Lesion of Primary Visual Cortex in Monkey Impairs the Inhibitory but Not the Facilitatory Cueing Effect on Saccade

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Abstract

■ Prior visual stimulus presentation induces immediate facilitation and subsequent inhibition of orienting to an ensuing target at the same location. Recent studies revealed that the superior colliculus (SC) is involved in these facilitatory and inhibitory cueing effects on saccade; however, as the SC receives inputs both directly from the retina (retino-tectal pathway) and indirectly from visual cortices (geniculostriate pathway), it is unclear which visual pathway contributes to the effects. We investigated this issue using monkeys with lesions in the primary visual cortex (V1), thus depriving the SC of the geniculostriate pathway and leaving the retino-tectal pathway intact. We found that the inhibitory cueing effect was selectively impaired and the facilitatory cueing effect was spared after V1 lesions. The results suggest that the geniculostriate and the retino-tectal pathways are differentially involved in the generation of cueing effects on saccade: The former is critically involved in the inhibitory effect whereas the latter alone can induce the facilitatory effect. The results provide the first direct evidence for the involvement of the geniculostriate pathway in the inhibitory cueing effect and further imply that the more recent evolution of the geniculostriate pathway in higher mammals improves the efficiency of visual search by inhibiting orienting to a previously attended location. ■

INTRODUCTION

Orienting is one of the most important behaviors of organisms and is often affected by past experience with the visual environment without any cognitive control. A spatially noninformative visual cue presented in the periphery modulates subsequent orienting to a target at the same location (Maylor, 1985). The modulation of orienting behavior significantly depends on the temporal relation between the cue and the target. If the target is presented immediately after the cue, usually less than a few hundred milliseconds, the orienting behavior is facilitated. However, if the time between the cue and the target is longer than approximately 100 msec (monkeys) or approximately 300 msec (humans), orienting is inhibited. Although recent works have suggested that these facilitatory and inhibitory cueing effects are important in visual search (Itti & Koch, 2001; Klein, 1988), the neural mechanisms underlying them are still unclear.

The superior colliculus (SC) is known to be a center of orienting behavior, converting sensory information into movement signals for the eyes and the head (Sparks, 1986; Wurtz & Albano, 1980). Several studies suggested that the SC is important for the cueing effects on saccade. Patients with damage to the SC showed reduced or no inhibitory cueing effect (Sereno, Briand, Amador, & Szapiel, 2006; Sapir, Soroker, Berger, & Henik, 1999; Posner, Rafal, Choate, & Vaughan, 1985), and neurophysiological studies using nonhuman primates revealed that neuronal activities in the SC are correlated with the cueing effects on saccadic RT (SRT) (Fecteau & Munoz, 2005; Dorris, Klein, Everling, & Munoz, 2002). However, these results do not necessarily mean that the SC is the original source of the cueing effects. As the SC receives inputs from various brain regions (Fries, 1984), it is possible that the cueing effects emerge initially in other brain regions and are relayed through the SC. Thus, it is important to know the functions of various inputs to the SC to understand the neural mechanisms underlying the cueing effects.

The visual inputs to the SC can be largely divided into two visual pathways. One is a direct input from the retina to the superficial layer of the SC (retino-tectal pathway). The other is an indirect input from the cerebral cortex (geniculostriate pathway), mostly through the primary visual cortex (V1). These two visual pathways are thought to be functionally different. A lesion study in hamsters showed that the retino-tectal pathway is concerned with locating and orienting, whereas the geniculostriate pathway is concerned with identification and recognition (Schneider, 1969). A similar functional dichotomy is also observed in human; patients with damage to V1 cannot report the presence of visual stimuli in their scotoma but still retain their ability to localize the stimulus (Sanders, Warrington, Marshall, & Wieskrantz, 1974; Poppel, Held,

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& Frost, 1973). How then are these two visual pathways involved in the cueing effects on saccade? It was reported that a hemianopic patient with damage to V1 still exhibited the inhibitory cueing effect, suggesting that the geniculostriate pathway is not necessary for this effect (Danziger, Fendrich, & Rafal, 1997). However, they also reported that another hemianopic patient failed to show the inhibitory cueing effect, suggesting that possible differences in the extent of lesion, recovery, or adaptive strategy among patients might influence the cueing effect. To clarify the functional role of the geniculostriate pathway in the cueing effect, better controlled studies are required.

In the present study, we investigated the neural mechanisms of the cueing effects using monkeys with controlled lesions. We made unilateral lesions of V1 in two monkeys, thus eliminating the geniculostriate pathway from visual processing in the visual field contralateral to the lesion (affected field) (Figure 1A). In contrast, both the geniculostriate and the retino-tectal pathways remain intact in the visual field ipsilateral to the lesion (normal field). Lesions in V1 lead to severe deficits in visual perception, although monkeys can still perform oculomotor tasks via the remaining visual pathways (Yoshida, Takaura, Kato, Ikeda, & Isa, 2008; Segraves et al., 1987; Mohler & Wurtz, 1977). We tested whether the monkeys with V1 lesions showed cueing effects on saccades using a saccadic cueing task modified for monkeys (Fecteau, Bell, & Munoz, 2004; Dorris et al., 2002).

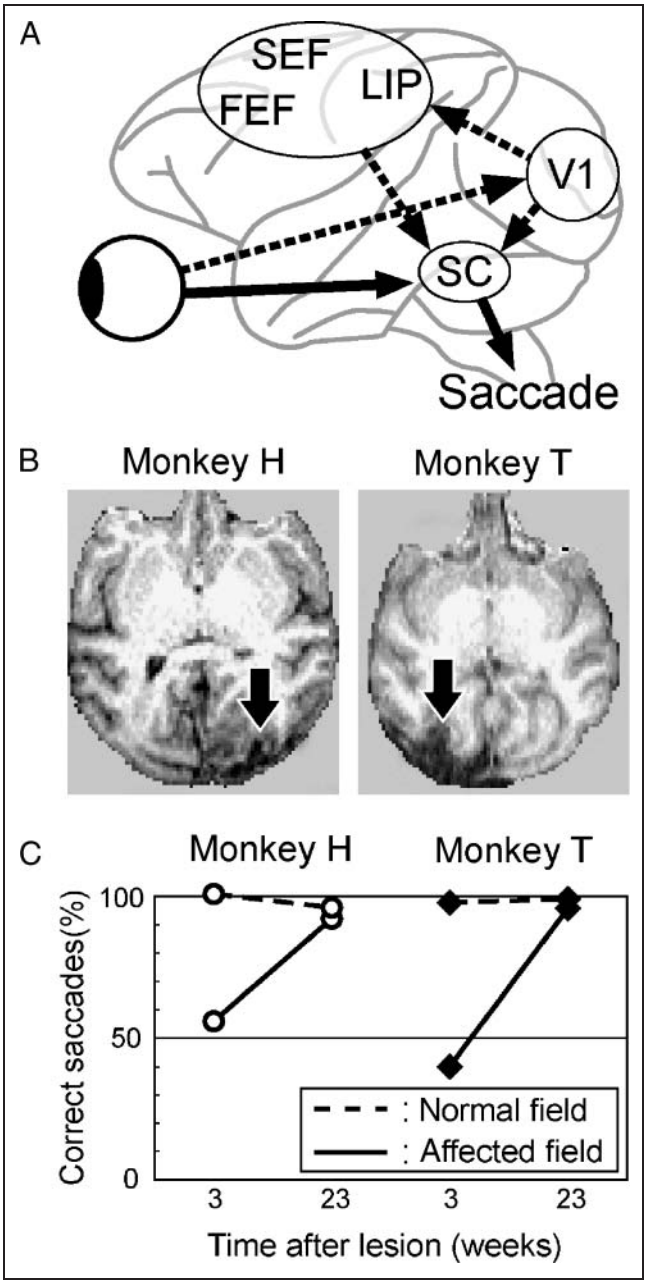

Figure 1. V1 lesion. (A) Schematic drawing of geniculostriate and retino-tectal pathways. Solid arrows represent the retino-tectal pathway mediated by a direct retino-tectal projection. Dashed arrows represent the geniculostriate pathway via primary visual cortex (V1) to the lateral intraparietal area (LIP), FEF, supplementary eye field (SEP), and other related areas that would be impaired after a V1 lesion. (B) Magnetic resonance images 1 week after V1 lesions (arrows). Left: Monkey H with right V1 lesion. Right: Monkey T with left V1 lesion. (C) Recovery after lesion. The rates for correctly discriminating two target locations presented in the affected field (solid line) and the normal field (dashed line) at 3 and 23 weeks after lesion are shown. Possible target locations were set 30° either up or down from the horizontal meridian and 10° from the fixation spot as in experiment 2.

METHODS

Animals and Surgery

Two Japanese monkeys (*Macaca fuscata*), Monkey T (female, body weight = 6.5 kg) and Monkey H (male, body weight = 8.0 kg), were used. All surgical and experimental procedures were performed in accordance with the *National Institutes of Health Guidelines for the Care and Use of Laboratory Animals and Basic Policies for the Conduct of Animal Experiments in Research Institutions* by MEXT, Japan, and approved by the Committee for Animal Experiments at the National Institutes of Natural Sciences, Japan. All surgeries were performed under aseptic conditions. The monkeys were anesthetized with xylazine hydrochloride (2 mg/kg) and ketamine hydrochloride (5 mg/kg), and anesthesia was maintained with isoflurane (1.0–1.5%). An eye coil was implanted in one eye for monitoring eye position, and a head holder was implanted to stabilize the head position. After several months of training (described below), V1 was unilaterally removed, under anesthesia, by aspiration using a metal suction tube as previously described (Yoshida et al., 2008). After the surgery, the monkeys were given penicillin G (80,000 U/day) and cefmetazole (0.5 g/day) as antibiotics and dexamethasone sodium phosphate (0.5 mg/kg) to minimize brain edema. Magnetic resonance images of the brains were acquired 1 week after lesion. The acquisition

sequence was MPRAGE-3D, with a voxel size of 0.82 × 0.82 × 0.81 mm.

Training before and after Lesion

A visually guided saccade task was used for training before and after the lesion. The possible target locations were widely distributed across the visual field (eccentricity: 10–20°; direction: between 60° up and down from the horizontal meridian). By changing the luminance contrast of the target, we confirmed that visual sensitivity was significantly impaired in the contralesional visual field (Yoshida et al., 2008). To exclude the possible contribution of light scattering in the affected field, we tested whether the monkeys could make a saccade to the target presented in their natural blind spot using the visually guided saccade task in a monocular condition (Moore, Rodman, Repp, & Gross, 1995; Campion, Latto, & Smith, 1983). The size of the target used in this test was the same or larger than that used in the subsequent experiments. Monkeys could not respond to the target in the blind spot, indicating that saccades to the target in the affected field were not due to light scattering.

Behavioral Task

The monkey sat in a primate chair in a sound-attenuated room with its head fixed. Eye movements were recorded using a magnetic search coil (MEL-25; Enzanshi Kogyo, Japan; Robinson, 1963), with a resolution of 0.1° of visual field. Eye positions were sampled at 1 kHz. Visual stimuli were presented on a CRT monitor (21 in.; Mitsubishi, Japan) positioned 28 cm in front of the eyes. The visual display and the data storage were controlled by computers running a real-time data acquisition system (Reflective computing; Tempo for Windows). Each trial of the saccadic cueing task started with the appearance of a central fixation point, on which the monkey had to fixate. After 700 msec of fixation, a task-irrelevant cue stimulus was presented briefly (50 msec). The central fixation point remained present for various delay periods after the cue appearance (SOAs; 100, 300, or 500 msec), and the target stimulus was presented concurrently with fixation disappearance, either at the same location as the cue (same condition) or at a different location (different condition). The monkey received a juice reward for making a correct saccade to the target within 400 msec and fixating on it for an additional 200 msec. The fixation point, cue, and target stimulus were small spots of red light (0.7° in diameter) with a luminance contrast of 11 (Weber Contrast) against the background (1 cd/m^2). The cue and the target were presented at one of two possible locations fixed in a given block of 60–100 successful trials. In the first experiment, the stimulus locations were to the right and left of the fixation point (eccentricity: 10°). In the second experiment, they were positioned in one hemifield, 30° up and down from the horizontal meridian (eccentricity: 10°). The target location and the cue condition (same or different) were pseudorandomized independently across trials such that every subblock of four trials contained two trials for each of the two locations and the two conditions. If the monkey did not maintain fixation within a window around the fixation point (usually 3° radius), the trial was aborted and the monkey did not receive the juice reward. We also set a window around the target (5° radius), and if the monkey's gaze failed to reach this window within 150 msec after initiating the saccade, the trial was aborted.

Data Analysis

The trials in which the monkey failed to acquire or maintain fixation accounted for 16% (Monkey T) and 22% (Monkey H) of total trials in the experiment. The eye velocity was calculated by the two-point central difference algorithm (Bahill, Kallman, & Lieberman, 1982). Saccades were detected when the eye velocity of the polar component exceeded 200 deg/sec after fixation disappearance. The onset time of the saccade was then defined as the time point at which the velocity exceeded 30 deg/sec, before reaching 200 deg/sec. The SRT was the time between fixation offset and saccade onset. The trials in which SRTs were less than 60 msec were considered as anticipatory saccades and excluded from the analysis. The end point of the saccade was defined as the spatial position at which the velocity of the saccade declined 30 deg/sec after the saccade onset. Saccade end-point error was defined by the distance between saccade end point and target, and trials were excluded from the following analysis if the saccade end-point error was >5°. The cueing effect on SRT was defined as the difference in mean SRTs between two target conditions (difference condition minus same condition). MATLAB and MATLAB statistical toolbox (MathWorks, Natick, MA) were used for off-line data analysis. Comparisons of SRTs were made using Student's t tests with Bonferroni corrections for six pairs (three SRTs for two monkeys) after confirming the normality of the distribution by the Kolmogorov–Smirnov test ($p < .05$).

RESULTS

Three weeks after unilateral V1 lesions (Figure 1B), the monkeys could not respond correctly to stimuli presented in the visual field contralateral to the lesion. However, after approximately 5 months of training, they were again able to make saccades to such stimuli (Figure 1C), as reported in previous studies (Yoshida et al., 2008; Segraves et al., 1987; Mohler & Wurtz, 1977). We then evaluated the spatial cueing effects on saccades using the saccadic cueing task (Figure 2).

In our initial experiment, we presented the cue and the target bilaterally, either to the left or to the right of central fixation, that is, either in the normal field (ipsilateral to the lesion) or in the affected field (contralateral to the lesion). SRTs to the targets in the normal and affected fields were analyzed separately, and the cueing effects

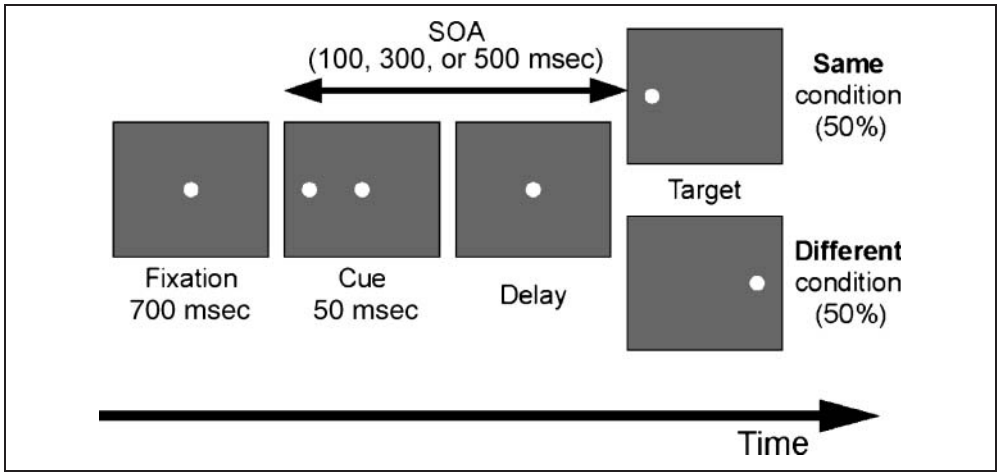

Figure 2. Experimental design. Time course of events in the saccadic cueing task. Monkeys had to make correct saccades to the target to obtain juice reward. The two possible locations for the cue and the target were fixed in each block of 60–100 successful trials.

were compared (Figure 3). When the target was presented in the normal field, both monkeys displayed cueing effects similar to those described in previous reports (Fecteau & Munoz, 2005; Dorris et al., 2002). SRTs tended to be longer in the same condition than that in the different condition (Figure 3A and C). On the other hand, when the target was presented in the affected field, there were no significant elongations of SRT to the target in the same condition, and both monkeys showed significantly shorter SRTs at 100 msec SOA (Figure 3B and D). Multiple comparisons between the same and the different cue conditions for each SOA and visual field confirmed these results (Figure 3A–D). Figure 3E and F shows the cueing effect on SRT defined by the difference between SRT in the same and different conditions (positive and negative values indicate facilitation and inhibition of reaction by the cue presentation, respectively). Although there were some differences in the effects between the two monkeys, such as shorter SRTs in Monkey H in the same condition at the 100-msec SOA, the cueing effects were significantly different between the two fields in both monkeys, suggesting the functional involvement of V1 in the cueing effects on saccades.

However, as the sensitivity to visual stimuli was altered after the V1 lesion (Isa & Yoshida, 2009; Yoshida et al., 2008), the difference between the same and the different cue conditions may have been due to differences in visual sensitivity to the cue, not to the spatial relation between the cue and the target. To exclude this possibility, a second experiment was conducted, in which the two possible spatial locations were matched by presenting the cue and the target unilaterally, only in one hemifield in a given block of trials (Figure 4). Two possible cue-target locations were set in each hemifield 30° up or down relative to the fixation point at 10° eccentricity. Thus, in a given block, both the cue and the target were always presented either in the normal field or in the affected field. When targets and cues were presented in the normal field, both monkeys showed longer SRTs in the same condition than that in the different condition (Figure 4A and C). On the other hand, when presented in the affected field, there was no elongation but rather a significant reduction in SRTs to the target in the same condition compared with the different condition (Figure 4B and D). As in the first experiment, we found a significant difference in the cueing effect between the normal and the affected field: an inhibitory cueing effect in the normal field and a facilitatory cueing effect in the affected field (Figure 4E, F). These results strongly suggest that V1 is important in the inhibitory cueing effect.

These experiments were conducted during two different periods of recovery in the two monkeys (Monkey H: 6 months after lesion; Monkey T: 40 months after lesion) that exhibited some differences in saccadic behavior. Saccades became less accurate after V1 lesions in both monkeys, but Monkey H showed a more severe deficit than Monkey T (Figure 5A). There were also differences in peak saccade velocities: Monkey T made faster saccades in the affected field, and Monkey H made faster saccades in the normal field (Figure 5B). There were also some differences in the time courses of the cueing effects in the two monkeys. For example, there was no inhibitory cueing effect in Monkey H at 100 msec SOA (Figure 4E), and there was a longer facilitatory cueing effect in Monkey T at 500 msec SOA (Figure 4F). These behavioral differences might have been caused by differences in structural or strategic changes during the course of recovery and adaptation after the V1 lesions and should be studied further. However, despite of these differences, both monkeys showed the same tendencies regarding the direction of cueing effects: inhibitory cueing effect in the normal field and no inhibitory but significant facilitatory cueing effect in the affected field. Thus, current results indicate that V1 is critically involved in the inhibitory cueing effect.

DISCUSSION

We found that the cueing effects on saccades were significantly affected by V1 lesions: the inhibitory effect disappeared, whereas the facilitatory effect still emerged after the V1 lesion. The results indicate a functional difference

between the geniculostriate pathway and other remaining visual pathways. Our results indicate that the geniculostriate pathway is critically involved in the inhibitory cueing effect, whereas the remaining pathways, most likely the retino-tectal pathway, might be more related to the facilitatory cueing effect.

Visual Processing after V1 Lesion

In primates, about 90% of the projections from retina are relayed to V1 through the LGN (Cowey & Stoerig, 1991). There is also a direct projection from the retina to the SC (retino-tectal pathway), which accounts for approximately 10% of the projections from the retina in normal monkeys (Perry & Cowey, 1984). This pathway is thought to serve as the main visual pathway after V1 lesions because joint lesions of V1 and the SC lead to the complete blindness (Mohler & Wurtz, 1977). Although these two visual pathways are thought to be phylogenetically and functionally different (Schneider, 1969; Trevarthen, 1968), they are not independent but interconnected with one another. In addition to these two pathways, there is another

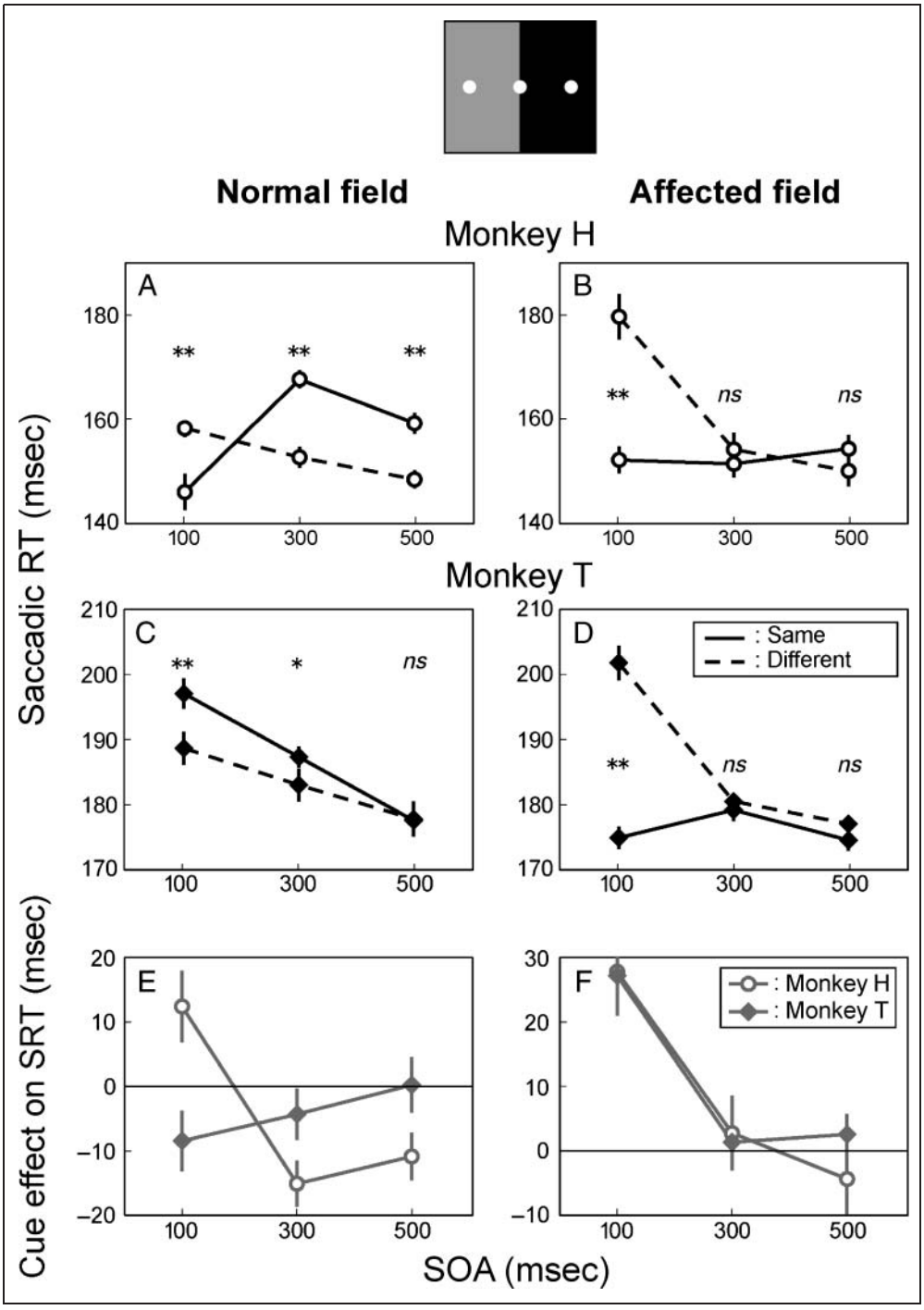

Figure 3. Cueing effects in the bilateral condition. Top figure shows the two possible locations for the cue and the target, which were presented either to the right or to the left of the fixation point (spot in the middle). Dark area represents the affected field. (A–D) SRTs when the target was presented in the normal field (A, C) or in the affected field (B, D) for the two monkeys. Solid lines indicate SRTs in the same condition and dashed lines those in the different condition. Error bars indicate the 95% confidence intervals of the mean. Asterisks indicate significant differences between the same and the different condition as determined by *t* test under Bonferroni correction for multiple tests, six pairs. $*p < .05$, $**p < .0001$. Each point represents an average of 312–431 (Monkey H) or 254–341 (Monkey T) trials. (E, F) Cueing effects on SRT, defined as the difference in mean SRTs between the two cue conditions (different condition minus same condition), in the normal and affected field, respectively. Open circle: Monkey H; filled diamond: Monkey T. Error bars indicate the 95% confidence intervals of the difference.

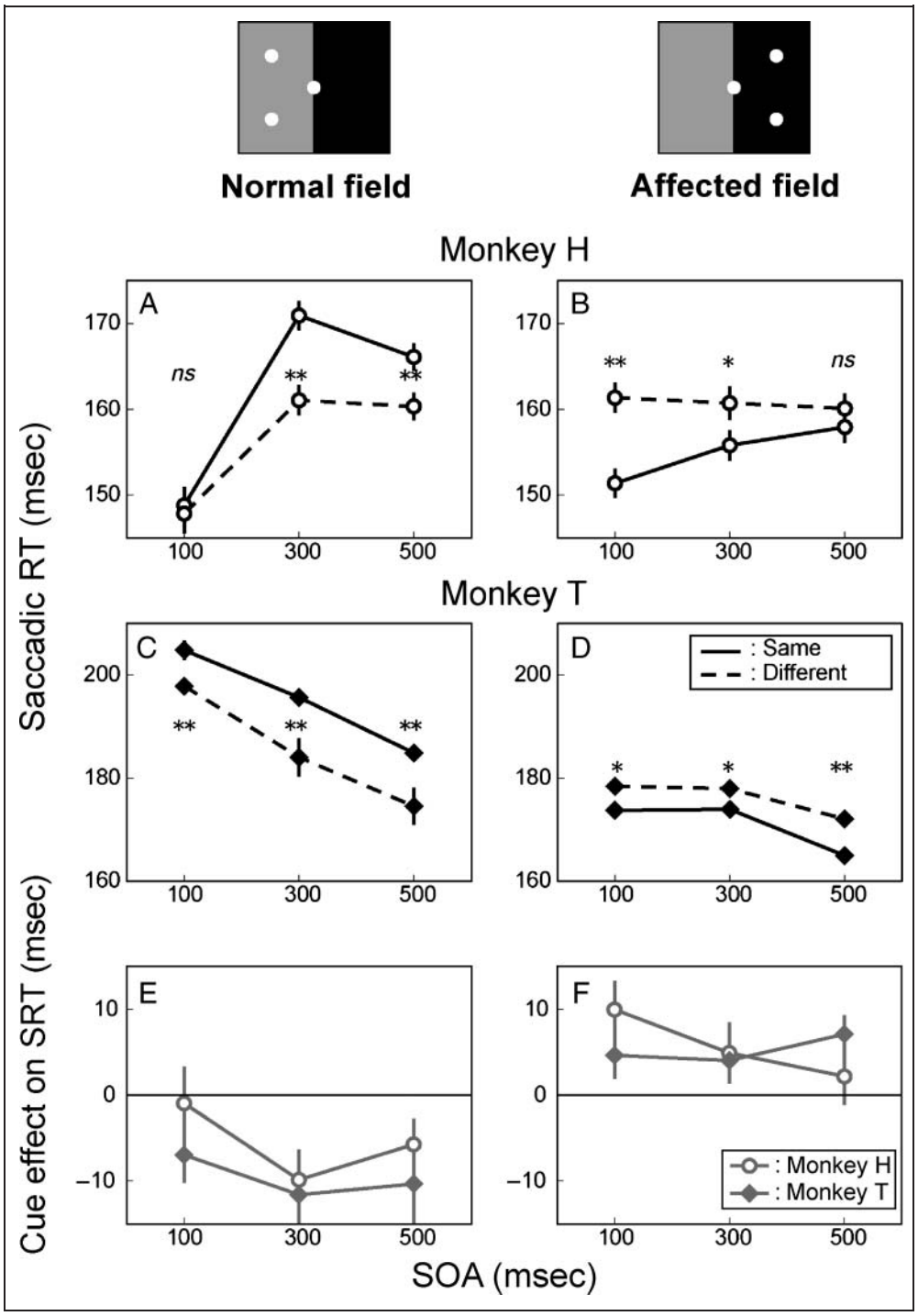

Figure 4. Cueing effects in the unilateral condition. Same format as Figure 3. Top figure shows the two possible locations for the cue and the target in the normal field block of trials (left) and in the affected field block (right). Each point represents the average of 315–420 (Monkey H) or 392–530 (Monkey T) trials.

visual pathway from LGN to extrastriate cortex (geniculo-extrastriate pathway; Sincich, Park, Wohlgemuth, & Horton, 2004; Bullier & Kennedy, 1983), which survives and may aid residual visual function after V1 lesion (Cowey & Stoerig, 1989). Thus, a V1 lesion has significant influences on the entire visual system in the brain, yet most of the visual-related areas still receive some information from remaining pathways. Furthermore, the fact that a V1 lesion leads to morphological and physiological changes in many brain areas (Rodman, Gross, & Albright, 1989; Dineen, Hendrickson, & Keating, 1982; Mohler & Wurtz, 1977; Cowey, 1974) makes the situation more complicated. In the following section, we discuss possible mechanisms that can explain our results in concert with previous behavioral and physiological findings.

Inhibitory Cueing Effect in the Normal Field

Previous studies suggested the critical involvement of the SC in the expression of the inhibitory cueing effect (Sereno et al., 2006; Sapir et al., 1999; Posner et al., 1985). However,

neurophysiological studies indicated that the SC itself is not the site of inhibition but rather reflects the reduced input from upstream regions (Fecteau & Munoz, 2005; Dorris et al., 2002). V1 is a possible candidate for the source of reduced visual response because it sends visual information to the superficial layer of the SC (Lui, Gregory, Blanks, & Giolli, 1995; Fries, 1984). Indeed, the visual response of V1 neurons is attenuated by a preceding visual stimulus at the same location (Macknik & Livingstone, 1998; Judge, Wurtz, & Richmond, 1980), and Müller and Kleinschmidt (2007) recently reported that the BOLD response of V1 to a visual stimulus is attenuated concurrently with the inhibitory cueing effect. These results suggest that V1 is involved in generating the inhibitory cueing effect. Our results provide the first direct evidence for a causal relationship between the V1 and the inhibitory cueing effect.

It should be noted that Sumner, Nachev, Vora, Husain, and Kennard (2004) suggested that the saccadic inhibitory cueing effect is mediated by the retino-tectal pathway, which is apparently inconsistent with our current results. Using a cueing task similar to that in the current experiment, they found that a cue stimulus visible only to short wavelength-sensitive cones (S-cone stimulus) did not elicit the inhibitory cueing effect on saccade. Because the S-cone stimuli are primarily processed by the geniculostriate pathway and not by the retino-tectal pathway (Schiller & Malpeli, 1977), this result seems to suggest that not the geniculostriate pathway but the retino-tectal pathway mediates the inhibitory cueing effect. However, the striate–collicular pathway, which is impaired by V1 lesions, is also blind to the S-cone stimuli (Schiller, Malpeli, & Schein, 1979). Thus, these two studies are not contradictory but complementary and strongly suggest the importance of the interaction between the geniculostriate pathway and the SC in the inhibitory cueing effect. We would like to propose that the geniculostriate pathway is responsible for the generation and the SC is involved in the expression of the inhibitory cueing effect on saccade.

Recent neuroimaging studies suggested that many cortical areas other than V1 are also involved in the inhibitory cueing effect (Müller & Kleinschmidt, 2007; Mayer, Seidenberg, Dorflinger, & Rao, 2004; Lepsien & Pollmann, 2002; Rosen et al., 1999). It is possible that these cortical areas modulate or strengthen the inhibitory cueing effect, although the visual input from the remaining pathways might be insufficient for generating it.

Facilitatory Cueing Effect in the Affected Field

It is known that a visual stimulus presentation with an abrupt onset is processed preferentially (Yantis & Jonides, 1984, 1990) and facilitates subsequent saccades to that location, often inducing reflexive eye movements (Godijn & Theeuwes, 2002; Irwin, Colcombe, Kramer, & Hahn, 2000; Theeuwes, Kramer, Hahn, Irwin, & Zelinsly, 1999; Theeuwes, Kramer, Hahn, & Irwin, 1998). This facilitatory effect is thought to be mediated by the local activation of an oculomotor map, possibly in the SC (Godijn & Theeuwes, 2002; Trappenberg, Dorris, Munoz, & Klein, 2001). Given the fact that this reflexive facilitatory effect can be induced by abrupt onset and luminance change but not by color cue (Irwin et al., 2000), the retino-tectal pathway should be the major, if not only, source of visual information for the facilitatory effect. Our results show a strong facilitatory cueing effect in the absence of the geniculostriate pathway and support this assumption, although it is still possible that other visual pathways can induce or modulate the facilitatory cueing effect as shown by the experiment using S-cone stimuli (Sumner, Adamjee, & Mollon, 2002).

In the conventional cueing task, the facilitatory cueing effect on saccades in monkeys fades quickly and is replaced by the inhibitory effect within 50–100 msec (Fecteau & Munoz, 2005; Dorris et al., 2002), which is consistent with our results in the normal field, although Monkey T did

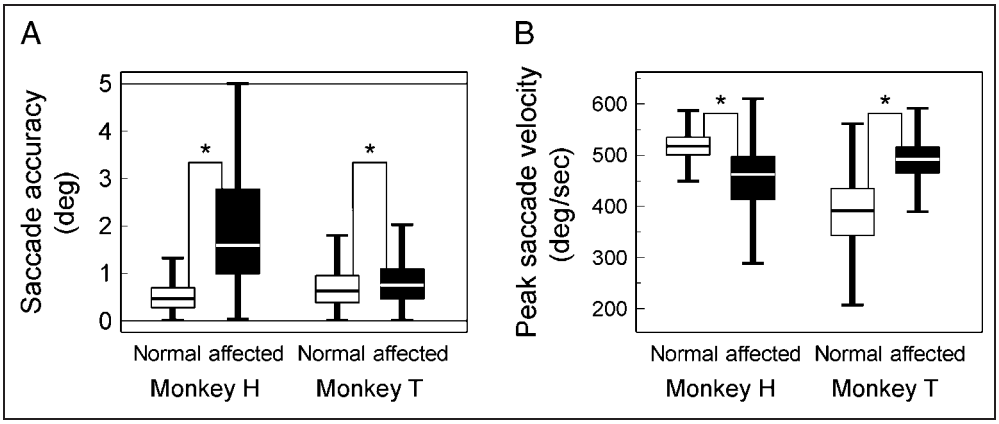

Figure 5. Saccade parameters in unilateral condition experiment. (A) Box plot showing the median and the quartile values of saccade accuracy in the unilateral condition experiment. Saccade accuracy was defined as the distance (in degrees) between the target and the end point of initial saccade. Saccades tended to be more accurate when the target was presented in the normal field than that in the affected field in both monkeys. However, the loss of accuracy in the affected field was much less in Monkey T than that in Monkey H. Asterisks indicate statistical significance by the Mann–Whitney *U* test ($*p < 10^{-10}$). (B) Box plot of peak saccade velocity in the unilateral condition experiment. Monkey H made faster saccades in the normal field, whereas Monkey T made faster saccades in the affected field. Asterisks indicate statistical significance by the Mann–Whitney *U* test ($*p < 10^{-10}$).

not show a facilitatory effect. On the other hand, in the affected field, the facilitatory cueing effect lasted more than 300 msec, much longer than shown in previous studies. It has been reported that a V1 lesion increases the attentional enhancement of neural activity in the SC (Mohler & Wurtz, 1977) and lowers the decision threshold for saccade initiation (Yoshida et al., 2008), which may enhance the facilitatory effect. This enhancement of facilitation may be a result of compensatory plasticity after the lesion (Moore, Rodman, & Gross, 1998; Moore, Rodman, Repp, Gross, & Mezrich, 1996). It is also possible that the enhancement of the facilitatory effect is merely a consequence of the decrement in the inhibitory effect because the cueing effects are the result of an integration of multiple processes (Lupiáñez, Ruz, Funes, & Milliken, 2007).

Relation to Shift of Attention

Since Posner and Cohen (1984) first reported the facilitatory and the inhibitory cueing effects, it is widely believed that these effects are the consequences of a shift in attention after cue presentation and are termed attentional capture and inhibition of return (IOR), respectively (Ruz & Lupiáñez, 2002; Klein, 2000; Posner et al., 1985). Recent studies suggested that several parallel processes, which might be mediated by different neural substrates, contribute to attentional capture and IOR (Lupiáñez et al., 2007; Khatoon, Briand, & Sereno, 2002; Tipper et al., 1997). Our study supports this parallel processing hypothesis and suggests that the geniculostriate pathway plays a central role in IOR, whereas the retino-tectal pathway may mediate attentional capture. However, as several studies have also demonstrated dissociation between the cueing effect on saccade and the shift of attention (Sumner et al., 2004; Hunt & Kingstone, 2003; Abrams & Dobkin, 1994), this should be considered carefully.

The inhibitory cueing effect on saccade is thought to improve the efficiency of visual searching by encouraging orienting to novel locations (Klein, 1988, 2000; Posner & Cohen, 1984). Considering that the geniculostriate pathway evolved much more recently than the primitive retino-tectal pathway and that lower animals like pigeons show no inhibitory cueing effect but only the facilitatory cueing effect (Gibson, Juricevic, Shettleworth, Pratt, & Klein, 2005), our finding raises an interesting speculation: The evolution of the geniculostriate pathway in higher mammals enhanced the inhibitory process, thus enabling animals to search complex environments more efficiently.

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