J Neurophysiol 100: 2757–2770, 2008.
First published September 24, 2008; doi:10.1152/jn.90238.2008.

Control of Predictive Error Correction During a Saccadic Double-Step Task

K. M. Sharika, Arjun Ramakrishnan, and Aditya Murthy
National Brain Research Centre, Nainwal More, Manesar, Haryana, India

Submitted 6 February 2008; accepted in final form 16 September 2008

Sharika KM, Ramakrishnan A, Murthy A. Control of predictive error correction during a saccadic double-step task. *J Neurophysiol* 100: 2757–2770, 2008. First published September 24, 2008; doi:10.1152/jn.90238.2008. We explored the nature of control during error correction using a modified saccadic double-step task in which subjects cancelled the initial saccade to the first target and redirected gaze to a second target. Failure to inhibit was associated with a quick corrective saccade, suggesting that errors and corrections may be planned concurrently. However, because saccade programming constitutes a visual and a motor stage of preparation, the extent to which parallel processing occurs in anticipation of the error is not known. To estimate the time course of error correction, a triple-step condition was introduced that displaced the second target during the error. In these trials, corrective saccades directed at the location of the target prior to the third step suggest motor preparation of the corrective saccade in parallel with the error. To estimate the time course of motor preparation of the corrective saccade, further, we used an accumulator model (LATER) to fit the reaction times to the triple-step stimuli; the best-fit data revealed that the onset of correction could occur even before the start of the error. The estimated start of motor correction was also observed to be delayed as target step delay decreased, suggesting a form of interference between concurrent motor programs. Taken together we interpret these results to indicate that predictive error correction may occur concurrently while the oculomotor system is trying to inhibit an unwanted movement and suggest how inhibitory control and error correction may interact to enable goal-directed behaviors.

INTRODUCTION

What makes the voluntary control of goal-directed behavior special is our capacity to suitably respond to abrupt changes that make the current goals inappropriate. When confronted with such a situation, we respond by inhibiting the ongoing, irrelevant action and programming a new response appropriate to the new context. However, errors are produced when there is a failure of inhibition and are most often followed by corrective measures that achieve the goal. Thus the abilities to inhibit inappropriate responses and detect/correct errors form two critical components of information processing that enable goal-directed behavior. A stimulus paradigm that has been widely used to study such voluntary control in the oculomotor system is the double-step task in which a single target is displaced to successive locations called "target steps," and subjects are asked to rapidly follow the targets and fixate afresh (Becker and Jurgens 1979; Findlay and Harris 1984; Hallett et al. 1976a,b; Hou and Fender 1979; Komoda et al. 1973; Wheeless et al. 1966). If the interval between the target displacements, called the target step delay, is short, subjects typically inhibit the planned saccade to the initial target location and respond with a single saccade directed toward the second target location. However, if the target step delay is long, subjects often respond with a sequence of two saccades; an initial erroneous saccade toward the initial target location and a second corrective saccade directed at the final target location.

Critical insights into the mechanism of error correction have been obtained by analyzing the pause between the two saccades generated in the preceding sequence at different target step delays. More specifically, it is now well established that as the duration between the appearance of the second target and the beginning of the first saccade, which is the time available to the saccadic system to reprogram the second saccade (known as the reprocessing time), increases, the interval between the two saccades decreases and may even fall below the normal reaction time. Such a pattern of responses has led to the hypothesis that the corrective saccade may be programmed in parallel with the erroneous saccade (Becker and Jurgens 1979; McPeek et al. 2000; Ray et al. 2004). However, because saccade programming is composed of at least two stages, a visual stage that identifies the location of a target and a motor stage that prepares and executes the oculomotor command (Hooge and Erkelens 1996; Ludwig et al. 2005; Thompson et al. 1996; Viviani 1990), the extent to which parallel processing of correction may occur in anticipation of an error is still not clear in double-step tasks.

An important contributor to successful performance in the double-step task is inhibitory control (Camalier et al. 2007; Joti et al. 2007; Kapoor and Murthy 2008). Understanding how inhibition and error detection/correction interact is of fundamental interest as they reflect the workings of an executive control system that has been hypothesized to flexibly coordinate goal-directed behavior (Baddeley and Della Sala 1996; Logan and Cowan 1984; Norman and Shallice 1980). To date, little is understood of this interaction and how this enables self-control of action. We examined these issues by using a modified version of the double-step task in the context of a race model widely used to interpret inhibitory control in saccadic reaction time tasks (Hanes and Carpenter 1999; Hanes and Schall 1996; Logan and Cowan 1984).

METHODS

Subjects and recording setup

Eye movements of 14 subjects (5 males and 9 females), with normal or corrected-to-normal vision, were recorded with their heads stabilized by means of chin, temple, and forehead rests. All subjects gave their informed consent in accordance with the institutional

Address for reprint requests and other correspondence: A. Murthy, National Brain Research Centre, Near NSG Campus, Nainwal More, Manesar - 122 050, Haryana, India (E-mail: aditya@nbrc.ac.in).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

human ethics committee of National Brain Research Centre and the Declaration of Helsinki. Subjects were rewarded monetarily for all sessions.

Experiments were computer-controlled using TEMPO/VIDEOSYNC software (Reflective Computing, St. Louis, MO) that displayed visual stimuli and sampled and stored eye position with other behavioral parameters. Eye position was recorded with an infra-red pupil tracker running at 240 Hz (ISCAN, Boston, MA) that interfaced with TEMPO software (Reflective Computing) in real time. The spatial resolution of our system was ~0.01°, and the median saccadic accuracy, as estimated by the SD of saccadic endpoints across three successive trials to single targets presented in the task, was ~0.9°. All stimuli were presented on a Sony Trinitron 500 GDM monitor (21 in.; 70 Hz refresh rate) placed 50 cm in front of the subject. Stimuli were calibrated with a Minolta CA-96 colorimeter.

Task and stimuli

The task used in this study is a modified version of the double-step task (Aslin and Shea 1987; Becker and Jurgens 1979; Hou and Fender 1979; Lisberger et al. 1975; Murthy et al. 2000; Ray et al. 2004) and consists of two kinds of trials: *no-step* trials in which only one target is presented (i.e., target is 'not stepped' to another location) and *step* trials in which two targets are presented in succession (or in other words, a target is presented and then "stepped" to a different location). Step trials in our task differ from those of earlier studies in two main respects: the initial and final targets are of different colors so that it is easier to spot the "second" target, especially at shorter target step delays, and redirect gaze to it; and the initial target does not disappear with the appearance of the final target. This manipulation implicitly encourages erroneous saccades to the initial target during trials when subjects fail to make a direct saccade to the final target.

No-step trials constituted 60% of the total number of trials in each session. In these trials (Fig. 1*B*), following fixation at a white box ($0.3 \times 0.3°$) on the center of the screen for a random duration (ranging from 300 to 800 ms), a single, green target ($0.5 \times 0.5°$; 0.9 cd/m^2) was presented. The location of targets was randomized such that they could appear in any one of the four positions specified by a radial distance of 21° and polar angles of 45, 135, 225, or 315° from the fixation point (Fig. 1*A*).

The remaining forty percent of trials in a session constituted of step trials, which were further categorized into two subtypes—*no-shift step* and *target-shift step* trials. Each of the two trial types occurred with an equal probability and was randomized with the no-step trials such that subjects could not predict or anticipate the appearance of the targets. In a no-shift step trial (Fig. 1*C*), following fixation for a random duration, and presentation of the initial green target at one of the four positions specified for a no-step trial, a final red target ($0.5 \times 0.5°$; 0.9 cd/m^2) appeared randomly at any one of the remaining three positions. In a target-shift step trial on the other hand, after the presentation of initial and final targets following fixation (just as in a no-shift step trial), the final red target was stepped to a new position during the execution of the first saccade (Fig. 2*A*). The "shifted" position of the final target (referred hereafter as the new position of the final target) was at a radial distance of 21° and a polar angle of either 0 or 180° from the fixation point depending on whether the original position of the final target (referred hereafter as the old position of the final target) was on the right or left hemi-field, respectively. In other words, for old positions of the final target specified by polar angles 45 and 315°, the new position was always at polar angle 0° (Fig. 2*B, left*) while for old positions of the final target specified by polar angles 135 and 225°, the new position was always at polar angle 180° (Fig. 2*B, right*). Notably, the polar angle shift of 45° in the location of the final target amounted to a vertical linear displacement of 14.5° from its old position. Only those trials in which the target shifted strictly during the execution of the first saccade, i.e., after it began but before it ended, were used for all analysis. Typically, this shift occurred ~46 ms after the first saccade

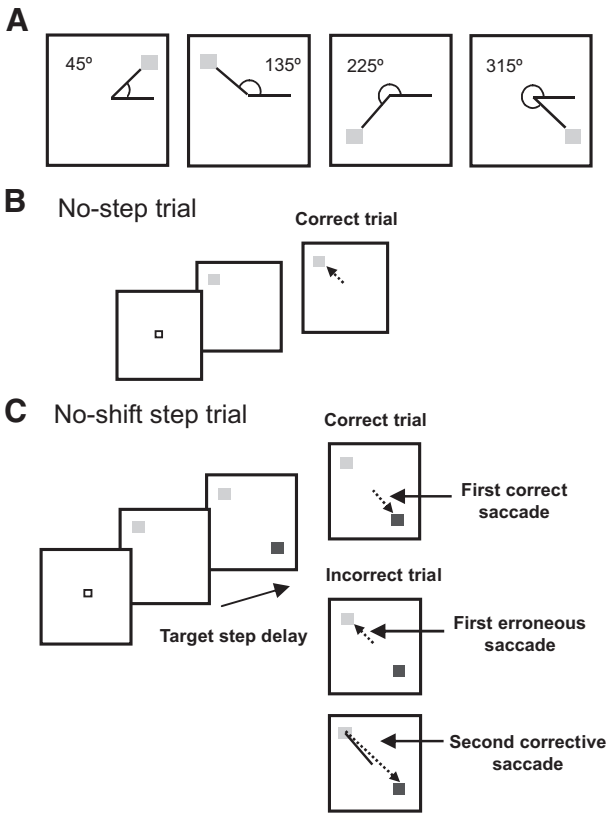

FIG. 1. *A*: probable locations of the target (light gray square) at polar angles 45, 135, 225, or 315° from the fixation spot. *B*: temporal sequence of events in a *no-step* trial: following fixation at a white box on the center of the screen for 300–800 ms, a green target (light gray square) appears at any one of the positions shown in *A*. The trial is scored as correct if subjects make a saccade to the target as soon as possible. *C*: temporal sequence of events in a *no-shift step* trial: fixation is followed by the appearance of an initial green target (light gray square) as in a *no-step* trial. After a variable target step delay (20–200 ms), a final red target (dark gray square) appears on any 1 of the 3 remaining locations shown in *A*. Subjects are instructed to cancel the saccade to the initial target and look directly at the final target. *Right*: dotted lines with arrowhead show different saccadic responses in a no-shift step trial—1st correct saccade to the final target (*top*), 1st erroneous saccade to the initial target (*middle*), and 2nd corrective saccade to the final target (*bottom*).

began but well before it terminated. Consistent with the phenomenon of saccadic suppression (Burr et al. 1994; Holt 1903; Krauskopf et al. 1966; Latour 1962; Riggs et al. 1974), the shift in the position of the final target was not perceived by subjects during the execution of the first saccade. Also luminosity of the target was kept low to minimize any after-shift flash effects, for example, due to phosphor persistence.

The target step delay, i.e., the time of the first appearance of the final target relative to the initial target, was varied randomly from ~20 to 200 ms. This manipulation controlled the extent of reprocessing time (i.e., the duration between the appearance of the second target and the beginning of the 1st saccade) such that in general, longer target step delays were associated with shorter reprocessing times and vice versa (Fig. 2*A*). Subjects were also encouraged to respond quickly by means of verbal feedback from the experimenter in case they took >400 ms to make the saccade in no-step trials.

Subjects were given both verbal and written instructions with some practice trials (~50) before data were collected. They were instructed to make quick saccades to the green target as soon as it appeared following a brief fixation. In case the red target appeared, they were asked to cancel the planned saccade to the green target and instead, make a direct saccade to the red target. This is why this version of the double-step task is also known as the redirect task (Ray et al. 2004).

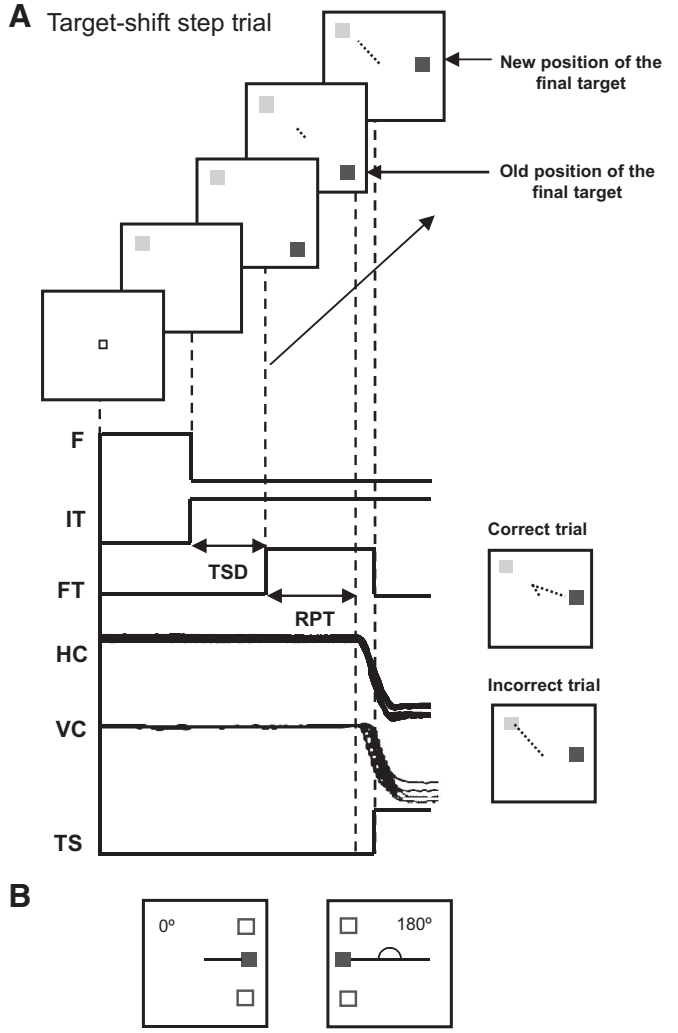

FIG. 2. *A*: sequence of events aligned to their representative time of occurrence in a *target-shift step* trial. *Top*: following fixation, the initial green target (light gray square) and the final red target (dark gray square) appear just as in a no-shift step trial. During the execution of the 1st saccade, the final target shifts vertically to a new position. *Bottom left*: solid vertical line denotes the beginning of the trial. Horizontal lines trace the time of presentation of the fixation box (F), initial target (IT), final target (FT), the occurrence of horizontal (HC) and vertical (VC) components of the 1st saccades, and the shift of the final target (TS). TSD, target step delay; RPT, reprocessing time. *Bottom right*: trials were scored as correct only if the subjects made the 1st saccade directly to the new position of the final target and deemed incorrect if the 1st saccade reached the initial target. *B*: final target locations before and after the position shift. Final target locations after the shift (dark gray square) are at polar angles 0° (*left*) or 180° (*right*) from the fixation point depending on whether the original position of the final target (unfilled square) were on the right or left hemi-field, respectively.

On average, each session lasted for ~45 min in which subjects performed ~500 trials with a 5- to 10-min break in between halves of the session. Each session was checked for a performance criterion mentioned in RESULTS before being combined for further analysis. The total number of sessions ranged from 4 to 12 for each subject to obtain sufficient number of trials to perform the analyses.

Trials were scored as successful, and conveyed to subjects by auditory feedback, if subjects made the first saccade to the "green" target in a no-step trial, to the "red" target in a no-shift step trial, and to the new position of the "red" target in a target-shift step trial, fixating the respective targets within an electronic window of ±6.5° centered on the target. Saccades to the new position of the target were scored as correct as against the old to dissociate effects of positive feedback on the programming of saccades to the old final target position. However, for off-line evaluation of subjects' performance and plotting the probability of an error as a function of the target step delays using step trials (see RESULTS), trials in which the first saccade went directly to the red target (irrespective of old or new position) were considered correct, whereas those in which the first saccade went to the green target were considered erroneous. Also corrective saccades were classified off-line based on their endpoints in the old versus new position of the final target using a spatial window of ±4° from the center of the respective target location. Because the old and new final target positions were always 14.5° apart and the spatial window used to delimit these saccades off-line were separated by 6.5°, the chance of misclassifying a saccade, considering that the spatial accuracy in the vertical direction was 0.9°, would occur only if the saccade endpoint were to be >7 SD of noise from the edge of the window demarcating the old and new positions of the final target.

Off-line analysis was done using Matlab (Mathworks). The analogue eye position data were smoothened and blinks removed. A velocity threshold of 30°/s was used to demarcate the beginning and end of saccades. All blink-perturbed saccades were eliminated from analysis. All statistical tests were done using Matlab.

RESULTS

Task performance analysis

Figure 1 describes the redirect task used to understand how behavioral control is exerted to achieve goal-directed movements. In this task, subjects were instructed to make a saccadic eye movement to an initial green target as soon as it appeared. However, in case a red target appeared subsequently, subjects were asked to cancel the planned saccade to the green target and direct their gaze to the red target. The critical variable that varied across trials was the time between the appearance of the initial and the final target, called target step delay, and was used to assess the performance of each subject. We plotted the probability of making a saccade to the initial target in step trials (known as the 1st erroneous saccade from hereon) as a function of increasing target step delays. Figure 3 shows the performance curves of fourteen subjects in the task quantified by fitting the best-fit cumulative Weibull function

$$W(t) = \gamma - (\gamma - \delta) \cdot e^{(-(t/\alpha)\beta)}$$

where t is the target step delay; α is the time at which the function reaches 64% of its full growth; β is the slope; γ is the maximum value of the function, and δ is the minimum value of the function. As expected, the inability to cancel the first erroneous saccade to the initial target, and thus the probability of making an error increases with an increase in the target step delay. Because the term $(\gamma - \delta)$ describes this increase in the probability of making an error as a function of target step delay, we used it as a value to quantify the degree of cancellation, and hence, the level of task performance among subjects. Only those individual sessions of each subject in which the degree of cancellation changed considerably with increasing target step delay and thus had a $(\gamma - \delta)$ value of ≥ 0.5 or more were pooled for the final analysis of performance. Because the probability of making an error is the lowest at the smallest target step delay, for the pooled data, we chose an additional criterion of $\delta < 0.3$ to sift out subjects who performed sufficiently well. Of the 14 subjects tested, 10 satisfied the preceding criteria and were included for subsequent anal-

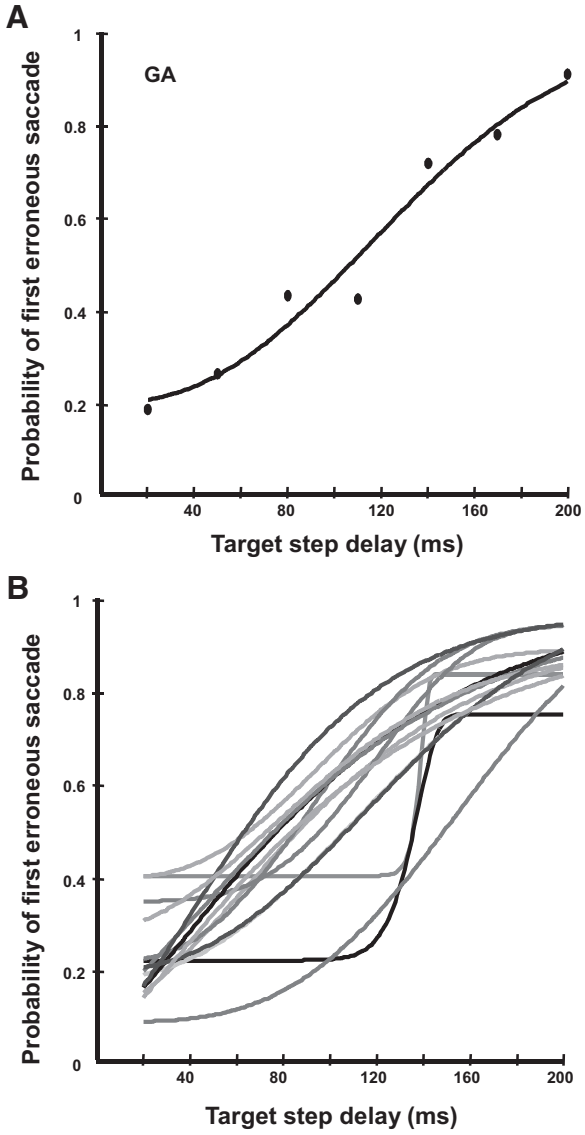

FIG. 3. Plots showing subjects' performance in *step* trials. Probability of making the 1st erroneous saccade to the initial target is plotted as a function of the target step delays (*A*) for a representative subject (*B*) for all 14 subjects. The target step delays were calculated taking into account the spatial location of the targets and separated into bin sizes of ± the refresh rate of the monitor (~14 ms). Data fit by Weibull function (see text for details). Probability of making the 1st erroneous saccade increases with increasing target step delays.

ysis. For eight of these subjects, >80% of the individual sessions were pooled in for final analysis; however, for *subjects GA* and *SP,* the final analysis is based on 50% of the total recorded sessions. The four subjects excluded from further analysis were *NS, CD, VK,* and *DT.*

Parallel programming during error correction

While the primary form of control required for goal-directed behavior in the redirect task is inhibition of a partially prepared saccade to the initial target, a secondary form of control is manifest on trials in which subjects fail to suppress this saccade. In such trials (see Fig. 1*C, bottom right*), subjects failing to cancel the initial saccade to the green target, achieve the "goal" of the task through a sequence of two saccades: the first erroneous saccade to the green target, followed by a quick second corrective saccade to the red target. According to the logic of parallel, independent programming of saccades, the preparation of the second saccade, following the appearance of the second target, begins while the first saccade is still being programmed and executed (Becker and Jurgens 1979; Ray et al. 2004). The degree to which parallel processing can take place depends on the interval between the appearance of the second target and the beginning of the first saccade called the reprocessing time (Fig. 2). This is the time available to the saccadic system to plan the second saccade while the first saccade is still in the pipeline. If the second saccade is programmed in parallel during the reprocessing time, increasing reprocessing times should be associated with decreasing intersaccadic intervals.

The no-shift step trials were used to test whether the second corrective saccades were being programmed in parallel with the first erroneous saccades. Those trials of a subject with intersaccadic intervals more than the no-step reaction time were removed from this analysis because saccades following such high intersaccadic intervals could not have possibly been processed in parallel. In addition, only trials with reprocessing times <200 ms were included for all analyses because the degree of parallel processing tends to reach a plateau at longer reprocessing times (Becker and Jurgens 1979; Ray et al. 2004). Figure 4*A* shows the plot of intersaccadic interval as a function of reprocessing time for a representative subject. We observed that shorter intersaccadic intervals were associated with longer reprocessing times and this inverse relation was quantified by fitting the data with a straight line (slope = −0.47, $r^2 = 0.17$, $n = 52$, $P < 0.002$). A similar significant trend was obtained for all ten subjects (Fig. 4*B,* slope median = −0.43, min = −0.16, max = −0.79, r^2 median = 0.18, min = 0.04, max = 0.41, $P < 0.02$). These data replicate previous work (Becker and Jurgens 1979; McPeek et al. 2000; Ray et al. 2004), indicating that some aspect of the second corrective saccade was processed during the preparation of the first erroneous saccade itself.

We used the target-shift step trials to determine specifically if the brain can begin the motor preparation for the second corrective saccade while the first erroneous saccade was being planned. The logic used was as follows: if motor preparation cannot occur in parallel and commences only after the first saccade, the second corrective saccades should always be directed to the new, shifted position of the final target. However, if motor preparation of the second corrective saccade can occur in parallel and begins before the final target shifts, one should find instances when these saccades end up at the old position of the final target. Across subjects we found that while in some trials the second corrective saccades landed up in the new position of the final target (Fig. 5*A*), in others, they were directed at the old location of the final target (Fig. 5*B*) consistent with the second alternative proposed above that motor preparation of the second corrective saccade may begin during the preparation of the first erroneous saccade itself.

To determine whether directing gaze at the old position of the final target is a consequence of parallel motor preparation, the propensity for such behavior was examined in relation to the reprocessing time. Because at longer reprocessing times the extent of preparation of the motor command is likely to be more advanced, the tendency to execute the second corrective saccade to the old final target position should be higher. We tested this by

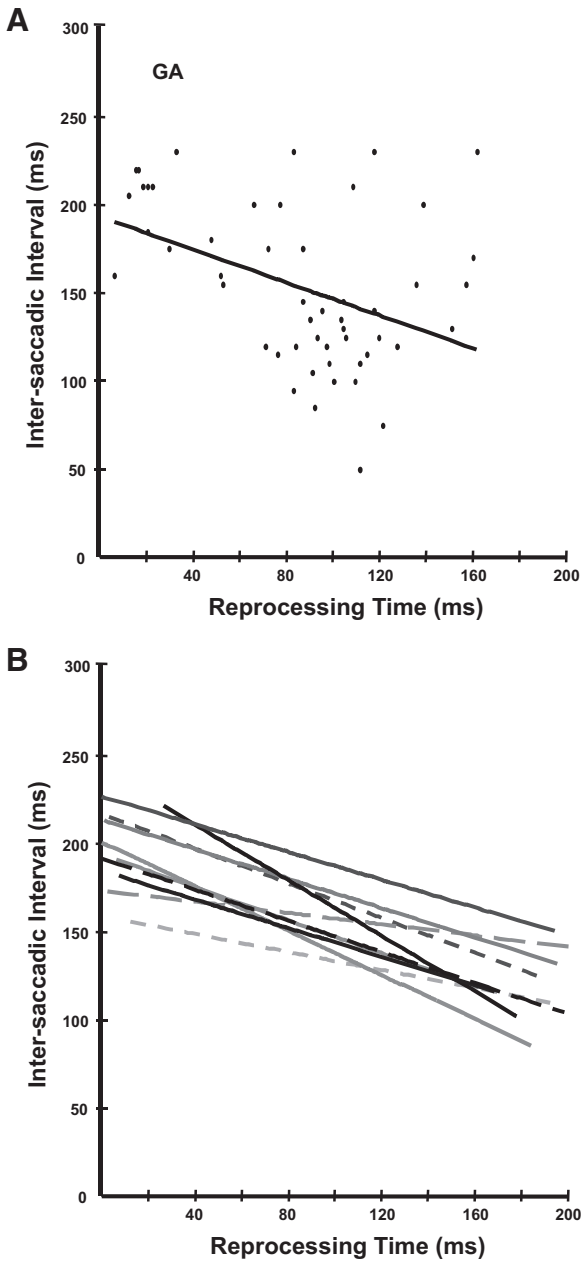

FIG. 4. Plots of the reprocessing time vs. intersaccadic interval between the error and the corrective saccade in no-shift step trials (*A*) for a representative subject (*B*) for 10 subjects. Data quantified by a linear fit highlight the decrease in intersaccadic intervals as the reprocessing time increases.

plotting the probability of reaching the old position of the final target as a function of reprocessing time. As described before, only those second corrective saccades the endpoints of which were within a vertical distance of ±4° from the center of the old final target location were defined as having gone to the old position of the final target. Trials were separated into equal bins on the basis of their reprocessing times and the probability of the second corrective saccade reaching the old final target position versus the new one was calculated. Figure 5*C* shows that for 7 of 10 subjects. this probability increased as a function of reprocessing time indicating that motor preparation of the corrective saccade may occur in parallel with the preparation of the first erroneous saccade. However, for three subjects the trend was variable. While for two subjects (*GA* and *MS*) the probability settled down to a lesser value after an initial increase, for *subject SP* the relationship showed a completely opposite trend; the probability started with an atypically high value at shorter reprocessing times (>0.6) and went on to decrease with increasing reprocessing times.

Dynamics of concurrent saccade preparation during error correction

A shift in the position of the final target during the programming of a second corrective saccade offers an excellent opportunity to study the nature of concurrent processing during error correction. Because of the shift in final target location, the oculomotor system is effectively faced with two alternatives for the corrective saccade's destination—either the old or the new location of the final target. Consistent with this assumption we often observed the second corrective saccade endpoints at either near the new position of the final target or near the old position of the final target. However, we also found that in many trials the second corrective saccades landed up mid-way between the old and new positions of the final target (Fig. 6*A*). This is contrary to what one would expect if motor preparation producing the second corrective saccade represents the outcome of a simple binary choice between the two potential targets.

We quantified the preceding behavior by plotting the endpoints of the second corrective saccades against increasing reprocessing time. The analysis was facilitated by the spatial arrangement of the shifted position of the final target with respect to its original position. As described earlier, the shift was always vertical (up or down) and to the horizontal right or left of the fixation box, corresponding to the hemi-field at which the final target was originally presented. Thus we plotted the vertical distance of the corrective saccade's endpoint from the corresponding old position of the final target for data from all four quadrants as a function of reprocessing time. Figure 6*B* shows the plot for a representative subject. The data points at 0 and 14.5° on the *y* axis (gray and black horizontal bars) denote saccadic endpoints with ordinate values corresponding to the center of the old and new positions of the final target, respectively**;** the vertical difference between the two being the magnitude of target displacement in target-shift step trials. At the shortest reprocessing time, the second corrective saccades' endpoints were found near the new position of the final target; while at longer reprocessing times, the endpoints were closer to the old final target position. Also, in all instances, we obtained a number of trials in which the second corrective saccades landed somewhere between the two potential target positions. Thus amid variability, we noticed a subtle but consistent shift in the mean endpoints as a function of reprocessing time which was quantified by fitting the data with a straight line (Fig. 6*B,* slope = -0.05, $r^2 = 0.15$, $n = 135$, $P < 0.001$). We observed a significant negative correlation for 8 of 10 subjects (Fig. 6*C,* slope median = -0.03, min = -0.03, max = -0.06, r^2 median = 0.085, min = 0.04, max = 0.24, $P < 0.02$), representing the gradual shift of the corrective saccades' endpoints from the new to the old position of the final target with increasing reprocessing time. These data suggest that when the oculomotor system has to choose between two potential alternatives during

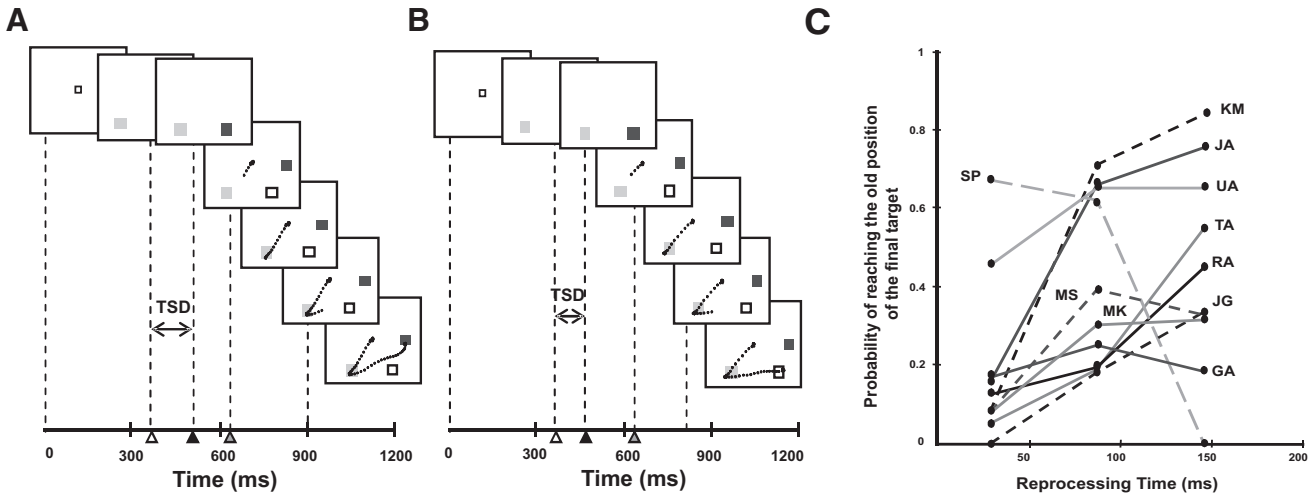

FIG. 5. Time-aligned sequence of events in a target-shift step trial when the corrective saccade is made to the new (*A*) and old position of the final target (*B*). The presentation of the initial target (light gray square), final target (dark gray square), and target-shift are denoted on the time axis by white, black and gray triangles, respectively. An unfilled square represents the old position of the final target for the convenience of the reader. *C*: plot showing the probability of the corrective saccade to the old position of the final target as a function of reprocessing time for all subjects.

error correction, the outcome of the decision process may be a consequence of some form of averaging. For the remaining two subjects, we observed either a correlation that was not significant (*subject GA*) or a positive trend (*subject SP;* slope = 0.06) of corrective saccades' endpoints as a function of reprocessing time. Because *SP*'s data in both analyses was not consistent with the notion of parallel processing of corrective saccades, it was excluded from subsequent analysis.

Time course of concurrent corrective saccade preparation

We estimated the time at which the second corrective saccade's preparation begins for each subject on the basis of the LATER model (Carpenter and Williams 1995; Hanes and Carpenter 1999; Hanes and Schall 1996; Reddi et al. 2003), using only corrective saccades to the old final target position for our estimation because concurrent planning is most likely to have begun the earliest for these trials. The LATER model envisions the delay responsible in generating a saccade as the time taken by the decision variable to linearly accumulate information from the environment till it reaches a criterion level of activation at which time the saccade is executed. We assumed the rates at which the decision variables responsible for generating a correct saccade in the no-step trial and a corrective saccade in a target-shift step trial, respectively, rise toward the activation threshold to vary with the same distribution and thus give rise to the same latency profiles. Now, if the second corrective saccade preparation began as soon as the final target was presented (Fig. 7*B*), then the predicted reaction times of these saccades fail to match the longer reaction times observed for all subjects. Thus it is likely that either the $GO_{Corrective}$ process itself started with a delay from the time of final target presentation or it began early but was slowed down later in the process. However, because the LATER model assumes the rate of accumulation to vary in a Gaussian fashion across trials but to be invariant during the latency of any one trial, the observed longer reaction times of corrective saccades was assumed to be caused by the delay in the onset of the $GO_{Corrective}$ process alone. By shifting the onset of $GO_{Corrective}$ process iteratively we calculated the minimum delay (denoted by *d* in Fig. 7*C*) required for the predicted reaction times to be the same as the observed reaction times of the corrective saccades for any set of trials.

Trials were separated on the basis of their reprocessing times, which were divided into bins of 40 ms each. Only bins with at least three trials were selected. By subtracting the onset delay from the mean reprocessing time of trials in the corresponding bin, we obtained the onset of corrective saccade preparation relative to the start of the error. Figure 8 shows the frequency histogram of the onset of corrective saccade preparation from the start of the first erroneous saccade to the initial target for all subjects across all reprocessing times. Consistent with the notion of parallel programming, 47% of the times, planning for correction was estimated to have begun before the onset of the first erroneous saccade itself, whereas in 97% of the cases, it was estimated to have begun before or during the execution of this erroneous saccade (mean erroneous saccade duration = 54 ms), i.e., in the absence of any sensory feedback.

If predictive processing of the corrective saccade fully depends on the time available for reprocessing the target step, one would expect a completely linear and inverse relation (slope of −1) between the onset of concurrent preparation of correction and reprocessing time (Fig. 9*A*) such that greater the available reprocessing time, the earlier the corrective saccade preparation would begin. We examined this possibility by plotting the onset of corrective saccade processing as a function of reprocessing time for each subject (Fig. 9) such that all negative values on the *y* axis refer to the onsets of correction before the start of the error while positive values correspond to the onsets after the error began. Trials were separated on the basis of their reprocessing times and only bins with at least three trials were selected. Figure 9*A* shows the data for one representative subject quantified by a linear fit (slope = −0.78, r^2 = 0.92, P = 0.02). A negative slope obtained for eight of nine subjects (Fig. 9*B,* slope median = −0.85, min = −0.03, max = −1.23,

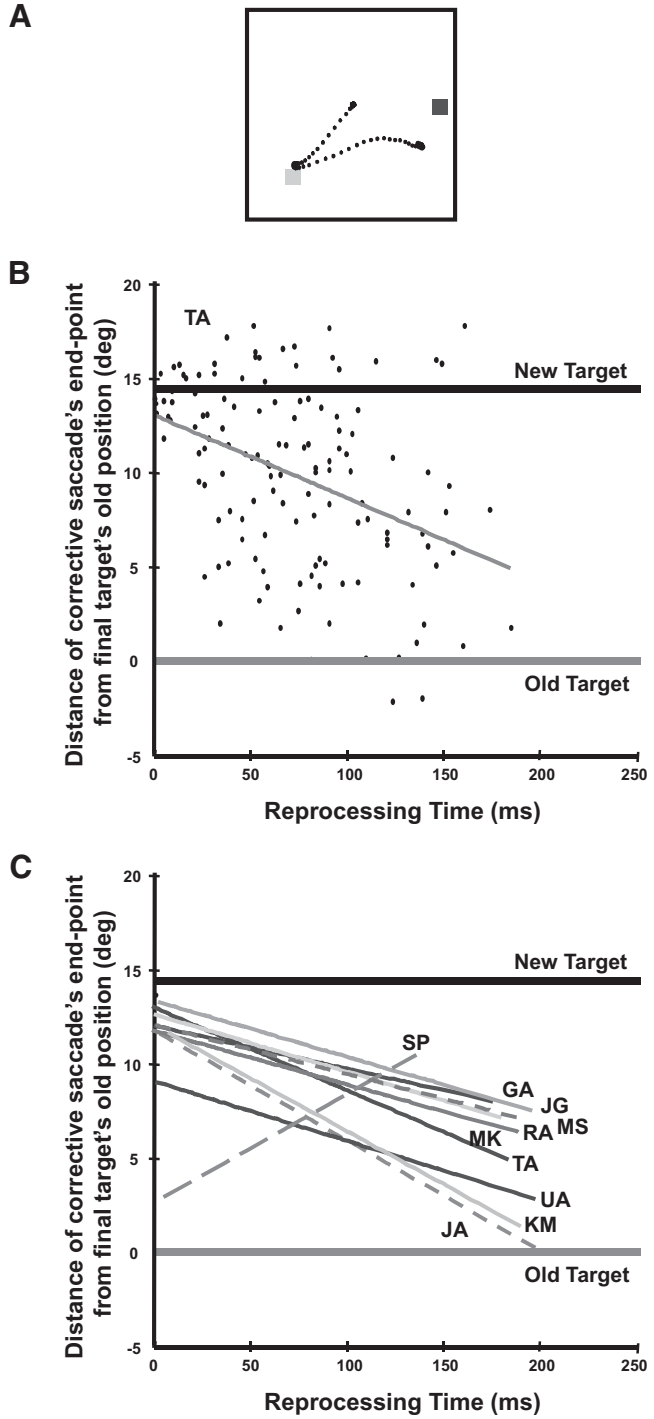

FIG. 6. *A*: behavioral instance of a corrective saccade made midway between the old and new positions of the final target. *B*: vertical distances of the corrective saccade endpoints from the corresponding old positions of the final target are plotted as a function of reprocessing time for a representative subject and for all subjects (*C*). Linear fits of data points for 9 of 10 subjects show a gradual shift of the corrective saccades' endpoints from the new to the old position of the final target with increasing reprocessing time.

r^2 median = 0.92, min = 0.3, max = 0.98) indicates that the preparation for correction began sooner with respect to error in trials with longer reprocessing times as compared with trials with shorter reprocessing times. However, negative slope values of less than unity suggest that in trials with high reprocessing time, planning for correction did not begin as early as was expected (Fig. 9*A*), and thus the onset for concurrent planning of the second corrective saccade is not completely governed by the time available for reprocessing the target step per se.

Because error correction is occurring while the oculomotor system is attempting to inhibit an inappropriate saccade, we also tested the relation between target step delay and the delay associated with the planning of the second corrective saccade. For this, we recalculated the onset delays as previously described (Fig. 7) for sets of trials separated on the basis of target step delay such that there were at least three trials per bin. Figure 10*A* shows the delay in the onset of corrective saccade preparation, relative to the final target presentation, plotted as a function of target step delay for a representative subject. Data are quantified by a linear fit (slope = −0.71, r^2 = 0.81, P = 0.05). A similar negative slope obtained for eight of nine subjects (Fig. 10*B,* slope median = −0.63, min = −0.01, max = −0.92, r^2 median = 0.66, min = 0.01, max = 0.92) suggests that at shorter target step delays, when the probability of successful inhibition is high, subjects tend to wait longer before preparing for correction. Conversely, at longer target step delays, when the likelihood of error is higher, subjects tend to wait less before preparing for correction.

DISCUSSION

In the present study, we used a modified double-step redirect task to probe the dynamics of error correction in relation to the erroneous initial saccade. In the process, we obtained four important results. First, we demonstrated that motor preparation for the second corrective saccade may proceed in parallel with the preparation of the first erroneous saccade. Second, we examined the nature of predictive saccade programming leading to error correction and found that the spatial outcome of the decision process with two target alternatives can be a consequence of averaging i.e., taking values between the two target locations. Third, we explored the time course of concurrent processing of error correction based on the LATER model and found that across subjects, 97% of the estimated onsets of corrective saccade preparation were before or during the execution of the erroneous saccade, i.e., without requiring sensory feedback. Fourth, we investigated the relation between the onset of programming correction in parallel and the reprocessing time/target step delay and observed longer delays in initiating correction at shorter target step delays and vice versa. We discuss and interpret these findings in the following text.

Parallel programming during error correction

While the occurrence of parallel visual analysis is known to occur during the preparation of a saccade in simple visual displays (Findlay and Harris 1984) and during reading (Morrison 1984; Rayner 1998; Tan et al. 2005), clear evidence of concurrent motor preparation is still forthcoming, particularly in the context of error correction. Previously, the presence of very brief intersaccadic intervals approaching 0 ms in the double-step and visual search paradigms have been cited as evidence of concurrent motor preparation (Becker and Jürgens 1979; Findlay and Harris 1984; Goossens and Van Opstal 1997; Hooge and Erkelens 1996; McPeek et al. 2000; McPeek

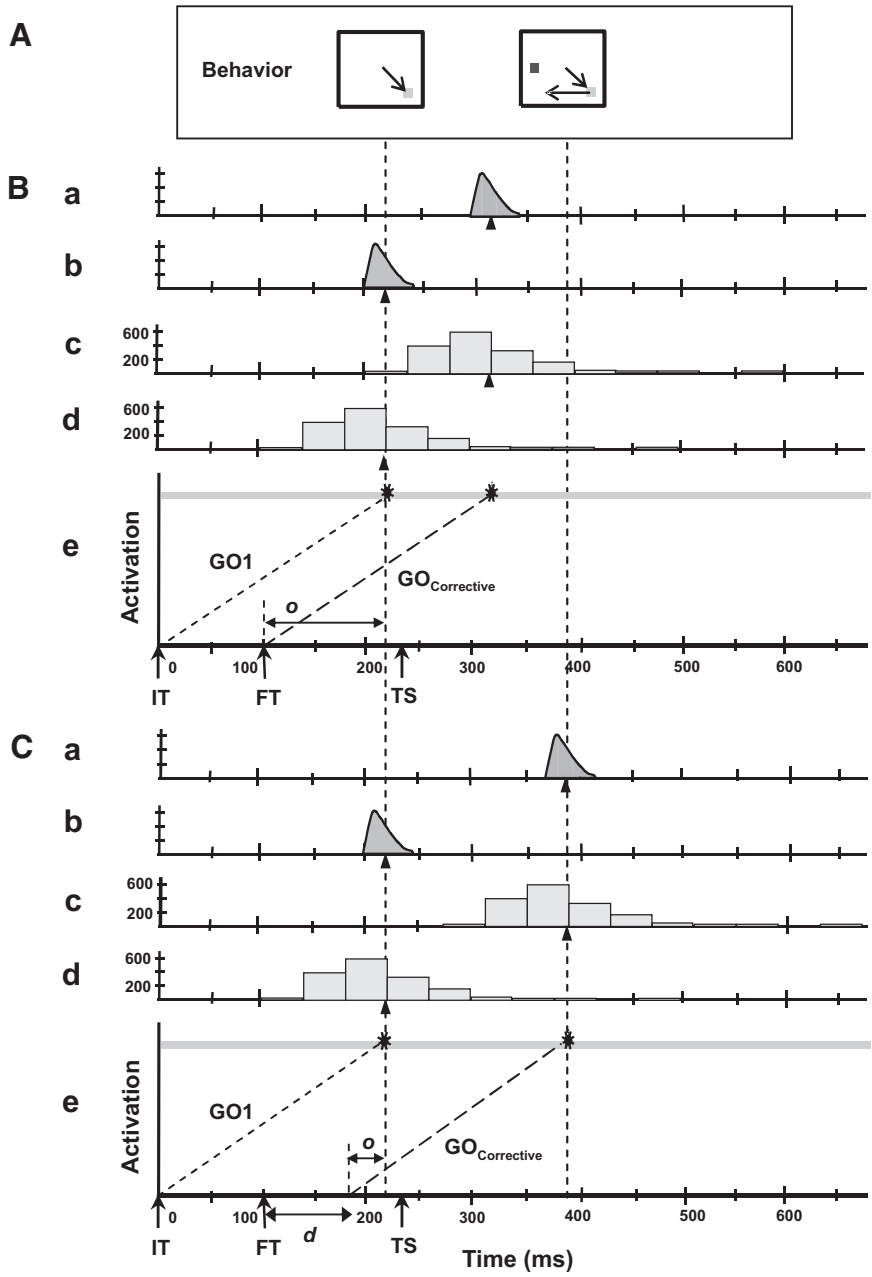

FIG. 7. LATER model based estimation of the onset of corrective saccade preparation. *A*: behavioral representation of a saccade to the green target (light gray square) in a no-step trial and a corrective saccade to the old position of the final target (dark gray square) aligned to their actual mean onset times (in a representative subject, *JA*) on the *x* axis of *B* and *C*. In *B* and *C*, the hashed, skewed reaction time distribution of *a* represents the latency profile of corrective saccades to the old position of the final target assumed to be the same as that of saccades made to single targets in no-step trials (represented by the gray, skewed reaction time distribution in *b*). *Panels c* and *d* show the actual no-step reaction time distribution of *subject JA* with respect to the presentation of the final target and initial target, respectively. Black triangles on the *x* axis of *a–d* mark the mean of the reaction time distributions. In *e,* the horizontal gray band represents the activation threshold and GO_1 (short-dashed line) and $GO_{Corrective}$ (long-dashed line) represent the rise to threshold of the decision variables responsible for generating a correct saccade in the no-step trial and a corrective saccade to the old final target position in a target-shift step trial, respectively. Vertical arrows on the *x* axis mark the typical time stamps of the following events: initial target presentation (IT), final target presentation (FT), and target shift (TS). *B*: if the $GO_{Corrective}$ process were to begin as soon as the final target was presented, then the predicted reaction times of these corrective saccades fail to match the longer reaction times observed in case of these saccades. Moreover, because the LATER model does not permit a change in the rate of accumulation within a trial, the longer reaction times can only be explained by assuming a delay in the onset of the $GO_{Corrective}$ process from the time of final target presentation. *o* represents the onset of corrective saccade preparation with respect to the start of the error at each such delay. *C*: by shifting the onset of the $GO_{Corrective}$ process iteratively, the minimum delay (denoted by *d* in Fig. 6*C*) required for the predicted reaction times to be the same as the observed reaction times of the corrective saccades is calculated.

and Keller 2002; Minken et al. 1993; Port and Wurtz 2003; Theeuwes et al. 1999; Viviani and Swensson 1982). However, other explanations cannot be ruled out. For example, in response to target steps in rapid succession, a package of two closely spaced saccades may be programmed (Becker and Jurgens 1979; Carlow et al. 1975; Levy-Schoen and Blanc-Garin 1974) as a chunk. Such a mode of packaged programming may be particularly facilitated if stimuli are all presented before the first gaze shift in a reasonably predictable manner so that the desired sequence of saccades is produced as a unit (Zingale and Kowler 1987). Alternatively, curved saccades with brief intersaccadic intervals, which have been observed in visual search as well as double-step tasks (e.g., Becker and Jurgens 1979; Findlay and Harris 1984; McPeek et al. 2003; Minken et al. 1993; Port and Wurtz 2003; Van Gisbergen et al. 1987), may not necessarily imply concurrent processing of two saccades but, rather, may reflect on-line correction of a single saccade.

However, other saccadic experiments have been conducted that raise the possibility of concurrent motor preparation (Vergilino and Beauvillain 2000). In the latter study, the authors examined the preprocessing of a refixation saccade to target words as the word length was either increased or reduced at different times after the primary saccade. It was found that refixation saccades produced within the first 150 ms after the length change were computed on the basis of the initial length whereas those triggered after 150 ms of the length change considered the final length of the target word. Evidence for concurrent processing of saccades was also shown by McPeek et al. (2000) in a visual search task where saccades were often erroneously made to distractors instead of the odd colored target as a result of priming. When the position of the target

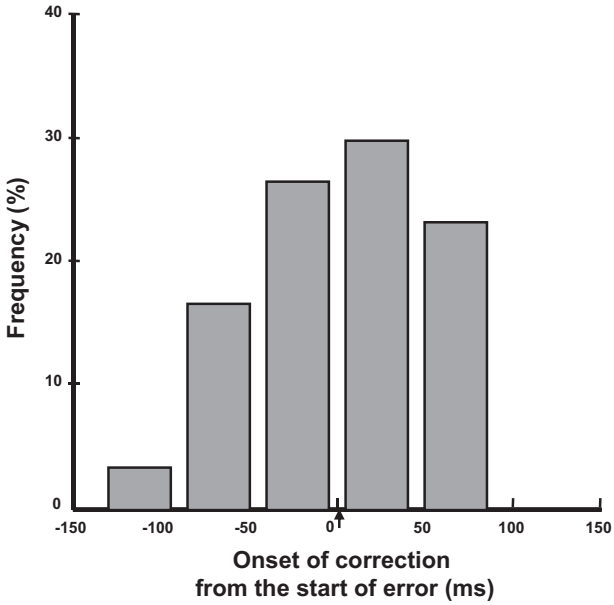

FIG. 8. Frequency histogram of the onsets of corrective saccade preparation with respect to the beginning of the erroneous saccade for all subjects, across all reprocessing times. Bars on the negative scale of the *x* axis represent onsets of corrective saccade preparation before the start of the error, whereas bars on the positive scale of the *x* axis denote onsets after the error began. The arrow on the *x* axis marks the mean onset of corrective saccade preparation across all subjects (1 ms after the start of the error).

was switched with the distractor during the execution of error, corrective saccades were often directed to the original position of the target. Although this is indicative of parallel motor preparation of a saccade in a sequence, because both the original target and the distractor were presented simultaneously before the first saccade and the corrective saccade preparation was internally triggered, the time course of the error correction could not be investigated. Polli et al. (2006) also reported short-latency "self-corrections" in the anti-saccade task. Although the intersaccadic interval of these saccades (~130 ms), produced without sensory feedback, was similar to what we obtained for corrective saccades to the old final target position (mean across subjects = 143 ms), being internally triggered, the extent of concurrent processing occurring in these corrections could not be studied in greater detail.

By using a modified double-step task, we have shown that when the position of the final target was shifted to a new location during the execution of the erroneous saccade, the probability of the corrective saccade to the original location of the final target increases with the reprocessing time—the time available for planning the second saccade before the error onset. Based on the LATER model as well as the positions of corrective saccade endpoints produced as a result of varying target step delays, we were also able to estimate the earliest onset of corrective saccade preparation across subjects. Because our estimates derive from behavioral data it is necessarily indirect, and its validity is based on two critical assumptions. The first assumes that the LATER model (Carpenter and Williams 1995; Reddi et al. 2003) is a fairly accurate descriptor of saccadic preparation. This assumption seems reasonable in light of the behavioral (Gold and Shadlen 2001; Hanes and Carpenter 1999; Reddi and Carpenter 2000) and neurophysiological (Gold and Shadlen 2000; Hanes and Schall 1996; Kim and Shadlen 1999) evidence in support of the LATER model. This notwithstanding, we acknowledge that the model has not been adequately tested on sequential saccades where there has been a report suggestive of a potential shortcoming (Van Loon et al. 2002). However, because the reported violations mainly concern fitted response distributions, whereas our estimates are based on fitting the central tendency, we assume that such shortcomings do not severely compromise our estimates. The second assumption concerns the use of the no-step saccade distribution to model corrective saccades despite evidence of a cognitive influence that facilitates saccade reaction times during error correction (Ray et al. 2004). However, taking this influence into account, by reducing the mean no-step saccadic reaction time by 25 ms, still places the onset of predictive programming before the end of the erroneous saccade or before sensory afferents have a chance to redirect gaze. The same would hold true even if we incorporate an afferent delay of 50–60 ms into the LATER model (Ludwig et al. 2007). Thus these potential shortcomings notwithstanding, this effort, to the best of our knowledge, represents the first instance where an estimate of error correction planning has been made in humans performing a motor task.

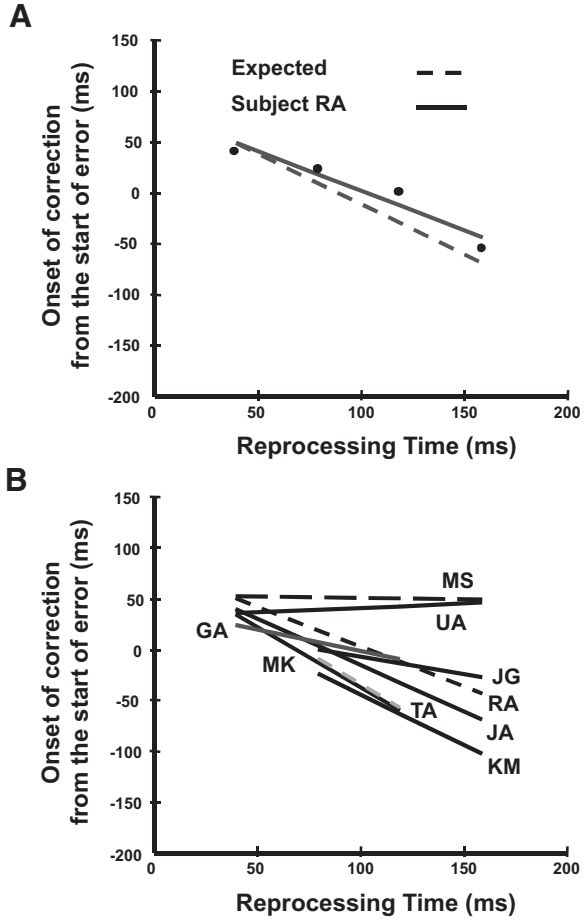

FIG. 9. Plots showing the onsets of corrective saccade preparation from the beginning of the erroneous saccade (represented by the 0 on the *y* axis) as a function of reprocessing time. All negative values on the *y* axis refer to the onsets of corrective saccade planning before the start of the error, whereas positive values correspond to the onsets after the error began. *A*: linear fit data from a representative subject (—) and the slope expected (- - -) if predictive processing of the corrective saccade completely depends on the reprocessing time. Subject's data has a negative slope of <1. *B*: negative slopes obtained for 8 of 9 subjects show that the preparation for correction begins earlier relative to the error as the reprocessing time increases.

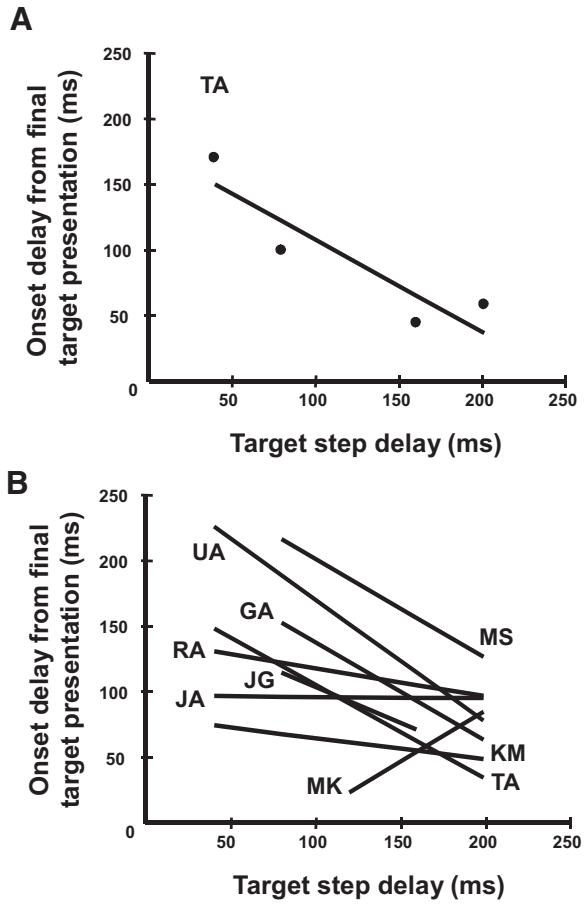

FIG. 10. Plot showing the delay in the onset of corrective saccade preparation from the final target presentation as a function of target step delay. The data are fit by a straight line (*A*) for a representative subject (*B*) for all subjects. Negative slopes obtained for 8 of 9 subjects show that delay in onset of corrective saccade preparation decreases with increasing target step delays.

A more direct evidence of concurrent motor preparation can only be revealed by physiological studies that can demarcate neural activity specifically linked to the generation of a saccade. In our mind, such motor preparation may correspond to the activity of movement-related cells in the frontal eye fields (Bruce and Goldberg 1985; Thompson et al. 1996), superior colliculus (Dorris et al. 1997; Munoz and Wurtz 1995; Paré and Hanes 2003), and possibly the lateral intraparietal cortex (Mazzoni et al. 1996). However, we presume that such motor preparation, if not sufficiently advanced, is not necessarily a commitment to make a saccade and hence may not include the presaccadic burst activity described in superior colliculus. Nevertheless because the activity of these cells is a good predictor of saccade reaction time (Hanes and Schall 1996) unlike visual cells (Murthy et al. 2001; Sato et al. 2001), we assume they reflect some aspect of motor preparation or motor intention. Our current results extend our findings from monkey neurophysiological experiments in which movement-related activity in the frontal eye fields obtained for corrective saccades during a similar double-step like task sometimes began before the completion of the error (Murthy et al. 2007).

Generation of accurate corrective saccades, with latencies less than the latency of visual feedback, has also received much empirical and theoretical interest (Hallett and Lightstone 1976a; Sparks and Mays 1983) because such correction is thought to require use of a movement vector, which must be updated for the change of eye position produced by the erroneous saccade. One mechanism hypothesized to account for fast on-line error correction may be a comparison of the spatial location of the goal with the current eye displacement. This potentially involves the use of an internal feedback control (Robinson 1968; Scudder 1988), allowing error correction to begin only after the erroneous saccade has occurred. Alternatively, fast on-line error correction may involve a comparison of the spatial location of the goal with the anticipated eye displacement using feedforward control (Bernstein et al. 1995; Desmurget and Grafton 2000; Shadmehr and Mussa-Ivaldi 1994; Vaziri et al. 2006; Wolpert et al. 1995) such that any deviations can be corrected without the delays associated with sensory feedback. In principle, such a mechanism allows for error correction to proceed in parallel with the erroneous saccade, even before the error is committed. The results of this study along with our previous work (Murthy et al. 2007) conform to the predictions of this parallel programming model of error correction.

Decisions and motor planning during error correction

The double-step task is particularly well suited to study oculomotor decisions (Aslin and Shea 1987; Becker and Jurgens 1979; Komoda et al. 1973; Lisberger et al. 1975; Ray et al. 2004; Westheimer 1954; Wheeless et al. 1966). Abruptly changing the location of the target and measuring the ability of the oculomotor system to compensate for the target shift can assess the temporal evolution of decision-making. If the target step is too late relative to the decision process, subjects shift gaze first to the original target position and may then look at the final target position. If the target step is early enough, subjects can cancel the first saccade and shift gaze to the new location. On the basis of our previous work (Camalier et al. 2007; Kapoor and Murthy 2008), we expected an explicit STOP process to get initiated, as soon as the new position of the final target was perceived, and race against the ongoing $GO_{Corrective}$ process (GO-STOP-GO/GO-GO-STOP model) to the old final target position. We anticipated the outcome of this race to determine whether the corrective saccade was directed at the new or old position of the final target. Within this context, corrective saccades to locations midway between the two final target positions were unexpected. Although the difference between the predicted and observed results are not entirely clear, one crucial difference between our earlier double-step work and the current study is that in the former, subjects were given explicit instructions about the need to cancel (STOP) a response, whereas in the current study, they were not told about the target step that occurred during the saccade. Hence, we speculate that different architectures may play a role in determining how decisions are averaged (i.e., winner take all/binary versus population based/continuous coding schemes) depending on the type of instructions given—(GO-STOP) (Camalier et al. 2007) versus GO-GO (this study). This interpretation is congruent with earlier work from our lab (Ray et al. 2004), suggesting that instructions play an important role in modulating saccadic reaction times.

In a typical double-step task, the amplitude of the resulting saccade as a function of reprocessing times is known to describe what is called an amplitude (AmpTF)/angle transition

function (AngTF) (Aslin and Shea 1987; Becker and Jurgens 1979; Findlay and Harris 1984; Ludwig et al. 2007). Because in our experiment the shift in the final target is akin to a traditional double-step, we calculated the time associated with the reprocessing of corrective saccade endpoints from the old to the new final target location. Assuming that the perception of the new final target position began only at the end of the first erroneous saccade, the intersaccadic interval between the error and the corrective saccades gave us the delay (*D*) (Becker and Jurgens 1979) that must elapse if the third step is to modify the corrective saccade that is being prepared in response to the second step. By fitting a cumulative Weibull function to the endpoints of the corrective saccades as a function of this delay (Aslin and Shea 1987), we calculated the time needed to complete the transition between the two limiting values of the amplitude known as transition time (Tw). We found a good agreement between our estimates of the transition time (Tw ~112 ms) and those reported by Becker and Jurgen (1979), where Tw ranged from 80 to 200 ms with a mean of 120 ms. However, our estimates where higher than those reported by Aslin and Shea (1987)'s study in which the Tw value of AmpTF was ~50 ms and that of AngTF ranged from 30 to 80 ms. The greater scatter of transition times in our study may be a result of subjects not being strictly instructed to follow the target shift and "fixate anew" on the shifted target as was the case in earlier studies.

The amplitude/angle transition functions have also been used to define what is called the saccadic dead time (SDT) (Ludwig et al. 2007) which is the point in time during saccadic preparation at which no new visual information can change the upcoming movement. Saccadic dead time is typically around 80 ms prior to the movement onset (Findlay and Harris 1984) and is assumed to be caused by the afferent and efferent delays in the transmission of information between the eye and the brain regions responsible for generating the oculomotor commands (Becker 1991; Ludwig et al. 2007; Van Loon et al. 2002). This being the case, are corrective saccades to the old final target location produced as a result of presenting the shifted target within the saccadic dead time? A simple examination of the reaction times of these corrective saccades after the perception of the target-shift rules out this possibility. Because the mean intersaccadic interval for corrective saccades to the old final target location across subjects was estimated to be ~143 ms, there is adequate time for visual information to modify the decision process. Instead, we argue that these trials must represent instances in which the preparation for the corrective saccade has already reached some "point of no-return" in decision-making.

Instances of saccades being directed at a location midway between two simultaneously or sequentially presented targets have been previously reported (Becker and Jurgens 1979; Chou et al. 1999; Ottes et al. 1984) and is particularly observed when the angular distance between two targets is <30° (Ottes et al. 1985) as in our study. However, averaging saccades, in general, have shorter latencies than target-directed saccades (Chou et al. 1999; Coeffe and O'Regan 1987; Findlay 1981a, 1997; Jacobs 1987; Ottes et al. 1984, 1985; Walker et al. 1997), whereas in our study, the latencies of the corrective saccades had an almost linear relation with their final destinations—increasing from the saccades that went to the old position to those that went mid-way and finally to those that went to the new position of the final target. In other words, for all subjects, the latencies of midway corrective saccades (mean across subjects = 308 ms) were longer than the latencies of those directed to the old position (mean across subjects = 298 ms) but shorter than the latencies of saccades directed to the new position of the final target (mean across subjects = 354 ms). This implies that the longer latencies of mid-way saccades were not used for accurate selection of the saccadic target, ruling out "insufficient time" for "perceptual selection" of saccadic destination as the possible cause for their production (Chou et al. 1999). Also, because the motor preparation of the corrective saccade is well underway when the shifted final target is perceived, it is unlikely that mid-way corrective saccades are produced on account of "perceptual grouping of targets" (amounting to sensory averaging) (Compton and Logan 1993; Kowler et al. 1995; Logan 1996; Van Oeffelen and Vos 1983). Rather, based on the finding that the visual information is integrated only over a relatively fixed period of time (Ludwig et al. 2005), following which it is assumed to be transmitted to an oculomotor decision unit, we propose that it is the weighted average of two motor preparations (or 2 decision processes) corresponding to the old and new positions that manifests in the form of midway saccades. Averaging at the motor stage of programming saccades is known to occur in cases wherein some aspect of the motor program (like amplitude, direction etc.) is known in advance (Coeffe and O'Regan 1987; Findlay 1981b; Viviani and Swensson 1982; Zambarbieri et al. 1987) so as to allow some prior motor preparation. Also, in microstimulation studies of the frontal eye fields and superior colliculus (Robinson and Fuchs 1969; Schiller and Sandell 1983), stimulation of a fixed vector saccade at different times during the preparation of an oculomotor command toward a selected target results in averaging saccades that are thought to reflect the weighted sum of the motor preparations toward the two potential targets. However, while we interpret our results as favoring a motor averaging hypothesis, we acknowledge that that they don't necessarily refute the possibility of some degree of averaging also occurring at the sensory stage.

Performance monitoring, inhibitory control, and error correction

That the brain maintains a representation of past performance is known since the first studies performed by Rabbitt (1966), who showed that the reaction times of subjects were much slower in trials following errors. In line with this view, neuronal representations of past performance have been recently recorded in the activity of single neurons in the prefrontal cortex of awake, behaving monkeys (Hasegawa et al. 2000). More recently, Brown and Braver (2005) used modeling and imaging studies to hypothesize and describe the role of anterior cingulate cortex (ACC), another executive control area of the brain, while subjects performed a high versus low conflict task. They obtained an increased metabolic activity in ACC during trials with no response conflict and high rate of error probability even when subjects performed correctly in them, leading them to propose that subjects learn to predict the likelihood of error based on the stimuli-outcome relationship of previous trials. In our study, we have shown the predictive nature of corrective saccade preparation in the redirect double step task. Unlike choice reaction time tasks where correction may be

construed as a delayed correct response, predictive error correction in a motor task raises a fundamental question about the nature of control. More specifically, if correction can start before the occurrence of the error, we propose that the brain must be predicting the likelihood of an error as it is trying to inhibit an unwanted movement. Because in the redirect task error likelihood is related to the degree of inhibitory control, we had the opportunity to examine the interaction between inhibition and error correction.

On the basis of the race model (Camalier et al. 2007; Kapoor and Murthy 2008; Logan and Cowan 1984) that describes saccade production or cancellation in a double-step task as an outcome of a race between a GO and a STOP process (initiated following the presentation of the initial and final target, respectively), it is plausible that at some point of time after the initiation of the STOP process, its likelihood of finishing first becomes so low that it is prudent for the oculomotor system to program a corrective saccade in parallel. If this was true and the onset of correction actually takes into account the likelihood of error, then one ought to expect the time of correction onsets to reflect such performance based stimulus-response relationship. That the estimated onset of correction is not completely determined by the available time of reprocessing alone (Fig. 9, *A* and *B*) is consistent with the stated hypothesis. A more specific test is that the onset of correction should vary systematically with target step delay. We found this to be true for eight of nine subjects where preparation for correction was increasingly delayed as target step delays became smaller and the likelihood of an error presumably reduced. These data are consistent with the idea of the brain estimating the likelihood of error for on-line monitoring of performance (Brown and Braver 2005) and suggests how such predictive estimation may influence the time course of corrective saccade preparation as well.

However, alternative hypotheses cannot be ruled out because it is possible that at shorter delays, the preparation of the corrective saccade may be attenuated by either the motor preparation of the first erroneous saccade and/or the preparation of the correct saccade that was never executed as a general consequence of there being a bottleneck at some stage of saccade processing. If the attenuation due to such a bottleneck varies as a function of target step delay such that interference increases as the temporal overlap between two saccades increases, then a similar relation between target step delay and onset of correction will result. Although at one level the error likelihood and bottleneck hypotheses may be distinct, these hypotheses need not necessarily be incompatible because models of executive control (Botvinick et al. 2001) postulate error/conflict detection in the brain to be a consequence of simultaneous programming of mutually incompatible motor programs. Our study suggests a model of how inhibitory control—generated either as a consequence of a general bottleneck or performance monitoring—and error detection/correction may interact for successful production of voluntary action. We propose that our findings, used in conjunction with electrophysiological recordings, may provide an important approach to study the interactions between error processing and inhibitory centers of the brain, two vital cogs in the executive system responsible for goal-directed behavior.

ACKNOWLEDGMENTS

We thank Drs. Shobini Rao, Jeffrey D. Schall, and Pierre Pouget and the anonymous reviewers for their valuable suggestions and J. Ahlawat for manuscript preparation.

GRANTS

This work was supported by grants from the Department of Science and Technology and the Department of Biotechnology, Government of India.

REFERENCES

Aslin RN, Shea SL. The amplitude and angle of saccades to double-step target displacements. *Vision Res* 27: 1925–1942, 1987.

Baddeley A, Della Sala S. Working memory and executive control. *Philos Trans R Soc Lond B Biol Sci* 351: 1397–1403, 1996.

Becker W. Saccades. In: *Vision and Visual Dysfunction*, edited by Carpenter RHS. Boca Raton, FL: CRC, 1991, p. 95–137.

Becker W, Jurgens R. An analysis of the saccadic system by means of double step stimuli. *Vision Res* 19: 967–983, 1979.

Bernstein P, Scheffers M, Coles M. Where did I go wrong? A psychophysiological analysis of error detection. *J Exp Psychol Hum Percept Perform* 21: 1312–1322, 1995.

Botvinick MM, Braver TS, Carter CS, Barch DM, Cohen JC. Conflict monitoring and cognitive control. *Psychol Rev* 108: 624–652, 2001.

Brown J, Braver T. Learned predictions of error likelihood in the anterior cingulate cortex. *Science* 307: 1118 –1121, 2005.

Bruce CJ, Goldberg ME. Primate frontal eye fields. I. Single neurons discharging before saccades. *J Neurophysiol* 53: 603–635, 1985.

Burr D, Morrone M, Ross J. Selective suppression of the magnocellular visual pathway during saccadic eye movements. *Nature* 371: 511–513, 1994.

Camalier CR, Gotler A, Murthy A, Thompson KG, Logan GD, Palmeri TJ, Schall JD. Dynamics of saccade target selection: Race model analysis of double step and search step saccade production in human and macaque. *Vision Res* 47: 2187–2211, 2007.

Carlow T, Dell'Osso L, Troost B, Daroff R, Birkett J. Saccadic eye movement latencies to multimodal stimuli. intersubject variability and temporal efficiency. *Vision Res* 15: 1257–1262, 1975.

Carpenter RHS, Williams MLL. Neural computation of log likelihood in the control of saccadic eye movements. *Nature* 377 59–62, 1995.

Chou I, Sommer M, Schiller P. Express averaging saccades in monkeys. *Vision Res* 39: 4200–4216, 1999.

Coeffe C, O'Regan J. Reducing the influence of non-target stimuli on saccade accuracy: predictability and latency effects. *Vision Res* 27: 227–240, 1987.

Compton BJ, Logan GD. Evaluating a computational model of perceptual grouping by proximity. *Percept Psychophys* 53: 403–21, 1993.

Desmurget M, Grafton S. Forward modeling allows feedback control for fast reaching movements. *Trends Cogn Sci* 4: 423–431, 2000.

Dorris MC, Pare′ M, Munoz DP. Neuronal activity in the monkey superior colliculus related to the initiation of saccadic eye movements. *J Neurosci* 17: 8566–8579,1997.

Findlay J, Harris L. Small saccades to double-stepped targets moving in two dimensions. In: *Theoretical and Applied Aspects of Eye Movement Research,* edited by Gale AG, Johnson F. Amsterdam: Elsevier, 1984, p. 71–78.

Findlay JM. Saccade target selection during visual search. *Vision Res* 37: 617–631, 1997.

Findlay JM. Local and global influences on saccadic eye movements. In: *Eye Movements, Cognition and Visual Perception,* edited by Fisher DF, Monty RA, Senders JW. Hillsdale, NJ: Erlbaum, 1981a, p. 171–179.

Findlay JM. Spatial and temporal factors in the predictive generation of saccadic eye movements. *Vision Res* 21: 347–354, 1981b.

Gold JL, Shadlen MN. Representation of a neural decision in developing oculomotor commands. *Nature* 404: 390–394, 2000.

Gold JL, Shadlen MN. Neural computations that underlie decisions about sensory stimuli *Trends Cogn Sci* 5: 10–16, 2001.

Goossens H, Van Opstal A. Local feedback signals are not distorted by prior eye movements: evidence from visually evoked double saccades. *J Neurophysiol* 78: 533–538, 1997.

Hallett PE, Lightstone AD. Saccadic eye movements toward stimuli triggered by prior saccades. *Vision Res* 16: 99–106, 1976a.

Hallett PE, Lightstone AD. Saccadic eye movements to flashed targets. *Vision Res* 16: 107–114, 1976b.

Hanes DP, Carpenter RHS. Countermanding saccades in humans. *Vision Res* 39: 2777–2791, 1999.

Hanes D, Schall J. Countermanding saccades in macaque. *Vis Neurosci* 12: 929–937, 1995.

Hanes DP, Schall JD. Neural control of voluntary movement initiation. *Science* 274: 427–430, 1996.

Hanes D, Patterson W, Schall J. Role of frontal eye fields in countermanding saccades: visual, movement, and fixation activity. *J Neurophysiol* 79: 817–834, 1998.

Hasegawa RP, Blitz AM, Geller NL, Goldberg ME. Neurons in monkey prefrontal cortex that track past or predict future performance. *Science* 290: 1786–1789, 2000.

Holt E. Eye movements and central anaesthesia. *Psychol Rev* 4: 3–45, 1903.

Hooge I, Erkelens C. Control of fixation duration in a simple search task. *Percept Psychophys* 58: 969–976, 1996.

Hou R, Fender D. Processing of direction and magnitude by the saccadic eye-movement system. *Vision Res* 19: 1421–1426, 1979.

Jacobs A. On localization and saccade programming. *Vision Res* 27: 1953–1966, 1987.

Joti P, Kulashekhar S, Behari M, Murthy A. Impaired inhibitory oculomotor control in patients with Parkinson's disease. *Exp Brain Res* 177: 447–457, 2007.

Kapoor V, Murthy A. Covert inhibition potentiates online control in a double-step task. *J Vis* 8: 1–16, 2008.

Kim JN, Shadlen MN. Neural correlates of a decision in the dorsolateral prefrontal cortex of the macaque. *Nat Neurosci* 2: 176–185, 1999.

Komoda M, Festinger L, Phillips L, Duckman R, Young R. Some observations concerning saccadic eye movements. *Vision Res*: 13: 1009–1020, 1973.

Kowler E, Anderson E, Dosher B, Blaser E. The role of attention in the programming of saccades. *Vision Res* 35: 1897–1916, 1995.

Krauskopf J, Graf V, Gaarder K. Lack of inhibition during involuntary saccades. *Am J Psychol* 79: 73–81, 1966.

Latour P. Visual threshold during eye movements. *Vision Res* 2: 261–262, 1962.

Levy-Schoen A, Blanc-Garin J. On oculomotor programming and perception. *Brain Res* 71: 443–450, 1974.

Lisberger S, Fuchs A, King W, Evinger L. Effect of mean reaction time on saccadic responses to two-step stimuli with horizontal and vertical components. *Vision Res* 15: 1021–1025, 1975.

Logan GD. The CODE theory of visual attention: an integration of space-based and object-based attention. *Psychol Rev* 103: 603–49, 1996.

Logan G, Cowan W. On the ability to inhibit thought and action: a theory of an act of control. *Psychol Rev* 91: 295–327, 1984.

Ludwig CJ, Gilchrist ID, McSorley E, Baddeley RJ. The temporal impulse response underlying saccadic decisions. *J Neurosci* 25: 9907–9912, 2005.

Ludwig CJ, Mildinhall JW, Gilchrist ID. A population coding account for systematic variation in saccadic dead time. *J Neurophysiol* 97: 795–805, 2007.

Mazzoni P, Bracewell RM, Barash S, Andersen RA. Motor intention activity in the macaque's lateral intraparietal area. I. Dissociation of motor plan from sensory memory. *J Neurophysiol* 76: 1439–1456, 1996.

McPeek R, Han J, Keller E. Competition between baccade goals in the superior colliculus produces saccade curvature. *J Neurophysiol* 89: 2577–2590, 2003.

McPeek RM, Keller EL. Superior colliculus activity related to concurrent processing of saccade goals in a visual search task. *J Neurophysiol* 87: 1805–1815, 2002.

McPeek R, Skavenski A, Nakayama K. Concurrent processing of saccades in visual search. *Vision Res* 40: 2499–2516, 2000.

Minken A, Opstal A, Gisbergen J. Three-dimensional analysis of strongly curved saccades elicited by double-step stimuli. *Exp Brain Res* 93: 521–533, 1993.

Morrison R. Manipulation of stimulus onset delay in reading: evidence for parallel programming of saccades. *J Exp Psychol Hum Percept Perform* 10: 667–682, 1984.

Munoz DP, Wurtz RH. Saccade-related activity in monkey superior colliculus. I. Characteristics of burst and buildup neurons. *J Neurophysiol* 73: 2313–2333, 1995.

Murthy A, Thompson KG, Schall JD. Neural control of saccade target selection during visual search: comparison of search-step and double-step. *Soc Neurosci Abstr* 25: 806, 2000.

Murthy A, Thompson, KG, Schall JD. Dynamic dissociation of visual selection from saccade programming. *J Neurophysiol* 86: 2634–2637, 2001.

Murthy A, Ray S, Shorter SM, Priddy EG, Schall JD, Thompson KG. Frontal eye field contributions to rapid corrective saccades. *J Neurophysiol* 97: 1457–1469, 2007.

Norman D, Shallice T. Attention to action: willed and automatic control of behavior. In: *Consciousness and Self-Regulation*, edited by Davidson RJ, Schwartz GE, Shapiro D. New York: Plenum, 1980, p. 1–18.

Ottes F, Van Gisbergen J, Eggermont J. Metrics of saccade responses to visual double stimuli: two different modes. *Vision Res* 24: 1169–1179, 1984.

Ottes F, Van Gisbergen J, Eggermont J. Latency dependence of colour-based target vs. nontarget discrimination by the saccadic system. *Vision Res* 25: 849–862, 1985.

Paré M, Hanes DP. Controlled movement processing: superior colliculus activity associated with countermanded saccades. *J Neurosci* 23: 6480–6489, 2003.

Polli FE, Barton JJS, Vangel M, Goff DC, Iguchi L, Manoach DS. Schizophrenia patients show intact immediate error-related performance adjustments on an antisaccade task. *Schizophr Res* 82: 191– 201, 2006.

Port N, Wurtz R. Sequential activity of simultaneously recorded neurons in the superior colliculus during curved saccades. *J Neurophysiol* 90: 1887–1903, 2003.

Rabbitt P. Errors and error correction in choice-response tasks. *J Exp Psychol* 71: 264–272, 1966.

Ray S, Schall J, Murthy A. Programming of double-step saccade sequences: modulation by cognitive control. *Vision Res* 44: 2707–2718, 2004.

Rayner K. Eye movements in reading and information processing: 20 years of research. *Psychol Bull* 124: 372–422, 1998.

Reddi BAJ, Asrress KN, Carpenter RHS. Accuracy, information and response time in a saccadic decision task. *J Neurophysiol* 90: 3538–3546, 2003.

Reddi B, Carpenter RHS. The influence of urgency on decision time. *Nat Neurosci* 3: 827–831, 2000.

Riggs L, Merton P, Morton H. Suppression of visual phosphenes during saccadic eye movements. *Vision Res* 14: 997–1011, 1974.

Robinson DA. The oculomotor control system: a review. *Proc IEEE* 56: 1032–1049, 1968.

Robinson DA, Fuchs AF. Eye movements evoked by stimulation of frontal eye fields. *J Neurophysiol* 32: 637–48, 1969.

Schiller PH, Sandell JH. Interactions between visually and electrically elicited saccades before and after superior colliculus and frontal eye field ablations in the rhesus monkey. *Exp Brain Res* 49: 381–392, 1983.

Scudder C. A new local feedback model of the saccadic burst generator. *J Neurophysiol* 59: 1455–1475, 1988.

Shadmehr R, Mussa-Ivaldi F. Adaptive representation of dynamics during learning of a motor task. *J Neurosci* 14: 3208–3224, 1994.

Sato T, Murthy A, Thompson KG, Schall JD. Search efficiency but not response interference affects visual selection in frontal eye field. *Neuron* 30: 583–591, 2001.

Sparks D, Mays L. Spatial localization of saccade targets. I. Compensation for stimulation-induced perturbations in eye position. *J Neurophysiol* 49: 45–63, 1983.

Tan LH, Spinks JA, Eden GF, Perfetti CA, Siok WT. Reading depends on writing, in Chinese. *Proc Natl Acad Sci USA* 102: 8781–8785, 2005.

Theeuwes J, Kramer A, Hahn S, Irwin D, Zelinsky G. Influence of attentional capture on oculomotor control. *J Exp Psychol Hum Percept Perform* 25: 1595–1608, 1999.

Thompson K, Hanes D, Bichot N, Schall JD. Perceptual and motor processing stages identified in the activity of macaque frontal eye field neurons during visual search. *J Neurophysiol* 76: 4040–4055, 1996.

Van Gisbergen J, Van Opstal A, Tax A. Collicular ensemble coding of saccades based on vector summation. *Neuroscience* 21: 541–555, 1987.

Van Loon EM, Hooge IT, Van den Berg AV. The timing of sequences of saccades in visual search. *Proc R Soc Lond B Biol Sci* 269: 1571–1579, 2002.

Van Oeffelen MP, Vos PG. An algorithm for pattern description on the level of relative proximity. *Pattern Recogn* 16: 341–348, 1983.

Vaziri S, Diedrichsen J, Shadmehr R. Why does the brain predict sensory consequences of oculomotor commands? Optimal integration of the predicted and the actual sensory feedback. *J Neurosci* 26: 4188–4197, 2006.

Vergilino D, Beauvillain C. The planning of refixation saccades in reading. *Vision Res* 40: 3527–3538, 2000.

Viviani P. Eye movements in visual search: cognitive, perceptual and motor control aspects. *Rev Oculomot Res* 4: 353–393, 1990.

Viviani P, Swensson R. Saccadic eye movements to peripherally discriminated visual targets. *J Exp Psychol Hum Percept Perform* 8: 113–126, 1982.

Walker R, Deubel H, Schneider W, Findlay J. Effect of remote distractors on saccade programming: evidence for an extended fixation zone. *J Neurophysiol* 78: 1108–1119, 1997.

Westheimer G. Eye movement responses to a horizontally moving visual stimulus. *Arch Opthalmol* 52: 932–941, 1954.

Wheeless L Jr, Boynton R, Cohen G. Eye-movement responses to step and pulse-step stimuli. *J Opt Soc Am* 56: 956–960, 1966.

Wolpert D, Ghahramani Z, Jordan M. An internal model for sensorimotor integration. *Science* 269: 1880, 1995.

Zambarbieri D, Schmid R, Ventre J. Saccadic eye movements to predictable visual and auditory targets. In: *Eye Movements: From Physiology to Cognition*, edited by O'Regan JK, Levy-Schoen A. Amsterdam: Elsevier, 1987, p. 131–140.

Zingale C, Kowler E. Planning sequences of saccades. *Vision Res* 27: 1327–1341, 1987.