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Parietal Damage Dissociates Saccade Planning from Presaccadic Perceptual Facilitation

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A well-known theory in the field of attention today is the premotor theory of attention which suggests that the mechanisms involved in eye movements are the same as those for spatial attention shifts. We tested a parietal damaged patient with unilateral optic ataxia and 4 controls on a dual saccade/attentional task and show a dissociation between saccadic eye movements and presaccadic perceptual enhancement at the saccade goal. Remarkably, though the patient was able to make the appropriate saccades to the left, impaired visual field (undistinguishable from saccades to his right, intact visual field), he was unable to discriminate the letter at the saccade goal (whereas his performance was like controls for letter discrimination in his right visual field). This suggests that saccade planning and presaccadic perceptual facilitation are separable—planning a saccade to a location does not necessitate that the processing of this location is enhanced. Based on these results, we suggest that the parietal cortex is necessary for the coupling between saccade planning and presaccadic perceptual facilitation.

Keywords: attention, optic ataxia, parietal cortex, saccades

Introduction

Eye movements and attention are very closely related. Normally, when we want to pay attention to an object, we move our eyes so as to foveate on it (i.e., we shift our gaze) and this is known as overt attention. However, it is also possible to shift attention without moving the eyes. This process is known as covert attention. Both overt and covert attention have the same objective: to enhance a location in space for further processing, either by bringing the object into higher-precision processing pathways associated with central vision (Anstis 1998), or by enhancing the efficiency of the processing in peripheral vision selectively at this specific location (Bushnell et al. 1981).

According to the premotor theory of attention (Rizzolatti 1983; Rizzolatti et al. 1987, 1994), a command to shift attention is the same as a command to shift gaze. In both cases, attention is shifted (covertly or overtly). This theory suggests that preparing a saccade to a peripheral target location involves the same process as orienting selective attention to this peripheral location, except that this process is then followed by the motor execution. Inversely, any saccade will be associated with a spatially specific enhancement of processing at the location of the saccade goal, i.e. saccades to a spatial location cannot occur without a previous shift of attention to the location (Hoffman and Subramaniam 1995; Kowler et al. 1995; Goldberg et al. 2006). In a behavioral study, Deubel and Schneider (1996) tested subjects' ability to discriminate letter symbols at locations around or at the saccade target location, before saccadic execution. They found that letter discrimination was best when it was in the same position as the saccade goal. The ability to discriminate a letter at any location other than the goal of the saccade decreased to chance level. By showing that when a saccade is executed toward a peripheral target, it is preceded by a phenomenon of "presaccadic facilitation" of letter discrimination specifically at this precise peripheral location, this paradigm confirmed the spatial and temporal relationship between saccades and attention: attention is oriented before saccadic execution toward the specific location of the saccade goal, and may thus share common mechanisms with saccadic preparation (Schneider and Deubel 2002).

Central to this theory is the finding that the same areas in the brain that are involved in gaze or eye movements are also involved in attention shifts (Ignashchenkova et al. 2004; Moore and Fallah 2004; Goldberg et al. 2006). One of the key areas shown to be involved in both attention and saccadic eye movements is the lateral intraparietal area (LIP) in the monkey posterior parietal cortex (Goldberg et al. 2006). We therefore tested involvement of the parietal cortex in the coupling between presaccadic attentional shifts and eye movements by observing the performance of a patient with unilateral optic ataxia due to damage to the right posterior parietal cortex. If saccade planning and attentional shifts involve the same mechanisms, we should find a similar pattern of performance in target discrimination and saccade execution to the damaged visual field. On the other hand, a dissociation between saccadic performance and target discrimination performance could suggest that separate neural substrates govern attention and saccade planning, but those processes could be coupled and occur simultaneously. A second possibility could be that the parietal cortex mediates the coupling between a network of areas involved in saccade planning and presaccadic facilitation, such as frontal and temporal areas. In normal behavior, it is impossible to distinguish between a coupling and a common substrate, however the impaired region/pathways in this patient may provide valuable insight into these mechanisms.

We tested the patient and 4 control subjects on a simplified version of the task used by Deubel and Schneider (1996). In our task the saccade was always directed to the same location (at 10 visual degrees of eccentricity) leftward or rightward. Subjects were then required to discriminate letters presented briefly during the saccade latency either at the saccade goal or at nonsaccade goal locations 3° closer or further away in the same visual field. This task allowed us to test the presence of presaccadic facilitation, 1) at the location of the saccade goal and 2) at surrounding locations, testing for potential spatial distortions in attentional shifts. Spatial distortions have been

observed in studies on reaching in optic ataxia where patients consistently misreach to the location of a perceived target in peripheral vision, suggesting a bias in reach space (Revol et al. 2003; Khan et al. 2005). This spatial distortion may also affect attentional shifts but not saccadic eye movements or vice versa (if these functions are dissociable).

Methods

Subjects

Patient CF is a right-handed 30-year-old male patient who, in 2003, suffered from a cerebral angeitis initially presenting with headache without fever. One week later, the patient was moderately confused with signs of Balint's syndrome. Cerebral angiography then showed vasospasm in the left middle cerebral artery territory and in the right posterior cerebral artery territory. MRI scans showed a posterior watershed infarct (ischemic lesion of junctional territories) resulting in distributed and asymmetrical bilateral lesions of the occipito-parietal region (Brodmann's areas 18, 19, 7, 5, and 2) with a minute extension to the centrum semiovale. None of the laboratory studies provided a clear etiology either in terms of inflammatory or autoimmune disease. During the following months, simultagnosia and neglect rapidly disappeared leaving the patient with a stable and isolated unilateral optic ataxia predominantly in his left visual field (LVF), thought to be the consequence of larger damage in the right hemisphere from both BA 7 lesions and a parieto-frontal disconnection from intrahemispheric fibers lesions—(Fig. 1*A*, see also Khan et al. 2005). He did not exhibit any purely motor, somatosensory or visual deficits (visual fields and visual perception were preserved including pattern recognition and color perception).

Four neurologically intact control subjects (age range: 25–35) also participated in the experiment. All subjects (including the patient) gave informed consent to participate in the experiment according to the French law (4 March 2002) on human subjects' rights.

Apparatus

Subjects sat in front of a specifically designed experimental device composed of a high speed CRT screen (frequency: 160 Hz, 21"), coupled with a real-time stimuli presentation device (Visual Stimulus Generator ViSaGe, Cambridge Research System, Rochester, UK). A High Speed Video Eyetracker (Cambridge Research System) fixed on a head and chin rest, registered the movements of the right eye (frequency: 250 Hz, resolution: 0.05°) by means of an infra-red camera. The subjects' eyes were at a distance of 57 cm from the screen. The Eyetracker, the ViSaGe and a button press (for the letter discrimination response) were synchronized by a program interface developed in our laboratory.

Procedure

We designed a simplified version of the original task by Deubel and Schneider (1996) as shown in Figure 1*B*. Each trial began with a fixation cross illuminated for 2 s. Next, 5 target locations (of which the central 3 on each side were surrounded by colored ovals) were presented as figure "8" symbols on both sides of the central visual fixation for a randomized period between 1.8 and 2.2 ms (average time = 2 s). A central cue (whose color corresponded to the center circled location) in the form of an arrow was then presented for 150 ms, which indicated the direction toward which the subjects were to make a saccade. Subjects were asked to always saccade to the central position either to the left or the right visual field (RVF) as indicated by the arrow (10 visual degrees of eccentricity—position 5). A target letter (E or ∃) was then flashed at this location during 60% of the trials, and at the other 2 circled locations for 20% of the trials each. The target letters were always flashed in the same visual field to which the subjects had to make a saccade. Symbols at the other locations also changed at the same time as the target letter, to either figure "2" or figure "5." The symbols were flashed for 250 ms and then subsequently masked by the reappearance of the "8" symbols. At the end of the trial, subjects were asked to respond using a button press to indicate whether the "E" faced left or right (forced choice).

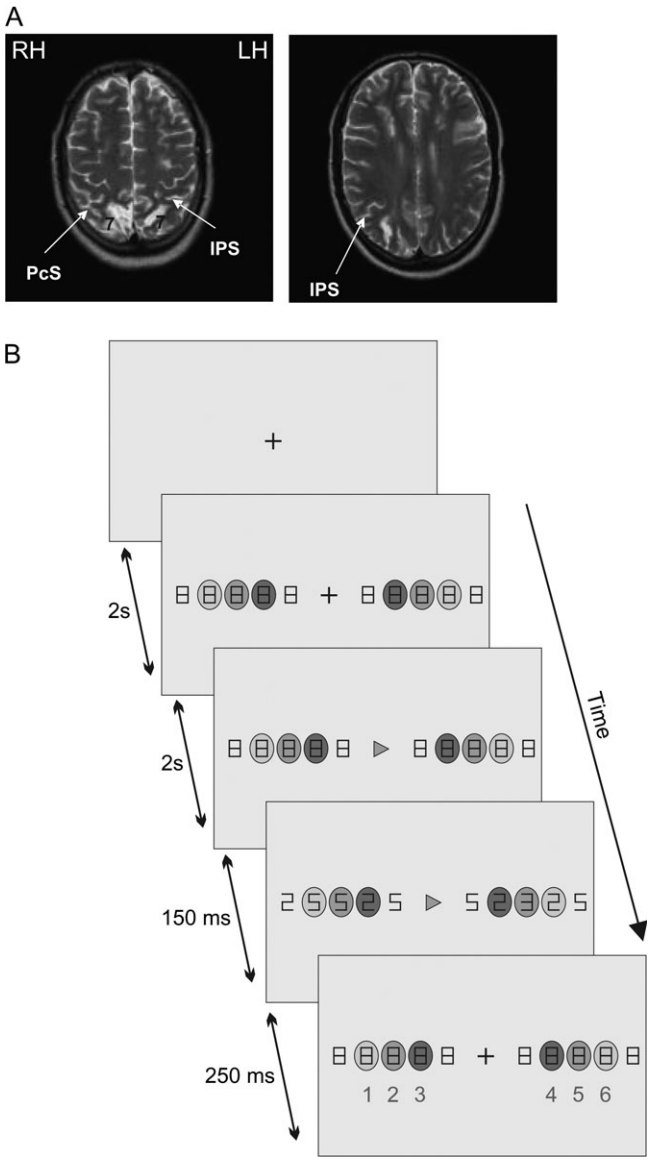

Figure 1. MRI slices of patient CF and task setup. (*A*) T2 scans for patient CF. The white areas at the bottom of the scans show asymmetrical damage mostly in the right hemisphere in the posterior parietal lobes (area 7 labeled in black). There is also some slight damage in the left premotor cortex. IPS = intraparietal sulcus, PcS = postcentral sulcus. (*B*) Task setup. Each trial began with the presentation of a central fixation cross. After 2 s, 6 of 10 target locations were presented in different colors on either side of the fixation spot (labeled positions 1–6 as shown in the last panel—these did not appear on the screen during the task). Within each target location, the figure 8 was displayed. After another 2 s, the central fixation cross changed into an arrow indicating the direction of the saccade. Subjects were instructed to always make saccades to the middle circle location (either position 2 or 5). In this case, the arrow indicated to the subjects to make a saccade to the right (position 5). Subjects were instructed to make a saccade as soon as they saw the arrow. After 150 ms, a letter was briefly presented at one of the 3 colored locations (gray ovals) in the direction of the arrow, either an E or ∃, whereas all other locations changed to figures "2" or "5". In this case, the letter was presented at the saccade goal. After another 250 ms, all locations changed back into a figure "8." After the saccade, subjects were required to report whether the letter was an E or an inverted E by a forced-choice button press.

Data Analysis

We recorded a total of 264 trials for patient CF and 667 trials for controls. We analyzed saccade movements offline and removed all trials in which subjects made erroneous saccades, for example, in the wrong direction, too soon (<80 ms) or too late (>1 s). This resulted in

200 trials for patient CF and 584 trials for controls. In addition, we noticed that overall, control subjects had shorter latencies than those of patient CF (see Results section). The timing of the experimental sequence was designed with patient CF in mind and was determined specifically to ensure that the target presentation occurred right before and during the saccade but was masked at the time at which the saccade landed on the target. Because the controls made faster saccades, to ensure equality in exposure time to the target between CF and the control subjects, we calculated the difference between saccade offset and target offset for all subjects across both experiments. We then removed all trials for controls where the difference was greater than that for CF. On average, CF landed on the target 2.8 ms after the target was masked with the earliest time at 59 ms before target offset. There were no differences between the leftward and rightward saccades (see Results section for latency analysis). We set this earliest time as the cut-off for our controls. With this cut-off, controls landed on average 21 ms before target offset. For subsequent analyses, we used the remaining 371 trials for the controls. We performed an additional analysis separating the data into trials in which the saccade landed on the target before target offset and those in which the saccade landed on the target after target offset. We found no differences between the 2 groups and all subsequent analyses include both groups. Further, as reported later in the results section, these differences in the average values (saccade offset-target offset) did not result in any performance discrimination differences between CF and the controls in trials toward his right intact visual field.

Results

To remind the reader, the order of events during a trial was as follows: subjects were presented with an arrow indicating the direction of the saccade (Fig. 1*B*). Although they were planning this saccade, the letter was displayed for a short interval at the saccade target (in 60% of the trials). After the saccade was made to the target location, subjects discriminated the letter they were presented. We compared performance for target discrimination to that for saccade execution.

Target Discrimination Performance

Figure 2*A* depicts the percentage of correct discriminations for the letter presented at one of the 6 possible locations shown on the *x*-axis. As can be seen in the figure, when the target was presented in the rightward 10° location (position 5)—the saccade goal, performance was very similar for both CF and the controls and was significantly different from chance level (CF = 80.9%, $\chi^2(1) = 21.04$, $P < 0.001$, Controls = 76.9%, $\chi^2(1) = 15.461$, $P < 0.001$). This can be seen in the gray region in the RVF in the figure. In contrast, when the discrimination target was at the saccade goal (position 2, 10°) in his impaired LVF, his performance was at chance level (46.2%, $\chi^2(1) = 0.296$, $P > 0.05$) and very different from the controls (average controls = 76.8%, $\chi^2(1) = 15.63$, $P < 0.001$).

We also tested for a spatial distortion in attention. If there is some kind of distortion in space for presaccadic attentional shifts, we may see an increase in performance in locations closer or further away from the saccade target within the damaged visual field. We found however that patient CF's performance was at chance for all letter locations (LVF). This is incompatible with the hypothesis of a distortion in attentional space; letter discrimination performance did not improve for either the location closer to fixation (position 3, 7°) or the location further past the saccade goal (position 1, 13°).

Interestingly, letter discrimination performance in the RVF for patient CF for the target closest from fixation (position 4) was also significantly different from chance level (73.9%, $\chi^2(1) = 12.13$, $P < 0.001$) whereas the controls performed at chance (48.8%, $\chi^2(1) = 0.03$, $P > 0.05$). It is unclear why this is the case, but it suggests a wider focus of attention compared with controls rather than a distortion in attentional space, as performance was also above chance for the location of the saccade goal. This may reflect a bias in attention toward the intact visual field.

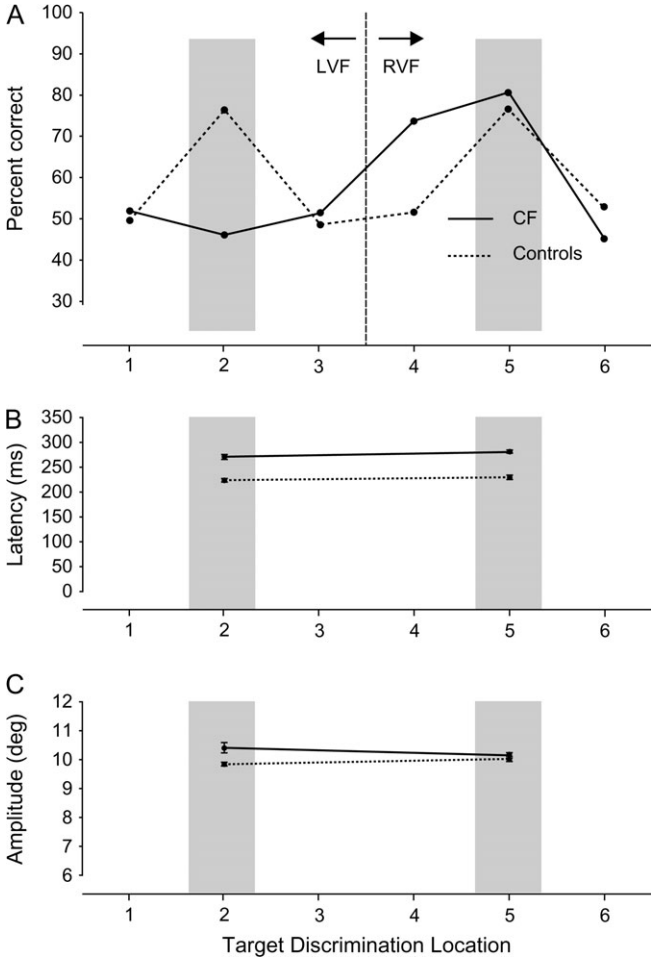

Figure 2. Experimental results. (*A*) Target discrimination. Percentage of correctly discriminated targets at the 6 locations (from leftmost to rightmost—positions 1–6, as shown in *B*) for patient CF (solid lines) and controls (dotted lines). The dashed vertical line partitions the LVF and RVF fields. The gray regions on either side schematically depict the location to which the saccade was made. (*B*) Saccade latencies. Saccade latencies in ms are plotted for patient CF (solid lines) and controls (dotted lines). (*C*) Saccade amplitudes. Saccade amplitudes are plotted in degrees for both the patient and the controls. Error bars are SEM.

Saccade Latency

Figure 2*B* depicts latencies for saccades performed by both patient CF and controls. Overall, patient CF's saccade latencies were significantly longer than the controls latencies (CF = 274.8 ms, controls = 226.5 ms, $F_{1,497} = 108.5$, $P < 0.001$). However, CF showed no differences between saccade latencies toward the left compared with the RVF (LVF = 269.5 ms, RVF = 281.1 ms, $F_{1,198} = 3.5$, $P > 0.05$). This was also the case for the controls (LVF = 223.5 ms, RVF = 229.6 ms, $F_{1,299} = 0.337$, $P > 0.05$). These results show that unlike target discrimination, saccade latency was indistinguishable between the left or RVFs for patient CF and did not differ from controls in this manner.

Saccade Amplitude

It could be the case that patient CF was making less accurate saccades to the LVF compared with the RVF which would explain the differences in target discrimination performance. We therefore measured saccade amplitude for left and right saccade target locations (Fig. 2*C*). We found no significant differences between saccade amplitudes for patient CF between leftward and rightward saccades (LVF = 10.11°, RVF = 9.84°, $F_{1,198}$ = 1.69, $P > 0.05$). Similarly, we found no significant differences between left and right saccade amplitudes in controls (LVF = 9.54°, RVF = 9.72°, $F_{1,299}$ = 2.565, $P > 0.05$). In addition, we also found no differences between the peak velocities for leftward vs. rightward saccades for patient CF ($F_{1,198}$ = 0.96, $P > 0.05$) and as well no differences between the main sequences (peak velocity vs. amplitude) for either direction (CF: leftward saccade slope = 95.04 ± 4.54(CI), rightward saccade slope = 103.95 ± 10.64, controls; leftward slope = 67.3 ± 20.65, rightward saccade slope = 83.27 ± 14.1).

As an additional analysis, we compared saccadic endpoints for leftward vs. rightward saccades for the patient. We found that the patient's leftward saccades had larger scatter in the *x* (horizontal) axis (leftward saccade *x* standard deviation = 1.8°, leftward saccade *y* (vertical) standard deviation = 0.51°, rightward saccade *x* standard deviation = 0.82°, rightward saccade *y* standard deviation = 0.49°, controls: leftward saccade *x* standard deviation = 0.82°, leftward saccade y (vertical) standard deviation = 1.01°, rightward saccade *x* standard deviation = 1.08°, rightward saccade *y* standard deviation = 0.87°). We therefore investigated CF's performance comparing whether saccade endpoint was correlated with target discrimination performance. We grouped saccade endpoints for both directions into 2 groups: those that landed within 1 (horizontal) degree of the center of the target and those that landed outside of this range. We then calculated target discrimination for this target separately for the 2 saccade endpoint groups. We found no differences between performance for either endpoint group (leftward saccades: within range group [*N* = 24] = 45.83%, outside group [28] = 46.63%, rightward saccades: within group [38] = 81.58%, outside group [7] = 77.78%). These results are in agreement with previous findings regarding this specific task which show that saccade endpoints are somewhat independent of target discrimination (Deubel and Schneider 1996).

Discussion

Taken together, our findings show that patient CF was unable to discriminate a letter at the saccade goal in his damaged visual field as opposed to the intact visual field where his performance was normal compared with controls. This deficit in letter discrimination in the damaged visual field corresponded to the damage to his right hemisphere. However, the damage did not affect saccade generation.

One explanation for our observations is a functional coupling between saccade preparation and attention rather than a common substrate. This is supported by neurophysiological findings within the posterior parietal cortex suggesting that this area is specifically involved in attention rather than saccade planning (Goldberg et al. 2002; Bisley and Goldberg 2003; Constantinidis and Steinmetz 2005). For example, Constantinidis and Steinmetz (2005) investigated the role of the parietal cortex in attention processing and found that neuronal activity was modulated by the presence of salient stimuli even when the stimuli were behaviorally irrelevant (no planned saccade). Goldberg et al. (2006) showed that activity in LIP accurately reflected the locus of attention, shifting to the presence of an irrelevant distractor before returning to the saccade goal, rather than continuously reflecting the location of the saccade goal. These findings imply that this area is involved in attention but not necessarily eye movements (Bisley and Goldberg 2003). The present results suggest that shifting attention and preparing a saccade involve separate neural networks and that accurate saccade can be generated without a preceding attentional shift to the goal location.

In the paradigm of Deubel and Schneider (1996), presaccadic orienting of attention at the peripheral target location is revealed by an improvement in accuracy in a perceptual discrimination task, and not by an effect on reaction time, as is often used to measure the location of spatial attention (e.g., Posner and Cohen 1984). Therefore, our findings could be viewed more specifically as reflecting different substrates for saccade planning and attentional facilitation, which may still involve common processes of target selection (spatial attention, Schneider and Deubel 2002). The advance shift of attention to the saccade goal may exist to aid the ventral processing of information in the visual periphery where the eyes are about to be directed (Findlay and Gilchrist 2003). Such coupling between saccade-related areas and perceptual ventral areas has been recently demonstrated by Moore and Armstrong (2003) who stimulated the Frontal Eye Field (FEF) in the monkey with a below-saccade-threshold current and simultaneously measured the activity of neurons in area V4, an area known to process the color and form of objects (as required by our task). They found that activity increased in the neurons in V4 when the motor fields of FEF and receptive fields of area V4 overlapped. In addition, discrimination of target luminance change improved at the same location (Moore and Fallah 2004). Importantly, a recent transcranial magnetic stimulation study on the human FEF during the Deubel and Schneider (1996) paradigm confirmed the role of the FEF in presaccadic facilitation (Neggers et al. 2007). Taken together, these studies strongly suggest that presaccadic facilitation involves the FEF, which direct attention in visual areas involved in perception. However, the functional pathways between the FEF and perceptual visual areas within the ventral stream remain unclear (Moore et al. 2003; Neggers et al. 2007). The FEF have been shown to have direct connections with areas such as V4 (Schall et al. 1995) and the anatomical location of the lesion may have directly disrupted this pathway. However, there is some evidence that this pathway is not likely to be used (Moore and Armstrong 2003). Instead, the FEF may send signals through a visuospatial network involving the parietal cortex (Moore and Armstrong 2003; Neggers et al. 2007). Indeed, neuroanatomical evidence shows strong interconnections between LIP and FEF (Schall et al. 1995; Stanton et al. 1995). Although the present work cannot speak to the specific pathway that is disrupted, it provides evidence of a disruption of the link between saccade execution and the enhancement of perceptual processes within the ventral visual stream.

In summary, we show that damage to the posterior parietal cortex dissociates the ability to make accurate saccades to a spatial location and presaccadic facilitation at that location. These findings thus highlight the involvement of distinct brain regions for presaccadic facilitation and voluntary saccadic execution, with the posterior parietal cortex as a crucial

interface between the motor and perceptual systems in mediating presaccadic facilitation.

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Notes

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