

## Anomalous global effects induced by ‘blind’ distractors in visual hemifield defects

S. Van der Stigchel<sup>a,\*</sup>, T.C.W. Nijboer<sup>a</sup>, D.P. Bergsma<sup>b</sup>, M. Abegg<sup>c</sup>, J.J.S. Barton<sup>c</sup>

<sup>a</sup> Department of Experimental Psychology, Helmholtz Institute, Utrecht University, Utrecht, The Netherlands

<sup>b</sup> Department of Functional Neurobiology, Helmholtz Institute, Utrecht University, Utrecht, The Netherlands

<sup>c</sup> Departments of Medicine (Neurology), Ophthalmology and Visual Sciences, University of British Columbia, Vancouver, BC, Canada

### ARTICLE INFO

#### Article history:

Accepted 21 June 2010

Available online 15 July 2010

#### Keywords:

Cerebral blindness  
Superior colliculus  
Visual system  
Eye movement

### ABSTRACT

Previous research has revealed that a stimulus presented in the blind visual field of participants with visual hemifield defects can evoke oculomotor competition, in the absence of awareness. Here we studied three cases to determine whether a distractor in a blind hemifield would be capable of inducing a *global effect*, a shift of saccade endpoint when target and distractor are close to each other, in participants with lesions of the optic radiations or striate cortex. We found that blind field distractors significantly shifted saccadic endpoints in two of three participants with lesions of either the striate cortex or distal optic radiations. The direction of the effect was paradoxical, however, in that saccadic endpoints shifted *away* from blind field distractors, whereas endpoints shifted *towards* distractors in the visible hemifields, which is the normal global effect. These results provide further evidence that elements presented in the blind visual field can generate modulatory interactions in the oculomotor system, which may differ from interactions in normal vision.

© 2010 Elsevier Inc. All rights reserved.

### 1. Introduction

The retinogeniculostriate pathway is the dominant route for mammalian visual processing, projecting from the retina to the lateral geniculate nucleus and then to the primary visual cortex (Cowey, 2004). In humans, a lesion to this pathway results in a loss of awareness of visual stimuli, which is confined to the contralateral hemifield if the lesion occurs after the optic chiasm. Despite this loss of visual awareness, numerous studies have reported that processing of visual information can still be demonstrated within the blind hemifield (Barbur, Ruddock, & Waterfield, 1980; Perenin, 1991; Pöppel, Held, & Frost, 1973; Sanders, Warrington, Marshall, & Weiskrantz, 1974; Van der Stigchel, van Zoest, Theeuwes, & Barton, 2008), a phenomenon called ‘blindsight’ (Weiskrantz, Warrington, Sanders, & Marshall, 1974). The anatomic and physiological correlates of blindsight remain areas of active investigation (Rees, 2008). Whereas different types of residual visual function may depend upon different processes (Danckert & Rossetti, 2005), a common element in several hypothesized pathways for blindsight is the involvement of the superior colliculus (Leh, Johansen-Berg, & Ptilito, 2006; Sahaie et al., 1997), a midbrain structure that is usually spared by lesions affecting either the optic radiations or striate cortex.

Since the superior colliculus plays a significant role in the generation of saccades, metrics of saccadic eye movements may be a

particularly sensitive measure of blindsight. Indeed, such logic guided one of the original studies of blindsight, which investigated the accuracy of saccades to the location of stimuli within a blind hemifield (Pöppel et al., 1973). Besides the localization of targets in the blind hemifield, which carries the awkward requirement of forcing participants to make a saccade to something they deny seeing, other types of collicular-mediated saccadic effects could be studied. For example, it is known that the modification of saccade metrics by experimental manipulations such as the presentation of a distractor reflect competition in the superior colliculus (Aizawa & Wurtz, 1998; McPeck, Han, & Keller, 2003).

Recently we tested the hypothesis that, in the absence of retinogeniculostriate processing, residual processing may still be detected as modifications of saccadic trajectories to seen targets by irrelevant distractors in the blind hemifield (Van der Stigchel et al., 2008). In healthy participants distractors at specific locations cause saccadic trajectories to deviate away from the distractor location (Van der Stigchel, Meeter, & Theeuwes, 2006). We therefore asked five participants with hemifield defects to make a saccade to a seen target while a distractor was presented simultaneously in either the blind or the intact field (Van der Stigchel et al., 2008). This is an ‘indirect’ strategy to study blindsight in that, rather than asking participants to respond to an unseen target, it measures the effect of unseen stimuli on responses to visible targets (Danziger, Fendrich, & Rafal, 1997; Intriligator, Xie, & Barton, 2002; Marcel, 1998; Rafal, Smith, Cohen, & Brennan, 1990; Walker, Mannan, Maurer, Pambakian, & Kennard, 2000). This strategy may be less artificial and less subjective than certain forced-choice methods (Barbur et al., 1980; Perenin, 1991; Sanders et al., 1974). All five of our previous participants with hemi-

\* Corresponding author. Address: Experimental Psychology, Helmholtz Institute, Heidelberglaan 2, 3584 CS Utrecht, The Netherlands. Fax: +31 30 253 4511.

E-mail address: [S.VanderStigchel@uu.nl](mailto:S.VanderStigchel@uu.nl) (S. Van der Stigchel).

field defects showed the expected trajectory deviation with distractors in their intact hemifield (Van der Stigchel et al., 2008). The key finding of interest was that two of the five also showed a significant deviation with distractors in their blind field. This was not seen in one control participant with a lesion of the optic chiasm, which would affect visual afferent fibers to the colliculus as well as to striate cortex. These results indicate that, in at least some participants with hemifield defects, competition between blind distractors and seen targets can occur, possibly at the level of the superior colliculus.

The aim of the current study was to further investigate oculomotor competition between elements in blind and intact regions of the visual field. This was done by examining a well-established phenomenon in healthy participants known as the *global effect* (Walker, Deubel, Schneider, & Findlay, 1997); when a target and a distractor are close to each other, within 20° or 30° of angular distance, the endpoint of the saccade to the target is shifted towards the distractor (Coren & Hoenig, 1972; Van der Stigchel & Theeuwes, 2005). Note that the global effect is different from the saccade trajectory deviations away from a distractor as described above, which is observed when the distractor is presented outside the zone for which the global effect is observed, causing an effect on the deviation of the path of the saccade, rather than a shift in saccade endpoint. The hypothesis that the global effect reflects tectal competition is supported by reports that activity in the superior colliculus is highest at a location between the target and the distractor on trials when the global effect occurs (Glimcher & Sparks, 1993; Van Opstal & Van Gisbergen, 1990).

Little is known about the global effect in participants with hemifield defects. In our initial study, we also tested two cases with the target and distractor closely aligned (Van der Stigchel et al., 2008). The target was always presented in the intact field, whereas the distractor was either in the intact or blind field. We found evidence for a global effect induced by a blind field distractor in both cases. The only other report, in the well-known blindsight patient G.Y. (Barbur, Forsyth, & Findlay, 1988), found a global effect when both target and distractor were in the 'blind' field, although it has to be noted that it was said that G.Y. was 'aware' of these targets.

Here we studied three new cases to determine whether a distractor in the blind hemifield induces a global effect when target and distractor are closely aligned. Participants were required to make a saccade to a single visible target. On two thirds of the trials, a distractor appeared simultaneously with the target. The distractor was presented in either the blind or the intact field. A healthy control group was run to ensure that the locations used in the experiment induced a significant global effect in the absence of visual field defects. The participants with hemifield defects served as their own control, because we compared the shift of saccadic endpoint induced by blind distractors to the endpoint shift induced by seen distractors. We hypothesized that, despite the lack of awareness of blind distractors, these stimuli would still be capable of inducing a global effect in participants with lesions of the optic radiations or striate cortex.

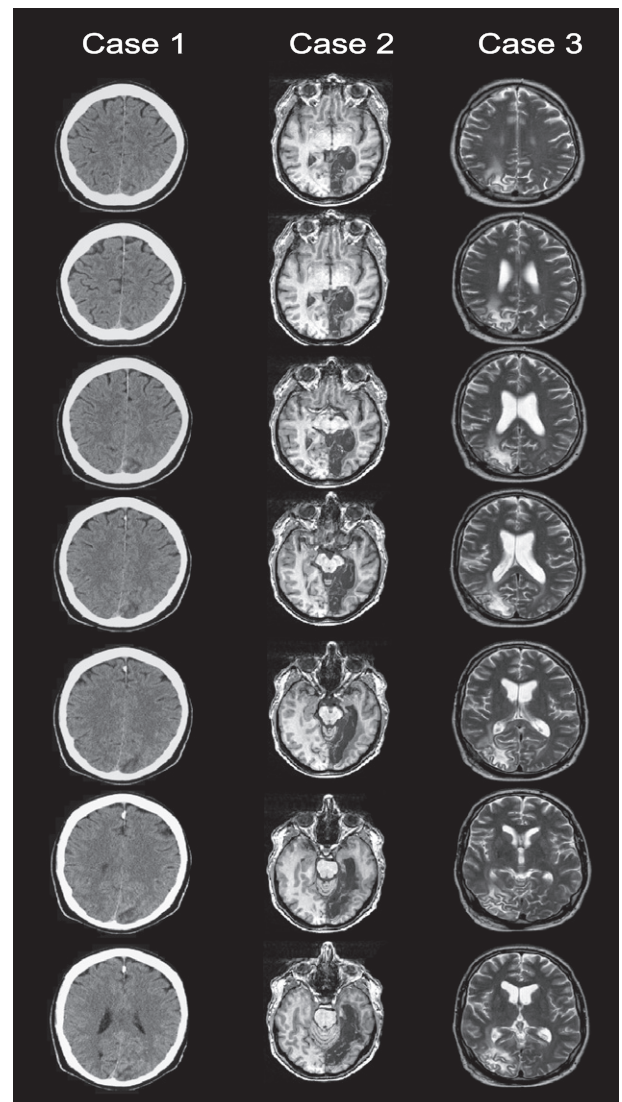
## 2. Methods

### 2.1. Participants

#### 2.1.1. Participants with hemifield defects

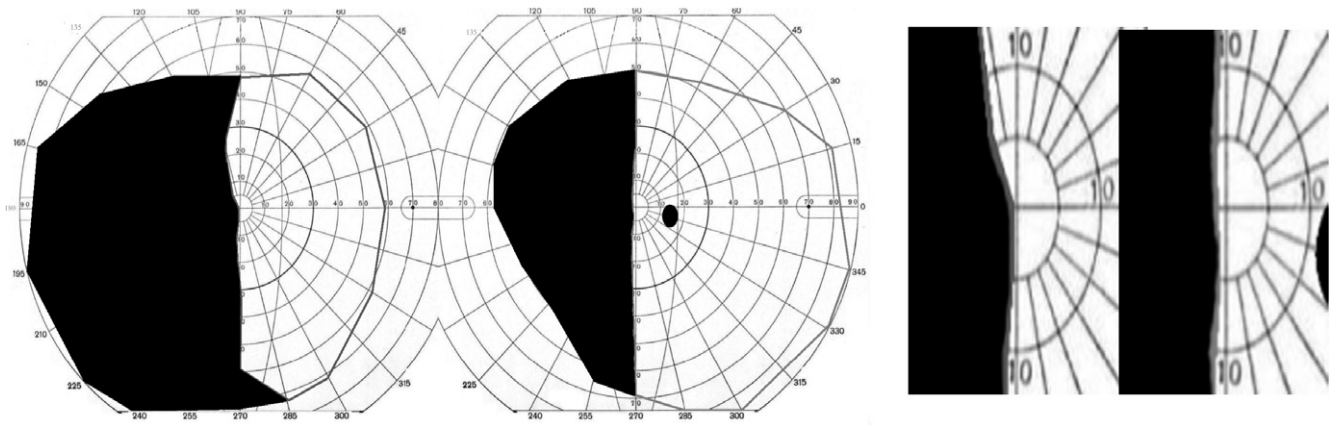
In total, three cases with homonymous hemifield defects from lesions of the optic radiations or striate cortex due to strokes or intracerebral hemorrhages were included. All had normal or corrected-to-normal visual acuity. These participants had lesions that affect the retinogeniculostriate pathway but not the retino-

tectal one. Fig. 1 shows MRI or CT images of the lesions of the participants. All cases had a complete neurologic and neuro-ophthalmologic examination. Visual fields were documented with perimetry (Fig. 2), and the brightest stimulus, the V4e target, was used to verify blindness in the retinotopic regions to be studied. Case 1 was a 53-year old male who had a small partial infarct of the posterior occipital lobe 38 months before testing. Case 2 was a 56-year old male who had a more extensive infarct in the territory of the posterior cerebral artery 62 months before testing, affecting medial occipital and temporal structures, including the lingual gyrus and parahippocampal cortex. Case 3 was a 56-year old male who had suffered an occipital hemorrhage 14 months before testing, leaving him with residual medial occipital damage to grey and white matter. At presentation and also at the time of testing, there were no signs of neglect or extinction in any of the three cases. All participants gave informed consent according to the standards of the Declaration of Helsinki for a protocol that was approved by the institutional review boards of the hospital and the university.

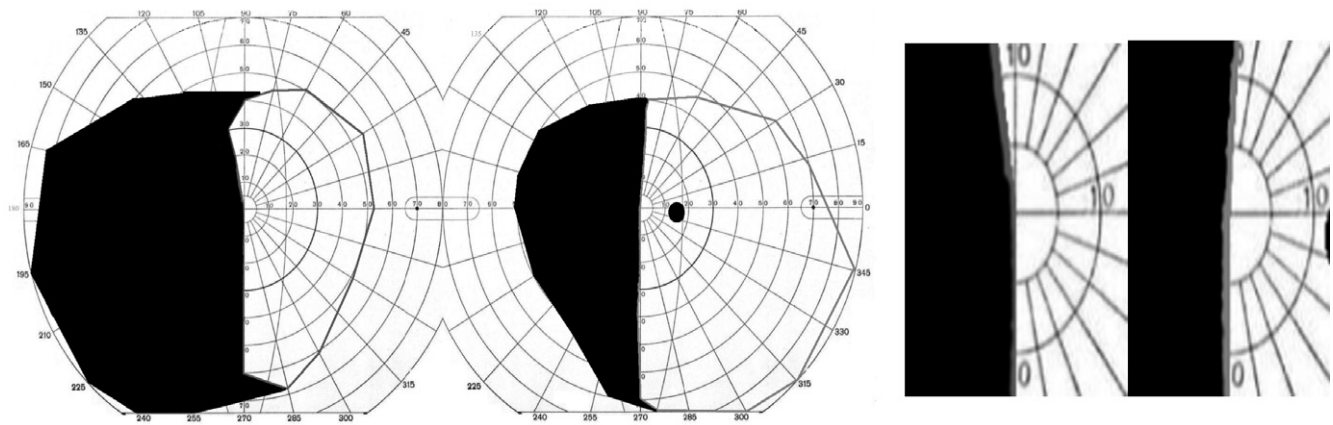


**Fig. 1.** Axial images of the lesions of the participants, with CT for Case 1, and T1-weighted MRI for Case 2, and T2-weighted MRI for Case 3. The left side of each slice represents the left side of the brain, and the images proceed from ventral slices at top to dorsal slices at bottom.

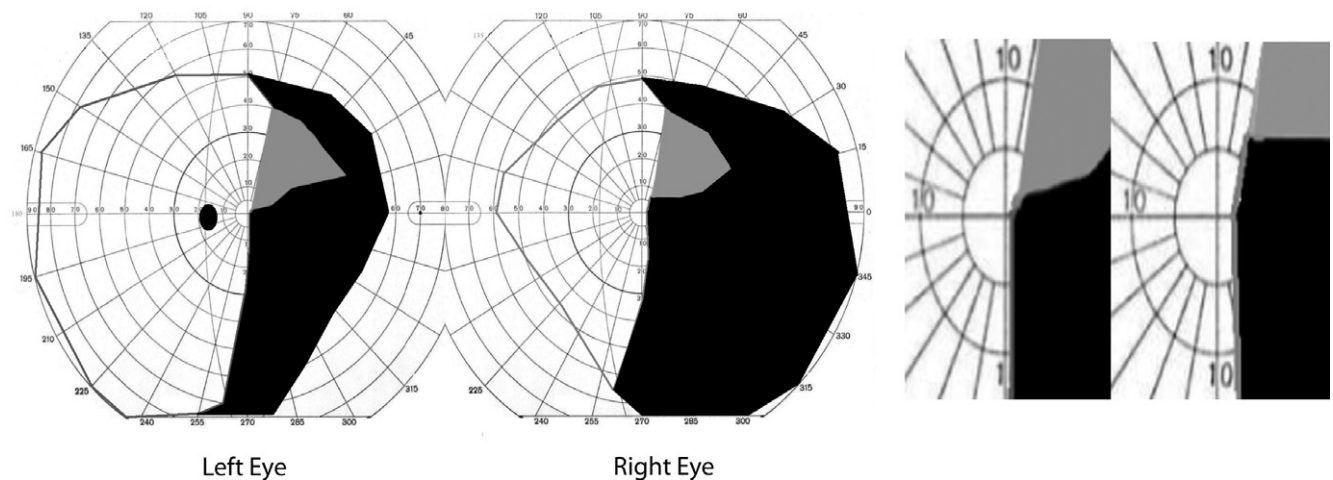
## Case 1



## Case 2



## Case 3



Left Eye

Right Eye

**Fig. 2.** Goldmann perimetry of visual fields of the three participants. Black regions indicate the scotoma as revealed by the V4e target. Gray regions indicate the areas in which a relative defect is present, indicating that the participant reported awareness of some of targets presented in this area during perimetry. On the right side, the central zone of 10° has been magnified for each participant.

### 2.1.2. Control group

Six observers (three males, three females; mean age = 22.2 - years; st. dev. = 1.5) participated in the experiment. All had normal or corrected-to-normal visual acuity.

### 2.2. Apparatus

Eye movements were registered by means of an infrared video-based eye tracker (SR Research Ltd., Canada). The EyeLink1000 sys-

tem has a 1000 Hz temporal resolution and an estimated spatial resolution of  $0.5^\circ$ . The left eye was recorded and analyzed. The participant's head was stabilized using a chin rest. The distance between monitor and chin rest was 65 cm.

### 2.3. Stimuli, procedure, and design

#### 2.3.1. Visual field test

A visual field test was performed to confirm our deductions from perimetry that subjects were not aware of stimuli of the size and luminance used in the distractor experiment, in the contralateral regions that were tested, and under the binocular conditions used. Hence the visual field test was done with both eyes open. A diamond of the same luminance and size as the distractor used in the experiment appeared at one of 16 possible locations, four in each quadrant (Fig. 3). In each quadrant, three possible locations were positioned  $6.26^\circ$  away in the vertical direction from fixation and either  $3.13^\circ$ ,  $2^\circ$  or  $1^\circ$  away in the horizontal direction. The first location (i.e.  $3.13^\circ$  away horizontally) was the location used in the distractor experiment. The other two locations (i.e.  $2^\circ$  and  $1^\circ$  away horizontally) were tested to investigate the border of the visual field defect with respect to the vertical meridian. The fourth location was positioned  $6.26^\circ$  away in the horizontal direction from fixation and  $4.69^\circ$  away in the vertical direction. This location was used in a previous study (Van der Stigchel et al., 2008) and was included to allow a comparison between the cases tested in the two studies. Cases 1 and 2 were given 96 trials, 48 of which contained no stimulus, and 48 of which contained the diamond stimulus. Case 3 was given 192 trials, 48 of which contained no stimulus, and 144 of which contained the diamond stimulus. The

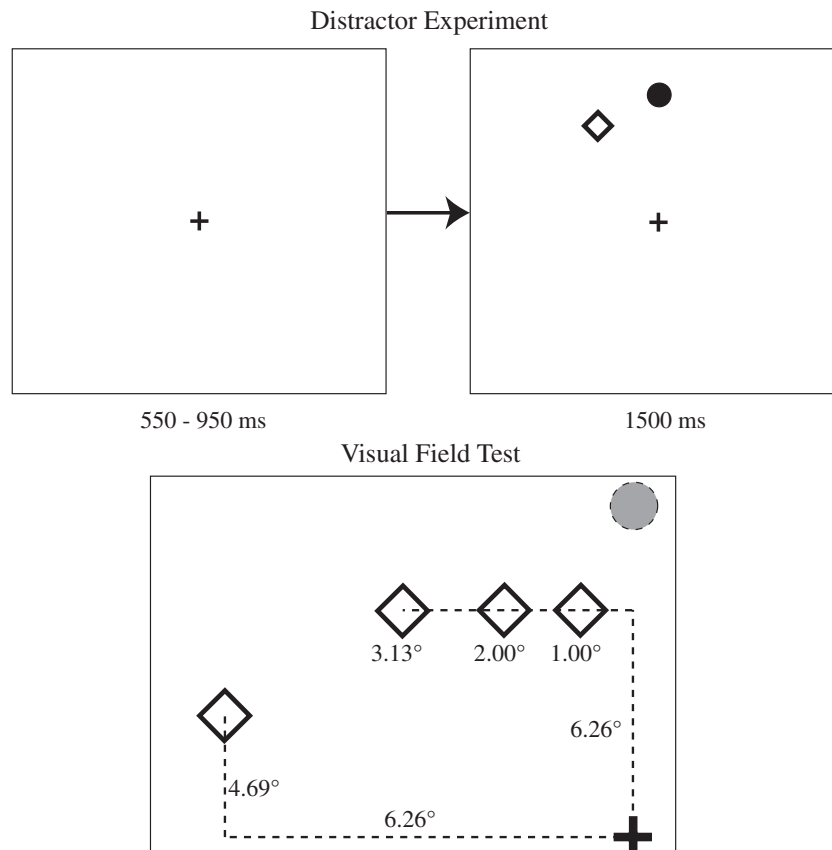
differences in the number of trials between the different hemianopic participants reflected variations in the amount of time that each participant was able to contribute.

At the end of each trial, participants had to indicate whether a diamond was present or not, pressing two different keys on a keyboard in response. Eye movements were recorded to monitor that the participants maintained steady fixation during the test.

#### 2.3.2. Distractor experiment

Each trial started with a central fixation cross ( $0.94^\circ \times 0.94^\circ$ ) (Fig. 3). After a variable period of 550–950 ms, the fixation cross disappeared and the target appeared (a disc with diameter of  $0.94^\circ$ ) located at an eccentricity of  $8.1^\circ$  on the vertical meridian, either above or below the location of the fixation cross. In a third of trials, the target was the only stimulus presented. In the remaining two-thirds, a diamond-shaped distractor (sides measuring  $1.09^\circ \times 1.09^\circ$ ) appeared at the same time as the target. The distractor was always located in the same upper or lower visual field as the target, but half the time it was in the horizontal hemifield ipsilateral to the lesion, and half the time in the contralateral horizontal hemifield. The distractor was located  $3.13^\circ$  away from fixation in the horizontal direction and  $6.26^\circ$  away in the vertical direction, or, in polar coordinates, at an eccentricity of  $7^\circ$  and angle of  $26^\circ$  away from the vertical axis. Both target and distractor were presented for 1500 ms.

To reduce possible effects of light scatter (i.e. a stimulus projected to the blind field is associated with light diffusing into the seeing field, Cowey, 2004), the experiment and visual field test were conducted in bright room lighting. Furthermore, all stimuli (fixation cross, target, and distractor) were black (luminance of



**Fig. 3.** Top of figure: Sequences of frames on a given trial in the present experiment. After 550–950 ms, the target appeared either above or below the central fixation point. In two-thirds of the trials, a diamond-shaped distractor appeared at the same time as the target. Bottom of figure: The four locations used in the visual field test. During the experiment, the distractor was presented  $3.13^\circ$  from fixation in the horizontal direction and  $6.26^\circ$  from fixation in the vertical direction.

0.58 cd/m<sup>2</sup>) on a gray background (luminance of 33.4 cd/m<sup>2</sup>). Both the visual field test and the distractor experiment were tested with participants having both eyes open.

Each session started with a nine-point grid calibration procedure. When the calibration point was positioned in the blind field, the participant was instructed verbally where the point was located. During calibration, we waited until the eyes of the participant no longer moved before performing the calibration for a specific point. The calibration was only accepted when the worst error point in the calibration was less than 1.5° and the average error is less than 1.0°. In addition, simultaneously fixating the fixation cross and pressing the space bar recalibrated the system by zeroing the offset of the measuring device at the start of each trial. When the fixation drift was larger than 2.0° (compared to the calibration), the drift correction failed and a complete new calibration was performed.

After calibration, the visual field test was performed before the start of the experiment in the participants with hemifield defects. The experiment consisted of a single training session of 30 trials and an experimental session of 300 trials. In order to obtain saccades that were not initiated before target onset, participants heard a short warning tone when saccade latency was longer than 800 ms or shorter than 80 ms. The sequence of trials was randomized for each participant, in terms of both target location (up or down) and distractor condition (none, right or left). Participants were instructed to look at the fixation cross until the target appeared, when they were to move their eyes to the target. It was stressed that they should make a single speeded saccade.

## 2.4. Data analysis

### 2.4.1. Distractor experiment

Saccade latency was defined as the interval between target onset and the initiation of a saccadic eye movement. An eye movement was considered a saccade either when the movement velocity exceeded 35°/s or when the movement acceleration exceeded 9500°/s<sup>2</sup>. If saccade latency was shorter than 80 ms, longer than 800 ms, or 2.5 standard deviations from the participant's mean latency, the trial was excluded. Trials were also excluded if there was no saccade or if the first saccade was too small (<3°). If the endpoint of the first saccade had an angular deviation of more than 22.5° from the vertical meridian (i.e. not in the direction of the target), the saccade was classified as an error and not analyzed. Furthermore, the initial saccade starting position had to be within 1.5° from the center fixation point for the horizontal direction. Altogether, these exclusion criteria led to a loss of 13.2% of trials for the control group. For the participants with visual field defects, 7.3% of trials were excluded in Case 1, 23.3% in Case 2 and 9.0% for Case 3. The majority of these trials were excluded because of poor fixations (i.e. too early saccades or a drift away from the fixation point). A too small first saccade (<3°) was observed in less than 1% of trials for Cases 1 and 3 and in 6.7% of trials for Case 2. A first saccade which endpoint had an angular deviation of more than 22.5° from the vertical meridian was observed in less than 1% of trials for all three cases.

In the remaining valid trials we measured endpoint deviation, defined in polar coordinates as the angular shift of the endpoint relative to the angle of the vector between the saccade starting position and the target location. For each distractor-present trial, we calculated endpoint deviation relative to the mean endpoint in the condition without a distractor, with a positive value indicating deviation towards the distractor, and a negative value deviation away from the distractor. By relating the distractor-present trials to the no-distractor trials, we can account for both potential small errors in the calibration and idiosyncratic patterns in saccade metrics (Van der Stigchel et al., 2006). We excluded trials in which the

deviation was further than 2.5 standard deviations away from the mean deviation for that condition (to exclude the influence of outliers).

Separate calculations of endpoint deviations were made for each distractor location ('Left Upper', 'Left Lower', 'Right Upper', and 'Right Lower' quadrant) and collapsed across the quadrants in each hemifield if both quadrants in the hemifield had the same visual status as determined by our visual field test (i.e. both 'seeing' or both 'blind' – Case 1). If only one quadrant was blind, to maintain equivalent power between blind and seeing hemifields we limited seeing hemifield data to the corresponding quadrant in the contralateral visual field (e.g. if the bottom-right was blind, this was matched by the seeing bottom-left). Thus, for Case 2, we matched the top-right quadrant with the top-left visual quadrant, whereas for Case 3, we matched the bottom-left quadrant with the bottom-right quadrant. For both distractors in the blind and intact fields, we then used *t*-tests to determine whether the induced deviations in saccadic endpoint were significantly different from zero. A mean saccade deviation of zero indicates no difference between the no-distractor and the distractor condition. Finally, we asked whether the presence of a distractor in either the blind or the seeing field influenced saccade latencies, using independent *t*-test to check for any difference from the mean latency in the condition with no-distractor.

## 3. Results

### 3.1. Visual field test

In all three cases, the visual field test produced results consistent with expectations from clinical perimetry. The visual field test was used to determine the status of each quadrant (i.e. intact or blind) for the stimuli we used. We only considered a quadrant to be blind if participants never reported any awareness of a stimulus presented at the location to be used in the experiment as distractor location. Conversely we considered a quadrant to be intact if participants always reported awareness of a stimulus at the distractor location. Quadrants which were neither intact or blind (i.e. mixed awareness) were not analyzed further. Case 1 was consistently unaware of a stimulus at the test location in both the upper and the lower quadrant of the contralateral hemifield. Case 2 lacked awareness for stimuli at the location in the top contralateral quadrant, whereas Case 3 lacked awareness for stimuli at the location in the bottom contralateral quadrant. For the ipsilateral quadrants, all three cases reported awareness for all stimuli at all tested locations. See Table 1 for performance in the visual field test for the quadrants which were considered blind.

### 3.2. Distractor experiment

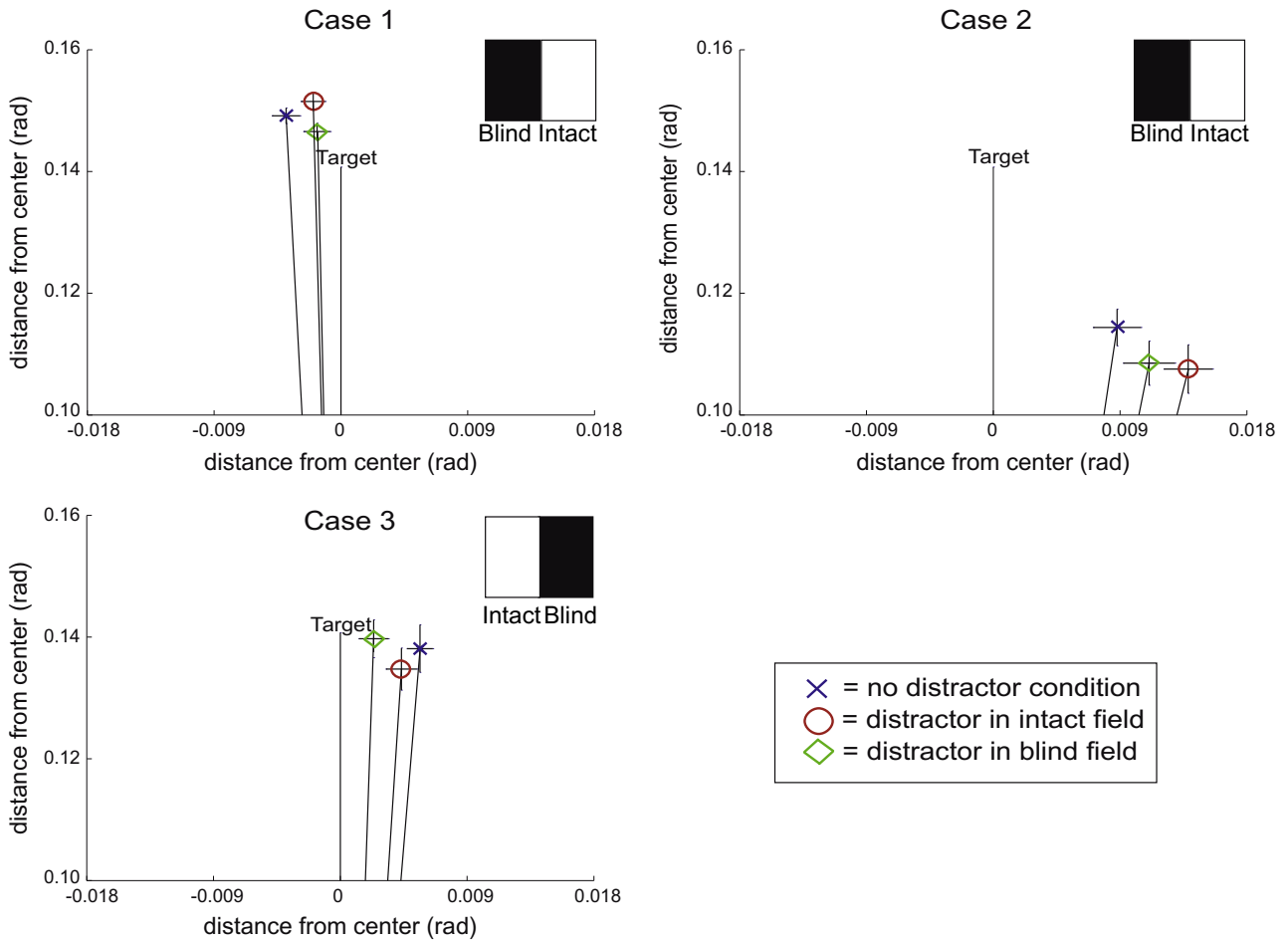
#### 3.2.1. Saccadic endpoint

For the control group, as expected, the endpoint of the eye movement deviated in the direction of the distractor (mean deviation = 0.038 rad; st. dev. = 0.031 rad; *t*(5) = 2.98; *p* < 0.05).

**Table 1**

Performance in the visual field test for the quadrants that were considered blind in the endpoint analyses. The top row indicates the horizontal distance from fixation. Values denote percentage correct for each of the four tested locations (see Fig. 3). In the distractor experiment, the distractor was presented at a horizontal distance of 3.13° away from fixation (indicated in bold).

Case	6.26° (%)	<b>3.13° (%)</b>	2.00° (%)	1.00° (%)
1	0	<b>0</b>	50	100
2	0	<b>0</b>	100	100
3	0	<b>0</b>	0	22



**Fig. 4.** Mean endpoints for the three conditions with SEM of x- and y-direction, collapsed across top and bottom of the visual field. Endpoint deviations are illustrated by a line between saccade startpoint (not shown in these graphs) and saccadic endpoint. The legend in the top right of each case illustrates the side of the visual field which was considered blind or intact. Note that the vertical and the horizontal axes use different scales.

Fig. 4 shows the mean endpoints of the participants with hemifield defects for the different conditions. The small errors in endpoint with respect to the target location in the no-distractor condition were also observed in the control group (see Appendix A). When related to the no-distractor condition, the effects of the distractor were generally quite small, but consistent within the three cases. Similar to the control group, visible distractors in the intact ipsilateral hemifield also influenced saccadic endpoint in all cases (Table 2), although only marginally in Case 3. For distractors in blind regions, we observed a deviation of the saccadic endpoint in Cases 1 and 3; however, in both cases this deviation was paradoxically away from the distractor, rather than towards it. Cases 2 showed no effect of distractors in the blind field.

3.2.2. Saccade latencies

In the control group, there was no difference in saccade latencies between the trials with a distractor (mean = 184 ms; st.

dev. = 24 ms) and the trials without a distractor (mean = 185 ms; st. dev. = 23 ms;  $t(5) = 0.36$ ;  $p = 0.73$ ). This is consistent with previous research that has shown that saccade latencies are not influenced by a distractor presented within 30° of the target (Walker et al., 1997).

Table 3 shows the results for the participants with hemifield defects. There was no effect of a distractor on saccade latencies in either the blind or seeing field in Case 1 (blind field:  $t(181) = 0.83$ ;  $p = 0.41$ ; seeing field:  $t(181) = 1.16$ ;  $p = 0.25$ ), or in Case 2 (blind field:  $t(77) = 0.33$ ;  $p = 0.74$ ; seeing field:  $t(76) = 0.91$ ;  $p = 0.37$ ). Case 3 did not show an effect on latency of a visible distractor ( $t(87) = 0.25$ ;  $p = 0.80$ ), but did have shorter latencies with a blind field distractor than in the absence of a distractor ( $t(88) = 2.38$ ;  $p < 0.02$ ).

4. Discussion

The current study investigated whether a stimulus in a blind visual field evokes oculomotor competition as measured by a shift in the endpoint of a saccade to a seen target. In our trials the target and distractor were near each other, a condition in which the global effect is evoked (Walker et al., 1997). Our results in both the control group and the intact ipsilateral hemifield of hemianopic participants confirmed that our parameters did generate a significant global effect. For distractors in blind regions, we also found a significant shift of saccadic endpoint in two of three participants with lesions of the striate cortex or distal optic radiations. The direction of the effect was paradoxical, however, in that the end-

**Table 2**  
Mean endpoint deviation for the participants in radians (polar coordinates). Note that positive and negative values refer to measurements towards and away of the distractor location, respectively.

Case	Blind field		Intact field	
	Endpoint deviation	t-test	Endpoint deviation	t-test
1	-0.0180	$p < 0.01$	+0.0178	$p < 0.01$
2	-0.0000	$p = 0.99$	+0.0403	$p < 0.01$
3	-0.0172	$p < 0.01$	+0.010	$p = 0.058$

**Table 3**  
Mean saccade latency for the three different conditions.

Case	No distractor		Distractor in blind field		Distractor in intact field	
	Mean (ms)	St. dev. (ms)	Mean (ms)	St. dev. (ms)	Mean (ms)	St. dev. (ms)
1	246	55	253	51	255	53
2	233	51	236	42	223	45
3	330	74	299	48	326	79

point shifted away from the distractor in these two cases. These results show that the normal global effect is not a general characteristic in hemianopic vision, although it provides further evidence that elements presented in the blind visual field can generate modulatory interactions in the oculomotor system, consistent with our prior study (Van der Stigchel et al., 2008).

The fact that endpoint deviation was not observed in one of the three hemianopic participants with a lesion of the optic radiations or striate cortex indicates a variability to the phenomenon that is consistent with other blindsight studies that examined a series of participants, which tend to show that blindsight abilities are found only in a minority of hemianopic participants (Barton & Sharpe, 1997; Kasten, Wuest, & Sabel, 1998; Scharli, Harman, & Hogben, 1999; Van der Stigchel et al., 2008). However, given the relatively modest size of the global effects we found, it is also possible that our paradigm was simply not sensitive enough to detect a similar effect in this remaining participant.

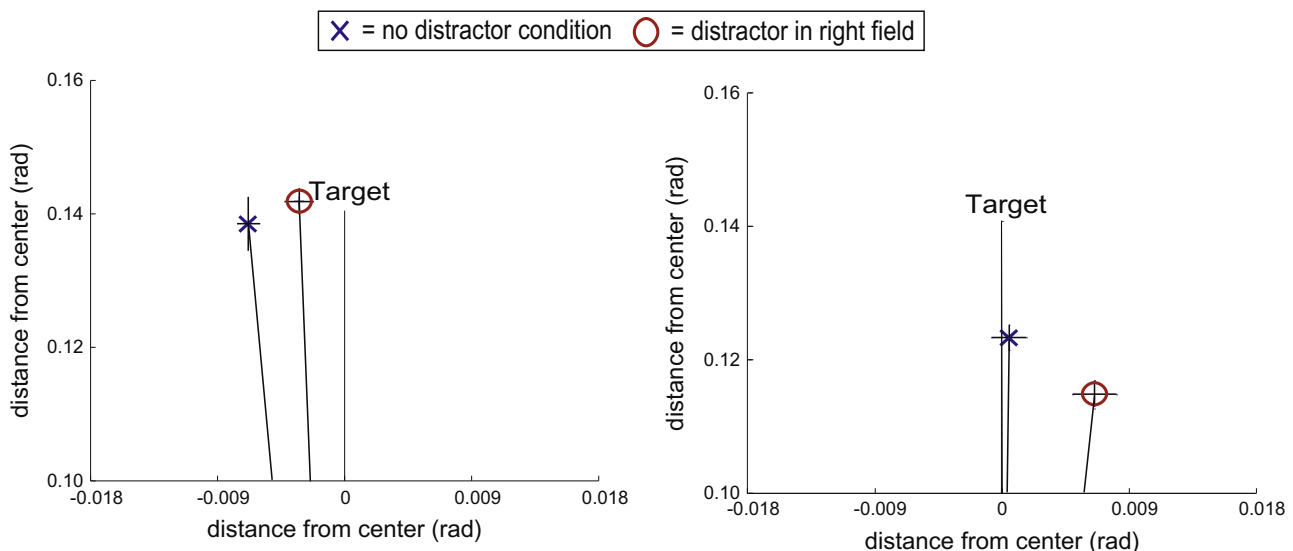
Shifts in saccadic endpoint are generally explained in terms of the ‘center of gravity account’, which states that the saccadic endpoint is based on the relative saliency of different elements in the saccade map (Coren & Hoenig, 1972). This account is in line with the finding that activity in the superior colliculus is highest at a location in between the two stimuli when a global effect occurs (Glimcher & Sparks, 1993; Van Opstal & Van Gisbergen, 1990). Because the distractor is presented simultaneously with the target, there is competition for selection in the oculomotor system between these two elements. The result of this competition is an eye movement, which is an average of the eye movements to the target and to the distractor. In the current study, the shift of saccadic endpoint was relatively small, which could be explained by the fact that the target could be easily selected on the basis of both

shape and location, resulting in a relatively weak competition between target and distractor. Nonetheless, the competition between target and distractor still evoked a shift of saccadic endpoint in the direction of the distractor when a distractor was in the intact visual field.

The result that the shift of saccadic endpoint was reversed for a distractor in the blind visual field in two out of three participants is consistent with earlier findings that hemianopic vision is different from normal vision, because information about the blind stimulus is processed in an unusual way (Azzopardi & Cowey, 1997). A possible explanation for this paradoxical effect is the absence of a zone in the blind hemifield in which the global effect occurs. Because shifts of saccadic endpoints away from a distractor have been related to strong inhibition of the distractor when presented outside the global effect zone (Van der Stigchel, Meeter, & Theeuwes, 2007), inhibition of the distractor in the absence of a global effect zone might explain why the endpoint deviated away from a distractor in the blind field. The resulting saccade is than an average of an eye movement to the target and an inhibited eye movement to the distractor, which will end away from the distractor location.

An explanation of the absence of a global effect zone might be the abnormal input to the superior colliculus. The normal global effect could represent an interaction between target and distractor that actually originates in cerebral cortex and is then reflected in neural patterns seen in collicular maps (Glimcher & Sparks, 1993; Van Opstal & Van Gisbergen, 1990). In the absence of cortical influences, the direct retino-collicular effect may be a different phenomenon, an interaction between an inhibitory zone (caused by the distractor) and an excitatory zone (caused by the target), rather than an interaction between two excitatory zones that is inherited from cortical activation patterns. As explained above, this interaction between the inhibitory and excitatory zone might then result in an ‘average’ eye movement that ends away from the distractor location.

Because target and distractor were closely aligned, care was taken to ensure that the observer was indeed unaware of distractors at the tested location in the blind hemifield, by performing a field test with concomitant monitoring of eye movements. Moreover, experimental trials were only included as valid when the start point of the eye movement was within 1.5° from screen center. Given that the horizontal distance of the distractor from the center fixation point was 3.13°, this ensured that the distractor was at



**Fig. A1.** Endpoint pattern of two individual participants in the healthy group. See Appendix A for more detail.

least 1.63° to the left of the participant's vertical meridian. Furthermore, to reduce possible effects of light scatter (i.e. a stimulus projected to the blind field is associated with light diffusing into the seeing field, Cowey, 2004), the experiment was performed with bright room background lighting, with the distractor being a black stimulus presented on a gray background. These experimental conditions appear to have successfully ensured the invisibility of blind field distractors, as no participant reported having seen distractors in the blind location when asked explicitly about this after completion of the experiment. Last, it is unlikely that inadvertent visibility of distractors accounted for our results, because that would be expected to lead to normal rather than paradoxical global effects.

Further investigation of the origins of this effect also have to reconcile the paradoxical global effect we found with the conflicting findings in two prior reports of normally directed global effects in two hemianopic participants (Barbur et al., 1988; Van der Stigchel et al., 2008). In addition to the absence of any global effect from some blind hemifields, these reports point to further heterogeneity in blindsight, and suggest complexities in the anatomic pathways and functional bases of visual function without a striate cortex.

## Appendix A

The small errors in endpoint with respect to the target location in the no-distractor condition were also observed in the control group. To account for possible failures in calibration or idiosyncratic patterns, the global effect is therefore always measured with respect to a no-distractor condition. Figure A1 shows the endpoint patterns of two individual participants in the healthy group. The plot on the left shows a participant which shows the same error in the no-distractor condition as our cases. The plot on the right shows a participant which shows the same hypometric saccades as Case 2. The small error in the no-distractor condition for the participants with hemifield defects seems to be in the range of the normal idiosyncratic variability in endpoint: a far larger sample of patients would be required to determine if there is a systematic change in endpoint beyond this normal variability.

## References

- Aizawa, H., & Wurtz, R. H. (1998). Reversible inactivation of monkey superior colliculus. I. Curvature of saccadic trajectory. *Journal of Neurophysiology*, 79(4), 2082–2096.
- Azzopardi, P., & Cowey, A. (1997). Is blindsight like normal, near-threshold vision? *Proceedings of the National Academy of Sciences*, 94(25), 14190–14194.
- Barbur, J. L., Forsyth, P. M., & Findlay, J. J. (1988). Human saccadic eye movements in the absence of the geniculocalcarine projection. *Brain*, 111, 63–82.
- Barbur, J. L., Ruddock, K. H., & Waterfield, V. A. (1980). Human visual responses in the absence of the geniculo-calcarine projection. *Brain*, 103, 905–928.
- Barton, J. J. S., & Sharpe, J. A. (1997). Motion direction discrimination in blind hemifields. *Annals of Neurology*, 41, 255–264.
- Coren, S., & Hoenig, P. (1972). Effect of non-target stimuli on the length of voluntary saccades. *Perceptual and Motor Skills*, 34, 499–508.
- Cowey, A. (2004). The 30th Sir Frederick Bartlett lecture: Fact, artefact, and myth about blindsight. *The Quarterly Journal of Experimental Psychology*, 57A(4), 577–609.
- Danckert, J., & Rossetti, Y. (2005). Blindsight in action: What can the different subtypes of blindsight tell us about the control of visually guided actions? *Neuroscience & Biobehavioral Reviews*, 29(7), 1035–1046.
- Danziger, S., Fendrich, R., & Rafal, R. (1997). Inhibitory tagging of locations in the blind field of hemianopic patients. *Consciousness and Cognition*, 6, 291–307.
- Glimcher, P. W., & Sparks, D. L. (1993). Representation of averaging saccades in the superior colliculus of the monkey. *Experimental Brain Research*, 429, 435.
- Intriligator, J. M., Xie, R., & Barton, J. J. (2002). Blindsight modulation of motion perception. *Journal of Cognitive Neuroscience*, 14, 1174–1183.
- Kasten, E., Wuest, S., & Sabel, B. (1998). Residual vision in transition zones in patients with cerebral blindness. *Journal of Clinical and Experimental Neuropsychology*, 20, 581–598.
- Leh, S. E., Johansen-Berg, H., & Ptito, A. (2006). Unconscious vision: new insights into the neuronal correlate of blindsight using diffusion tractography. *Brain*, 129, 1822–1832.
- Marcel, A. J. (1998). Blindsight and shape perception: Deficit of visual consciousness or of visual function? *Brain*, 121, 1565–1588.
- McPeck, R. M., Han, J. H., & Keller, E. L. (2003). Competition between saccade goals in the superior colliculus produces saccade curvature. *Journal of Neurophysiology*, 89(5), 2577–2590.
- Perenin, M. T. (1991). Discrimination of motion direction in perimetrically blind fields. *Neuroreport*, 2, 397–400.
- Pöppel, E., Held, R., & Frost, D. (1973). Residual visual function after brain wounds involving the central visual pathways in man. *Nature*, 243, 295–296.
- Rafal, R. D., Smith, J., Cohen, A., & Brennan, C. (1990). Extrageniculate vision in hemianopic humans: Saccade inhibition by signals in the blind field. *Science*, 250, 118–121.
- Rees, G. (2008). The anatomy of blindsight. *Brain*, 131(6), 1414–1415.
- Sahraie, A., Weiskrantz, L., Barbur, J. L., Simmons, A., Williams, S. C. R., & Brammer, M. J. (1997). Pattern of neuronal activity associated with conscious and unconscious processing of visual signals. *Proceedings of the National Academy of Sciences*, 94, 9406–9411.
- Sanders, M. D., Warrington, E. K., Marshall, J., & Weiskrantz, L. (1974). 'Blindsight': Vision in a field defect. *Lancet*, 1, 707–708.
- Scharli, H., Harman, A., & Hogben, J. (1999). Blindsight in subjects with homonymous visual field defects. *Journal of Cognitive Neuroscience*, 11, 52–66.
- Van der Stigchel, S., Meeter, M., & Theeuwes, J. (2006). Eye movement trajectories and what they tell us. *Neuroscience & Biobehavioral Reviews*, 30(5), 666–679.
- Van der Stigchel, S., Meeter, M., & Theeuwes, J. (2007). The spatial coding of the inhibition evoked by distractors. *Vision Research*, 47(2), 210–218.
- Van der Stigchel, S., & Theeuwes, J. (2005). Relation between saccade trajectories and spatial distractor locations. *Cognitive Brain Research*, 25(2), 579–582.
- Van der Stigchel, S., van Zoest, W., Theeuwes, J., & Barton, J. J. S. (2008). The influence of 'blind' distractors on eye movement trajectories in visual hemifield defects. *Journal of Cognitive Neuroscience*, 20(11), 2025–2036.
- Van Opstal, A. J., & Van Gisbergen, J. A. M. (1990). Role of monkey superior colliculus in saccade averaging. *Experimental Brain Research*, 79, 143–149.
- Walker, R., Deubel, H., Schneider, W. X., & Findlay, J. M. (1997). Effect of remote distractors on saccade programming: Evidence for an extended fixation zone. *Journal of Neurophysiology*, 78(2), 1108–1119.
- Walker, R., Mannan, S., Maurer, D., Pambakian, A. L. M., & Kennard, C. (2000). The oculomotor distractor effect in normal and hemianopic vision. *Proceedings of the Royal Society of London B*, 267, 431–438.
- Weiskrantz, L., Warrington, E. K., Sanders, M. D., & Marshall, J. (1974). Visual capacity in the hemianopic field following a restricted occipital ablation. *Brain*, 97, 709–728.