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Attentional sensitivity and asymmetries of vertical saccade generation in monkey

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Abstract

The first goal of this study was to systematically document asymmetries in vertical saccade generation. We found that visually guided upward saccades have not only shorter latencies, but higher peak velocities, shorter durations and smaller errors. The second goal was to identify possible mechanisms underlying the asymmetry in vertical saccade latencies. Based on a recent model of saccade generation, three stages of saccade generation were investigated using specific behavioral paradigms: attention shift to a visual target (CUED paradigm), initiation of saccade generation (GAP paradigm) and release of the motor command to execute the saccade (DELAY paradigm). Our results suggest that initiation of a saccade (or "ocular disengagement") and its motor release contribute little to the asymmetry in vertical saccade latency. However, analysis of saccades made in the CUED paradigm indicated that it took less time to shift attention to a target in the upper visual field than to a target in the lower visual field. These data suggest that higher attentional sensitivity to targets in the upper visual field may contribute to shorter latencies of upward saccades.

Keywords: Eye movement; Hemifield asymmetry; Saccade generation; Attention

1. Introduction

Recently, functional specialization in the lower and upper visual fields in primates has been analyzed in relation to the ecological function of the primate visual system (Previc, 1990). Visual information processing differences between the upper and lower visual hemifields appear to be related to the distinction between near (peripersonal) and far (extrapersonal) space, whose representations are biased toward the lower and upper visual fields, respectively. On the one hand, the lower visual field advantage with respect to visual scene segmentation (Rubin, Nakayama, & Shapely, 1996) is related to the fact that occlusion situations occur more frequently in the lower visual field. Thus, it is of a greater survival importance to develop an enhanced capacity for visual scene segmentation in the ground plane (Gibson, 1950). On the other hand, the upper visual field advantage with respect to saccade latency (Heywood & Churcher, 1980; Schlykowa, Hoffmann, Bremmer, Thiele, & Ehrenstein, 1996) is related to the importance of saccades in scanning and visual search in extrapersonal space that is usually directed to the upper visual field.

Attentional systems play important roles in both visual information processing and in generation of visually guided saccades (Posner, 1980). It was reported recently that there is a lower visual field advantage in the spatial resolving power of attention (He, Cavanagh, & Intriligator, 1996), which may contribute to a lower visual field advantage for global shape perception (Previc, 1990; Rubin et al., 1996). However, little is known about whether the temporal resolving power of attention, which relates to how fast the attentional system responds to the appearance of peripheral targets, may favor one vertical hemifield over the other. Remington (1980) showed that in the presence of a stimulus that elicits eye movement, a subject's attention tends to shift prior to his eye movement. This attentional shift may facilitate the processing of peripheral visual events and the generation of visually guided saccades. Furthermore,

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Posner (1980) showed that attentional shifts could be assessed by comparing the reduction in mean saccade latency of eye movements whose targets were correctly cued with respect to those whose targets were incorrectly cued. The present study investigates whether attentional shifts favor saccades to the upper or lower visual field and whether such a bias, if it exists, contributes to an upper visual field advantage with respect to saccade latency (Heywood & Churcher, 1980; Schlykowa et al., 1996), i.e. saccades in the upper visual field have shorter latencies than those to the lower visual field.

The rationale of our experiments is based on a recent model of saccade generation that features parallel central processes organized in a hierarchy of levels (Fig. 1 adapted from Findlay & Walker, 1999). The model in Fig. 1 was simplified for visually elicited saccades that are the focus of the current study. In the model, a visual stimulus first elicits a shift of attention that is followed by parallel computation of saccade metrics (WHERE) and decision making (WHEN). The outputs of the WHERE processes are motor commands that determine the dynamics of the saccades. The outputs of the WHEN process control a GATE that prevents the outputs of the motor command center from executing unwanted saccades. According to this model, saccade latency includes components related to sensory information processing, attention shift, temporal and spatial programming of a possible saccade, a decision to open the GATE and execution of the motor command. The primary goal of the present study is to examine the contributions of these stages to the asymmetry in vertical saccade latencies. As reviewed by Previc (1990), it is unlikely that the sensory processing of target information (see Fig. 1, "visual stimulus") is involved in the asymmetry because manual reaction times show an opposite pattern, being generally faster for targets in the lower field. The contribution of the last stage (motor execution) to the asymmetry in vertical saccade generation is evaluated by employing a DELAY paradigm, in which a saccade target and a fixation point are presented simultaneously for a variable time and the monkey is required to withhold its saccade until the fixation point is extinguished. In the DELAY paradigm, the saccade generation system is given sufficient time to complete the earlier stages of saccade programming prior to execution of the motor command. Thus, any differences in saccade latency to targets in the upper or lower visual fields during this paradigm should reflect the contribution of the motor execution process. The contribution of the GATE stage is evaluated by a GAP paradigm. A decision to "open the gate" in the model is assumed to be equivalent to what has been called "ocular disengagement" in the literature (for a review, see Fischer & Weber, 1993). Saslow (1967) showed that "opening the gate" could be assessed by measuring a reduction in saccade latency that results from the introduction of a time delay (GAP) between when the fixation point is extinguished and the target is turned on. A comparison of saccade latencies to targets in the upper or lower visual fields in the GAP paradigm can assess involvement of the GATE stage. Finally, contributions of attentional shift to the asymmetry are assessed by employing a CUED eye movement paradigm.

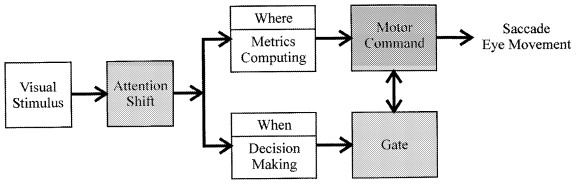

Fig. 1. A working model of the generation of visually guided saccades. Adapted from Findlay and Walker (1999).

2. Methods

2.1. General

Two macaque monkeys (*Macaca mulatta*) were fitted with a head stabilization platform that was attached to the skull using stainless steel orthopedic bone plates and screws. Eye movements were monitored by a magnetic search coil technique (Judge, Richmond, & Chu, 1980; Robinson, 1963) with a resolution of 0.1° (CNC Engineering, Seattle, WA). Surgical procedures and experimental protocols adhered to the *NIH Guide for the Care and Use of Laboratory Animals* and were approved by the Institutional Animal Care and Use Committee at the University of Mississippi Medical Center. After recovery from surgery, monkeys were trained to fixate and track laser generated target spots for juice rewards. Eye position was calibrated by requiring the monkey to fixate target spots at known horizontal and vertical eccentricities on a tangent screen located about two meters in front of the monkey.

2.2. Experiment 1: asymmetry in the DELAY paradigm

To evaluate asymmetries in the motor command stage (Fig. 1), monkeys were trained to make saccades to peripheral targets in CONTROL and DELAY conditions. In the CONTROL condition, the peripheral target appeared immediately after the central fixation light was extinguished (Fig. 2A). In the DELAY [1] condition, the peripheral target was illuminated for a variable period

[1] In the saccade literature, the DELAY condition is often used to study memory-guided saccade by turning on the target only briefly. In this study, the target stays on and the visually guided saccade is triggered by the offset of the fixation point.

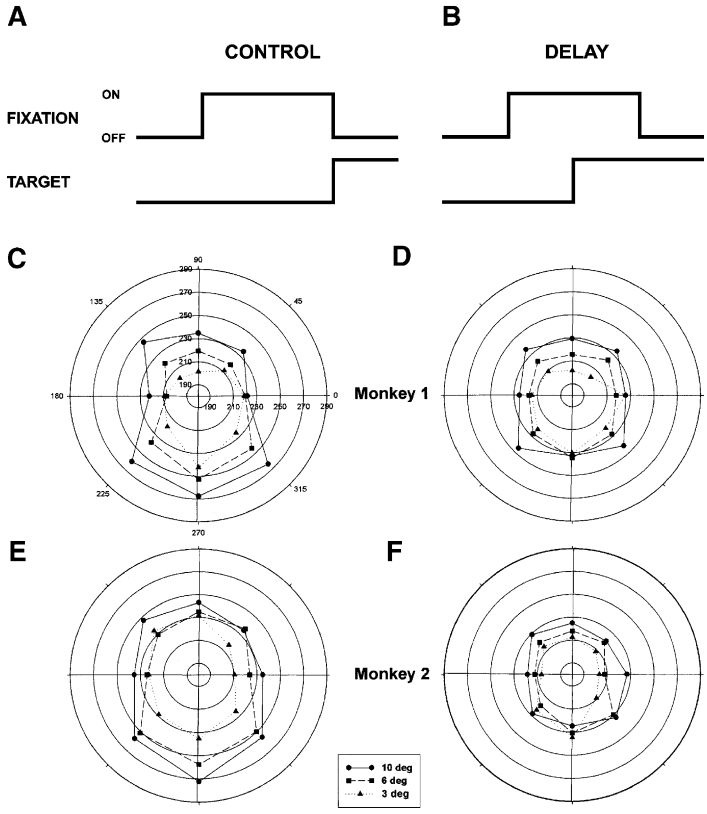

Fig. 2. Saccade latency plotted as a function of direction and eccentricity in CONTROL and DELAY conditions. Schematic illustration of stimuli conditions is shown in A for CONTROL condition, and in B for DELAY condition. Data from two monkeys in both conditions are shown in C and D (Monkey 1) and E and F (Monkey 2). Formats of C, D and E are as same as that of A.

of time (1.2, 1.4, or 1.6 s) before the central fixation target was extinguished (Fig. 1B). In the DELAY paradigm, a saccade is triggered by the offset of the fixation point rather than by the onset of the peripheral target. Peripheral targets appeared randomly in four cardinal and four oblique directions relative to the fixation point and at three different eccentricities (3°, 6°, and 10°). Fifty trials were obtained for each condition. Data in CONTROL and DELAY conditions were collected in separate batches.

2.3. Experiment 2: asymmetry in the GAP paradigm

To evaluate if asymmetry exists in the gating stage (Fig. 1), monkeys were trained to make saccades to peripheral targets in CONTROL and GAP conditions. The CONTROL condition was identical to that described above. In the GAP condition, a 200 ms time delay is introduced between the fixation light's offset and the peripheral target's onset. The two conditions were interleaved randomly with equal probability. Targets were presented randomly at two locations (8° up, 8° down) in either task. There were 200 trials of each condition.

2.4. Experiment 3: asymmetry in the CUED paradigm

To evaluate asymmetries in the attentional shift stage (Fig. 1), monkeys were trained to make saccades in CONTROL and CUED conditions. In the CUED condition, a target was flashed briefly (~100 ms) in the upper or lower visual field 700 ms after the central spot was fixated. About 200 ms after presenting the cue, the target was turned on and the central fixation point was simultaneously extinguished. The target appeared either in the cued location (valid cue, 80% of trials) or in the opposite visual field (invalid cue, 20% of trials). Targets were presented randomly with equal probability at one of two locations: either 8° up or 8° down. CUED and CONTROL conditions were interleaved randomly with equal probability. Prior to data collection, monkeys were exposed to 3 or 4 practice sessions to assure their familiarity with the behavioral paradigm. To discourage anticipatory errors, trials were aborted if the monkey broke fixation prior to the target's onset, and trials were not rewarded if the monkey did not make a saccade to the target. A few anticipatory saccades did occur despite these precautions (<10%). Anticipatory saccades, defined as having latencies <70 ms after target onset, were excluded from the analysis (see Section 2.5).

Interpretation of cued trials is complicated because cues can cause an attention shift as well as allowing preprogramming by signaling target location and its impending onset. These effects, however, should be similar for both valid and invalid cue conditions. Thus, any differences between the mean reaction times of valid and invalid cue conditions are assumed to be related to attentional shift (Kustov & Robinson, 1996; Posner, 1980; Remington, 1980).

2.5. Data analysis

Data were acquired and behavior was controlled using custom software on an IBM compatible personal computer operating under Microsoft Windows 3.11. Analysis of the collected data was performed off-line on a Sun workstation (Sparc 20) using interactive computer programs. Eye position data were differentiated to obtain velocity using a two-point central difference filter with a step size of +8 ms (Bahill & McDonald, 1983). During visual fixation, eye position signals recorded by search coils show no drifting in either horizontal or vertical directions. Saccade onset was determined as the time when eye velocity first surpassed a velocity threshold (20 deg/s) and saccade end was determined as the time when eye velocity first declined below the same threshold. Saccade latency was determined as the interval from the onset of the target to the onset of the saccade. Analysis of saccade latency was restricted to on-target saccades with latencies between 70 and 500 ms. As in previous studies, saccades with latencies shorter than 70 ms were arbitrarily classified as anticipatory and were excluded from our analysis (Bronstein & Kennard, 1987; Fischer & Weber, 1993; Fischer et al., 1993; Kalesnykas & Hallet, 1987; Pare & Munoz, 1996; Smith & Van Gisbergen, 1989; Wenban-Smith & Findlay, 1991). The values of saccade latency are presented as means ± SEM. The accuracy of the first saccade was computed as the difference between final eye position and eye position at the end of the initial saccade. The results were evaluated by analysis of variance followed by a pairwise multiple comparison procedure (SYSTAT for Windows).

3. Results

3.1. Asymmetry in DELAY and CONTROL conditions

Fig. 2C–F show saccade latency as polar diagrams in the CONTROL (Fig. 2C and E) and DELAY (Fig. 2D and F) conditions for two monkeys. The timing of the fixation and peripheral targets in the two conditions is schematically illustrated in Fig. 2A for the CONTROL condition and in Fig. 2B for the DELAY condition. Saccade latency is plotted as a function of eccentricity and direction in each plot. In the CONTROL condition, there is a marked asymmetry in vertical saccade latency in both monkeys (Fig. 2C and E) at each of the three eccentricities tested ($P < 0.001$). For example, the mean latency of upward 10° saccades (235 ± 2 ms) of Monkey 1 was significantly shorter than the mean latency of downward 10° saccades (267 ± 2 ms) ($P < 0.001$) (Fig. 2C). Similarly, for Monkey 2, the mean latency of upward 10° saccades (243 ± 4 ms) was significantly shorter than the mean latency of downward 10° saccades (272 ± 2 ms) ($P < 0.001$) (Fig. 2E). In the DELAY condition, the target and the central fixation point were simultaneously present for variable intervals (1.2, 1.4, 1.6 s) (Fig. 2B). The variable intervals were interleaved randomly to prevent anticipation of fixation point offset. The saccade latencies from the three intervals were not statistically different and were pooled. Fig. 2D and F shows that, in the DELAY condition, the asymmetry of vertical saccade latency was abolished. For Monkey 1, the mean latencies of upward and downward 10° saccades were not statistically different (229 ± 3 ms for upward saccades, 233 ± 3 ms for downward saccades, $P > 0.05$) (Fig. 2D). For Monkey 2, the mean latency of upward and downward 10° saccades was the same (225 ± 3 ms for either direction) (Fig. 2F). These data suggest that the asymmetry in vertical saccade latency is not due to an asymmetry in the motor output mechanism. However, these data do not rule out the possibility that there may be motor asymmetries that appear only within a brief interval between target appearance and saccade onset.

Normally, monkeys make more than one saccade to accurately foveate a target. We used the difference between the eye position at the end of the initial saccade and final eye position to estimate initial saccade error. Fig. 3 shows the accuracy of initial saccades in CONTROL and DELAY conditions (means of the absolute values of the errors). The polar diagrams illustrate that in both monkeys, there is a marked asymmetry in vertical saccade accuracy. In general, errors of saccades to targets in the upper visual field were smaller than errors of saccades of similar amplitude to targets in the lower visual field. Interestingly, the asymmetry in vertical saccade accuracy was still present in the DELAY condition. The accuracy of saccades to lower targets was not improved when exposure to the target was longer. Fig. 4 shows the distributions of errors for 10° upward and downward saccades in the two monkeys. A positive error indicates an overshoot saccade and a negative error indicates an undershoot saccade. In both monkeys, upward saccades tended to overshoot in both CONTROL and DELAY conditions. However, downward saccades tended to undershoot in both conditions. In fact, in Monkey 2, downward saccade errors in the DELAY condition were increased ($P < 0.001$) (Fig. 4b). These data suggest that undershooting for downward

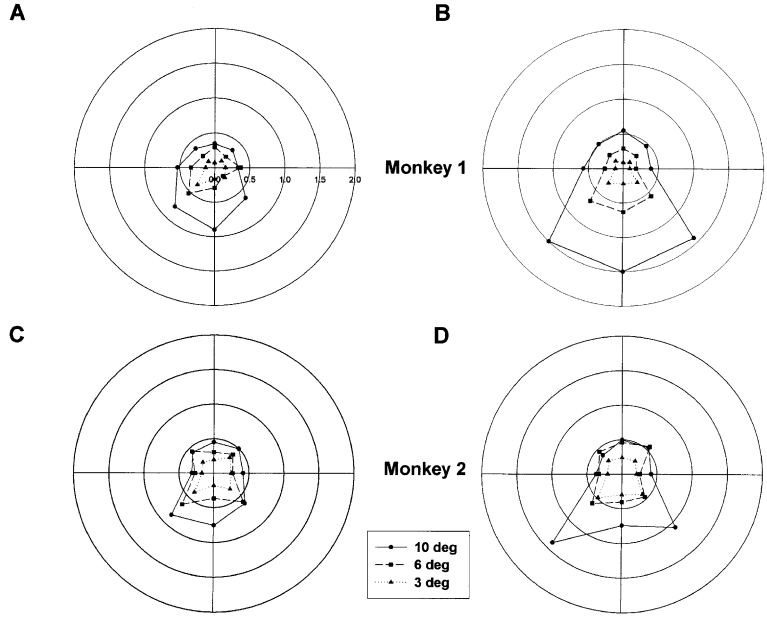

Fig. 3. Saccade accuracy plotted as a function of direction and eccentricity in CONTROL (A, C) and DELAY (B, D) conditions for Monkey 1 (A, B) and Monkey 2 (C, D). The radial axis is from 0° to 2° at every 0.5°. Means were computed using the absolute value of saccade errors.

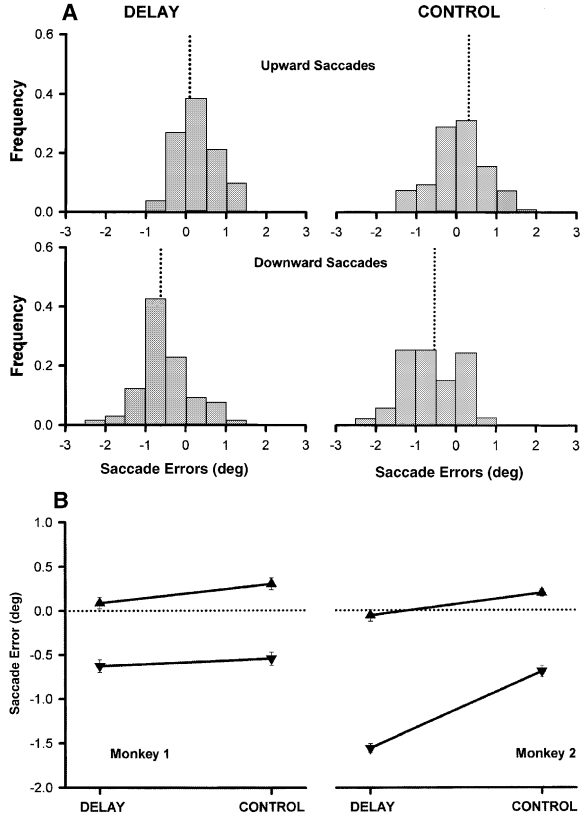

Fig. 4. Distributions of saccade errors (10° upward/downward saccades) in CONTROL and DELAY conditions. (A) Histograms of saccade errors in Monkey 1, the dotted lines show the means of the data sample. (B) Means and standard errors from both monkeys under the two conditions (upward triangles for 10° upward saccades, downward triangles for 10° downward saccades).

saccades and overshooting for upward saccades may be a deliberate strategy. If deliberate, such a strategy would confine a target's neural representation to the lower visual field that has been suggested to specialize in visual information processing for texture and shape (He et al., 1996; Henson, 1978; Robinson, 1973; Rubin et al., 1996). As a control, Fig. 5 shows that saccade peak velocity and duration were similar in CONTROL and DELAY conditions. However, there was a clear asymmetry between upward or downward peak saccade velocity and duration. Thus, these data show that upward saccades not only have shorter latencies, but also are faster, take less time to finish, and are more accurate.

3.2. Asymmetry in ocular disengagement

Fig. 6 shows that there was a significant asymmetry in vertical saccade latencies in both CONTROL and GAP conditions in both monkeys ($P < 0.0001$). However, there was no significant interaction between the GAP effect and the location of the target in the upper or lower visual field (for Monkey 1, $P > 0.6$; Monkey 2, $P > 0.06$), indicating that the introduction of a gap between the target onset and fixation offset resulted in similar reductions in the mean latencies of saccades to targets in either the upper or lower visual fields.

3.3. Asymmetry in attentional shift

Fig. 7 shows latencies of up or down saccades in three conditions: CONTROL, VALID CUES, and INVALID CUES. In all three conditions, up saccades in both monkeys had shorter latencies ($P < 0.0001$). As

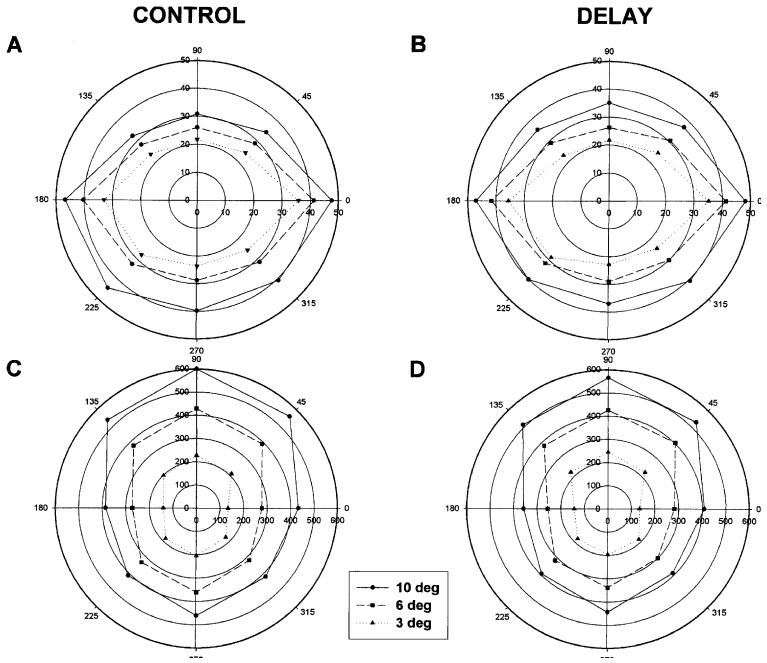

Fig. 5. Polar diagrams of saccade duration (A, B) and peak velocity (C, D) as a function of direction and eccentricity in CONTROL (A, C) and DELAY (B, D) conditions.

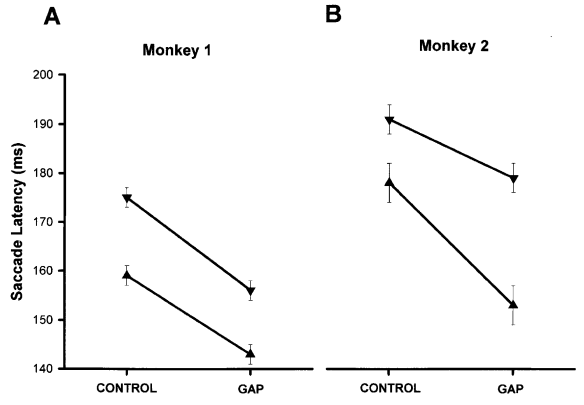

Fig. 6. The interaction between the GAP effect and the asymmetry of vertical saccade latency.

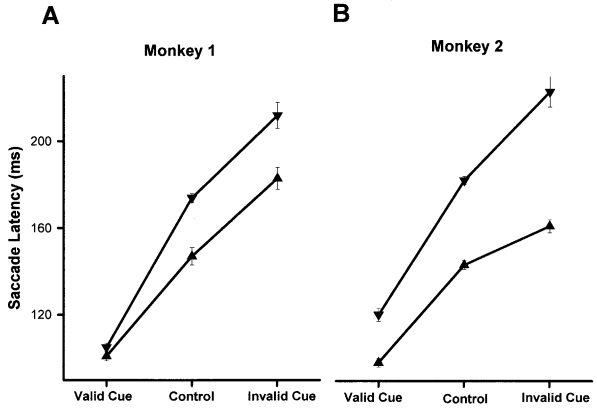

Fig. 7. The interaction between the CUE effect and the asymmetry of vertical saccade latency.

expected, saccades with valid cues also had significantly shorter latencies in both monkeys ($P < 0.0001$), demonstrating the effectiveness of the cue in shifting attention. In both monkeys, the interaction between the CUE effect and asymmetry in vertical saccade latencies was significant ($P < 0.001$). The cue validity effect, measured as the difference in mean latency between valid and invalid cue conditions, was larger for downward than for upward saccades (Monkey 1: 69 ± 4 vs. 46 ± 6 ms, $P < 0.01$; Monkey 2: 62 ± 5 vs. 45 ± 4, $P < 0.01$). In Monkey 1, the difference in mean saccade latency between upward or downward saccades was 27 ms in the CONTROL condition but this difference was reduced to 4 ms in the VALID CUE condition. For this monkey, an asymmetry in shifting attention to targets in the upper or lower visual fields could account for about 85% of the asymmetry in vertical saccade latency to these targets. In Monkey 2, the difference in mean latency between upward or downward saccades was 39 ms and this difference was reduced to 22 ms in the VALID CUE condition. For this monkey, the asymmetry in shifting attention to targets in the upper or lower visual fields could account for about 44% of the asymmetry in vertical saccade latency. Thus, attention shift contributes significantly to asymmetries in vertical saccade latencies.

4. Discussion

The model shown in Fig. 1 illustrates two parallel information and command streams in a hierarchy of

processing levels. Information in the WHERE stream is transmitted in spatially mapped pathways (for more details, see Findlay & Walker, 1999), but in the WHEN stream, a single non-spatial signal is involved. Sensory processing of the stimulus within the two streams is preceded by an attentional shift to the target. This attentional shift controls target selection and facilitates sensory processing and motor programming. The motor command is produced in the WHERE stream. The saccadic eye movement occurs when a trigger signal opens the GATE. For visually guided saccades, the sensory processing of the visual target involves subcortical and cortical visual pathways including the retina, lateral geniculate nucleus, striate and extrastriate cortex. The WHERE and WHEN streams involve cortical and subcortical sensory-motor pathways including the parietal eye fields, frontal eye fields, supplemental eye fields and the superior colliculus. The motor command and GATE stages involve the brainstem circuitry of omnipause cells and pre-motor burst cells (for a review, see Fischer & Weber, 1993).

In this study, we demonstrated that there is a robust asymmetry in vertical saccade latencies (Fig. 2C and E). Although there is evidence for asymmetries in sensory processing mechanisms, they favor the lower rather than the upper visual field. For example, the receptor density in the upper retina is greater than that in the lower retina (Skrandies, 1987). Similar asymmetries, biased toward the lower visual field, were also found in primary visual cortex, middle temporal visual area (MT) and area 7a (Maunsell & Van Essen, 1987; Robinson, Goldberg, & Stanton, 1978; Tootell, Switkes, Silverman, & Hamilton, 1988; Van Essen, Newsome, & Maunsell, 1984). Shape discrimination and feature detection are similar in the upper and lower visual fields, but the perception of illusory contours is enhanced in the lower visual field (Rubin et al., 1996).

The absence of vertical saccade latency asymmetries in the DELAY condition (Fig. 2D and F) suggests that the motor command center plays a minor role in producing vertical saccade latency asymmetries. These results are consistent with single unit studies in which "burst" neurons that encode either upward or downward components of saccades are found in about equal proportions in the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) (Buttner, Buttner-Ennever, & Henn, 1977; King & Fuchs, 1979; Moschovakis, Scudder, & Highstein, 1991; Moschovakis, Scudder, Highstein, & Warren, 1991; Vilis, Hepp, Schwarz, & Henn, 1989).

The lack of a significant interaction between the GAP effect on saccade latency and the occurrence of asymmetries in vertical saccade latencies suggest that the GATE stage is also not a major source of the difference in latency. These data are similar to a prior study that used human subjects and which found no interaction between the GAP effect and the latency asymmetries (Honda & Findlay, 1992). In another study in humans, however, Goldring and Fischer (1997) reported a larger gap effect for downward saccades as compared to upward saccades in 10/12 tested subjects. A difference in experimental design may have contributed to the discrepancy between these studies. In the study of Goldring and Fischer (1997), the gap effect was assessed by comparing saccade latencies in the gap condition to those occurring in the overlap condition. In the study of Honda and Findlay (1992) and in the present study, the gap effect was assessed by comparing saccade latencies in the gap condition to those occurring in the CONTROL (i.e. no-gap) condition. In the overlap condition, delays in ocular disengagement or attentional shift may contribute to the longer saccade latencies that are observed. Since there is an asymmetry in shifting visual attention to a target in the upper or lower visual field, the larger gap effect reported by Goldring and Fischer (1997) may be due to an interaction between the processes producing delays in the overlap condition and vertical saccade latency. Furthermore, a population of inhibitory neurons located within the midline raphe nuclei (Evinger, Kaneko, Johanson, & Fuchs, 1977; Keller, 1974, 1977; King & Fuchs, 1979) has been suggested to be the neural substrate of the GATE. The inhibitory neurons have been hypothesized to act as a "gate" on the saccadic burst generator network in the brainstem (for a review, see Fuchs, Kaneko, & Scudder, 1985). These cells are called "omnipause neurons", because their activity ceases prior to and during every saccade, regardless of its direction. The absence of any directional asymmetry in their responses is consistent with a minimal role of the GATE circuitry in generating the vertical saccade latency asymmetries.

The significant interaction between the CUE effect and the asymmetries in vertical saccade latency implies a possible asymmetry in attentional mechanisms. In particular, it may take less time to shift attention to targets in the upper visual field than to targets in the lower visual field. Higher *attention sensitivity* to objects in the upper visual fields may make the appearance of a target in the upper field a more powerful stimulus for an attentional shift and thus result in a saccade with a shorter latency than if the target had appeared in the lower field. What are the likely sites for such an asymmetry? The first site where there are reported asymmetries in processing sensory information from the upper and lower visual hemifields is the third visual area (V3) (Burkhalter, Felleman, Newsome, & Van Essen, 1986). In the macaque, both areas are anterior to V2, but V3 differs anatomically and functionally from ventral area VP. VP contains a topographic map for the upper visual field and projects mainly to the ventral pathway. However, V3 contains a topographical map of the lower visual field and projects mainly to the dorsal pathway. Thus,

an asymmetry in vertical saccade processing could lie in the cortical networks at the level of V3 and beyond. This network could include the frontal eye fields (FEF), parietal eye fields (PEF) and supplemental eye fields (SEF) (Tian & Lynch, 1996). There is, however, no evidence for asymmetries in the neural representations of the upper and lower visual fields in these cortical regions (Felleman & Van Essen, 1991; Horton & Hoyt, 1991). The dorsal parietal system has classically been associated with attentional processes (Gazzaniga & Ladavas, 1987), and it has been demonstrated that the projections from early visual areas to the parietal regions are more numerous for the lower visual field than the upper field (Maunsell & Newsome, 1987). The dense projections for the lower visual field is consistent with a higher spatial attentional resolution in the lower visual field (He et al., 1996) that is of survival importance in interpreting richly textured visual information in peripersonal space (lower visual field) (Gibson, 1950; Previc, 1990). Interestingly, our data suggest that the upper visual field may have a higher temporal attentional sensitivity that is of survival importance in the initiation of rapid responses to visual targets in the extrapersonal space (upper visual field).

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