is visual attention a mechanism through which a deficient magnocellular pathway might cause reading disability? Optom Vis Sci 1998; 75: 674–81.

Tallal P, Piercy M. Defects of non-verbal auditory perception in children with developmental aphasia. Nature 1973; 241: 468–9.

Tallal P, Merzenich MM, Miller S, Jenkins W. Language learning impairments: integrating basic science, technology, and remediation. [Review]. Exp Brain Res 1998; 123: 210–9.

Vidyasagar TR. A neuronal model of attentional spotlight: parietal guiding the temporal. [Review]. Brain Res Brain Res Rev 1999; 30: 66–76.

Received October 31, 2000. Revised February 27, 2001. Accepted March 5, 2001