Eye movements in hemianopia and the rehabilitation of hemianopic dyslexia

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EYE MOVEMENTS IN HEMIANOPIA AND THE REHABILITATION OF HEMIANOPIC DYSLEXIA

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ABSTRACT

This thesis is a study of the nature and rehabilitation of the functional impairments in unilateral homonymous hemianopia (HH), with a major focus on hemianopic dyslexia. The reading, visual exploration and line bisection impairments associated with homonymous visual field loss are frequent and well-established clinical phenomena. Yet, it is still unknown whether the reading and visual exploration impairments are caused by the visual field defect or by additional extrastriate injury preventing efficient spontaneous oculomotor adaptation. It is also unclear whether the line bisection impairment directly arises from the visual field defect or its adaptive oculomotor consequences, or whether it indicates an associated visual-spatial deficit that is caused by injury to regions involved in visual-spatial perception (*Introduction*). Based on a critical review of research into hemianopic dyslexia since its original description in 1881, it is suggested that the visual field defect is a major component of hemianopic dyslexia but possibly not its sole cause (*Chapter 1*). This assumption was confirmed in six experiments whose purpose was to establish the extent to which the reading, visual exploration and line bisection impairments associated with HH are purely visually elicited. To study the behavioural changes associated with the visual field defect that are not caused by brain injury, a gaze-contingent display paradigm was used to simulate HH in healthy participants. Simulated HH induced the reading and visual exploration impairments of hemianopic patients. However, all participants showed efficient spontaneous oculomotor adaptation to simulated HH which was associated with highly specific and task-dependent improvements in reading and visual exploration (*Chapters 2 and 3*). Moreover, simulated HH did not induce the main feature of the hemianopic line bisection impairment, i.e., the contralateral line bisection error, albeit it nevertheless impaired line bisection performance (*Chapter 4*). The final study investigated the basis and specificity of the therapeutic effect of an efficient compensatory oculomotor treatment method for hemianopic dyslexia in patients with unilateral homonymous visual field loss. The results demonstrate that using text-material and, thus, lexical-semantic processes, is not critical to the treatment effect, which was also found to be specific to reading (*Chapter 5*). The concluding chapter reviews the main findings and suggests that the functional impairments associated with visual field loss may not simply be failures of vision. Although the hemianopic visual field defect is a major component of hemianopic dyslexia and possibly contributes to the visual exploration and line bisection impairments, additional injury to specific extrastriate regions seems to be the critical causative factor. The implications for understanding, assessing and rehabilitating functional impairments in homonymous visual field disorders are discussed. The important future research directions arising from this thesis are also identified and presented (*Conclusion*).

DECLARATION

I hereby declare that no part of this thesis has been submitted for a degree at this or any other university. This thesis has been composed by myself and the research reported herein has been conducted by myself.

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Durham City, 2009, Susanne Schütt.

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This thesis is a testimony of my passion for visual disorders after brain injury and using eye-movements as a tool to help us better understand, assess and rehabilitate their disabling functional sequelae. This thesis is also a contemporary witness of the most challenging and most instructive years I have had in my life thus far. My PhD years in England have been of great importance to me, both from a professional and personal point of view. They were life-changing. In many other ways, this thesis reflects the support of several people who influenced my work and shaped my ideas during that important time. I owe a particular debt of gratitude to my supervisors and mentors Charlie Heywood, Bob Kentridge, and Josef Zihl. It was Josef Zihl who introduced me to and taught me about visual disorders, and it was during my patient work with him that my passion for visual disorders and using eye-movements as an assessment and rehabilitation tool was born. Charlie Heywood and Bob Kentridge made my "simulated hemianopia" happen, and it was in their Vision Laboratory where my passion for using eye-movements in research and clinical practice deepened. It has been a great privilege to work with my supervisors whose advice I seek and value greatly. I would like to thank them for their support in any aspect of my research. I have also enjoyed the privilege of getting valuable advice and support from several other people, about which I am truly grateful: I would like to thank Alan Cowey, John Findlay, Simon Liversedge and Keith Rayner for supporting my work on simulated hemianopia and the rehabilitation of hemianopic dyslexia since its beginnings. I am also grateful to Robert Metcalf for writing the software program to analyse my eye-movement data. I also would like to thank the many other people who supported me during my PhD years at Durham University's Psychology Department, at Munich University's Neuropsychology Department, particularly Karin Münzel and Ruth Dauner, and at Josef Zihl's Neuropsychology Unit at the Max-Planck-Institute of Psychiatry in Munich. Moreover, I am grateful to everyone who participated in my experimental studies. The German Academic Exchange Service (DAAD) was generous in supporting my research with a PhD scholarship and I am pleased to acknowledge its support. Finally, I would like to thank my parents, Marianne and Josef Hitzenberger, who have always believed in me in whatever I pursued, and I am particularly grateful for their generous support throughout my PhD. I also wish to thank my parents-in-law, Ilona and Manfred Schütt, who always provided me with a second home and supported me during my PhD years.

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For Robert,
without whom I would never be
where I am today.

TABLE OF CONTENTS

IMPAIRMENTS OF VISUAL FUNCTIONING IN HEMIANOPIA: FAILURES OF VISION?

1. Unilateral homonymous hemianopia and its functional impairments

In 1824 William Hyde Wollaston presented himself as the subject of a case to the Royal Society of London and described a "peculiar state of vision": "I could see but half the face of a man whom I met; and it was the same with respect to every object I looked at. In attempting to read the name JOHNSON, over a door, I saw only SON; the commencement of the name being wholly obliterated to my view. (...) The loss of sight was toward my left, and was the same whether I looked with the right eye or the left" (quoted in Lawrence, 1854, p. 681). This case became known as the first report of unilateral homonymous hemianopia (Simpson & Crompton, 2008), the most frequent visual disorder after brain injury (Zihl, 2000, 2003).

Unilateral homonymous hemianopia (HH) is a visual field disorder in which vision is lost in both monocular hemifields contralateral to the side of brain injury. It is caused by postchiasmatic visual pathway injury that is frequently accompanied by extrastriate lesions. Posterior cerebral artery infarction is the most common aetiology (Hebel & von Cramon, 1987; Zhang, Kedar, Lynn, Newman, & Biousse, 2006a; Zihl, 2000). Sufficient spontaneous recovery of the visual field or spontaneous oculomotor adaptation to visual field loss occurs rarely (Zhang, Kedar, Lynn, Newman, & Biousse, 2006b; Zihl, 2000; Zihl & Kennard, 1996). Visual field disorders therefore represent a chronic visual handicap that greatly compromises patients' occupational and daily life. Commonly, patients complain of persistent and severe difficulties with reading, orienting and navigating as well as with locating objects and avoiding obstacles (Papageorgiou et al., 2007; Zihl, 2000). In addition to impaired reading and visual exploration, patients frequently show an impairment of line bisection, i.e., a contralateral bisection error during manual line bisection, which seems to be

associated with difficulties in maintaining the straight-ahead direction during walking (Zihl, 2000).

This thesis is primarily concerned with the nature and rehabilitation of these functional impairments in HH. While a high degree of consensus about the characteristics of the reading, visual exploration and line bisection impairments associated with homonymous visual field disorders has been reached, the causes of these well-established and frequently reported clinical phenomena remain largely unknown. There is still considerable debate whether the reading and visual exploration impairments are caused by the visual field defect or by additional extrastriate injury preventing efficient spontaneous oculomotor adaptation. It is also unclear whether the line bisection impairment arises from the visual field defect or its adaptive oculomotor consequences, or whether it is an indicator of an associated visual-spatial deficit that is caused by additional injury to regions and fibre pathways involved in visual-spatial perception (Zihl, 2000). Thus, the impairments of vision-related functioning in homonymous visual field disorders may not simply be failures of vision. Yet, as long as their causes are unclear, our understanding of these functional impairments remains incomplete. Consequently, current practice of assessment and rehabilitation of homonymous visual field disorders after brain injury is imperfect since a clear understanding of the nature of the frequently associated reading, visual exploration and line bisection impairments is essential for assessment and rehabilitation effectiveness.

Thus far, the reading impairment associated with homonymous visual field disorders (hemianopic dyslexia) has received least attention, both as a functional impairment as well as an acquired reading disorder. This is surprising given that hemianopic dyslexia is one of the most important functional impairments after brain injury (Papageorgiou et al., 2007; Zihl, 2000). Since the ability to read is fundamental to daily living and an essential prerequisite to success in our modern society (Rayner & Pollatsek, 1989), hemianopic dyslexia has a major impact on patients' lives. Although reading depends as much on an intact visual field and efficient eye-movement control as on intact language functions, most research on acquired

reading disorders has focussed on aphasic reading impairments, the so-called higher-level reading disorders (Snowling & Hulme, 2005). Hemianopic dyslexia, the most elementary and possibly most frequent lower-level reading disorder, has been neglected. Even though hemianopic dyslexia is also the most frequent and disabling functional impairment in homonymous visual field loss (Zihl, 2000), the majority of studies on visual field disorders has been concerned with the visual exploration impairment and its rehabilitation (Bouwmeester et al., 2007). Since Poppelreuter's (1917/1990) first systematic attempt to rehabilitate the reading impairment in hemianopic patients, only five studies have dealt with this important matter of research (Kerkhoff, Münßinger, Eberle-Strauss, & Stögerer, 1992; Spitzyna et al., 2007; Zihl, 1995, 2000; Zihl, Krischer, & Meißen, 1984). This thesis seeks to remediate this unfortunate state of affairs and therefore puts its major focus on hemianopic dyslexia and its rehabilitation.

2. Thesis structure

The first report of hemianopic dyslexia dates back to 1881 when Mauthner described this important functional impairment in patients with HH. Chapter 1 provides a comprehensive review of the research that has been carried out since this original description. It attempts to develop a theoretical explanation of the reading impairment associated with homonymous visual field loss and to clarify its functional and anatomical bases. The critical examination and discussion of findings from research into hemianopic dyslexia and its rehabilitation suggests that the basis of this reading impairment may not be purely visual. Although the visual field defect seems to be a major component of hemianopic dyslexia, it may not be its sole cause.

This assumption may not only apply to the impairment of reading but also to that of visual exploration and line bisection. The purpose of the experiments reported in Chapters 2, 3 and 4 therefore was to establish the extent to which these impairments are visually elicited. To study the behavioural changes associated with the hemianopic visual field defect that are not caused by brain injury in reading, visual exploration and line bisection, a gaze-contingent

display paradigm (McConkie & Rayner, 1975; Rayner & Bertera, 1979) was used to simulate HH in healthy participants. Chapter 2 presents three experiments, of which the first investigated the effects of simulated HH on reading and visual exploration performance and eye-movements as well as on saccadic accuracy, an indicator of efficiency of visual exploration (Meienberg, Zangemeister, Rosenberg, Hoyt, & Stark, 1981; Zihl, 2000); the second and third experiments were conducted to determine whether and to what extent healthy participants spontaneously adapt their eye-movements to simulated HH in reading and in visual exploration. Chapter 3 reports an experimental study that explored whether spontaneous oculomotor adaptation to simulated HH is task-specific, or whether there is a transfer of adaptation-related changes in eye-movements and performance improvements between reading and visual exploration. The two experiments presented in Chapter 4 investigated the effect of simulated HH on line bisection performance and associated eye-movements. The first experiment examined this effect in a manual line bisection task; the second experiment used an ocular line bisection task without manual response (Ishiai, Koyama, & Seki, 1998) to explore the significance of manual and oculomotor factors in line bisection with simulated HH.

The final experimental chapter deals with the rehabilitation of the most important but most neglected of the functional impairments in homonymous visual field loss that were under investigation in the preceding chapters, i.e., hemianopic dyslexia. The study presented in Chapter 5 investigates the basis and specificity of the therapeutic effect of a compensatory oculomotor treatment method for hemianopic dyslexia (Zihl et al., 1984), which proved its effectiveness in a number of investigations (Kerkhoff et al., 1992; Spitzyna et al., 2007; Zihl, 1995, 2000). Since it is still unclear whether the treatment effect associated with this method critically depends on using text material, the effectiveness of systematic oculomotor training with non-text material in comparison with conventional oculomotor training using text material was evaluated in patients with unilateral homonymous visual field loss and hemianopic dyslexia.

The concluding chapter reviews the main findings and suggests that the functional impairments associated with visual field loss may not simply be failures of vision. Although the hemianopic visual field defect is a major component of hemianopic dyslexia and possibly contributes to the visual exploration and line bisection impairments, additional injury to specific extrastriate regions seems to be the critical causative factor. The implications for understanding, assessing and rehabilitating functional impairments in homonymous visual field disorders are discussed and important future research directions arising from this thesis identified and presented.

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Chapter 1

THE SIGNIFICANCE OF VISUAL INFORMATION PROCESSING IN READING: INSIGHTS FROM HEMIANOPIC DYSLEXIA

This chapter presents the first comprehensive review of research into hemianopic dyslexia since Mauthner's original description of 1881. It offers an explanation of the reading impairment in patients with unilateral homonymous visual field disorders and clarifies its functional and anatomical bases. The major focus of this review is on visual information processing, visuospatial attention and eye-movement control during reading. An advanced understanding of the basis of hemianopic dyslexia and its rehabilitation also increases our knowledge about normal reading and its underlying neural mechanisms. By drawing together various sources of evidence this review illustrates the significance of bottom-up and attentional top-down control of visual information processing and saccadic eye-movements in reading. Reading depends critically on the cortical-subcortical network subserving the integration of visual, attentional and oculomotor processes involved in text processing.

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1. Introduction

Reading is a complex skill which can be disturbed at any of its visual, lexical-semantic and phonological processing stages. A wide variety of quantitatively and qualitatively different reading disorders following brain injury has been identified (for reviews, see Ellis & Young, 1996; Hillis & Caramazza, 1992; Shallice, 1988). Acquired impairments of reading in subjects with previously well-established reading skills immediately draw to mind the aphasic reading disorders which involve disturbances of lexical and/or post-lexical processes. These higher-level reading disorders (central dyslexias) rank high in neuropsychology's research agenda and have substantially contributed to the development of models of the normal reading process.

Unfortunately, the acquired lower-level reading disorders, which involve impairments of pre-lexical (visual) processes, have been largely neglected. These so-called peripheral dyslexias arise from disturbances at the more peripheral levels of visual text information processing. Visual field disorders, deficits of visual acuity, spatial contrast sensitivity and visual adaptation, disorders in visuo-spatial perception, spatial restriction of the field of visual attention (a prominent symptom of visual neglect and Balint's syndrome), visual agnosia, and visual illusions and hallucinations can all impair reading at various levels of visual processing (Baylis, Driver, Baylis, & Rafal, 1994; Behrmann, Moscovitch, Black, & Mozer, 1990; Behrmann, Shomstein, Black, & Barton, 2001; De Luca, Spinelli, & Zoccolotti, 1996; Hess, Zihl, Pointer, & Schmid, 1990; Zihl, 1989, 1995a; Zihl & Kerkhoff, 1990; Zihl & von Cramon, 1986). Although the peripheral dyslexias have been attracting increasing attention recently, the chief focus has been on the clinical syndromes of neglect dyslexia and pure alexia or visual agnosia for letters.

Surprisingly, hemianopic dyslexia, the most elementary and frequent peripheral dyslexia (present in ~15% of patients in neurological rehabilitation centres, see Kerkhoff, 1999; Prosiegel, 1988), is hardly considered in reviews or text books dealing with peripheral dyslexias (e.g., Ellis & Young, 1996; Riddoch, 1991; Shallice, 1988). It perhaps counts as

the most important visual impairment following brain injury affecting the patients' occupational and daily life as a pronounced visual handicap (Papageorgiou et al., 2007; Zihl, 2000). Reading becomes so laborious that many patients give up recreational reading; if reading is essential for their occupation, continuing employment may be at risk (Leff, Spitzyna, Plant, & Wise, 2006). Hemianopic dyslexia (also called hemianopic alexia) is an acquired reading disorder in which 80% of patients with homonymous visual field defects affecting parafoveal (and foveal) vision have severe reading difficulties despite intact language functions (Zihl, 2000). In these patients, word identification and the ability to plan and guide reading eye-movements is disturbed (McDonald, Spitzyna, Shillcock, Wise, & Leff, 2006; Spitzyna et al., 2007; Zihl, 1995a).

This chapter offers the first comprehensive review of research into hemianopic dyslexia. It explains the nature of hemianopic dyslexia and clarifies its functional and anatomical bases. Furthermore, it considers what hemianopic dyslexia can tell us about normal reading and its neural basis. In this manner this review hopes to provide a coherent framework for future work. It is organised into six sections. The first section (i.e., Section 2) gives a brief survey of the themes relevant for the critical examination of the findings from hemianopic dyslexia research by introducing reading as a complex skill entailing coordinated visual information processing, eye-movement control, visuospatial attention and linguistic processing. Section 3 describes the features of homonymous visual field disorders and reviews the findings from hemianopic dyslexia research since Mauthner's original description of 1881. Section 4 demonstrates the significance of parafoveal vision for reading by discussing the effects of unilateral homonymous parafoveal visual field loss on word identification and oculomotor control in reading, both at the behavioural and neural level. Section 5 presents an examination of the anatomy of hemianopic dyslexia and shows that parafoveal visual field loss in itself cannot completely account for this reading impairment. Section 6 discusses a compensatory treatment approach for rehabilitating hemianopic dyslexia, which reveals important insights into the functional plasticity of the visual, attentional and oculomotor systems involved in text processing. Section 7 provides a

synopsis of all sources of evidence that demonstrates the important insights studying hemianopic dyslexia generates into the normal reading process and its neural basis, which may be useful in informing theories and models of reading and eye-movement control.

2. Reading: Vision, attention, eye-movements and language in (inter-)action

Poppelreuter (1917/1990) remarked that "it should theoretically be possible (...) to conclude a priori that a hemianopia (...) must impair reading" (p. 223) and regarded a detailed consideration of theories of visual information processing and eye-movements in normal reading as essential. However, he also firmly believed that hemianopic dyslexia could not be explained as "merely a consequence of (...) hemianopia" (p. 226). Thus, the basis of hemianopic dyslexia may not be purely visual. This section therefore introduces the visual, attentional, oculomotor and language processes involved in normal reading and their underlying neural mechanisms.

2.1. Eye-movements and visual information processing in reading

Reading is the process of understanding written language. This requires our eyes to move in such a way as to allow for the extraction of spatially distributed visual information which is in harmony with the speed of comprehension (Findlay & Gilchrist, 2003). The eyes follow a typical scan path across the text, in the direction depending upon the language of the text (i.e., from left-to-right and from top-to-bottom for Western cultures). Plotting eye position against time reveals a staircase pattern as saccadic eye-movements regularly alternate with periods of fixations. Whereas the majority of the words in a text are fixated, sometimes even twice (i.e., refixation; 15% of total fixations), many words are skipped; 2–3 letter words, for instance, only receive a fixation about 25% of the time. On average, a fixation during reading lasts for about 200–250 ms and is followed by a saccade to some 7–9 characters forward (~2–3°). About 10–15% of our reading saccades are regressive. Towards the end of a line of text, a large right-to-left slightly oblique saccadic eye-movement is made close to the beginning of the next line. The size of the return-sweep depends upon line length

(usually about 50 characters, ~17°) (Rayner, 1998; Rayner & Pollatsek, 1989). In continual information sampling, eye-movements may be coupled with head-movements. As most studies of reading eye-movements immobilize the head, relatively little is known about eye-head coordination and the role and pattern of head-movements in reading (Lee, 1999).

Eye-movements during reading are systematically influenced by visual and lexical characteristics of the text information extracted during a fixation (Rayner & Pollatsek, 1981). The region of effective processing during reading, the perceptual span, extends about 3–4 characters to the left and up to 15 characters to the right of fixation (in left-to-right writing systems). As one degree of visual angle encompasses about 3 characters for most normal text (Leff et al., 2000), these values are equivalent to ~1.3° to the left and 5° to the right of fixation (McConkie & Rayner, 1975, 1976; Rayner & Bertera, 1979). Visual acuity falls symmetrically to either side of foveal vision and the distribution of the perceptual span is therefore likely to reflect an attentional asymmetry in reading. Acuity limitations determine only its right boundary. Discriminating fine detail such as letters is only possible within the foveal region, which extends out 1° to either side of fixation. Visual acuity and processing speed decrease sharply with increasing eccentricity in the horizontal direction, and even more so in the vertical direction (Anstis, 1974). Therefore, readers are able to gain letter identity information up to 7–8 characters to the right of fixation (McConkie & Zola, 1987; Rayner, Well, Pollatsek, & Bertera, 1982; Underwood & McConkie, 1985). Beyond this, only coarse textual features can be discerned up to the rightward boundary of the parafoveal visual field (Rayner, 1998). The range of letters that can be reliably identified without moving the eyes, i.e., shifts of fixation, is called word identification span or visual span. This range depends, of course, on print size; larger fonts are more discriminable but, with increasing font size, letter strings will fall further into the visual periphery with a concomitant drop in acuity (Legge et al., 2007). The perceptual span for text processing, composed of the foveal and parafoveal visual field, is illustrated in Fig. 1. Since the perceptual span's spatial extent exceeds the average-sized word at a given fixation and the

mean amplitude of reading saccades, text material is scanned in a highly overlapping manner (Ikeda & Saida, 1978; Rayner & Bertera, 1979).

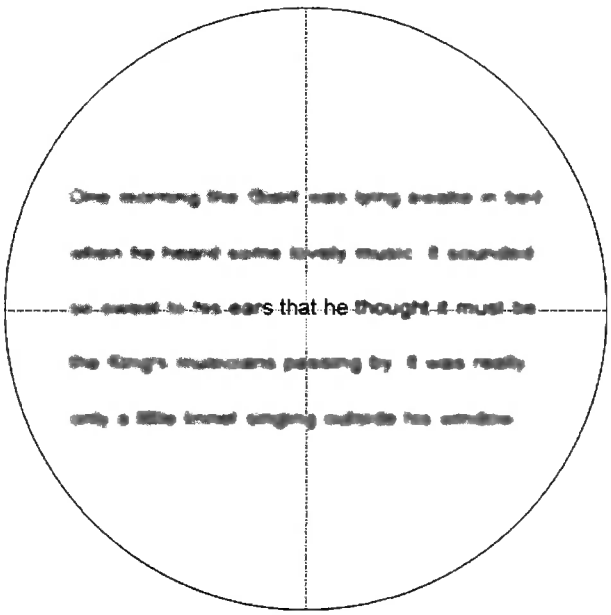

Fig. 1 Schematic illustration of the visual field and perceptual span for text processing in normal readers. During a fixation, readers extract visual information from the foveal visual field (central white oval) and the parafoveal visual field (grey ellipse). Note that the drawing is schematic and not drawn to scale; the cross-hairs indicating fixation position do not resemble the actual initial fixation position, which would be probably on the "h" of the fixated word "that" in normal readers (optimal viewing position).

Foveal processing of fixated words enables lexical access and hence word identification. Fixation duration is influenced by factors such as word frequency, predictability and age-of-acquisition (Rayner, 1998). During successive saccades, foveal processing is facilitated by information that has been extracted from the right parafovea on the preceding fixation, i.e., the so-called parafoveal preview benefit (Rayner, 1975; Rayner, White, Kambe, Miller, & Liversedge, 2003). Such information includes that of word-length, which is used for the selection of the to-be-fixated word and the specification of the saccadic amplitude (Ducrot & Pynte, 2002; Inhoff, Radach, Eiter, & Juhasz, 2003).

2.2. The neural basis of text reading

Our knowledge about the neural basis of reading continuous text is sparse compared with what is known about the neural mechanisms underlying single-word reading (for a recent review on word identification, see Jobard, Crivello, & Tzourio-Mazoyer, 2003). The neural mechanisms involved in text reading, which involves the initiation and maintenance of an oculomotor scanpath in addition to word identification, has been investigated in only two studies (Leff et al., 2000; Leff, Scott, Rothwell, & Wise, 2001). Reading involves visual, attentional, oculomotor and language processes (Rayner & Pollatsek, 1989), which are supported by large-scale neural networks (Mesulam, 1990). Distributed and coordinated processing relying on multiple cortical and subcortical brain regions suggests that white matter pathways connecting these regions play a crucial role (Ben-Shachar, Dougherty, & Wandell, 2007; Binder & Mohr, 1992).

Visual information is transmitted from the retinae to the primary visual (striate) cortex via the optic nerves, the optic chiasm, the optic tracts, the lateral geniculate nucleus, and the optic radiation (Grüsser & Landis, 1991). The striate cortex (V1), the prestriate visual area V2, the posterior parietal cortex and frontal eye fields, as well as the supplementary eye fields and the dorsolateral prefrontal cortex form a network which integrates vision, attention and eye-movements. Subcortical structures, particularly the superior colliculus and thalamus, also contribute to saccade control (for a more detailed discussion, see Leigh & Zee, 2006; Pierrot-Deseilligny, Rivaud, Gaymard, Müri, & Vermersch, 1995). This distributed neural system subserves the bottom-up (i.e., stimulus-driven) and top-down (i.e., goal-directed) control of visual-spatial attention and saccadic eye-movements via feedforward and feedback connections (Corbetta, 1998; Corbetta & Shulman, 2002). Although "attention and ocular control did not evolve for reading (...), reading is a special application of the attentional/ocular control system" (Kliegl & Engbert, 2003, p. 492).

The primary visual cortex (V1) appears indispensable for visually guided eye-movements and word identification during reading since it represents the foveal and

parafoveal visual field (Leff, 2004). There is evidence that the eyes are disparate on 40–50% of fixations during reading (Kirkby, Webster, Blythe, & Liversedge, 2008). It has therefore been suggested that a single perceptual representation is achieved through the visual integration of the two disparate retinal signals at a very early stage in the visual pathway (Liversedge, Rayner, White, Findlay, & McSorley, 2006). Word identification involves the activation of left and right striate and ventral prestriate cortex where foveal vision is represented. The guidance of reading eye-movements requires the representation of right parafoveal vision in the left primary visual cortex and neighbouring V2. The asymmetric activation of left parafoveal V1/V2 during text reading has been interpreted as physiological confirmation of the perceptual span's asymmetry, which is controlled by top-down attentional factors (Leff et al., 2000). This top-down attentional modulation of early visual information processing is mediated by fronto-parietal activity (Kastner, Pinsk, De Weerd, Desimone, & Ungerleider, 1999; Russell, Malhotra, & Husain, 2004) and results in the directing of visual attention to the right of fixation during reading (Upton, Hodgson, Plant, Wise, & Leff, 2003). Attentional processes facilitate visual processing in the striate and extrastriate cortices (Martinez et al., 2001) and in the ventral occipito-temporal stream (Mangun, Hopfinger, Kussmaul, Flechter, & Heinze, 1997), which is crucially involved in high-resolution, local processing of visual features and object identification (Milner & Goodale, 2006). Thus, "attention during reading acts early in the visual hierarchy" (Leff et al., 2000).

As words can be regarded as visual objects, the ventral stream has been implicated in word processing and identification processes (Poldrack, Desmond, Glover, & Gabrieli, 1998) which are associated with an activation of the foveal part of the left and right occipital cortex (V1/V2) (Brewer, Liu, Wade, & Wandell, 2005) and the left posterior occipito-temporal junction in the inferior temporal gyrus (Leff, Crewes, et al., 2001). Word identification is also the first stage of linguistic processing; its successful accomplishment provides the basis for intact language comprehension as it makes semantic, syntactic and thematic information available (Liversedge & Blythe, 2007). Left occipito-temporal activation might also be

mediated by top-down influences from the left-lateralized major language-processing areas involved in reading (Powell et al., 2006), i.e., the posterior superior temporal gyri, implicated in lexical and semantic processing, and the inferior frontal cortex, implicated in syntactic processing (Binder et al., 1997).

The posterior parietal cortex (PPC) is crucial for the generation of a visuospatial representation (based on bottom-up visual input from the parafoveal visual field) which then can be used by prefrontal mechanisms to guide attention and eye-movements concerned with visual information sampling from the top-down, i.e., visuo-motor integration (Leigh & Kennard, 2004; Zihl & Hebel, 1997). The projections from the parafoveal part of V1/V2 to posterior parietal regions illustrate the significance of the parafoveal visual field for the visual-spatial control of reading saccades. The transformations carried out in the dorsal processing stream mediate visuomotor control, and are thus an interface between perception and action (eye-movements) (Milner & Goodale, 2006). Bilateral activation of the PPC, with a greater signal on the left is associated with efficient reading saccades from left-to-right, i.e., into contralateral hemispace. Evidence suggests that it controls the online maintenance and modification of a sensorimotor plan which is required to read along each single line of text (Leff et al., 2000; Leff, Scott, et al., 2001).

Bilateral activation of the frontal eye fields (FEF), with a greater signal on the right is associated with the preparation of this sensorimotor plan at the beginning of each new line and with performing the return-sweep, which interrupts the oculomotor scanpath and requires a change of the sensorimotor plan (oculomotor flexibility). FEF activation is minimal for the continued generation of saccadic reading eye-movements along a line of text. The FEF seem to be less important for visually guided saccades but are crucial for intentional, voluntary generated saccades irrespective of their direction (Leff et al., 2000; Leff, Scott, et al., 2001). The FEF plays a key role in the top-down control of oculomotor scanpaths that follow a previously learned rule (e.g., reading direction imposed by the writing system). In addition, the oculomotor aspects of eye-movement control interact with

cognitive processes underlying visual word identification, which may also determine how long attention maintained at a specific position, i.e., the temporal aspect of saccade programming (Heinzle, Hepp, & Martin, 2007). Higher-level linguistic processing activities in the left anterior inferior prefrontal and left temporo-parietal cortex may also influence the duration of a fixation from the top-down (Posner, Abdullaev, McCandliss, & Sereno, 1999).

3. Hemianopic dyslexia: Reading when the visual world shrinks

Mauthner (1881) was the first to describe the effects of unilateral homonymous visual field defects on reading. His classic description marks the starting point of research into hemianopic dyslexia. Wilbrand (1907) termed this reading impairment associated with unilateral homonymous visual field loss "macular-*hemianopic* reading disorder" since hemianopia is the typical and most frequent visual disorder after brain injury (see also Poppelreuter, 1917/1990). It is the "cardinal symptom" which dominates all postchiasmatic visual pathway pathologies (Lenz, 1909).

3.1. Introducing cerebral visual field disorders

Homonymous visual field disorders account for about 20% of functional impairments after brain damage (Zihl, 2000, 2003). They are caused by injury to the postchiasmatic visual pathway, i.e., to the optic tract, the lateral geniculate nucleus, the optic radiation, or to the primary visual cortex (located at the calcarine sulcus) (Zhang, Kedar, Lynn, Newman, & Biousse, 2006). For these patients the "visual world shrinks" as vision is lost in both monocular hemifields contralateral to the side of brain injury (Grüsser & Landis, 1991, p. 136). Sufficient spontaneous recovery of the visual field occurs rarely and, therefore, homonymous visual field deficits can be regarded as chronic manifestations (Zihl & Kennard, 1996).

In addition, posterior cerebral artery infarctions, the most common aetiology underlying homonymous visual field loss (~70%, see Zhang et al., 2006; Zihl, 2000), are seldom restricted to calcarine cortex only. Additional lesions to the occipital white matter,

which might affect fibre pathways connecting occipital, parietal, temporal and frontal cortical regions, as well as to the posterior thalamus are the rule rather than the exception for these patients (Hebel & von Cramon, 1987). As a consequence, the majority of patients (about 70%) show persistent and severe impairments of reading and visual exploration (for oculomotor scanning in hemianopia, see Pambakian et al., 2000; Tant, Cornelissen, Kooijman, & Brouwer, 2002; Zihl, 1995b, 1999, 2000).

Visual field disorders can be measured quantitatively by perimetric techniques (see, e.g., Aulhorn & Harms, 1972) and are classified according to the portion of the visual field affected. After unilateral damage, the most common type is hemianopia, the loss of vision in one hemifield (of both eyes), followed by quadranopia, the loss of vision in one quadrant, and paracentral scotoma, a small island-like field defect in the parafoveal visual field. Left-sided lesions result in right-sided visual field defects, and *vice versa*. After bilateral brain injury, corresponding portions in both visual hemifields may be affected. The resulting disorders are analogously termed: bilateral hemianopia (tunnel vision), bilateral upper or lower quadranopia, and bilateral paracentral scotoma. The loss of vision in the central visual field region is referred to as central scotoma. Unilateral visual field disorders are much more common than those resulting from bilateral brain injury (~90% of patients with visual field disorders). Depending on the quality of the deficit, vision can either be completely lost (anopia) or one or more visual functions in the affected visual field can be reduced (amblyopia). In cerebral amblyopia, light sensitivity is reduced whereas form and/or colour vision is lost (Zihl, 2000). The selective loss of colour vision is referred to as achromatopsia (Zeki, 1990). Testing visual functions like colour and form vision requires the use of special targets and procedures in perimetric testing (see Aulhorn & Harms, 1972).

The extent of visual field sparing in the affected hemifield is measured in degrees of visual angle from the fovea. In unilateral postchiasmatic damage, the foveal or central visual field (± 0.5–1.0°) is always spared. Macular sparing (visual sparing between 1–5° to the left or right of fixation) is seldom and most likely results from incomplete damage to the striate

cortex or its afferent connections (Zihl, 1989; Zihl & von Cramon, 1986). Approximately 75% of patients with unilateral homonymous visual field disorders present a parafoveal visual field sparing of less than 4°. Visual field sparing (co-)determines the resulting functional visual impairment. As a rule, patients with a smaller field sparing are more disabled, especially with regard to visual functions that crucially depend on the parafoveal region, such as reading (Zihl, 1989, 2000). When parafoveal visual field sparing is smaller than 4° 75% of patients with left-sided field loss and as many as 92% of patients with right-sided defects show pronounced reading difficulties, i.e., hemianopic dyslexia (Zihl, 1994). When visual field sparing ranges between 5–10°, reading is still yet less disturbed in about 25% of cases. Reading is rarely impaired when field sparing exceeds 10° (Zihl, 2000). Fig. 2 schematically illustrates the visual field and perceptual span for text processing in left- or right-sided hemianopia, quadranopia, and paracentral scotoma.

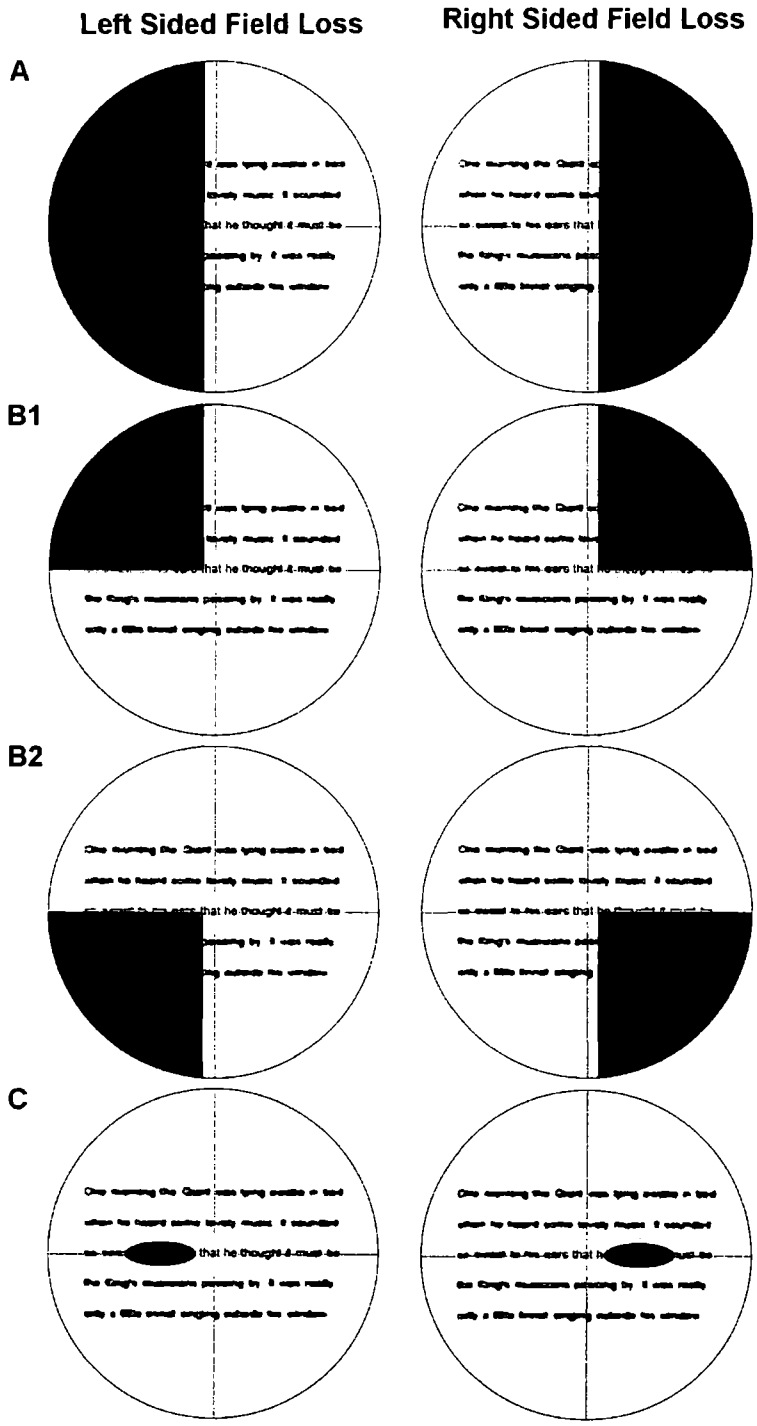

Fig. 2 Schematic illustration of the visual field and perceptual span (comprising of foveal (central white oval) and parafoveal vision (grey ellipse)) for text processing in patients with left- or right-sided unilateral homonymous parafoveal visual field loss (field sparing: ~1°) (affected binocular regions in black). **A**: hemianopia; **B1**: upper quadranopia; **B2**: lower quadranopia; **C**: paracentral scotoma. Note that the drawing is schematic and not drawn to scale. The cross-hairs indicating fixation position do not resemble the actual initial fixation position, which would be probably located to the left of the optimal viewing position (i.e., left of the "h" in "that") in right-sided field loss (McDonald et al., 2006; Spitzyna et al., 2007); for left-sided field loss, the initial fixation position has not yet been investigated.

Diagnosing hemianopic dyslexia requires the presence of a homonymous parafoveal visual field defect (as confirmed by detailed perimetric testing). It is essential to exclude disorders of visual acuity, spatial contrast sensitivity, visual adaptation, disturbances of the anterior visual pathways or the oculomotor system, macular disease (as revealed by ophthalmological examination) and language disorders that could interfere with the correct processing of text material. Wilbrand (1907) clearly differentiated hemianopic dyslexia from aphasic reading disorders. In hemianopic dyslexia, reading is impaired despite intact lexical and post-lexical processes (see also Best, 1917; Poppelreuter, 1917/1990).

A hemianopic reading impairment also must be clearly distinguished from pure alexia (letter-by-letter reading) (for the first report, see Dejerine, 1891). Although pure alexia is usually accompanied by a right-sided hemianopia, the visual field defect is not causally linked to it (for a collection of key articles, see Coltheart, 1998). Pure alexia seems to be associated with a serial encoding of letters (Behrmann et al., 2001; Rayner & Johnson, 2005). The diagnosis of hemianopic dyslexia also requires the absence of any signs of visual-spatial neglect in standard tests. Left-sided hemianopia and visual-spatial neglect often coexist and can be difficult to disentangle (Walker, Findlay, Young, & Welch, 1991). Despite the absence of neglect symptoms, however, patients may nevertheless exhibit neglect dyslexia (for the first report, see Brain, 1941). Evidence suggests a clear double dissociation between neglect symptoms and neglect dyslexia (for a review, see Haywood & Coltheart, 2000; Riddoch, 1991). Recently, neglect dyslexia was interpreted as a deficit of extracting visual information from the left side of space (Behrmann, Black, McKeeff, & Barton, 2002). Explaining neglect dyslexia in this manner may be reminiscent of hemianopic dyslexia. Yet, both reading impairments are distinct disorders and have to be differentiated.

3.2. Reading performance and eye-movements in hemianopic dyslexia

Since Mauthner's (1881) first description, several studies have dealt with hemianopic dyslexia and a high degree of consensus about its characteristics has been reached. It has consistently been shown that a visual field defect "is a disturbing obstacle and, depending on

its location to the right or left of the fixation position, unpleasantly manifests itself in different ways" (Wilbrand, 1907, p. 6, my translation). Yet, not only whether right- or left-sided parafoveal vision is affected but also how much of it is spared (co-)determines type and severity of the resulting reading impairment in homonymous visual field disorders (Mackensen, 1962). It is noteworthy that most findings are based on investigations of left-to-right text reading in patients with unilateral left- or right-sided unilateral homonymous hemianopia (LH, RH).

Reading performance

In hemianopic dyslexia research reading speed (correctly read words *per* minute, wpm) and accuracy (uncorrected reading errors), the standard measures of reading performance, are recorded while patients engage in reading aloud standardised texts, as quickly and accurately as possible. These texts are easy to comprehend and letter size, spacing between lines, words and letters are maintained as optimal for reading. Reading speed is significantly reduced in both LH and RH, in comparison with age-matched normal readers. Slowness of reading is the distinctive attribute of hemianopic dyslexia, and applies not only to text but also to reading single words (Kerkhoff, Münßinger, Eberle-Strauss, & Stögerer, 1992; Spitzyna et al., 2007; Zihl, 1995a, 2000; Zihl, Krischer, & Meißen, 1984; Zihl & von Cramon, 1986). Reading time increases with each additional letter, especially in patients with small visual field sparing. Yet the effect is not as pronounced as in letter-by-letter reading (Behrmann et al., 2001; Leff, Crewes, et al., 2001; Rayner & Johnson, 2005).

Patients make only relatively few reading errors and are, therefore, often overlooked in neuropsychological examinations. Nevertheless, reading errors in hemianopic dyslexia do occur and can be characterised as visual omissions of letters, syllables, and even short words. Patients also make meaningful completions of only partially seen words by adding syllables to their beginning or to their end. As a result, errors are introduced by guesses. Patients do not show letter-by-letter reading or spelling errors. Reading errors are caused by the visual field defect at the visual-sensory level. They are visually related to the actual word being

read and consistently affect just one side of the word, i.e., the side of the blind field (Zihl, 1995a, 2000). Examples of oral reading in patients with hemianopic dyslexia are presented in Table 1.

Table 1 Examples of oral reading in patients with left- (**A**) or right-sided (**B**) unilateral homonymous parafoveal visual field loss (field sparing: ~2°) (**[~~abe~~]** indicates visual omission errors; *[pause]* indicates reading interruptions; guessing errors are ***italicised***).

Original text
The trees were in leaf, and the rumps of the tourist buses were thick and fat in the traffic, and all the farmers wanted fertilizer admixes rather than storehouse insulation when Sixsmith finally made his call. In the interim, Alistair had convinced himself of the following: before returning his aggrieved letter, Sixsmith had steamed it open and then resealed it. During this period also, Alistair had grimly got engaged to Hazel. But the call came.
A
[~~The~~] trees were in leaf, and **[~~the~~]** rumps of the tourist **[~~b~~]uses** [*pause*] buses were thick and fat **[~~in~~]** the traffic, and all the [*pause*] farmers wanted fertilizer **[~~ad~~]mixes** rather than **[~~store~~]house** insulation when **[~~Six~~]smith** finally made his call. In the interim, Alistair had convinced **[~~him~~]self** [*pause*] himself of the following: before **[~~re~~]turning** his **[~~ag~~]grieved** [*pause*] aggrieved letter, **[~~Six~~]smith** had steamed it open and then **[~~re~~]sealed** [*pause*] **[~~re~~]sealed** it. During this period also, Alistair had grimly got **[~~en~~]gaged** [*pause*] engaged to Hazel. But the call came.
B
The trees were in **[~~leaf~~]**, and the rumps of **[~~the~~]** tourist buses were thick and fat **[~~in~~]** the traffic, and all **[~~the~~]** farmers **want[~~ed~~]** ***fertile*** [*pause*] wanted ***to be fertile*** [*pause*] **admix[~~es~~]** [*pause*] ***admixture*** [*pause*] rather than **store[~~house~~]** [*pause*] insulation when **Six[~~smith~~]** [*pause*] finally made his call. In the interim, **Ali[~~stair~~]** [*pause*] Alistair had **convince[~~ed~~]** ***to*** **convince[~~d~~]** [*pause*] himself of the following: before **return[~~ing~~]** ***the*** **return[~~ing~~]** ***of*** his aggrieved letter, **Six[~~smith~~]** had steamed it open and then resealed it. During this period also, **Ali[~~stair~~]** had grimly got **engage[~~d~~]** to Hazel [*pause*] ***an engagement with*** Hazel. But the call came.

Note. The text is taken from Martin Amis (1994). Career move. In G. Gordon & D. Hughes (Eds.), The Minerva book of short stories (p. 14). London: Mandarin Paperbacks. Figure is adapted from Zihl (2000, p. 72).

Oral reading performance (reading speed and errors) considerably differs between left- and right-sided parafoveal visual field defects. Patients with a left-sided defect require about twice as much reading time as normal readers. An average reading speed of 78 wpm was measured in a sample of left-sided hemianopic patients whereas the corresponding figure for normal subjects (N) was 174 wpm (Zihl, 2000). Reading errors mainly consisted of omissions of prefixes and small words, especially at the beginning of lines (~4 errors, Zihl, 1995a) (see Table 1, A). In patients with a right-sided defect reading speed was only ~56

wpm (Zihl, 2000). They also made three times as many errors as patients with left-sided field loss (~13 errors, Zihl, 1995a). These errors can be characterised as omissions and substitutions of suffixes and small words, especially at the end of lines (see Table 1, B).

The reading impairment as defined by reading rate and number of errors is not only related to the side but also to the severity of the parafoveal visual field loss. Reading time and errors increase with decreasing visual field sparing. This inverse relationship holds for both left- and right-sided parafoveal visual field loss but is more pronounced in right-sided field loss (LH: <3°: 53 wpm, >5°: 124 wpm; RH: <3°: 43 wpm, >5°: 98 wpm) (Zihl, 2000).

Reading eye-movements

The first formal electro-oculographic investigations of hemianopic dyslexia were carried out by Remond, Lesevre and Gabersek (1957) (cited in Ciuffreda, 1994), followed by Mackensen (1962) and Gassel and Williams (1963a). Mackensen (1962) viewed reading as a sensorimotor ability and therefore regarded the study of eye-movements in hemianopic dyslexia as indispensable. His eye-movement recordings revealed a dramatic increase in the number of fixations and saccades. Gassel and Williams (1963a) observed similar irregularities in a larger sample of patients with unilateral homonymous hemianopia. The severe alteration of the oculomotor reading pattern is the most objective behavioural manifestation of the reading impairment in hemianopic dyslexia.

Detailed eye-movement analyses have provided a comprehensive understanding of the global temporal and spatial oculomotor measures associated with text processing during silent reading in hemianopic dyslexia (Zihl, 1995a, 2000). Overall, prolonged fixation durations (LH: 310 ms; RH: 410 ms; N: 250 ms), smaller amplitudes (LH: 4.0°; RH: 3.2°; N: 4.3°), more fixations (LH: 76; RH: 87; N: 56), and a much higher percentage of refixations (LH: 37%; RH: 44%; N: 15%) have been reported (Zihl, 2000). The increased number and duration of fixations and especially the increased likelihood to refixate words seem to account for the slowness of reading in hemianopic dyslexia (McDonald et al., 2006; Zihl, 1995a).

Although word-based analyses of text reading are standard in experimental reading research (Rayner, 1998), it is only recently that local spatial and temporal oculomotor measures have been obtained in patients with right-sided hemianopia. The initial landing position for longer words moves from the centre towards the beginning and small words are less likely to be skipped (RH: 22%; N: 63% (3-letter-words)). First fixation and gaze durations as well as the total fixation time are about twice as long as in normal readers (McDonald et al., 2006; see also Spitzyna et al., 2007). Like experimental reading research all hemianopic dyslexia research has been based on monocular eye-movement recordings. Binocular recordings of eye-movements in hemianopic dyslexia may provide further insights into the binocular coordination of reading eye-movements and the mechanisms underlying the formation of a single perceptual representation from disparate retinal signals (Liversedge et al., 2006).

The differential effects of left- and right-sided parafoveal visual field loss on reading eye-movement patterns were reported by Mauthner (1881). Patients with left-sided hemianopia showed difficulties to find the beginning of a new line. When compared to the impairments associated with right-sided hemianopia, Mauthner regarded these difficulties as negligible. In left-sided hemianopia, to him the "more pleasant" disorder, "only the words which have already been read disappear, and looking ahead at the upcoming words is not disturbed" (p. 370, my translation) whereas in right-sided hemianopia "the despair is enormous that from the point of fixation the visual field is cut off completely in the direction of reading; hence nothing can be read ahead" (Mauthner, 1881, p. 370, my translation). Wilbrand (1907) described a considerable uncertainty and hesitation about where to move the eyes next in patients with right-sided parafoveal visual field loss; to him, it looked as if their eyes were stuck at the currently fixated word (see also Best, 1917).

The differences between left- and right-sided parafoveal visual field loss in the majority of global eye-movement parameters are illustrated in Fig. 3.

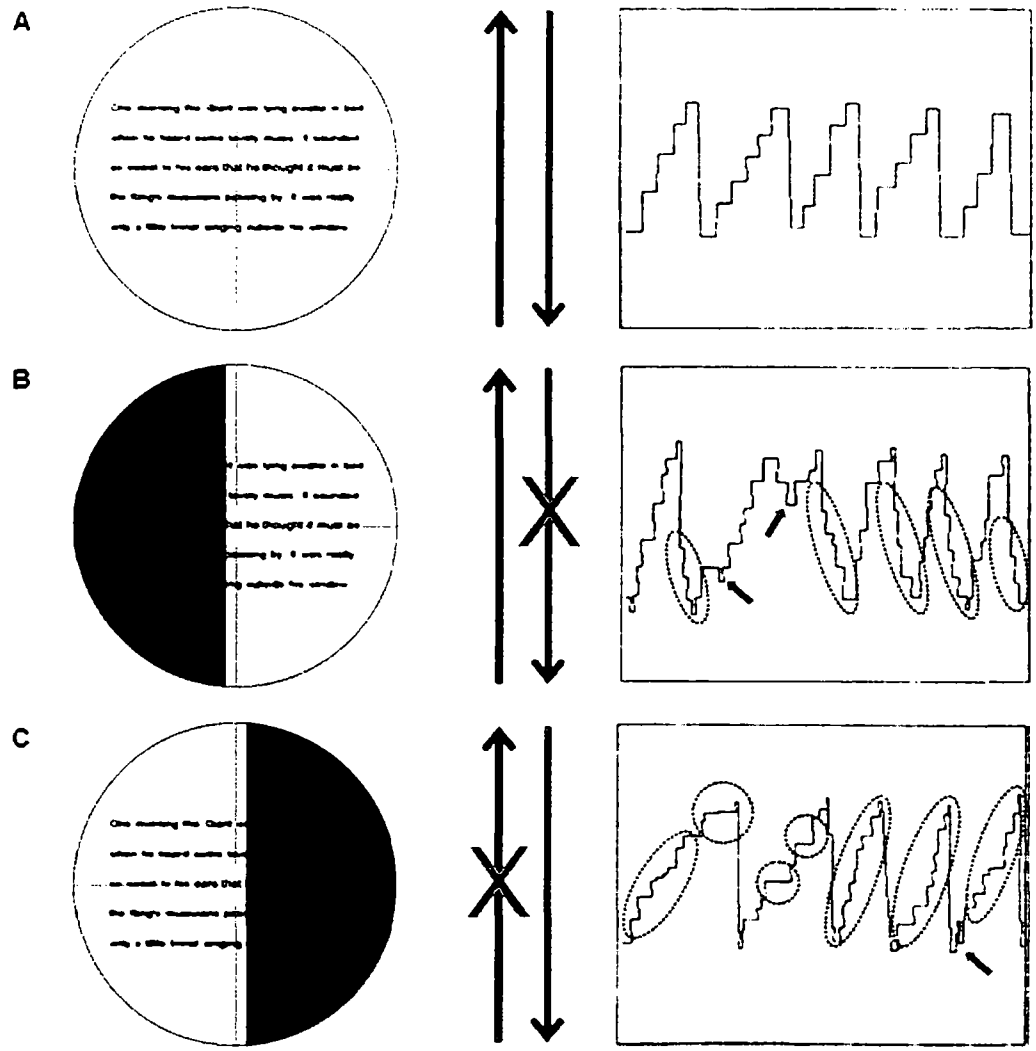

Fig. 3 Infra-red eye-movement recordings in normal readers (**A**) and in patients with left- (**B**) or right-sided (**C**) unilateral homonymous hemianopia (field sparing: ~1°). For illustration purposes, eye-movement patterns for reading five lines (x-axis: time period of recording; y-axis: horizontal extension of line from left to right) are shown in relation to the visual field and perceptual span for text processing (**B**, **C**: affected binocular regions in black). Downward arrows indicate moving the eyes from the end to the beginning of a new line (which is disturbed in **B** (crossed arrow) as indicated by ellipses); upward arrows indicate moving the eyes from the beginning to the end of a line (which is disturbed in **C** (crossed arrow) as indicated by ellipses). Ovals indicate prolonged fixations, small arrows indicate regressions. Eye-movement recordings are adapted from Zihl (1995a).

In left-sided field visual field loss, the oculomotor reading pattern differs from the typical staircase pattern of normal readers (see Fig. 3, A) although overall reading performance can be regarded as slowed yet, more or less, fluent reading. The return-sweep appears fragmented. It is reduced to half of its normal size (LH: 9.4°; N: 17.3°) (Zihl, 1995a) and is broken down into many small saccades (Mackensen, 1962). Patients make many more smaller leftward saccades and show a higher percentage of repetitions of saccades and

fixations to the left (Zihl, 2000) (see Fig. 3, B). A right-sided visual field defect, on the contrary, impairs shifting the gaze systematically from left-to-right while the return-sweep remains unaffected. The staircase-like oculomotor reading pattern is severely deteriorated and replaced by many small and irregular saccadic eye-movements to the right (see Fig. 3, C: ellipse). The amplitude of rightward saccades is significantly reduced and the total number of saccades increases (Zihl, 2000). Numerous leftward-directed regressions occur (see Fig. 3, C: small arrow) and fixation durations are considerably prolonged (up to 1.5 s, Zihl, 1995a) (see Fig. 3, C: ovals). These findings have been confirmed and replicated elsewhere (De Luca et al., 1996; Eber, Metz-Lutz, Bataillard, & Collard, 1987; Leff et al., 2000; McDonald et al., 2006; Schoepf & Zangemeister, 1993; Spitzyna et al., 2007; Trauzettel-Klosinski & Brendler, 1998).

The degree of visual field sparing also clearly contributes to the irregularities of the oculomotor pattern in terms of an inverse relationship. Patients with only 1–2° of field sparing show the most disturbed oculomotor reading pattern, in particular when the right hemifield is affected. Patients with a right-sided defect and 5° of sparing are still much more disabled than patients with a left-sided defect of the same extent who show a close to normal reading eye-movement pattern (Fig. 4) (De Luca et al., 1996; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a, 2000).

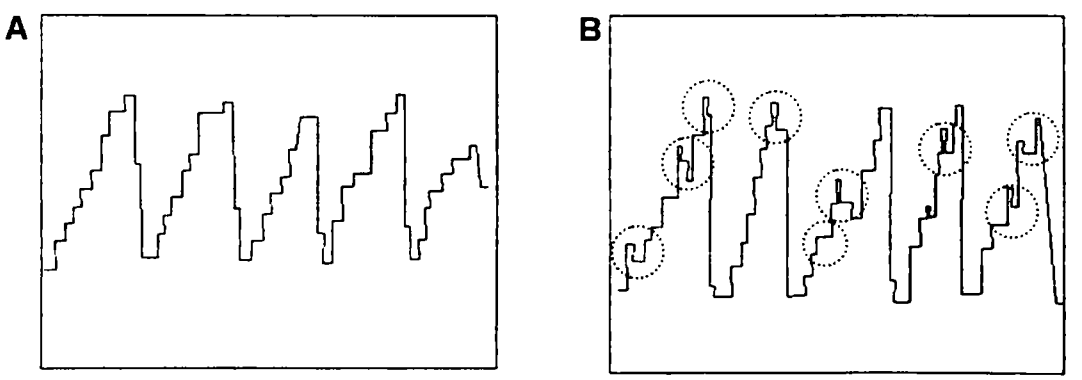

Fig. 4 Infra-red eye-movement recordings in patients with left- (**A**) or right-sided (**B**) unilateral homonymous hemianopia (field sparing: ~5°). Eye-movement patterns during reading of five lines are shown (x-axis: time period of recording; y-axis: horizontal extension of line from left to right). Note the more or less regular oculomotor reading pattern in **A** in comparison to the distorted oculomotor pattern in **B** (ovals indicate regressions and prolonged fixations). Eye-movement recordings are adapted from Zihl (1995a).

If at least 3° to the left and 5° to the right of fixation are spared in homonymous visual field loss, reading is nearly unimpaired (Mackensen, 1962; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a, 2000). Yet, how much visual field sparing is required for reading to be unimpaired might be better described in number-of-characters than in degrees-of-visual-angle. It is the number of characters (in relation to print size) that determines saccade size in reading (Morrison & Rayner, 1981; O'Regan, 1983) and sets the spatial boundary of the perceptual and word identification span. Describing visual sparing in terms of number of characters rather than visual angle may also explain better Wilbrand's (1907) finding that the reading impairment of his patients with a paracentral scotoma was more pronounced for small print, despite normal visual acuity. Print size determines the number of letters which can be identified at a glance; the smaller the print, the smaller is the word identification span (Anstis, 1974). The values given above as critical visual field requirement for unimpaired reading in patients with visual field defects hold for text that has 3 characters per degree. Thus, according to hemianopic dyslexia research the perceptual span extends 15 characters to the right and 9 to the left of fixation, confirming the asymmetry as well as the right boundary of the perceptual span in normal readers (McConkie & Rayner, 1975, 1976; Rayner & Bertera, 1979).

The left boundary may vary depending on whether readers engage with a text passage or with a single sentence. Only in the former case return-sweeps have to be performed, which may require 9 rather than 3–4 characters to the left of fixation. One may speculate that the perceptual span flexibly adapts to the changed reading direction (right-to-left) and becomes asymmetric to the left of fixation. This would also explain the finding that the first word is often not fixated in normal reading (Rayner & Pollatsek, 1989) and usually omitted in left-sided parafoveal visual field loss (Wilbrand, 1907).

Most studies investigated reading in patients with hemianopia and, therefore, less is known about the distinctive effects of a quadranopia or paracentral scotoma (see Fig. 2: B1/2, C). Nevertheless, there are some data available suggesting that the characteristic

reading difficulties are present in all patients with unilateral homonymous visual field disorders if parafoveal vision is affected by brain injury (Mackensen, 1962; Wilbrand, 1907; Zihl, 2000). Although seemingly small and negligible, paracentral scotomata can disturb reading substantially: Wilbrand's (1907) patients reported a "notorious impediment to their usual speed when gliding along the lines of text" (p. 1, my translation) (see also Mackensen, 1962). Yet investigations of single cases suggest that reading speed is higher and the number of fixations and refixations much smaller than in patients with a hemianopia. A quadranopia seems to affect reading performance and oculomotor parameters even less than paracentral scotoma (Zihl, 2000).

Furthermore, it seems that no complete loss of vision (anopia) is necessary for a reading impairment to emerge. Amblyopic forms of unilateral homonymous visual field disorders can cause hemianopic dyslexia if the residual visual field for form vision is smaller than 4–5°. Since text processing requires the visual discrimination of forms (letters), the reading impairment in homonymous hemi–amblyopia is almost identical with the impairment in hemi–anopia (Wilbrand, 1907; Zihl, 2000). Hemianopic dyslexia is quite common in homonymous hemi–amblyopia as only 25% of these patients show at least 5° of visual sparing (Zihl, 1994). Only recently a case of subtle hemianopic dyslexia in right-sided unilateral homonymous quadrant–amblyopia has been reported in detail. The threshold for luminance detection was elevated and form vision (especially for letters) was severely reduced in the upper right quadrant. Text reading was slowed down and the number of reading errors significantly increased. Minor abnormalities in the oculomotor reading pattern were found (Habekost & Starrfelt, 2006).

4. Reading without a parafovea: Seeing only half the wor(l)d

Despite a growing literature on reading impairments in unilateral homonymous visual field defects and relatively consistent results, the explanation of hemianopic dyslexia is still in its infancy. Mauthner (1881) and Wilbrand (1907) suggested that it is the loss of the parafoveal visual field in unilateral homonymous visual field disorders which causes hemianopic

dyslexia. The discussion of the effects of parafoveal visual field loss on word identification and oculomotor control in reading, both at the behavioural and neural level, demonstrates the significance of parafoveal vision for reading. Yet, we have to keep Poppelreuter's (1917/1990) remark in mind that explaining hemianopic dyslexia as merely a functional consequence of parafoveal visual field loss may not provide the full story.

4.1. Word identification without a parafovea

The activation of the left occipito-temporal junction associated with word identification processes is still present in patients with right-sided homonymous hemianopia. Even patients with a very small visual sparing show this activation although the necessary input from left striate cortex representing right foveal and parafoveal vision is missing (Leff, Crewes, et al., 2001). In contrast to pure alexia, the left occipito-temporal junction as well as its afferents from left and right striate cortex is spared in hemianopic dyslexia (Leff et al., 2006). Hence, information from the intact contralateral (i.e., right) striate cortex (representing the left visual field) must be transferred to the left occipito-temporal junction via the splenium of the corpus callosum. Intact afferent connections from the right occipital cortex to the left occipito-temporal junction appear sufficient to support word identification in patients whose right parafoveal vision is compromised (Leff, Crewes, et al., 2001).

Word identification through this indirect route, however, can be regarded as less efficient. In left-to-right readers, words are processed and identified best in the right foveal/parafoveal visual field represented in the left striate cortex (Brysbaert & Nazir, 2005; Nazir, 2000; Nazir, Ben-Boutayab, Decoppet, Deutsch, & Frost, 2004). This may also explain the finding that patients with larger right-sided visual field sparing and patients with left-sided visual field loss (i.e., right-sided injury) are less impaired in word identification (Upton et al., 2003).

In most patients with unilateral homonymous parafoveal visual field loss the perceptual and visual span may be no more than 3–4 characters. Yet the more letters can be identified at a single fixation, the larger is the amplitude of reading saccades, which

facilitates faster reading. The visual span imposes a limit on reading speed and is also referred to as the "sensory bottleneck" in reading (Legge et al., 2007). If this bottleneck is additionally restricted by parafoveal visual field loss, forward saccades become smaller and many more saccades have to be made to extract the same amount of text information for correct word identification. This effect is most pronounced when reading longer words (Leff, Crewes, et al., 2001; McDonald et al., 2006). Converging evidence stems from low vision readers whose visual and perceptual span is restricted by foveal processing difficulties due to macular disease (Chung, Legge, & Cheung, 2004; Crossland & Rubin, 2006; Legge, Ahn, Klitz, & Luebker, 1997).

Parafoveal visual field loss prevents that the beginning and end of a word are simultaneously visually apprehended. Especially longer words are never seen as a whole and parallel letter processing, which is required for efficient lexical word identification (Rayner & Pollatsek, 1989), is disturbed. Incomplete visual percepts of half-seen words are encoded and forwarded to higher-level linguistic processing units. Often, the visual input is insufficient to activate corresponding representations in the mental lexicon. If an incompletely encoded word makes sense and activates a lexical representation, visual omission errors emerge. Guessing errors occur if the predictive value of the incomplete percept is used for a meaningful completion of the word (Zihl, 2000). For instance, words which can be misread by omission or substitution of the first letter (e.g., peach: each or beach) increase the likelihood of errors in left-sided parafoveal visual field loss (Ellis, Flude, & Young, 1987).

Patients seem to over-rely on higher-level linguistic processes to compensate for the missing visual information when trying to identify words. Extracting meaning from an incompletely perceived word (comprehension) rather than inspecting the entire word first (visual apprehension) is the preferred strategy. Higher-level linguistic processes come into play too early which disrupts further acquisition and processing of text information located in the blind hemifield. Overall, processing words when the parafoveal visual field is

compromised requires more time. Difficulties in word identification, which also affect language comprehension, are reflected by longer fixation durations. As regressions occur as attempts to correct linguistic processing difficulties (Rayner & Pollatsek, 1989), their increased number in parafoveal visual field loss is not surprising.

Hence, for processing text information, patients make use of their residual foveal/parafoveal vision and linguistic processes. Reading in parafoveal visual field loss can, therefore, still be regarded as non-random visual information sampling (see also McDonald et al., 2006). Further evidence stems from a small sample of patients with a left- or right-sided homonymous hemianopia (De Luca et al., 1996). Patients identified high-frequency words much quicker than low-frequency words. Reading passages containing low-frequency words was associated with an increased number of saccades and regressions, longer fixation durations and smaller saccadic amplitudes. Words embedded in a textual context were identified quicker than words in isolation (contextual constraints). Meaningful words were inspected and read quicker than non-words (lexical constraints) (De Luca et al., 1996). Similarly, reading multi-digit numbers is much more prone to visual omission errors than is reading meaningful text material as much less facilitating top-down information becomes available (Zihl, 2000). One of Poppelreuter's (1917/1990) patients with a right-sided paracentral scotoma showed a pronounced reading impairment when confronted with meaningless or Latin text but "read familiar text (...) like a normal" (p. 224). High contextual constraint (as determined by word predictability) facilitates word skipping, reduces fixation durations and refixations (Binder, Pollatsek, & Rayner, 1999; Ehrlich & Rayner, 1981; Pynte & Kennedy, 2006; Rayner, 1998; Rayner & Well, 1996).

Right-sided parafoveal visual field loss affects not only processing the foveal word but also impairs preprocessing of the to-be-identified word located in the parafovea. During a fixation, readers process information from the fovea and parafovea; attentional top-down processes facilitate processing of the foveal text information first and the attentional focus then shifts to the parafoveal visual field (Reichle, Rayner, & Pollatsek, 2003). Parafoveal

preprocessing is indispensable for maintaining fast and fluent reading (Blanchard, Pollatsek, & Rayner, 1989; Inhoff, 1987; Rayner, 1979; Rayner et al., 1982; Rayner et al., 2003). The missing parafoveal preview benefit in right-sided parafoveal visual field loss contributes to the decreased likelihood of word skipping as well as to the overall increase in fixation duration and number of fixations (McDonald et al., 2006). Multiple words are no longer able to be processed during a single fixation and, therefore, a larger proportion of words have to be fixated. Furthermore, guiding reading saccades towards the centre of the to-be-fixated word where word processing is optimal (O'Regan & Lévy-Schoen, 1987; Vitu, O'Regan, & Mittau, 1990) becomes increasingly difficult as it requires right parafoveal word-length information. The initial fixation position wanders towards the beginning of the word and thus further away from the optimal viewing location. The resulting difficulties in word processing are reflected by longer fixation durations and an increased number of refixations (McDonald et al., 2006).

4.2. Visual guidance of reading eye-movements without a parafovea

Parafoveal visual field loss disturbs the integration of visual and motor processes: "Successive gaze-shifts from left to right (...) are no longer in the order dictated by the visual information, but occur irregularly" (Poppelreuter, 1917/1990, p. 224). Visual information extraction from the parafoveal (and peripheral) visual field regions that provides the basis for a top-down control of visual attention and eye-movements in space and further local processing of fine details is impaired (Hochstein & Ahissar, 2002; Juan & Walsh, 2003; Upton et al., 2003). Word- and line-length as well as page boundary information may be represented at higher levels and form a coordinate system containing the relative spatial location of word-objects (Kennedy, Brooks, Flynn, & Prophet, 2003). This spatial coordinate system enables the attentional selection of the to-be-identified word. Saccades are computed accordingly and identification processes via local information processing of fine detail are initiated (Deubel, O'Regan, & Radach, 2000; Deubel & Schneider, 1996; McConkie & Zola, 1987).

Mackensen's (1962) observation that a paracentral scotoma produces a less pronounced reading impairment than a hemianopia may confirm such an assumption. He speculated that the lines above and below the paracentral scotoma may be used for visual guidance of reading eye-movements. A hemianopia, on the contrary, prevents the extraction of this visual information (compare Fig. 2: A vs. C). Although the information below and above the current line does not affect oculomotor control in normal readers (Pollatsek, Raney, LaGasse, & Rayner, 1993), it may alleviate the reading impairment in patients with a paracentral scotoma.

A functional neuroimaging study (PET) investigated reading eye-movements in three patients with right-sided homonymous hemianopia and complete loss of right parafoveal vision (Leff et al., 2000). Eye-movement recordings of text reading revealed abnormal oculomotor reading parameters and reading speed was severely reduced. Instead of the left-lateralised PPC and right-lateralised FEF activation observed in normal readers, PPC was symmetrically activated and FEF activation was left-lateralised. Interestingly, a patient with a right-sided homonymous hemianopia that spared parafoveal vision showed the normal patterns of activation. His reading speed was in the range of age-matched controls and the oculomotor reading pattern was, despite a slight increase in the number of rightward saccades, more or less normal. Hence, the extent of the visual field defect seems to determine the level of functioning of the neural systems (PPC, FEF) subserving eye-movement control during text reading. Based on these results hemianopic dyslexia was interpreted as a disconnection of the motor systems involved in planning and guiding reading saccades from the representation of right parafoveal vision in the left striate and prestriate cortex (Leff et al., 2000).

In left-to-right reading, the left hemisphere (left striate and prestriate cortex, left-lateralised PPC activation) seems to be of greater importance for controlling oculomotor activities along a line of text than the right hemisphere (Leff et al., 2000; Leff, Scott, et al., 2001). The observation that right-sided parafoveal visual field loss (left-sided injury) impairs

left-to-right reading more severely than a left-sided field loss (right-sided injury) is in line with this finding. However, one might question a fundamental, hard-wired asymmetry in cortical activities associated with text reading. Reading-related brain activation and its lateralisation appears to be functionally determined as indicated by investigations of the neural basis of reading across writing systems (Al-Hamouri et al., 2005; Bolger, Perfetti, & Schneider, 2005; Skoyles, 1988). Evidence suggests that cultural differences in writing systems are reflected by differential activation across the neural network(s) mediating reading-related processes (Schlaggar & McCandliss, 2007). The influence of reading direction on text information processing and related eye-movements might be mediated by a top-down control which determines the dynamics of visuospatial attention allocation, i.e., the size and location of the perceptual span (Osaka, 2003). The reversed asymmetry of the perceptual span in right-to-left writing systems such as Hebrew (Pollatsek, Bolozky, Well, & Rayner, 1981) supports this assumption.

Converging evidence stems from a case study that reports a skilled bilingual reader with a left-sided hemianopia who had pronounced reading difficulties in his mother tongue Hebrew (right-to-left reading) but not in his second language English (left-to-right reading) (Leker & Biran, 1999; Mohamed, Elsherbiny, & Goulding, 2000). That the asymmetry of the perceptual span in bilinguals flexibly adapts according to the reading direction of the language which is currently being read is in line with this study (Pollatsek et al., 1981). Already Mauthner (1881) speculated that the differences in reading impairment between left- and right-sided parafoveal visual field loss might be functional: in right-to-left writing systems a "right-sided hemianopia appears to be more desirable" (Mauthner, 1881, p. 370, my translation). Functional neuroimaging (and behavioural) studies of hemianopic dyslexia in right-to-left writing systems would be very illuminating in this regard. Comparing patients with right-sided parafoveal visual field loss in left-to-right writing systems with patients showing left-sided parafoveal visual field loss in right-to-left writing systems might clarify the (relative) significance of left-lateralized activation of the cortical structures involved in text processing and related eye-movements.

Planning and guiding the return-sweep is associated with right FEF activation (Leff et al., 2000; Leff, Scott, et al., 2001) and depends on the extraction of line-length information from the left parafoveal visual field (McConkie & Zola, 1987). Left-sided parafoveal visual field loss impairs the accurate discrimination of the beginning of the line and, therefore, affects the visual guidance of the return-sweep. The observation that overly long lines disrupt the return-sweeps of normal readers supports this assumption (Gassel & Williams, 1963a; Rayner & Pollatsek, 1989). Gassel and Williams (1963a) reported that the return-sweep of their left-sided hemianopic patients improved after reading a few lines. In contrast to left-to-right reading saccades, the return-sweep's saccadic target, i.e., the first word of the next line, is almost always at a fixed horizontal position (most print text is left justified). After some practice with a text the spatial coordinates of the left text boundary might be represented within a higher-level framework, which may mitigate the effects of a left-sided parafoveal visual field loss on the visual guidance of the return-sweep.

5. Looking beyond parafoveal visual field loss: Is hemianopic dyslexia purely visually elicited?

Poppelreuter (1917/1990) pointed out that "the impairment caused by the hemianopia itself is not that substantial" (p. 223), and "the disturbance of the co-ordination of the reading gaze-shifts" (p. 224) associated with hemianopic dyslexia may not be solely visually elicited. Examining adaptation processes in homonymous parafoveal visual field loss and the anatomical conditions that are responsible for the severe and long-lasting reading impairments in patients with hemianopic dyslexia will show that parafoveal visual field loss is a necessary yet not a sufficient condition that causes hemianopic dyslexia.

5.1. Hemianopic dyslexia and the question of spontaneous oculomotor adaptation

Moving masks and window studies with normal readers may suggest that hemianopic dyslexia is purely visually elicited. Visual masks or windows occluding either the foveal or parafoveal visual field produce reading impairments in normal readers similar to those

caused by homonymous visual field disorders (Cummings & Rubin, 1992; Fine & Rubin, 1999a, 1999b, 1999c; Ikeda & Saida, 1978; McConkie & Rayner, 1975, 1976; Rayner & Bertera, 1979; Rayner, Inhoff, Morrison, Sowiaczek, & Bertera, 1981).

Reading using parafoveal and peripheral vision, i.e., "reading without a fovea" (Rayner & Bertera, 1979), is almost impossible (see also Fine & Rubin, 1999a; Fine & Rubin, 1999b, 1999c; Rayner et al., 1981) as is found by patients with a central scotoma (Teuber, Battersby, & Bender, 1960). Two single cases have been reported where reading speed was as low as 3 and 12 words per minute (see Zihl, 2000, pp. 151-164). Having to rely exclusively on foveal vision (reading without both parafoveas) also makes reading difficult. A one-letter moving window forces normal readers into letter-by-letter reading (Rayner & Bertera, 1979; Rayner et al., 1981), similar to the reading-style of pure alexics (Johnson & Rayner, 2007; Rayner & Johnson, 2005). The 'natural' counterparts of these experimental moving windows are bilateral homonymous visual field disorders, which affect both left and right parafoveal vision. Mackensen (1962) found the distinctive reading impairments of left- and right-sided parafoveal visual field loss appear in combination in such patients. Reading performance is worst in patients with a bilateral hemianopia (tunnel vision) as their residual vision may be nothing else than a one-letter moving window (Zihl, 2000). Reading without *a* parafovea may be less difficult. Yet, obliterating the left or right parafoveal visual field in normal readers produces reading impairments similar to hemianopic dyslexia (Cummings & Rubin, 1992; Ikeda & Saida, 1978; McConkie & Rayner, 1975, 1976; Rayner et al., 1981; Rayner, Liversedge, & White, 2006).

One must not forget that if an artificial visual field defect is imposed the resulting reading impairments are not as severe and long-lasting as in hemianopic dyslexia. Normal subjects seem to adapt quickly to visual field loss (Poppelreuter, 1917/1990), although interindividual differences may be substantial (Zangemeister & Utz, 2002). Furthermore, not all patients with unilateral homonymous parafoveal visual field loss necessarily show impaired reading. Adequate reading performance was found in 16% (out of 50 cases) about

six weeks after brain injury (Zihl, 1995a), and in 29% (out of 35 cases) when followed over a period of three years (Gassel & Williams, 1963a). Despite the prevailing parafoveal visual field defect, impaired reading performance as well as the concomitant abnormalities of the oculomotor parameters were no longer evident. Furthermore, Mackensen (1962) reported a case with a remarkable reading performance despite a severe right-sided homonymous hemianopia with only 0.5° visual field sparing. Such observations contradict the assumption that parafoveal visual field loss is the sole cause of hemianopic dyslexia and raise the question of the extent to which hemianopic dyslexia has a purely visual basis.

From his investigations Mackensen (1962) concluded that the severity of the reading impairment is not only determined by the presence of parafoveal visual field loss but also by whether and how well one has learnt to compensate for the visual defect. To overcome their visual impairment the most obvious solution for patients seems to be using appropriate eye-movement strategies. Patients consistently shift their gaze, thus their visual field border, into the area corresponding to their blind hemifield, thereby bringing obscured visual information briefly into the seeing field. It was Poppelreuter (1917/1990) who first reported spontaneous oculomotor compensation in visual field loss.

There is a consistent set of compensatory oculomotor strategies to which patients resort in order to cope with their lost part of the visual field. Targets located in the blind hemifield are approached with a safe-but-slow staircase strategy (series of small stepwise, hypometric saccadic eye-movements) especially if the target is unpredictable (Meienberg, Zangemeister, Rosenberg, Hoyt, & Stark, 1981). Most patients resort to such strategy, which is, however, time-consuming, laborious and simply insufficient to effectively compensate for parafoveal visual field loss (see also Poppelreuter, 1917/1990; Williams & Gassel, 1962; Zihl, 2000). They also employ this careful, safe-but-slow staircase strategy in reading ("beginning and end of line detective") – their reading rate is considerably reduced, and the number of errors is increased, in comparison with normal readers. More efficient adaptive reading strategies are characterised by top-down guided, predictive overshooting saccades in

the direction of the blind field ("blind hemifield overshooting") (Meienberg et al., 1981; Zangemeister, Oechsner, & Freska, 1995; Zangemeister & Utz, 2002).

Such spontaneous adaptative strategies are, however, rarely found (Schoepf & Zangemeister, 1993). A common observation is rather that patients with homonymous visual field loss shift their head towards the affected side (Zihl, 2000). As head movements normally follow and depend on saccadic eye-movements (Uemura, Arai, & Shimazaki, 1980), reversing this normal physiological sequence to compensate for visual field loss is maladaptive and might even increase the resulting functional visual impairment (Kerkhoff et al., 1992). Although some patients with parafoveal scotomas regain normal reading performance despite only 1–2° of visual field sparing, in the majority of patients reading impairments persist (Zihl, 2000). So, parafoveal visual field loss in itself cannot completely account for hemianopic dyslexia.

Consequently, there must be specific requirements for the ability to develop a compensatory eye-movement strategy with time (see also Kennard, 2002). Unquestionably, effective compensation implies some (implicit) knowledge of how to compensate (Zihl, 2000). Furthermore, learning to cope with a homonymous visual field loss and developing spontaneous compensatory strategies should require some time: "the complicated processes of compensation (...) can come to light as only slowly and gradually acquired improvements" (Poppelreuter, 1917/1990, p. 239). Evidence suggests that reading performance and the concomitant eye-movement parameters can improve with time after the onset of visual field loss (Gassel & Williams, 1963a). However, patients seem to either start very early spontaneously compensating for their parafoveal field loss or they do not regain normal reading performance even several weeks or months after their initial visual field loss (Zihl, 1995a). Thus, patients can be classified into two categories according to whether or not they develop spontaneous compensation strategies.

The decisive factor seems to be whether injury to the postchiasmatic visual pathway is accompanied by additional injury to the occipital white matter, occipitoparietal structures, or

the posterior thalamus. Patients in which these structures are spared show very efficient spontaneous oculomotor compensation, notwithstanding very small degrees of visual field sparing. Even after posterior cerebral artery infarction extra-striate injury is the rule rather than the exception (Hebel & von Cramon, 1987), which may explain the high percentage of patients showing little or no spontaneous compensation (Zihl, 1995a). Support for this hypothesis stems from the fact that these structures and their reciprocal connections are assumed to be part of a cortical-subcortical network subserving the bottom-up and top-down control of visual-spatial attention and related saccadic eye-movements (Corbetta, 1998; Corbetta & Shulman, 2002), which may be crucial for the development of spontaneous compensatory oculomotor strategies. If the structures and interconnecting callosal fibre pathways of this network are spared, parafoveal visual field loss can be mitigated by a specific set of top-down controlled intentional saccades into the blind hemifield. As a consequence, the regular eye-movement pattern required for effective text processing can be regained (Zihl, 1995a).

5.2. Hemianopic dyslexia and its anatomical basis

From our knowledge of the anatomy of the retino-striate visual pathway, we can infer the anatomical loci in which damage gave rise to a specific pattern of visual field loss. Injury to central, i.e., postchiasmatic portions of the pathway leads to characteristic homonymous visual field defects, which can be predicted from the retinotopic organization of the pathway. Most commonly, lesions are located in the optic radiations and the striate cortex (Harrington, 1976; Zhang et al., 2006), typically resulting in a hemianopia with and without macular sparing. Injury to the posterior part of the optic radiations and the striate cortex results in congruous homonymous visual field defects, i.e., they share the same location, extent and shape in the two monocular visual fields. Incongruous and incomplete defects typically occur in cases with injury to the anterior parts of the postchiasmatic pathway (optic tract, lateral geniculate body, and the anterior part of optic radiation) (Harrington, 1976; Zihl & von Cramon, 1986).

However, although no empirical data are available, it appears reasonable to assume that for hemianopic dyslexia to emerge the locus of damage along the postchiasmatic visual pathway is not decisive and has no differential effects on the resulting reading impairment. Hemianopic dyslexia can be caused either by injury to the optic tract, the lateral geniculate body, the optic radiation, or the striate cortex – presupposed this injury is accompanied by additional damage to the fibre pathways and/or structures constituting the neural network subserving the bottom-up and attentional top-down control of visual information processing and saccadic eye-movements in reading (Zihl, 1995a). Injury to the primary visual cortex (or its geniculostriate afferents) in itself (Leff et al., 2006) cannot completely account for hemianopic dyslexia.

So far, only one study has analysed the anatomical basis of hemianopic dyslexia (Zihl, 1995a). In a sample of 50 patients with left- or right-sided homonymous hemianopia, reading performance of patients whose brain injury was restricted to calcarine cortex (location of primary visual cortex) (16% of patients) was close to normal (155 wpm; ~89% of normal reading performance, N: 175 wpm), and sufficient for their occupational and daily life. In cases with larger lesions involving the striate cortex and partially the occipital white matter (44% of patients) a moderate reading impairment was found (108 wpm; ~62% of normal reading performance). Extensive unilateral injuries involving the occipital white matter (in 26% of patients) and the posterior thalamus (in 14% of patients) resulted in a severe and long-lasting reading impairment and pronounced visual handicap (56 wpm; ~32% of normal reading performance) (Zihl, 1995a).

Reports of three single cases with right-sided homonymous hemianopia (visual field sparing: 2°) further confirm and illustrate more clearly that hemianopic dyslexia is not purely visually elicited (Zihl, 1995a). Despite showing the same visual field defect and the same field sparing, these patients differed greatly with regard to their reading speed (A: 120 wpm, B: 82 wpm, C: 32 wpm). In addition, they did not differ with regard to age (A: 50 years, B: 46 years, C: 46 years) nor time since lesion (A: 8 weeks, B: 9 weeks, C: 14 weeks).

Although patient C had the longest time since lesion, and thus the longest time to adapt to his defect, he nevertheless showed the most severe reading impairment. A comparison of their underlying lesions revealed that in patient A, who showed almost normal reading performance, the lesion was restricted to calcarine cortex; in patient B, who showed a moderate reading impairment, occipital white matter was, in addition, partly affected; in patient C, whose reading impairment was worst, a major portion of the occipital white matter and the posterior thalamus were affected. The same finding was reported for three patients (D, E, F) with left-sided homonymous hemianopia (LH; 2°) (Zihl, 1995a).

The pronounced differences in reading speed between right- and left-sided parafoveal visual field losses seem to diminish if the extent and site of lesions is controlled for when making the comparison. The lesions of patient A (RH) and D (LH; age: 46 years, time since lesion: 7 weeks) were restricted to calcarine cortex, and both showed almost normal reading performance with almost similar reading speeds (A: 120 wpm, D: 105 wpm); Patient B (RH) and E (LH; age: 52 years, time since lesion: 8 weeks) had both partly occipital white matter involvement and showed a similar reduction in reading speed (B: 82 wpm, E: 87 wpm). Patient C (RH) and F (LH; age: 58 years, time since lesion: 9 weeks) showed both extensive occipital white matter involvement and a severe reading impairment. Yet, the greater reduction of reading speed in patient C (32 wpm; F: 68 wpm) cannot be fully explained by the difference between right- and left-sided parafoveal visual field loss – the additional involvement of damage to the posterior thalamus in patient C has to be taken into account (Zihl, 1995a).

The posterior thalamus and its connections to the occipital, parietal and frontal lobes, and the limbic neocortex are involved in the visual guidance of eye-movements (Ogren, Mateer, & Wyler, 1984; Robinson & Petersen, 1992; Zihl & von Cramon, 1979). Injury to the occipital white matter might damage the fibre pathways which connect the visual areas of the brain to motor areas for the visual control of eye-movements. In addition to the cortico–cortical fibre connections between visual, parietal and frontal areas, the subcortical pathways

connecting visual cortical areas and pontine cells, which also receive input from the superior colliculus, may also be affected by damage to the occipital white matter (Glickstein, 2000).

Injury to the striate cortex or its geniculostriate afferents, the occipital white matter comprising subcortical–cortical reciprocal connections, and/or the posterior thalamus, causes parafoveal visual field loss. These injuries may impair, to varying degrees, the bottom-up and attentional top-down control of visual information processing in the fovea and parafovea and the eye-movements involved in reading. Lesions that are confined to calcarine cortex result in parafoveal visual field loss which may disturb visual information processing and bottom-up oculomotor control (Leff et al., 2000). However, restricted calcarine cortex lesions preserve the neural network that mediates efficient visual processing and related oculomotor processes from the top-down. Intact attentional top-down control can facilitate visual information processing and the guidance of eye-movements into the blind field via feedback connections. The interactive flow of activation between V1/V2 and parietal as well as frontal cortical regions via feedforward (bottom-up) and feedback connections (attentional top-down modulation of V1/V2) supports such a view (Foxe & Simpson, 2002; Hochstein & Ahissar, 2002; Juan & Walsh, 2003). Where top-down attentional mechanisms are intact, an oculomotor strategy can be developed and adjusted to compile a complete percept of each word, even though each fixation provides only an incomplete view. Spontaneous oculomotor adaptation efficiently substitutes the lost visual field region via top-down processing and the ability to read remains more or less intact (Zihl, 1995a, 2000).

Evidence on the anatomical basis of hemianopic dyslexia suggests that this reading impairment is more than purely visually determined. Hemianopic dyslexia is not caused by parafoveal visual field loss resulting from unilateral postchiasmatic injury alone. Severe and long-lasting saccadic eye-movement abnormalities in reading and related impairments of text processing require widespread damage to the distributed neural network subserving the bottom-up and attentional top-down control of visual information processing and saccadic eye-movements in reading. In contrast, patients with sparing of the structures belonging to

this neural network usually compensate for their parafoveal visual field loss and show a close to normal reading performance. The high frequency of combined striate/white matter lesions in patients with homonymous visual field defects (Hebel & von Cramon, 1987) nevertheless justifies the further usage of the term hemianopic dyslexia to characterize this special type of reading impairment (Zihl, 1995a).

Our current knowledge of the anatomical basis of hemianopic dyslexia is based on an analysis of CT and MRI scans (Zihl, 1995a). These methods may underestimate the extent of lesions. An unilateral lesion to the optic radiation or striate cortex might change glucose metabolism in the intact ipsilateral thalamus and visual association areas as revealed by PET studies (Bosley et al., 1985). Such 'remote' effects are interpreted as interruption of the fibre pathways interconnecting both structures, which leads to a deactivation of the primary intact structure (Grüsser & Landis, 1991). These effects have to be differentiated from primary lesion sites for a valid interpretation of behavioural deficits and for developing a model of the functional organisation of the processes underlying complex behaviour such as reading (Zihl, 1995a). Consequently, we may (re-)interpret the effects on reading-related PPC and FEF activation patterns in patients with right-sided homonymous hemianopia (Leff et al., 2000). Preparation of reading saccades may not be disrupted solely by right-sided hemianopia arising from left V1/V2 damage. It is also possible that fibres connecting cortical visual areas with parietal and frontal areas may have been affected in these patients.

6. The rehabilitation of hemianopic dyslexia: Re-learning eye-movement control in reading

Poppelreuter (1917/1990) was the first who systematically attempted training patients with hemianopic dyslexia to learn, or re-learn, oculomotor control in reading. He showed convincingly that in patients with a lost parafoveal visual field "relearning of reading was successful" (p. 249). As Poppelreuter (1917/1990) said, the main goal for patients is "to make the preserved paracentral portion (...) a field for reading" and to move "the location of the position of the clearest vision further to the right or to the left" (p. 248). He taught his

patients to use a wooden reading stick which they moved successively from word to word of the text they read off a board. Patients with a right-sided visual field loss were asked to place the reading stick at the end of the word that is currently being read, patients with a left-sided defect had to place it at the beginning of words. Patients therefore learn to shift their attention and gaze intentionally into their blind field. After a few weeks of training, hesitant reading gave way to regular reading with correct intonation (a valuable behavioural indicator of rehabilitation of hemianopic dyslexia, see Mackensen, 1962; Zihl, 2000), and difficulties in identifying words and text comprehension were reduced. Reading speed increased and errors were reduced (Poppelreuter, 1917/1990).

Gassel and Williams (1963b) also found that the refinement and employment of attentional and gaze-shifts are the basis for oculomotor compensation in patients with homonymous visual field defects. To regain reading performance, patients have to intentionally shift their gaze further than they can actually see, i.e., into their blind field, so that they can perceive the entire word or text passage again; they learn "to keep the 'blind side' in sight" (Zangemeister & Oechsner, 1999, p. 89). Well-timed gaze shifts can re-establish the temporal and spatial coherence of successive extracted parts of visual information, which leads to the experience of seeing words at one glance again (Zihl, 1995a). Intentionally shifting attention and gaze so as to perceive each word as a whole before reading it is of particular importance in ameliorating word processing and identification difficulties. It is crucial that patients learn to visually apprehend before comprehend a word (Zihl, 2000).

Although Poppelreuter's (1917/1990) wooden reading stick has not stood the test of time, the rationale behind his quirky procedure is still valid. It has survived in the form of a compensatory treatment approach for rehabilitating patients with hemianopic dyslexia using, instead of a wooden stick, an electronic reading aid with gliding text (Zihl, 1995a; Zihl et al., 1984). An alternative yet more flexible and efficient treatment method is the PC-based, tachistoscopic presentation of text material (Zihl, 2000). Regular and systematic massed

practice allows new oculomotor strategies to be consolidated into flexible oculomotor reading routines (Ofen-Noy, Dudai, & Karni, 2003). Over-learning gradually leads to the 'automatization' of this strategy and hence comfortable reading (see also Bäckman, 1999).

Eye-movement recordings after only a few training sessions (about 10 to 15 sessions, 45 min.) reveal more or less normal oculomotor reading patterns and reading performance in the majority of cases (Zihl, 1995a, 2000). Overall, patients make fewer and shorter fixations, and show fewer regressions and refixations within words. The amplitude of rightward saccades increases especially in right-sided visual field loss. Patients with left-sided field loss make larger leftward saccades (return-sweeps) (see Fig. 5) (Zihl, 1995a, 2000).

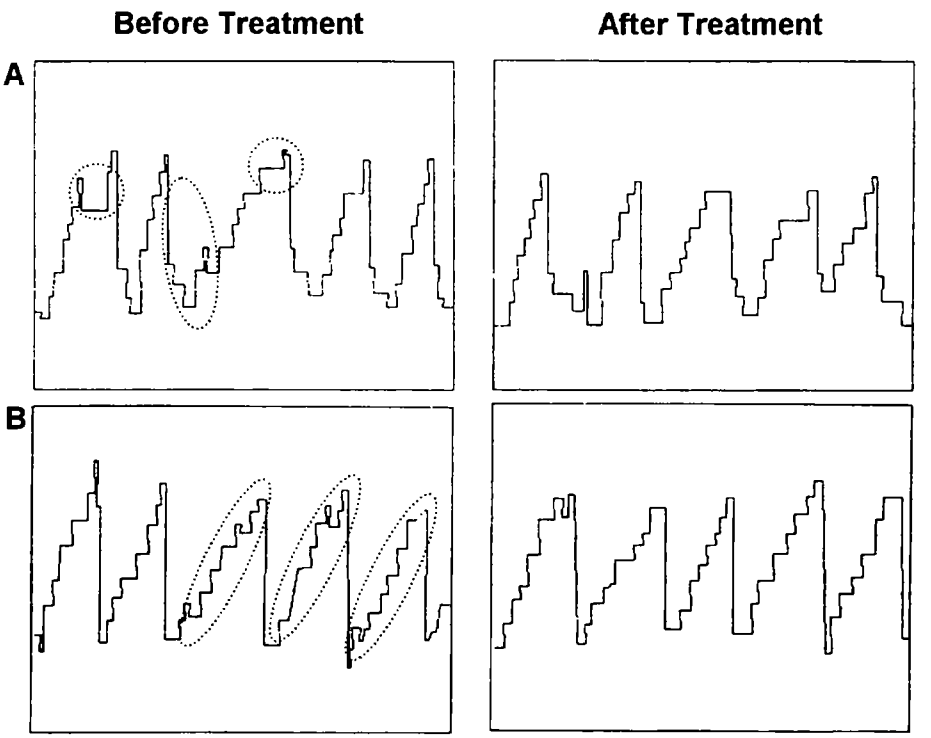

Fig. 5 Infra-red eye-movement recordings before and after treatment in patients with left- (**A**) or right-sided (**B**) unilateral homonymous hemianopia (field sparing: ~3°). Eye-movement patterns during reading of five lines are shown (x-axis: time period of recording; y-axis: horizontal extension of line from left to right). **A**: Note the prolonged fixations and regressions (ovals) as well as the interrupted return sweep (ellipse) before and the normalisation of the oculomotor pattern after treatment. **B**: Note the distorted left-to-right oculomotor reading pattern (prolonged fixations, smaller forward saccades, and regressions (ellipses)) before and its normalisation after treatment. Eye-movement recordings are adapted from Zihl (1995a).

Reading fluency is regained; reading speed increases (before treatment: 76 wpm (LH), 53 wpm (RH); after treatment: 113 wpm (LH), 96 wpm (RH) (Zihl, 1995a)) and fewer errors are made. Follow-up assessments show that these treatment effects remain stable (Kerkhoff et al., 1992; Spitzyna et al., 2007; Zihl, 1995a, 2000). After treatment, reading performance in hemianopic dyslexia is markedly improved although parafoveal vision is still lost. Re-organizing oculomotor control is decisive for making our 'optical instruments' useful once again for reading (Gassel & Williams, 1963b).

Most patients benefit from systematic oculomotor practice. Patients with right-sided parafoveal visual field loss, however, require almost twice as many training sessions as patients with a similar left-sided field loss. Even then, they still show a poorer outcome in comparison with patients with left-sided field loss (Zihl, 1995a, 2000). The differential or "asymmetrical" effect of left- and right-sided parafoveal visual field loss on rehabilitation outcome appears to be specific to reading. When scanning a visual scene there are no performance differences between left- and right-sided visual field defects as in reading (Tant et al., 2002; Zihl, 1995b, 1999). Furthermore, oculomotor scanning performance, in contrast to reading performance, is not associated with the degree of visual field sparing in such a way that the smaller the sparing, the more impaired is oculomotor scanning. Also the location of the visual defect within the visual field is much more important in determining the resulting reading impairment than the scanning impairment (Poppelreuter, 1917/1990; Zihl, 1995b). Wilbrand (1907) reported that small paracentral scotomas only posed an impediment to reading but not to exploring surroundings in his patients. Furthermore, spontaneous oculomotor adaptation to a homonymous visual field disorder in visual exploration is more likely (40%) than compensating for the reading impairment (~20%). In addition, both abilities seem to require specific training for their improvement and there appears to be no obvious transfer effect between both domains. One may speculate that the control of the oculomotor scanpath for reading is mediated by different neural networks than the scanpath for visual exploration, although both networks probably overlap (Zihl, 1995a, 1995b, 2000).

The effect of top-down text processing strategies on inter- and intraindividual variation in reading ability might be marginal in normal readers (see O'Regan, 1992). Yet, differences in factors such as self-control may be crucial when a new reading strategy has to be learnt. 'Risky' readers (who flexibly deploy fewer fixations over a larger span of words) often adopt new strategies to cope with visual field loss more quickly than 'cautious' word-by-word readers do (although possibly at the expense of omitting words or syllables).

Rehabilitation of hemianopic dyslexia depends on perceptual and oculomotor (i.e., procedural) learning processes. Learning and consolidation of new oculomotor reading strategies are top-down guided and modulated by attention. PPC function may play an important role in mediating these learning processes. Right PPC is crucial for perceptual learning and attention, and practice-related decrease of activation has been observed for the practice of visual search tasks (Walsh, Ashbridge, & Cowey, 1998; Walsh, Ellison, Ashbridge, & Cowey, 1999).

That patients with additional extensive injuries to the occipital white matter and/or to occipitoparietal regions require the largest amount of training (Zihl, 1995a, 2000) also indicates the relevance of parietal mechanisms for the rehabilitation of hemianopic dyslexia and demonstrates the importance of intact functional connections between the cortical visual areas and the areas that supposedly mediate the treatment effect.

Interestingly, patients with normal visual fields but posterior parietal damage reported difficulties in finding their way through lines of text on a page (Zihl & Hebel, 1997). Comparing this reading impairment to the reading difficulties of patients who have a similar posterior parietal involvement but an accompanying unilateral homonymous parafoveal visual field loss could illuminate the relative contributions of attentional posterior parietal and sensory striate cortex functions to reading and also to learning new oculomotor reading strategies.

Further evidence for PPC involvement stems from a recent investigation of brain representation of visually guided saccades in a small group of patients with pure striate

cortex lesions resulting in right- or left-sided homonymous hemianopia. These patients showed no impairments of visual exploration or reading. Making saccades to targets presented in the intact and compromised hemifield was associated with a bilateral activation of the frontal and parietal eye fields, albeit to a lesser degree than in normal observers. Increased activation in patients was found in the posterior parietal cortex of the unaffected hemisphere, i.e., contralateral to the side of the intact hemifield, suggesting that visual field defects after striate lesions are associated with changes in the fronto-parietal network underlying the cortical control of saccades. Whether this activation represents a neural correlate of (spontaneous and/or training-induced) oculomotor compensatory processes needs further study (Nelles et al., 2007).

Mirror reading provides further insights into the involvement of parietal and also frontal mechanisms in the rehabilitation of hemianopic dyslexia. The acquisition of mirror-reading skill in normal subjects is associated with changes in activation patterns of posterior brain regions and stronger activation in the parietal associative cortex and the frontal eye fields. After training when reading strategies have been learned successfully and become routine, a practice-related decrease of activation in prefrontal and posterior parietal areas is observed (Kassubek, Schmidtke, Kimmig, Lücking, & Greenlee, 2001; Poldrack et al., 1998; Poldrack & Gabrieli, 2001). Prefrontal cortex activity is critical for procedural learning (Beldarrain, Grafman, Pascal-Leone, & Garcia-Monco, 1999; Jueptner et al., 1997; Miller & Cohen, 2001) and the FEF in particular are involved in intentional, voluntary generated attentional and eye-movement shifts according to a rule (Heinzle et al., 2007).

Many assumptions about the underlying mechanisms of the resulting improvement in rehabilitation of hemianopic dyslexia must remain speculative without evidence from functional neuroimaging. Nevertheless, the finding that the lost parafoveal visual field region can be successfully substituted by spontaneous or training-induced oculomotor adaptation shows the functional plasticity of the visual, attentional and oculomotor systems and their underlying neural mechanisms involved in text reading. Reading eye-movements can be

controlled either from bottom-up (parafoveal visual field) or via an attentional top-down text processing strategy.

7. Synopsis: Insights from and into hemianopic dyslexia

A great deal has been learnt about hemianopic dyslexia since it was first reported by Mauthner in 1881. Studying patients with hemianopic dyslexia offers important insights into the normal reading process and its neural basis, which may be useful in informing theories and models of reading and eye-movement control. Hemianopic dyslexia is a special type of reading impairment that is caused by injury to systems subserving the bottom-up and attentional top-down control of visual information processing in the foveal and parafoveal visual field and saccadic eye-movements involved in reading. The anatomical basis of hemianopic dyslexia involves left- or right-sided injury to the striate cortex or its geniculostriate afferents compromising the representation of parafoveal vision. Yet, the critical lesion location for the severe and long-lasting reading impairments lies elsewhere. It is in the fibre pathways that reciprocally connect the visual areas of the brain to the parietal, frontal, and temporal areas, as well as to the subcortical areas involved in the control of visuospatial attention and the guidance of the scanpath in text processing.

Hemianopic dyslexia provides valuable neuropsychological insights into the neural mechanisms essential for normal reading. It shows that the visual field is not only a sensory surface or passive information intake zone but "as much a measure of the attention (...) as of the anatomical substrate" (Williams & Gassel, 1962, p. 243). Visual information processing in reading requires attentional top-down control which, together with higher-level linguistic processes, facilitates visual processing at the early stages of the reading process for word identification and eye-movement control. Such careful coordination of visual information processing, eye-movement control, visuospatial attention, and linguistic processing requires coordinated parallel processing in multiple cortical brain regions supported by large-scale neural networks.

Hemianopic dyslexia shows that parafoveal vision is crucially involved in reading although it is not absolutely essential. It is crucially involved insofar as it subserves word processing and identification and the visual guidance of reading eye-movements. Obliterating parafoveal vision, either by injury to the striate cortex or its geniculostriate afferents or by experimental masks in normal subjects impairs text processing and alters the oculomotor reading scanpath from bottom-up. Furthermore, the side and extent of the artificial or natural visual field defect determine, together with the functional demands of the writing system and the reading task *per se*, the quality and severity of the resulting reading impairments. Purely visually elicited impairments are, however, not severe or long-lasting. The guidance of reading eye-movements can be adjusted to re-establish sufficient visual information processing for reading to proceed in a regular fashion, although each fixation still only provides an incomplete view. An attentional top-down control of visual sampling can successfully 'substitute' parafoveal vision. The representation of parafoveal vision in striate and prestriate cortex may not be essential to reading in so far as its (artificial or brain injury-related) loss can be compensated for. Parafoveal visual field loss is a necessary yet not sufficient component for the emergence of hemianopic dyslexia.

Successful spontaneous and training-induced oculomotor compensation for parafoveal visual field loss in reading suggests that there is a discrepancy between involvement and absolute necessity of the cortical and subcortical areas involved in reading. This discrepancy demonstrates the functional plasticity of the visual, attentional and oculomotor systems involved in reading and "may reflect significant functional reserve and plasticity within the cortical network as a whole" (Leigh & Kennard, 2004, p. 474). Oculomotor adaptation to parafoveal visual field loss in reading requires intact attentional and oculomotor systems along with their reciprocal connections to visual areas. These systems and their inter-connections form a distributed network that subserves visuo-motor integration and the attentional top-down modulation of visual information processing which are required for reading. This network is therefore not only involved but necessary for normal reading to occur. It consists of visual cortical, parietal (esp. PPC) and frontal (esp. FEF) areas.

If, in addition to unilateral homonymous parafoveal visual field loss, the functional connections within this network, and hence the functioning of its components, are affected by brain injury, hemianopic dyslexia results. The level of functioning of this network determines the extent to which the residual visual field can be utilised via a top-down attentional strategy for word identification and guiding reading eye-movements. Depending on which network components are affected as well as which part of the visual field and how much of it is spared, hemianopic dyslexia is more or less severe and qualitatively different reading impairments result. Hemianopic dyslexia demonstrates the importance to reading of white matter pathways reciprocally connecting the foveal/parafoveal parts of V1 with the parietal, frontal, and temporal cortices and the subcortical areas involved in saccade control. Despite different contributions of parietal and frontal areas to the control of saccadic activity, both areas and their close cooperation are essential in sampling the visual world in reading. Hemianopic dyslexia may be interpreted best as a *visual-attentional-oculomotor-network disorder*.

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Chapter 2

ARE HEMIANOPIC READING AND VISUAL EXPLORATION IMPAIRMENTS VISUALLY ELICITED? NEW INSIGHTS FROM EYE-MOVEMENTS IN SIMULATED HEMIANOPIA

The three experiments presented in this chapter investigated whether the hemianopic reading and visual exploration impairments are primarily caused by the hemianopic visual field defect itself or by additional brain injury preventing efficient spontaneous oculomotor adaptation. To establish the extent to which these impairments are visually elicited, unilateral homonymous hemianopia was simulated in healthy participants, using a gaze-contingent display paradigm. Simulated hemianopia was found to induce the reading and visual exploration impairments of hemianopic patients in all participants. Over time, however, all participants showed efficient spontaneous oculomotor adaptation to the visual-sensory deficit, which improved their reading and visual exploration performance and eye-movements. Thus, although the hemianopic visual field defect may be a major component of the chronic impairments of reading and visual and exploration found in hemianopic patients, it does not seem to be their primary cause.

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1. Introduction

Unilateral homonymous hemianopia (HH) is a common functional impairment after brain damage. It is a visual field disorder caused by injury to the postchiasmatic visual pathway, which leads to loss of vision in both monocular hemifields contralateral to the side of brain injury. Posterior cerebral artery infarction is its most frequent aetiology and seldom restricted to striate cortex (Zhang, Kedar, Lynn, Newman, & Biousse, 2006; Zihl, 2000). Sufficient spontaneous recovery of the visual field occurs rarely (Zihl & Kennard, 1996). The majority of hemianopic patients show persistent and severe impairments of reading (hemianopic dyslexia) and visual exploration (Zihl, 2000, 2003). Hemianopic reading and visual exploration impairments are well-established clinical phenomena with a long history (for early descriptions, see Mauthner, 1881; Pfeifer, 1919; Poppelreuter, 1917/1990; Wilbrand, 1907).

Hemianopic dyslexia is an acquired reading disorder which is frequently associated with HH affecting parafoveal vision. Difficulties in word identification and reading eye-movement control impair the ability to read text quickly and efficiently despite intact language functions. The main behavioural feature of hemianopic dyslexia is very slow reading that is characterised by visual omission and guessing errors as well as severe alterations in the pattern of reading eye-movements. Patients show an increased number and duration of fixations and repeated fixations as well as much smaller saccadic eye-movements (e.g., Leff et al., 2000; McDonald, Spitzyna, Shillcock, Wise, & Leff, 2006; Spitzyna et al., 2007; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a, 2000). Hemianopic patients also typically show a severe impairment of visual exploration. It disturbs the ability to gain a complete overview of the visual surroundings and leads to difficulties in detecting and locating objects, avoiding obstacles and in orienting and navigating in unfamiliar surroundings. The hemianopic visual exploration impairment is distinguished by considerably increased visual search and scanning times, as well as target omissions, longer and unsystematic scanpaths, a higher number of fixations, smaller saccades and, at least in

part, longer fixation durations (e.g., Mort & Kennard, 2003; Pambakian et al., 2000; Tant, Cornelissen, Kooijman, & Brouwer, 2002; Zihl, 1995b, 1999, 2000).

Although a high degree of consensus about the characteristics of the hemianopic reading and visual exploration impairments has been reached, the causes of these impairments are, however, still unknown. It is a matter of debate whether hemianopic reading and visual exploration impairments are consequences of the hemianopic visual field defect itself, or whether they are caused by additional brain injury preventing efficient spontaneous oculomotor adaptation. Moreover, the dissociability of hemianopic reading and visual exploration impairments (Zihl, 2000) raises the question as to whether these impairments are caused by a common underlying mechanism. The visual origin of hemianopic dyslexia is supported by studies that investigate the significance of parafoveal vision for reading in normal readers; occluding the parafoveal visual field by paracentral masks induces behavioural changes in reading that correspond with the hemianopic reading impairment (Cummings & Rubin, 1992; Fine & Rubin, 1999a; Ikeda & Saida, 1978; McConkie & Rayner, 1975, 1976; Rayner & Bertera, 1979; Rayner, Inhoff, Morrison, Sowiaczek, & Bertera, 1981; Rayner, Liversedge, & White, 2006). Studies investigating the effects of a simulated hemianopic visual field defect on visual exploration in healthy individuals provide additional evidence that the visual exploration impairment associated with HH may be a consequence of the visual field loss rather than of additional brain damage (Tant et al., 2002; Zangemeister & Oechsner, 1999; Zangemeister & Utz, 2002). Yet, observations of patients showing normal reading and visual exploration performance despite visual field loss indicate that the hemianopic visual field defect may be a necessary but not sufficient condition that causes the hemianopic reading and visual exploration impairments. Very soon after brain injury, these patients seem to spontaneously adopt eye-movement strategies which allow them to efficiently compensate for their visual-sensory dysfunction (Gassel & Williams, 1963; Zihl, 2000, 2003). It has therefore been suggested that additional

lesions preventing efficient spontaneous oculomotor adaptation may be required for the hemianopic reading and visual exploration impairments to persist (Zihl, 1995a, 1995b).

As long as it is unclear whether the hemianopic reading and visual exploration impairments are caused by the visual field loss itself or by additional brain injury, and whether they are caused by a common underlying mechanism, our understanding of these functional impairments remains incomplete. Consequently, current practice of assessment and rehabilitation of visual field loss after brain injury is imperfect. Thus, investigating the causes of these functional impairments is both of theoretical but also of high clinical-practical relevance. The purpose of the experiments reported in this chapter therefore was to identify the visual components that may constitute the hemianopic reading and visual exploration impairment as well as to establish the extent to which these impairments are visually elicited. A gaze-contingent display paradigm (McConkie & Rayner, 1975; Rayner & Bertera, 1979) was used to simulate HH in healthy participants, which allows studying the behavioural changes associated with the hemianopic visual field defect that are not caused by brain injury. Experiment 1 investigated the effects of simulated HH on reading and visual exploration. In addition, it examined the effects of simulated HH on saccadic accuracy, which is regarded as an indicator of efficiency of visual exploration and is often impaired in hemianopic patients (Meienberg, Zangemeister, Rosenberg, Hoyt, & Stark, 1981; Zihl, 2000). Experiment 2 investigated whether and to what extent healthy participants spontaneously adapt to simulated HH in reading (Experiment 2a) and in visual exploration (Experiment 2b).

2. Experiment 1: The effects of simulated hemianopia on reading, visual exploration, and saccadic accuracy

2.1. Methods

Participants

For each of the three experiments (Experiments 1, 2a, 2b), a new group of naïve, healthy participants with normal or corrected-to-normal vision was tested. All participants were

native English speakers and had no reading disorders, visual disorders or any other neurological disease or psychiatric condition, and gave their informed consent in accordance with the Declaration of Helsinki and with local ethical committee approval. In Experiment 1, 17 participants (8 males, 9 females; mean age: 38.7 years (SD: 11.6); years of education: 11.2 years (SD: 3.5)) were tested in order to investigate the effects of simulated HH on reading, visual exploration, and saccadic accuracy.

Eye-movement recording and simulating hemianopia

Eye-movements were recorded using a pupil and dual Purkinje image video eye-tracker (HS-VET, Cambridge Research Systems) with a sampling frequency of 250 Hz and a spatial resolution of 0.05° of visual angle. Since previous research on reading and visual exploration in hemianopic patients is based on monocular eye-movement recordings during binocular viewing (e.g., Leff et al., 2000; McDonald, et al., 2006; Mort & Kennard, 2003; Pambakian et al., 2000; Spitzyna et al., 2007; Tant, et al., 2002; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a, 1995b, 1999, 2000), the position of the right eye was sampled under binocular viewing conditions. Prior to each recording session, the equipment was calibrated using a 16-point grid; calibration was repeated before each task and block of trials. Stimuli were presented on an Eizo FlexScan F56 monitor (100Hz, 17'', 800x600 pixels) which subtended 40° horizontally and 32° vertically. Participants were seated comfortably at a viewing distance of 38 cm with the centre of the screen at eye level. To prevent head movements, each participant's head was tightly strapped to a circular head holder that was firmly attached to a forehead- and chinrest. Ambient room illumination was 1 lux. Stimulus presentation and eye-tracking was controlled by a visual stimulus generator (Cambridge Research Systems) running custom software.

Left- and right-sided HH (LHH, RHH) was simulated with a gaze-contingent visual display paradigm which completely blanks one side of the screen relative to the current eye position, i.e., the side to the left or right of current fixation (to simulate LHH or RHH respectively) assumes the colour of the background (see Figure 1).

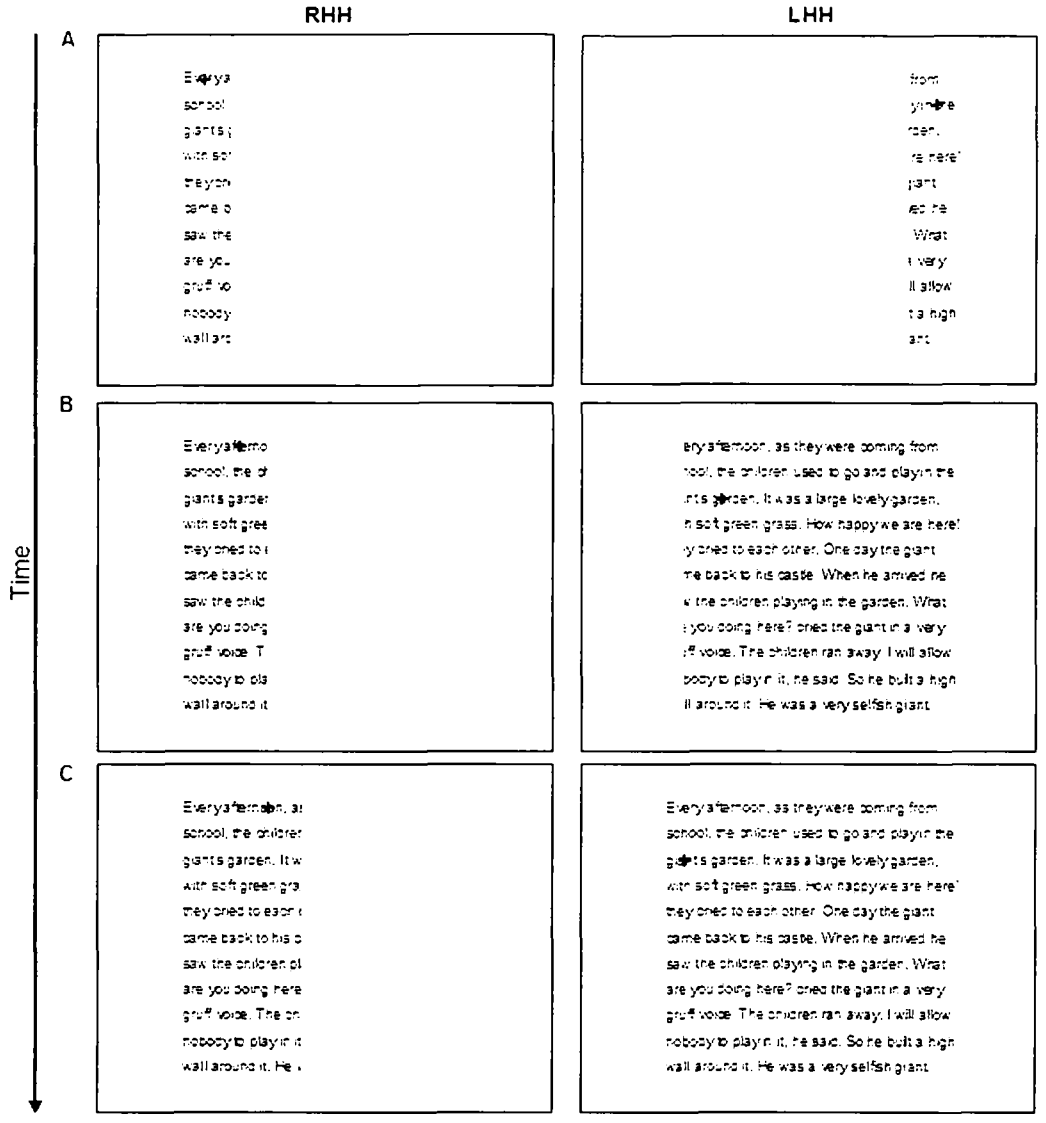

Fig. 1. Schematic illustration of right- and left-sided simulated hemianopia during reading (RHH, LHH); the gaze-contingent display paradigm blanks the side to the right or left of current fixation (visual field sparing: 1°). Potential fixation sequences are illustrated (the cross indicates potential fixation positions of a participant): RHH: reading the first line (fixating the first word (**A**), the beginning (**B**) and end of the second word (**C**)); LHH: moving the eyes from the end of the second line (fixating the last word (**A**)) to the beginning of the third line (fixating the second word due to a too short return-sweep (**B**) and fixating the first word after a corrective saccade towards the beginning of the line (**C**)).

In patients with HH after unilateral postchiasmatic damage, the foveal visual field (±0.5–1.0° to the left or right of fixation) is spared and macular sparing (±1–5°) is infrequent (Gray, Galetta, Siegal, & Schatz, 1997; Reinhard & Trauzettel-Klosinski, 2003; Zihl, 1989, 2000). Therefore, a visual field sparing of 1° was chosen for the simulated HH, i.e., between each participant's foveal eye position and the left or right border of the simulated HH 1° of

the visual field (~3 letters in the reading task) remained visible. When saccadic eye shifts landed at positions outside the registration area, the complete screen area was blanked. An update of the entire display occurred within a single frame (maximum lag: 10ms) based on current eye position (acquired at 2.5 times frame rate). For developing the simulated HH paradigm, patients with HH after brain injury were consulted in order to obtain their subjective experience of the visual field loss. To match patient's descriptions of their subjective experience, the simulated visual field defect was created so that it did not convey any visual information (blank defect with the colour of the background) rather than using textured (e.g., Rayner et al., 1981) or black masks on white background (e.g., Fine & Rubin, 1999a). This is also in line with a recent finding suggesting that a textured mask obliterating visual information to the right of fixation in reading attracts attention and leads to an attentional shift to the mask (Rayner et al., 2006), which is not the case in cerebral visual field defects.

Prior to each task and block of trials, calibration and the accuracy of the simulated visual field border were validated; a nine-point grid validation was used to assess the offset between actual and measured gaze location. Calibration and validation were repeated if the validation error was greater than 1° on average or greater than 0.5° at each point. During trials, the match between actual and measured gaze location was continuously monitored on a control display; in cases of mismatch, calibration and validation were repeated. Trials with >20% loss of eye-movement data (as a result of lid closures or saccadic eye shifts to positions outside the registration area) were not included in the analysis.

Assessment of reading and eye-movements

Materials for assessing reading performance and eye-movements during silent text reading consisted of six text passages taken from Oscar Wilde's (1931) "The selfish giant" (pp. 479-483). None of the participants had read this fairy tale before. Each text consisted of 100 words arranged in eleven, left-aligned lines. Number of characters (including spaces) was similar across the selected text passages (mean: 507.7, SD: 15.0). Letter size was 0.8°, letter

width 0.3°; spacing between letters was 0.1° and 0.4° between words. About three characters subtended 1° of visual angle. Single lines were separated vertically by 2°. Luminance of the black letters was 0.2cd/m^2, against a white background of 27cd/m^2. The texts were characterised by short sentences with a low semantic and syntactic complexity level. The difficulty level of the texts was well below the education level of the participants.

There were no differences among the selected six text passages in any of the parameters describing reading performance and eye-movements, as assessed in a control sample of 25 participants (12 males, 13 females; mean age: 19.0 years (SD: 1.2); years of education: 12.4 (SD: 0.8)). There was no significant effect of text passage (6-level within-subject factor) for reading time ($F_{(5,144)}$=0.59, p=0.707) or for any oculomotor reading measure (number and duration of fixations and repeated fixations, mean amplitude of forward and return-sweep saccades, scanpath length) (largest $F_{(5,144)}$=2.03, p=0.078; ANOVA); the maximal difference in reading time between any two of the six text passages was 2.3 s.

For assessing reading performance and eye-movements, participants were asked to read one of these texts passages silently and only once, with the goal of understanding the text's content. No further instructions were given on how to proceed. For testing comprehension and to confirm that participants had read the text, they were also asked to reiterate its content after reading, which all participants did correctly. Eye-movement recording started with the onset of text presentation and ended after the participant indicated completion of reading. A similar reading test (in German) has been found to be sensitive to changes in reading performance and related oculomotor measures during treatment of hemianopic dyslexia (Zihl, 1995a, 2000).

Reading performance was defined as the time required to read one text passage (reading time), i.e., time elapsed between reading the first and the last word of the text. For the assessment of reading eye-movements, the following global temporal and spatial oculomotor parameters were analysed: number and mean duration (ms) of fixations,

percentage of fixation repetitions (i.e., fixations at previously fixated points), number and mean amplitude (°) of forward (i.e., rightward) saccades, mean amplitude of return-sweep saccades (i.e., the mean first amplitude of eye-movements from the end to the beginning of the next line (°)) and scanpath length (i.e., the sum of all saccadic amplitudes (°)).

Assessment of visual exploration and eye-movements

For assessing visual exploration and eye-movements, irregular stimulus patterns consisting of 19, 20 or 21 black dots (diameter: 1°) on a white background were presented in randomized order. This task has been found to be sensitive to changes in oculomotor visual exploration measures during treatment in patients with HH (Zihl, 1995b, 1999, 2000). Dot luminance was 0.2cd/m^2, against a white background of 27cd/m^2. Dot patterns were created by randomly assigning the dots to any of 24 possible positions in a rectangular imaginary 6 x 4 grid (subtending 18.6° horizontally and 12.4° vertically); minimal spatial separation of any pair of adjacent dots was 6°. Each dot pattern was preceded by the presentation of a fixation spot (0.5°) displayed in the centre of the screen which, once fixated, initiated the trial. Participants were asked to silently count the presented dots as accurately and as quickly as possible, and to report their number. This test is similar to the dot cancellation test (Lezak, Howieson, & Loring, 2004) but did not include feedback on which dots had already been counted. No instruction was given on the number of dots or how to proceed with counting or search; participants received no feedback on the number of counted dots. Eye-movement recording started with the onset of the dot pattern and was ended when the participant indicated completion of dot counting and reported their number.

Visual exploration performance was defined as visual exploration time (the time required to perform one trial) and number of errors (all errors committed were omission errors). For the assessment of visual exploration eye-movements, the following global temporal and spatial oculomotor parameters were analysed: number and mean duration (ms) of fixations, mean saccadic amplitude (°) and scanpath length (i.e., the sum of all saccadic amplitudes (°)). In addition, directional and hemispace analyses were performed (Tant et al.,

2002; Zihl, 1995b). Number and mean amplitude (°) of left- and rightward saccades (directional analysis) as well as number and mean duration (ms) of fixations spent in left and right hemispace defined with respect to the centre of the screen (hemispace analysis) were analysed.

Assessment of saccadic accuracy

For assessing the accuracy of intentional saccadic eye-movements to visual targets, two simultaneously presented black dots (diameter: 1°) were used; one was presented 10° to the left, the other 10° to the right of the screen's centre in the horizontal plane (distance between dots: 20°). Dot luminance was 0.2cd/m^2 against a white background of 27cd/m^2. The simultaneous presentation of the two dots was preceded by a fixation dot (0.5°) in the centre of the screen. Participants were asked to alternate their gaze back and forth between the two simultaneously presented dots as accurately as possible. They were informed that the target-dot located in their blind hemifield is presented at the same distance from the centre in the horizontal plane as the target-dot located in their seeing hemifield (Zihl, 2000; Zihl & Hebel, 1997). Eye-movement recording started with the onset of the display and ended when the participant had performed at least 10 saccadic eye shifts.

Saccadic accuracy was defined as mean saccadic gain, i.e., the quotient of initial saccadic amplitude and target distance for left- and rightward saccades. A saccadic gain of 1 indicates perfect correspondence between target and eye position. Under- or over-shooting of the target is referred to as saccadic dysmetria, i.e., hypo- or hypermetria, respectively. Accuracy of each saccade was considered as normal when saccadic gain was between 0.88 and 1.06, hypometric when the gain was <0.88 and as hypermetric when the gain was >1.06. These cut-off values were derived from the average gain ± one standard deviation of participants' left- and rightward initial saccades in the non-simulation condition (mean: 0.97°, SD: 0.09) (Zihl, 2000). For each participant, the mean amplitude (°) and saccadic gain of initial left- and rightward saccades as well as frequency of normal, hypo- and hypermetric initial left- and rightward saccades were analysed.

Procedure

All participants performed each task, i.e., reading (1 text passage out of 3), visual exploration (10 trials) and saccadic accuracy (3 trials) with simulated LHH, RHH and in a normal viewing condition, i.e., without any simulated HH (N). Task performance in the normal viewing condition as well as reports on each participant's subjective experience with simulated HH was obtained at the end of the experiment. The sequence of simulation conditions (starting with LHH or RHH), tasks and text passages used for reading assessment were counterbalanced across participants to eliminate order effects. To avoid adaptation and practice effects, the same simulation condition (LHH or RHH) was never imposed in succession and the same task was never performed consecutively; before performing the same task again in a different simulation condition, the two other tasks had to be performed.

Data analyses

For testing the effects of simulated HH on reading, visual exploration and saccadic accuracy, a repeated measures ANOVA with simulation condition (LHH, RHH, N) as a within-subject factor was performed for each task. For hemispace and directional analyses of the visual exploration data, repeated measures ANOVAs with simulation condition (LHH, RHH, N) and space/direction (left, right) as within-subject factors were performed. Where sphericity assumptions were violated as assessed by Mauchly's W test, the Greenhouse-Geisser correction to the degrees of freedom was applied. Post-hoc paired comparisons between simulation conditions and space/directions were performed using repeated measures t-tests. As multiple tests were carried out, the significance level was adjusted using a Bonferroni correction to an alpha-level of 0.05 for multiple comparisons. 4.3% of trials were excluded from the visual exploration data analyses.

2.2. Results

The effect of simulated hemianopia on reading

Reading and eye-movements of healthy participants were adversely affected by simulated HH (Table 1), as indicated by a significant effect of simulation condition for reading time

and all oculomotor parameters (smallest $F_{(1.2,19.3)}$=4.49, p=0.041). Reading with simulated LHH or RHH was characterised by significantly longer reading times, a higher number and duration of fixations and repeated fixations, many more and smaller forward saccades, a smaller return-sweep and a prolonged scanpath when compared with normal performance. Reading performance also differed significantly between LHH and RHH, except for the rate of fixation repetitions and the return-sweep amplitude. Reading with RHH was much more impaired than reading with LHH.

Table 1 Reading performance and related oculomotor parameters in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [mean (SD, range)].

	LHH	RHH	N	N–LHH	N–RHH	LHH–RHH
Reading time (s)	34.1 (15.2, 14.6–79.4)	57.7 (23.9, 25.5–115.3.0)	19.0 (4.2, 14.1–29.0)	*	*	*
Total fixations						
Number	111.1 (32.7, 57.0–173.0)	155.1 (36.5, 73.0–233.0)	79.8 (16.5, 54.0–122.0)	*	*	*
Duration (ms)	244 (58.6, 181–401)	316 (105.4, 192–631)	180 (17.4, 153–225)	*	*	*
Repeated fixations (%)	18.5 (9.5, 3.5–38.2)	20.3 (10.0, 3.1–40.4)	12.3 (5.5, 4.6–25.4)	*	*	n.s.
Forward saccades						
Number	70.4 (15.8, 43.0–108.0)	101.5 (30.0, 47.0–154.0)	53.1 (9.6, 39.0–71.0)	*	*	*
Amplitude (°)	3.8 (0.6, 3.0–5.1)	3.3 (1.3, 1.9–5.4)	4.3 (0.6, 3.3–5.4)	*	*	p=0.031
Return-sweep amplitude (°)	15.8 (1.9, 11.8–19.1)	15.4 (2.4, 10.0–18.5)	17.2 (1.4, 14.7–19.7)	*	*	n.s.
Scanpath length (°)	529.0 (84.3, 401.6–667.9)	604.2 (132.1, 437.2–860.3)	457.0 (50.2, 373.6–544.8)	*	*	p=0.028

Statistical comparisons were made between LHH, RHH, and N (one-tailed dependent samples t-tests). n.s. indicates non-significant comparisons. * $p<0.017$ (α_{corr}); p-values are given for marginally significant results.

The effect of simulated hemianopia on visual exploration

Simulated HH also had a detrimental effect on visual exploration and eye-movements of healthy participants (Table 2), as indicated by a significant effect of simulation condition for

visual exploration time, number of errors, and for the majority of oculomotor parameters (smallest $F_{(2,32)}=3.85$, $p=0.032$). Visual exploration with simulated LHH and RHH was characterised by significantly longer visual exploration times, more errors, a higher number and duration of fixations, smaller saccades (significant for RHH only), and a prolonged scanpath. There were no significant differences between LHH and RHH for these performance measures.

Table 2 Visual exploration performance and related oculomotor parameters in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [mean (SD, range)].

	LHH	RHH	N	N–LHH	N–RHH	LHH–RHH
Visual exploration time (s)	15.5 (5.3, 8.5–28.8)	18.6 (15.6, 10.7–74.5)	8.7 (1.8, 5.4–12.4)	*	*	n.s.
Number of errors	0.7 (0.7, 0–2.3)	0.8 (0.6, 0–2.4)	0.1 (0.1, 0–0.3)	*	*	n.s.
Total fixations						
Number	27.3 (9.4, 16.1–46.7)	31.8 (17.7, 17.5–76.2)	18.7 (3.8, 14.1–26.0)	*	*	n.s.
Duration (ms)	452 (70.4, 319–591)	439 (114.3, 267–752)	356 (90.3, 234–609)	*	*	n.s.
Saccadic amplitude (°)	4.2 (0.9, 2.7–7.1)	4.2 (0.9, 2.3–6.5)	4.5 (0.6, 3.1–5.7)	n.s.	*	n.s.
Scanpath length (°)	118.6 (58.7, 54.1–293.5)	128.4 (67.6, 60.7–321.7)	85.2 23.8, 45.2–120.3)	*	*	n.s.
Right hemispace fixations						
Number	12.7 (5.3, 6.8–22.7)	18.3 (12.0, 10.0–50.1)	8.7 (2.2, 5.6–12.4)	*	*	p=0.027
Duration (ms)	449 (94.8, 318–583)	457 (141.4, 284–877)	400 (113.1, 236–733)	p=0.035	p=0.032	n.s.
Left hemispace fixations						
Number	14.6 (5.8, 9.2–30.1)	13.5 (6.2, 6.8–28.0)	10.1 (2.3, 7.6–17.1)	*	*	n.s.
Duration (ms)	474 (104.3, 320–719)	436 (131.1, 255–750)	330 (83.3, 232–528)	*	*	n.s.
Rightward saccades						
Number	13.0 (5.3, 3.9–27.3)	16.9 (11.1, 4.4–45.9)	11.4 (2.3, 8.3–18.4)	n.s.	*	n.s.
Amplitude (°)	4.2 (0.8, 2.7–6.1)	4.7 (1.6, 2.1–9.2)	4.5 (0.8, 3.0–5.9)	n.s.	n.s.	p=0.062
Leftward saccades						
Number	14.3 (7.5, 3.3–28.1)	14.9 (8.0, 6.2–37.0)	7.3 (3.3, 3.4–15.4)	*	*	n.s.
Amplitude (°)	4.8 (1.7, 2.7–9.2)	4.0 (0.8, 2.9–5.6)	4.8 (0.7, 3.3–6.1)	n.s.	*	p=0.027

Statistical comparisons were made between LHH, RHH, and N (one-tailed dependent samples t-tests). n.s. indicates non-significant comparisons. * $p<0.017$ (α_{corr}); p-values are given for marginally significant results.

Although there was no significant effect of simulation condition for saccadic amplitude, number and amplitude of left- and rightward saccades and for duration of right-hemispace fixations (largest $F_{(1.3,20.6)}$=3.65, p=0.061), hemispace and directional analyses revealed a significant interaction between simulation condition and hemispace/direction for fixation number and saccadic amplitude (smaller $F_{(2,32)}$=4.49, p=0.019). During visual exploration with RHH, significantly more fixations were spent in right than in left hemispace, and rightward saccadic amplitudes were significantly larger than leftward amplitudes (smaller $t_{(16)}$= –2.44, p=0.014, one-tailed). In LHH, leftward saccadic amplitudes were marginally larger than rightward ($t_{(16)}$=1.66, p=0.059, one-tailed). Visual exploration with RHH was associated with the highest number and duration of right-hemispace fixations and more and larger rightward saccades whereas visual exploration with LHH was associated with the highest number and duration of left-hemispace fixations. Eye-movement patterns during visual exploration with LHH and RHH were both distinguished by a higher number of leftward saccades than in the normal viewing condition (Table 2).

The effect of simulated hemianopia on saccadic accuracy

Saccadic accuracy of healthy participants was also affected by simulated HH (Table 3), as indicated by a significant effect of simulation condition for the majority of saccadic accuracy measures (smallest $F_{(2,30)}$=3.41, p=0.046). The amplitude and gain of initial left- and rightward saccades was smaller when confronted with simulated LHH or RHH than in the normal viewing condition (yet, the LHH–N difference for leftward saccades and the RHH–N difference for rightward saccades were only marginally significant). Although there was no significant effect of simulation condition for the frequency of hypermetric left- and rightward saccades (larger $F_{(1.3,21.3)}$=2.37, p=0.132), the frequency of hypometric left- and rightward saccades was significantly higher, and that of normal saccades lower, when confronted with LHH and RHH. There were no significant differences between LHH and RHH (except for the frequency of normal rightward saccades that was lower with RHH).

Table 3 Saccadic accuracy in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [mean (SD, range)].

	LHH	RHH	N	N–LHH	N–RHH	LHH–RHH
Initial rightward saccades						
Amplitude (°)	18.7 (1.0, 16.0–20.4)	18.6 (1.4, 14.3–20.6)	19.3 (0.7, 17.8–20.5)	*	p=0.034	n.s.
Saccadic gain	0.97 (0.05, 0.83–1.05)	0.96 (0.07, 0.74–1.06)	1.00 (0.04, 0.92–1.06)	*	p=0.034	n.s.
Normal saccades (%)	81.6 (20.1, 22.4–100.0)	64.8 (24.8, 4.8–95.2)	86.0 (15.5, 57.9–100.0)	n.s.	*	*
Hypometric saccades (%)	10.5 (17.3, 0–72.9)	18.6 (22.4, 0–95.2)	2.5 (7.9, 0–32.5)	*	*	n.s.
Hypermetric saccades (%)	7.9 (11.5, 0–41.7)	16.6 (20.4, 0–63.5)	11.5 (13.7, 0–40.0)	n.s.	n.s.	n.s.
Initial leftward saccades						
Amplitude (°)	18.0 (1.1, 15.6–19.6)	17.7 (1.0, 16.2–19.3)	18.5 (0.7, 16.9–19.4)	p=0.059	*	n.s.
Saccadic gain	0.93 (0.06, 0.80–1.01)	0.91 (0.05, 0.83–0.99)	0.95 (0.04, 0.87–1.00)	p=0.059	*	n.s.
Normal saccades (%)	72.5 (17.0, 33.3–90.7)	75.0 (14.9, 47.6–97.0)	86.1 (12.0, 61.1–100.0)	*	*	n.s.
Hypometric saccades (%)	20.0 (16.0, 0–57.4)	20.0 (12.8, 3.0–42.1)	7.3 (7.0, 0–23.1)	*	*	n.s.
Hypermetric saccades (%)	7.5 (9.2, 0–30.4)	4.9 (7.2, 0–23.3)	6.7 (10.9, 0–34.1)	n.s.	n.s.	n.s.

Statistical comparisons were made between LHH, RHH, and N (one-tailed dependent samples t-tests). n.s. indicates non-significant comparisons. * $p<0.017$ (α_{corr}); p-values are given for marginally significant results.

Subjective reports

Participants' reports on the effects of simulated HH on reading were in close agreement with the objective test results (for a selection of representative quotes, see Table 4).

Table 4 Subjective reports on the effects of simulated HH on reading, visual exploration and saccadic accuracy (selection of representative verbatim quotes).

Reading
- "The text consisted of half-words and reading was hesitant."
- "It was very difficult to make an eye-movement to the next word that was always covered by the visual defect."
- "It was extremely difficult to concentrate on moving the eyes and understanding text at the same time."
- "Reading with left-sided blindness was easier than with right-sided blindness because as soon as one knows where the lines begin sweeping the eyes back becomes less difficult."
Visual exploration
- "One could never be certain whether one had missed dots or not whereas missing a word instantly resulted in comprehension difficulties."
- "Eye-movements don't have to be as precise as in reading because you don't have to fixate each dot whereas in reading each word has to be fixated for understanding the text."
Saccadic accuracy
- "Although one could not see the dot on the side of the simulated HH, its location was predictable after performing a few gaze shifts."

All participants reported severe impairments of reading, visual exploration, and saccadic accuracy when confronted with simulated HH. They found reading with simulated HH more difficult than visual exploration (except for three participants). Reading with simulated RHH was more difficult than reading with simulated LHH, yet, participants experienced no such differential effects in the visual exploration and saccadic accuracy task. Reading with simulated HH was described as extremely slow, laborious and fatiguing, and participants reported that they missed syllables and words on the side of the simulated HH. RHH greatly impaired the ability to move the eyes smoothly along each line of text whereas LHH impaired the ability to find the beginning of the new line. During visual exploration, participants experienced difficulties in finding the way through the dots without losing their place; concentrating on moving the eyes and keeping count at the same time was described as very difficult. Participants considered the effect of simulated HH on saccadic accuracy to be minor.

2.3. Discussion

The effect of simulated hemianopia on reading

The main effect of simulated HH on reading performance was to induce a pronounced increase in reading time, which was paralleled by a severe alteration of the oculomotor reading pattern. Simulated HH led to a considerable increase in number and duration of fixations and repeated fixations. The decrease in forward and return-sweep saccadic amplitude and the consequent increase in number of forward saccades further contributed to the reduction in reading performance. Simulated HH seemed to provoke an inefficient oculomotor text processing strategy, which was also reflected by significantly prolonged and disorganised scanpaths. The side of the simulated visual field defect determined the severity of the resulting reading impairment. Reading a text passage with simulated RH was three times longer than under normal viewing conditions whereas it required only twice as much time with simulated LHH. The oculomotor reading patterns associated with simulated RHH were distinguished by a much higher number and duration of fixations, smaller and many more saccades and a much longer scanpath than those associated with simulated LHH; only the rate of repeated fixations was equally affected by simulated LHH and RHH. These observations replicate those obtained in hemianopic patients with hemianopic dyslexia (e.g., Leff et al., 2000; McDonald et al., 2006; Spitzyna et al., 2007; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a, 2000) and are consistent with prior studies using gaze-contingent display paradigms to examine reading without parafoveal vision in healthy people (Cummings & Rubin, 1992; Fine & Rubin, 1999a; Ikeda & Saida, 1978; McConkie & Rayner, 1975, 1976; Rayner & Bertera, 1979; Rayner et al., 1981; Rayner, Liversedge, & White, 2006). Moreover, subjective reports are also in accordance with those of hemianopic patients (Kerkhoff, Münßinger, Eberle-Strauss, & Stögerer, 1992; Kerkhoff, Schaub, & Zihl, 1990; Zihl, 2000). Thus, these findings suggest that simulated HH induces the hemianopic reading impairment in healthy participants.

Yet, the observation that simulated LHH and RHH led to a similar decrease of the return-sweep amplitude departs from evidence obtained from hemianopic patients showing that only left-sided visual field defects are associated with smaller return-sweep saccades (Mackensen, 1962; Zihl, 1995a). Inter-individual differences regarding the impact of simulated HH on the return-sweep, as indicated by a large variation in individual return-sweep amplitudes (range: 11.8–19.1), may account for this inconsistent finding. One may speculate that, at least in some participants, the return-sweep might have quickly improved after reading a few lines. The fixed horizontal position of the return-sweep's saccadic target, i.e., the first word of the next line, may have alleviated the adverse effects of simulated LHH on the visual guidance of the return-sweep. This has been reported for some patients with LHH after brain injury (Gassel & Williams, 1963), and is consistent with participants' subjective reports.

The effect of simulated hemianopia on visual exploration

Simulated HH also had a profound effect on visual exploration. It led to elevated visual exploration times and a higher number of errors, which were paralleled by alterations of the oculomotor visual exploration pattern. Exploring and counting the presented dots with simulated LHH or RHH required twice as much time as under normal viewing conditions, and participants made more errors in counting the dots. Simulated HH induced an inefficient and unsystematic oculomotor scanpath for exploring and processing the visual information in the visual exploration task, as indicated by the increase in number and duration of fixations as well as in scanpath length. Simulated HH also affected saccadic amplitudes, albeit to a much lesser degree. Unlike in reading, there were no performance differences between simulated LHH and RHH. The side of the simulated visual field defect only seemed to determine the horizontal fixation distribution, i.e., whether more and longer fixations are spent in left or right hemispace, as well as the properties of directional oculomotor measures, i.e., whether more left- or rightward saccades are being made. These observations are consistent with those obtained in hemianopic patients (Gassel & Williams, 1963; Ishiai,

Furukawa, & Tsukagoshi, 1987; Meienberg et al., 1981; Mort & Kennard, 2003; Pambakian, Mannan, Hodgson, & Kennard, 2004; Pambakian et al., 2000; Tant et al., 2002; Zihl, 1995b, 1999) as well as with studies dealing with visual exploration in simulated and real HH (Tant et al., 2002; Zangemeister & Oechsner, 1999; Zangemeister & Utz, 2002). Furthermore, subjective reports are also in accordance with those of hemianopic patients (Zihl, 1995b, 2000). Thus, these findings suggest that simulated HH also induces the hemianopic visual exploration impairment in healthy participants.

Yet, contrary to the common observation in hemianopic patients that saccades directed to the affected hemifield are smaller (hypometric) than those of saccades to the unaffected field (Ishiai et al., 1987; Meienberg et al., 1981; Tant et al., 2002; Zihl, 1995b, 1999), simulated HH resulted in participants making *larger* (hypermetric or overshooting) saccades in the direction of the affected hemifield. This discrepancy may be explained by inter-individual differences regarding the impact of simulated HH on visual exploration. Large variations in individual saccadic amplitudes to the right during visual exploration with simulated RHH (range: 2.1–9.2) and in those to the left during visual exploration with simulated LHH (range: 2.7–9.2) suggest that some participants quickly have adopted an efficient oculomotor strategy to compensate for simulated HH by making large saccades into the affected hemifield while others have not.

The effect of simulated hemianopia on saccadic accuracy

Saccadic accuracy was also affected by simulated HH, albeit to a lesser extent than reading and visual exploration. Simulated HH induced saccadic dysmetria in healthy participants while they performed voluntary horizontal saccadic eye-movements to visual targets, leading to a reduction in saccadic accuracy. When confronted with simulated LHH or RHH, participants showed hypometric saccades in the direction of their affected hemifield, i.e., participants' saccades undershoot the position of visual targets located in their blind hemifield whereas, during normal viewing, participants made only few hypometric saccades. As in visual exploration, the side of simulated HH did not determine the severity of saccadic

dysmetria. These observations are in accordance with reports on saccadic dysmetria in hemianopic patients (Meienberg et al., 1981; Schoepf & Zangemeister, 1993; Zangemeister, Oechsner, & Freska, 1995; Zangemeister & Utz, 2002; Zihl, 2000) and replicate a recent study that investigated saccadic accuracy in simulated HH (Zangemeister & Utz, 2002).

Yet, the saccadic accuracy impairment seemed to be less pronounced in simulated HH than in hemianopic patients. Group means indicate that hypometric saccades to the affected hemifield were less frequent and normal saccades more frequent in simulated HH than in real HH (hypometria: ~20% vs. ~45%, normal saccades: ~67% vs. 30%, respectively) (Zihl, 2000). This inconsistent finding may be accounted for by inter-individual differences in the impact of simulated HH. The large variation in the frequency of hypometric saccades to the affected hemifield (range: 0–95.2%), together with participants' reports, suggest that some participants quickly made use of the fixed target positions to accurately guide predictive saccades to the visual targets (Zangemeister & Utz, 2002).

3. Experiment 2: Spontaneous oculomotor adaptation to simulated hemianopia in reading and visual exploration

To determine whether and to what extent healthy participants spontaneously adapt to simulated HH in reading and visual exploration, two further experiments were conducted that investigated the effect of uninstructed reading practice (Experiment 2a) and visual exploration practice (Experiment 2b) on reading and visual exploration with simulated HH, respectively.

3.1. Methods

Participants

In Experiment 2a, a group of 12 participants (3 males, 9 females; mean age: 19.4 years (SD: 1.3); years of education: 12.6 years (SD: 0.8)) was tested for investigating spontaneous oculomotor adaptation to simulated HH in reading. In Experiment 2b, a new group of 13 participants (3 males, 10 females; mean age: 18.7 years (SD: 0.9); years of education: 12.2

years (SD: 0.6)) was tested for investigating spontaneous oculomotor adaptation in visual exploration.

Eye-movement recording, simulating hemianopia, and the assessment of reading and visual exploration

Methods for eye-movement recording, simulating HH and for assessing reading and visual exploration performance were identical to those used in Experiment 1.

Procedure

The procedures of Experiments 2a and 2b were identical. In Experiment 2a, participants performed two reading practice sessions: one session with simulated LHH, one with RHH (time spent practicing reading was ~15 min. in each case). The sequence of simulation conditions, i.e., starting with LHH or RHH, was counterbalanced. Reading performance and eye-movements (one text passage out of four) were assessed before and after the LHH-practice session and before and after the RHH-practice session. Between sessions, i.e., after the first post-practice assessment, a short break of 10 minutes was given. Task performance without any simulated HH (N) as well as each participant's subjective experience was obtained at the end of the experiment. In Experiment 2b, participants performed two visual exploration practice sessions: one session with simulated LHH, one with RHH (time spent practicing visual exploration was ~15 min. in each case). Visual exploration performance and eye-movements (5 trials) were assessed before and after the LHH- and RHH-practice session.

Materials for the reading practice sessions (Experiment 2a) consisted of two sets of ten text passages taken from Michael Ende's (1974) "The grey gentlemen"; the text sets were counterbalanced between LHH- and RHH-practice sessions. None of the participants had read this novel before. Characteristics and presentation mode of the practice text passages were identical to those of the text passages used for the assessment of reading performance. During a practice session, participants were asked to read 10 consecutively presented texts. They were asked to read each text silently and only once, with the goal of understanding the

text's content. No further instructions were given on how to proceed. For testing comprehension and to provide evidence that participants had read each text, participants were asked to reiterate its content immediately after reading the text, which all participants did correctly. The practice session gave participants the opportunity to learn how to read with a simulated HH without specific advice.

Materials for visual exploration practice sessions (Experiment 2b) consisted of 2 sets of 30 trials of the visual exploration task used for assessing visual exploration performance. During a practice session, patients were asked to silently count the dots of each of the 30 consecutively presented stimulus patterns as accurately and quickly as possible and to report the number of counted dots. No instruction was given on the number of dots or how to proceed with counting or searching; participants received no feedback on the number of counted dots. The practice session gave participants the opportunity to learn how to explore abstract patterns with a simulated HH without specific advice.

In order to disentangle the effects of adaptation to simulated HH from performance changes due to mere practice effects, a new group of six participants (6 females; mean age: 18.8 (SD: 0.8); all had 12 years of education) performed the same experimental protocol without any simulated HH in Experiment 2a (control condition). The control sample in Experiment 2b consisted of five participants (1 male, 4 females; mean age: 18.6 (SD: 0.5); all with 12 years of education).

Data analyses

For testing the effects of simulated HH on pre- and post-practice reading (Experiment 2a) and visual exploration performance (Experiment 2b), the same analyses as in Experiment 1 were conducted. For testing the effects of practice, a repeated measures ANOVA with simulation condition (LHH, RHH) and time (pre-, post-practice) as a within-subject factors was performed for both experiments. Post-hoc paired comparisons between simulation conditions and time points were performed using repeated measures t-tests. Corrections for violations of sphericity assumptions and multiple comparisons were identical to those used

in Experiment 1. In the control samples, Friedman nonparametric analyses of variance were used to test for overall effects of time (pre-, post-practice1, pre-, post-practice2, N-condition) because of the small sample size. Post-hoc paired comparisons were performed using Wilcoxon tests (two-tailed, $p<0.05$, Bonferroni-correction). In Experiment 2b, 4.3% of trials were excluded.

3.2. Results

Reading and visual exploration with simulated hemianopia before practice

The effects of simulated HH on reading before practice (Experiment 2a) were identical to those found in Experiment 1 (Tables 5, 6), as indicated by a significant effect of simulation condition (LHH, RHH, N) for reading time and all oculomotor parameters (smallest $F_{(2,22)}=8.57$, $p=0.002$). In addition, significant differences between simulation conditions for the amplitude of return-sweep were obtained; reading with simulated LHH was characterised by the smallest return-sweeps.

Table 5 Pre- and post-practice reading performance and related oculomotor measures in left- and right-sided simulated hemianopia (LHH, RHH) in comparison with the normal viewing condition (N) [mean (SD, range)].

	LHH		RHH		N
	Pre	Post	Pre	Post	
Reading time (s)	32.4	20.7	63.8	35.6	16.9
	(12.3, 12.7–56.5)	(5.5, 11.2–27.4)	(30.8, 43.2–156.3)	(13.4, 22.8–63.1)	(4.4, 9.9–26.1)
Total fixations					
Number	106.4	80.9	164.8	127.8	70.9
	(40.5, 56.0–210.0)	(19.5, 54.0–111.0)	(71.7, 100.0–380.0)	(48.5, 84.0–241.0)	(21.6, 50.0–130.0)
Duration (ms)	254	214	320	234	192
	(49.6, 186–347)	(37.0, 164–274)	(50.5, 263–431)	(36.9, 177–287)	(25.2, 149–245)
Repeated fixations	22.3	13.6	22.9	16.4	11.8
(%)	(10.8, 5.4–48.1)	(6.4, 5.6–23.6)	(10.3, 4.4–39.2)	(8.8, 2.7–28.6)	(6.8, 3.5–23.1)
Forward saccades					
Number	63.5	50.9	110.9	84.5	48.5
	(19.2, 34.0–98.0)	(13.2, 35.0–72.0)	(42.4, 53.0–211.0)	(32.8, 51.0–150.0)	(13.7, 29.0–85.0)
Amplitude (°)	4.0	4.4	2.8	3.5	4.4
	(1.0, 2.5–5.5)	(0.9, 3.2–5.6)	(1.0, 1.7–4.8)	(1.0, 1.9–5.3)	(0.8, 3.3–5.8)
Return-sweep	14.2	16.6	16.7	17.5	17.1
amplitude (°)	(1.8, 11.0–17.0)	(1.7, 13.8–20.2)	(1.6, 14.6–20.1)	(1.6, 15.4–20.5)	(1.8, 13.9–19.6)
Scanpath length (°)	483.7	410.2	586.8	503.8	403.6
	(82.4, 369.2–680.2)	(48.9, 283.4–459.1)	(119.1, 460.1–918.6)	(88.6, 373.9–745.6)	(67.9, 307.2–540.5)

Table 6 Dependent samples t-tests (one-tailed) for analysing mean differences in reading performance and oculomotor measures between left- and right-sided simulated hemianopia (LHH, RHH) and the normal viewing condition (N) before and after practice (pre, post).

	N–LHH		N–RHH		LHH–RHH	
	Pre	Post	Pre	Post	Pre	Post
Reading time (s)	*	*	*	*	*	*
Total fixations						
Number	*	0.049	*	*	*	*
Duration (ms)	*	0.038	*	*	*	n.s.
Repeated fixations (%)	*	n.s.	*	0.045	n.s.	n.s.
Forward saccades						
Number	*	n.s.	*	*	*	*
Amplitude (°)	n.s.	n.s.	*	*	*	0.021
Return-sweep amplitude (°)	*	n.s.	n.s.	n.s.	*	n.s.
Scanpath length (°)	*	n.s.	*	*	*	*

n.s. indicates non-significant comparisons. * $p<0.017$ (α_{corr}); p-values are given for marginally significant results.

The effects of simulated HH on visual exploration before practice (Experiment 2b) were also identical to those found in Experiment 1 (Tables 7, 8), as indicated by a significant effect of simulation condition for visual exploration time, number of errors, and for all oculomotor parameters (smallest $F_{(2,24)}=3.56$, $p=0.044$); consistent with Experiment 1, there was no significant effect for overall, left- and rightward saccadic amplitude (largest $F_{(2,24)}=2.17$, $p=0.136$). The results of the directional and hemispace analyses were also replicated; although only the interaction between simulation condition and direction for number of saccades reached statistical significance ($F_{(1.2,14.3)}=11.38$, $p=0.003$), post-hoc comparisons revealed that visual exploration with simulated RHH was associated not only with significantly more right- than leftward saccades but also with more right- than left-hemispace fixations (vice versa for LHH-performance; smallest $t_{(12)}= -2.60$, $p=0.012$; one-tailed).

Table 7 Pre- and post-practice visual exploration performance and related oculomotor measures in left- and right-sided simulated hemianopia (LHH, RHH) in comparison with the normal viewing condition (N) [mean (SD, range)].

	LHH		RHH		N
	Pre	Post	Pre	Post	
Visual exploration	12.0	9.6	13.6	9.4	6.8
time (s)	(2.9, 6.9–13.8)	(2.1, 6.1–11.8)	(2.7, 8.8–18.5)	(1.5, 6.9–11.9)	(1.1, 4.9–8.2)
Number of errors	0.6	0.1	0.8	0.1	0.2
	(0.6, 0–1.8)	(0.1, 0–0.4)	(0.8, 0–2.4)	(0.1, 0–0.3)	(0.1, 0–0.4)
Total fixations					
Number	24.0	19.8	26.8	21.1	16.1
	(6.5, 14.6–36.3)	(5.3, 9.2–28.4)	(8.0, 15.0–47.6)	(4.3, 14.8–28.0)	(3.0, 10.6–20.8)
Duration (ms)	463	407	449	381	361
	(100.6, 349–699)	(113.6, 265–666)	(116.2, 317–749)	(117.8, 252–719)	(60.1, 299–481)
Saccadic amplitude	3.9	3.5	3.7	3.5	4.0
(°)	(0.8, 2.4–5.2)	(0.8, 2.0–5.3)	(0.9, 2.1–4.8)	(0.9, 2.2–5.5)	(0.5, 2.6–4.6)
Scanpath length (°)	89.4	73.2	95.5	74.0	64.0
	(29.2, 51.4–155.8)	(29.6, 30.7–132.0)	(32.0, 45.2–168.9)	(21.0, 42.4–117.3)	(14.5, 37.2–91.8)
Right hemispace fixations					
Number	11.1	10.0	13.6	13.0	8.6
	(3.4, 7.8–17.0)	(4.4, 5.0–23.0)	(3.6, 7.0–19.7)	(4.6, 6.4–24.0)	(1.9, 5.0–11.6)
Duration (ms)	468	411	461	382	385
	(86.0, 360–583)	(94.6, 244–580)	(135.3, 307–793)	(109.1, 232–658)	(91.8, 311–643)
Left hemispace fixations					
Number	11.4	11.2	10.9	10.3	7.6
	(3.4, 6.8–16.2)	(3.9, 4.2–17.6)	(2.8, 4.4–14.7)	(4.4, 6.0–23.6)	(2.2, 5.0–12.2)
Duration (ms)	446	433	444	387	350
	(145.6, 289–778)	(154.4, 284–852)	(103.0, 331–708)	(142.4, 260–796)	(58.8, 273–430)
Rightward saccades					
Number	8.7	7.6	16.8	15.5	10.9
	(3.7, 4.0–17.0)	(5.8, 3.0–25.3)	(6.2, 4.0–24.3)	(6.9, 3.6–26.4)	(3.1, 5.4–16.0)
Amplitude (°)	3.9	3.7	3.7	3.9	4.0
	(1.1, 2.4–7.0)	(1.0, 2.4–6.1)	(1.0, 2.0–5.3)	(1.4, 2.1–6.2)	(0.6, 2.5–4.7)
Leftward saccades					
Number	14.3	14.0	7.8	7.9	5.2
	(5.4, 4.2–22.2)	(5.5, 4.4–20.0)	(2.8, 3.0–14.0)	(5.0, 4.0–21.2)	(2.4, 1.6–10.6)
Amplitude (°)	4.0	4.2	3.7	3.5	4.2
	(1.1, 2.4–5.3)	(2.3, 1.9–10.6)	(0.7, 2.1–4.8)	(0.8, 2.4–5.3)	(0.9, 2.7–6.1)

Table 8 Dependent samples t-tests (one-tailed) for analysing mean differences in visual exploration performance and oculomotor measures between left- and right-sided simulated hemianopia (LHH, RHH) and the normal viewing condition (N) before and after practice.

	N–LHH		N–RHH		LHH–RHH	
	Pre	Post	Pre	Post	Pre	Post
Visual exploration time (s)	*	*	*	*	n.s.	n.s.
Number of errors	*	n.s.	*	n.s.	n.s.	n.s.
Total fixations						
Number	*	*	*	*	n.s.	n.s.
Duration (ms)	*	n.s.	*	n.s.	n.s.	n.s.
Saccadic amplitude (°)	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
Scanpath length (°)	*	n.s.	*	n.s.	n.s.	n.s.
Right hemispace fixations						
Number	*	n.s.	*	*	0.048	n.s.
Duration (ms)	*	n.s.	*	n.s.	n.s.	n.s.
Left hemispace fixations						
Number	*	*	*	0.018	n.s.	n.s.
Duration (ms)	*	0.030	*	n.s.	n.s.	n.s.
Rightward saccades						
Number	n.s.	n.s.	*	*	*	*
Amplitude (°)	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
Leftward saccades						
Number	*	*	*	0.026	*	*
Amplitude (°)	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.

n.s. indicates non-significant comparisons. * $p<0.017$ (α_{corr}); p-values are given for marginally significant results.

The effect of practice on reading and visual exploration with simulated hemianopia

Practicing reading with simulated LHH or RHH (Experiment 2a) led to an improvement in reading performance and related eye-movements (Table 5), as indicated by a significant effect of time for reading time and all oculomotor parameters (smallest $F_{(1,11)}=7.79$, $p=0.018$). Significant pre–post-differences for both LHH and RHH confirm this finding (smallest $t_{(11)}= -2.20$, $p=0.025$; marginal significance for the amplitude of forward saccades in LHH ($t_{(11)}= -1.37$, $p=0.061$)). There was a significant effect of simulation condition (LHH, RHH) for reading time and all oculomotor parameters (smallest $F_{(1,11)}=4.90$, $p=0.049$), except for fixation repetitions ($F_{(1,11)}=0.37$, $p=0.558$). The significant interaction between time and

simulation condition for reading time and return-sweep amplitude (smaller $F_{(1,11)}$=7.11, p=0.022) can be explained by a significantly larger decrease in reading time for RHH (–28.2s) than for LHH (–11.6s) ($t_{(11)}$=2.81, p=0.017), and by a significantly larger increase in return-sweep amplitude for LHH (+2.4°) than for RHH (+0.8°) ($t_{(11)}$=2.67, p=0.022).

After reading practice, there was still a significant effect of simulation condition (LHH, RHH, N) for reading time and all oculomotor parameters (smallest $F_{(2,22)}$=5.73, p=0.010), except for fixation repetitions and the return-sweep amplitude (larger $F_{(2,22)}$=1.73, p=0.20). Yet, mean differences in reading time between the simulated HH and normal viewing condition were much smaller (LHH: 3.8 s, RHH: 18.7 s) than before practice (LHH: 15.5 s, RHH: 45.5 s). Analysing the differential effects of LHH and RHH on practice outcome revealed that practicing reading with RHH led to greater improvements than practicing reading with LHH. However, the reading performance participants regained was closer to normal during with LHH than with RHH. Yet, although practicing reading with LHH or RHH significantly reduced the reading impairment caused by the hemianopic visual field defect, reading performance and eye-movements still differed from normal reading after practice (Table 6).

Practicing visual exploration with simulated LHH or RHH (Experiment 2b) led to a significant improvement in visual exploration performance and related eye-movements (Table 7), as indicated by a significant effect of time for visual exploration time, number of errors, and for number and duration of fixations and scanpath length (smallest $F_{(1,12)}$=5.13, p=0.043). Significant pre–post-differences for LHH and RHH confirm this finding (smallest $t_{(11)}$= –2.20, p=0.025). Consistent with pre–practice analyses, there was no significant effect for overall, left- and rightward saccadic amplitude; practice did also not affect number and duration of left- and right-hemispace fixations (largest $F_{(1,12)}$=2.49, p=0.141). In contrast to reading practice, visual exploration performance and eye-movement measures as well as the overall practice outcome were not differentially affected by the side of simulated HH (non-significant effect of simulation condition and of its interaction with time, largest $F_{(1,12)}$=3.60,

p=0.082); only the number of left- and rightward saccades differed significantly between LHH and RHH, both before and after practice (significant effect of simulation condition, smaller $F_{(1,12)}$=8.89, p=0.011).

The absence of a significant effect of simulation condition (LHH, RHH, N) for number of errors, duration of overall, left- and right-hemispace fixations, scanpath length, and duration of left- and right-hemispace fixations after practice indicates that participants regained normal performance with regard to these visual exploration measures despite LHH or RHH (largest $F_{(2,24)}$=2.54, p=0.100; see also Tables 7, 8). Yet, visual exploration time and the number of overall, left- and right-hemispace fixations and of left- and rightward saccades were still elevated, albeit to a lesser extent (smallest $F_{(1.2,14.6)}$=5.03, p=0.035). Although the differences for visual exploration time still reached statistical significance, they were very small (LHH–N: 2.9 s, RHH–N: 2.6 s) and are unlikely to reflect any meaningful performance difference, especially when considering that visual exploration with LHH and RHH was as accurate as normal performance after practice. However, visual exploration with RHH was still characterised by significantly more right-hemispace fixations and rightward saccades that were also more frequent than left-hemispace fixations and leftward saccades; the converse pattern was obtained for visual exploration with LHH (see Table 8; significant interaction between simulation condition and hemispace/direction, smaller $F_{(2,24)}$=3.77, p=0.038; smallest $t_{(12)}$=2.51, p=0.014; one-tailed).

Subjective reports

Participants' subjective reports were in close agreement with the effects of simulated HH on reading (Experiment 2a) and visual exploration (Experiment 2b) as well as with the effects of reading and visual exploration practice as verified by objective test results (for a selection of representative quotes, see Table 9). Subjective reports on pre–practice reading and visual exploration performance were similar to those obtained in Experiment 1. After reading practice (Experiment 2a), all participants reported an improvement in reading, which was described as an increase in the ability to efficiently identify words and guide eye-movements

through the text despite simulated HH. Participants reported to have developed specific reading strategies which reduced omission and guessing errors, diminished the need to re-read words, and improved text comprehension; to guide their eye-movements during reading with simulated LHH, they reported to have made use of the fixed left text boundary. Reading with simulated LHH was experienced as more or less normal after practice whereas reading with simulated RHH was still considered as impaired, albeit to a lesser extent. After visual exploration practice (Experiment 2b), all participants reported an improvement in visual exploration performance, which was described as an increase in the ability to quickly gain a complete overview of each stimulus pattern and accurately count all dots despite simulated HH; participants also stated that they were much more confident about which dots have already been seen and counted than before practice. Participants reported to have quickly adopted a more efficient eye-movement strategy for dot counting. After practice, visual exploration with simulated HH was described as being normal.

Table 9 Subjective reports on the effects of practicing reading and visual exploration with simulated HH (selection of representative verbatim quotes).

Reading practice

- "I got used to reading with half-blindness and reading became much easier."
- "Over time, the technique for unveiling words and sentences got better."
- "I tried to look past each word and see it as a whole before reading it."
- "I tried to carry on in the flow of reading by imagining that there are more words to come that need to be looked at."
- "I forced myself to follow each sentence although the rest of the sentence was not there."

Visual exploration practice

- "After practice, exploring and counting dots with left or right half-blindness was normal."
- "After practice, dot counting was much easier and quicker than in the beginning"
- "Concentrating on eye-movements to unveil the dots and keeping count at the same time became less effortful."
- "I tried to get a quick overview of the entire dot pattern by making large eye-movements and grouping dots."
- "I overcompensated with the eyes into the blind field."

Practice effects in the control condition

In the control samples, there was no significant effect of time (Experiment 2a: largest $\chi^2_{(4)}$=7.07, p=0.132; Experiment 2b: largest $\chi^2_{(4)}$=9.36, p=0.053). Although there was a significant effect for forward and return-sweep saccadic amplitude and scanpath length in Experiment 2a (smaller $\chi^2_{(4)}$=10.40, p=0.024), no difference between any two of the four time points was significant (largest Z=2.20, p=0.031 (corrected level of significance: p=0.01)); even if significant, these differences would be either too small to reflect any meaningful difference (0.5° and 0.6° for the amplitudes of forward and return-sweep saccades respectively) or even indicate maladaptation since scanpath length increased by 41.3°. In Experiment 2b, there was a significant effect for number of fixations and forward saccades, fixation duration and scanpath length (smallest $\chi^2_{(4)}$=9.76, p=0.045); yet, again, no difference between any two of the four time points reached statistical significance (largest Z= –2.02, p=0.063).

3.3. Discussion

The main result of Experiment 2 is that reading (Experiment 2a) and visual exploration practice (Experiment 2b) without specific instruction led to significant improvements in reading and visual exploration with simulated HH, respectively. In addition, the effect of simulated HH on reading and visual exploration performance and associated eye-movement patterns found in Experiment 1 was replicated, which is also congruent with previous reports on the hemianopic reading and visual exploration impairments in patients with HH. Moreover, the findings from Experiment 1 were complemented by obtaining the differential effect of simulated LHH and RHH on the return-sweep in reading (Experiment 2a) as well as on the horizontal fixation distribution and directional oculomotor measures in visual exploration (Experiment 2b), which are typical for the hemianopic reading and visual exploration impairments (Zihl, 1995a, 1995b, 1999, 2000).

Reading practice effects were characterised by a considerable decrease in reading time, the effects of visual exploration practice by a decrease in exploration times and number

of errors despite simulated LHH or RHH. Both improvements were accompanied by changes in the respective eye-movement patterns. In reading (Experiment 2a) participants made significantly fewer fixations and fixation repetitions and showed much shorter fixation durations. The amplitude of forward saccades and that of the return-sweeps increased, which led to a much smaller number of forward saccades. Participants seemed to extract the same amount of text information by using a much more efficient oculomotor text processing strategy, which is also reflected by the significant decrease in scanpath length. In visual exploration (Experiment 2b), they also showed significantly fewer fixations and shorter fixation durations. Although the differential distribution of fixations as well as the differential effect on directional oculomotor measures pertained after practice, participants seemed to have adopted a much more efficient oculomotor strategy for exploring and processing visual information, which is also reflected by significantly shorter and more systematic scanpaths. Although inter-individual differences of these changes were substantial (as indicated by a large variation in individual means before and after practice, see Table 4 (Experiment 2a) and 6 (Experiment 2b)) reading and visual exploration performance as well as oculomotor parameters improved in all participants.

It is important to note that the improvements in reading and visual exploration and associated eye-movements cannot be attributed to increases in visual field sparing during the experimental sessions since the accuracy of the simulated visual field border was continuously monitored. The absence of performance changes during reading and visual exploration practice under normal viewing conditions shows that mere practice effects cannot account for the performance changes during reading practice with simulated HH. In addition, there was no evidence of a trade-off between speed and accuracy after practice, neither for reading nor for visual exploration performance. Before and after reading practice, participants reiterated the content of each text equally correctly, and visual exploration practice led to a significant decrease in number of errors.

Practice-related changes of oculomotor measures in reading (Experiment 2a) and visual exploration (Experiment 2b) seem to reflect spontaneous oculomotor adaptation to simulated HH, which is possibly best understood as functional reorganisation of eye-movement control in reading (see also Chapter 1) and visual exploration (Mort & Kennard, 2003). Spontaneous oculomotor adaptation to simulated HH in reading and visual exploration possibly emerges as a result of perceptual and oculomotor (procedural) learning processes in reading (Ofen-Noy, Dudai, & Karni, 2003) and visual exploration (Rogers, Lee, & Fisk, 1995), which are modulated by attention. These processes seem to occur spontaneously and rapidly when healthy participants are first confronted with a simulated HH, even in the absence of any instruction aimed at improving performance. Reading as few as only 10 short text passages and practicing visual exploration for as few as only thirty trials seems to suffice to facilitate spontaneous oculomotor adaptation processes, which alleviate the reading and visual exploration impairments resulting from this simulated visual-sensory deficit. Since eye-movements were not recorded binocularly, it remains possible that the improvements during reading practice were based on changes in fixation disparity. Although participants may have compensated for simulated HH by increasing the magnitude and/or frequency of fixation disparity, the effects of such a strategy cannot fully account for the obtained improvements. During normal reading, average fixation disparity ranges between 1–2 characters (40-50% of fixations) (Liversedge, Rayner, White, Findlay, & McSorley, 2006a; Liversedge, White, Findlay, & Rayner, 2006b). Since the visual system may tolerate fixation disparity only up to a certain point and reduced convergence leading to increased fixation disparity seems to be associated with a reduction in reading performance (Kirkby, Webster, Blythe, & Liversedge, 2008), the adaptation of fixation disparity during reading with simulated HH is limited. The resulting improvement of ~2 characters *per* fixation is, however, too small to explain the obtained improvement in reading performance.

Hemianopic patients with impairments of reading and visual exploration in contrast require specific and systematic treatment to reinforce these oculomotor adaptation processes

(Gassel & Williams, 1963; Zihl, 2000, 2003). About 10–15 oculomotor reading training sessions (a 45 min.) and an equal amount of oculomotor scanning training is necessary for patients to regain sufficient reading and visual exploration performance (Zihl, 2000). The changes related to spontaneous oculomotor adaptation in this study's participants are consistent with the treatment-related changes of hemianopic patients in reading and visual exploration (Zihl, 1995a, 1995b, 2000). This finding is also in accordance with previous studies investigating spontaneous oculomotor adaptation to simulated central visual field loss in reading (Bernard, Scherlen, & Castet, 2007; Fornos, Sommerhalder, Rappaz, Pelizzone, & Safran, 2006; Sommerhalder et al., 2003, 2004) and reports on spontaneous oculomotor adaptation to simulated hemianopic visual field loss in visual exploration (Zangemeister & Oechsner, 1999; Zangemeister & Utz, 2002).

Yet, there seems to be a differential effect of simulated LHH and RHH on the outcome of practice that is specific to reading. Reading 10 text passages with RHH led to greater improvements than reading the same amount of text with LHH. After practice, however, reading with LHH was closer to normal than reading with RHH, albeit that in either case reading still differed from that under normal viewing conditions. In contrast to reading, there was no such differential effect on the outcome of visual exploration practice. Practicing visual exploration for 30 trials led to the same improvements in visual exploration with simulated LHH and RHH. This finding is consistent with the differential effect of left- and right-sided visual field loss on the rehabilitation outcome of hemianopic patients receiving specific treatment for their reading and visual exploration impairments. Patients with RHH require twice as many reading training sessions to reach the same outcome as patients with LHH whereas an equal amount of training leads to the same improvements in visual exploration (Zihl, 1995a, 2000).

4. General discussion

The purpose of the reported experiments was to identify the visual components that may constitute the hemianopic reading and visual exploration impairments as well as to determine

whether these impairments are purely visually elicited. Experiment 1 examined the effects of simulated HH on reading, visual exploration and saccadic accuracy in healthy participants. Experiment 2 investigated whether and to what extent healthy participants may spontaneously adapt to simulated HH in reading (Experiment 2a) and in visual exploration (Experiment 2b). The results suggest that the hemianopic visual field defect clearly contributes to the chronic impairments of reading and visual and exploration found in hemianopic patients although it may not be their sole cause.

Experiment 1 demonstrated that simulated HH produces the main features of the hemianopic reading and visual exploration impairments (as well as of its indicator saccadic accuracy) in healthy participants. This result shows that the bottom-up restriction of the visual field clearly affects reading and visual exploration performance. Reading critically depends on the parafoveal visual field, which provides the basis for word identification and eye-movement control (Rayner, 1998), whereas efficient visual exploration requires global visual information extraction from the parafoveal and peripheral visual field for the attentional top-down control of eye-movements in space and local processing of fine details (Hochstein & Ahissar, 2002; Juan & Walsh, 2003). If vision in these visual field regions is affected, either by simulated HH or by brain injury, efficient word identification and the visual control of eye-movements in reading are impaired; since visual scenes are only partly visible, quickly gaining a complete overview becomes increasingly difficult and consequent impairments of global processing affect guiding the eyes through a scene for further local processing (Zihl, 2000).

The differential effect of simulated (or real) LHH and RHH on reading performance provides additional evidence for the visual basis of the hemianopic reading impairment. In left-to-right reading, right parafoveal vision is of greater importance than left parafoveal vision (McConkie & Rayner, 1976). Visual information to the right of fixation is critical to eye-movement control and enables efficient processing of the foveal and preprocessing of the parafoveal word whereas visual information to the left of fixation is mainly required for

planning and guiding the return-sweep (Rayner, 1998). This explains why the hemianopic reading impairment is more pronounced in simulated (or real) RHH than in LHH. These results are substantiated by a prior study showing that masking the right visual field imposes a greater limit to reading performance than masking the left visual field (Fine & Rubin, 1999a; see also Cummings & Rubin, 1992; Ikeda & Saida, 1978; McConkie & Rayner, 1975, 1976; Rayner et al., 1981; Rayner, Liversedge, & White, 2006). However, since the foveal visual field and parts of the contralateral parafoveal visual field were additionally obliterated in this study (Fine & Rubin, 1999a), the resulting reading impairment was more pronounced than in the present experiment. Occluding foveal vision, which is essential for word identification, makes reading almost impossible (Fine & Rubin, 1999b, 1999c; Rayner & Bertera, 1979; Rayner et al., 1981). That the greatest impairments of reading associated with a visual field disorder are found in patients with a central scotoma is consistent with this finding (Teuber, Battersby, & Bender, 1960; Zihl, 2000).

Yet, this differential effect seems to be specific to reading. Although the side of the hemianopic visual field defect determines the horizontal fixation distribution and properties of directional oculomotor measures in visual exploration, there are no performance differences between LHH and RHH. It does not determine the severity of the resulting impairment as it does in reading. Thus, there seems to be a stronger relationship between the visual-sensory defect and the resulting impairments in reading than in visual exploration. Further evidence stems from the observation that the extent of a visual field defect (as determined by visual field sparing) determines the severity of the resulting reading impairment but not that of the visual exploration (and saccadic accuracy) impairment (Zihl, 1995a, 1995b, 2000). Poppelreuter (1917/1990) therefore concluded that "the visual field defect as such does not itself significantly impair the process of visual search" (p. 113) and dismissed it as primary cause of the hemianopic visual exploration impairment; he also suggested that the reading impairment "caused by the hemianopia itself is not that substantial" (p. 223).

Experiments 2a and 2b demonstrated that the hemianopic visual field defect is a necessary but possibly not a sufficient condition that causes the severe and long-lasting reading and visual exploration impairments in hemianopic patients. When participants were confronted with simulated HH, they initially presented the main features of the hemianopic reading and visual exploration impairments. Yet, relatively quickly, they spontaneously adapted to simulated HH by developing efficient oculomotor compensation strategies that alleviated the reading and visual exploration impairments caused by this pure visual-sensory deficit. Participants regained close to normal visual exploration performance but reading with simulated HH, particularly with simulated RHH, remained impaired. Yet, since the reading performance level was still higher than that of hemianopic patients, visually elicited hemianopic reading and visual exploration impairments do not seem to be as severe and long-lasting as those found in hemianopic patients whose reading and visual exploration performance remains severely impaired even years after the occurrence of visual field loss (Gassel & Williams, 1963).

These findings are consistent with observations that some hemianopic patients show efficient spontaneous oculomotor adaptation and regain normal performance very soon after brain injury (Gassel & Williams, 1963; Zihl, 2000, 2003). Interestingly, patients are more likely to adapt to their visual field defect in visual exploration (~40% of cases) than in reading (~20%). Moreover, there seems to be a clear double dissociation between spontaneous oculomotor adaptation to visual field loss in visual exploration and reading (Zihl, 2000), suggesting task-specificity of spontaneous oculomotor adaptation to visual field loss. This may be explained by a task-specific functional specialisation of the (cortical) oculomotor system (Alahyane et al., 2007) and is consistent with the view that control of visual processing and eye-movements in reading may be mediated by different neural networks than in visual exploration, albeit both networks probably overlap (Zihl, 1995a, 1995b, 2000).

Yet, successful spontaneous oculomotor adaptation to visual field loss occurs only very rarely in patients. It seems to depend on whether postchiasmatic visual pathway injury is accompanied by injury to the fibre pathways and/or structures involved in the visual bottom-up and attentional top-down control of visual information processing and saccadic eye-movements in reading (see also Chapter 1) and visual exploration (Zihl, 1995b, 2000). Patients whose brain injury is confined to the postchiasmatic visual pathway spontaneously adapt to their visual field loss and show normal reading and visual exploration performance (Zihl, 1995a, 1995b). Thus, vision is what the eyes (can) make of it. If the occipital white matter comprising subcortical–cortical reciprocal connections and/or the posterior thalamus is additionally affected by brain injury, patients show severe and chronic impairments of reading (Zihl, 1995a). Impairments of visual exploration emerge and persist if patients show additional injury to the ipsilateral occipito-parietal cortex and/or posterior thalamus (Zihl, 1995b).

Observations of patients with normal visual fields and posterior parietal damage showing the hemianopic visual exploration (and saccadic accuracy) impairment (Poppelreuter, 1917/1990; Zihl & Hebel, 1997) suggest that it is not the visual field defect but additional extrastriate brain injury that causes this impairment; a comparison between these patients and hemianopic patients with a similar posterior parietal involvement might clarify whether an accompanying visual field defect may exacerbate the visual exploration impairment. The hemianopic reading impairment, in contrast, seems to critically depend on the presence of a visual field defect. Although patients with normal visual fields and posterior parietal damage also reported difficulties in finding their way through lines of text on a page (Zihl & Hebel, 1997), no case of hemianopic dyslexia in patients with normal visual fields and occipital white matter and/or posterior thalamus injury has been reported thus far.

The high frequency of extrastriate lesions in patients with homonymous visual field loss (Hebel & von Cramon, 1987) explains why impairments of reading and visual

exploration are commonly associated with hemianopic visual field defects. That these patients require systematic oculomotor training for at least 8 hours (Zihl, 2000), whereas this study's participants showed improved reading or visual exploration performance after only 15 minutes of uninstructed practice, provides further evidence that the visual field defect is an important but not the sole cause of the hemianopic reading and visual exploration impairments. That patients with extensive lesions involving the occipital white matter and/or occipito-parietal regions require the largest amount of training (Zihl, 1995a, 1995b, 2000) is consistent with this assumption. The greater importance of the visual field defect for the hemianopic reading impairment than for the visual exploration impairment is substantiated by the differential effect of left- and right-sided visual field loss on the treatment outcome in reading but not in visual exploration (Zihl, 1995a, 1995b, 2000). Yet, the findings obtained from the experiments presented in this chapter may be limited by the fact that the evidence was obtained on the basis of relatively young and well-educated healthy participants. The majority of hemianopic patients are over the age of 55 (Zihl, 2000) and age-related processes appear to play a significant role in spontaneous oculomotor adaptation to visual field loss (Tant et al., 2002).

In conclusion, this study suggests that the visual field defect is a major component of the hemianopic reading impairment. It is likely, however, that additional injury to the occipital white matter and/or posterior thalamus is required for this impairment to persist. Although the visual field defect contributes to the hemianopic visual exploration impairment, it does not seem to be causative. In contrast to the hemianopic reading impairment, injury to the ipsilateral occipito-parietal cortex and/or posterior thalamus seems to be the primary cause. Hemianopic dyslexia and the impairment of visual exploration may be interpreted as disorders of the visual bottom-up and attentional top-down control of visual processing and eye-movements which masquerade as failures of vision.

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Chapter 3

ADAPTATION OF EYE-MOVEMENTS TO SIMULATED HEMIANOPIA IN READING AND VISUAL EXPLORATION: TRANSFER OR SPECIFICITY?

The experiment reported in this chapter further investigated whether spontaneous oculomotor adaptation to visual field loss is task-specific or whether there is a transfer of adaptation-related improvements between reading and visual exploration. It explored the specificity with which oculomotor adaptation to simulated hemianopia during uninstructed reading or visual exploration practice leads to improvements in both abilities. Since there was no transfer of adaptation-related performance and oculomotor improvements between reading and visual exploration, it is concluded that efficient oculomotor adaptation to visual field loss is highly specific and task-dependent.

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1. Introduction

Unilateral homonymous hemianopia (HH) is the most frequent visual disorder after brain damage (Zihl, 2000). It is commonly caused by posterior cerebral artery infarction affecting the postchiasmatic visual pathway. In HH, vision is lost in both monocular hemifields contralateral to the side of brain injury (Zhang, Kedar, Lynn, Newman, & Biousse, 2006a; Zihl, 2000). Homonymous visual field defects are chronic manifestations since sufficient spontaneous recovery of the visual field is seldom seen (Zhang, Kedar, Lynn, Newman, & Biousse, 2006b; Zihl & Kennard, 1996). The majority of patients show persistent and severe impairments of reading (i.e., hemianopic dyslexia) and visual exploration (Zihl, 2000, 2003).

The cardinal symptoms of hemianopic dyslexia are slowed reading, visual omission and guessing errors as well as a severely altered reading eye-movement pattern (e.g., Leff et al., 2000; McDonald, Spitzyna, Shillcock, Wise, & Leff, 2006; Spitzyna et al., 2007; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a, 2000). The visual exploration impairment is characterised by considerably increased exploration times, target omissions as well as longer and unsystematic oculomotor scanning patterns (e.g., Mort & Kennard, 2003; Pambakian et al., 2000; Tant, Cornelissen, Kooijman, & Brouwer, 2002; Zihl, 1995b, 1999, 2000). These hemianopic reading and visual exploration impairments have been reported early in the literature and are now well-established clinical phenomena (for early clinical reports, see Mauthner, 1881; Pfeifer, 1919; Poppelreuter, 1917/1990; Wilbrand, 1907).

Spontaneous adaptation of eye-movements to visual field loss and consequent improvements in reading and visual exploration performance is an equally well-known phenomenon with a long history. Poppelreuter (1917/1990) was the first to report spontaneous oculomotor adaptation in hemianopic patients. Very soon after brain injury, some patients spontaneously adopt eye-movement strategies allowing them to efficiently compensate for their visual-sensory dysfunction. As a consequence, even patients with the most severe visual field defect can regain normal reading and visual exploration performance (Gassel & Williams, 1963; Mackensen, 1962; Meienberg, Zangemeister, Rosenberg, Hoyt,

& Stark, 1981; Zangemeister, Oechsner, & Freska, 1995; Zangemeister & Utz, 2002; Zihl, 2000, 2003). Yet, it is still unclear whether efficient spontaneous oculomotor adaptation to visual field loss in reading and visual exploration is task-specific, or whether there is a transfer of adaptation-related improvements between reading and visual exploration. Consequently, our understanding of oculomotor adaptation processes in homonymous visual field loss and thus current rehabilitation practice remains imperfect.

The experiments reported in Chapter 2 demonstrated that simulated HH successfully induces the hemianopic reading and visual exploration impairments in healthy participants. Over time, however, all participants showed efficient spontaneous oculomotor adaptation to this pure visual-sensory defect which led to improvements in reading and visual exploration performance. These adaptation processes seemed to occur spontaneously and rapidly, even in the absence of any instruction aimed at improving participants' performance (see also Poppelreuter, 1917/1990). To investigate whether spontaneous oculomotor adaptation is task-specific, or whether there is a transfer of adaptation-related improvements between reading and visual exploration, another experiment was conducted that compares the effects of uninstructed reading and visual exploration practice on reading and visual exploration performance with simulated HH in a cross-over design.

2. Methods

2.1. Participants

Twenty-four naïve, healthy participants (8 males, 16 females) participated in this experiment. Mean age was 19.1 years (SD: 1.0) and subjects had on average 12.5 years of education (SD: 0.7). All participants were native English speakers with normal or corrected-to-normal vision, had no reading disorders, visual disorders or any other neurological disease or psychiatric condition, and gave their informed consent in accordance with the Declaration of Helsinki and with local ethical committee approval.

2.2. Eye movement recording and simulating hemianopia

The methods used for eye-movement recording and simulating left- and right-sided HH (LHH, RHH) in healthy participants were identical to those used in the experiments presented in the previous Chapter 2.

2.3. Assessment of reading performance and eye-movements

Reading and eye-movements during silent text reading were assessed using four texts of the reading task used in the experiments presented in Chapter 2. This task was demonstrated to be sensitive to adaptation-related changes during uninstructed reading practice with a simulated HH (see Chapter 2). Reading performance and eye-movement analyses were also identical to those in Chapter 2; i.e., reading performance was defined as the time required to read one text passage (reading time) and the following global temporal and spatial oculomotor parameters were analysed for each text: number and mean duration (ms) of fixations, percentage of fixation repetitions (i.e. fixations at previously fixated points), number and mean amplitude (°) of forward (i.e. rightward) saccades, mean amplitude of return-sweep saccades (i.e. the mean first amplitude of eye-movements from the end to the beginning of the next line (°)) and scanpath length (i.e. the sum of all saccadic amplitudes (°)).

2.4. Assessment of visual exploration performance and eye-movements

Visual exploration and related eye-movements were also assessed using the same task as in Chapter 2, which demonstrated the sensitivity of this task to adaptation-related changes during uninstructed visual exploration practice with a simulated HH. Visual exploration performance and eye-movement analyses were also identical to those performed in the previous chapter, i.e., visual exploration performance was defined as exploration time (the time required to perform one trial) and number of errors (all errors committed were omission errors) and the following global temporal and spatial oculomotor parameters were analysed

for each trial (five trials in total): number and mean duration (ms) of fixations, mean saccadic amplitude (°) and scanpath length (i.e., the sum of all saccadic amplitudes (°)).

2.5. Reading and visual exploration practice

The reading and visual exploration practice sessions (RP, VP) were identical to those used in the experiments presented in Chapter 2. All participants performed one RP and one VP session.

2.6. Procedure

Participants were randomly allocated into two equal groups: Group A (n=12) first performed the reading practice (RP), then the visual exploration practice (VP) session; Group B (n=12) did the converse and first performed the VP, then the RP session in a cross-over design. Half of each group (n=6) performed the two practice sessions with a RHH, the other half with a LHH. Reading and visual exploration performance and eye-movements were assessed before (T1) and after (T2) the first practice session, after the second practice session (T3), and then in a normal viewing condition (N), i.e., without any simulated HH (see Fig. 1).

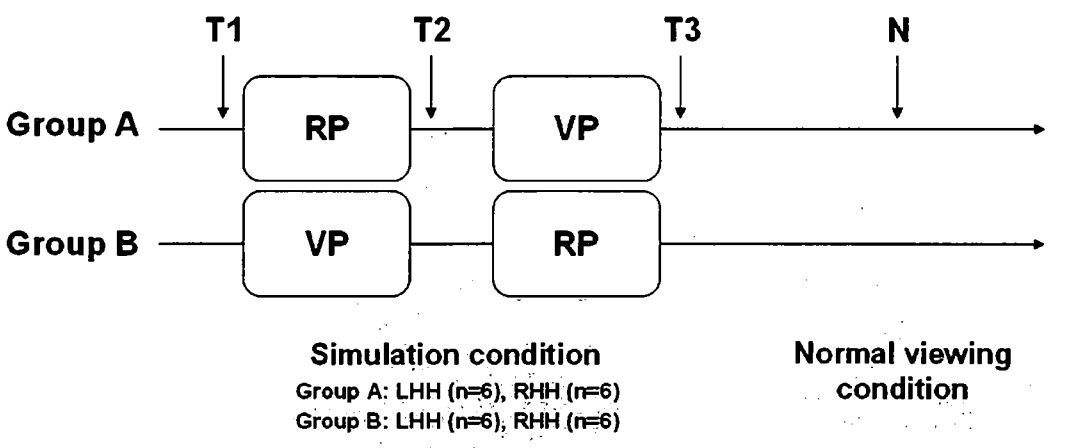

Fig. 1. Illustration of the experimental procedure. Practice sessions in the simulation condition (left- or right-sided hemianopia (LHH, RHH)) were either uninstructed reading practice (RP) or visual exploration practice (VP). T1-T3 indicate the three time points at which reading and visual exploration with simulated hemianopia was assessed; N indicates the time point at which reading and visual exploration was assessed under normal viewing conditions.

Both the sequence of assessment tasks (performing the reading or visual exploration task first) and that of texts (passage 1-4) used for reading assessment were counterbalanced across participants to eliminate order effects. There were no differences between Group A and B either for demographic variables or for reading and visual exploration performance and oculomotor measures before practice (T1) and in the normal viewing condition (N) (see Table 1).

Table 1 Demographic details and reading and visual exploration performance with simulated HH before practice (T1) and in the normal viewing condition (N) for Group A and B [mean (SD, range)].

	Group A (n=12)	Group B (n=12)	
Age (years)	19.2 (1.0, 18–21)	19.0 (1.0, 18–21)	+p=.748
Education (years)	12.5 (0.8, 12–14)	12.6 (0.7, 12–14)	+p=.665
Sex (female : male)	8 : 4	8 : 4	
Side of simulated HH (LHH : RHH)	6 : 6	6 : 6	
Reading time (s)*			
T1	59.9 (31.3, 16.9–136.3)	65.3 (35.3, 26.4–136.0)	p=.708
N	17.5 (3.8, 12.3–23.3)	19.8 (6.1, 12.5–34.9)	p=.280
Exploration time (s)*			
T1	16.6 (5.4, 8.6–26.4)	14.7 (3.9, 8.3–19.4)	p=.329
N	7.0 (1.2, 5.7–10.3)	7.5 (1.5, 5.2–10.9)	p=.431
Number of errors			
T1	0.52 (0.55, 0–2.0)	0.55 (0.47, 0–1.4)	p=.874
N	0.03 (0.05, 0–0.1)	0.02 (0.04, 0–0.1)	p=.368

Statistical comparisons were made between groups. P-values for two-tailed independent samples t-tests or +Mann-Whitney-U-tests (where normality assumptions were violated as assessed by Shapiro-Wilk tests) are given. * There were also no differences for oculomotor reading and visual exploration measures between groups (largest $t_{(22)}=1.81$, p=0.085).

In order to disentangle the effects of adaptation to simulated HH from performance changes due to mere practise effects, a new group of six participants (6 females; mean age: 19.3 (SD: 1.0); mean years of education: 12.2 years (SD: 0.4)) that performed the same experimental protocol without any simulated HH was studied (control condition).

2.7. Data analyses

The data were analysed by repeated measures ANOVAs (for details on factor variables, see the results section (3.)). Separate analyses were performed for analysing reading and visual exploration performance and oculomotor measures. For the comparisons, either the largest or smallest F value is reported. In the control sample, Friedman nonparametric analyses of variance were performed to test for overall effects of time (T1, T2, T3, N); for post-hoc paired comparisons, Wilcoxon tests were used (two-tailed, $p<0.05$, Bonferroni-correction). 2.3% of trials were discarded from the analyses.

3. Results

3.1. The effect of simulated hemianopia on reading and visual exploration before practice

To test whether simulated HH affected reading and visual exploration performance and associated eye-movements before practice (i.e., at T1), and to determine whether there were any order effects reflected in differences between participants who first performed reading practice (Group A) and those who first performed visual exploration practice (Group B), simulation condition was used as a within-subject factor (simulated HH, normal viewing condition) and group as a between-subject factor (Group A, B). Simulated HH had the expected adverse effect on reading and visual exploration which did not differ between groups (non-significant main and interaction effects: largest $F_{(1,22)}=3.39$, $p=0.079$). During reading with simulated HH participants showed significantly longer reading times, a higher number and duration of fixations and refixations, many more and smaller forward saccades and a prolonged scanpath when compared with normal performance (significant effect of simulation condition: smallest $F_{(1,22)}=23.57$, $p<0.001$). During visual exploration with simulated HH, participants showed elevated exploration times, made many more errors and the prolonged scanpath was characterised by a higher number and duration of fixations (smallest $F_{(1,22)}=20.18$, $p<0.001$). However, participants failed to show the expected decrease in return-sweep and exploration saccadic amplitude (smaller $F_{(1,22)}=2.53$, $p=0.126$).

3.2. The specificity of practice-related changes in reading and visual exploration with simulated hemianopia

First, it was tested whether the order in which reading and visual exploration practice was carried out had an effect on the changes in reading and visual exploration performance and eye-movements. Time was therefore used as a within-subject factor (before vs. after the two practice sessions (T1/T3)) and group as between-subject factor (reading practice first vs. visual exploration practice first (Group A, B)). With a single exception, there were no order effects of whether reading or visual exploration practice occurred first on practice-related changes (non-significant main and interaction effects: largest $F_{(1,22)}=2.93$, $p=0.101$). The only exception was that, following the completion of practice, participants who practiced reading first (Group A) showed slightly larger improvements in return-sweep and exploration saccadic amplitude than participants who practiced visual exploration first (Group B) (significant interaction: smaller $F_{(1,22)}=6.34$, $p=0.020$).

Secondly, it was investigated whether there were any carry-over effects from reading practice or visual exploration practice, i.e., it was tested whether practicing visual exploration was beneficial (or disadvantageous) to the outcome of subsequent reading practice and *vice versa*. Therefore, two repeated measures ANOVAs were conducted with time as a within-subject factor (pre-/post-reading-practice; pre-/post-visual-exploration-practice) and group as a between-subject factor (Group A, B). The effect of reading practice did not differ between participants who first practiced reading (Group A) and those who received visual exploration practice before practicing reading (Group B) (non-significant interaction effect: largest $F_{(1,22)}=2.93$, $p=0.101$). Exploration times and numbers of errors before and after reading practice were significantly larger in participants who had not yet received visual exploration practice (Group A) than those who practiced visual exploration before reading (Group B) (significant main effect of group, smaller $F_{(1,22)}=8.30$, $p=0.009$).

The same result was obtained for visual exploration practice (non-significant main and interaction effects: largest $F_{(1,22)}=2.19$, $p=0.153$). Pre- and post-exploration-practice reading

times, fixation durations, number of repeated fixations and saccadic amplitudes were significantly larger in participants who had not yet received reading practice (Group B) than in those who had already practiced reading (Group A) (significant main effect of group, smallest $F_{(1,22)}$=5.47, p=0.029). The only carry-over effect that was found was that participants who practiced reading first (Group A) showed a decrease in exploration saccadic amplitude after visual exploration practice whereas those who had not yet received reading practice (Group B) showed an increase in saccadic amplitude (significant interaction: $F_{(1,22)}$=9.23, p=0.006). Thus, there were no order effects or carry-over effects (with a single exception), and the measures for Groups A and B were therefore essentially indistinguishable.

The main result of the three analyses was that performing both reading and visual exploration practice sessions led to significant improvements in all reading and visual exploration performance and oculomotor measures (significant effect of time (T1/T3): smallest $F_{(1,22)}$=4.67, p=0.042). More importantly, the analyses revealed that these improvements were task-specific. Practicing reading and visual exploration with simulated HH led to specific improvements in performance and oculomotor measures of reading (see Fig. 2, 3) and visual exploration (see Fig. 4), respectively.

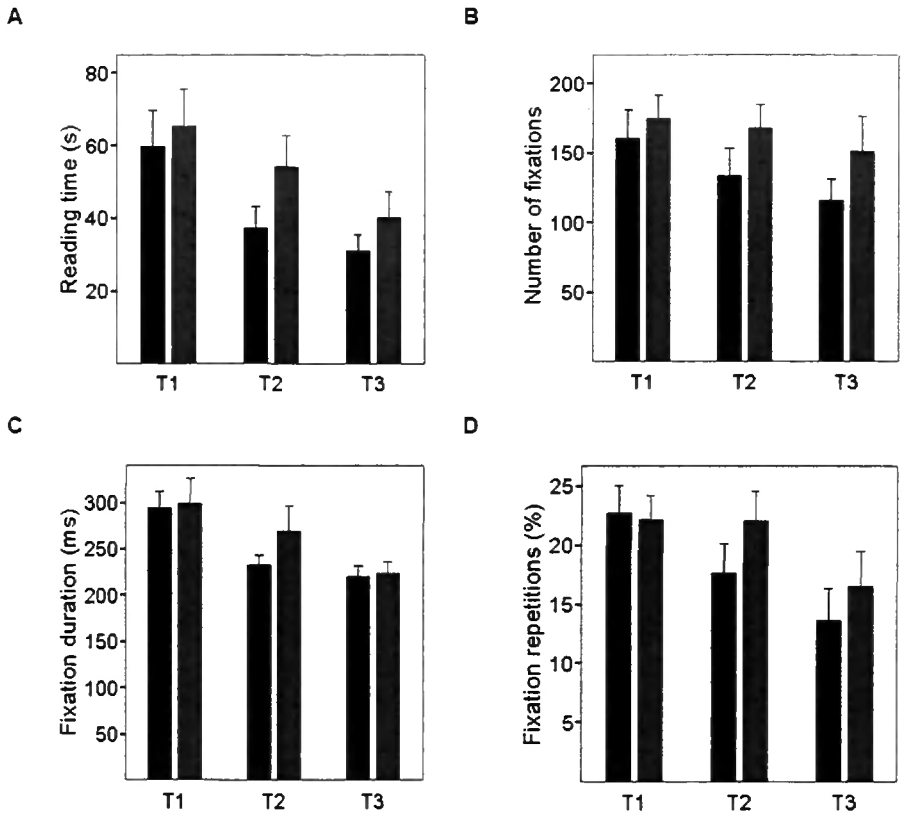

Fig. 2. Mean reading time (s) (**A**), number of fixations (**B**), fixation duration (ms) (**C**), and repeated fixations (%) (**D**) before practice (T1), after the first (T2) and second practice session (T3). The black bars at T1-T2 (Group A, practice sequence: reading→visual exploration) and the grey bars at T2-T3 (Group B, practice sequence: visual exploration→reading) illustrate the major improvements that were associated with reading practice but not with visual exploration practice (black bars: T2-T3, grey bars: T1-T2).

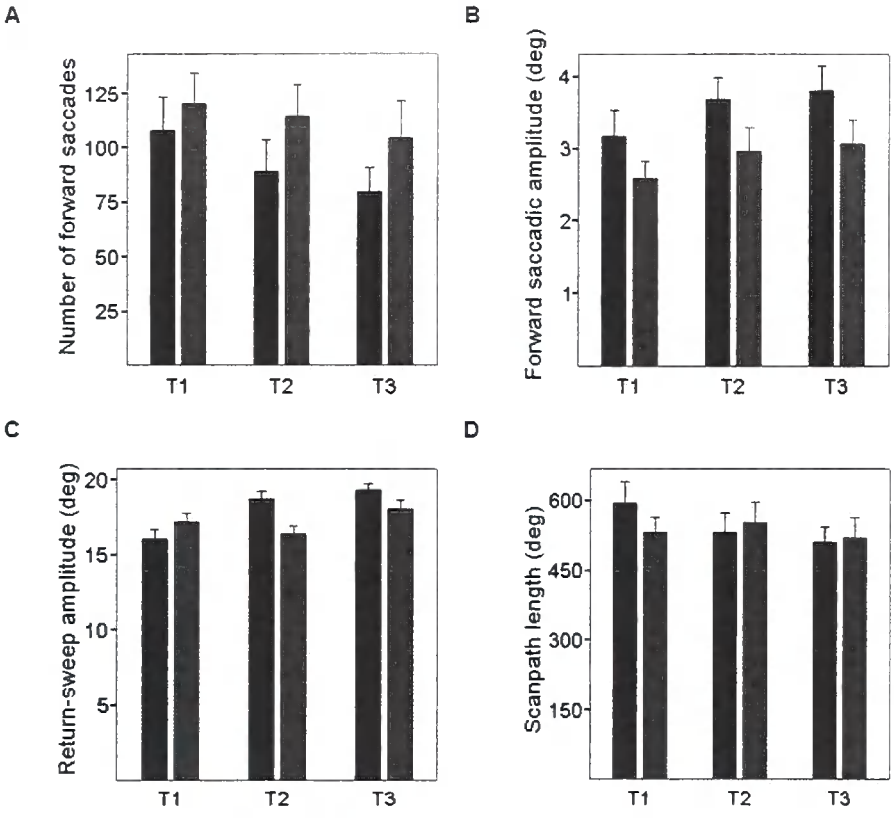

Fig. 3. Mean number (**A**) and amplitude of forward saccades (°) (**B**), return-sweep amplitude (°) (**C**), and scanpath length (°) (**D**) before practice (T1), after the first (T2) and second practice session (T3). The black bars at T1-T2 (Group A, practice sequence: reading→visual exploration) and the grey bars at T2-T3 (Group B, practice sequence: visual exploration→reading) illustrate the major improvements that were associated with reading practice but not with visual exploration practice (black bars: T2-T3, grey bars: T1-T2).

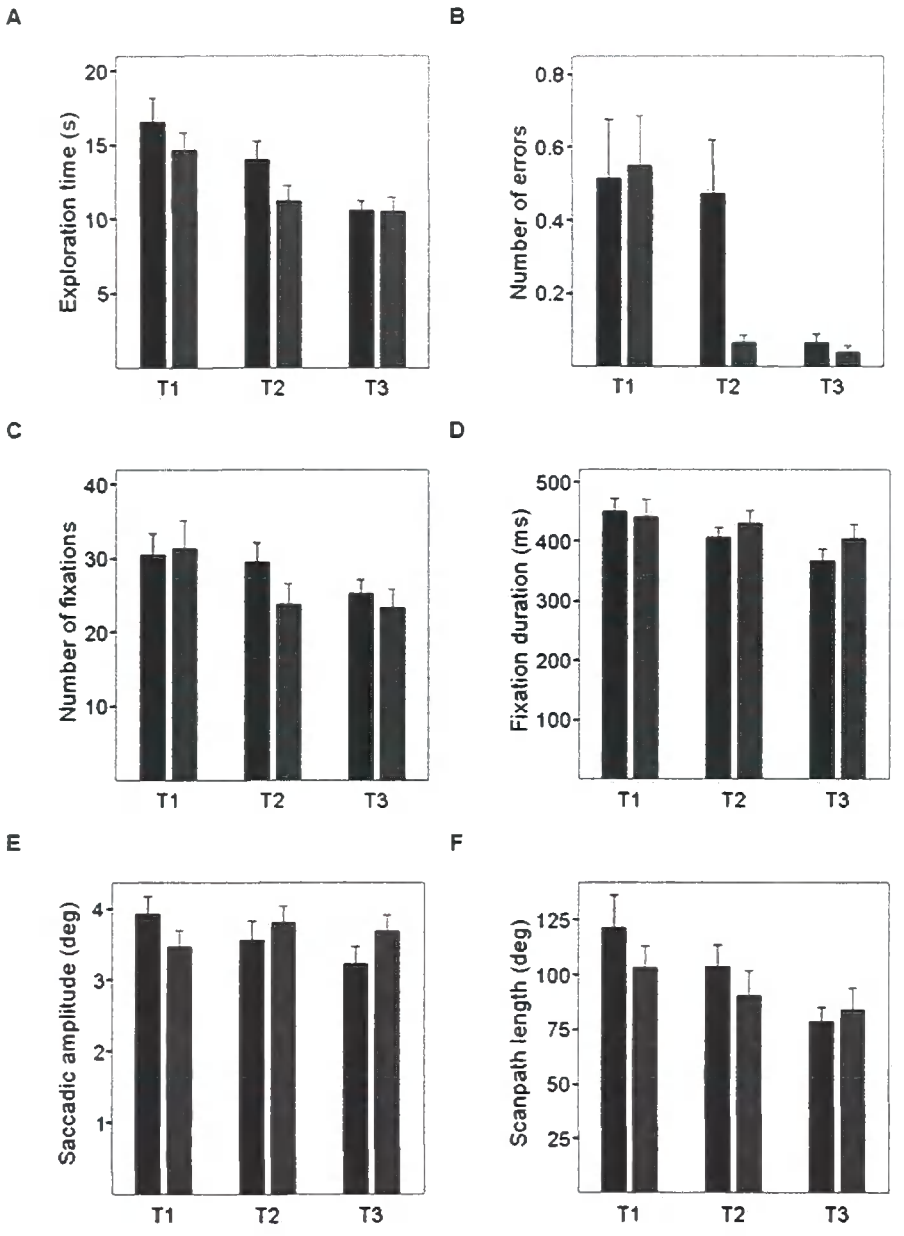

Fig. 4. Mean exploration time (s) (**A**), number of errors (**B**), number of fixations (**C**), fixation duration (ms) (**D**), saccadic amplitude (°) (**E**), and mean scanpath length (°) (**F**) before practice (T1), after the first (T2) and second practice session (T3). The grey bars (Group B, practice sequence: visual exploration→reading) at T1-T2 and the black bars (Group A, practice sequence: reading→visual exploration) at T2-T3 illustrate the major improvements that were associated with visual exploration practice but not with reading practice (grey bars: T2-T3, black bars: T1-T2).

Reading practice led to a significant decrease in reading time (significant effect of time (pre-/post-reading-practice): $F_{(1,22)}=19.89$, $p<0.001$) but did not affect visual exploration times and number of errors (larger $F_{(1,22)}=2.33$, $p=0.141$). Visual exploration practice, in contrast, induced a significant decrease in exploration time and number of errors (significant effect of time (pre-/post-visual-exploration-practice): smaller $F_{(1,22)}=25.18$, $p<0.001$).

Although it also led to a significant decrease in reading time ($F_{(1,22)}$=16.87, p<0.001), this decrease was very small (–8.7s) and significantly smaller than that after reading practice (–18.2s; $t_{(46)}$=2.05, p=0.045, two-tailed independent samples t-test).

These findings were mirrored in oculomotor measures. Reading practice led to a significant improvement in all oculomotor reading measures (except scanpath length) but not in oculomotor visual exploration measures. After reading practice, there was a significant decrease in number and duration of fixations and forward saccades as well as an increase in the amplitudes of forward saccades and return-sweeps (significant effect of time (pre-/post-reading-practice): smallest $F_{(1,22)}$=4.41, p=0.047). Yet, practicing reading had no effect on oculomotor visual exploration measures (largest $F_{(1,22)}$=2.16, p=0.156), with the exception of a slight decrease in fixation duration ($F_{(1,22)}$=7.21, p=0.014).

The converse pattern of results was obtained for visual exploration practice. After visual exploration practice, participants showed a significant decrease in the number of fixations and scanpath length during visual exploration (significant effect of time (pre-/post-visual-exploration-practice): smaller $F_{(1,22)}$=6.90, p=0.015). Oculomotor reading measures, however, remained unchanged after practicing visual exploration (largest $F_{(1,22)}$=2.35, p=0.140), with the exception of slight decreases in the number and duration of fixations and forward saccades during reading (smallest $F_{(1,22)}$=6.30, p=0.020). This improvement in fixation duration was significantly smaller than that induced by reading practice ($t_{(46)}$=2.34, p=0.023, two-tailed independent samples t-test).

In addition, it was investigated whether there were any differences in performance and practice-related improvements between left- and right-sided simulated HH in reading and visual exploration time, and whether these differences were task-dependent. Task (reading, visual exploration) and time (before and after the two practice sessions (T1/T3)) were used as within-subject factors and the side of simulated HH as a between-subject factor (left, right). Consistent with previous reports on HH (e.g., Zihl, 1995a, 2000), it was found that reading with a right-sided simulated HH was much more impaired and showed greater

improvements after reading practice than reading with a left-sided HH. More importantly, it was found that there were no such differences for visual exploration (significant 3-way-interaction: $F_{(1,22)}=6.97$, $p=0.015$). The decrease in reading time after reading practice was significantly larger in right-sided HH (–69.5s (SD: 24.8)) than in left-sided HH (–18.4s (SD: 12.0); $t_{(22)}=6.41$, $p<0.001$). The decrease in exploration time after visual exploration practice, in contrast, was the same for right-sided HH (–6.0s (SD: 4.8)) and left-sided HH (–4.2s (SD: 4.0); $t_{(22)}=1.05$, $p=0.307$) (two-tailed independent samples t-tests).

In summary, the main finding was that the order of reading and visual exploration practice had no effect on the practice-related improvements in reading and visual exploration performance and eye-movements. More importantly, however, these improvements were found to be task-specific.

3.3. The effect of simulated HH on reading and visual exploration after practice

Finally, it was tested whether the effects of simulated HH on reading and visual exploration performance and eye-movements that were obtained before practice were alleviated by performing reading and visual exploration practice (i.e., at T3), and whether there were any differences between participants who first performed reading practice (Group A) and those who first performed visual exploration practice (Group B). Simulation condition was used as a within-subject factor (simulated HH, normal viewing condition) and group as a between-subject factor (Group A, B). The effect of simulated HH on reading and visual exploration did not differ between groups (non-significant main and interaction effects: largest $F_{(1,22)}=3.17$, $p=0.089$). Although practicing reading and visual exploration with simulated HH led to significant improvements in reading and visual exploration performance and oculomotor measures, the adverse effect of simulated HH on reading and visual exploration remained after practice (significant effect of simulation condition: smallest $F_{(1,22)}=6.70$, $p=0.017$). Yet, mean performance differences between the simulated HH and normal viewing condition were much smaller (reading time: 17s; exploration time: 3.3s, errors: 0.03) than before practice (reading time: 43.9s; exploration time: 8.4s, errors: 0.51).

3.4. Practice effects in the control condition

Analysing the data obtained from the control sample that performed the same experimental protocol without any simulated HH revealed that there were no significant changes in reading or visual exploration performance and eye-movement measures (non-significant effect of time (T1/T2/T3/N): reading: largest $\chi^2_{(3)}=4.60$, p=0.218; visual exploration: largest $\chi^2_{(3)}=7.20$, p=0.060). Although there was a significant effect for number of fixations in reading ($\chi^2_{(3)}=10.16$, p=0.010), no difference between any two of the four time points was significant (Z= –2.21, p=0.124). Moreover, the obtained decrease was very small (–10%) and was not associated with improvements in reading and visual exploration performance measures since these remained unchanged.

4. Discussion

The purpose of this experiment was to determine the specificity of efficient oculomotor adaptation to visual field loss in reading and visual exploration. It was therefore investigated whether spontaneous oculomotor adaptation to simulated HH during reading practice and visual exploration practice is task-specific, or whether there is a transfer of practice-related improvements between reading and visual exploration.

The finding that practice-related improvements in reading and visual exploration performance were accompanied by changes of the respective oculomotor measures indicates efficient spontaneous oculomotor adaptation to simulated HH. Even in the absence of any instruction aimed at improving performance, participants spontaneously adapted to simulated HH by developing efficient oculomotor compensation strategies that alleviated their hemianopic reading and visual exploration impairments. It is important to note that these improvements cannot be explained by increases in visual field sparing during the experimental sessions since the accuracy of the simulated visual field border was continuously monitored. Moreover, they can neither be attributed to mere practice effects since performing the RP and VP sessions under normal viewing conditions was not

associated with any performance or oculomotor changes. In addition, there was no evidence of a speed-accuracy trade-off after practice, neither for reading nor for visual exploration performance; participants reiterated the content of each text equally correctly before and after practice and the number of errors during visual exploration decreased significantly. This finding replicates the experiments presented in Chapter 2 and is consistent with previous reports that investigated adaptation processes in artificial visual field loss during reading (Bernard, Scherlen, & Castet, 2007; Fornos, Sommerhalder, Rappaz, Pelizzone, & Safran, 2006; Sommerhalder et al., 2003, 2004) or visual exploration (Zangemeister & Oechsner, 1999; Zangemeister & Utz, 2002).

Yet, more importantly, this experiment demonstrated that efficient spontaneous oculomotor adaptation to visual field loss is highly specific and task-dependent. Uninstructed RP with simulated HH led to significant improvements in reading performance and associated eye-movements but had no effect on visual exploration; likewise, while VP could significantly improve visual exploration performance and associated eye-movements, it had no effect on reading. This lack of transfer of practice-related improvements in performance and eye-movement measures between reading and visual exploration suggests that both visuo-motor abilities require specific oculomotor adaptation processes for their improvement. Neither efficient oculomotor adaptation to visual field loss in reading nor efficient adaptation in visual exploration alone is sufficient to improve both abilities. Efficient spontaneous oculomotor adaptation to a pure visual-sensory dysfunction is task-specific. The finding that the effect of the side of simulated HH on the resulting impairment and practice-related improvement was also task-dependent confirms this assumption and is consistent with previous reports on hemianopic patients (Zihl, 1995a, 2000).

Although reading and visual exploration are both visuo-motor abilities, they are special applications of the visual, attentional and oculomotor systems. The visually and linguistically structured environment as well as the visual material involved in reading imposes a notably different visual sampling strategy than a complex and less systematic

scene. Moreover, the cognitive demands differ quite substantially between reading and visual exploration. In contrast to visual exploration, reading requires not only visual, attentional and oculomotor but also linguistic processes; it is the process of understanding written language (Liversedge & Findlay, 2000; Rayner, 1998). Thus, visual information sampling and processing in reading serve quite different purposes than those in visual exploration and are therefore task-specific.

The finding that visual field loss can be successfully alleviated by oculomotor adaptation shows the functional plasticity of the visual, attentional and oculomotor processes involved in reading and visual exploration. Yet, specificity rather than generality in transfer of adaptation-related oculomotor changes and performance improvements between both abilities suggests that the functional plasticity of these processes is task-dependent. Task-specific limitations in neural and cognitive plasticity across the adult lifespan support this assumption; age-associated reductions in cognitive plasticity seem to be task-specific (Jones et al., 2006). Further evidence stems from mirror reading. The acquisition of mirror reading skill requires specific and systematic practice (Ofen-Noy, Dudai, & Karni, 2003) and seems to be associated with gray matter increase in task-specific processing areas (Ilg et al., 2008). Moreover, the present experiments also may indicate task-specificity in the functional specialisation of the (cortical) oculomotor system (Alahyane et al., 2007).

Task-specificity in spontaneous oculomotor adaptation explains the double dissociation between spontaneous oculomotor adaptation to visual field loss in reading and visual exploration and consequently that of the hemianopic reading and visual exploration impairments (Zihl, 2000), which has been unclear thus far. Analyses of the anatomical basis of these impairments further support the findings presented in this chapter. If injury to the postchiasmatic visual pathway is accompanied by additional injury to the occipital white matter comprising subcortical-cortical reciprocal connections and/or to the posterior thalamus, hemianopic patients do not show efficient spontaneous oculomotor adaptation to visual field loss in reading and their ability to read remains severely impaired (Zihl, 1995a).

The hemianopic visual exploration impairment emerges if the additional injury involves the ipsilateral occipito-parietal cortex and/or posterior thalamus; these patients do not spontaneously adapt to their visual field loss in visual exploration (Zihl, 1995b). These structures are assumed to be part of the distinctive though overlapping networks subserving the control of visual and oculomotor processes in reading (see Chapter 1) or visual exploration (Mort & Kennard, 2003), respectively. Yet, they may also play a significant role in spontaneous oculomotor adaptation to visual field loss in the respective visuo-motor abilities.

Efficient spontaneous oculomotor adaptation and consequent improvements in reading and visual exploration seem to occur only if brain injury is restricted to the postchiasmatic visual pathway. If injury to the postchiasmatic visual pathway is accompanied by additional lesions affecting the occipital white matter, occipitoparietal structures, and/or the posterior thalamus, hemianopic patients either show insufficient or no spontaneous oculomotor adaptation (Zihl, 1995a, 1995b). It is important to note, however, that there are rare reports of hemianopic patients with confined postchiasmatic lesions who nevertheless do not spontaneously compensate for their visual field defect in reading (Upton, Hodgson, Plant, Wise, & Leff, 2003). The high frequency of combined striate/extrastriate lesions in patients with homonymous visual field loss (Hebel & von Cramon, 1987) may explain why efficient spontaneous oculomotor adaptation to visual field loss occurs rarely. Moreover, it is consistent with the observation that patients either start compensating for their visual field defect soon after brain injury or never regain normal reading and visual exploration performance, at least not without systematic treatment (Zihl, 1995a, 1995b, 2000).

In current clinical practice, hemianopic patients with reading and visual exploration impairments receive two distinct compensatory treatments for improving their impaired reading and visual exploration performance. Improving reading in hemianopic patients seems to require practising rather smaller, very precise, systematic and regular horizontal saccadic eye-movements with single words. Improving the hemianopic visual exploration

impairment, in contrast, requires practicing the use of large saccadic eye-movements to enlarge the field of view as well as practicing more systematic and spatially-organised scanning strategies. Treatment-related oculomotor adaptation seems to transfer from processing abstract visual stimulus arrays and visual search displays during training sessions to natural scene viewing, orienting and navigating (Zihl, 2000). Recent evidence suggests, however, that it does not transfer to text reading (Spitzyna et al., 2007). Although a compensatory visual exploration training involving audio-visual stimulation was found to improve reading in hemianopic patients, it is important to note that the evaluation of reading improvement was based only on single-word reading accuracy (Bolognini, Rasi, Coccia, & Làdavas, 2005), which is not sufficient for an ecologically valid assessment of hemianopic dyslexia and related treatment effects (see also Chapter 1).

The finding of specificity rather than generality in transfer of adaptation-related improvements between reading and visual exploration is consistent with current rehabilitation practice and suggests that not only spontaneous but also treatment-related oculomotor adaptation to visual field loss is task-specific. Moreover, since the cumulative effect of practicing reading and visual exploration with simulated HH did not differ between participants who first practiced reading and those who first practiced visual exploration, one may speculate that the treatment sequence in the rehabilitation of the hemianopic reading and visual exploration impairments may not determine the overall treatment outcome.

However, it requires cross-over rehabilitation studies to determine whether these hemianopic impairments are best treated using specific methods and whether there is an optimal treatment sequence. The oculomotor changes and performance improvements that occurred spontaneously in the present experiment's participants may be similar to those of hemianopic patients who receive systematic treatment to reinforce these adaptation processes (Zihl, 1995a, 1995b, 2000). However, this evidence was obtained on the basis of relatively young and well-educated healthy participants but the majority of hemianopic patients are over the age of 55 (Zihl, 2000). Moreover, since neural, functional and cognitive plasticity

changes across the lifespan (Burke & Barnes, 2006; Craik, 2006; Hedden & Gabrieli, 2004; Reuter-Lorenz, 2002; Sowell et al., 2003), age or age-related processes may play a significant role in oculomotor adaptation to visual field loss and therefore (co-)determine not only patients' functional impairments but also the amount of treatment required and the overall rehabilitation outcome. Yet, apart from a single report on the effect of age on spontaneous oculomotor adaptation to simulated and real HH in visual exploration (Tant et al., 2002), it remains to be investigated whether and to what extent age can influence spontaneous and treatment-related oculomotor adaptation to visual field loss.

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Chapter 4

IS THE BASIS OF THE HEMIANOPIC LINE BISECTION ERROR PURELY VISUAL? EVIDENCE FROM EYE-MOVEMENTS IN SIMULATED HEMIANOPIA

The two experiments presented in this chapter investigated whether the hemianopic line bisection error is caused by the visual field defect itself, by strategic adaptation of eye-movements to contralateral hemispace or by additional extrastriate brain injury. To study the behavioural changes associated with the hemianopic visual field defect that are not caused by brain injury, unilateral homonymous hemianopia was simulated in healthy participants. Studying manual and ocular line bisection in simulated hemianopia demonstrated that this visual-sensory deficit impaired line bisection and induced the contralaterally deviated eye-movement pattern of hemianopic patients. However, it did not induce the contralateral hemianopic bisection error. These results suggest that although the visual field defect and oculomotor adaptation to it may contribute to the hemianopic bisection error, they are not its primary causes.

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1. Introduction

Unilateral homonymous hemianopia (HH) is a visual field disorder in which vision is lost in both monocular hemifields contralateral to the side of brain injury. It is caused by postchiasmatic visual pathway injury that is frequently accompanied by extrastriate lesions; posterior cerebral artery infarction is the most common aetiology (Hebel & von Cramon, 1987; Zhang, Kedar, Lynn, Newman, & Biousse, 2006; Zihl, 2000). Hemianopic patients commonly complain of persistent and severe impairments of reading (see Chapter 1) and visual exploration (Zihl, 2000). Evidence suggests that these functional impairments are determined both by the visual field defect and by the degree of strategic oculomotor adaptation to visual field loss. The hemianopic reading and visual exploration impairments have therefore been interpreted as disorders of the visual bottom-up and attentional top-down control of visual processing and eye-movements, which masquerade as failures of vision (see Chapter 2).

It is rather striking that these patients also frequently seem to suffer from a spatial distortion which is reflected by a reliable contralateral deviation in the manual bisection of horizontal lines towards the side of their blind hemifield. This contralateral hemianopic bisection error may be understood as a disorder of the egocentric visual midline in the horizontal plane which becomes manifest as a systematic, contralateral shift of the visual midline or subjective straight-ahead direction in visual-spatial judgements as well as in spatial orientation problems in daily life, such as difficulties with maintaining the straight-ahead direction during walking (Ferber & Karnath, 1999; Kerkhoff, 1999; Zihl, 2000). The hemianopic bisection error is not a deficit in an everyday life task but an indicator of a potentially underlying visual-spatial deficit in HH and therefore also needs to be distinguished from the hemianopic reading and visual exploration impairments. Thus, the line bisection task is a diagnostic and experimental tool to investigate this apparent visual-spatial disorder.

Such a visual-spatial disorder would not be expected with a pure visual-perceptual deficit such as HH and it is therefore not surprising that unfortunately, and despite a much longer history, this contralateral hemianopic line bisection error is less well-known than the ipsilateral bisection error that is frequently associated with visuospatial neglect (Kerkhoff & Bucher, 2008). Axenfeld (1894) was the first to report the hemianopic bisection error. Liepmann and Kalmus (1900) confirmed his report a few years later and termed this contralateral bisection error "hemianopic measurement error". This error is significantly larger than that of normal observers, who typically bisect horizontal lines more or less accurately (Jewell & McCourt, 2000; for the first report on line bisection in normal observers, see Wolfe, 1923). The contralateral bisection error represents a robust symptom that is frequently associated with HH and persists even years after the occurrence of brain injury (Barton, Behrmann, & Black, 1998; Barton & Black, 1998; Doricchi et al., 2005; Hausmann, Waldie, Allison, & Corballis, 2003; Kerkhoff, 1993; Zihl, 2000; Zihl & von Cramon, 1986).

The origin of the hemianopic bisection error, however, remains unclear. Barton and Black (1998) investigated line bisection in a small group of hemianopic patients as well as in patients with unilateral cerebral hemispheric lesions who showed normal visual fields. Based on their finding that the contralateral bisection error was present only in hemianopic patients but not in those with normal visual fields, they suggested two possible explanations for the hemianopic bisection error, which, however, have never been investigated.

The first explanation is that the hemianopic bisection error is a direct consequence of the visual field defect. The contralateral bisection error results from a non-veridical spatial representation within a visual hemifield, since in HH the line is viewed in only one hemifield (Barton & Black, 1998). Evidence from hemifield line bisection in normal participants seems to support the visual origin of the hemianopic bisection error, i.e., that the field defect is a necessary prerequisite for the contralateral bisection error. Bisecting lines viewed in only one hemifield by instructing participants to fixate the left or right line end induces the

contralateral bisection error found in hemianopic patients (Best, 1910a, 1910b; Nielsen, Intriligator, & Barton, 1999). Yet, Best (1910b) found that the bisection error in hemianopic patients was significantly larger than that of healthy observers during hemifield line bisection and therefore dismissed his original hypothesis of a visual origin of the contralateral bisection error. Observations of dissociations between HH and the contralateral bisection error also suggest that the hemianopic visual field defect may not be a necessary condition that causes the contralateral bisection error (Best, 1919; Zihl, 1988, 2000).

According to Barton and Black's (1998) second explanation, the hemianopic bisection error is a manifestation of strategic oculomotor adaptation to visual field loss. Patients who show oculomotor adaptation to visual field loss consistently shift their gaze and, thus, their visual field border, into the area corresponding to their blind hemifield, enabling them to regain sufficient reading and visual exploration performance (Zihl, 2000). Oculomotor adaptation becomes manifest as a change of oculomotor patterns and is possibly best explained as a functional reorganisation of the attentional top-down eye-movement control in reading (see Chapter 1) and visual exploration (Zihl, 2000). Oculomotor adaptation to visual field loss possibly indicates an adaptive attentional bias to contralateral hemispace, which might cause the contralateral line bisection error (Barton & Black, 1998). The slight leftward error normal observers typically show during line bisection (i.e., pseudoneglect), has also been interpreted as reflecting an attentional bias to left hemispace (Fischer, 2001; Jewell & McCourt, 2000). Barton, Behrmann, and Black (1998) studied eye-movements in seven hemianopic patients showing the contralateral bisection error. In contrast to the fixation pattern of normal observers that is concentrated around the centre of the line (Barton et al., 1998; Ishiai, Furukawa, & Tsukagoshi, 1987, 1989), all patients showed a contralateral deviation in the pattern of eye-movements. Although this finding seems to support Barton and Black's (1998) second explanation, i.e. that an adaptive attentional bias to contralateral hemispace is a necessary prerequisite for the contralateral bisection error, their hypothesis was challenged by observations of dissociations between oculomotor adaptation to visual

field loss and the contralateral bisection error (Gassel & Williams, 1963a, 1963b; Williams & Gassel, 1962).

Thus, although the contralateral bisection error is frequently associated with HH, it seems to be separable from both the visual field defect and oculomotor adaptation to it. Alternatively, it has been suggested that additional extrastriate brain injury to regions that are involved in visual-spatial perception might result in the hemianopic bisection error (Best, 1919; Ferber & Karnath, 1999; Kerkhoff, 1993; Zihl, 2000). However, the critical lesion location remains to be investigated. It may include posterior occipito-parietal structures (Best, 1919; Ferber & Karnath, 1999; Kerkhoff, 1993; Zihl, 2000) and/or cortical and subcortical white matter pathways, particularly splenial fibres (Hausmann et al., 2003). The high frequency of extrastriate lesions in patients with HH resulting from postchiasmatic visual pathway injury (Hebel & von Cramon, 1987) may explain why the contralateral bisection error is frequently associated with, but separable from, HH and oculomotor adaptation to it.

In summary, it is still unclear whether the contralateral line bisection error in HH is caused by the visual field defect and/or oculomotor adaptation to visual field loss, or whether hemianopic patients additionally have to deal with the consequences of a visual-spatial deficit caused by additional extrastriate brain injury. Yet, as long as the origin of the hemianopic bisection error is unknown, our understanding of functional impairment in visual field loss remains incomplete and current practice of assessment and rehabilitation imperfect. The purpose of the reported experiments therefore was to identify the visual and adaptive oculomotor (and thus attentional) components that may constitute the hemianopic bisection error and to establish the extent to which this bisection error is purely visually elicited. To do this, HH was simulated in healthy participants by means of a gaze-contingent display. As the experiments in the previous two Chapters 2 and 3 have shown, simulating HH allows studying the behavioural changes associated with the hemianopic visual field defect in the absence of brain injury.

Experiment 1 investigated the effects of simulated HH on manual line bisection performance and associated eye-movements. Measurement of eye-movements helps to elucidate the role of adaptive oculomotor (and thus attentional) factors in causing the hemianopic bisection error. For the same purpose, it was also examined whether the point of bisection may be predicted by the ocular fixation at the time of bisection. A computerised manual line bisection task was developed, and it was determined whether it resembles the conventional paper-and-pencil task that is commonly used to assess line bisection in hemianopic patients.

Experiment 2 studied the effects of simulated HH on line bisection performance and associated eye-movements, not only in a manual bisection task but also in an ocular bisection task without a manual response ("line bisection task by fixation", see Ishiai, Koyama, & Seki, 1998). Investigating ocular line bisection in simulated HH allows establishing both the role of adaptive oculomotor factors in causing the hemianopic bisection error, as well as further investigation of the assumption that the point of bisection may be predicted by the ocular fixation at the subjective line centre. Comparing ocular and manual line bisection performance and eye-movements also allows disentangling the contributions of adaptive oculomotor/attentional factors from the possible impact of manual motor factors. In addition, it was investigated whether performing the ocular bisection task may influence line bisection performance in a subsequent manual bisection task (and vice versa).

2. Experiment 1: The effects of simulated hemianopia on manual line bisection

2.1. Methods

Participants

In Experiments 1 and 2 two different groups of naïve, healthy participants with normal or corrected-to-normal vision were tested. Only right-handed participants with a laterality quotient of >+80 in the Edinburgh Handedness Inventory (Oldfield, 1971) were included in order to eliminate the effects of handedness, which is a significant factor modulating

bisection performance in line bisection (Jewell & McCourt, 2000). All participants were native English speakers and had no reading disorders, visual disorders or any other neurological disease or psychiatric condition, and gave their informed consent in accordance with the Declaration of Helsinki and with local ethical committee approval. In Experiment 1 twelve participants were tested (9 males, 3 females; mean age: 32.0 years (SD: 13.3); years of education: 11.2 years (SD: 3.5)).

Eye-movement recording and simulating hemianopia

The methods used for eye-movement recording and simulating left- and right-sided HH (LHH, RHH) in healthy participants were identical to those used in the experiments presented in the previous Chapters 2 and 3, which demonstrated that these methods successfully induce the reading and visual exploration impairments matching those of hemianopic patients (see Fig. 1). The monitor used for stimulus presentation was also identical, except that a Keytech touch screen (KTMT-1700, 17'') was mounted upon the monitor.

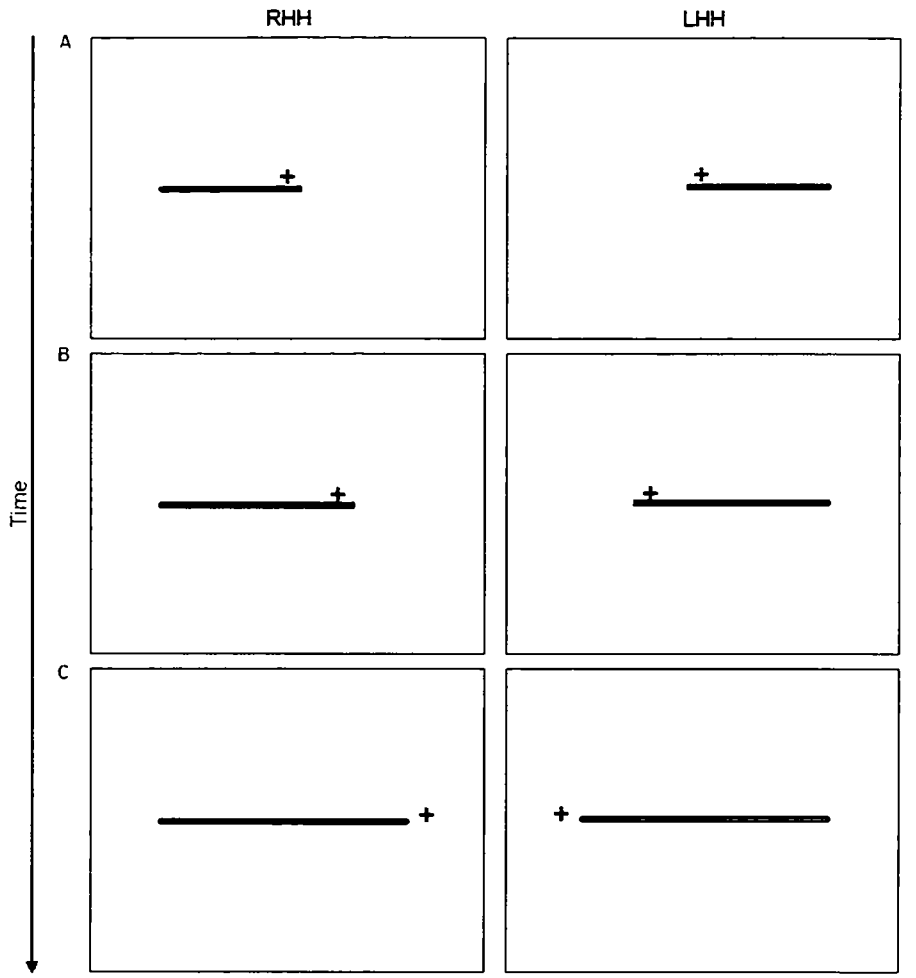

Fig. 1. Schematic illustration of right- and left-sided simulated hemianopia during line bisection (RHH, LHH); the gaze-contingent display paradigm blanks the side to the right or left of current fixation (visual field sparing: 1°). Potential fixation sequences are illustrated (the cross indicates potential fixation positions of a participant); RHH: scanning the line from the centre (**A**) to its right end (**C**), LHH: scanning the line from the centre (**A**) to its left end (**C**).

Assessment of manual line bisection

For assessing manual line bisection and associated eye-movements a computerised manual line bisection task was devised that resembles the conventional paper-and-pencil bisection task in which lines are presented on a paper sheet and are bisected using a pencil; this task is typically used with hemianopic patients (for the only exceptions, see Barton et al., 1998; Kerkhoff, 1993). The most common computerised line bisection task, in contrast, involves using a mouse-controlled cursor for line bisection. Since this task involves different cognitive and motor demands than a line bisection task using a reaching action (Dellatolas,

Vanluchene, & Coutin, 1996; Luh, 1995; Rolfe, Hamm, & Waldie, 2008), it was not used for assessing manual line bisection in the present experiments.

Short (5.3 cm, 8° of visual angle), medium (8.1 cm, 12°) and long (10.9 cm, 16°) horizontal lines (width: 0.3 cm) were presented, one at time, in the centre of a touch-sensitive monitor screen. Luminance of the black lines was 0.2cd/m^2, against a white background of 27cd/m^2. Ten lines of each length were presented in randomised sequence. The centre of each line was aligned with the participants' midsagittal plane. Participants were instructed to touch the centre of each line (i.e., subjective line centre) as accurately as possible by using a fine touch screen pen (Palm Inc.). There was no preceding fixation dot. Participants were asked to make sure to have seen the entire line, i.e., both line ends, before touching the position they perceived to be its centre (Liepmann & Kalmus, 1900). Viewing time was unlimited and participants were free to move their eyes. Touching the line initiated the next trial (ISI=1000 ms). Participants received no visual feedback on their touch position or its accuracy in order to eliminate practice effects and to ensure that subsequent bisections were not biased. Eye-movement recording started with the onset of line presentation and ended after the participant touched the line.

For assessing line bisection performance the response position was used to calculate the deviation from the left or right of the objective line centre. The signed error (°) is reported as a measure of error direction. A negative or positive value indicates a leftward or rightward bisection error, respectively. In addition, the absolute error (°) is reported as a measure of error magnitude. The time required to bisect each line was also measured, i.e., the time elapsed between onset of line presentation and the response (bisection time).

For assessing eye-movements during line bisection the horizontal positions (°) of the following fixations were analysed: (1) the bisection fixation (i.e., the fixation at the time of bisection), (2) the maximum fixation (i.e., the fixation with the longest duration), and (3) the left- and right-most fixations (negative and positive values indicate fixation positions to the left and right of the lines' centre, respectively). The (4) horizontal fixation range (the

distance between left- and right-most fixation positions) as well as the (5) number and (6) duration (ms) of left- and right-hemispace fixations (i.e., the fixations spent in left and right hemispace defined with respect to the centre of the screen) were also analysed. In addition to analysing measures indicating the horizontal fixation distribution, the (7) number and (8) mean amplitude (°) of left- and rightward saccades (indicating the direction of the eye-movements used to inspect each line) were analysed. (9) The scanpath length (the sum of saccadic amplitudes) (°), which indicates the efficacy of visual information extraction in visual field loss (Zihl, 2000), is also reported.

Assessment of touch position measurement accuracy and paper-based line bisection

For assessing the accuracy of the touch position measurement in the manual line bisection task, a pre-transected manual line bisection task was devised. This task was identical to the manual line bisection task, except that pre-transected lines in which the lines' centres were marked with small, vertical transection marks were presented (data were obtained from participants in Experiment 2 (n=20) who performed this task at the end of the experiment). This pre-transected manual line bisection task is similar to the "Landmark Task" (Milner, Brechmann, & Pagliarini, 1992), except that the transection marks were always at the centre of each line and participants were instructed to touch the centre-mark of each presented line as accurately as possible. The absolute deviation of each touch position to the centre mark was calculated.

To investigate whether the computerised manual bisection task resembles the conventional paper-and-pencil line bisection task, paper-and-pencil line bisection performance was assessed. Materials, instruction and procedure were identical to those used in the computerised manual bisection task, except that lines were presented in the centre of separate white paper sheets, one at a time; test sheets were aligned with the participant's midsagittal plane. After marking the subjective line centre, the experimenter immediately exchanged the test sheet and presented the next line. The paper-and-pencil line bisection task

was performed under normal daylight conditions. The position of each bisection mark was measured to 0.5 mm (0.08°) accuracy and expressed in °.

Procedure

Participants were instructed to bisect each line using their right hand in order to eliminate the effects of hand use, which is also a significant factor modulating bisection performance (Jewell & McCourt, 2000). To control the initial starting position of oculomotor and gross motor scanning participants were instructed to begin visually scanning the line in the centre of the screen and to rest their hand on the table in a position aligned with the screen centre between trials. All participants performed the computerised manual line bisection task with simulated LHH, RHH and in a normal viewing condition, i.e., without any simulated HH (N). Task performance in the normal viewing condition was obtained at the end of the task. The sequence of simulation-conditions (starting with LHH or RHH) was counterbalanced across participants to eliminate order effects. After completion of the computerised manual line bisection task and a short break, participants performed the conventional paper-based line bisection task under normal viewing conditions.

Data analyses

To evaluate whether line bisection performance in the computerised and paper-and-pencil bisection task is comparable a repeated measures ANOVA was performed on the measurements of signed and absolute error, with task (computerised, paper-based) and line length (small, medium, long) as within-subject factors. To investigate the effects of simulated HH on line bisection performance and eye-movements, a repeated measures ANOVA was performed, with simulation-condition (LHH, RHH, N) and line length (small, medium, long) as within-subject factors. Where sphericity assumptions were violated as assessed by Mauchly's W test, the Greenhouse-Geisser correction to the degrees of freedom was applied. Post-hoc paired comparisons between simulation-conditions, line lengths and tasks were performed using repeated measures t-tests. As multiple tests were carried out, the significance level was adjusted using a Bonferroni correction to an alpha-level of 0.05 for

multiple comparisons. In addition, Pearson's correlations (two-tailed) between the horizontal bisection point and the position of the fixation at the time of bisection for each simulation-condition were calculated. 3.4% of trials were excluded from the analyses.

2.2. Results

The effects of simulated hemianopia on manual line bisection performance

Before assessing the effects of simulated hemianopia on line bisection the accuracy of the touch-screen system was measured using the pre-transected line bisection task. The mean absolute error between the marked centres and the measured touch positions was 0.10° (SD: 0.04) for all simulation conditions. Moreover, the touch-screen based manual line bisection task can also reasonably be used as a substitute for the conventional paper-based bisection task since there were no differences in error magnitude (absolute error) and direction (signed error) between tasks (larger $F_{(1.0,11.0)}=0.36$, $p=0.561$). The significant effect of line length for absolute error ($F_{(1.5,16.3)}=26.05$, $p<0.001$) disappeared when the error was expressed as a proportion of line length (largest $F_{(1.3,14.3)}=3.54$, $p=0.072$) as would be expected given Weber's Law for Position.

This experiment's main result was that in standard (non pre-transected) manual line-bisection simulated HH of either sort induced an ipsilateral bisection error (i.e., towards the intact hemifield), as well as increased bisection times (see Table 1); although contralateral errors did occur, they were less frequent (see Table 1) and smaller than ipsilateral errors (RHH: $t_{(10)}=3.16$, $p=0.010$, non-significant for LHH: $t_{(9)}=-1.83$, $p=0.147$; two-tailed repeated measures t-tests).

Table 1 Manual line bisection performance in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [means (SD) calculated over all line lengths].

	LHH	RHH	N	N-LHH	N-RHH	LHH-RHH
Overall bisection error						
Signed error						
(')	+0.4 (1.0)	–0.4 (0.7)	–0.1 (0.2)	*	*	*
[% of line length]	[+3.4 (8.3)]	[–3.9 (5.9)]	[–0.8 (1.7)]			
Absolute error						
(')	0.7 (0.8)	0.6 (0.6)	0.2 (0.1)	*	*	n.s.
[% of line length]	[6.2 (6.5)]	[5.2 (4.9)]	[1.5 (1.1)]			
Leftward bisection error						
(%)	42.1	75.4	66.7	*	n.s.	*
(°)	0.3 (0.3)	0.7 (0.6)	0.2 (0.1)	*	*	*
[% of line length]	[3.3 (2.3)]	[6.0 (5.2)]	[1.7 (1.2)]	*	*	*
Rightward bisection error						
(%)	57.3	24.0	29.9	*	n.s.	*
(°)	1.0 (1.0)	0.3 (0.2)	0.1 (0.1)	*	*	*
[% of line length]	[8.4 (7.7)]	[2.5 (2.4)]	[1.2 (0.8)]	*	*	*
Correct bisections (%)	0.6	0.6	3.4	n.s.	n.s.	n.s.
Bisection time (s)	6.6 (3.5)	7.1 (2.6)	4.4 (2.8)	*	*	n.s.

Statistical comparisons were made between LHH, RHH, and N (two-tailed dependent samples t-tests, except for frequency of left- and rightward errors and correct bisections: two-tailed Pearson's chi-square test). * indicates $p<0.017$ (α_{corr}), n.s. indicates non-significant comparisons.

Under normal viewing conditions, in contrast, lines were bisected quickly and more or less accurately (see Table 1); although a slight leftward error was obtained, it was significantly smaller than the bisection errors induced by simulated HH (significant effect of simulation-condition; smallest $F_{(2,22)}=5.25$, $p=0.014$). Leftward errors were more frequent but not larger than rightward errors (see Table 1; $t_{(9)}=0.90$, $p=0.393$; two-tailed repeated measures t-test). These results are substantiated by the finding that error direction was determined by simulation-condition ($\chi^2_{(4)}=28.00$, $p<0.001$; two-tailed Pearson's chi-square test). Line length had no effect on line bisection performance. Although errors increased with increasing line length (absolute error: $F_{(1.1,12.6)}=11.00$, $p<0.001$; signed error: $F_{(1.3,14.3)}=3.73$, $p=0.065$), errors remained invariant across line lengths when expressed as a proportion of line length (largest $F_{(1.4,14.9)}=1.82$, $p=0.20$).

The effects of simulated hemianopia on eye-movements during manual line bisection

Under normal viewing conditions, participants showed a symmetrical distribution of fixations that was concentrated around the objective line centre. Simulated HH of either sort induced a contralateral deviation of the eye-movement pattern (significant effect of simulation-condition for all oculomotor parameters; smallest $F_{(2,22)}=9.19$, $p=0.001$) (see Table 2).

Table 2 Eye-movements during manual line bisection in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [means (SD) calculated over all line lengths].

	LHH	RHH	N	N-LHH	N-RHH	LHH-RHH
Horizontal position (°) of the						
Bisection fixation	−1.3 (1.6)	+1.6 (1.7)	−0.1 (0.6)	*	*	*
Maximum fixation	−3.7 (2.3)	+2.6 (2.4)	−0.2 (0.7)	*	*	*
Leftmost fixation	−8.9 (3.4)	−4.0 (2.3)	−3.9 (3.8)	*	n.s.	*
Rightmost fixation	+3.1 (2.9)	+9.3 (3.2)	+3.0 (3.3)	n.s.	*	*
Fixation range (°)	12.0 (4.9)	13.3 (4.2)	6.9 (5.8)	*	*	n.s.
Right-hemispace fixations						
Number	17.9 (15.2)	58.2 (21.9)	9.89 (9.9)	*	*	*
Duration (ms)	500.6 (311.7)	453.7 (159.9)	419.2 (170.1)	*	*	*
Left-hemispace fixations						
Number	48.3 (23.3)	22.4 (12.1)	9.36 (7.0)	*	*	*
Duration (ms)	560.7 (270.4)	448.9 (165.3)	493.5 (269.1)	*	*	*
Rightward saccades						
Number	34.6 (17.9)	38.8 (13.4)	10.4 (7.9)	*	*	n.s.
Amplitude (°)	2.7 (1.1)	3.5 (1.5)	2.3 (1.0)	n.s.	*	*
Leftward saccades						
Number	30.4 (16.6)	43.5 (16.1)	8.7 (7.0)	*	*	n.s.
Amplitude (°)	3.5 (1.9)	2.4 (0.6)	2.5 (1.1)	*	n.s.	*
Scanpath length (°)	191.5 (113.1)	229.1 (89.6)	50.0 (49.0)	*	*	n.s.

Statistical comparisons were made between LHH, RHH, and N (two-tailed dependent samples t-tests).
* indicates $p<0.017$ (α_{corr}), n.s. indicates non-significant comparisons.

Analysing the left- and rightmost fixation positions revealed that participants scanned further into their blind hemifield than into their intact field; the fixation with the longest duration also showed a contralateral deviation. Consistent with this observation analyses revealed a contralaterally skewed horizontal fixation distribution during line bisection with simulated HH of either sort. Participants made significantly more fixations on the side of space corresponding to their blind hemifield (smaller $t_{(11)}$=4.95, p<0.001). Under normal viewing conditions, however, fixations were equally distributed in left- and right-hemispace ($t_{(11)}$= –0.28, p=0.788) (two-tailed repeated measures t-tests).

Although there was no significant effect of simulation-condition on fixation duration and saccadic amplitudes (largest $F_{(1.4,15.1)}$=2.50, p=0.105), post-hoc comparisons revealed that these measures were significantly and differentially affected by simulated HH (see Table 2). During line bisection with simulated HH fixation durations increased and participants made larger saccades towards the blind field than towards the intact hemifield (RHH: $t_{(11)}$= –2.55, p=0.027; LHH: $t_{(11)}$=1.88, p=0.087); under normal viewing conditions, however, saccadic amplitudes did not differ between directions ($t_{(11)}$=1.29, p=0.225) (two-tailed repeated measures t-tests). As would be expected given these results, it was found that the spatial range covered by fixations was considerably larger, scanpaths significantly longer and participants made more saccades (both to the left and right) during line bisection with simulated HH than under normal viewing conditions (see Table 2).

The horizontal range of fixations increased with increasing line length under normal viewing conditions (significant difference between the small and long line; $t_{(11)}$= –8.07, p<0.001) but remained constant across lengths during line bisection with simulated HH (RHH: largest $t_{(11)}$= –1.19, p=0.260; LHH: largest $t_{(11)}$= –2.14, p=0.056); the same effect was obtained for the positions of the left- and rightmost fixation positions (significant main and interaction effect line length smallest $F_{int(4,44)}$=3.41, p=0.016). Line length did not affect the contralateral deviation of the leftmost fixation in LHH or that of the rightmost fixation in RHH (largest $t_{(11)}$=0.70, p=0.499). It did, however, affect the rightmost fixation in LHH and

the leftmost fixation in RHH as well as both fixation positions under normal viewing conditions; both fixations were shifted further to the left or right, respectively, with increasing line length (smallest $t_{(11)}= -2.85$, $p=0.016$).

The relationship between the point of bisection and the fixation at the time of bisection

Simulated HH of either sort induced a contralateral deviation of the fixation at the time of bisection (see Table 2). During line bisection with RHH, the same large deviation was present irrespective of the direction of the bisection error (largest $t_{(10)}=0.42$, $p=0.686$). During line bisection with LHH, the magnitude of the contralateral deviation depended on error direction; it was significantly larger for contralaterally deviated bisections than for ipsilateral bisections ($t_{(9)}= -2.41$, $p=0.039$). Under normal viewing conditions, the fixation at the time of bisection showed only a slight deviation whose direction depended on the direction of the error. For leftward bisections, it was shifted to the left; for rightward bisections, it was shifted slightly to the right. Yet, the magnitude of this deviation did not differ between left- and rightward bisections ($t_{(9)}= -1.20$, $p=0.260$) (two-tailed repeated measures t-tests).

There was a significant correlation between the position of the fixation at the time of bisection and the manual bisection position for both types of simulated HH (smaller $r=0.17$, $p=0.001$) and under normal viewing conditions ($r=0.11$, $p=0.047$). These effects nevertheless differed depending on direction of the bisection error with simulated HH. During line bisection with simulated HH, significant correlations were only found when subjects made ipsilateral bisection errors (smaller $r=0.24$, $p<0.001$; contralateral errors: larger $r= -0.13$, $p=0.127$). Under normal viewing conditions significant correlations were only found for rightward errors ($r=0.20$, $p=0.045$; leftward errors: $r= -0.01$, $p=0.929$).

2.3. Discussion

The results demonstrate that simulated HH of either sort induced an ipsilateral bisection error that was significantly larger than the typical, small leftward bisection error that was obtained

under normal viewing conditions (Jewell & McCourt, 2000). The contralateral bisection errors that did occur were smaller and less frequent than ipsilateral errors. These effects differ from the common observation of a reliable and much larger contralateral bisection error in hemianopic patients (Barton et al., 1998; Barton & Black, 1998; Doricchi et al., 2005; Hausmann et al., 2003; Kerkhoff, 1993; Zihl, 2000; Zihl & von Cramon, 1986). Although simulated HH did not induce the bisection error found in hemianopic patients it produced the same contralateral deviation in the pattern of eye-movements that is shown by patients during line bisection; this deviation suggests the presence of strategic oculomotor adaptation to contralateral hemispace (Barton et al., 1998; Ishiai et al., 1987, 1989).

The observation of large, predictive overshooting saccades into the blind hemifield (i.e., a contra-directional saccadic bias) further supports the presence of oculomotor adaptation to simulated HH (Gassel & Williams, 1963a; Meienberg, Zangemeister, Rosenberg, Hoyt, & Stark, 1981; Williams & Gassel, 1962; Zangemeister, Oechsner, & Freska, 1995; Zangemeister & Utz, 2002; Zihl, 2000). By shifting gaze, and thus the simulated visual field boundary, towards the blind hemifield participants can bring obscured visual information about the extent of the presented line into their seeing hemifield. The experiments presented in Chapters 2 and 3 demonstrated that oculomotor adaptation to simulated HH occurs spontaneously and rapidly, even in the absence of any instruction aimed at improving participants' performance. The finding of a symmetrical and centred oculomotor scanning pattern under normal viewing conditions confirms prior observations that healthy participants mainly scan the centre of the lines (Barton et al., 1998; Ishiai et al., 1987, 1989).

Fixation position at the time of bisection may be an important factor in predicting the ipsilateral bisection error in simulated HH as indicated by the significant correlations that were found between the ipsilaterally deviated point of bisection and the position of the fixation at bisection. The contralateral deviation of this fixational measure was more pronounced for contralateral errors but these were not predicted by the fixation at the time of

bisection. Under normal viewing conditions, the fixation at the time of bisection deviated in the same direction as the bisection error but it seems only to predict the bisection positions in rightward errors. These findings are consistent with evidence from line bisection in visual neglect suggesting that the placement of the bisection mark may be predicted by an ocular fixation at the time of bisection (Ishiai et al., 1989; Ishiai et al., 1998).

3. Experiment 2: The effects of simulated hemianopia on ocular line bisection

To further investigate the significance of oculomotor (and thus attentional) factors in line bisection with simulated HH and to establish the extent to which line bisection performance is determined by the manual motor component of the bisection task, Experiment 2 was conducted. Here line bisection was studied both in computerised and paper-based manual bisection tasks as well as in an ocular bisection task without manual response (Ishiai et al., 1998). In addition, it was investigated whether performing the ocular bisection task may influence line bisection performance in a subsequent manual bisection task (and *vice versa*).

3.1. Methods

Participants

Twenty participants were tested (12 males, 8 females; mean age: 19.1 years (SD: 1.3); years of education: 12.4 years (SD: 0.7)).

Eye-movement recording and simulating hemianopia

Methods for eye-movement recording and simulating HH were identical to those used in Experiment 1.

Assessment of ocular line bisection

For examining ocular line bisection a computerised version of Ishiai, Koyama and Seki's (1998) "line bisection task by fixation" was devised. The ocular line bisection task was identical to the manual line bisection task used in Experiment 1, except that the response-mode was ocular; in addition, longer lines (small: 13.6 cm (19.7°), medium: 16.6 cm (23.6°),

long: 19.6 (27.3°)) were used and five instead of ten lines were presented for each length. Participants were instructed to fixate the centre of each presented line as accurately as possible. Upon stable fixation of the position they perceived to be the line's centre, the next trial was initiated via mouse-click. Eye-movement recording started with the onset of line presentation and ended by mouse-click.

The analysis of ocular line bisection performance and eye-movement parameters was identical to Experiment 1, except that the horizontal positions of the 'bisection'-fixation were used instead of the touch positions.

Assessment of manual line bisection

The manual line bisection task and methods that were used to assess and analyse manual line bisection performance and oculomotor parameters was identical to Experiment 1.

Assessment of 'bisection'-fixation and touch position measurement accuracy and paper-based line bisection

In order to assess the accuracy of 'bisection'-fixation and touch position measurements the pre-transected manual line bisection task described in Experiment 1 was used, except that for assessing 'bisection'-fixation position measurement accuracy (pre-transected ocular line bisection task) participants were instructed to fixate the centre-mark of each presented line as accurately as possible. The results of the manual version of the task have already been presented in Experiment 1.

In addition, paper-based line bisection performance was assessed to establish the extent to which paper-based line bisection performance is predicted by the manual motor component of the bisection task. The same paper-and-pencil line bisection task was used as in Experiment 1, except that line lengths were larger (small: 13.6 cm (19.7°), medium: 16.6 cm (23.6°), long: 19.6 (27.3°)) and five instead of ten lines were presented for each length.

Procedure

All participants performed the ocular and manual line bisection task with LHH, RHH and in a normal viewing condition, i.e., without any simulated HH (N). Normal viewing condition was the final test condition for every participant. The sequence of simulation-conditions (starting with LHH or RHH) was counterbalanced across participants to eliminate order effects. Since performing the ocular bisection task may influence line bisection performance in a subsequent manual bisection task (and *vice versa*), participants were randomly allocated into two equal groups (n=10); Group A first performed the manual, then the ocular line bisection task (mean age: 19.4 years (1.7); years of education: 12.5 (0.8); 2 females, 8 males), Group B performed the tasks in the opposite order (mean age: 18.8 years (0.6); years of education: 12.3 (0.6); 6 females, 4 males). After completion of the computerised line bisection tasks, the baseline accuracy of manual and ocular line bisection performance was assessed using pre-transected lines. Finally, participants performed the paper-and-pencil line bisection task under normal viewing conditions.

Data analyses

The analyses for testing the effects of simulated HH on ocular and manual line bisection performance and eye-movements were identical to Experiment 1, except that task-sequence (Group A, B) was used as an additional between-subject factor. The same analysis was used for testing the effects of response-mode by including response-mode (manual, ocular) as an additional within-subject factor. In addition, bisection performance was compared between the computerised manual, ocular and paper-and-pencil bisection task (signed and absolute error under normal viewing conditions) by performing a repeated measures ANOVA with task and line length as within-subjects factors. Task-sequence was a between-subject factor in both analyses. Post-hoc paired comparisons between simulation-conditions, tasks and line lengths were performed using repeated measures t-tests. Corrections for violations of sphericity assumptions and multiple comparisons were identical to those used in Experiment 1. The analyses to further investigate the hypothesis that the point of bisection may be

predicted by the ocular fixation at the subjective line centre were also identical to those used in Experiment 1; in addition Pearson's correlations (two-tailed) between the manual and ocular signed bisection errors were calculated. 1.3% of trials were excluded from the analyses of the manual line bisection data, 2.3% of trials from the analyses of the ocular line bisection data.

3.2. Results

The effects of simulated hemianopia and task-sequence on ocular and manual line bisection performance, and the effects of response-mode

The effects of simulated hemianopia and task-sequence on ocular line bisection

The accuracy of the 'bisection'-fixation position measurements in the pre-transected ocular line bisection task was 0.15° (SD: 0.21) for all viewing conditions (mean absolute deviation for all line lengths).

The patterns of effects of simulated HH on the magnitude and direction of the bisection error and bisection time during ocular line bisection were identical to those observed in Experiment 1, except that ocular bisection errors were slightly larger. The analyses also revealed the same slight leftward error under normal viewing conditions (see Table 3; significant effect of simulation-condition, smallest $F_{(1.2,22.3)}=15.00$, $p<0.001$; $\chi^2_{(4)}=75.20$, $p<0.001$). The ipsilateral errors during line bisection with a simulated HH of either sort were not only more frequent (see Table 3) but also significantly larger than the contralateral errors (smaller $t_{(16)}= -3.26$, $p=0.005$). Under normal viewing conditions, the leftward errors were more frequent but not larger than rightward errors ($t_{(15)}=1.24$, $p=0.233$) (repeated measures t-tests) (see Table 3). As with manual line bisection, ocular line bisection was not affected by line length (largest $F_{(1.4,26.1)}=0.95$, $p=0.372$).

Table 3 Ocular line bisection performance in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [means (SD) calculated over all line lengths].

	LHH	RHH	N	N-LHH	N-RHH	LHH-RHH
Overall bisection error						
Signed error						
(°)	+1.0 (1.7)	−1.4 (1.6)	−0.4 (0.8)	*	*	*
[% of line length]	[+4.2 (7.1)]	[−5.8 (7.1)]	[−1.5 (3.5)]			
Absolute error						
(°)	1.4 (1.4)	1.6 (1.4)	0.7 (0.6)	*	*	n.s.
[% of line length]	[6.0 (5.7)]	[6.9 (6.1)]	[2.9 (2.5)]			
Leftward bisection error						
(%)	23.7	80.6	64.1	*	n.s.	*
(°)	0.9 (0.7)	1.8 (1.4)	0.8 (0.6)	n.s.	*	*
[% of line length]	[3.8 (2.8)]	[7.9 (6.3)]	[3.5 (2.8)]	n.s.	*	*
Rightward bisection error						
(%)	73.9	16.3	31.7	*	*	*
(°)	1.6 (1.5)	0.8 (0.6)	0.5 (0.3)	*	*	*
[% of line length]	[7.0 (6.1)]	[3.3 (2.8)]	[2.1 (1.4)]	*	*	*
Correct bisections (%)	2.4	3.1	4.1	n.s.	n.s.	n.s.
Bisection time (s)	7.0 (3.3)	7.2 (3.7)	4.6 (2.4)	*	*	n.s.

Statistical comparisons were made between LHH, RHH, and N (two-tailed dependent samples t-tests, except for frequency of left- and rightward errors and correct bisections: two-tailed Pearson's chi-square test). * indicates $p<0.017$ (α_{corr}), n.s. indicates non-significant comparisons.

There was no effect of the order in which participants undertook the manual and ocular bisection tasks on ocular bisection performance (the largest task-sequence main or interaction effect is non-significant: $F_{(2,36)}=2.06$, $p=0.143$).

The effects of simulated hemianopia and task-sequence on manual line bisection

Although the effects of simulated HH on the magnitude of the manual bisection error and bisection time were identical to those found in Experiment 1 (see Table 4; significant effect of simulation-condition; smaller $F_{(2,36)}=34.57$, $p<0.001$) and the non-significant effect of line length was replicated (largest $F_{(2,36)}=2.50$, $p=0.10$), the ipsilateral bisection error during line bisection with simulated HH was not obtained ($F_{(1.1,20.0)}=0.02$, $p=0.919$); ipsi- and contralateral errors were equally frequent (see Table 4) and of equal magnitude (larger $t_{(16)}=0.19$, $p=0.850$; repeated measures t-tests).

Table 4 Manual line bisection performance in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [means (SD) calculated over all line lengths].

	LHH	RHH	N	N-LHH	N-RHH	LHH-RHH
Overall bisection error						
Signed error						
(°)	–0.07 (1.0)	–0.05 (1.2)	–0.03 (0.4)	n.s.	n.s.	n.s.
[% of line length]	[–0.3 (4.2)]	[–0.3 (4.7)]	[–0.1 (1.7)]			
Absolute error						
(°)	0.8 (0.6)	0.9 (1.2)	0.3 (0.3)	*	*	n.s.
[% of line length]	[3.3 (2.6)]	[3.5 (3.1)]	[1.3 (1.0)]			
Leftward bisection error						
(%)	56.0	50.7	52.0	n.s.	n.s.	n.s.
(°)	0.8 (0.8)	0.9 (0.7)	0.3 (0.2)	*	*	n.s.
[% of line length]	[3.2 (2.1)]	[3.7 (3.0)]	[1.4 (0.9)]	*	*	n.s.
Rightward bisection error						
(%)	43.7	49.0	45.7	n.s.	n.s.	n.s.
(°)	0.9 (0.7)	0.8 (0.8)	0.3 (0.3)	*	*	n.s.
[% of line length]	[3.5 (3.1)]	[3.3 (3.1)]	[1.3 (1.1)]	*	*	n.s.
Correct bisections (%)	0.3	0.3	2.3	n.s.	n.s.	n.s.
Bisection time (s)	6.9 (2.7)	6.8 (2.5)	3.6 (1.5)	*	*	n.s.

Statistical comparisons were made between LHH, RHH, and N (two-tailed dependent samples t-tests, except for frequency of left- and rightward errors and correct bisections: two-tailed Pearson's chi-square test). * indicates $p<0.017$ (α_{corr}), n.s. indicates non-significant comparisons.

There was a slight leftward error not only under normal viewing conditions but also for line bisection with simulated HH (see Table 4). The leftward errors under normal viewing conditions were slightly larger than rightward errors ($t_{(18)}=1.95$, $p=0.068$, marginal; repeated measures t-test). These results are substantiated by the finding that error direction was not determined by simulation-condition ($\chi^2_{(4)}=4.54$, $p=0.371$; two-tailed Pearson's chi-square test).

It was examined whether the absence of an ipsilateral bisection error during line bisection with simulated HH was accounted for by task-sequence. It was found that the main result of Experiment 1 was replicated in participants who performed the ocular bisection task first (n=10). They showed slightly more and larger ipsilateral than contralateral bisection errors during manual line bisection with simulated HH (LHH: $t_{(8)}=3.88$, $p=0.006$; non-

significant for RHH: $t_{(8)}= -1.11$, p=0.297); these effects were not evident in participants who first performed the manual bisection task (larger $t_{(8)}=0.89$, p=0.401) (repeated measures t-tests).

Moreover, it was found that participants who first performed the ocular bisection task showed slightly smaller bisection errors during line bisection with simulated HH (RHH: 0.70° (SD: 0.42), LHH: 0.79° (SD: 0.29)) than those who performed the manual bisection task first (RHH: 1.04° (SD: 0.53), LHH: 0.84° (SD: 0.41)), although this difference only reached marginal significance for RHH ($t_{(18)}=1.89$, p=0.075; LHH: $t_{(18)}=0.46$, p=0.652); this tendency was not evident under normal viewing conditions ($t_{(18)}= -0.48$, p=0.641) (independent samples t-tests; significant interaction between task-sequence and simulation condition: $F_{(2,36)}=3.72$, p=0.034).

The effects of response-mode

The differences in the effects of simulated HH on line bisection performance between the ocular and manual line bisection task obtained in the present experiment are substantiated by a significant effect of response-mode for the absolute error (measure of error magnitude) and its significant interaction with simulation-condition for the signed error (measure of direction and magnitude) (smaller $F_{(1,18)}=32.35$, p<0.001).

Conducting the same analysis (i.e., repeated measures ANOVA with response-mode, simulation-condition and length as within-subject factors and task-sequence as a between-subject factor) but using the manual line bisection data obtained in Experiment 1 showed, however, that line bisection performance with simulated HH did not differ between the ocular and manual task. In contrast to the previous analysis, the significant main and interaction effects only indicate a difference in magnitude but not in direction between ocular and manual bisection errors with simulated HH (Tables 1, 3; smaller $F_{(2,22)}=7.35$, p=0.004).

Despite these differences significant correlations were obtained between ocular and manual bisection errors for line bisection with simulated HH of either sort (smaller r=0.33, p=0.009) but not under normal viewing conditions (r= –0.12, p=0.356). Moreover,

participants required the same amount of time for manual and ocular line bisection (Tables 3, 4; larger $F_{(1,18)}$=2.61, p=0.124).

Comparing computerised ocular, manual and paper-based line bisection performance under normal viewing conditions revealed a slight leftward bisection error, irrespective of the task used to assess bisection performance. This error was largest in the ocular bisection task (smaller $t_{(19)}$= –4.24, p<0.001) and did not differ between the two manual bisection tasks ($t_{(19)}$= –0.61, p=0.552) (repeated measures t-tests); significant effect of task: absolute error $F_{(1.2,21.8)}$=17.93, p<0.001, signed error $F_{(1.2,21.8)}$=3.88, p=0.055). The significant effect of line length for absolute error ($F_{(1.8,32.7)}$=5.61, p=0.010) disappeared when expressed as a proportion of line length ($F_{(1.8,32.3)}$=2.01, p=0.154); there was no effect of task-sequence (largest $F_{(1.4,44.2)}$=1.68, p=0.165).

The effects of simulated hemianopia and task-sequence on eye-movements during ocular and manual line bisection, and the effects of response-mode

The effects of simulated hemianopia and task-sequence on ocular and manual line bisection

The use of longer lines explains the greater left- and rightward deviation of fixational measures, the larger range of fixations and the longer scanpaths that were obtained in the present experiment when compared to Experiment 1. Eye-movement patterns during ocular line bisection with simulated HH showed the same contralateral deviation that was obtained during manual line bisection in the previous and present experiment (Tables 5, 6) (significant main effect of simulation-condition for all oculomotor parameters; ocular: smallest $F_{(2,36)}$=6.02, p=0.006; manual: smallest $F_{(2,36)}$=4.54, p=0.017). In contrast to manual line bisection, however, the horizontal range of fixations did not differ between viewing conditions ($F_{(1.5,26.3)}$=0.24, p=0.785) and the differences in scanpath length were less consistent ($F_{(2,36)}$=3.03, p=0.061).

Table 5 Eye-movements during ocular line bisection in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [means (SD) calculated over all line lengths].

	LHH	RHH	N	N-LHH	N-RHH	LHH-RHH
Horizontal position (°) of the						
Maximum fixation	–3.0 (4.2)	+2.4 (4.5)	–0.2 (4.1)	*	*	*
Leftmost fixation	–15.9 (2.6)	–9.6 (5.2)	–13.2 (2.3)	*	*	*
Rightmost fixation	+9.7 (4.4)	+16.6 (2.6)	+12.3 (2.3)	*	*	*
Fixation range (°)	25.6 (6.1)	26.2 (6.5)	25.5 (4.3)	n.s.	n.s.	n.s.
Right-hemispace fixations						
Number	24.4 (21.4)	38.2 (19.6)	16.9 (9.8)	n.s.	*	*
Duration (ms)	389.0 (121.2)	316.2 (92.0)	298.8 (109.5)	*	n.s.	n.s.
Left-hemispace fixations						
Number	40.7 (19.6)	28.5 (31.1)	21.5 (15.8)	*	n.s.	*
Duration (ms)	366.1 (175.6)	468.7 (239.8)	353.7 (157.8)	n.s.	*	n.s.
Rightward saccades						
Number	31.2 (12.5)	30.8 (19.7)	20.6 (13.2)	*	*	n.s.
Amplitude (°)	5.4 (1.2)	4.9 (1.8)	6.7 (2.7)	*	*	n.s.
Leftward saccades						
Number	33.8 (25.4)	35.9 (26.1)	17.9 (10.5)	*	*	n.s.
Amplitude (°)	3.9 (1.2)	4.6 (1.4)	6.0 (2.0)	*	*	n.s.
Scanpath length (°)	295.2 (168.1)	290.6 (181.2)	226.0 (121.4)	n.s.	n.s.	n.s.

Statistical comparisons were made between LHH, RHH, and N (two-tailed dependent samples t-tests).
* indicates $p<0.017$ (α_{corr}), n.s. indicates non-significant comparisons.

Table 6 Eye-movements during manual line bisection in left- and right-sided simulated hemianopia (LHH, RHH) and in the normal viewing condition (N) [means (SD) calculated over all line lengths].

	LHH	RHH	N	N-LHH	N-RHH	LHH-RHH
Horizontal position (°) of the						
Bisection fixation	–2.1 (4.5)	+1.7 (4.7)	–0.2 (0.6)	*	*	*
Maximum fixation	–6.5 (3.9)	+6.8 (4.5)	–0.5 (4.5)	*	*	*
Leftmost fixation	–16.5 (2.8)	–10.3 (5.1)	–11.4 (4.3)	*	n.s.	*
Rightmost fixation	+11.0 (4.0)	+17.4 (2.7)	+11.6 (3.8)	n.s.	*	*
Fixation range (°)	27.5 (6.1)	27.7 (6.7)	23.0 (7.6)	*	*	n.s.
Right-hemispace fixations						
Number	17.9 (10.9)	46.4 (22.6)	15.8 (9.7)	n.s.	*	*
Duration (ms)	324.8 (82.9)	341.5 (94.4)	280.0 (92.3)	*	*	n.s.
Left-hemispace fixations						
Number	50.5 (25.8)	17.5 (12.5)	15.1 (8.9)	*	n.s.	*
Duration (ms)	355.3 (84.0)	376.0 (123.0)	345.1 (129.7)	n.s.	n.s.	n.s.
Rightward saccades						
Number	34.1 (15.7)	29.7 (17.1)	16.3 (9.0)	*	*	n.s.
Amplitude (°)	4.8 (1.5)	5.8 (1.6)	5.9 (2.5)	n.s.	n.s.	n.s.
Leftward saccades						
Number	34.3 (17.2)	34.2 (15.7)	14.6 (8.1)	*	*	n.s.
Amplitude (°)	4.8 (1.5)	5.3 (1.4)	6.0 (2.7)	n.s.	n.s.	n.s.
Scanpath length (°)	358.1 (202.2)	344.6 (188.9)	182.8 (114.6)	*	*	n.s.

Statistical comparisons were made between LHH, RHH, and N (two-tailed dependent samples t-tests). * indicates $p<0.017$ (α_{corr}), n.s. indicates non-significant comparisons.

Hemispace analyses revealed the differential effect of simulated HH on the horizontal fixation distribution for ocular (Table 5) and manual line bisection (Table 6). Fixations were more frequent in contralateral than in ipsilateral hemispace (ocular: smaller $t_{(19)}=4.39$, $p<0.001$; manual: smaller $t_{(19)}= -10.76$, $p<0.001$). Under normal viewing conditions fixations were symmetrically distributed during manual line bisection ($t_{(19)}= -0.55$, $p=0.586$). During ocular line bisection, however, participants showed a tendency to fixate more frequently in left- than right-hemispace ($t_{(19)}=1.80$, $p=0.088$) (repeated measures t-tests). There was also a differential effect for fixation durations during ocular line bisection that was not evident

during manual line bisection (Table 5). Simulated HH of either sort induced significantly longer fixation durations in ipsilateral than in contralateral hemispace (RHH: $t_{(19)}$=3.79, p=0.001; LHH: $t_{(19)}$= –0.53, p=0.603 (the non-significant result may possibly be due to a large variation in individual fixation durations)). Under normal viewing conditions left-hemispace fixation duration was significantly longer than right-hemispace fixation duration ($t_{(19)}$=2.46, p=0.023) (repeated measures t-tests). This result is consistent with the finding of ipsilateral ocular bisection errors during line bisection with simulated HH and leftward bisection errors under normal viewing conditions.

The interaction between simulation-condition and line length for the left- and right-most fixation positions and the range of fixations during manual line bisection was also replicated. This effect was also confirmed for the maximum fixation position (smallest $F_{(4,72)}$=3.28, p=0.016; marginal significance for fixational range: $F_{(4,72)}$=2.12, p=0.087). During ocular line bisection, however, this length effect was present in all simulation-conditions (smallest $F_{(1.8,32.5)}$=4.19, p=0.028).

Although performing the ocular bisection task had a considerable effect on manual line bisection performance (but not *vice versa*), eye-movement measures were not significantly affected by task-sequence (non-significant main and interaction effects; largest $F_{(1,18)}$=1.48, p=0.240). Eye-movement patterns during ocular line bisection also did not differ between participants who first performed manual line bisection and those who first performed ocular bisection (largest $F_{(1,18)}$=2.14, p=0.161), except that the maximum fixation position showed a slight rightward deviation in the former group but a leftward deviation in the latter group ($F_{(1,18)}$=7.37, p=0.014).

The effects of response-mode

The differences between ocular and manual line bisection in the effects of simulated HH on the range of fixations, scanpath length and left- and right-hemispace fixations are confirmed by a significant interaction of response-mode and simulation-condition (smallest $F_{(2,36)}$=3.67, p=0.035): the increased fixation range and longer scanpaths were present only during manual

line bisection whereas the differential effect on fixation durations was associated with ocular line bisection only. In addition it was found that simulated HH induced a greater deviation of the maximum fixation position during manual line bisection than during ocular bisection (Tables 4, 6; $F_{(2,36)}$=5.69, p=0.007). Eye-movement patterns under normal viewing conditions were not, however, affected by response-mode (largest $F_{(1,18)}$=3.56, p=0.075), except that bisecting lines by fixation seemed to induce a slight leftward deviation in oculomotor patterns that was not present during manual line bisection as well as slightly larger saccades (significant effect of response-mode for left-hemispace fixations and saccadic amplitudes, smallest $F_{(1,18)}$=4.79, p=0.042).

The relationship between the point of bisection and the fixation at the time of bisection during manual line bisection

The contralateral deviation of the fixation at the time of bisection during manual line bisection with simulated HH and the slight leftward deviation under normal viewing conditions were replicated (Table 6; $F_{(2,36)}$=16.12, p<0.001). The observation that ipsilateral errors were accompanied by a smaller fixational deviation (LHH: –1.6° (SD: 3.6), RHH: 1.4° (SD: 2.8)) than contralateral errors (LHH: –2.6° (SD: 3.2), RHH: 1.9° (SD: 3.9)) is also consistent with Experiment 1. These differences did not, however, reach statistical significance (larger $t_{(16)}$=1.17, p=0.261). It was also found that the fixation at the time of bisection deviated in the same direction as the point of bisection under normal viewing conditions ($t_{(18)}$= –4.03, p=0.001) (repeated measures t-tests).

Since performing the ocular bisection task improved (albeit not significantly) in subsequent manual line bisection performance (but not *vice versa*), it was investigated whether participants might have used the bisection-by-fixation strategy they must have adopted during ocular line bisection to perform manual line bisection. Although the position of the fixation at the time of bisection was not affected by task-sequence (largest $F_{(2,72)}$=2.01, p=0.149), it was found that only participants who first performed the ocular bisection task (n=10) showed the relationship between this fixational measure and the point of bisection

during line bisection with simulated HH (smaller r=0.19, p=0.018; manual task first: larger r=0.09, p=0.264). Consistent with Experiment 1, this relationship was more pronounced for ipsilateral errors (smaller r=0.19, p=0.099) than for contralateral errors (larger r= –0.02, p=0.891). Under normal viewing conditions, however, this relationship was obtained irrespective of whether participants first performed the ocular or manual bisection task (smaller r=0.31, p<0.001). Yet, in the former group, it reached statistical significance for rightward errors only (r=0.30; p=0.014) (as in Experiment 1); in the latter group, it was significant for leftward errors only (r=0.25, p=0.023).

3.3. Discussion

Although the ipsilateral manual bisection error found in Experiment 1 was not fully replicated, it was shown again that simulated HH induces the contralaterally deviated eye-movement pattern of hemianopic patients during line bisection (Barton et al., 1998; Ishiai et al., 1987, 1989) but not their contralateral line bisection error (Barton et al., 1998; Barton & Black, 1998; Doricchi et al., 2005; Hausmann et al., 2003; Kerkhoff, 1993; Zihl, 2000; Zihl & von Cramon, 1986). Interindividual differences in the impact of a simulated visual field defect (Zangemeister & Utz, 2002; see also Chapter 3), the use of longer lines, which increases the difficulty of line bisection with a visual field defect, and fewer trials may account for the differences between experiments.

Studying ocular line bisection in simulated HH demonstrated that the ipsilateral bisection error and the contralateral deviation in the pattern of eye-movements found in Experiment 1 also occur without manual response. The significant correlation between ocular and manual bisection errors and the finding that ocular and manual line bisection require the same amount of time is consistent with this finding. Moreover, irrespective of whether the ocular, manual or the classic paper-and-pencil bisection task was used to assess line bisection performance under normal viewing conditions, participants showed the same bisection times, the small leftward bisection error and the symmetrical oculomotor scanning pattern that is typical of healthy subjects (Barton et al., 1998; Ishiai et al., 1987, 1989; Jewell

& McCourt, 2000). Although ocular bisection errors were slightly larger and a slight leftward directional bias in the otherwise symmetrical eye-movement patterns was obtained under normal viewing conditions, this result nevertheless suggests that the manual motor component of the line bisection task, i.e., the actual hand movement, seems not to be critical to the bisection error and oculomotor behaviour of healthy participants when confronted with a pure visual field defect or under normal viewing conditions. This conclusion is supported by findings from ocular line bisection in visual neglect indicating that the placement of the bisection mark is predicted by the fixation at the time of bisection (Ishiai et al., 1989; Ishiai et al., 1998). Based on these findings the "line bisection task by fixation" has been proposed as a substitute for the manual line bisection test (Ishiai et al., 1998) which may be particularly useful in cases where upper extremity disorders impede the assessment of line bisection performance. Examining ocular line bisection in simulated HH has shown that this task might also be a useful experimental and diagnostic tool for assessing line bisection in patients with visual field loss.

The importance of oculomotor factors in line bisection with simulated HH is further emphasised by the effects of ocular line bisection on subsequent manual bisection. Performing the ocular line bisection task led to smaller bisection errors and seemed to increase the frequency and magnitude of ipsilateral relative to contralateral errors. Performing the manual bisection task, in contrast, had no effect on subsequent ocular bisection. These findings suggest that participants may adopt the bisection-by-fixation strategy they used during ocular line bisection for performing the manual bisection task with simulated HH. Participants may use an ocular fixation to guide their manual bisection response, which seems to improve manual line bisection performance. The significant correlation that was obtained between the fixation at the time of bisection and the point of bisection during manual line bisection with simulated HH only after participants had performed the ocular bisection task supports this assumption. It remains possible, however, that these improvements did not result from adopting a specific bisection strategy but from

increased oculomotor adaptation to simulated HH or from simple practice effects. Yet, line bisection performance and eye-movements as well as the close relationship between the fixation at the time of bisection and the bisection position under normal viewing conditions remained unchanged after performing the oculomotor task. Moreover, ocular line bisection did not improve after performing the manual bisection task, neither when participants were confronted with simulated HH nor under normal viewing conditions. These findings contradict the latter two explanations and that line bisection performance has been found to be robust to retest effects further supports this chapter's assumption (Kerkhoff & Marquardt, 1998; Pierce, Jewell, & Mennemeier, 2003).

4. General Discussion

The purpose of the experiments reported in this chapter was to identify the visual and oculomotor (and thus attentional) components that may constitute the hemianopic bisection error as well as to establish whether the origin of the contralateral bisection error in hemianopic patients is purely visual.

The results demonstrate that a pure hemianopic visual field defect does not induce the reliable contralateral deviation during line bisection that has been reported for hemianopic patients (Barton et al., 1998; Barton & Black, 1998; Doricchi et al., 2005; Hausmann et al., 2003; Kerkhoff, 1993; Zihl, 2000; Zihl & von Cramon, 1986). Although it induces significantly larger bisection errors than under normal viewing conditions, these errors are smaller than those of hemianopic patients and participants showed both, contra- and ipsilateral errors; ipsilateral errors were even larger and more frequent than contralateral errors, resulting in an overall ipsilateral error. Although the presence of a pure hemianopic visual field defect impairs line bisection performance in healthy participants, it seems not sufficient for the reliable contralateral bisection error to emerge. This finding contradicts the hypothesis that the hemianopic bisection error is a direct consequence of the visual field defect (Barton et al., 1998; Barton & Black, 1998; Best, 1910a; Nielsen et al., 1999).

Yet the presence of strategic oculomotor adaptation to visual field loss indicating an attentional bias to contralateral hemispace also does not seem to be the causative factor in the hemianopic bisection error. It was demonstrated that line bisection with simulated HH was associated with a contralateral deviation in the pattern of eye-movements. This deviation indicates strategic oculomotor (and thus attentional) adaptation to visual field loss and mirrors the oculomotor behaviour of hemianopic patients during line bisection (Barton et al., 1998; Ishiai et al., 1987, 1989). Despite strategic oculomotor adaptation to contralateral hemispace, participants did not show the reliable bisection error in the same direction. Thus, compensatory shifts of eye-movements towards the blind field and the contralateral bisection error can dissociate. This finding challenges the view that the hemianopic bisection error arises from oculomotor adaptation indicating an adaptive attentional bias to contralateral hemispace (Barton et al., 1998; Barton & Black, 1998).

Although neither the visual field defect nor oculomotor adaptation to it seems to be the causative factor in the hemianopic bisection error, they may nevertheless contribute to it. The line bisection task has long been used as an experimental tool to study the perceptual, attentional and motor factors affecting visuospatial performance both in patients with visual neglect and normal subjects (Fischer, 2001) but surprisingly not in patients with visual field loss. Thus, it remains unknown exactly which factors determine line bisection performance in visual field loss. Investigating the role of the visual field defect in relation to perceptual, attentional and (ocular and manual) motor factors seems to be of particular interest in this regard, not least since patients with visual neglect frequently show a concomitant visual field disorder (Walker, Findlay, Young, & Welch, 1991).

The fact that the magnitude and direction of the bisection errors that were observed in simulated HH are not the same as in real HH suggests a differential contribution of visual and adaptive oculomotor (and thus attentional) factors to the respective bisection errors. Since error magnitude does not differ between left- and right-sided visual field loss, neither in real HH (Kerkhoff, 1993; Zihl, 1995, 2000; Zihl & von Cramon, 1986) nor in simulated

HH, it may be the severity of the visual field defect that determines the magnitude of the bisection error. If the visual field defect contributes to the error, the degree of visual field sparing should be negatively correlated with error magnitude (Barton & Black, 1998). Although preliminary evidence suggests that there is no such relationship in hemianopic patients (Kerkhoff, 1999; Zihl, 2000), no systematic study has been carried out thus far, and since these experiments studied line bisection in simulated HH with a constant visual field sparing, this relationship still requires further investigation in both real and simulated HH. The side of the visual field defect seems to determine the direction of the error in hemianopic patients; patients with a left-sided HH show leftward errors, patients with a right-sided HH show rightward errors (Barton et al., 1998; Barton & Black, 1998; Doricchi et al., 2005; Hausmann et al., 2003; Kerkhoff, 1993; Zihl, 2000; Zihl & von Cramon, 1986). In simulated HH, however, the relationship between side of visual field loss and error direction was less pronounced. The effect of the side of visual field loss on the hemianopic contralateral bisection error may not be purely visual. It may rather be the side of brain injury that determines error direction but masquerades as a visual effect.

It is also important to consider the possibility that hemianopic visual field defects result in a chronic differential lateralised or asymmetric visual-sensory input and, thus, an imbalance in visual-spatial processing efficiency, which can give rise to an attentional bias in the direction of the seeing hemifield, i.e., to ipsilateral hemispace (Tant, Kuks, Kooijman, Cornelissen, & Brouwer, 2002). Such ipsilateral attentional bias arising from a visual-sensory deficit might explain the ipsilateral bisection errors participants showed when confronted with a simulated HH. Another factor contributing to the ipsilateral errors may be a geometric bias that is introduced by the fact that the visual angles subtended by each of the two halves of a line are unequal when the line is viewed in one hemifield on a flat surface perpendicular to the direction of gaze at fixation. Although the error arising from this geometric bias is in the wrong direction to account for the ipsilateral bias in simulated HH, its magnitude is comparable to that of the ipsilateral error in our participants. Since its

magnitude also increases with increasing line length, this error could account for the absence of a consistent ipsilateral bias when longer lines were used (Experiment 2). The difference in distance from the eye to the two halves of the line with a flat display is another potential influence on line length perception (Norman, Todd, Perotti, & Tittle, 1996). However, again the difference in distance between the near and far end lines in this study's tasks is negligible compared to the depth differences one would expect in order to account for the ipsilateral bisection errors found in the present study (Norman et al., 1996). It nevertheless remains possible that retinal eccentricity effects on perceived line length may contribute to these errors. Bisecting lines viewed in only one hemifield by instructing participants to fixate the left or right line end induces a contralateral bisection error which has been explained as being mediated by the relationship between retinal eccentricity and cortical magnification. The representation of space may be distorted in the periphery and the portion of the stimulus in central vision may be overestimated (central magnification) (Nielsen et al., 1999). The similarities in magnitude between the errors found in hemifield line bisection and the errors associated with simulated HH seem to support this argument. Yet, since both errors were in opposing directions, it remains to be seen exactly which factors determine a systematic change in the bias (in addition to the systematic change in the accuracy) of position judgments as eccentricity increases.

Although the bisection error in simulated and real HH does not seem to be a manifestation of strategic oculomotor adaptation indicating an adaptive attentional bias to contralateral hemispace, oculomotor factors may nevertheless contribute to the resulting bisection error. The fixation at the time of bisection was identified as an important oculomotor factor that seems to be critical to the ipsilateral bisection error found in simulated HH. The significance of oculomotor factors in manual line bisection is further supported by the findings from ocular line bisection in simulated HH and under normal viewing conditions. Participants showed the same line bisection error and oculomotor behaviour as in the manual line bisection task indicating that the manual motor component

seems not to be integral to the ipsilateral bisection error associated with simulated HH and the small leftward error under normal viewing conditions. Significant correlations between ocular and manual bisection errors are consistent with this view. Further investigation is required in order to determine the extent to which the fixation at the time of bisection and the manual motor component contribute to the contralateral bisection error found in hemianopic patients.

The finding that performing the ocular bisection task with simulated HH, i.e., bisecting lines by fixating instead of marking the subjective line centre, improved performance in the subsequent manual bisection task but not *vice versa*, provides additional evidence for the importance of oculomotor factors in manual line bisection. However, since no improvements were observed under normal viewing conditions, oculomotor factors may be of particular importance if vision is compromised. Performing ocular line bisection with simulated HH may allow participants to adopt an oculomotor strategy that helps guiding their manual bisection response in a condition where lines can never be seen in their entirety. The consequent improvements in line bisection suggest that this strategy alleviates the line bisection impairment caused by this pure visual field defect. It remains to be determined whether such oculomotor strategies suffice to alleviate the contralateral line bisection error in hemianopic patients.

In conclusion, these findings suggest that the hemianopic visual field defect and its adaptive oculomotor (and thus attentional) consequences may contribute to the contralateral bisection error found in hemianopic patients but they do not seem to be its primary causes. The bottom-up restriction of the visual field clearly affects line bisection performance, suggesting that the ability to accurately bisect lines requires visual information extraction from the parafoveal and peripheral visual field. If vision in these visual field regions is affected, either by simulated HH or by brain injury, lines are only partly visible, which impairs efficient line bisection. However, a pure hemianopic visual field defect and its adaptive oculomotor (and thus attentional) consequences did not suffice to induce the

contralateral bisection error. Thus, the basis of the hemianopic bisection error does not seem to be purely visual. These results are consistent with reports that the contralateral bisection error can dissociate from visual field loss (Best, 1919; Zihl, 1988, 2000) as well as from successful strategic oculomotor adaptation indicating an adaptive attentional bias to contralateral hemispace in patients (Gassel & Williams, 1963a, 1963b; Williams & Gassel, 1962). Although the contralateral bisection error is frequently associated with HH, it is separable from both, the visual field defect and its adaptive oculomotor (and thus attentional) consequences.

The hemianopic line bisection impairment is not simply a failure of vision but an indicator of a visual-spatial deficit which is frequently associated with HH but not primarily caused by it. It seems to require additional extrastriate brain injury, possibly to regions that are involved in visual-spatial perception. Axenfeld (1894) advocated the line bisection task as "a simple method to diagnose hemianopia", particularly in cases where there is no access to a perimeter or when patients are not able to undergo perimetric visual field testing (see also Liepmann & Kalmus, 1900). The dissociability of the contralateral line bisection error and HH indicates, however, that the diagnostic value of the line bisection task in the assessment of HH is limited. Yet, although the line bisection task is not an appropriate substitute for perimetric testing and can only complement perimetric diagnosis, it is an important tool to assess visual-spatial perception which is frequently impaired in hemianopic patients. Since visual-spatial deficits interact with visual deficits and increase resulting functional impairments, studying visual-spatial deficits in patients with visual field loss, as well as developing effective treatment methods, is of great importance. Although strategic oculomotor adaptation and the contralateral bisection error can dissociate, treatment-induced oculomotor adaptation in reading and visual exploration (Zihl, 2000) may help patients to overcome their shift of the egocentric visual midline. Yet, evidence from patients with visual neglect suggests that visual-spatial deficits require specific treatment for their improvement (Kerkhoff, 1998). It is therefore also important to study the natural course of the visual-

spatial deficit associated with visual field loss since spontaneous recovery of perception of spatial axes has been reported in patients with right posterior cerebral infarctions (Zihl, 2000).

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Chapter 5

REHABILITATION OF HEMIANOPIC DYSLEXIA: ARE WORDS NECESSARY FOR RE-LEARNING OCULOMOTOR CONTROL?

The study presented in this chapter investigated whether the therapeutic effect of a specific compensatory treatment method for rehabilitating hemianopic dyslexia critically depends on using text material. The effectiveness of systematic oculomotor training using non-text material (Arabic digits) was therefore evaluated in comparison with the conventional oculomotor training method that uses text material (words) in 40 patients with unilateral homonymous visual field disorders showing hemianopic dyslexia. Non-text training was found to be as effective as conventional text training in improving reading performance and associated eye-movements. This result suggests that using words is not critical to the treatment effect of this training procedure. Thus, lexical-semantic processes seem not to be necessary for re-learning eye-movement control in hemianopic dyslexia.

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1. Introduction

Unilateral homonymous visual field disorders are common functional impairments after acquired injury to the postchiasmatic visual pathway. Sufficient spontaneous recovery of the visual field occurs rarely (Zhang, Kedar, Lynn, Newman, & Biousse, 2006; Zihl & Kennard, 1996), and most patients show severe impairments of reading (80%) and visual exploration (60%) (Zihl, 2000, 2003). Mauthner (1881) was the first to describe the acquired reading disorder in which patients with unilateral homonymous visual field disorders have severe reading difficulties despite intact language functions. Wilbrand (1907) termed it "macular-*hemianopic* reading disorder" (see also Poppelreuter, 1917/1990) since hemianopia is the most frequent visual field disorder, followed by quadranopia and paracentral scotoma (Zihl, 2000).

In hemianopic dyslexia, the visual bottom-up and attentional top-down control of text processing and eye-movements involved in reading is disturbed (see Chapter 1). Consequent impairments of word identification and the ability to plan and guide reading eye-movements become manifest as pronounced slowness of reading, visual omission and guessing errors as well as a severely disorganised oculomotor scan-pattern in reading–the cardinal symptoms of hemianopic dyslexia (De Luca, Spinelli, & Zoccolotti, 1996; Eber, Metz-Lutz, Bataillard, & Collard, 1987; Gassel & Williams, 1963; Leff et al., 2000; Mackensen, 1962; McDonald, Spitzyna, Shillcock, Wise, & Leff, 2006; Schoepf & Zangemeister, 1993; Spitzyna et al., 2007; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a, 2000). Hemianopic dyslexia represents a substantial impediment to patients' vocational, educational and daily life activities and counts as an important cerebral visual impairment (Papageorgiou et al., 2007; Zihl, 2000).

Although spontaneous oculomotor adaptation to visual field loss in visual exploration is more likely (40%) than patients compensating for their reading impairment (20%) (Zihl, 2000), the majority of neuropsychological rehabilitation studies on visual field disorders has focussed on the visual exploration impairment (for a systematic review, see Bouwmeester,

Heutink, & Lucas, 2007). To date, only five studies have dealt with the rehabilitation of hemianopic dyslexia (Kerkhoff, Münßinger, Eberle-Strauss, & Stögerer, 1992; Spitzyna et al., 2007; Zihl, 1995a, 2000; Zihl, Krischer, & Meißen, 1984). The first systematic attempt to improve reading in patients with visual field loss dates back to Poppelreuter (1917/1990). He developed a special reading training for addressing the "disturbance of the co-ordination of the reading gaze-shifts" (p. 224) he observed in his patients. Poppelreuter showed convincingly that by systematic practice of oculomotor control "relearning of reading was successful" (p. 249).

Poppelreuter's treatment rationale led to the development of a compensatory oculomotor treatment method for hemianopic dyslexia (Zihl et al., 1984), which proved its effectiveness in a number of investigations (Kerkhoff et al., 1992; Spitzyna et al., 2007; Zihl, 1995a, 2000). It involves supervised, systematic practice of reading eye-movements with text material (words) to overcome the effects of parafoveal visual field loss in reading. Patients learn to efficiently use saccadic eye-movements to bring the entire word from the blind into the seeing hemifield for identification. As a consequence, patients regain sufficient reading performance with long-term stability, confirming the importance of effective oculomotor control in reading. Treatment effects are characterised by an increase in reading speed and accuracy, and the re-establishment of a systematic oculomotor scan-pattern in reading. These effects were attributed to training-related oculomotor adaptation to parafoveal visual field loss in reading (Zihl, 1995a, 2000).

Thus far only text material, either moving (Kerkhoff et al., 1992; Spitzyna et al., 2007; Zihl, 1995a, 2000; Zihl et al., 1984) or static (Zihl, 2000), has been used in this treatment procedure. However, it is still unclear whether the treatment effect associated with this treatment procedure for hemianopic dyslexia critically depends on using text material (words). The study reported in this chapter therefore investigated whether words and thus lexical-semantic processes are necessary for re-learning reading eye-movement control in parafoveal visual field loss, or whether non-text material lacking lexical-level linguistic

information (Arabic digits) is sufficient. The effectiveness of oculomotor training using time-limited presentation of static non-text material was evaluated in comparison with conventional oculomotor training using text material (Zihl, 2000). In addition to assessing the treatment effects on reading performance and associated eye-movements in patients with hemianopic dyslexia, it was investigated whether these effects are specific to reading, or whether there is a transfer of training-related improvement to visual exploration performance.

Investigating the therapeutic potential of non-text training is also an attempt to improve current rehabilitative efforts. Clinical observations suggest that patients with hemianopic dyslexia seem to over-rely on linguistic processes when attempting to identify words. Their common yet maladaptive strategy is to elaborate the meaning of an incompletely perceived word by guessing rather than first inspecting the entire word. Lexical-semantic processing comes into play too early, which disrupts further acquisition of text information located in the blind hemifield and interferes with the treatment goal, i.e., that patients learn to visually apprehend before comprehending text (Zihl, 2000). Avoiding text material in the treatment of hemianopic dyslexia may eliminate not only such undesired linguistic top-down interference but also the additional cognitive load associated with word processing itself (Lien, Ruthruff, Cornett, Goodin, & Allen, 2008; McCann, Remington, & Van Selst, 2000; Shaywitz et al., 2001). Reading-related oculomotor training with non-text material may therefore be less effortful for the patient.

2. Methods

2.1. Participants

40 patients with left- (n=16) or right-sided (n=24) homonymous parafoveal visual field loss and hemianopic dyslexia participated in this study. Homonymous hemianopia was the most frequent cause of parafoveal visual field loss; 12 patients had a left-sided, 12 a right-sided hemianopia. Six patients had a right-sided upper and two a right-sided lower quadranopia.

Six patients had a right-sided, two a left-sided paracentral scotoma. The parafoveal visual field was compromised in all patients. Mean visual field sparing, i.e., the extent of the visual field in degrees between the fovea and the visual field border along the left or right horizontal axes, was 2.1° (range: 1–4°). In all patients, aetiology of brain injury, as verified by cranial CT and/or MRI, was an infarction (82.5%) or haemorrhage (17.5%) in the territory of the posterior cerebral artery causing a lesion to the occipital cortex. Time between the occurrence of brain injury and initial assessment was on average 30 weeks (range: 5–220). None of the patients had received any treatment for their visual field defect. Patients showed no evidence of associated cerebral visual disorders, including reduced visual acuity (<0.90 for near and far binocular vision), impaired spatial contrast sensitivity (Vistech contrast sensitivity test, 1988), visual adaptation, disturbances of the anterior visual pathways or of the oculomotor system, macular disease (according to ophthalmologic examination), nor aphasia, premorbid reading disorders, pure alexia (vertical word reading test, see Zihl, 1995a), impairments of visual-lexical numerical processing (horizontal and vertical number reading, see Zihl, 1995a), or verbal memory deficits (WMS-R (Logical Memory I/II), see Wechsler, 1987). None of the patients had visual neglect as assessed by tests in accordance with the Behavioural Inattention Test, composed of line bisection, letter and star cancellation, figure and shape copying, and drawing from memory (see Halligan, Cockburn, & Wilson, 1991). All patients were native German speakers and had at least five years of education.

All patients complained of moderate to severe difficulties in reading and showed impaired reading performance. Patients were therefore systematically treated to compensate better for their parafoveal visual field loss in reading. Half of patients received treatment with text material (text training, Group A, n=20), the other half was treated with non-text material (non-text training, Group B, n=20). For treatment allocation, age, type, side and severity (i.e., visual field sparing) of visual field loss were used as stratifying variables before testing was carried out. Before treatment, there were no differences between both

groups either for demographic and clinical variables or for reading and visual exploration performance (see Table 1).

Table 1 Demographic and clinical details and behavioural measurements for both treatment groups [mean (SD, range)]

	Text training (Group A: n=20)	Non-text training (Group B: n=20)	
Age (years)	58.8 (11.8, 28–80)	58.7 (13.8, 23–83)	p=.980
Sex			
Female	3 (15%)	3 (15%)	
Male	17 (85%)	17 (85%)	
Education (years)	9.7 (3.2, 5–18)	10.0 (4.1, 5–19)	+p=.989
Time since lesion (weeks)	28.9 (28.4, 5–97)	31.0 (47.0, 5–220)	+p=.839
Aetiology			
Posterior infarction	18 (90%)	15 (75%)	
Occipital haemorrhage	2 (10%)	5 (25%)	
Type of visual field loss			
Hemianopia	12 (60%)	12 (60%)	
Upper quadranopia	3 (15%)	3 (15%)	
Lower quadranopia	1 (5%)	1 (5%)	
Paracentral scotoma	4 (20%)	4 (20%)	
Side of visual field loss			
Left	8 (40%)	8 (40%)	
Right	12 (60%)	12 (60%)	
Visual field sparing (°) (pre-treatment)	1.9 (0.9, 1–4)	2.2 (1.0, 1–4)	p=.562
Reading speed (wpm) (pre-treatment)	92.8 (40.7, 33–162)	100.9 (27.2, 50–148)	p=.464
Visual exploration time (s) (pre-treatment)	31.8 (14.6, 14–65)	34.5 (13.9, 15–72)	p=.315
Interval T1 – T2 (weeks)	6.0 (3.5, 2–15)	4.6 (3.0, 1–13)	+p=.155
Interval T2 – T3 (number of training sessions within 2 weeks)	10.5 (2.0, 7–14)	9.6 (2.0, 6–12)	p=.141
Interval T3 – T4 (weeks)	10.9 (2.6, 6–15)	11.4 (2.7, 7–16)	p=.518

Statistical comparisons were made between treatment groups. P-values for two-tailed independent t-tests or +Mann-Whitney-U-tests (where normality assumptions were violated as assessed by Shapiro-Wilk tests) are given.

Mean near Snellen visual acuity was 0.97 (SD: 0.05, range: 0.9–1.0) in Group A, and 0.98 (SD: 0.04, range: 0.9–1.0) in Group B. A single subject baseline design with a

treatment-free interval before and after oculomotor training with text or non-text material was used; thus, every patient served as his or her own control. Visual fields and reading performance were assessed at four time-points, i.e., at initial assessment (T1), before (T2) and after (T3) treatment, and after a follow-up interval (T4). Time intervals between assessments did not differ between groups (see Table 1). In addition, visual exploration performance was assessed and subjective reports were obtained (T2, T3). In a representative sub-sample of 7 patients for each treatment group, eye-movements during reading (T2, T3, T4) and visual exploration (T2, T3) were recorded. All patients gave informed consent to participate in this study.

2.2. Visual field testing

Monocular and binocular visual fields were measured using kinetic perimetry with a standard Tübingen perimeter (Aulhorn & Harms, 1972; Zihl, 1989). Target diameter was 1.2°, its luminance was 102 cd/m^2; background luminance was 3.2 cd/m^2. The target was moved with a speed of ~2°/s from the periphery towards the perimeter's centre. Patients were instructed to fixate a small red spot of light (diameter: 0.5°) in the centre of the sphere and to press a response button as soon as they detected the target. Fixation accuracy was monitored through a telescope. The visual field border was determined along 16 meridians. Perimetric resolution was 0.5° and measurement error was 0.5° within the central 15° of the visual field, which is relevant for reading.

2.3. Assessment of reading and visual exploration performance; subjective reports

Reading performance was assessed by using four parallel versions of a standardized reading test shown to be sensitive to changes in reading performance during treatment (Zihl, 1995a, 2000). Each text consisted of 200 words (in 14pt Arial font) arranged in 20 double spaced, left-aligned lines printed on a white sheet of paper. The texts were characterised by short sentences and simple syntactic structure and were standardised for content (taken from Gotthold E. Lessing's animal fables (in German)). The frequency of each word-length (in

number of characters) was the same for each text. Patients were instructed to read the text aloud as accurately and quickly as possible. Reading time and errors were recorded. Reading performance was defined as number of words correctly read per minute (wpm); this measure incorporates both (oral) reading speed and accuracy. The number of reading errors is therefore not reported in the results section. Normative data were available from a sample of 80 control participants (40 females, 40 males; mean age: 41.3 years (SD: 13.4)). Average corrected reading speed was 161.1 wpm (SD: 21.3, range: 121–218).

For assessing visual exploration performance stimulus patterns consisting of simple forms have proven to be a valuable test, which is sensitive to changes in visual exploration performance during treatment (Zihl, 2000). A cancellation task with 20 black diamonds (targets) randomly embedded in 22 black dots and crosses (distractors) presented on a sheet of white paper was used. At a viewing distance of 30 cm the stimulus array subtended 44.6° horizontally and 35° vertically; stimulus diameter was 0.8°. Patients were asked to mark all diamonds with a pencil as quickly as possible with their right hand. No instruction was given on how to proceed and patients were not informed about the number of targets. Visual exploration performance was defined as the time required to perform the task. Since all patients performed the task errorless, errors are not reported in the results section. Normative data were available from 25 control participants (12 females, 13 males; mean age: 38.0 years (SD: 10.7)), who required on average 13.2s (SD: 1.3, range: 9.1–17.2) to perform this task errorless.

The reading and visual exploration tests were administered under normal daylight conditions. The experimenter sat to the right of the patient and centred the test sheets to the patient's body axis at a distance of 30 cm. Eye and head movements were not restricted. In addition informal subjective reports on reading and visual exploration performance were obtained by using the corresponding questions of a validated questionnaire (Kerkhoff, Schaub, & Zihl, 1990). In addition, patients were observed during training sessions and their subjective impressions of the training method were collected (subjective rehabilitation

experience); after treatment, they were also asked whether they were satisfied with the treatment outcome.

2.4. Recording of eye-movements in text reading and visual exploration

In a sub-sample of 14 patients, oculomotor measures for silent reading (T2, T3, and T4 (except for 1 patient)) and visual exploration (T2, T3) were obtained. Eye-movements were recorded using a video-based, infrared remote eye tracking system (iView X RED, SensoMotoric Instruments GmbH, Teltow, Germany). Viewing was binocular and the position of the dominant eye was sampled at 50 Hz, with a spatial resolution of 0.1°. Prior to the recording session of each patient, the equipment was calibrated using a nine-point grid. During the registration of eye-movements, patients sat in front of a screen which subtended 40° horizontally and 32° vertically, with the head fixed at a distance of 140 cm. Room illumination was very low (1 lux) to avoid cues from the surroundings.

Materials for recording eye-movements during silent text reading consisted of three parallel versions of a standardized reading test shown to be sensitive to changes in oculomotor reading measures during treatment (Zihl, 1995a, 2000). Each text consisted of 61 words arranged in nine, left-aligned lines. Letter size was 1.0°, allowing for the maximum reading rate (Legge, Pelli, Rubin, & Schleske, 1985); letter width subtended 0.5°; spacing between letters was 0.2° and 1° between words. Single lines were separated vertically by 2°. Luminance of the black letters was 0.2cd/m^2, that of the white background was 27cd/m^2. The texts were characterised by short sentences and simple syntactic structure and were also standardised for content. Patients were asked to read the text silently and only once, with no further instructions on how to proceed. For testing comprehension and to provide evidence that patients actually read the texts, they were also asked to reiterate its content after reading the text, which all patients did correctly. Eye-movement recording was started at the onset of text presentation and was ended after the patient indicated completion of reading. At each time-point (T2, T3, T4), one text was presented. Normative data were available from a sample of 25 control participants (12 females, 13 males; mean age: 38.0 years (SD: 10.7)).

For eye-movement recording during visual exploration, irregular stimulus patterns consisting of 20 white dots (diameter: 0.9°) on a black screen were used, which have been found to be sensitive to changes in oculomotor visual exploration measures during treatment (Zihl, 1995b, 1999, 2000). Dot luminance was 27cd/m^2; background luminance was 0.2cd/m^2. The minimal spatial separation of any pair of adjacent dots was 7° (maximum distance: 10.5°). Patients were asked to silently count the dots presented on a screen; no instruction was given on the number of dots or how to proceed with counting or searching. This test is similar to the dot cancellation test (Lezak, Howieson, & Loring, 2004) but did not include feedback on which dots have already been processed. Eye-movement recording in the visual exploration condition was started with the onset of dot pattern presentation and was ended when the patient indicated to have counted all dots. At the end of recording, each patient was asked to report the number of dots. Since all patients reported the 20 dots correctly, errors are not reported in the results section. One trial was carried out at each time-point (T2, T3). Normative data were available from 30 control participants (15 females, 15 males; mean age: 51.6 years (SD: 10.1)).

For each participant, individual calibration measurements were used as a basis for further data analysis. Successive points of measurement were combined into fixations if they fell into a window of 1.5° of visual angle. The minimum fixation duration was set at 100ms. Recordings with >15% loss of eye-movement data (due to lid closures or saccadic eye shifts to positions outside the registration area) were not included in the analysis. The following global temporal and spatial oculomotor measures were analysed for the assessment of reading eye-movements: Mean number and duration (ms) of fixations, percentage of fixation repetitions (fixations at previously fixated points, i.e., regressions), number of forward saccades, mean amplitude of all saccades (°) and scanpath length (i.e., the sum of saccadic amplitudes (°) between the appearance of the text and the verbal report by the patients that reading had been completed). For assessing oculomotor visual exploration performance, the

mean number and duration (ms) of fixations, percentage of fixation repetitions, and the mean amplitude (°) of all saccades were analysed.

2.5. Method of treatment: Reading training with text and non-text material

The treatment was performed using the software-based training program as developed by Zihl (2000, pp. 81-89). Text and non-text training material was presented in the centre of a 17-inch high-resolution monitor. Letter and digit size was 2°, and width subtended 1°; spacing between letters (text material) was 0.4°. Yellow was used for the training material and a dark blue for the background. These size and colour specifications have shown to allow for comfortable reading and oculomotor practice (Zihl, 2000). Room illumination was low (< 5 lux) in order to minimise the effects of glare from the monitor. Patients were seated in front of the screen, at a distance of 50cm. The treatment was administered and supervised by the experimenter, who sat beside the patient to give verbal feedback on reading errors during training (supervised learning). Reading errors were always immediately corrected by the experimenter after each trial. In addition, the experimenter monitored that patients did not resort to the common strategy of guessing only half-seen words instead of first using eye-movements to perceive words as a whole. Moreover, she monitored that patients did not use head- instead of eye-movements, another maladaptive strategy patients often resort to. Preventing such maladaptations is of great importance in the rehabilitation of visual field disorders since they increase functional visual impairment, interfere with the acquisition of an adaptive oculomotor strategy and delay treatment progress (Zihl, 2000).

Text training (Group A)

Single words of different lengths, ranging from 3 to 12 letters, were used as training material for Group A. Each training trial was composed of the time-limited presentation of one single word in the centre of the screen. Patients were instructed to perceive each word as a whole before reading it aloud by intentionally shifting their gaze, as quickly as possible, from the screen's centre to the beginning (in cases with left-sided visual field loss) or to the end (in cases with right-sided visual field loss) of each word. This paradigm allows reading-related

eye-movements to be trained and reinforced by the patient's normal internal visual feedback and feedback given by the experimenter. During the course of training, the length of the presented words was systematically increased from 3- to 13-letter-words. When a patient was able to read at least 90% of the words of a given length correctly, presentation time was reduced from ~1000 to eventually 300 to 400 ms. The final training stage involved the randomised presentation of words of different lengths. By adopting this procedure, patients were forced to make quicker and more efficient saccades in order to perceive and read the whole word before its disappearance. In addition, patients learned to flexibly adjust the size of saccades according to word-length. This training protocol was adjusted to individual reading performance and training progress. Training was completed when patients reached a defined criterion (at least 90% correct responses) for any level of difficulty used. An individual training session lasted ~ 45 minutes; it consisted of 10 practice units (30 trials each) and short or, if required, longer breaks between units. Patients required on average 11 training sessions, which were carried out within 2 weeks for each patient (interval T2–T3; see Table 1).

Non-text training (Group B)

Non-text training required saccadic eye-movements that are arguably similar to those made during text training but did not involve lexical-semantic linguistic processing. In the design of the non-text training material special care was taken to preserve the main visual feature of a word that is critical for inducing reading saccades, i.e., word-length (Ducrot & Pynte, 2002; Inhoff, Radach, Eiter, & Juhasz, 2003). Word-like units that are variable in length and comprise of a beginning and end were created. They can be expected to support similar saccadic activity as real words. For excluding lexical-level linguistic information and thus lexical-semantic processes 'digit-words' consisting of Arabic digits were created. Arabic digits do not contain any semantic information (Dehaene & Cohen, 1995; Dehaene, Molko, Cohen, & Wilson, 2004). Each digit word consisted of two Arabic digits, i.e., a 'beginning'-digit (1–9) and an 'end'-digit (0–9). Different stimulus lengths were created by varying the

space between the two digits; the spatial extent of a 12-'letter'-digit-word, for an example, resembles the average spatial extent of a 12-letter word. The second type of digit-words contains an additional digit which is inserted at random positions between the beginning- and end-digit. For each length (3 to 12-'letter'-widths), a different digit-word selection out of 90 possible beginning- and end-digit combinations were created; yet, adjacent digits were never identical. Each training trial was composed of the time-limited presentation of a single digit-word in the centre of the screen. Patients were instructed to intentionally shift their gaze, as quickly as possible, to the 'beginning', i.e., left, digit (in cases with left-sided visual field loss) or to the 'end', i.e., right, digit (in cases with right-sided visual field loss) of each digit-word before reading the two (or three) digits aloud sequentially (e.g., digit-word "2 8 3" is to be read as "2, 8, 3"). The training was carried out exactly according to the same training protocol and procedure as in Group A, with only the training material being exchanged. Patients required on average 10 training sessions, which were carried out within 2 weeks for each patient (interval T2–T3; see Table 1).

2.6. Data analyses

For testing the treatment effects of text and non-text training, a repeated measures analysis of variance was performed with time as within-subject factor for each group (within-group effects). The same analysis was conducted with treatment group as between-subject factor (between-group effects). Where sphericity assumptions were violated as assessed by Mauchly's W test, the Greenhouse-Geisser correction to the degrees of freedom was applied. Post-hoc paired comparisons between time-points were performed using two-tailed related samples t-tests. Comparisons between treatment groups were performed using two-tailed independent samples t-tests. As multiple tests were carried out, the significance level was adjusted using a Bonferroni correction to an alpha-level of 0.05 for multiple comparisons.

3. Results

3.1. Reading and visual exploration before treatment

Before treatment, all patients in both treatment groups complained of difficulties in reading and visual exploration. Patients reported that reading had become an extremely laborious and fatiguing activity. They described reading as being very slow and reported missing syllables and words as well as difficulties in finding the beginning of a new line (especially in left-sided visual field loss) and in moving the eyes smoothly along a line of text (especially in right-sided visual field loss). In addition, patients complained about colliding with obstacles, missing objects or persons located in the blind field, and losing orientation especially in unfamiliar surroundings. These reports were in close agreement with patients' objective test results as well as corresponding eye-movement recordings and were similar in both groups. All patients showed impaired reading and visual exploration performance and severely altered eye-movement patterns.

Reading performance

Before treatment, corrected reading speed was considerably reduced in all patients of both treatment groups (see Table 2, T2); there were no differences between groups for reading speed (see Table 1). The reading errors of patients consisted mainly of visual omissions of pre- or suffixes and small words or guessing errors, i.e., meaningful completion of only partially seen words.

Table 2 Reading performance and related oculomotor measures before (T2) and after treatment (T3) and at follow-up (T4) [mean (SD), [range]]. Normative data from control samples are given for comparison (N)

	Text training (Group A: n=20):			Non-text training (Group B: n=20)			
	T2	T3	T4	T2	T3	T4	N
Reading speed (wpm)	92.8 (40.7) [33–162]	127.8 (36.8) [64–187]	134.1 (35.3) [69–189]	100.9 (27.2) [50–148]	136.6 (27.7) [79–179]	142.5 (27.8) [82–183]	161.1 (21.3)
Number of fixations	105.3 (18.1) [83–135]	66.6 (18.5) [50–97]	62.8 (18.8) [46–89]	76.7 (14.0) [59–93]	51.6 (12.9) [33–71]	46.7 (11.3) [34–63]	56.0 (11.0)
Fixation repetitions (%)	26.3 (7.6) [16.3–37.5]	18.5 (6.4) [10.7–28.6]	15.1 (5.8) [8.7–22.6]	23.9 (12.9) [10.1–40.9]	14.0 (11.5) [4.2–35.7]	9.0 (7.8) [3.2–23.5]	15.2 (9.6)
Fixation duration (ms)	360.0 (108.2) [240–510]	274.3 (50.0) [220–360]	266.7 (38.3) [230–320]	340.0 (84.7) [260–460]	232.9 (47.2) [160–300]	233.3 (31.4) [190–280]	250.0 (20.0)
Number of forward saccades	68.6 (28.4) [45–121]	45.9 (23.7) [27–88]	43.5 (20.2) [25–74]	53.6 (16.5) [25–72]	40.7 (11.9) [24–60]	37.7 (11.4) [22–52]	41.0 (8.0)
Saccadic amplitude (°)	3.5 (1.0) [2.5–5.4]	4.7 (1.3) [2.9–6.7]	4.6 (1.4) [3.1–6.6]	3.7 (0.8) [2.8–5.2]	4.3 (1.2) [3.0–6.8]	4.5 (1.1) [3.4–6.5]	4.3 (0.7)
Scanpath length (°)	540.8 (87.3) [432.9–698.4]	445.1 (110.9) [321.9–608.1]	408.9 (88.9) [322.4–525.6]	528.4 (172.6) [334.7–809.9]	409.5 (107.2) [287.3–619.7]	362.0 (59.9) [278.3–431.2]	358.4 (74.2)

Yet, the individual reading speeds of 12 patients were classed as unimpaired in that they fell within two standard deviations of the average performance of control participants (A (n=7): 140.1 wpm (SD: 11.2, range: 127–162); B (n=5): 133.4 wpm (SD: 11.4, range: 120–148). However, these patients nevertheless complained of a reading impairment, especially when comparing reading performance with their premorbid performance as very skilled and avid readers. After treatment, they also showed a significant improvement in reading performance, which they were satisfied with. Their mean reading speed increased to 166.3 wpm (SD: 10.7, range: 156–187) in Group A (n=7) and to 160.2 wpm (SD: 12.0, range: 148–179) in Group B (n=5); their mean increase in reading speed was on average (A) 26.1 wpm (SD: 9.3, range: 17–45) and (B) 26.8 wpm (SD: 5.6, range: 18–32), respectively, and reached statistical significance (A: $t_{(6)}= -7.42$, $p<0.001$; B: $t_{(4)}= -10.64$, $p<0.001$).

Oculomotor reading measures

The reading eye-movement patterns (recorded in a representative sub-sample of 7 patients for each treatment group) were characterised by an increased number of fixations, a higher percentage of fixation repetitions as well as prolonged fixation durations. Saccades were much smaller and patients made many more forward saccades. Length of reading scanpaths was markedly increased (see Table 2, T2). There were no significant differences in these oculomotor measures between treatment groups (largest $t_{(12)}=1.59$, $p=0.138$), except for number of fixations ($t_{(12)}=3.30$, $p=0.006$).

Visual exploration performance

All patients in both treatment groups showed markedly elevated visual exploration times (see Table 3, T2), and there were no significant differences between groups (see Table 1).

Table 3 Visual exploration performance and related oculomotor measures before (T2) and after treatment (T3) [mean (SD), [range]]. Normative data from control samples are given for comparison (N)

	Text training (Group A: n=20)		Non-text training (Group B: n=20)		
	T2	T3	T2	T3	N
Time (s)	31.8 (14.6) [14–65]	30.7 (14.0) [12–59]	34.5 (13.9) [15–72]	34.2 (13.6) [14–69]	13.2 (1.3)
Number of fixations	50.4 (10.4) [32–59]	49.1 (11.6) [27–58]	47.3 (7.0) [39–58]	44.9 (6.2) [38–53]	21.0 (4.0)
Fixation repetitions (%)	23.8 (8.8) [9.4–36.5]	23.8 (8.2) [14.6–35.4]	25.4 (9.6) [12.2–39.6]	25.8 (7.1) [15.8–34.0]	12.7 (6.2)
Fixation duration (ms)	300.0 (57.7) [200–400]	278.6 (27.9) [250–330]	314.3 (69.0) [200–400]	305.7 (48.3) [260–380]	270.0 (20.0)
Saccadic amplitude (°)	4.7 (0.4) [4.1–5.2]	5.0 (0.8) [4.0–6.5]	4.5 (0.7) [3.4–5.3]	4.4 (0.7) [3.3–5.2]	5.6 (0.7)

Although the individual visual exploration times of 2 patients (A: 1; B: 1) were classed as unimpaired in that they fell within two standard deviations of the average

performance of control participants, these patients nevertheless complained about colliding with objects and navigation difficulties.

Oculomotor visual exploration measures

The eye-movement patterns in visual exploration (recorded in a representative sub-sample of 7 patients for each treatment group) were characterised by an increased number of fixations and a higher percentage of fixation repetitions. There was also a modest decrease in saccadic amplitude and increase in fixation duration (see Table 3, T2). There were no significant differences in these oculomotor measures between treatment groups (largest $t_{(12)}=0.77$, $p=0.455$).

3.2. The effect of text and non-text training: Within- and between-group analyses

All patients in both treatment groups reported an improvement in reading after training. Patients described reading to be much quicker, more fluent and less effortful than before training; they also reported that omitting syllables and words occurred only very rarely and reading became much more accurate. In addition, they reported to be more efficient in guiding eye-movements through the text and that comfortable reading time increased substantially. However, all patients still complained of the same difficulties in visual exploration that were reported before treatment. These subjective reports were in close agreement with the treatment effects as verified by objective test results and similar in both training groups: All patients showed an increase in reading speed and accuracy as well as more systematic reading eye-movement patterns whereas visual exploration performance and related eye-movement patterns remained impaired.

During training sessions, patients who practiced eye-movements with text material often tried to guess the presented yet only half-seen word rather than following the instruction to first inspect each word by making an eye-movement, which is consistent with previous observations (Zihl, 2000); moreover, the reading task itself, i.e., processing, identifying and reading the presented words, often seemed to distress patients. Patients who

practiced eye-movements with non-text material less frequently reported to be distressed, tired or frustrated during training sessions than patients who received text training. When asked whether they were satisfied with the treatment outcome, all patients of both groups replied in the affirmative.

Reading performance

The results are illustrated by Figure 1 (see also Table 2). Both, text and non-text training led to a significant improvement in reading performance (A: n=20, B: n=20), as indicated by a significant effect of time on corrected reading speed in both treatment groups (A: $F_{(1.0,19.9)}$=73.49, p<0.001; B: $F_{(1.1,20.3)}$=90.96, p<0.001).

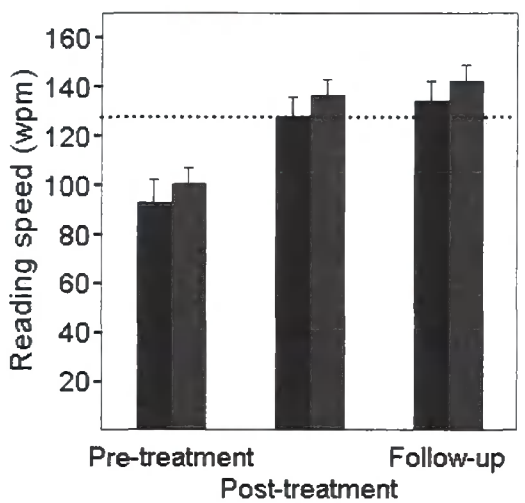

Fig. 1 Mean reading speed (wpm) before and after treatment and at follow-up for both training groups (text training: black bars, non-text training: grey bars; vertical bars indicate 1 SE). Dotted line indicates cut-off value based on control sample (n=80).

Reading speed remained unchanged between initial and pre-treatment assessment (A: $t_{(19)}$= –1.72, p=0.101; B: $t_{(19)}$= –0.81, p=0.426). After treatment, reading speed increased significantly (A: $t_{(19)}$= –7.62, p<0.001; B: $t_{(19)}$= –8.87, p<0.001); in addition, patients did not show visual omission and guessing errors any longer. Although patients of both groups showed another very small yet significant increase after follow-up (A: +6.4 wpm (SD: 3.6,

range: 2–15), $t_{(19)}$= –7.92, p<0.001; B: +5.9 wpm (SD: 3.7, range: 1–16), $t_{(19)}$= –7.12, p<0.001), the major improvement in reading performance was confined to the treatment interval (see Table 4). The pre–post-treatment increase in reading speed was consistently and significantly larger than the very small increase after follow-up, which is also unlikely to reflect any meaningful difference in reading performance (A: $t_{(19)}$=5.89, p<0.001; B: $t_{(19)}$=6.79, p<0.001 [two-tailed related samples t-test]).

Table 4 The effects of text and non-text training on reading performance and related oculomotor measures during the treatment interval [mean (SD, range); magnitude of mean improvements are also given in %]

	Text training (Group A: n=20)	Non-text training (Group B: n=20)
Increase in reading speed (wpm)	+35.0 (20.5, 13–91) [+38%]	+35.7 (18.0, 13–82) [+35%]
Decrease in number of fixations	–38.7 (9.5, 25–48) [–37%]	–25.1 (8.1, 12–37) [–33%]
Decrease in fixation repetitions (%)	–7.8% (1.9, 5.3–10.4) [–30%]	–11.7% (8.6, 0–23.7) [–41%]
Decrease in fixation duration (ms)	–85.7 (66.3, 0–190.0) [–24%]	–107.1 (76.1, 0–210.0) [–31%]
Decrease in number of forward saccades	–22.7 (9.3, 9–36) [–33%]	–12.9 (8.7, 1–28) [–24%]
Increase in saccadic amplitude (°)	+1.2 (0.7, 0.4–2.3) [+34%]	+0.6 (0.6, 0.1–1.6) [+16%]
Decrease in scanpath length (°)	–95.8 (62.1, 30.8–187.4) [–18%]	–118.9 (99.5, 36.1–294.0) [–23%]

The individual reading speeds of 8 patients in Group A (89.0 wpm (SD: 16.8, range: 64–113) and of 6 patients in Group B (101.2 wpm (SD: 16.6, range: 79–118) were still classed as impaired in that they fell below two standard deviations of the average performance of control participants after treatment. However, these patients showed a significant improvement in reading performance (A (n=8): +26.1 wpm (SD: 9.0, range: 13–41), $t_{(7)}$= –8.24, p<0.001); B (n=6): +22.7 wpm (SD: 11.0, range: 13–38), $t_{(5)}$= –5.04,

p=0.004) and reported to be satisfied with this outcome when compared with their pre-treatment performance (A: 62.9 wpm (SD: 23.2, range: 33–96); B: 78.5 wpm (SD: 21.4, range: 50–105)).

Between-group analyses (n=40) revealed that these treatment effects of text and non-text training were the same (see Table 4). There was a significant main effect of time across treatment groups ($F_{(1.06,40.1)}$=162.73, p<0.001), and neither the effect of treatment group nor its interaction with time were significant ($F_{trgroup(1,38)}$=0.87, p=0.358; $F_{int(1.06,40.1)}$=0.01, p=0.938). Mean increases in reading speed did not differ between groups ($t_{(38)}$= –0.12, p=0.903).

Oculomotor reading measures

Likewise, both text and non-text training led to a significant improvement in reading eye-movements (recorded in 7 patients for each treatment group), as reflected in a significant effect of time for all oculomotor reading measures in both groups (A: smallest $F_{(1.1,5.3)}$=8.92, p=0.028; B: smallest $F_{(1.0,5.0)}$=6.94, p=0.046). The results are illustrated by Figure 2 (see also Table 2).

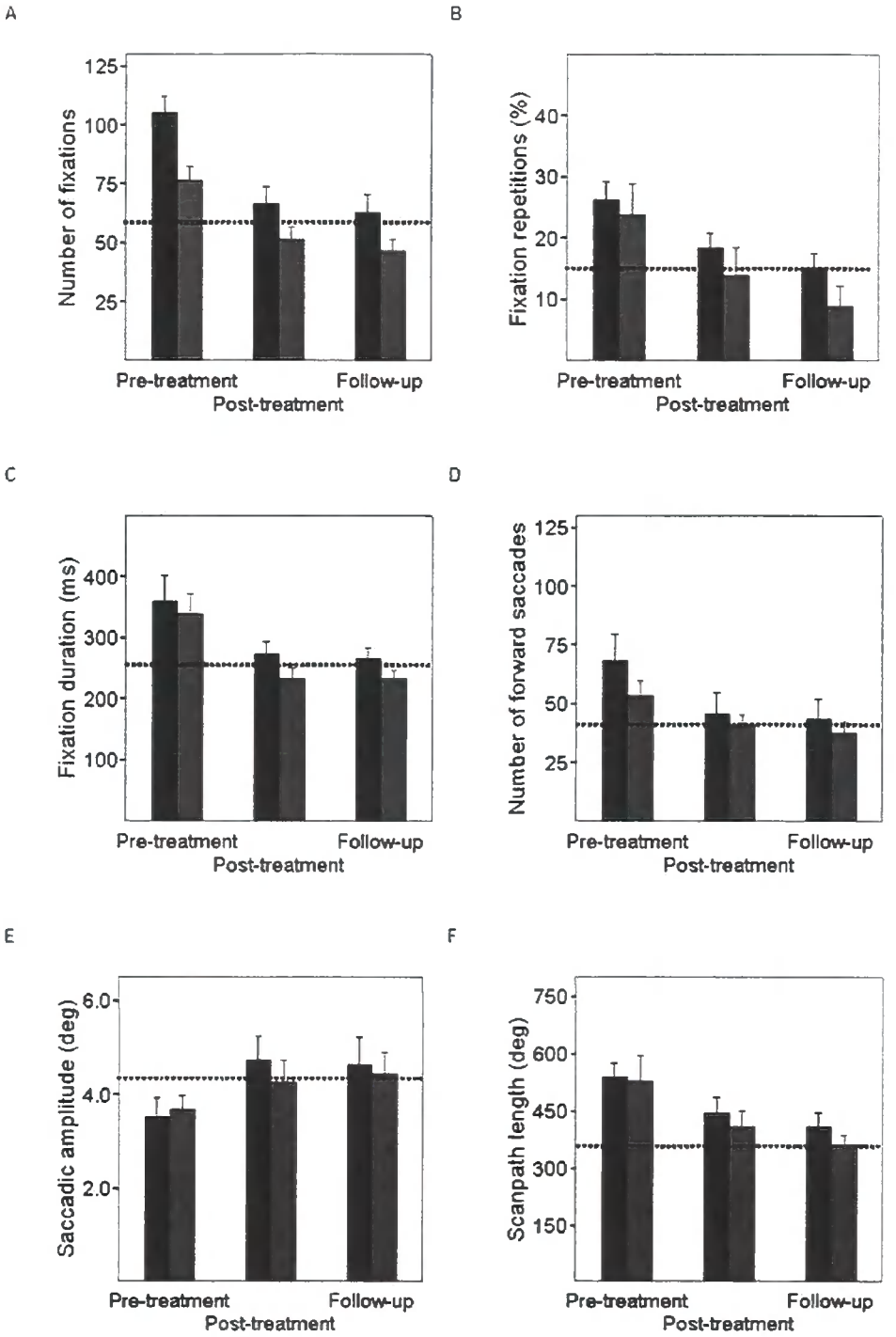

Fig. 2 Mean number of fixations (**A**), fixation repetitions (%) (**B**), mean fixation duration (ms) (**C**), mean number of forward saccades (**D**), mean saccadic amplitude (°) (**E**) and scanpath length (°) (**F**) before and after treatment and at follow-up for both training groups (text training: black bars, non-text training: grey bars; vertical bars indicate 1 SE). Dotted lines indicate average values for normal, age-matched controls (n=25).

Pre- and post-treatment comparisons revealed a significant decrease in number of fixations, percentage of fixation repetitions, fixation duration, number of forward saccades

and scanpath length as well as a significant increase in saccadic amplitude (A: smallest $t_{(6)}$=3.42, p=0.014; B: smallest $t_{(6)}$= –2.67, p=0.037). After follow-up, all oculomotor measures remained unchanged (A: largest $t_{(5)}$=1.17, p=0.296; B: largest $t_{(5)}$= –.877, p=0.421).

Although there was another very small yet significant mean decrease after follow-up in both groups for number of fixations (A: –5.5 (SD: 1.6, range: 4–8); B: –4.5 (SD: 2.3, range: 1–8)), number of forward saccades (A: –5.5 (SD: 4.5, range: 1–14); B: –3.8 (SD: 2.6, range: 2–8)) and scanpath length (A: –9.1° (SD: 5.4, range: 0.5–15.8); B: –12.5° (SD: 5.8, range: 9.0–23.4)) (A: smallest $t_{(5)}$=2.99, p=0.030; B: smallest $t_{(5)}$=3.41, p=0.019), the major decrease was confined to the treatment interval: The pre–post-treatment decrease in these oculomotor measures was consistently and significantly larger (see Table 4) than their very small decrease after follow-up, which is also unlikely to reflect any meaningful difference in eye-movement measures (A: smallest $t_{(5)}$=2.95, p=0.032; B: smallest $t_{(5)}$=2.34, p=0.066 (marginal significance, possibly due to a large variation in individual pre–post-treatment decreases in scanpath length (see Table 4) [two-tailed related samples t-test]).

The treatment effects of text and non-text training were the same (see Table 4), which is supported by the significant main effect of time across treatment groups for all oculomotor reading measures (smallest $F_{(1.3,12.9)}$=17.53, p=0.001) and the absence of a significant effect of treatment group and its interaction with time (largest $F_{trgroup(1,10)}$=1.81, p=0.208; largest $F_{int(1.1,10.9)}$=3.16, p=0.101). The significant main and interaction effects for number of fixations ($F_{trgroup(1,10)}$=5.56, p=0.040; $F_{int(1.1,11.3)}$=9.00, p=0.010)) were caused by a significant difference between both groups in mean number of fixations, which was confined to pre-treatment assessment only: Group A showed a higher mean number of fixations (105.3, SD: 18.1) than Group B (76.7, SD: 14.0) (one-way ANOVA, $F_{(1,13)}$=10.89, p=0.006). Mean improvements in reading eye-movements did not differ between groups (largest $t_{(12)}$=1.73, p=0.110). Only the improvements in mean number of fixations differed significantly between treatment groups (A: –38.7, SD: 9.5 (mean decrease relative to pre-treatment assessment: –37%); B: –25.1, SD: 8.1 (mean relative decrease: –33%); $t_{(12)}$=2.88, p=0.014).

The magnitude of the difference between groups is, however, so small (a 4% difference in the relative decrease of mean number of fixations) that it is unlikely to reflect any meaningful difference in the treatment effects of text and non-text training.

Visual exploration performance

Neither text nor non-text training had an effect on visual exploration performance (A: n=20, B: n=20) (see Table 3). Although Group A showed a significant decrease in mean visual exploration time after treatment ($F_{(1,19)}$=6.31, p=0.021), this improvement was very small (–1.11s (SD: 2.00)) and is unlikely to reflect any meaningful difference in visual exploration performance; visual exploration performance of all patients was still impaired (except for 1 patient, see above). There were no significant pre–post-treatment changes for Group B ($F_{(1,19)}$=0.33, p=0.573). The statistically (but not clinically) significant effect found for Group A explains the significant main effect of time across treatment groups ($F_{(1,38)}$=4.80, p=0.035). Yet, again, neither the effect of treatment group nor its interaction with time were significant ($F_{trgroup(1,38)}$=.48, p=0.492; $F_{int(1,38)}$=1.92, p=0.174).

Oculomotor visual exploration measures

Likewise, text and non-text training had no effect on any of the oculomotor visual exploration measures (obtained in 7 patients for each treatment group), as indicated by the non-significant of effect of time in both groups (A: largest $F_{(1,6)}$=2.22, p=0.187; B: largest $F_{(1,6)}$=1.00, p=0.356) (see Table 3). The only significant effect was found for mean number of fixations in Group B ($F_{(1,6)}$=21.15, p=0.004); patients showed a very small yet significant decrease after treatment (–2.4 (SD: 1.4); $t_{(6)}$=4.60, p=0.004), which is, however, unlikely to reflect any meaningful difference in visual exploration performance. The absence of a significant effect of time across treatment groups (largest $F_{(1,12)}$=2.31, p=0.155), except for mean number of fixations ($F_{(1,12)}$=9.18, p=0.01), confirmed this result. Again, neither the effect of treatment group nor its interaction with time were significant (largest $F_{trgroup(1,12)}$=1.57, smallest p=0.234; largest $F_{int(1,12)}$=1.71, p=0.216).

Visual field extent

None of the patients' visual fields changed between initial, pre- and post-treatment assessments, with the exception of one patient who showed a small increase of 0.5° in visual field sparing after treatment. The same increase was found in 6 patients of Group A and in 2 patients of Group B after the follow-up interval. The size of this change however lies within perimetric measurement error. Neither text nor non-text training had an effect on visual field extent (A: n=20, B: n=20): There was no significant effect of time ($F_{(1.5,29.3)}$=2.59, p=0.104) in Group B, and the significant effect of time for Group A ($F_{(1.4,25.7)}$=10.60, p=0.001) was accounted for by a small yet significant increase in visual field sparing during follow-up ($t_{(19)}$= −3.20, p=0.005). This increase also explains the significant main effect of time across treatment groups ($F_{(1.4,54.9)}$=12.65, p<0.001). Again, neither the effect of treatment group nor its interaction with time were significant ($F_{trgroup(1,38)}$=0.87, p=0.358; $F_{int(1.4,54.9)}$=2.87, p=0.081).

4. Discussion

The main result of this study is that systematic oculomotor training using time-limited presentation of non-text material has strong therapeutic effects on reading performance and associated eye-movements in patients with hemianopic dyslexia. It is as effective as conventional oculomotor training with text material in alleviating the reading difficulties associated with homonymous visual field disorders. In addition, these treatment effects were found to be specific to reading; there was no transfer of training-related improvement to visual exploration performance and associated eye-movements.

Before treatment, all patients showed considerably reduced reading speeds, visual omission and guessing errors and severely disorganised eye-movement patterns, which is consistent with previous reports on hemianopic dyslexia (De Luca et al., 1996; Eber et al., 1987; Kerkhoff et al., 1992; Leff et al., 2000; Mackensen, 1962; McDonald et al., 2006; Schoepf & Zangemeister, 1993; Spitzyna et al., 2007; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a, 2000). In agreement with investigations of visual exploration in

homonymous visual field disorders, patients also showed impaired visual exploration performance (Tant, Cornelissen, Kooijman, & Brouwer, 2002; Zihl, 1995b, 1999, 2000).

During the period of treatment, non-text reading training led to the same statistically as well as clinically significant improvements in reading performance and eye-movement measures as did text training. These treatment effects were characterised by an increase in reading speed, which was accompanied by a normalization of the oculomotor scan-pattern in reading. Patients made significantly fewer fixations and fixation repetitions and showed much shorter fixation durations. Saccadic amplitudes increased, leading to a much smaller number of forward saccades. After training, patients seemed to extract the same amount of text information by using a much more efficient oculomotor text processing strategy, which is also reflected by the significant decrease in scanpath length.

It is important to note that the improvements in reading performance and associated eye-movements cannot be attributed to spontaneous recovery of the visual field or spontaneous oculomotor adaptation. No patient showed any major change in the parafoveal visual field border. There was no change in reading performance between initial and pre-treatment assessment. The major improvement in reading performance and eye-movement parameters was confined to the treatment interval. The additional small increase of reading speed as well as the small decrease of number of fixations, forward saccades and scanpath length after follow-up were very small and are unlikely to reflect any meaningful difference in reading performance and eye-movement parameters; these changes possibly resulted from continued regular reading at home (Zihl, 2000). The improvements in reading with long-term stability (at least for a period of 12 weeks) are therefore attributable to systematic reading-related oculomotor training with text or non-text material.

These results are consistent with the findings of earlier studies (Kerkhoff et al., 1992; Spitzyna et al., 2007; Zihl, 1995a, 2000; Zihl et al., 1984) and confirm the single report on the therapeutic effect of systematic oculomotor practice using time-limited presentation of static text material in the rehabilitation of hemianopic dyslexia (Zihl, 2000). Yet, more

importantly, this is the first study to show that the therapeutic effect of this treatment procedure does not critically depend on using words as training material. Systematic reading-related oculomotor training using time-limited presentation of non-text material suffices to facilitate oculomotor adaptation to parafoveal visual field loss, which alleviates the impairments of word identification and oculomotor control during text processing.

Oculomotor adaptation to parafoveal visual field loss is reflected in the training-related changes of oculomotor reading measures (see also Zihl, 1995a, 2000), and is possibly best understood as functional reorganisation of reading eye-movement control. Hemianopic dyslexia is caused by a disturbance of the visual bottom-up and attentional top-down control of text processing and eye-movements (see Chapter 1). This disturbance becomes manifest as a severely disorganised eye-movement pattern in reading, impairments of word identification and slowness of reading. The results of the present study confirm that by oculomotor training, patients can regain the systematic and regular staircase-like eye-movement pattern of normal readers, leading to an improvement in reading performance. These training-related oculomotor changes might emerge as an adaptive solution to the problem of learning how to read efficiently, i.e., to process text information correctly and at the same time as quickly as possible (Reichle & Laurent, 2006), without parafoveal vision. Re-learning reading eye-movement control with parafoveal visual field loss and the consequent improvements in reading performance confirm the importance of precise and effective oculomotor control in reading. It shows that, ultimately, it is not the visual span (the range of letters that can be identified without moving the eyes) (Legge et al., 2007) or simple fixation disengagement (Liversedge et al., 2004) which imposes a limit on reading speed. The effectiveness of reading eye-movement control which brings the visual span 'in action' is decisive. The bottom-up control of text processing and eye-movements, which is based on parafoveal vision in normal readers, can be substituted by an attentional top-down control, suggesting the functional plasticity of the visual, attentional, oculomotor and linguistic systems involved in reading (see Chapter 1).

(Re-)learning reading eye-movement control implies (re-)learning to coordinate not only the visual, attentional, and oculomotor processes but also the linguistic processes that control text processing and eye-movements in reading (Rayner, 1998; Rayner & Pollatsek, 1989). Interestingly, learning oculomotor control in beginning readers is accompanied by changes in reading speed and eye-movements (McConkie et al., 1991; Rayner, 1985, 1986) which may be similar to the training-related changes in patients who re-learned oculomotor control in reading. These developmental changes in reading speed and eye-movements and, thus, in oculomotor control are commonly assumed to follow from linguistic skill acquisition during years of extensive reading practice with linguistic material (Rayner & Pollatsek, 1989). In the present study's patients, however, the training-related changes in reading speed and eye-movements cannot be explained by improvements in linguistic skills. These patients have already had acquired the linguistic skills necessary for sufficient reading performance. In addition, re-learning reading eye-movement control to make these premorbidly acquired intact linguistic skills useful for reading again does not seem to require reading practice with linguistic material. The finding that lexical-semantic linguistic processes are not critical to the training-related changes associated with re-learning oculomotor control in skilled readers without parafoveal vision becomes therefore all the more interesting.

This finding also suggests a transfer of training-related oculomotor adaptation from processing visual symbols (Arabic digits) to reading words, sentences and even text passages. No direct practice with text material seems to be necessary for integrating the training-related oculomotor changes into visual and linguistic processing of text information. The lack of transfer to visual exploration indicates, however, that the training-related oculomotor adaptation is nevertheless highly specific and task-dependent. This study showed for the first time that the treatment effects of systematic oculomotor training with text (words) or non-text material (Arabic digits) are specific to reading. While this training procedure could significantly improve reading performance and associated eye-movements, it had no effect on patients' visual exploration impairment. Visual exploration performance

remained markedly reduced and the associated eye-movement pattern severely disorganised. This lack of transfer between reading and visual exploration suggests that both visuo-motor abilities require specific training for their improvement. This finding is however not only of high clinical relevance but also indicates that control of visual processing and eye-movements in reading may be mediated by different neural networks than in visual exploration. Although these networks probably overlap, the result illustrates the dissociability of reading- and visual exploration-related visual, attentional and oculomotor processes (Zihl, 1995a, 1995b, 2000). Task-specificity of oculomotor adaptation may also suggest functional specialisation of the (cortical) oculomotor system in a task-specific way (Alahyane et al., 2007).

Further support stems from the clear double dissociation between spontaneous oculomotor adaptation to homonymous visual field loss in visual exploration and reading. It has been found that patients may successfully overcome their visual exploration impairment while their ability to read remains impaired, and vice versa. In addition, patients are more likely to overcome their impairment in visual exploration (40%) than in reading (20%) (Zihl, 2000). The differences between eye-movement patterns during reading and visual exploration provide additional evidence for this study's claim; the visually and linguistically structured environment in reading requires a notably different visual sampling strategy than a complex and less systematic scene (Liversedge & Findlay, 2000; Rayner, 1998). Moreover, the oculomotor pattern in visual exploration seems to become adult-like early in infant development (Shea, 1992) whereas the regular staircase-like oculomotor reading pattern requires years of laborious reading practice to develop (Rayner, 1998). These differences in developmental trajectories of eye-movement patterns between visual exploration and reading further substantiate this study's claim.

A recent report showing that an oculomotor training regime that involved practising visual exploration of pictures had no effect on the reading impairment of patients with parafoveal visual field loss complements this study's finding (Spitzyna et al., 2007).

Hemianopic dyslexia cannot be alleviated by practising any voluntary eye-movements with any visual material. Practising rather smaller, very precise, systematic and regular horizontal saccadic eye-movements with words seems to be essential (Zihl, 2000). This study's results show, however, that the treatment effect does not depend on their linguistic properties; their visual properties are essential. This finding is consistent with the significance of visual word-length information for spatial eye-movement control in reading (Ducrot & Pynte, 2002; Inhoff et al., 2003; Inhoff, Radach, Starr, & Greenberg, 2000; Rayner, 1998). Converging evidence stems from studies investigating the effect of pure oculomotor training tasks on reading performance in patients with age-related macular degeneration (Seiple, Szlyk, McMahon, Pulido, & Fishman, 2005) and in patients with reading difficulties of oculomotor and/or visual origin after acquired brain injury (Ciuffreda, Han, Kapoor, & Ficarra, 2006).

Remediation of hemianopic dyslexia may solely depend on perceptual and oculomotor (procedural) learning processes (Ofen-Noy, Dudai, & Karni, 2003), which are modulated by attention. Training-related oculomotor adaptation possibly emerges as a result of motor learning. Motor performance improves through specific practice with error-related feedback (Lisberger, 1988), which enables patients to acquire a flexible eye-movement pattern optimal for efficient text processing without parafoveal vision. Interestingly, the training-related oculomotor changes were characterised by interindividual variability, suggesting that regaining successful text processing and reading performance may not necessarily depend on one specific combination of oculomotor changes. The same outcome can be reached by different combinations, which is in line with the concept of equifinality in motor learning (Cicchetti & Blender, 2006).

The neural mechanisms mediating these learning processes and thus the therapeutic effect in the rehabilitation of hemianopic dyslexia are still unknown (for a discussion of potential mechanisms, see Chapter 1). The findings of the present study suggest that the cortical structures supporting lexical-semantic processing of words, i.e., the left inferior temporal gyrus (Leff et al., 2001) and the left posterior superior temporal gyrus (Binder et

al., 1997; Powell et al., 2006), may not be involved. Whether activation of the left and right fusiform gyrus located in the occipito-temporal region implicated in visual identification of single Arabic digits (Dehaene & Cohen, 1995; Dehaene et al., 2004) is critical for mediating the therapeutic effect remains to be investigated.

Although the effects of reading-related oculomotor training using non-text material were not superior to those obtained with text material, there were fewer reports and observations of frustration, distress and tiredness during training with non-text material than with text material. The clinical observations from training sessions suggest that practising eye-movements with non-text material (Arabic digits) may enhance the rehabilitation experience as patients need not to be confronted with a reading task (and the additional cognitive load associated with it, see Lien et al., 2008; McCann et al., 2000; Shaywitz et al., 2001) where they may be distressed by learning to compensate for their visual impairment. As the use of text material confers no advantage in the rehabilitation of hemianopic dyslexia and may be less preferred by patients than non-text material there seems little reason to select text rather than non-text material in this oculomotor training protocol. Reading-related oculomotor training with non-text material may also be a useful treatment option for children with visual field disorders after brain injury (see also Han, Ciuffreda, & Kapoor, 2004). Unfortunately, cerebral visual field disorders often remain undiagnosed in the paediatric population (Kedar, Zhang, Lynn, Newman, & Biousse, 2006) and no report has dealt with the effects of parafoveal visual field loss on reading in children thus far, let alone potential therapeutic interventions (Zihl & Priglinger, 2002). Children with parafoveal visual field loss are not only confronted with learning to compensate for their visual impairment but have yet to acquire the visual, linguistic and oculomotor skills involved in reading. Since even healthy beginning readers seem to benefit from oculomotor training with non-text material (e.g., Lehtimäki & Reilly, 2005), it may be all the more useful to improve oculomotor control in children suffering from visual field disorders.

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VISUAL FUNCTIONING IN HEMIANOPIA: VISION IS WHAT THE EYES MAKE OF IT

1. The eyes have it: Part I. Understanding functional impairments in homonymous visual field disorders

This thesis studied the nature and rehabilitation of the functional impairments in HH. Although the hemianopic reading, visual exploration and line bisection impairments are frequent and well-established clinical phenomena, their causes are largely unknown. There is still considerable debate whether the reading and visual exploration impairments are caused by the visual field defect or by additional extrastriate injury preventing efficient spontaneous oculomotor adaptation. It is also unclear whether the line bisection impairment arises from the visual field defect or its adaptive oculomotor consequences, or whether it is an indicator of an associated visual-spatial deficit that is caused by additional injury to regions and fibre pathways involved in visual-spatial perception. Since hemianopic dyslexia is the most important but most neglected of these impairments, the major focus of this thesis was on hemianopic dyslexia and its rehabilitation.

The purpose of Chapter 1 was to develop a theoretical explanation of hemianopic dyslexia as well as to clarify its functional and anatomical bases by critically examining research into hemianopic dyslexia and its rehabilitation since its original description in 1881. The hemianopic visual field defect resulting from left- or right-sided postchiasmatic visual pathway injury was identified as necessary but not sufficient to cause hemianopic dyslexia. The review showed that it possibly requires additional extrastriate injury to structures involved in the control of visuospatial attention and eye-movements in text processing for this impairment to persist. In this regard, injury to the occipital white matter and/or posterior thalamus has been implicated. Hemianopic dyslexia was therefore explained as a special type of reading disorder that is caused by injury to the neural network subserving the visual bottom-up and attentional top-down control of visual information processing and eye-movements involved in reading, i.e. a *visual-attentional-oculomotor-network disorder.*

To establish the extent to which the hemianopic reading, visual exploration and line bisection impairments are purely visually elicited, HH was simulated in normal observers by using a gaze-contingent display paradigm. This experimental paradigm allows studying the behavioural changes associated with the hemianopic visual field defect in reading, visual exploration and line bisection that are not caused by brain injury.

The experiments presented in Chapter 2 investigated the effects of simulated HH on reading, visual exploration and saccadic accuracy and determined whether and to what extent normal observers spontaneously adapt their eye-movements to simulated HH in reading and in visual exploration. Simulated HH was found to induce the reading, visual exploration and saccadic accuracy impairments of hemianopic patients in normal observers. However, all normal observers showed efficient spontaneous oculomotor adaptation to simulated HH which led to close to normal visual exploration performance and associated eye-movements. Although normal observers also showed improved reading performance and eye-movements, reading with simulated HH remained impaired. These findings suggest that the hemianopic visual field defect is a major component of the hemianopic reading impairment but is unlikely to be the causative factor in the hemianopic visual exploration impairment, although it must contribute to it. Both functional impairments rather seem to be caused by additional extrastriate injury preventing efficient spontaneous oculomotor adaptation. Injury to the occipital white matter and/or posterior thalamus seems to be associated with hemianopic dyslexia whereas injury to the ipsilateral occipito-parietal cortex and/or posterior thalamus appears to be critical for the visual exploration impairment. The cross-over study of spontaneous oculomotor adaptation to simulated HH in reading and visual exploration presented in Chapter 3 further investigated whether spontaneous oculomotor adaptation is task-specific. Since there was no transfer of adaptation-related improvements between reading and visual exploration, it was concluded that oculomotor adaptation to hemianopic visual field loss is highly specific and task-dependent, suggesting that the reading and visual exploration impairments have distinct but possibly overlapping neural and functional bases.

The two experiments reported in Chapter 4 investigated the effect of simulated HH on line bisection performance and associated eye-movements in a manual and ocular line bisection task. Although simulated HH was found to impair line bisection and induced the contralaterally deviated eye-movement pattern of hemianopic patients, it did not induce the main feature of the hemianopic line bisection impairment, i.e. the contralateral bisection error. Thus, although the hemianopic visual field defect and its adaptive oculomotor (and thus attentional) consequences may contribute to the contralateral bisection error found in hemianopic patients, they do not seem to be its primary causes. The hemianopic line bisection impairment rather seems to be an indicator of a visual-spatial deficit which is, as with the reading and visual exploration impairments, frequently associated with HH but not primarily caused by it. It seems to require additional extrastriate brain injury, possibly to regions that are involved in visual-spatial perception.

Chapter 5 dealt with the rehabilitation of hemianopic dyslexia. The presented study investigated the basis and specificity of the therapeutic effect of an efficient compensatory treatment method for hemianopic dyslexia which has been unclear thus far. Systematic oculomotor reading training using time-limited presentation of non-text material was shown to be as effective as conventional oculomotor training with text material in alleviating the hemianopic reading impairment of patients with unilateral homonymous visual field loss. There was no transfer of treatment-related oculomotor adaptation and consequent improvements in reading performance to visual exploration, which is consistent with the task-specificity of spontaneous oculomotor adaptation to visual field loss found in Chapter 3. It was therefore concluded that lexical-semantic processes are not critical to the treatment effect which is specific to reading.

By combining behavioural and oculomotor techniques, this thesis demonstrated that the hemianopic reading, visual exploration and line bisection impairments may not simply be failures of vision. Although the hemianopic visual field defect is a major component of hemianopic dyslexia and possibly contributes to the hemianopic visual exploration and line

bisection impairment, additional injury to specific extrastriate regions seems to be the critical factor that causes these functional impairments. These findings are of great importance for improving our understanding of the functional impairments associated with visual field disorders after brain injury.

Studying reading and visual exploration and associated eye-movements in simulated and real visual field loss shows that vision and vision-related functioning in visual field loss are "what the eyes (can) make of it". The locus and extent of brain damage in patients seems to determine not only the extent and quality of vision loss but also the degree of oculomotor adaptation to visual field loss. It determines what patients can make of their remaining visual field by utilising eye-movements to compensate for the visual field defect and, thus, the degree and quality of their reading and visual exploration impairments. Alterations in the eye-movements are therefore the most objective behavioural manifestation of these functional impairments that are frequently associated with visual field loss. Hemianopic dyslexia and the impairment of visual exploration may be interpreted as disorders of the visual bottom-up and attentional top-down control of visual processing and eye-movements in reading and visual exploration, respectively, which masquerade as failures of vision. The line bisection impairment, in contrast, seems to be independent of the presence or absence of oculomotor adaptation to visual field loss. It may be interpreted as a visual-spatial deficit which is frequently associated with visual field loss and its adaptive oculomotor (and thus attentional) consequences. The line bisection impairment may indicate a disorder of the egocentric visual midline in the horizontal plane.

The major future research direction arising from this thesis is to investigate the lower-level visual dysfunction (i.e. the visual field defect) and the higher-level impairment of the attentional top-down control of visual processing and eye-movements, their relative roles and interactions in causing and modulating the reading and visual exploration impairments associated with homonymous visual field loss. Likewise, the relative and interactive contributions of the lower-level visual field deficit and the higher-level impairment of both

the attentional top-down control of visual processing and eye-movements and that of visual-spatial perception to the line bisection impairment in visual field loss require further investigation. Knowledge from these investigations will advance our understanding of the nature of these functional impairments and improve the current practice of assessment and rehabilitation of the most important visual disorder after brain injury.

The lower-level visual dysfunction that contributes to the reading, visual exploration and line bisection impairments in visual field loss is characterised by the portion of the visual field affected (hemianopia, quadranopia, scotoma), by whether vision is lost in one or both visual hemifields (unilateral or bilateral field defects) and whether, in cases of unilateral field loss, the left or right hemifield is affected, as well as by the extent (visual field sparing) and quality of the visual field loss (anopia or amblyopia) (Zihl, 2000). To understand the exact contribution of the visual field defect to these functional impairments, it is essential to study the relative and interactive effects of these variables on reading, visual exploration and line bisection.

Since our knowledge about functional impairments in homonymous visual field disorders is mainly based on evidence from patients with unilateral homonymous hemianopia, the distinctive effects of bilateral hemianopias, uni- and bilateral upper and lower quadranopias as well as of paracentral and central scotomas on reading, visual exploration and line bisection remain unknown. The functional specialisation of the visual field and its effect on behaviour (Pflugshaupt et al., 2009) suggests that the effects of the type and uni- or bilaterality of visual field loss on visual information processing may be task-specific. It is therefore very important to explore the task-specificity of these effects, particularly since the visual field requirements differ quite substantially among reading, visual exploration and line bisection.

Although the effects of the side and extent of visual field loss have been studied in reading and visual exploration, it is still unclear whether line bisection is differentially affected by these variables. The impairment of visual exploration that is frequently

associated with homonymous visual field loss seems not to be determined by the side and extent of the visual field defect (Zihl, 1995b). The severity and quality of hemianopic dyslexia, in contrast, is determined by the side and extent of the visual field defect (Zihl, 1995a). In addition, these variables seem to interact with the functional demands of the writing system and the reading task. Yet, this interaction between the properties of the visual field defect and the direction of the writing system requires further investigation. Although Mauthner (1881) has already suggested that the differences in severity and quality of the reading impairment between left- and right-sided visual field loss and the effects of its extent depend on reading direction, there exists only one case study to date that confirms this assumption (Leker & Biran, 1999). Behavioural and functional neuroimaging studies of hemianopic dyslexia in right-to-left writing systems as well as direct comparisons between writing systems will help to further determine whether the difference between left- and right-sided visual field loss in reading is purely visual and to elucidate the relative importance of left-lateralized activation of the cortical structures involved in text processing and reading eye-movements (Leff et al., 2000; Leff, Scott, Rothwell, & Wise, 2001b). Since the effects of the side and extent of visual field loss have been studied only in patients with unilateral homonymous hemianopia, it remains to be investigated whether the effects of the side and extent of visual field loss also interact with the type and uni- or bilaterality of visual field loss.

The exact effects of the quality of the visual field defect, i.e. whether vision is completely lost (anopia) or one or more visual functions in the affected visual field are only reduced (amblyopia), on reading, visual exploration and line bisection also remain an open issue that requires further study. Although no report has dealt with line bisection in amblyopic visual field defects, preliminary evidence indicates that the effects of the quality of visual field loss may be task-specific and depend on visual task requirements. If the residual visual field for form vision in cerebral amblyopia is smaller than 4-5°, the quality and severity of hemianopic dyslexia does not seem to differ between amblyopic and anopic

visual field defects. The resulting visual exploration impairments, however, seem to differ between amblyopia and anopia. Visual exploration was found to be less impaired in amblyopic than in anopic visual field defects, suggesting that residual visual functions can reduce the functional impairment associated with visual field loss (Zihl, 2000). Yet, it remains to be determined whether and how residual visual functions in amblyopia may affect the resulting functional impairment.

If and to what extent 'blindsight' occurs in anopic visual field loss (Weiskrantz, Warrington, Sanders, & Marshall, 1974), i.e. the residual visual capacity to detect, localize, and discriminate visual stimuli in the affected visual field region in the absence of acknowledged visual experience, may also be used to characterise a visual field defect, although this frequently reported visual cognitive phenomenon is still considered as controversial (Cowey, 2004). Its substrate is also still debated, i.e. whether it is mediated by unaffected extrageniculo-striate mechanisms bypassing the lesion site or by surviving fibre connections within the affected damaged visual cortex (Cowey & Stoerig, 1991). Blindsight is present in 15-20% of patients with visual field disorders (Blythe, Kennard, & Ruddock, 1987) and has also been identified in children with hemianopia (Boyle, Jones, Hamilton, Spowart, & Dutton, 2005). Although preliminary evidence suggests that blindsight does not reduce patients' functional impairments (Zihl, 1980), the exact effects of blindsight on visual functioning in visual field loss remain to be determined.

This thesis also demonstrated that simulating visual field loss in normal observers may be an effective tool in studying the role of the lower-level visual dysfunction, i.e. the visual field defect, in the functional impairments associated with visual field loss. It is an attractive alternative approach to investigating patients with real visual field loss after brain injury. Simulating visual field loss allows controlling the attributes of the visual field defect, such as shape (hemianopia, quadranopia, scotoma), extent (visual field sparing), and location (uni- or bilateral, left- or right-sided field loss). Recruiting a sufficiently large sample of patients with visual field defects that are homogeneous in these variables, in contrast, is very difficult or

even impossible. In addition, using normal observers allows conducting far more extensive behavioural measurements, such as eye-movement recordings, than is practical with patients (see also Bowers & Reid, 1997; Fine & Rubin, 1999). The use of simulations is therefore of particular advantage for testing potential diagnostic tests and treatments since proper clinical trials of diagnostic tests and behavioural therapies are difficult to achieve and developing efficient training techniques in the first place is a considerable challenge.

Simulating visual field defects in patients with visual field loss may be an innovative approach in elucidating the effects of residual visual functions on functional impairments in amblyopic visual field defects as well as the impact of blindsight in this regard. Studying potential performance changes when the visual field defect of patients who show amblyopic field loss or of those with anopic field loss showing blindsight are superimposed with a simulated field defect that is equal in shape, size and location may elucidate the role of residual visual functions and blindsight capacity in the functional impairments associated with visual field loss as well as their relevance for spontaneous oculomotor adaptation. Visual neglect/neglect dyslexia, the Balint's syndrome and pure alexia are frequently accompanied by visual field defects (Coltheart, 1998; Leff et al., 2001a; Müller-Oehring et al., 2007; Walker, Findlay, Young, & Welch, 1991; Zihl, 2000). Thus, simulating visual field loss in patients with visual neglect, the Balint's syndrome or in those with pure alexia who do not show an additional visual field loss may help clarifying the role of visual field defects in the symptoms that are frequently associated with these common disorders.

However, simulating visual field loss in normal observers cannot substitute but only complement the study of real visual field loss in patients after brain injury. Simulations of visual field defects have shortcomings which limit the generalisability of the obtained results to patients with real visual field loss. A simulated visual field defect may not fully mimic all the characteristics of a real field defect and resembles only the early stages of sudden onset, acquired visual field loss (Bowers & Reid, 1997). The sharp boundary of the simulated field defect may be a feature that is unlikely to be seen in real visual field defects. Normal

observers may use the clearly visible boundary that results from the sharp transition to control their eye-movements. Patients with a real visual field loss, in contrast, are not able to resort to such strategy because they do not perceive such a boundary. However, creating a smooth transition at the visual field boundary when simulating field loss may alleviate this shortcoming (Lingnau & Schwarzbach, 2008). Moreover, an experimentally induced field defect is only present while the participant is looking at a computer screen during an experimental session and also lacks the 24-hour presence of a real visual field defect. Although it appears that patients with visual field loss experience their deficit as an obvious absence of vision, patients are not constantly made aware visually of their deficit, which is also in contrast to normal observers when being confronted with a simulated visual field defect. It is also important to note that real visual field defects are frequently accompanied by concomitant (central or peripheral) visual disorders and sometimes by oculomotor deficits or other cognitive disorders affecting attention, memory, language or executive functions (Anderson, 2003; Rowe et al., 2009; Zihl, 2000). Thus, patients who suffered from brain injury are naturally in a different condition than normal observers with a pure simulated visual field defect.

The higher-level impairment of the attentional top-down control of visual processing and eye-movements that contributes to the reading and visual exploration impairments in visual field loss is determined by the locus and extent of extrastriate brain lesions that frequently accompany postchiasmatic visual pathway injury (Zihl, 2000). Studying reading and visual exploration in simulated hemianopia and in patients with visual field loss whose injury is restricted to striate cortex showed that a "visually elicited" reading and visual exploration impairment can be quite substantial. The visual field defect impairs efficient word processing and thus identification and the visual control of reading eye-movements as well as the ability to quickly gain a complete overview and thus global processing of a scene. However, these impairments do not persist. Both populations show efficient spontaneous oculomotor adaptation which considerably improves reading and visual exploration. In

addition, this thesis demonstrated that oculomotor adaptation to visual field loss in visual exploration seems to be more likely and induces greater performance improvements than in reading. Although this finding is consistent with clinical observations of patients with visual field loss (Zihl, 2000), it remains to be verified in larger patient samples. Hence, the reading and visual exploration impairments that are frequently associated with visual field loss seem to require not only a visual field defect but also a specific additional ipsilateral extrastriate injury. However, only very few studies have analysed the anatomical basis of the reading and visual exploration impairments and, thus, of spontaneous oculomotor adaptation in patients with visual field loss. Preliminary evidence suggests that the occipital white matter comprising subcortical-cortical reciprocal connections and/or posterior thalamus may be the critical lesion locations for hemianopic dyslexia (Zihl, 1995a) whereas the occipito-parietal cortex and/or posterior thalamus seem to be the anatomical basis of the visual exploration impairment (Zihl, 1995b). Since the findings were based on an analysis of CT and MRI scans only, the extent of lesions and particularly the role of white matter injury affecting fibre pathways may still be underestimated.

To date, no report has dealt with the anatomy of the line bisection impairment. This thesis demonstrated that this impairment is neither a consequence of the visual field defect nor a manifestation of spontaneous oculomotor adaptation to visual field loss. The critical factor in causing the contralateral bisection error seems to be additional extrastriate brain injury to regions that are involved in visual-spatial perception (Best, 1919; Kerkhoff, 1993; Zihl, 2000). Although injury to posterior occipital and parietal structures have been suggested as a causative factor (Ferber & Karnath, 1999; Kerkhoff, 1993; Zihl, 2000), the critical lesion location remains to be investigated, which may also include cortical and subcortical white matter pathways, including splenial fibres (Hausmann, Waldie, Allison, & Corballis, 2003a).

Investigating the relative roles and potential interactions between the lower-level visual dysfunction and the higher-level impairment of the attentional top-down control of

visual processing and eye-movements in causing and modulating the reading and visual exploration impairments requires comparing patients with visual field loss and additional injury to the critical extrastriate areas with cases who show a similar visual field defect but no extrastriate damage as well as with patients who have normal visual fields but show a comparable injury to extrastriate areas. The same investigative approach applies to the study of the relative and interactive contributions of the lower-level visual field deficit and the higher-level impairment of both, the attentional top-down control of visual processing and eye-movements and that of visual-spatial perception in causing and modulating the line bisection impairment in visual field loss. Combining transcranial magnetic stimulation (TMS) (Stewart, Ellison, Walsh, & Cowey, 2001; Walsh & Cowey, 2000) with the simulation of visual field loss in normal observers, i.e. applying TMS to the relevant extrastriate areas in normal observers while they are confronted with simulated visual field loss, may be an alternative approach in this regard. Comparing these observers with those who are confronted with a simulated visual field defect in a no-TMS condition as well as with observers receiving TMS under normal viewing conditions might help clarifying the relative and interactive roles of the lower-level and higher-level deficits causing the functional impairments in visual field loss.

Although additional extrastriate injury seems to be the decisive factor that determines spontaneous oculomotor adaptation and thus functional impairment, awareness, time since brain injury, and age or age-related processes might also be important contributing factors. Spontaneously compensating for visual field loss by developing efficient adaptive eye-movement strategies may require awareness of the visual field defect. However, patients with visual field loss are not always aware of their visual deficit (Bisiach, Vallar, Perani, Papagno, & Berti, 1986; Celesia, Brigell, & Vaphiades, 1997; Gassel & Williams, 1963a, 1963b; Koehler, Endtz, Te Velde, & Hekster, 1986; Vallar & Ronchi, 2006). 'Anosognosia' for visual field loss is a common phenomenon since the vision loss in a particular field region is not necessarily associated with immediate sensation (Levine, 1990). Direct

experience of visual field loss is based on acquiring knowledge about failures resulting from the loss of vision, which requires curiosity, self-observation, inference, and memory (Zihl, 2000). Yet, it remains to be investigated whether awareness of the visual field defect is a prerequisite of spontaneous oculomotor adaptation.

In this regard, time since brain injury may also be an important factor to consider since developing adaptive or maladaptive compensatory strategies in visual field loss requires time (Gassel & Williams, 1963a; Poppelreuter, 1917/1990; Zihl, 2000). Moreover, age or age-related processes may also play a significant role in spontaneous oculomotor adaptation to visual field loss. This assumption is consistent with evidence for age-related changes in neural, functional and cognitive plasticity (Burke & Barnes, 2006; Craik, 2006; Hedden & Gabrieli, 2004; Reuter-Lorenz, 2002; Sowell et al., 2003) as well as in white matter pathways (Wozniak & Lim, 2006), which seem to be of particular importance for efficient oculomotor adaptation to visual field loss (Zihl, 2000). Further support stems from a single report on the effect of age on spontaneous oculomotor adaptation to simulated and real HH in visual exploration (Tant, Cornelissen, Kooijman, & Brouwer, 2002b). Hence, another important future research direction is to investigate whether and to what extent the presence or absence of awareness, time since injury and age can influence oculomotor adaptation to visual field loss and therefore co-determine patients' functional impairments. In this regard, it is also important to determine whether this influence is task-specific.

Using analyses of global spatial and temporal eye-movement measures in the study of reading in patients with visual field loss has greatly advanced our understanding of hemianopic dyslexia and its rehabilitation (Zihl, 1995a, 2000). To further advance our knowledge about the associated impairments in word identification and eye-movement control as well as treatment-related improvement, it is essential to investigate exactly which visual and lexical text information is extracted during a fixation and influences the eye-movements of patients with visual field loss during reading, and whether and how information extraction may change after treatment. Of particular interest in this regard are

the effects of word length (i.e. number of letters) as well as inter-line spacing, which possibly both interact with the side and extent of the visual field defect. Patients with visual field disorders seem to over-rely on higher-level linguistic processes to compensate for their visual field loss when trying to identify words, which disrupts further acquisition and processing of text information located in the blind hemifield and even interferes with rehabilitation. Investigating lexical effects on eye-movements in hemianopic dyslexia (e.g. word frequency, lexical constraint) seems therefore even more important. In this regard, it is of great importance to apply the word-based analyses of local spatial and temporal eye-movement measures which are standard in experimental reading research (Rayner, 1998).

Moreover, binocular eye-movement recordings may also be a valuable research tool in the study of hemianopic dyslexia since eye-movements are not fully conjugate during normal reading (Kirkby, Webster, Blythe, & Liversedge, 2008; Liversedge, Rayner, White, Findlay, & McSorley, 2006; Liversedge, White, Findlay, & Rayner, 2006) and hemianopic dyslexia research has been based on monocular eye-movement recordings only. Using binocular eye-movement recordings in the study of hemianopic dyslexia would provide insights into the binocular coordination of reading eye-movements in visual field disorders and may help determining whether and to what extent changes in fixation disparity account for spontaneous or treatment-related improvements in hemianopic dyslexia. Moreover, there is not much known about eye-movements in oral reading (Rayner & Juhasz, 2004). Studying oral reading eye-movements in hemianopic dyslexia is essential, not only because the assessment of reading speed and accuracy in hemianopic dyslexia is mainly based on oral reading tests, but also because younger children and beginning readers spend much time reading aloud. Moreover, it further helps to elucidate the typical reading errors made by these patients. With the recent technological advances leading to accurate eye-movement recording devices that do not require a fixed head, pursuing such research has become much more feasible.

Similarly to reading, it is still unclear which visual information and higher-level cognitive factors and processes influence patients' guidance of eye-movements when performing tasks involving visual exploration. In normal observers, visual exploration eye-movements are controlled by visual features, cognitive factors such as planning and sequencing, visuo-spatial attention and visuo-spatial working memory as well as on the top-down influences resulting from task requirements (Kennard, 2002; Leigh & Kennard, 2004). It has been suggested recently that patients with visual field loss may rely more on visuo-spatial memory representations of their visual environment than normal observers (Martin, Riley, Kelly, Hayhoe, & Huxlin, 2007). Visual field loss seems to induce a shift from visually-guided eye-movements to memory-guided eye-movements. Such shift from a bottom-up to a top-down control of eye-movements is consistent with findings from reading where patients show a greater reliance on higher-level linguistic processes than on lower-level visual information extraction. Yet, it remains to be determined exactly which lower-level visual information and which higher-level cognitive factors are being used for guiding eye-movements during visual exploration, and whether and how such strategies may change after treatment.

Likewise, it remains unknown exactly which factors determine line bisection performance in visual field loss. Investigating the role of the visual field defect in relation to perceptual, attentional and motor biases seems to be of particular interest in this regard. Visual field defects result in a chronic differential lateralised or asymmetric visual-sensory input, which can give rise to an attentional bias in the direction of the seeing hemifield, i.e. to ipsilateral hemispace (Tant, Kuks, Kooijman, Cornelissen, & Brouwer, 2002c). If patients with visual field loss show strategic oculomotor adaptation to their visual deficit, visual field loss seems to be associated with an additional attentional bias. This adaptive attentional bias is, however, in the direction of contralateral hemispace (Barton, Behrmann, & Black, 1998). Thus, attentional biases or imbalances do not necessarily result from a higher-level

attentional right hemisphere dysfunction but also can arise from a lower-level visual deficit such as visual field loss and its adaptive oculomotor consequences (Tant et al., 2002c).

For assessing the visual-perceptual, attentional and motor effects on the perception of line-midpoints in visual field loss, it is important to consider particularly the factors that have been found to modulate performance in the line bisection task in visual neglect as well as in normal observers, i.e. handedness and hand-use, eye of regard under monocular viewing conditions, directional (oculo-)motor scanning (left-to-right vs. right-to-left), as well as the spatial location (left, right, superior, or inferior hemispace), orientation (horizontal, vertical, diagonal, and radial), salience, and length of the line (Jewell & McCourt, 2000). Although directional oculomotor scanning, line location and length have been demonstrated to influence line bisection in HH on a single case basis (Hausmann et al., 2003a), the effects of these factors require further investigation. Our knowledge of line bisection in visual field loss is largely based on evidence from hemianopic patients bisecting horizontal lines. To further elucidate the origin of the bisection error associated with visual field loss, it is essential to examine line bisection in unilateral upper and lower quadranopia and paracentral scotoma as well as in bilateral visual field disorders using horizontal, vertical and diagonal lines. This becomes evident in two early case reports of patients with altitudinal homonymous visual field defects who show a vertical bisection error in the direction of their blind visual field as would be expected if the bisection error was a consequence of the visual field defect and/or oculomotor adaptation to it (Best, 1919; Teuber, Battersby, & Bender, 1960). Since then only one study has dealt with horizontal and vertical line bisection in five patients with upper or lower altitudinal visual field loss and additional left- or right-sided hemianopic visual field defects (Kerkhoff, 1993). In addition to confirming these early reports by demonstrating horizontal and vertical line bisection errors in the direction of the blind visual field, this study also reported an interesting case of a patient with a very small bilateral paracentral scotoma who showed a large bisection error towards the lower hemifield, which provides additional evidence against visual and adaptive

oculomotor/attentional factors as the primary causes of the visual-spatial deficit in visual field loss.

Visual field disorders are frequently accompanied by concomitant (peripheral or central) visual disorders and sometimes by oculomotor deficits or other cognitive disorders, which increases the resulting functional impairments (Anderson, 2003; Patel, Duncan, Lai, & Studenski, 2000; Rowe et al., 2009; Zihl, 2000). Since visual deficits account for the most elementary and frequent sequelae of brain injury (Rowe et al., 2009; Zihl, 2000), it is of great importance to explore the effects of other central visual disorders on reading, visual exploration and line bisection performance and eye-movements as well as their interactions with visual field defects and peripheral visual disorders. In this regard, it is essential to investigate disorders in visual acuity, spatial contrast sensitivity and visual adaptation, colour vision deficits, disorders in visual-spatial perception, the visual neglect and Balint's syndromes, visual agnosia, visual illusions and hallucinations, and visual discomfort. These visual disorders predominantly affect the visual and attentional prerequisites for the ability to read. Thus, studying their effects on reading as well as developing appropriate assessment and treatment techniques is of great importance, particularly since 'visual' reading disorders still receive too little attention (with the exception of neglect dyslexia and pure alexia, see Leff & Behrmann, 2008).

Patients with visual neglect/neglect dyslexia, Balint's syndrome or pure alexia frequently show a concomitant visual field defect. The similarities between the reading impairments associated with visual neglect/neglect dyslexia and unilateral hemianopic visual field loss and between those associated with the Balint's syndrome as well as pure alexia and bilateral hemianopic visual field loss additionally underscore the significance of investigating reading and eye-movements in these disorders (Zihl, 2000). Such investigations would not only further elucidate the role of visual and attentional processes in hemianopic dyslexia but also improve our understanding of the reading impairments in visual neglect/neglect dyslexia, the Balint's syndrome as well as in pure alexia, particularly since

studies of reading eye-movements in these disorders are rare (for exceptions, see Baylis, Driver, Baylis, & Rafal, 1994; Behrmann, Black, McKeeff, & Barton, 2002; Behrmann, Plaut, & Nelson, 1998; Behrmann, Shomstein, Black, & Barton, 2001; di Pellegrino, Làdavas, & Galletti, 2001/2002; Johnson & Rayner, 2007; Karnath & Huber, 1992; Kerkhoff & Heldmann, 1999; Lee et al., 2009; Leff et al., 2001a; Rayner & Johnson, 2005; Zihl, 2000).

2. The eyes have it: Part II. Assessing functional impairments in homonymous visual field disorders

These findings are of great importance not only for our understanding of the functional impairments associated with visual field disorders but also for improving current practice of assessment and rehabilitation. Understanding what components contribute to the reading, visual exploration and line bisection impairments is essential for assessment and rehabilitation effectiveness. This thesis advocates that the eyes and not the visual field "have it": studying eye-movements in visual field loss provides the means not only to better understand the functional impairments associated with visual field disorders but also to assess and rehabilitate them in a more efficient way.

The findings of the theoretical review and experimental studies suggest that the extent of visual field loss does not translate into patients' actual functional impairments and assessing the visual field only may therefore not be sufficient to accurately predict residual vision-related functioning (see also Papageorgiou et al., 2007; Zihl, 2000). An ecologically valid assessment, which is essential for predicting patients' functional impairments and planning of treatment, requires not only a comprehensive assessment of the visual field defect but also a detailed examination of patients' eye-movements as well as considering the locus and extent of their brain injury.

The standard assessment of patients with homonymous visual field loss should include detailed perimetric testing of monocular and binocular visual fields using light, colour and form targets in order to determine the portion of the visual field affected, whether vision is

lost in one or both visual hemifields and whether, in cases of unilateral field loss, the left or right hemifield is affected, as well as the extent (visual field sparing) and quality of the visual field loss (anopia or amblyopia) (Barton & Benatar, 2003; Simpson & Crompton, 2008a, 2008b; Zihl, 2000). Since these variables (co-)determine the resulting functional impairments, a detailed examination of these variables is of particular importance for predicting functional impairment and planning of treatment. If blindsight was found to influence the functional impairments or spontaneous visual field recovery and oculomotor adaptation, the examination of blindsight should be included in the assessment of visual field loss (see also Lane, Smith, & Schenk, 2008).

Since measuring the visual field does not suffice to accurately predict functional impairment in visual field loss, the assessment of the useful field of gaze (or visual search field) should be regarded as an obligatory complement of perimetric visual field testing. The field of gaze is the area within the visual field which patients can explore with their eyes when asked to search for a light target that is moved slowly from the periphery towards the centre of the perimeter while their head is fixed. The extent of the field of gaze is measured in degrees of visual angle in the blind and intact visual hemifield. It indicates the degree of oculomotor adaptation to visual field loss, with a small field of gaze indicating poor oculomotor adaptative strategies in visual field loss (Kerkhoff, 1999; Zihl, 2000). Determining the field of gaze, in addition to assessing the visual field, is therefore of great importance since it allows a more accurate prediction of patients' functional impairments in vision-related tasks.

However, task-specificity of oculomotor adaptation to visual field loss suggests that measuring the visual field and the field of gaze may not be sufficient for an ecologically valid assessment of visual field disorders and the associated functional impairments. It is therefore essential to obtain performance and eye-movement measures during reading, visual exploration and line bisection using appropriate and specific tasks that assess these visuo-motor functions. The development of standardised tests is of great importance, particularly

for standardising outcome measures in evaluations of potential treatment methods. To further increase the ecological validity of the assessment (and, thus, rehabilitation) of visual field disorders, it is important to establish the implications of these functional impairments for daily life and to develop corresponding diagnostic tests (and treatment methods). For evaluating potential tests and treatments, simulating visual field loss in normal observers is of great advantage.

The development of portable eye-trackers has allowed the study of everyday activities, ranging from food preparation to driving, which produced important knowledge of the significance of eye-movements for these activities (Land, 1994, 2001, 2006). Although patients with visual field loss frequently report great difficulties in daily life activities, particularly those involving orienting and navigating, only very few reports have investigated the effects of visual field loss on naturalistic task performance and eye-movements (Coeckelbergh, Cornelissen, Brouwer, & Kooijman, 2002; Martin et al., 2007; Riley, Kelly, Martin, Hayhoe, & Huxlin, 2007; Schulte, Strasburger, Müller-Oehring, Kasten, & Sabel, 1999; Tant, Brouwer, Cornelissen, & Kooijman, 2002a). Most research has established these effects with laboratory-based tasks in a two-dimensional environment, subjective reports and activities of daily living questionnaires. It is therefore of great importance to extend the study of everyday activities and associated eye-movements to patients with visual field loss. Pursuing such research will not only increase our understanding of the visual exploration impairment and its clinical significance and improve the ecological validity of its assessment and rehabilitation, but also complement the findings obtained from normal observers. Recent attempts to simulate visual field loss in virtual three-dimensional environments as a means of studying human navigation is also an interesting lead in this regard (Fortenbaugh, Hicks, Hao, & Turano, 2007).

Reading performance in visual field loss is commonly assessed using paper-based reading tests that involve reading aloud single words, sentences and short paragraphs which are easy to comprehend and where the letter size, font, colour, contrast, spacing between

letters, words and lines, spatial layout and text alignment as well as reading distance are maintained as optimal for reading (e.g. Zihl, 2000). The recent development of a reading test that is standardised for layout, content, length (in characters), word frequency and syntactic complexity and available in four European languages with normative data for different age groups is to be commended in this regard (Hahn et al., 2006). Yet, reading in daily life usually involves silent reading of text material that is presented in different formats and spatial arrangements, varies in its linguistic characteristics and may not always be static. In addition, it often involves the combination of reading and visual exploration skills as, for instance, studies of newspaper, net paper and map reading demonstrate (Holmqvist, Holsanova, Barthelson, & Lundqvist, 2003; Lobben, 2007). Since the range of day-to-day electronic applications for a variety of purposes is expanding, the display of text on (computer) screens has become more commonplace than the display of text on paper. However, processing and reading text on monitors differs quite substantially from reading printed text. Monitor reading has been found to be slower and more visually fatiguing than reading from paper. Physical aspects of monitor text presentation as well as software design (e.g., navigation structure) seem to account for this performance difference and may also explain why monitor reading involves different reading strategies for extracting text information than reading from paper (Holmqvist et al., 2003; Kruk, 1993). Further evidence stems from studies investigating the behavioural strategies involved in web interaction and human-computer interaction in general (Grainger, 2003; Stenfors, Morén, & Balkenius, 2003). It is therefore of great importance to take these considerations and findings into account when studying, assessing and rehabilitating the reading impairment associated with visual field loss. In addition, it is essential to assess not only text reading but also reading multi-digit numbers since processing and reading numbers differs from word and text reading and has also been found to be more impaired than text reading in patients with visual field loss (Zihl, 2000).

Interestingly, Axenfeld (1894), who was the first to report the hemianopic line bisection impairment, advocated the line bisection task as "a simple method to diagnose hemianopia", particularly in cases where there is no access to a perimeter or when patients are not able to undergo perimetric visual field testing (see also Liepmann & Kalmus, 1900). However, the limited diagnostic value of the line bisection task in the assessment of visual field disorders is indicated by the dissociability of the contralateral line bisection error and visual field loss. Although the line bisection task is not an appropriate substitute for perimetric testing and can only complement perimetric diagnosis, it is an important tool to assess the egocentric visual midline, which is frequently impaired in visual field loss.

Yet, while the line bisection task is an established diagnostic tool in the assessment of visual neglect (Fischer, 2001), it is rarely being used in the assessment of visual field disorders. The "Landmark Task" (Milner, Brechmann, & Pagliarini, 1992), i.e. the perceptual variant of the classic manual line bisection task, has been frequently used to study line bisection in visual neglect as well as in normal observers. However, it has, with a single exception, never been used in patients with visual field loss (Doricchi, Onida, & Guariglia, 2002). Like the ocular bisection task, it might be a useful diagnostic instrument for assessing visual-spatial deficits in hemianopic patients, particularly in cases where upper extremity disorders impede the assessment of line bisection performance. In addition, it might also be a useful experimental tool to disentangle motor from perceptual and attentional biases in the study of line bisection in visual field loss. Clinical reports suggest that visual field defects are frequently associated with disorders in visual-spatial perception, which are likely to interact with visual deficits and increase resulting functional impairments in abilities requiring visual-spatial functions which also include reading and visual exploration (Zihl, 2000). Visual-spatial test performance has even been found to better predict visual performance during driving than the characteristics of the visual field defect (Tant et al., 2002a). This finding further supports this thesis's conclusion that it is not simply the visual field that "has it". It is patients' eye-movements and possibly also their visual-spatial

performance that help us better understand, assess and rehabilitate the functional impairments associated with visual field loss. It is therefore of great importance to investigate further visual-spatial perception in patients with visual field loss as well as to include a detailed examination of visual-spatial perception in the assessment of visual field disorders.

Since the contralateral bisection error is only an indicator of an underlying disorder of the egocentric visual midline, it is essential to study its implications for activities of daily living, which are, however, still unknown but important for an ecologically valid assessment and rehabilitation. Preliminary evidence obtained from hemianopic patients suggests that the contralateral bisection error is associated with impairments in visual-spatial judgements and spatial orientation problems in daily life, such as difficulties with maintaining the straight-ahead direction during walking (Kerkhoff, 1999; Zihl, 2000). Although a contralateral deviation of the visual subjective straight-ahead direction (i.e. one index of perceived body orientation in the horizontal plane) has been demonstrated in patients with left-sided HH (Ferber & Karnath, 1999), its relationship to the contralateral bisection error remains unclear. Investigations of this relationship in patients with visual neglect showed a strong positive correlation between the bisection error and the subjective straight-ahead deviation (e.g. Chokron & Bartolomeo, 1999; Richard, Honoré, Bernati, & Rousseaux, 2004). This finding suggests that both phenomena may arise from the same visual-spatial disorder, although evidence from studies that failed to show this relationship contradicts this assumption (e.g. Bartolomeo & Chokron, 1999; Chokron, 2003).

Yet, differences in the assessment of the subjective straight-ahead direction and deficiencies in methodology may account for these negative results (Richard et al., 2004), which seem surprising, particularly when considering the similarities between the manual line bisection and straight-ahead pointing tasks. Both tasks require the division of a symmetrical body-centred space into two equal left and right halves as well as programming a motor response towards a 'virtual' target, i.e. the midpoint (Halligan & Marshall, 1998).

The only difference seems to be the visual 'extra-corporeal' stimulus that is present in the line bisection but not in the straight-ahead task, suggesting that line bisection additionally involves object-based processing (Galati et al., 2000). Investigating the relative contributions of object- and space-based mechanisms to the contralateral bisection error associated with visual field loss by using line and spatial-interval bisection tasks (i.e. bisecting a specific spatial extent indicated by two markers) might be illuminating in this regard (Post, Caufield, & Welch, 2001).

In addition to assessing patients' eye-movements, it is also important to observe patients' head movements, which are restricted during perimetric testing of the visual field and the field of view, while they perform these vision-related tasks. Often patients use head- instead of eye-movements as a means to compensate for visual field loss (Zihl, 2000), thereby reversing the normal physiological sequence that head movements follow saccadic eye-movements (Uemura, Arai, & Shimazaki, 1980). Since this strategy increases the functional impairments in visual field loss (Kerkhoff, Münßinger, Eberle-Strauss, & Stögerer, 1992a), it is essential to identify maladaptive head movements in the assessment of visual field disorders.

This thesis suggests that the assessment of visual field disorders should not only include perimetric visual field testing. An ecologically valid assessment of visual field loss, which is crucial for predicting functional impairment in daily life and planning of treatment, requires determining the field of gaze as well as assessing eye-movements and performance measures separately in reading, visual exploration and line bisection. The additional consideration of the locus and extent of patients' brain injury should complement this multifaceted assessment approach since it determines the functional impairments in visual field loss. Although the effect of time since brain injury, age, and awareness on spontaneous oculomotor adaptation and, thus, functional impairment in visual field loss remains to be investigated, it may be useful to consider these factors as well. Awareness can be assessed by determining the degree of agreement between patients' subjective (vision-related)

difficulties, which can be established using specific questionnaires (Kerkhoff, Schaub, & Zihl, 1990; Papageorgiou et al., 2007), and their objective diagnostic results.

For an (ecologically) valid assessment (and treatment) of functional impairments in visual field loss, it is also important to consider the frequent co-occurrence of visual field disorders and other (peripheral or central) visual deficits, oculomotor deficits or other cognitive disorders, particularly since multiple deficits commonly increase the resulting functional impairments (Anderson, 2003; Patel et al., 2000; Rowe et al., 2009; Zihl, 2000). The similarities between the functional impairments associated with visual neglect/neglect dyslexia and unilateral hemianopic visual field loss, between those associated with the Balint's syndrome/pure alexia and bilateral hemianopic visual field loss as well as between those associated with visual acuity/spatial contrast sensitivity disorders and central scotomas indicate the importance of differential diagnosis in the assessment of visual disorders after brain injury (Kerkhoff et al., 1992a; Zihl, 1995a, 2000). Differential diagnosis is not only essential for accurately predicting functional impairments in daily life but also for efficient rehabilitation practice.

3. The eyes have it: Part III. Rehabilitating functional impairments in homonymous visual field disorders

Eye-movements are the tool not only to help better understand and more effectively assess the functional impairments in visual field disorders but also to rehabilitate them in a more efficient way, at least for reading and visual exploration. The first systematic attempt to treat the functional impairments of hemianopic patients dates back to Poppelreuter (1917/1990). The oculomotor training he devised to improve reading in patients with visual field loss is probably the first compensatory treatment method in the rehabilitation of visual field disorders. Compensatory treatment approaches aim at substituting the lost visual field region by eye-movements, i.e. at re-learning eye-movement control in visual field loss. Restorative treatment approaches, in contrast, aim at (partial) restitution of the lost visual field region, thus, at re-building the visual brain (Anderson, 2003; Bouwmeester, Heutink, & Lucas,

2007; Kerkhoff, 1999, 2000; Lane et al., 2008; Pambakian, Currie, & Kennard, 2005; Pambakian & Kennard, 1997; Pelak, Dubin, & Whitney, 2007; Schofield & Leff, 2009; Stoerig, 2008; Zihl, 2000, 2003).

After experiments in primates with visual field defects caused by striate cortex injury demonstrated that the visual field can, at least partly, be restored (Cowey & Weiskrantz, 1963; Mohler & Wurtz, 1977), attempts were made to induce visual field recovery in human patients. Systematic repetitive stimulation with light stimuli at the border of the visual field defect as well as systematic practice of detection, saccadic localisation and identification of targets (its luminance, form, colour, or motion) presented in the blind field have been found to reduce the size of visual field defects or improve the sensitivity to specific stimuli in the affected visual field; visual attention allocation towards the stimulated visual field region seems to be crucial in this regard (Zihl, 1981; Zihl & von Cramon, 1979, 1985). These seemingly promising results have been replicated in a number of investigations (Huxlin et al., 2009; Hyvärinen, Raninen, & Näsänen, 2002; Julkunen, Tenovuo, Jääskeläinen, & Hämäläinen, 2003; Julkunen et al., 2006; Kasten, Poggel, & Sabel, 2000; Kasten, Wüst, Behrens-Baumann, & Sabel, 1998a; Mueller, Mast, & Sabel, 2007; Sahraie et al., 2006; Schmielau & Wong, 2007).

Yet, despite single cases showing remarkable and stable visual field recovery including form and colour vision, treatment-induced visual field enlargement seldom exceeds 5° (Hyvärinen, Raninen, & Näsänen, 2002; Julkunen et al., 2003, 2006; Kasten, Poggel, & Sabel, 2000; Kasten et al., 1998a; Mueller, Mast, & Sabel, 2007; Schmielau & Wong, 2007; Zihl, 1981; Zihl & von Cramon, 1979, 1985). Although this increase may be sufficient to improve reading in some patients with visual field defects, it is too small to reduce the visual exploration impairment. Whereas normal reading performance requires only a visual field sparing of 3° to the left and 5° to the right of fixation (Mackensen, 1962; Trauzettel-Klosinski & Brendler, 1998; Zihl, 1995a), the critical left- and right-sided visual field requirement in visual exploration is at least 20° (Lovie-Kitchin, Mainstone, Riobinson,

& Brown, 1990). However, the improvements in reading performance that have been obtained in some patients were very small. In addition, they were not related to the treatment-induced visual field enlargement but to a change in patients' oculomotor text processing strategy (Reinhard et al., 2005; Zihl & von Cramon, 1985). Moreover, although patients report subjective improvements in daily life activities after treatment (Kasten et al., 1998a; Mueller, Poggel, Kenkel, Kasten, & Sabel, 2003; Sabel, Kenkel, & Kasten, 2004; Zihl & von Cramon, 1985), it remains to be investigated whether the treatment-induced visual field enlargement is also of behavioural significance and translates into improved functioning in vision-related tasks such as reading, visual exploration and line bisection.

Moreover, not all patients show visual field recovery after restorative visual field training. Treatment-induced visual field enlargement can be found only in patients with incomplete or partly reversible postchiasmatic lesions, a shallow gradient in the profile of light sensitivity and amblyopic transition zones in perimetry, or residual metabolism and/or fMRI activations in the affected striate cortex (Bosley et al., 1985; Kerkhoff, 1999, 2000; Poggel, Mueller, Kasten, & Sabel, 2008; Zihl & von Cramon, 1985). It was suggested that systematic stimulation of spared visual cortical neurons surviving brain injury at the border of the lesion ("transition zone" or "area of residual vision") may reactivate visual processing in this critical region, thereby restoring parts of the visual field (Kasten, Wüst, & Sabel, 1998b; Poggel, Kasten, Müller-Oehring, Sabel, & Brandt, 2001; Sabel, 1999; Zihl & von Cramon, 1985). Evidence from functional neuroimaging studies suggests that vision restoration in visual field loss is associated with altered neuronal activity in surviving neural networks in ipsi- and contralesional striate, peristriate and extrastriate visual areas (Henriksson, Raninen, & Nasanen, 2007; Julkunen et al., 2006; Nelles et al., 2007a; Nelles et al., 2002; Yoshida et al., 2006) and may also involve, at least initially, (pre-)frontal and temporal areas (Marshall et al., 2008). Yet, the exact neural basis of spontaneous and treatment-induced visual field recovery nevertheless remains to be determined. Since not all patients show residual neuronal functions and sharply demarcated visual field defects are

very common, restorative visual training is unlikely to be a treatment option for the majority of patients with visual field disorders (Pambakian & Kennard, 1997; Zihl, 2000). Moreover, it is still a matter of controversy whether the obtained visual field enlargements indicate true visual field recovery, or whether they are merely perimetric measurement artefacts and might also be explained by changes in attentional and oculomotor measures (Glisson, 2006; Horton, 2005a, 2005b; McFadzean, 2006; Plant, 2005; Reinhard et al., 2005; Schreiber et al., 2006).

Since the functional impairments in visual field disorders are not simply failures of vision, restorative treatment methods might not suffice to restore functioning in vision-related tasks such as reading, visual exploration and line bisection. Although the visual field defect is a major component of the functional impairments in visual field loss, it is not their primary cause. Consequently, treatment methods ought not aim at restoring the visual field but at re-organizing the control of visual information processing and eye-movements. Compensatory treatment methods may therefore be preferred, even more so since their therapeutic effects seem to be superior to those of restorative methods. In addition, they have been found to induce behaviourally significant improvements in reading and visual exploration in almost all patients with visual field loss. Moreover, they involve considerably fewer treatment sessions (10-25 sessions) than restorative approaches (30-500 sessions) (Kerkhoff, 1999; Zihl, 2000). Thus, compensatory treatment approaches seem to be more efficient and can be applied to all patients with visual field loss, which makes them the first choice for routine rehabilitation. For the same reasons, compensatory approaches also outweigh the use of optical aids in the rehabilitation of functional impairments in visual field disorders. Optical therapies aim at substituting the lost part of the visual field by using customised spectacles, fitted with either mirror or prism systems, that induce visual field expansions (Bowers, Keeney, & Peli, 2008; Gottlieb & Miesner, 2004; Peli, 2000). Despite increasing efforts to demonstrate the benefit of this treatment approach for patients with visual field loss (Bowers, Keeney, & Peli, 2008; Szlyk, Seiple, Stelmack, & McMahon,

2005), the efficacy of optical aids is still controversial. Moreover, optical aids are expensive and require time-consuming and effortful fitting and training procedures (Zihl, 2000). It remains to be determined whether compensatory treatment methods are superior to restorative treatment methods (and the use of optical aids) or whether it is a combination of treatments that most efficiently reduces the functional impairments in visual field loss.

Since most of our knowledge about the rehabilitation of visual field disorders is based on evidence from hemianopic patients, it is important to investigate the effects of the lower-level visual dysfunction (i.e. the visual field defect) and the higher-level impairment of the attentional top-down control of visual processing and eye-movements on the amount and outcome of compensatory (and restorative) treatment methods. Although most patients benefit from compensatory oculomotor treatment, preliminary evidence suggests that patients with bilateral visual field defects require at least twice as many training sessions as patients with unilateral field defects and show only little improvement. As it would be expected, the side and extent of unilateral visual field defects only seem to determine the treatment requirements for rehabilitating hemianopic dyslexia; patients with right-sided field defects and smaller visual field sparing require a larger amount of treatment and show a poorer outcome than those with left-sided field defects. Furthermore, patients with hemianopic visual field loss seem to require a larger amount of treatment than patients with quadranopias or paracentral scotomas. The only exceptions to this rule are patients with a central scotoma. Since these patients additionally show a severe impairment of visual acuity/spatial contrast sensitivity and ocular fixation difficulties, they require not only the largest amount of treatment and still show the poorest outcome but also need special rehabilitation measures. Moreover, patients with additional injury to the white matter or to occipito-parietal structures seem to require more compensatory practice sessions and show a poorer treatment outcome than patients who do not have these additional extrastriate injuries. The quality of the visual field loss also seems to play an important role in determining the amount of compensatory treatment since rehabilitating functional impairments in anopic

visual field loss involves more treatment than amblyopic field defects (Zihl, 2000). Although blindsight does not seem to reduce patients' functional impairments (Zihl, 1980), practising blindsight has been suggested as a potential rehabilitation strategy (Boyle et al., 2005; Kerkhoff, 1999, 2000). Preliminary evidence indicates that training blindsight may be associated with an enlargement of the visual field as well as with improvements in visual detection and letter identification (Chokron et al., 2008; Sahraie et al., 2006). Yet, the exact impact of blindsight on the amount and outcome of compensatory and restorative treatment as well as the usefulness of blindsight training in the rehabilitation of functional impairments in visual field loss remains to be determined.

Time since brain injury, age, and the presence or absence of awareness may also be important factors that co-determine the amount of treatment required and the overall rehabilitation outcome. Although chronic patients (>2 years since brain injury) or elderly patients seem to benefit from treatment to the same extent as more acute or younger patients (Kerkhoff, 2000; Kerkhoff, Münßinger, Haaf, Eberle-Strauss, & Stögerer, 1992b; Zihl, 2000), the relevance of time since injury and age as factors for treatment prognosis has yet to be established. The exact relationship between awareness of deficits and treatment outcome following acquired brain injury also remains to be investigated (Ownsworth & Clare, 2006). The presence of awareness, however, i.e. knowledge about the disorder and possible coping strategies, appears to be beneficial for the progress and outcome of treatment as well as the emotional well-being of patients, and several interventions have been developed to improve awareness in patients (Fleming & Ownsworth, 2006). For improving awareness in patients with visual field defects, it may be important to repeatedly inform the patient about the cause of visual field loss and to demonstrate the field deficit (e.g., by using visual field charts), its functional consequences as well as relative improvements during treatment (Kerkhoff, 2000). The presence of compensation strategies that involve head- instead of eye-movements seems to be another factor that is relevant for treatment prognosis since such strategies seem to interfere with the acquisition of an adaptive oculomotor strategy and to delay treatment

progress. Clinical evidence also suggests that concomitant (peripheral or central) visual disorders, oculomotor deficits, other cognitive disorders as well as the presence of depressive symptoms after acquired brain injury diminish treatment success in the rehabilitation of visual field disorders, particularly if the neural and cognitive prerequisites of perceptual and motor learning (or skill acquisition) are affected. Moreover, patients with multiple disorders require a larger amount of treatment for effectively improving their functional impairments (Zihl, 2000).

Thus, depending on the characteristics of the visual field defect, the locus and extent of brain injury, time since injury, the patient's age, awareness and the presence of maladaptive strategies and/or comorbidities, specific modifications of compensatory (and restorative) treatment methods, special rehabilitation measures and even a combination of treatments may be necessary to effectively reduce functional impairments and maximise the outcome for the patient. The rehabilitation of visual field disorders is often idiosyncratic and highly individualised and has to be tailored to the patient's unique combination of deficits and preserved functions as well as to the functional impairment that is to be rehabilitated. Studies attempting to identify "which type and amount of treatment works best for which visual field disorder and functional impairment under what conditions" would greatly improve the current practice of rehabilitating functional impairments in patients with visual field disorders.

In contrast to restorative treatment approaches using bottom-up stimulation of the visual field, compensatory treatment methods involve supervised, systematic practice of an intentional top-down-directed eye-movement strategy to compensate for the effects of visual field loss (Zihl, 2000, 2003). This thesis's findings suggest that reading and visual exploration impairments require specific treatments for their improvement and confirm current rehabilitation practice in visual field disorders. The study presented in Chapter 3 demonstrated that there is no transfer of practice-related improvements in performance and eye-movements between reading and visual exploration with simulated HH. Supporting

evidence stems from the study reported in Chapter 5, where the treatment effects of compensatory oculomotor reading training did not generalise to visual exploration and were found to be specific to reading. Thus, the reading and visual exploration impairments associated with visual field loss cannot be alleviated by practising any voluntary eye-movements with any visual material. Yet, it nevertheless requires cross-over rehabilitation studies to determine whether these functional impairments are best treated using specific methods.

The rehabilitation of hemianopic dyslexia normally involves the systematic practice of small, very precise, systematic and regular horizontal saccadic eye-movements with either moving or static text material (words). Evidence from skill acquisition in the rehabilitation of memory disorders (Beaunieux et al., 2006) indicates the superiority of massed systematic oculomotor training over using distributed oculomotor training sessions, which confirms the current practice of hemianopic dyslexia rehabilitation. The effectiveness of this compensatory oculomotor reading training to reduce the hemianopic reading impairment has been confirmed in a number of investigations (Kerkhoff et al., 1992a; Poppelreuter, 1917/1990; Zihl, 1995a, 2000; Zihl, Krischer, & Meißen, 1984) as well as in a placebo-controlled clinical evaluation (Spitzyna et al., 2007). The study presented in Chapter 5 was not only the first to show that the treatment effect is specific to reading but it also demonstrated that the treatment effect associated with this method does not critically depend on using text material (words). Using non-text material that preserves the main visual features of a word seems to be sufficient to improve reading in unilateral homonymous visual disorders.

It remains unclear, however, whether using gliding text material that moves against reading direction (optokinetic therapy) or the time-limited presentation of static text material is to be preferred in the rehabilitation of hemianopic dyslexia. It seems that systematic oculomotor practice with moving text material (Kerkhoff et al., 1992a; Zihl, 1995a, 2000; Zihl et al., 1984) is equally effective as using static text material in improving reading

performance (Zihl, 2000). Hence, the mode of presenting training material (moving vs. static text), and thus the related differences in treatment-induced eye-movements (bottom-up optokinetic nystagmus stimulation inducing 'involuntary' saccades vs. practicing an attentional top-down strategy for guiding reading eye-movements, i.e. 'voluntary' saccades) and underlying mechanisms, does not seem to have an impact on the magnitude of improvement in reading. Clinical evidence suggests, however, that the effects of using static text material may be superior to those obtained with moving text material since it accelerates the acquisition of a successful compensatory oculomotor strategy. Systematic practice with static text material reaches similar improvements with a considerably smaller number of treatment sessions (Zihl, 2000). Another unresolved issue in the rehabilitation of hemianopic dyslexia is whether saccade size in oculomotor reading training has an effect on the treatment outcome. It is still unknown on which size of saccadic eye-movements oculomotor training has to focus in order to reach the greatest improvements in reading performance that transfer equally effectively to all reading situations, ranging from laboratory- and paper-based reading tests to reading books, newspapers, net-papers, maps, and interacting with word- and number-processing software as well as web-pages. It is therefore also important to establish these transfer effects not only by using subjective reports and questionnaires but also by using behavioural measures.

Moreover, the much smaller and less stable improvements found in the latest study investigating the effect of systematic practice using moving text (Spitzyna et al., 2007) compared with previous and the present results are surprising. The authors explained this result by differences between studies in demographic and clinical variables, particularly time since lesion. Yet, chronic patients seem to benefit from treatment to the same extent as more acute patients (Kerkhoff, 2000; Kerkhoff et al., 1992b; Zihl, 2000). A more appropriate explanation for the critical difference between Spitzyna et al.'s study (2007) and all previous reports, including the present study (Chapter 5), is that Spitzyna et al.'s patients performed systematic oculomotor reading practise in their own home without supervision. The lack of a

supervised learning condition may account for the much smaller treatment effect obtained with such a home-based procedure, suggesting the significance of supervision and immediate feedback on reading performance, eye and head movements for the rehabilitation of hemianopic dyslexia. Without appropriate instruction, supervision and immediate feedback preventing errors that occur in systematic practice procedures (i.e. errorless learning in contrast to trial-and-error learning) (Clare & Jones, 2008; Mount et al., 2007), patients may not only benefit less from the same amount of oculomotor practice but may even develop maladaptive strategies which prevent the acquisition of an adaptive strategy, delay treatment progress, increase functional visual impairment and impede subsequent rehabilitation efforts. However, compensatory treatment programmes that can be administered by patients themselves in their own homes have become increasingly popular and are now being made available freely online (e.g. Spitzyna et al., 2007). Although this development seems appealing, particularly from a patient's and health economic viewpoint, it should be treated with caution until the efficacy of home-based treatment programmes is evaluated in comparison with programmes that are administered in a supervised, errorless learning condition.

Investigating the role of supervision and errorless learning in the rehabilitation of functional impairments in visual field loss is not only of importance for the treatment of hemianopic dyslexia but also for the rehabilitation of the visual exploration impairment where a similar development can be observed (e.g. Pambakian, Mannan, Hodgson, & Kennard, 2004). In contrast to improving reading in visual field loss, improving visual exploration requires the systematic practice of large saccadic eye-movements, which helps enlarging the useful field of gaze. Moreover, it requires practicing more systematic and spatially-organised oculomotor scanning strategies using visual search tasks. This compensatory treatment procedure has been found to effectively reduce the hemianopic visual exploration impairment (Kerkhoff et al., 1992b; Kerkhoff, Münßinger, & Meier, 1994; Kooijman et al., 2004; Nelles et al., 2001; Pambakian et al., 2004; Zihl, 1988, 1995b, 2000).

However, since many different visual stimulus arrays and visual search displays of various sizes have been used in different compensatory oculomotor visual exploration training programmes, the optimal training regime and display, and thus the optimal saccade size in oculomotor training, remains to be determined. Moreover, although treatment-related oculomotor adaptation to visual field loss seems to transfer from processing abstract stimulus displays during training to natural scene viewing and patients report improvements in orienting, navigating and searching for objects or persons (Zihl, 2000), it is important to establish this transfer of functional training benefits to activities of daily living using behavioural measures from more naturalistic tasks. Commonly, the functional benefits of training are established with subjective reports and activities of daily living questionnaires, which is however unreliable. Investigating whether laboratory-based, pure visual exploration performance is a good predictor of visuo-motor performance in daily life activities is of great importance in this regard. Nevertheless, it has been demonstrated that the treatment effect is specific to visual exploration and does not transfer to text reading (Spitzyna et al., 2007). Although recent evaluations of a compensatory visual exploration training based on systematic audio-visual stimulation showed treatment-related improvements in single-word reading accuracy (Bolognini, Rasi, Coccia, & Làdavas, 2005), text reading and associated eye-movements (Passamonti, Bertini, & Làdavas, 2009), these improvements were very small in comparison to those found after compensatory oculomotor reading training (Zihl, 2000). However, the treatment-related improvements that have been demonstrated for visual exploration performance and eye-movements suggest that systematic audio-visual stimulation of the blind hemifield may be a promising treatment method for the visual exploration impairment associated with visual field loss (Bolognini et al., 2005; Passamonti et al., 2009). Thus, as in the rehabilitation of hemianopic dyslexia, both compensatory bottom-up (i.e. systematic audio-visual stimulation) and top-down approaches (i.e. systematic practice of an attentional top-down strategy for guiding visual exploration eye-movements) may be useful in the treatment of the visual exploration impairment in visual field loss. However, again it remains to be investigated which approach is to be preferred.

Although visual field enlargement has occasionally been observed after compensatory oculomotor treatment in reading and visual exploration (Kerkhoff et al., 1992a; Kerkhoff et al., 1992b; Kerkhoff et al., 1994) and is considered to be the basis of the therapeutic effect of restorative rehabilitative methods (Zihl & von Cramon, 1985), it cannot account for the treatment-related improvements in reading and visual exploration performance and eye-movements associated with compensatory methods. These improvements are based on treatment-related oculomotor adaptation to visual field loss in reading and visual exploration. It is possibly best understood as functional reorganisation of eye-movement control. The bottom-up control of visual information processing and eye-movements, which is normally based on parafoveal and peripheral vision, is substituted by an attentional top-down control (Zihl, 1995a, 1995b, 2000). Changes of activation in the fronto-parietal network underlying the cortical control of saccades may represent the neural correlate of oculomotor adaptation to visual field loss (Nelles et al., 2007b). That patients with additional extensive injuries to the occipital white matter and/or to occipitoparietal regions require the largest amount of compensatory treatment supports the relevance of parietal mechanisms and indicates the significance of functional connections between cortical visual areas and the areas supposedly mediating the treatment effect (Zihl, 2000). However, the exact neural basis of spontaneous and treatment-induced oculomotor adaptation to visual field loss is still unclear and requires further investigation. Moreover, it remains to be determined whether there are differences, both at the neural and behavioural level, between spontaneous and treatment-induced oculomotor adaptation to visual field loss. Behavioural and functional neuroimaging studies of reading and visual exploration performance and eye-movements before and after uninstructed as well as instructed specific oculomotor practice both in real and simulated visual field loss may be illuminating in this regard. When considering the potential benefits of combining behavioural interventions with non-invasive brain stimulation for improving the rehabilitation of patients after brain injury (Fregni & Pascual-Leone, 2007; Hummel & Cohen, 2005, 2006), investigating the neural mechanisms that underlie spontaneous and treatment-induced oculomotor adaptation (and visual field recovery) seems even more

important. Yet, such investigations are not only of clinical relevance since they also improve our knowledge about the functional (re-)organisation of the brain and its plasticity.

Spontaneous and treatment-induced visual field recovery and oculomotor adaptation to visual field loss indicate a remarkable neural and functional plasticity in the visual and oculomotor system. Plasticity in cortical/subcortical areas involved in visual processing and eye-movement control, and thus patients' rehabilitation potential, may not be as limited as previously assumed (Duffau, 2006; Hopp & Fuchs, 2004; Hummel & Cohen, 2005; Huxlin, 2008; Leigh & Kennard, 2004; Sabel, 2008; Safran & Landis, 1996; Stoerig, 2008; Ward, 2005). Yet, specificity rather than generality in transfer of the improvements associated with compensatory oculomotor treatment suggests task-specific limitations of neural and functional plasticity in visual, attentional and oculomotor processes (Ilg et al., 2008; Jones et al., 2006). It may also indicate task-specificity in the functional specialisation of the (cortical) oculomotor system (Alahyane et al., 2007), which is in line with evidence indicating that the information the oculomotor and visual processing systems require is highly task-specific (Land & Furneaux, 1997). The limited effects of restorative treatment methods compared with compensatory methods in reducing the functional impairments in visual field loss suggest that the neural and functional plasticity for restitutional changes in early visual areas is much lower than for cortical reorganisation in regions involved in oculomotor control during reading and visual exploration (Huxlin, 2008).

Although restorative visual field training may therefore not be the treatment of choice for rehabilitating the functional impairments in adults, it may be an effective treatment for children with visual field disorders after brain injury. Restorative visual field training was found to induce a mean visual field increase of 65° in children aged between 1 and 15 years suffering from visual field loss. However, since conventional perimetry could not be performed, changes in target-directed eye-movements were used to estimate the extent of treatment-induced visual field recovery (Werth & Moehrenschlager, 1999; Werth & Seelos, 2005). Although it therefore remains unclear whether the reported visual field increase

reflects restitution of the visual field or oculomotor adaptation to visual field loss, restorative visual field training seems to be promising for the rehabilitation of visual field disorders in children. It may be particularly useful for children since it does not require practicing an intentional top-down strategy but uses bottom-up stimulation of the visual field (Zihl, 2000, 2003). However, no report has dealt with compensatory treatment methods in the rehabilitation of children with visual field disorders after brain injury. Thus, it remains to be determined whether restorative or compensatory visual rehabilitation is to be preferred. Conducting research in assessment and rehabilitation of cerebral visual field disorders in children is of particular importance. Visual field disorders often remain undiagnosed in the paediatric population (Kedar, Zhang, Lynn, Newman, & Biousse, 2006) and have far-reaching consequences for children's development in the domains of language, (visuo-)motor functions, attention, and memory as well as for their social behaviour development. Yet, the exact effects of visual field loss on these domains and their development remain to be established. Moreover, bilateral visual field disorders, which are much more disabling than their unilateral counterparts, are the rule rather than the exception in early brain injury (Zihl & Priglinger, 2002).

That children seem to benefit more from restorative visual field training than adults suggests that the neural and functional plasticity and thus the potential for spontaneous and treatment-induced visual field recovery and oculomotor adaptation to visual field loss may be much greater after brain injury sustained in early life. This assumption is consistent with the common view that increasing age is associated with a decrease in neural, functional and cognitive plasticity (Burke & Barnes, 2006; Craik, 2006; Hedden & Gabrieli, 2004; Payne & Lomber, 2002; Sowell et al., 2003) and a deterioration of myelin affecting white matter pathways (Wozniak & Lim, 2006). Whether the potential for spontaneous and treatment-induced visual field recovery and oculomotor adaptation to visual field loss changes corresponding to this alleged age-related decrease in neural, functional and cognitive plasticity remains unknown and individual differences may be substantial (Celesia, 2005).

Despite reports of remarkable spontaneous visual field recovery (Bova et al., 2008; Celesia, 2005; Werth, 2006, 2007) and efficient spontaneous oculomotor adaptation in children (Zihl & Priglinger, 2002), injury to the developing brain can disturb normal developmental plasticity and may therefore not necessarily be associated with a better recovery or treatment outcome than injury to the mature and ageing brain (Giza & Prins, 2006). Hence, "young is not always better" (Giza & Prins, 2006, p. 364) when considering patients' potential for spontaneous and treatment-induced visual field recovery and oculomotor adaptation to visual field loss. The effectiveness of restitutive and compensatory oculomotor treatment methods to reduce the functional impairments in middle- and old-aged patients with visual field loss (Zihl, 2000) adds to the growing evidence for a life-long potential for functional reorganisation and plasticity (Craik, 2006; Jones et al., 2006; Reuter-Lorenz, 2002).

Yet, the role of visual, attentional, and oculomotor routines, which have already been established in the mature and ageing brain but not yet in the immature and developing brain, may also be important to consider in spontaneous and treatment-induced oculomotor adaptation to visual field loss. For exploring, orienting and navigating with a visual field defect, middle- and old-aged patients, in contrast to children, may be able to use already established visual memory representations of their visual environments. This assumption may also explain why these patients report to be more impaired in unfamiliar than in familiar environments (Zihl, 2000). Likewise, visual field loss in children is likely to have greater impact on reading since children have yet to learn the visual, linguistic and oculomotor skills involved in reading that have already been acquired by skilled readers.

However, no report has dealt with the effects of visual field loss on reading and visual exploration in children thus far. The impact of visual field loss on the development of these visuo-motor abilities also remains unknown. Although only few studies examined reading performance and eye-movements in young children (Kwon, Legge, & Dubbels, 2007; McConkie et al., 1991; Rayner, 1986) and older readers (Kliegl, Grabner, Rolfs, & Engbert, 2004), these studies nevertheless suggest that reading performance and eye-movements

change across the lifespan (Laubrock, Kliegl, & Engbert, 2006). Age-related performance changes have also been demonstrated in visual exploration (Coeckelbergh, Cornelissen, Brouwer, & Kooijman, 2004; Hommel, Li, & Li, 2004) and line bisection (Beste, Hamm, & Hausmann, 2006; Hausmann, Waldie, & Corballis, 2003b). However, it remains unclear whether these developmental changes are accompanied by changes in eye-movement patterns. Moreover, it is still unknown whether the developmental trajectories differ between reading, visual exploration and line bisection. Thus, in addition to determining whether and to what extent age influences spontaneous oculomotor adaptation in visual field loss, the resulting functional impairments, the outcome and required amount of treatment, it is essential to further investigate the development of reading, visual exploration and line bisection as well as the impact of visual field loss. Using eye-movements as research tool in this regard will not only improve our understanding of the functional impairments in visual field loss as well as current assessment and rehabilitation practice but also provide insights into neural and functional plasticity across the lifespan.

It remains to be investigated whether compensatory treatment approaches are also suitable for the rehabilitation of the line bisection impairment associated with visual field disorders. Although investigating line bisection in simulated and real HH demonstrated that the visual field defect and oculomotor adaptation to it and the contralateral bisection error can dissociate, it remains to be determined whether treatment-induced oculomotor adaptation can alleviate the line bisection impairment in patients with visual field loss. Efficient oculomotor adaptation to visual field loss may help patients overcome their visual-spatial deficit, i.e. their contralateral shift of the visual midline or subjective straight-ahead direction in visual-spatial judgements and in spatial orientation. During walking, for instance, patients may monitor more carefully their walking direction and orientation in space. Continuously re-adjusting their straight-ahead direction may be mediated by executive functions until routines are established. The similarities of the visual sampling strategies in line bisection (or adjusting straight-ahead direction) and visual exploration suggest that oculomotor visual

exploration training may be a more appropriate treatment option for the line bisection impairment than oculomotor reading training. Yet, since the effects of systematic oculomotor reading and visual exploration training on the line bisection impairment are unknown, investigating line bisection and associated eye-movements before and after treatment is required.

However, task-specificity of spontaneous and treatment-related oculomotor adaptation in visual field loss indicates that line bisection may require specific treatment for its improvement. Supporting evidence stems from a cross-over rehabilitation study which demonstrated the necessity of specific and differential treatments for the rehabilitation of the visual exploration impairment and the visual-spatial deficits associated with visual neglect (Kerkhoff, 1998). Yet, although repetitive training with contingent verbal or visual feedback has been found to be effective in reducing visual-spatial deficits in visual neglect (see also Kerkhoff, 2000), it remains to be investigated whether systematic and repetitive feedback-based practice of line bisection is an appropriate treatment method for the line bisection impairment associated with visual field loss. Since visual-spatial deficits are also likely to interact with visual deficits and increase resulting functional impairments, it is even more important to study potential treatment methods for the rehabilitation of the line bisection impairment associated with visual field loss.

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