The Influence of ''Blind'' Distractors on Eye Movement Trajectories in Visual Hemifield Defects

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Abstract

■ There is evidence that some visual information in blind regions may still be processed in patients with hemifield defects after cerebral lesions (''blindsight''). We tested the hypothesis that, in the absence of retinogeniculostriate processing, residual retinotectal processing may still be detected as modifications of saccades to seen targets by irrelevant distractors in the blind hemifield. Six patients were presented with distractors in the blind and intact portions of their visual field and participants were instructed to make eye movements to targets in the intact field. Eye movements were recorded to determine if blind-field distractors caused deviation in saccadic trajectories. No deviation was found in one patient with an optic chiasm lesion, which affect both retinotectal and retinogeniculostriate pathways. In five patients with lesions of the optic radiations or the striate cortex, the results were mixed, with two of the five patients showing significant deviations of saccadic trajectory away from the ''blind'' distractor. In a second experiment, two of the five patients were tested with the target and the distractor more closely aligned. Both patients showed a ''global effect,'' in that saccades deviated toward the distractor, but the effect was stronger in the patient who also showed significant trajectory deviation in the first experiment. Although our study confirms that distractor effects on saccadic trajectory can occur in patients with damage to the retinogeniculostriate visual pathway but preserved retinotectal projections, there remain questions regarding what additional factors are required for these effects to manifest themselves in a given patient. ■

INTRODUCTION

Although the predominant visual pathway in humans is the retinogeniculostriate pathway, which projects from the retina to the lateral geniculate nucleus and then to the primary visual cortex, other pathways also exist, including in particular the retinotectal pathway, which projects from the retina to the superior colliculus in the midbrain (Cowey, 2004). In humans, a lesion of the retinogeniculostriate pathway beyond the optic chiasm results in a hemifield defect, in which visual loss is present in both eyes but is limited to the field contralateral to the lesion. Extensive research over the past 30 years suggests that, despite lack of conscious visual perception in the blind field, some residual unconscious visual function may still persist in at least some patients, a phenomenon named ''blindsight'' (Weiskrantz, 1986).

Studies of blindsight have used numerous methods to investigate a variety of visual functions. Most common are studies of saccadic or manual pointing accuracy to luminant targets in the blind hemifield (Blythe, Kennard, & Ruddock, 1987; Perenin & Jeannerod, 1975; Weiskrantz, Warrington, Sanders, & Marshall, 1974; Pöppel, Held, & Frost, 1973), and forced-choice discrimination regarding stimulus attributes such as motion, color, or form (Perenin, 1991; Barbur, Ruddock, & Waterfield, 1980; Sanders, Warrington, Marshall, & Weiskrantz, 1974; Weiskrantz et al., 1974). More recently, studies also turned to ''indirect'' strategies that circumvent the awkward need to ask patients to respond to something they denied seeing. Rather, these indirect methods measure the effect of blind-field stimuli on the patient's responses to targets in the seeing visual field (Intriligator, Xie, & Barton, 2002; Marcel, 1998; Danziger, Fendrich, & Rafal, 1997; Tomaiuolo, Ptito, Marzi, Paus, & Ptito, 1997; Corbetta, Marzi, Tassinari, & Aglioti, 1990; Rafal, Smith, Cohen, & Brennan, 1990; Weiskrantz, 1990; Marzi, Tassinari, Aglioti, & Lutzemberger, 1986; Pizzamiglio, Antonucci, & Francia, 1984).

Some blindsight experiments were motivated by the hypothesis that, in the absence of retinogeniculostriate processing, residual processing of the retinotectal pathway may still be detected. For example, saccadic localization was considered a function that could reflect retinotectal processing, and hence, was the subject of the first blindsight study (i.e., Zihl, 1980; Pöppel et al., 1973). Putative functions of the superior colliculus (i.e., Leh, Johansen-Berg, & Ptito, 2006; Sahraie et al., 1997)

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continue to motivate more recent studies of blindsight that have used indirect strategies. However, evidence for these functions is controversial. For instance, Rafal et al. (1990) showed that distractors in the blind field of three hemianopic patients increased saccade latencies to a target in the intact field. In normal subjects, when target and distractor are presented far apart, saccade latency is increased relative to a single target condition, a finding known as the remote distractor effect (Levy-Schoen, 1969), an effect that is likely mediated by the superior colliculus (Olivier, Dorris, & Munoz, 1999). However, these results were not replicated in another study (Walker, Mannan, Maurer, Pambakian, & Kennard, 2000), and another (unpublished) study observed a remote distractor effect for blindsight patient G.Y. but not in several other hemianopic patients (Cochrane, 1995; described in Weiskrantz, 1997).

Given these conflicting results, the existence of retinotectal function in blindsight still remains uncertain, particularly because there are other potential explanations of blindsight, including, for example, direct projections from the geniculate to the extrastriate cortex (Stoerig & Cowey, 1997). Therefore, to further investigate retinotectal visual processing in blindsight, we studied hemianopic vision using a new measure of distractor effects on saccadic eye movements.

In recent years, there has been an increasing interest in modifications of the trajectory of saccades (for a review, see Van der Stigchel, Meeter, & Theeuwes, 2006). These studies have revealed that these modifications are a measure of visual processing and can yield valuable information about the mechanisms that control eye movements. In visual search experiments involving either humans (Walker, McSorley, & Haggard, 2006; Godijn & Theeuwes, 2002; McPeek, Skavenski, & Nakayama, 2000) or monkeys (McPeek, Han, & Keller, 2003; Port & Wurtz, 2003; McPeek & Keller, 2001), saccades to a target deviate toward the location of a salient distractor. In addition, irrelevant distractors may also cause saccade trajectories to deviate away from the location of the distractor (Van der Stigchel & Theeuwes, 2005; Ludwig & Gilchrist, 2003; Doyle & Walker, 2001, 2002). Specifically, the direction of the saccadic deviation (toward or away from a distractor) depends on top–down influences, such as whether the participant has prior knowledge about the location of either the target or the distractor (Van der Stigchel et al., 2006; Walker et al., 2006).

Similar to the remote distractor effect, these deviations in saccadic trajectory are thought to reflect competition between saccade goals in the superior colliculus. McPeek et al. (2003) showed that when a saccade deviated toward a distractor during visual search, there was increased presaccadic activity at the location of the distractor. Also, microstimulation of the superior colliculus below the threshold for saccade generation caused saccades to deviate toward the stimulated location, and the magnitude of this deviation correlated with the activity induced at the stimulated location.

Given the evidence that the superior colliculus plays a role in generating the deviations in saccade trajectory that are induced by distractors, we hypothesized that such deviations could be used to probe for residual retinotectal function in blindsight. Similar to previous studies investigating the interference evoked by ''blind'' distractors (Walker et al., 2000; Cochrane, 1995; Rafal et al., 1990), we recorded saccades to visible targets, with and without distractors that could be located in either blind or intact portions of the visual field. The influence of the distractor was examined by measuring deviations in saccadic trajectory.

EXPERIMENT 1

Methods

Subjects

We studied five patients with homonymous hemifield defects from lesions of the optic radiations or the striate cortex due to strokes or intracerebral hemorrhages (Table 1, Figure 1). These subjects have lesions that affect the retinogeniculostriate pathway but not the retinotectal one. As a control, we tested a sixth patient with a pituitary tumor that compressed his optic chiasm, causing bitemporal hemianopia, which affects both retinogeniculate and retinotectal projections. Compared to most stroke patients, all six patients were relatively young, being between the ages of 20 and 50 years. All patients had a complete neurologic and neuro-ophthalmologic examination excluding other ocular or neurologic conditions. Visual fields were documented with Goldmann perimetry (Figure 2), and the brightest stimulus, the V4e target, was used to verify blindness in the retinotopic regions to be studied. Hemineglect was excluded by normal performance on the Sunnybrook Neglect Assessment Battery (Weintraub & Mesulam, 1985). All subjects 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.

As expected with naturally occurring lesions, the five patients with cerebral lesions varied in the extent of both their hemifield loss and lesions. Case 1 had an extensive lesion affecting the left medial occipital lobe, fusiform gyri, lateral occipito-temporal cortex, and anterior temporal cortex. Case 2 had a very focal infarct of the striate cortex and underlying white matter, with a correspondingly modest homonymous paracentral scotoma. Case 3 had a typical right posterior cerebral arterial infarction, affecting striate and medial occipito-temporal structures, including the posterior lingual gyrus. Case 4 had an occipital lobar hemorrhage that left residual damage to the white matter underlying the striate cortex, lingual gyrus, and dorsomedial occipital cortex, including the cuneus.

Table 1. Patients Studied

Case	*Age (years)*	*Sex*	*Duration (months)*	*Lesion*	*Visual Structure*
1	42	F	60	infarct, middle cerebral artery	optic radiations
2	40	F	14	infarct, medial occipital cortex	striate cortex
3	47	M	1	infarct, medial occipital cortex	optic radiations/striate
4	40	M	144	hemorrhage, occipital lobe	optic radiations
5	27	M	60	infarct, medial occipital	striate cortex
6	47	M	1	pituitary adenoma	optic chiasm

Case 5 had a restricted left posterior cerebral arterial infarction affecting primarily the striate cortex, the posterior aspect of the medial occipital lobe, and the distal termination of the optic radiations.

The five patients with homonymous hemifield defects were tested with both eyes open, and the patient with bitemporal hemianopia was tested with only the left eye viewing. Four patients had incomplete hemifield defects (Cases 1, 2, 3, and 6) and in three of these (Cases 1, 3, and 6) the hemifield defect affected one contralateral visual quadrant primarily, with residual vision in the region where our experimental stimuli were located in the other quadrant. For these patients, analysis of the incomplete hemifield was limited to the blind quadrant. In all patients, the degree of saccade deviation induced by distractors in the blind portion of the visual field was compared to that induced by distractors in the ipsilateral (sighted) hemifield to verify that the distractors were indeed capable of inducing saccade deviation.

Apparatus

Participants performed the experiment in a sound-attenuated setting with bright background lighting (10–20 lx), viewing a display monitor from a distance of 70 cm. Eye movements were recorded by an Eyelink-II system (SR Research, Canada), an infrared video-based eye tracker that has a 500-Hz temporal resolution and a spatial resolution of 0.01°. The subject's head was stabilized with a chin and forehead rest, and an infrared head-mounted tracking system compensated for any residual head motion. The left eye was monitored in all subjects. An eye movement was considered a saccade when either eye velocity exceeded 35°/sec or eye acceleration exceeded 9500°/sec^2.

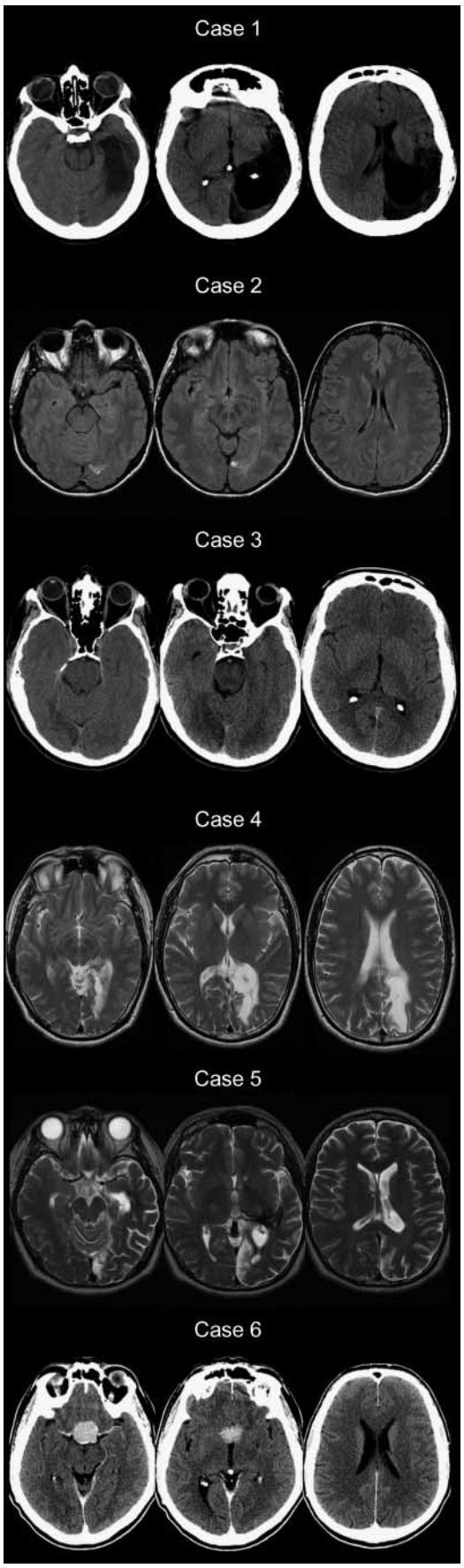
Stimuli

All figures (fixation, target, distractor) were white (40 cd/m^2) on a black background of luminance (0.6 cd/m^2), as measured with an OptiCal photometer (Model OP200-E, Cambridge Research Systems). Each trial started with the presentation of a ''plus'' character (0.94° × 0.94°) in the center of the screen that functioned as the fixation stimulus (Figure 3). After 600 msec, an arrow (of 1.09° height and 0.13° width) pointing up or down appeared directly above or below the fixation position (''cue''). After a variable period of 800 to 1200 msec, the target appeared (a solid circle with a diameter of 0.94°) located at an eccentricity of 8.1° on the vertical meridian, in the direction indicated by the cue. In a third of trials, the target was the only element presented. In the remaining two-thirds, a diamond-shaped distractor (sides measuring 1.09° × 1.09°) appeared at the same time as the target. The distractor was always located in the same vertical hemifield as the target, but half the time it was in the spared hemifield ipsilateral to the lesion, and half the time in the contralateral hemifield. The distractor was presented 6.26° away from fixation in the horizontal direction and 4.69° away in the vertical direction. Both elements were presented for 1200 msec.

Procedure and Design

Participants were instructed to fixate the center fixation point until the target appeared, when they were to move their eyes to the target. It was stressed that they should try to make a single accurate saccade. Each session started with a 9-point grid calibration procedure. In addition, simultaneously fixating the center fixation point and pressing the space bar recalibrated the system by zeroing the offset of the measuring device at the start of each trial.

After calibration, we administered a short visual field test to confirm our estimations from Goldmann perimetry about which contralateral target locations were located in seeing versus blind regions. Subjects were given 52 trials, 26 of which contained no stimulus and 26 of which contained a circle of the same luminance as the experimental targets, shown at one of 26 possible locations in either hemifield, including the locations of potential targets and distractors in the experiment. Participants had to report at the end of each trial whether they saw a circle. The question, ''Did you see a gray circle? If YES, press 'z'. If NO press '/'.'' was presented on the screen and participants were required to report their awareness by a key response. For all patients, this test replicated the Goldmann perimetry with patients responding ''yes'' to



stimuli in the intact visual field and responding "no" to stimuli in the blind visual field. None of the subjects reported any awareness of stimuli in their blind regions with this technique, or during the actual experiment.

The experiment consisted of a training session of 30 trials and an experimental session of 300 trials. Participants heard a short tone when the saccade latency was higher than 600 msec or lower than 80 msec. The sequence of trials was randomized for each participant, in terms of both target location (up or down) and distractor condition (none, contralateral, or ipsilateral).

Data Analysis

Saccade latency was defined as the interval between target onset and the initiation of a saccadic eye movement. If saccade latency was shorter than 80 msec, or longer than 600 msec or 2.5 standard deviations from the subject's mean latency, the trial was removed from the analysis. Trials were excluded if there was no saccade or the first saccade was too small ($<3^\circ$). If the endpoint of the first saccade had an angular deviation of more than 22.5° from the center of the target, the saccade was classified as an error and was not analyzed. Furthermore, the initial saccade starting position had to be within 2° from the center fixation point for the vertical and 1° for the horizontal direction. These exclusion criteria led to a loss of 18% of trials. With respect to saccade latency, 1.94% of trials were removed because latency was shorter than 80 msec and 2.44% of trials were removed because the latency was longer than 600 msec.

In the remaining trials, we measured saccade deviation, defined as the mean angle of the saccade path relative to the angle of a straight line between the saccade starting position and the target location. The mean angle of the saccade path was calculated by averaging the angles of the straight lines between the saccade starting position and the different sample points (for a more detailed overview of saccade trajectory computation, see Van der Stigchel et al., 2006). For each saccade in a trial with a distractor, we compared its mean path angle to that of the averaged mean-path-angles of all saccades in trials without a distractor, to determine if the saccade in the presence of a distractor deviated toward or away from the location of the distractor. Deviations were signed so that a positive value indicated deviation toward the distractor, and a negative value indicated deviation away from the distractor.

Separate calculations were made for each distractor location ("left upper," "left lower," "right upper," and

Figure 1. Axial MRI or CT images of lesions in the patients. Case 1 has a large occipito-temporo-parietal infarct. Cases 2 and 5 have small infarcts of the striate cortex. Case 3 has a more extensive medial occipito-temporal infarct. Case 4 shows residual damage after an occipital hemorrhage. Case 6 has a pituitary adenoma compressing the optic chiasm, shown as the bright mass on this CT scan with contrast.

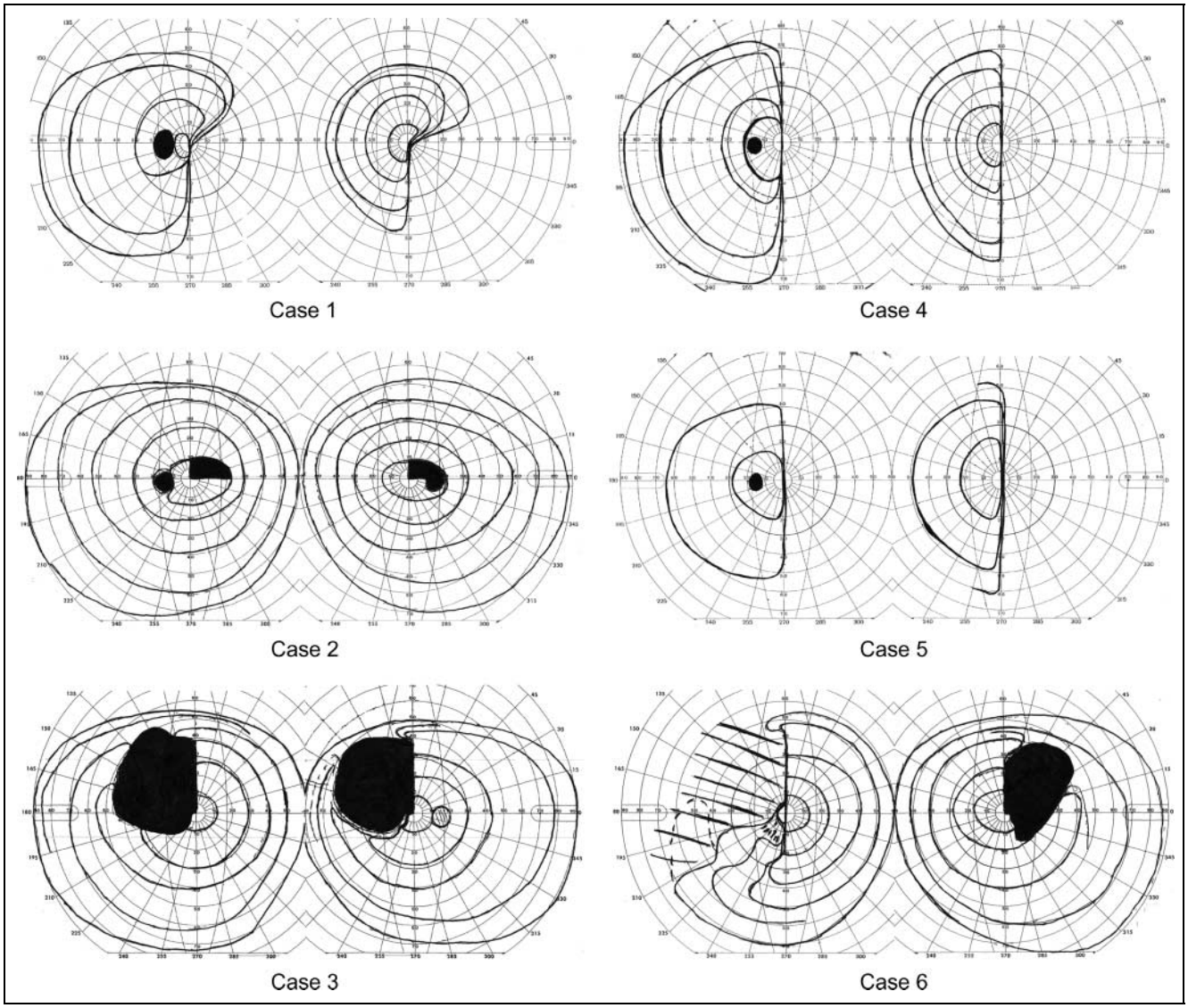


Figure 2. Goldmann perimetry of visual fields of six patients. The largest isopter in all cases represents the V4e target. Black regions indicate scotomata. Likewise, the oblique slashes for Case 6 indicate regions where the patient does not respond to visual stimuli.

''right lower''), but then collapsed in each hemifield if both vertical quadrants had the same visual status (seeing vs. blind). We excluded trials in which the deviation of mean-path-angle was further than two-and-a-half standard deviations away from the mean deviation. Using the remaining data, for both the blind and intact field, we used *t* tests to determine whether the deviations in saccade trajectory were significantly different from zero. Note that a mean saccade deviation of zero indicates no difference between the no-distractor and the distractor condition.

In order to validate our within-subject analysis, we reanalyzed data from a different experiment in a non-patient population. This experiment was run for a different reason but had exactly the same set-up as the present experiment. The only difference was that, besides vertical eye movements, horizontal movements were also included. This experiment included nine healthy participants (aged 17–22). We analyzed only the upward and downward conditions which consisted of the same amount of trials as the present experiment. Results showed that, for all participants, eye movement trajectories significantly deviated away from the distractor ($p < .05$). This is in line with numerous findings in similar experiments (Van der Stigchel & Theeuwes, 2005, 2006; Ludwig & Gilchrist, 2003; Doyle & Walker, 2001, 2002), although these studies did not analyze the data in a within-subject manner. The current analysis therefore shows that this type of set-up enables to investigate deviations within a single subject.

Results

Distractors in the seeing hemifield influenced saccade trajectories in all participants (Table 2). In two subjects (Cases 1 and 3), saccades deviated toward the distractor, whereas deviation away from the distractor

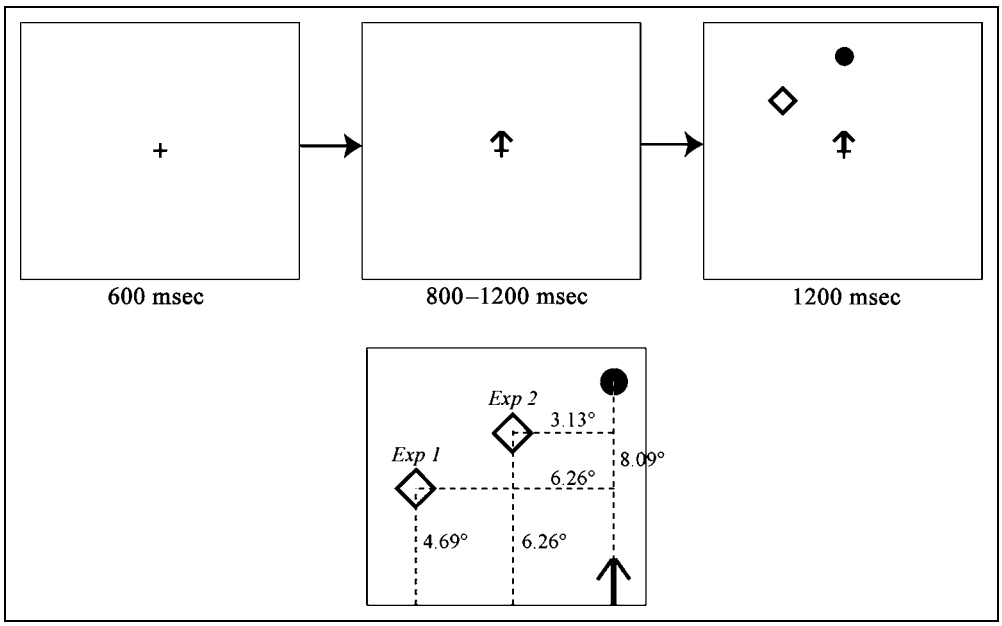


Figure 3. Sequences of frames on a given trial in the present experiment. After 600 msec, an arrow cue pointing up or down appeared. After a variable period of 800 to 1200 msec, the target appeared in the direction indicated by the cue. In two-thirds of the trials, a diamond-shaped distractor appeared at the same time as the target. The bottom of the figure shows the exact location of the distractor for both experiments.

was observed for the four other patients. Figure 4 shows mean saccade trajectories for three subjects.

For distractors in the blind hemifield, we observed no saccade deviation in Case 6, the patient with a lesion of the optic chiasm that would be expected to deafferent both the retinogeniculostriate and retinotectal pathways. In the other five patients, with potentially spared retinotectal function, the results were mixed. Two patients (Cases 1 and 2) showed no saccade deviation induction by blind-field distractors. However, two other patients (Cases 3 and 4) showed significant induction of saccade deviation and another (Case 5) showed a trend to saccade deviation.

Comment

We found that a distractor in the intact field influenced saccadic trajectory in all patients, consistent with the numerous reports on the effect of an irrelevant distractor on saccadic trajectories in healthy subjects (Van der Stigchel & Theeuwes, 2005, 2006; Ludwig & Gilchrist, 2003; Doyle & Walker, 2001, 2002). This was an important control, as it ensured that lack of an effect in the blind hemifield was not due to problems with our paradigm in generating deviations in trajectory.

Of note, though, these intact-field distractors caused different deviations in different patients, with four patients showing deviation of saccades away from the distractor, and two showing deviation toward. In normal subjects, deviation away is generally observed (Van der Stigchel & Theeuwes, 2005, 2006; McSorley, Haggard, & Walker, 2005; Doyle & Walker, 2001, 2002), as was also found in our within-subject reanalysis of data from a different experiment in a nonpatient population. In patients there may be pathologic reasons for differences in the effect of distractors on saccadic trajectory. Although the effects of distractors are seen in the superior colliculus, top–down inhibition from cortical regions may contribute to deviation away from irrelevant distractors (Van der Stigchel et al., 2006; McSorley, Haggard, & Walker, 2004; Sheliga, Riggio, & Rizzolatti, 1994). Because frontal lobe (Guitton, Buchtel, & Douglas, 1985) and temporal–parietal damage (Butler, Gilchrist, Ludwig, Muir, & Harvey, 2006) may reduce inhibition in some oculomotor tasks, the lesions in some of our patients may have reduced top–down effects on saccadic deviation. Of note, the two patients with deviation toward the distractor in the seeing field (Cases 1 and 3) were those with the largest strokes in our group, with significant temporoparietal or occipito-temporal damage.

Regarding the blind field, we confirmed that no deviation occurred in the patient with a lesion of the optic chiasm. Our results in patients with lesions of the optic

Table 2. Mean Saccade Deviation for the Subjects in Radians

	Blind Field		*Intact Field*	
Case	*Saccade Deviation*	*t Test*	*Saccade Deviation*	*t Test*
1	−0.003	$p = .64$	+0.018	$p < .03$
2	+0.005	$p = .46$	−0.025	$p < .0001$
3	−0.027	$p < .03$	+0.036	$p < .01$
4	−0.035	$p < .001$	−0.046	$p < .0001$
5	+0.020	$p = .06$	−0.046	$p < .01$
6	−0.011	$p = .33$	−0.033	$p < .0001$

Note that positive and negative values refer to measurements toward the distractor location and away from the distractor location, respectively.

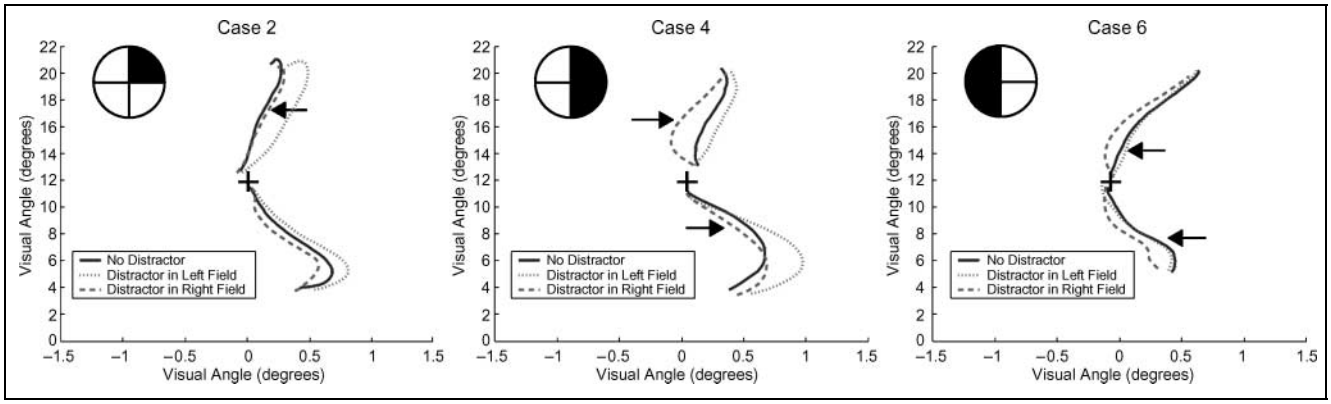


Figure 4. Mean saccade trajectories of each condition for three patients. In the top left of each figure, it is shown in which quadrant of the stimulus screen the patient could not see the distractor. Fixation is indicated by the central cross. The arrows indicate which trajectory is in response to a distractor in the blind field.

radiation or the striate cortex were mixed. Nevertheless, in two of our patients, saccades significantly deviated away from a distractor which was not consciously reported, supporting our hypothesis that in at least some patients with lesions affecting the retinogeniculostriate but not the retinotectal pathway, distractors could induce trajectory deviations.

Because saccade trajectory modifications are thought to be due to competition between possible saccade target locations in the oculomotor system, this indicates that visual stimuli in the blind visual field are still represented in the oculomotor system. To further investigate this issue, we ran another experiment looking at a different distractor effect. When distractors are located close to the target—within 20° or 30° of angular distance (Walker, Deubel, Schneider, & Findlay, 1997)—a ''global effect'' results, in that the eyes land on an intermediate location between the target and the distractor (Coren & Hoenig, 1972). The global effect is explained in terms of a ''center of gravity'' account, which states that the saccade endpoint is based on the relative saliency of the elements in the visual field (Coren & Hoenig, 1972). Recently, it has been shown that not only the endpoint is affected in this situation but the whole trajectory deviates toward the distractor (Van der Stigchel & Theeuwes, 2005). In Experiment 2, we tested for this deviation toward the distractor in two contrasting patients (Cases 2 and 4). Due to availability of the patients, only these two could be tested in the second experiment. One (Case 2) did not show an effect of a blind distractor on saccadic trajectory in Experiment 1, whereas the other (Case 4) did. Our hypothesis was that the global effect on trajectory would also be found in the latter patient but not the former. Moreover, it has been demonstrated that the global effect is greatly influenced by saccadic latency such that the deviation decreases as saccadic latencies increase (Van der Stigchel & Theeuwes, 2005). Time-course analyses were performed in Experiment 2 to investigate whether the patients show a similar decrease in deviation as a function of saccadic latency and if this varies depending on whether a distractor is presented in the blind or intact field of vision.

EXPERIMENT 2

Methods

Procedure

The current experiment was identical to Experiment 1, except for two aspects. First, the distractor was presented 3.13° away from fixation in the horizontal direction and 6.26° away in the vertical direction, and thus, was located close to the target location at 8.09° eccentricity on the vertical meridian (see Figure 3). Second, the target location was not cued in advance but was presented at one of the two target locations without any advance indication. The experiment consisted of a training session of 30 trials and an experimental session of 300 trials. Among the 26 locations tested in the short visual field test described in Experiment 1 were the locations of potential targets and distractors of the current experiment.

Data Analysis

We used the same exclusion criteria to eliminate trials of inappropriate latency, accuracy, or size, resulting in a loss of 7% of trials. With respect to saccade latency, no trials were removed because latency was shorter than 80 msec and 0.33% of trials were removed because the latency was longer than 600 msec. Again, we measured saccade deviation as defined in Experiment 1. This was done separately for distractors in blind regions of the contralateral hemifield and in seeing regions of the ipsilateral (sighted) hemifield. We then divided saccades in trials with distractors into three latency bins: the fastest third, the middle third, and the slowest third,

and averaged saccade deviation for all saccades within each bin. We used t tests to determine if the average saccade deviations within a bin were significantly different from zero. Analyses of variance were run to investigate whether there was a main effect of latency bin. If the bins were different, post hoc linear contrasts were used to investigate whether the deviation effect decreased with increasing latency.

Results

Case 2 showed in her intact field a consistent global effect: that is, a deviation toward the distractor that was significant for all three latency bins ($p < .02$; see Figure 5). There was a main effect of latency bin [$F(2, 90) = 10.25, p < .001$]: In line with previous research (Van der Stigchel & Theeuwes, 2005; Ottes, Van Gisbergen, & Eggermont, 1985), the deviation effect decreased when saccade latencies lengthened, as indicated by a post hoc linear contrast ($p < .001$). In contrast, distractors in the blind field did not generate a consistent deviation, with only a single significant effect in the fastest latency bin [$t(15) = 2.51, p < .05$], and a trend for a main effect of latency bin [$F(2, 28) = 3.17, p = .057$].

Case 4 showed a global effect in the intact field, but only for the fastest responses [$t(30) = 3.10, p < .01$]. There was a main effect of latency bin [$F(2, 58) = 7.88, p < .001$]: This effect again decreased when saccade latencies lengthened ($p < .001$) and even showed the opposite effect for the slowest responses [$t(29) = 2.52, p < .02$]. Distractors in the blind field also generated a consistent deviation toward for the first two latency bins [first bin: $t(29) = 5.57, p < .001$; second bin: $t(29) = 3.88, p < .001$] and a main effect of latency bin [$F(2, 56) = 8.30, p < .001$]. This effect again decreased when saccade latencies lengthened, as indicated by a post hoc linear contrast ($p < .001$). Of note, for the first two latency bins, the deviation toward was larger with distractors in the blind hemifield than with distractors in the seeing hemifield, although this effect was only marginally significant for the first bin [first bin: $t(29) = 1.85$, $p = .07$; second bin: $t(29) = 2.92, p < .01$].

In summary, Case 2, who did not show a significant effect of distractors on the deviation of saccadic trajectories, showed only a weak deviation toward for short-latency saccades, whereas Case 4, who did have a significant distractor-induced deviation effect, showed a strong deviation toward for both short- and medium-latency saccades.

Comment

This second experiment showed that a global effect could be elicited by blind-field distractors in both patients; however, this deviation was larger relative to the effect in the seeing field in Case 4, the patient who had demonstrated a significant effect of blind-field distractors on saccadic trajectory in Experiment 1, but not in Case 2, the patient without a significant effect in Experiment 1. In line with behavioral studies of the global effect, the deviation decreased with increasing saccade latency (Van der Stigchel & Theeuwes, 2005; Ottes et al., 1985), a finding that was also observed in the only other report of the global effect in a single

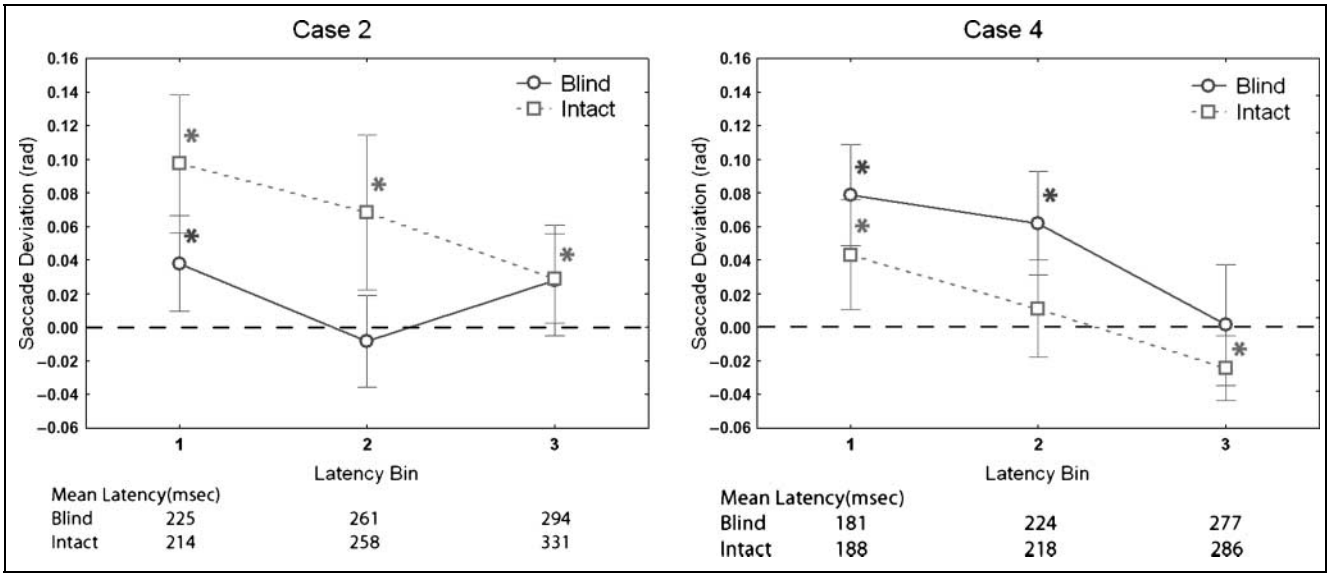


Figure 5. Results of Experiment 2. Mean deviation is shown for eye movements in response to distractors in both the blind and intact part of the visual field. Mean deviations are divided in three latency bins. Both patients showed trajectory deviation toward the distractor that decreased with slower saccadic responses, but the effect of the ''blind'' distractor was stronger in the patient who showed significant trajectory deviation in the first experiment.

hemianopic subject, the blindsight patient G.Y. (Barbur, Forsyth, & Findlay, 1988).

DISCUSSION

We tested the hypothesis that in the absence of retinogeniculostriate processing, residual processing of the retinotectal pathway may still be detected as a modification of saccades to seen targets by irrelevant distractors in the blind hemifield, given the known role of activity in the superior colliculus in generating deviations of saccade trajectory (McPeek et al., 2003). Experiment 1 showed that in two of the five patients with hemifield defects, saccade trajectories to a target on the vertical meridian significantly deviated away from a ''blind'' distractor, and a similar trend was seen in a third patient. Because saccade trajectory modifications are the result of competition between possible saccade targets in the oculomotor system (Van der Stigchel et al., 2006), this indicates that, although unconscious, visual stimuli in the blind visual field of these patients are still treated by the oculomotor system as possible targets for subsequent eye movements. To further test this hypothesis, two of the patients were tested again in Experiment 2, in which the distractor was presented close enough to the target to elicit a global effect in normal subjects (Walker et al., 1997) and in the intact hemifield of these two patients. Both patients showed a deviation toward the distractor that decreased with slower saccadic responses, but the effect was stronger in the patient who showed significant trajectory deviation in Experiment 1.

One of the six patients (Case 6) served as an anatomic control, in that his lesion likely affected both retinogeniculate and retinotectal projections at the optic chiasm. As expected, he showed no deviation of saccadic trajectory away from blind-field distractors. His result also serves as an intersubject control for light scatter. Blindsight-like performance in some experimental conditions may occur because a stimulus projected to the blind field is associated with light diffusing into the seeing field (Cowey, 2004). The fact that Case 6 does not show any deviation of saccadic trajectory from distractors in the blind field suggests that any light scatter in our experiment is insufficient to generate the deviations seen in Cases 3 and 4 on an artifactual basis. Likewise, the lack of a distractor effect in Cases 1 and 2 argues against light scatter as an explanation of the results in Cases 3 and 4, particularly because Case 2 had the smallest hemifield defect, hence, the greatest vulnerability to the effects of light scatter.

The present study is consistent with a previous study of the influence of blind-field distractors on saccades to seen stimuli, which found that latencies to seen targets were increased by a distractor in the blind field (Rafal et al., 1990). Because the superior colliculus plays an important role in the remote distractor effect (Olivier et al., 1999), it was hypothesized that the surviving retinotectal pathways was responsible for the presence of this effect when the distractor was located in a blind hemifield. Further indirect evidence for the role of the superior colliculus in mediating distractor effects on saccades was recently produced by a transcranial magnetic stimulation study (Ro, Shelton, Lee, & Chang, 2004). Stimulation of the primary visual cortex caused a blind spot in which participants could not consciously perceive visual stimuli. Despite this, saccades were delayed when distractors were located within the blind spot. Furthermore, monkeys with removal of the primary visual cortex showed increased reaction times of a reach to a visual target presented in the normal hemifield when a distractor was presented in the blind hemifield (Cowey, Stoerig, & Le Mare, 1998). This effect was only observed when the distractor was presented before the target. These results further suggest that the distractor effect for saccades does not depend upon the retinogeniculostriate pathway.

Of note, we observed deviation of trajectories away from blind-field distractors in the first experiment and saccade deviation towards blind-field distractors in the second experiment. An explanation of these contrasting effects may be found in the population coding theory of Tipper, Howard, and Houghton (2000) and Tipper, Howard, and Jackson (1997). This theory states that possible target objects are represented by a large population of neurons that encode the movement toward each target object as a vector. The strength of a population code is related to the saliency of the corresponding object. When two possible targets are positioned in close proximity, the populations corresponding to these targets will be combined to one mean population, resulting in a target vector with an intermediate location (i.e., the global effect). On the other hand, competition between two widely separated active populations has to be resolved by inhibiting one of them. Inhibition of one population may distort the resulting movement vector to the selected target, represented by the other population. The amount of deviation is related to the inhibition applied to the cancelled vector: the stronger the inhibition, the greater the deviation will be.

Deviations away from a distractor, as observed in the first experiment, are therefore generally associated with a large amount of inhibition (McSorley et al., 2004; Sheliga et al., 1994). Inhibition of the distractor vector shifts the target vector away from the distractor location. In at least two of our patients, a distractor in the blind hemifield was able to generate such inhibition, in the absence of retinogeniculostriate processing.

In the case of the global effect observed in the second experiment, with two targets in close proximity, the weighted average of the mean population is located at an intermediate location. The competition between the two elements is not resolved and the saccade deviates to a position between the target and the distractor. Indeed,

in situations in which the global effect occurs, activity in the superior colliculus has been found to be highest at a location in between the two targets (Glimcher & Sparks, 1993; Van Opstal & Van Gisbergen, 1990). The fact that the global effect is generally observed for only the fastest saccades may be explained by the time-course of visual selection. For shorter latencies, target selection is more stimulus-driven (Van Zoest, Donk, & Theeuwes, 2004), whereas at longer latencies the selection process may be affected more by goal-driven processes such as inhibition. In these situations, the distractor is successfully inhibited and the global effect is eliminated. The present results show that similar mechanisms play a role in the blind visual field of patients with hemifield defects.

A remaining issue is why distractor effects were only found in some patients with putative sparing of the retinotectal pathway and not others. This variability is consistent with the literature, however: Whenever larger series of patients have been examined for blindsight, the general rule has been that it is variably present (Scharli, Harman, & Hogben, 1999; Kasten, Wuest, & Sabel, 1998; Barton & Sharpe, 1997a). Explaining this variability is a challenge. One possibility advanced is that blindsight may depend on how much additional damage to the extrastriate visual cortex exists in a given patient (Weiskrantz, 1990). However, correlations between blindsight and cortical lesion anatomy have proven elusive (Barton & Sharpe, 1997a, 1997b; Magnussen & Mathiesen, 1989; Blythe, Bromley, Kennard, & Ruddock, 1986; Marzi et al., 1986). In our study, blind-field influences did not appear to correlate with lesion size either: One patient (Case 3) with a large posterior cerebral artery infarct showed a distractor effect from the blindfield, whereas another patient with a very focal striate lesion (Case 2) did not. Furthermore, one can question the relevance of additional cortical damage to phenomena that are thought to depend on retinotectal function, such as the effect of distractors on saccades. However, a recent study with diffusion tensor imaging of four hemispherectomized patients found that the two patients in whom blindsight could be demonstrated with a spatial summation paradigm had projections from the superior colliculus to the visual association cortex bilaterally, whereas the two who did not have blindsight had only ipsilateral projections to the cortex (Leh et al., 2006). Whether similar connections between the superior colliculus and the visual association cortex contribute to the saccadic phenomenon we studied is not known.

Other sources of variables are timing parameters, including age at onset (Moore, Rodman, Repp, Gross, & Mezrich, 1996; Payne, Lomber, Macneil, & Cornwell, 1996; Blythe et al., 1987; Perenin & Jeannerod, 1978) (but see, Ptito, Lassonde, Lepore, & Ptito, 1987), duration of lesion, and extent of training if any (Magnussen & Mathiesen, 1989; Zihl & Werth, 1984a, 1984b; Bridgeman & Staggs, 1982; Zihl, 1980; but see, Blythe et al., 1987; Balliet, Blood, & Bach-y-Rita, 1985). These factors could influence the potential for neural plasticity in a given subject, and may be relevant to blindsight if this phenomenon requires modifications to the neural system. Again, these factors do not seem to account for the variability in our data. First, Cases 1 to 4 were all in their fifth decade; second, although one of the two subjects with distractor effects from the blind field had had his defect for 12 years, the other had his for only a month; and third, no subject had received training. Therefore, although our study does confirm that distractor effects on saccadic trajectory and endpoint can occur in patients with damage to the retinogeniculostriate visual pathway, there remain questions regarding what additional factors are required for these effects to manifest themselves in a given patient.

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