Voluntary Control of Multisaccade Gaze Shifts During Movement Preparation and Execution

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Ramakrishnan A, Chokhandre S, Murthy A. Voluntary control of multisaccade gaze shifts during movement preparation and execution. J Neurophysiol 103: 2400–2416, 2010. First published February 17, 2010; doi:10.1152/jn.00843.2009. Although the nature of gaze control regulating single saccades is relatively well documented, how such control is implemented to regulate multisaccade gaze shifts is not known. We used highly eccentric targets to elicit multisaccade gaze shifts and tested the ability of subjects to control the saccade sequence by presenting a second target on random trials. Their response allowed us to test the nature of control at many levels: before, during, and between saccades. Although the saccade sequence could be inhibited before it began, we observed clear signs of truncation of the first saccade, which confirmed that it could be inhibited in midflight as well. Using a race model that explains the control of single saccades, we estimated that it took about 100 ms to inhibit a planned saccade but took about 150 ms to inhibit a saccade during its execution. Although the time taken to inhibit was different, the high subject-wise correlation suggests a unitary inhibitory control acting at different levels in the oculomotor system. We also frequently observed responses that consisted of hypometric initial saccades, followed by secondary saccades to the initial target. Given the estimates of the inhibitory process provided by the model that also took into account the variances of the processes as well, the secondary saccades (average latency ~215 ms) should have been inhibited. Failure to inhibit the secondary saccade suggests that the intersaccadic interval in a multisaccade response is a ballistic stage. Collectively, these data indicate that the oculomotor system can control a response until a very late stage in its execution. However, if the response consists of multiple movements then the preparation of the second movement becomes refractory to new visual input, either because it is part of a preprogrammed sequence or as a consequence of being a corrective response to a motor error.

INTRODUCTION

All voluntary acts entail a decision process, whether implicit or explicit. Since a fundamental goal of the oculomotor system is to choose objects of interest in the visual scene to which gaze is directed, saccadic eye movements provide a logical paradigm to study the decision process that precedes movement execution. A task particularly well suited to study oculomotor decisions is the double-step task (Aslin and Shea 1987; Becker and Jurgens 1979; Komoda et al. 1973; Lisberger et al. 1975; Ray et al. 2004; Westheimer 1954; Wheless et al. 1966). Here on most trials subjects are shown a target to which a saccade is made. Abruptly changing the location of the target on some random infrequent trials and measuring the ability of the oculomotor system to compensate for the target shift assesses 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 to the final position. If the target step is early enough, subjects can cancel the first saccade and shift gaze to the new location.

Many studies have found that performance during double-step tasks is stochastic and that the probability of compensating for the target step by directing gaze to the final target location decreases with the delay of the step, presumably because of the advancing commitment to shift gaze to the initial target location. More recently it has been shown that performance during double-step tasks can be accounted for by a race between three stochastically independent processes: 1) a GO process producing the saccade to the initial target location, 2) a STOP process interrupting that GO process, and 3) an ongoing GO process producing the saccade to the final target location (Becker and Jurgens 1979; Camalier et al. 2007; Kapoor and Murthy 2008; Lisberger et al. 1975; Murthy et al. 2009). Using this race model, the duration required to cancel the planned saccade can be estimated and has been called the target step/switch reaction time (TSRT), which is analogous to the stop signal reaction time used in the countermanding task (Hanes et al. 1998; Logan and Cowan 1984) to estimate the time it takes to inhibit a planned saccade (Camalier et al. 2007; Joti et al. 2007; Murthy et al. 2001) Therefore in addition to studying how decisions are made, the double-step task can be used to study how control is implemented by the oculomotor system, using a similar race model framework that has been successful in modeling performance in countermanding tasks.

The goal of this study was to extend behavioral predictions arising from the race model framework that have been proposed to underlie the control of single saccades to instances involving multisaccade gaze shifts, which often occur when subjects make saccades to highly eccentric targets (eccentricity >15°; Becker 1972; Becker and Fuchs 1969; Henson 1978; Prablanc and Jeannerod 1975; Prablanc et al. 1978; Weber and Daroff 1972). In such instances the target is foveated by an initial hypometric saccade that undershoots the target and is then followed with a secondary saccade. Therefore on double-step trials if subjects fail to inhibit the initial hypometric response to the initial target, they have the opportunity to inhibit the secondary response and direct gaze to the newly specified target. Since control can manifest at multiple levels—either before the gaze shift begins or during the intersaccadic interval separating intervening saccades—we asked whether a unitary mechanism would suffice to explain oculomotor control.

In addition to understanding how our brains control multisaccade gaze shifts, we also used this paradigm to assess whether the programming of multisaccade gaze shift might also involve a ballistic stage of processing that is refractory to voluntary control. Although previous behavioral (e.g., Logan 1994) and
neurophysiological studies (Hanes and Schall 1995; Hanes et al. 1998; Paré and Hanes 2003) support the notion of a race to a single threshold, the location of the threshold in relation to movement execution is unclear. One possibility is that the threshold occurs before saccade execution, providing a distinct point of no return in saccadic decision making (e.g., Osman et al. 1986). Modeling countermanding behavior supports this model and has estimated the point of no return at 28–60 ms before saccade execution (Kornyro et al. 2003). Alternatively, it is possible that saccade programming has no ballistic stage and can be controlled even during the execution of saccades. Evidence in support of this derives from countermanding studies in which hypometric saccades are generated, presumably from an interaction of the STOP process during the execution of the noncanceled saccade (Camalier et al. 2007; Colonius et al. 2001; Ozyurt et al. 2003; Walton and Gandhi 2006). In addition, many double-step studies have provided evidence of gaze errors being corrected very rapidly, sometimes resulting in trajectories that are curved toward the target following initial errors (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). In this study we examine which of these two alternatives explains performance during multisaccade gaze shifts.

METHODS

Subjects

Fifteen naive subjects (ages between 18 and 32 yr), with normal or corrected vision, performed the redirect task (see Task for details). Their eye movements were recorded with their heads stabilized by means of a chin and forehead rest. All subjects gave their informed consent in accordance with the institutional human ethics committee of the National Brain Research Centre. Subjects were monetarily rewarded after every session to keep them motivated.

Task

The redirect task (Murthy et al. 2001; Ray et al. 2004) is a modified version of the classic double-step task (Aslin and Shea 1987; Becker and Jurgens 1979; Komoda et al. 1973; Lisberger et al. 1975; Ray et al. 2004; Westheimer 1954; Wheelless et al. 1966). The task consists of two kinds of trials: no-step trials in which a single target is presented (i.e., target is “not stepped” to another location) and step trials in which two targets are presented in succession.

On 50% of the trials, referred to as no-step trials, following fixation for a random duration that ranged from 300 to 800 ms, a single green target (1° × 1°), with the International Commission on Illumination (CIE) chromaticity coordinates [273 615 7.1], appeared on the screen on a background, with CIE chromaticity coordinates of [220 300 0.06] (see Fig. 1A, top left). The location of the target was randomized such that it could appear in any one of eight locations centered on an imaginary circle with a radius of 30°. Subjects were instructed to quickly make a saccade to the target. They were given verbal instructions to increase the speed if their saccade latencies exceeded 400 ms.

In step trials, after the presentation of the first target, a second (1° × 1°) red target, with CIE chromaticity coordinates of [632 330 7.8], appeared randomly at another location on the screen (see Fig. 1B, top right) at an angular separation equal to or >90° to avoid averaging of the saccades to the two target locations (Ottes et al. 1984). The time of appearance of the final target relative to the initial target, called target step delay (TSD), was varied randomly from about 20 to 230 ms. The appearance of the second target served as a “redirect” signal instructing subjects to inhibit the partially planned saccade to the initial target and direct gaze to the final target. Random interleaving of the two trial types prevented the subjects from delaying the initial saccade, in anticipation of the second target. Step trials in our task differed from the earlier double-step studies in two main respects: the initial and final targets were of different colors, to ease distinction between the targets, especially at shorter TSDs; further, the initial target did not disappear with the appearance of the final target.

Trials were scored as successful, and conveyed to subjects by auditory feedback, if subjects made the saccade to the green target in a no-step trial and to the red target in a step trial, fixating the respective targets within an electronic window of ±5° centered on the target.

Data collection and analysis

Experiments were under computer control using TEMPO/VIDEOSYNC software (Reflective Computing, St. Louis, MO) that displayed visual stimuli and stored sampled eye position. Eye position was sampled at 240 Hz with an infrared pupil tracker (ISCAN, Boston, MA) that interfaced with the TEMPO software in real time. All stimuli were presented on a Sony Trinitron 500 GDM monitor (21 in.; 70 Hz refresh rate) placed 34 cm in front of the subject. Stimuli were calibrated with a Minolta CA-96 colorimeter.

To calibrate our eye tracker (see also Kornyro et al. 2007) subjects made saccades with increased fixation times (mean = 500 ms ± 30°) and postsaccadic times (mean = 500 ms ± 30%); for these trials we measured the SD of the eye tracker positions during a 100 ms fixation period. The estimate of the inherent noise of the tracker was 0.01°. The spatial accuracy of the tracker was estimated by measuring the mean saccade endpoint location during 100 ms of postsaccadic fixation time. The median of these endpoints on three different trials to the same target was about 0.9°, which was an estimate of saccadic accuracy.

At the beginning of each session, subjects were given written and verbal instructions followed by some practice trials (~50). On average, subjects performed about 500 trials per session, with breaks every 200 trials. A typical session lasted about 1 h, with each subject performing four to six such sessions.

All analysis and statistical tests were performed off-line using MATLAB (The MathWorks, Natick, MA). Blinks were removed from the eye position data and a velocity threshold of 30°/s was used to demarcate the beginning and end of saccades. The accuracy of saccade detection was subsequently verified manually.

Classification of hypometric trials

Any trial in which the amplitude of the primary saccade fell short of the target by ≥6° was considered a hypometric response. We have used a fairly large amplitude cutoff to include only those trials that involved large corrections. Previous work has shown that when the error is large, the secondary saccade is executed much faster than when the error is small (<20% of the target eccentricity, which in our case is 6°; Becker 1972; Becker and Fuchs 1969; Becker and Jurgens 1979; Henson 1978; Prablanc and Jeanmerod 1975; Prablanc et al. 1978; Weber and Daroff 1972). These differences in reaction times have been suggested to arise as a consequence of the oculomotor system using two different algorithms for error correction. In the case of large errors, fast correction can occur without waiting for the retinal reafferent signal. On the other hand, when the error is small the retinal information is awaited because the predictive correction is likely to be too noisy to prepare precise small corrections. Since our study focused on comparing hypometric errors with hypometric corrections (see Fig. 1, B3 and B4), which entailed fairly large corrections, we sought to ensure the same for the former so that we could contrast the effect of control, independent of potential differences in the algorithms that might be used to prepare the corrections.
Race model and estimation of target step reaction time

Performance in the double-step redirect task has been recently modeled as a race between three stochastically independent processes: 1) a GO process (GO1), producing the saccade to the initial target location; 2) a process inhibiting or interrupting the preparation of this saccade to the initial target (STOP); and 3) another GO process (GO2), producing the saccade to the final target location (Camalier et al. 2007; see Fig. 2). On step trials, the STOP process is assumed to be initiated following the appearance of the final target that attempts to cancel the partially prepared response to the initial target (GO1). The two processes, assumed to be independent of each other, race toward the threshold. The winner of the race decides the behavioral outcome. When the STOP process wins the race, the saccade to the initial target is inhibited. This allows the second GO process (GO2) to elicit a saccade to the final target, resulting in a successful response (see Fig. 2A). When the STOP process loses the race, the first saccade is made to the initial target and is referred to as an erroneous response. Subsequently, a corrective saccade redirects gaze to the final target (see Fig. 2B).

The race model provides a measure of the duration of the inhibitory process, which we refer to as the target step reaction time (TSRT). TSRT is analogous to the stop signal reaction time.
before the GO1 process, the initial response is successfully inhibited and a second GO2 process directs gaze to the final target (referred to as a successful response). The panels above A and B depict the behavior at the relevant time points. C: the finish times of the GO1 process give rise to a right-skewed no-step reaction time distribution. In step trials, assuming that the GO process is independent of the STOP process, the no-step reaction time distribution would be partitioned into a faster fraction of saccades that escaped inhibition (green) and a slower fraction that did not (red). The time from the onset of final target until the time of partition is the TSRT for this TSD (see calculation of TSRT by the integration method in METHODS for details).

(A) Successful response
(B) Erroneous response
(C) No-step reaction time distribution

FIG. 2. The race model framework and calculation of target step reaction time (TSRT). In A and B, following the appearance of the first target (green arrow on the x-axis) and a visual delay of 60 ms, a GO1 process (green solid line) is initiated. Execution of the saccade to the target occurs once the GO1 process crosses the threshold (thick horizontal gray line). Following a TSD, when a second target appears (red arrow on the x-axis), the STOP process (red solid line) is initiated following the visual delay, which attempts to cancel the GO1 process. A: sometimes when the STOP process reaches the threshold before the GO1 process, the initial response is successfully inhibited and a second GO2 process directs gaze to the final target (referred to as a successful response). B: on the other hand, when the GO1 process wins the race, the saccade to the initial target is executed (referred to as an erroneous response). Execution of the saccade to the final target is only possible following the appearance of the second target (red arrow on the x-axis) and a visual delay of 60 ms, a GO2 process (green solid line) is initiated following the visual delay, which attempts to cancel the GO1 process. In step trials, assuming that the GO process is independent of the STOP process, the no-step reaction time distribution would be partitioned into a faster fraction of saccades that escaped inhibition (green) and a slower fraction that did not (red). The time from the onset of final target until the time of partition is the TSRT for this TSD (see calculation of TSRT by the integration method in METHODS for details).
MAXIMUM LIKELIHOOD METHOD. We also performed parameter estimation using a maximum likelihood approach as described by previous studies (Corneil and Elsley 2005; Kornryo et al. 2003; Walton and Gandhi 2006). To estimate the parameters that defined rGO, we initially computed the inverse of the no-step reaction time distribution. The mean and SD of the rates of the GO process were directly obtained running the "fmincon" optimization routine in MATLAB. We repeated this procedure 50–75 times, with different sets of initial parameter values, to ensure convergence to the global minima.

Testing the validity of the race model

The race model assumes that the GO and STOP processes are stochastically independent (Logan 1984)—i.e., the underlying GO process is not altered by the presence of the STOP process in the step trials. If this assumption holds, then for a given TSD, the finish times of the STOP process partitions the no-step reaction time distribution such that only those saccades that have shorter latencies than the sum of the TSD and the average latency of the STOP process (TSRT), escape inhibition (see Fig. 2C). Testing the validity of the race model has been traditionally performed using the mean latency of the STOP process. However, given that the processes involved are stochastic we therefore included the variances for a stringent and conservative method that was primarily aimed at establishing violations of the race model. For this it was important to show that the timing of erroneous saccades could not be accounted for by the subset of even the slowest STOP processes. In practice this was tested by STOP2σ, (see Fig. 3), which is the latency of the STOP process that is slower than the mean STOP rate by 2σ, is denoted by STOP2σ, and corresponds to the end of the STOP reaction time distribution. Almost all (>97.5%) step trials should be successfully inhibited to the left of STOP2σ. This makes STOP2σ, given by the sum of the TSD, the target step reaction time (TSRT) and the time equivalent to 2SDs of rSTOP, the cutoff for the latency of the slowest erroneous response.

taken as unity. Starting 60 ms after the appearance of the target, corresponding to the latency of visual cell response in the visuomotor system (e.g., Goldberg and Wurtz 1972; Thompson et al. 1997), the GO process activation increased at the rate of GO every millisecond. The saccade was assumed to be executed when this process reached the threshold (i.e., at 1/rGO ms after the visual delay). To estimate the parameters (mean and SD) of this Gaussian distribution, we simulated about 2,000 no-step trials. We used the Kolmogorov–Smirnov (KS) statistic to compare the simulated cumulative no-step reaction time distribution with the observed one. The KS statistic served as an index of the error that was minimized in the parameter space using a nonlinear minimization procedure running in MATLAB. Convergence was decided based on parameter values that minimized the KS statistic between the simulated and observed data. We repeated this procedure 50–75 times, with different sets of initial parameter values, which was done to ensure convergence to the global minima, before choosing the best set of parameters.

On step trials, following the appearance of the final target and a visual latency of 60 ms, a STOP process with rate of accumulation rSTOP Hz (chosen arbitrarily from a Gaussian distribution with mean μSTOP and SD σSTOP) began. Since this defines the slope of the STOP process (see Fig. 2A and B) the latency of the STOP process would be 1/rSTOP ms plus the visual delay. If the STOP process reached the threshold value before the GO process, then the saccade was successfully inhibited. On the other hand, if the GO process reached the threshold before the STOP process, then the saccade was taken to have escaped inhibition and the time of reaching the threshold was taken as the simulated latency of the erroneous saccade. By determining the probability of erroneous saccades as a function of TSD, we plotted the simulated compensation functions along with the subjects’ observed compensation functions. A least squares method was used to determine the error between the predicted and the observed functions, which was minimized in the parameter space to iteratively obtain the parameters for the rate distribution of the STOP process (by using the “fmincon” optimization routine in MATLAB). We repeated this procedure 50–75 times, with different sets of initial parameter values, to ensure convergence to the global minima.

Testing the validity of the race model

The race model assumes that the GO and STOP processes are stochastically independent (Logan 1994)—i.e., the underlying GO process is not altered by the presence of the STOP process in the step trials. If this assumption holds, then for a given TSD, the finish times of the STOP process partitions the no-step reaction time distribution such that only those saccades that have shorter latencies than the sum of the TSD and the average latency of the STOP process (TSRT), escape inhibition (see Fig. 2C). Testing the validity of the race model has been traditionally performed using the mean latency of the STOP process. However, given that the processes involved are stochastic we therefore included the variances for a stringent and conservative method that was primarily aimed at establishing violations of the race model. For this it was important to show that the timing of erroneous saccades could not be accounted for by the subset of even the slowest STOP processes. In practice this was tested by STOP2σ, (see Fig. 3), which is the latency of the STOP process that is slower than the mean STOP rate by 2σ. Since 2σ accounts for 97.5% (leaving the upper tail of 2.5%) of the STOP distribution, we intuitively expected a 2–3% violation by using this criterion. However, it is important to note that because of the statistics of sampling, this percentage violation represents the average expected violation. Since the purpose of the analysis was aimed at testing the limits of the race model, it is important to establish the upper bound of the expected violation. This number is given by Chebyshev’s theorem, which states that at most 1/k2 proportion of trials can occur in the range μ ± kσ. Therefore when k = 2 we get 0.25. However, since this includes both tails of the distribution, this has to be halved if just the upper tail is considered. Thus values of violation >12.5% would invalidate the race model. In practice a trial was declared a violation if its latency exceeded the sum of the TSD, the target step reaction time, and the time equivalent to 2SDs of the rate of the STOP process by at least 15 ms, the latter being the resolution of the TSD bin size.
Idealizing compensation functions

In practice, compensation functions seldom spanned the entire psychometric range (from 0 to 1). This means that at shorter TSDs subjects sometimes failed to compensate when they should have and, at longer TSDs, they managed to compensate unexpectedly. Such behavior can systematically affect the compensation function and lead to biased parameter estimates. We therefore corrected for this bias by making certain assumptions about the subject’s strategy. If $\alpha$ (the lower asymptote to the Weibull fit of the compensation function) is the proportion of trials a subject failed to inhibit at the shortest TSD, we considered $\alpha$ to be the proportion of trials when subjects ignored the final target. In simulations, these trials can be incorporated by withholding the STOP process. Similarly, if $\beta$ (the upper asymptote to the Weibull fit of the compensation function) is the maximum value of the compensation function, we considered $(1 - \beta)$ to be the proportion of trials when the subjects ignored the initial target. In simulations, these trials can be incorporated by not initiating the GO process. An equivalent approach that yields the same outcome is to treat the subjects’ performance in the remaining fraction of trials ($\beta - \alpha$) to be ideal. We rescaled subjects’ compensation functions between 0 and 1 to reflect the ideal compensation function, which was used to obtain bias free parameter estimates. However, we also carried out analyses without rescaling. Although this changed the parameter values of the STOP and the GO processes and the estimated fraction of violations, the percentage of secondary saccades violating the cutoff was nevertheless very high, compared with erroneous first saccades. Thus the basic results did not change due to rescaling.

RESULTS

We used the redirect task to understand how inhibitory control is exerted to achieve goal directed movements. In this task, subjects were instructed to make a saccadic eye movement to the initial target as soon as it appeared (no-step trials). However, if a second target subsequently appeared (step trials), subjects were asked to cancel the partially planned saccade to the first target and direct their gaze to the second target. In step trials, subjects sometimes compensated for the target step—i.e., inhibited the initial saccade and directed gaze toward the final target (see Fig. 1B1: successful response). However, at other times, they failed to inhibit the saccade toward the initial target—i.e., their response was not successfully inhibited (see Fig. 1B2: erroneous response). On reaching the initial target, however, they made a subsequent corrective saccade toward the final target.

The targets appeared at 30° eccentricities. Subjects attempting to foveate often made (on average across subjects 11.5 ± 2SE%) a set of two saccades instead of a single saccade: the primary saccade followed by a secondary saccade (see Fig. 1A2, hypometric response in a no-step trial). We embedded this behavior in the context of the redirect task to assess inhibitory control during multisaccade gaze shifts, where control can manifest at multiple levels: either before the saccades began, during the saccade, or even during the intersaccadic interval.

Performance of subjects

In the redirect task, the TSD was varied across trials. Although subjects typically find it easier to inhibit at shorter TSDs, they find it harder to do the same at longer TSDs, presumably because they are more committed to the initial response. We plotted the probability of making a saccade to the initial target in step trials as a function of increasing TSDs. Only trials in which responses were nonhypometric were used for this analysis. Figure 4 shows the performance curves, referred to as compensation functions (Becker and Jurgens 1979; Camalier et al. 2007), of nine typical subjects in the task. Superimposed are the best-fit cumulative Weibull function fitted to quantify performance.
where \( t \) is the TSD, \( \alpha \) is the time at which the function reaches 64% of its full growth, \( \beta \) is the slope, \( \gamma \) is the maximum value of the function, and \( \delta \) is the minimum value of the function. Given the nature of the task, \( \gamma - \delta \) can be used as an index to assess how the subjects performed. In all our sessions \((\gamma - \delta)\) was \(>0.5\).

**Race model analysis: quantifying the ability to inhibit**

We obtained the parameters of the rates of the GO process by simulating the cumulative no-step reaction time distribution using the Monte Carlo method as well as the maximum likelihood based method (see METHODS). The simulated and the observed cumulative no-step reaction times for the nine typical subjects are shown in Fig. 5. The goodness-of-fit was ascertained by computing the correlation coefficient \((r^2)\), which was on average \(0.98\) (max = \(0.99\); min = \(0.95\)). The \(r^2\) value was used to decide the better of the estimates from the two methods, which was then used for all further analyses. Columns 1 and 2 of Table 1 and Table 2 list the parameters (mean and SD) obtained using the Monte Carlo approach and the maximum likelihood based approach, respectively.

Step trials were simulated on the basis of the race model described in Fig. 2. The parameters of the GO process that were obtained as mentioned earlier were used to estimate the parameters of the STOP process using the Monte Carlo method (see Table 1, column under STOP), which were then used to simulate the compensation functions (see Fig. 6). The goodness-of-fit was ascertained by computing the correlation coefficient \((r^2)\), which was on average \(0.89 \pm 0.02\) (max = \(0.95\); min = \(0.65\)). We also performed the same procedure using the maximum likelihood based procedure, which also yielded similar results (see Table 2, column under STOP). The goodness-of-fit was on average \(0.88 \pm 0.02\) (max = \(0.95\); min = \(0.64\)).

We used the parameters of the rate of STOP process to find an estimate of the latency to STOP or the target step reaction time (TSRT; see METHODS) by computing the time taken by the simulated cumulative no-step reaction time distribution (black) and, when compared with the observed distribution (light gray), shows good correlation. Data (binned every 25 ms) are shown for the 9 representative subjects.

**TABLE 1. Parameter estimation based on the Monte Carlo method**

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<th>Subject</th>
<th>Nonhypometric</th>
<th>Hypometric</th>
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<td>GO((\sigma))</td>
<td>STOP((\mu))</td>
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<tr>
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Values are means (\(\mu\)) and SD (\(\sigma\)) for the nonhypometric GO process, the hypometric GO process, and the STOP process for each subject.

![FIG. 5. Comparison of the observed and simulated cumulative no-step reaction time distributions. Parameter estimates (mean and SD) for the GO process were obtained from both the Monte Carlo method and the maximum likelihood based method. The better of the estimates from the 2 methods (see RESULTS for details) was used to simulate the cumulative no-step reaction time distribution (black) and, when compared with the observed distribution (light gray), shows good correlation. Data (binned every 25 ms) are shown for the 9 representative subjects.](http://jn.physiology.org/doi/10.1152/jn.00306.2010)
reach the threshold (i.e., 1/μ \text{stop} ms after the visual delay). The average TSRT across subjects calculated in this fashion (101.2 ± 5.9 ms) was comparable to the TSRT estimate (107 ± 5 ms) that was derived by conventional methods using the race model (described earlier in Race model and estimation of target step reaction time in METHODS). In addition, the two TSRT estimates were strongly correlated [Pearson’s r = 0.86, P = 0.000036] with the mean slope of the regression line close to unity (slope = 0.94; intercept = 0.083), indicating that our simulated parameters provided a good description of the behavioral data (see Fig. 7).

We next tested the validity of the race model for all the subjects by checking the percentage of erroneous saccades whose reaction times violated the STOP2 \text{cutoff}. The percentage of violation for every subject is tabulated in column 1 of Table 3 and Table 4 for the percentage of violations calculated by the Monte Carlo method and the maximum likelihood based method, respectively. The percentage of trials that violated the cutoff was on average 10.29% (min 3.1%; max 17.4%). Eleven of 15 subjects are within the prescribed cutoff demarcated by the STOP2 \text{criterion}. However, these percentages are an average across TSDs. To get a better picture we separated these trials that violated the cutoff into three different groups based on the TSDs. On closer inspection, the percentage of

<table>
<thead>
<tr>
<th>Subject</th>
<th>Nonhypometric GO(μ)</th>
<th>Hypometric GO(μ)</th>
<th>STOP(μ)</th>
<th>Nonhypometric GO(σ)</th>
<th>Hypometric GO(σ)</th>
<th>STOP(σ)</th>
</tr>
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<tbody>
<tr>
<td>AS</td>
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<td>2.7</td>
<td>5.60</td>
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<td>3.1</td>
<td>2.2</td>
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</tr>
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<td>80.00</td>
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<td>1.60</td>
</tr>
<tr>
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<td>64.00</td>
<td>3.1</td>
<td>1.6</td>
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<td>76.00</td>
<td>3.1</td>
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<td>8.60</td>
</tr>
<tr>
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<td>6.7</td>
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<td>2.2</td>
<td>2.4</td>
<td>1.00</td>
</tr>
<tr>
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<td>3.2</td>
<td>3.0</td>
<td>1.00</td>
</tr>
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<td>19.20</td>
<td>4.0</td>
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<td>2.20</td>
</tr>
<tr>
<td>DC</td>
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</tr>
<tr>
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<td>80.00</td>
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<tr>
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<td>7.2</td>
<td>18.10</td>
<td>2.1</td>
<td>1.4</td>
<td>1.70</td>
</tr>
<tr>
<td>VJ</td>
<td>6.7</td>
<td>7.5</td>
<td>19.45</td>
<td>3.7</td>
<td>1.8</td>
<td>1.60</td>
</tr>
<tr>
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<td>29.08</td>
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<td>TS</td>
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<td>5.1</td>
<td>17.40</td>
<td>2.5</td>
<td>1.2</td>
<td>1.30</td>
</tr>
</tbody>
</table>

Values are means (μ) and SD (σ) for the nonhypometric GO process, the hypometric GO process, and the STOP process for each subject.

FIG. 6. Comparison between simulated and observed compensation functions. Observed compensation functions (solid gray circles) are best fit with Weibull functions (adapted from Fig. 4). Simulated compensation functions (solid black triangles) generated for the 9 representative subjects using the parameters obtained based on Monte Carlo simulation (see Monte Carlo method in METHODS) show good correlation with the observed data.

FIG. 7. Comparison of TSRT obtained by conventional methods vs. TSRT obtained by simulating the race model. Each circle represents datum from one subject. The broken line represents the fit derived from a linear regression of the data. The simulated and the observed TSRTs are strongly correlated, indicating that simulation provides a good description of the behavioral data.
We were interested to assess the ability to inhibit the multisaccade gaze shift at the level of the secondary saccade. We reasoned that the secondary saccade, which was erroneous in the given context, should be inhibited by the STOP process and be superseded by a corrective saccade to the final target, resulting in a hypometric correction. Although we do not know when the secondary saccade preparation starts, the extent of its inhibition is still expected to conform to the parameters of the STOP process. Assuming the same parameters of the STOP process, as in the earlier case, we determined the percentage of secondary saccades that occurred beyond the latency predicted by STOP$_2$ and found it to consist of a large proportion (average = 78.29%; min = 31.74%; max = 100%) for every subject examined (see column 3 of Tables 3 and 4 for results using the Monte Carlo method and the maximum likelihood based method, respectively). At all TSDs every subject violated the cutoff at early (20, 50, and 80 ms; min = 87%; max = 100%; mean = 99%; 15/15 subjects violated the cutoff), intermediate (110 and 140 ms; min = 50%; max = 100%; mean = 84%; 15/15 subjects violated the cutoff), and late (>170 ms; min = 0%; max = 100%; mean = 53%; 14/15 subjects violated the cutoff) TSDs. In contrast to the first saccades, a large percentage of secondary saccades (78%) violated the cutoff that could not be accounted for by the upper bound established by the STOP$_2$ criterion. Thus the race model failed to account for the timing of the secondary saccades of hypometric errors.

Next, to understand the magnitude of this violation we quantified the latencies of the saccades that violated the cutoff. The extent of violation of erroneous first saccades that violated the cutoff was 51 ms (min = 20 ms; max = 87 ms). Likewise, the extent of violation of erroneous primary saccades that violated the cutoff was 38 ms (min = 12 ms; max = 52 ms). In contrast, the secondary saccade reaction times occurred well beyond the cutoff time. The extent of violation of the secondary saccades that violated the cutoff was 114 ms (min = 73 ms; max = 157 ms). To compare and contrast the extent of violations were higher for the shorter TSDs (20, 50, and 80 ms; min = 19%; max = 70%; mean = 42%; 15/15 subjects violated the cutoff). However, for the majority of erroneous trials that occurred at intermediate and longer TSDs there was no significant violation (at 110 and 140 ms; min = 0%; max = 12%; mean = 5%; no subjects violated the cutoff and >170 ms; min = 0%; max = 2%; mean = 0.9%; no subjects violated the cutoff). These results validate the race model for the erroneous first saccade of a hypometric response.

### Inhibitory control during a hypometric response

**PRIMARY SACCADE.** Analogous to the earlier analysis, we tested the validity of the race model for primary saccades of the hypometric response. The percentage of violation for every subject is tabulated in column 2 of Tables 3 and 4. The percentage of trials that violated the cutoff was on average 13.0% (min = 3.4%; max = 23.8%). Eight of 15 subjects are within the prescribed cutoff demarcated by the STOP$_2\alpha$ criterion. Again, on closer inspection the percentage of violations was higher for the shorter TSDs (20, 50, and 80 ms; min = 12%; max = 67%; mean = 34%; 15/15 subjects violated the cutoff). However, for the majority of erroneous trials that occurred at intermediate and longer TSDs there was little or no significant violation (at 110 and 140 ms; min = 0%; max = 14%; mean = 5%; 1/15 subjects violated the cutoff and for >170 ms; min = 0%; max = 9%; mean = 1%; no subjects violated the cutoff). These results validate the general applicability of the race model for the primary saccade of the hypometric response.

**SECONDARY SACCADE.** On step trials, when subjects made an initial hypometric saccade toward the first target, two interesting behavioral patterns resulted. Although on some trials they managed to correct midway following the initial erroneous saccade (referred to as a hypometric correction; see Fig. 1B3), on others, they failed to do so and instead made a secondary saccade to the initial target, foveating the final target only in the subsequent saccade (referred to as hypometric errors; see Fig. 1B4).

### Table 3. Percentage of violation from the Monte Carlo method

<table>
<thead>
<tr>
<th>Subject</th>
<th>Nonhypometric</th>
<th>Hypometric</th>
<th>Secondary Saccade</th>
</tr>
</thead>
<tbody>
<tr>
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<td>70.0000</td>
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<td>BS</td>
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<td>100.0000</td>
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<td>94.4444</td>
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<tr>
<td>JA</td>
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<td>23.8288</td>
<td>97.3684</td>
</tr>
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</tr>
<tr>
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<td>17.6415</td>
<td>94.4444</td>
</tr>
<tr>
<td>VI</td>
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</table>

Percentage of violation for first saccades of nonhypometric trials, hypometric trials, and the secondary saccades of hypometric errors.

### Table 4. Percentage of violation from the maximum likelihood–based method

<table>
<thead>
<tr>
<th>Subject</th>
<th>Nonhypometric</th>
<th>Hypometric</th>
<th>Secondary Saccade</th>
</tr>
</thead>
<tbody>
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<tr>
<td>NC</td>
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<td>64.7059</td>
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<td>92.0000</td>
</tr>
<tr>
<td>TS</td>
<td>12.7820</td>
<td>7.1429</td>
<td>100.0000</td>
</tr>
</tbody>
</table>

Percentage of violation for first saccades of nonhypometric trials, hypometric trials, and the secondary saccades of hypometric errors.
violation we have plotted a typical subject’s data (latencies of erroneous first saccades along with those of secondary saccades) for a particular TSD (see Fig. 8A). In Fig. 8B we pooled the data across TSDs and aligned it with respect to STOP2\(2\) (depicted by “0” on the x-axis). In contrast to the erroneous first saccades a significantly greater proportion of secondary saccades occur at latencies greater than STOP2\(2\) consistently across subjects (Wilcoxon rank sum between the two distributions, \(P < 0.001\) for all subjects). The extent of violation signifies the inability of the oculomotor system to inhibit the secondary saccade and is indicative of the failure of voluntary control. In other words, these data point to the existence of a ballistic stage of saccade programming during the intersaccadic interval in a multisaccade gaze shift.

If the secondary saccade is not inhibited then what could explain the production of hypometric corrections? We hypothesized that hypometric corrections could be a result of the STOP process interrupting the first saccade during execution, following which a GO\(2\) process redirects gaze to the final target. This hypothesis has three predictions, which we examined in turn. First, a midflight truncation by STOP process implies that the frequency of hypometria must be greater in step trials that engage a STOP process, compared with no-step trials, which do not. To test this prediction, we plotted the percentage of hypometria during step trials and no-step trials for each subject (see Fig. 9A) and found this value to be significantly greater (Wilcoxon rank sum, \(P = 0.046\) for the pooled data) in step trials (mean = 17.36\%), compared with no-step trials (mean = 11.47\%) for all subjects (15/15), favoring the role of STOP process in midflight truncation.

Second, we examined whether the primary saccades in hypometric corrections are qualitatively different from their no-step counterparts, as a result of being generated as a consequence of STOP mediation. To test this, we plotted their amplitudes (see Fig. 9B) and found the median amplitude of primary saccades in hypometric correction trials to be less than the same in no-step trials for 14/15 subjects; the difference was significant for 11/15 subjects (Mann–Whitney \(U\) test, \(P < 0.05\)), conforming to the hypothesis that the STOP process interrupts the first saccade during execution, leading to a hypometric correction.

Third, if the primary saccade (the first saccade in the hypometric correction) began as a normal saccade but was truncated midflight by STOP process, it would modify the typical bell-
shaped velocity profile to that of a skewed one. We used the ratio of acceleration duration to deceleration duration to quantify the degree of this skewness in the saccade velocity profile (Van Gisbergen et al. 1987; Van Opstal and Van Gisbergen 1987). We calculated the acceleration duration and the deceleration duration separately for four different amplitude bins (<7, 7–12, 12–18, 18–25°) for primary saccades and their amplitude-matched no-step counterparts, including only horizontal saccades in the analyses, as has been done in past studies (Van Gisbergen et al. 1987; Van Opstal and Van Gisbergen 1987). Figure 10 shows the acceleration durations (top panel), deceleration durations (middle panel), and the ratio of the two (bottom panel) as a function of amplitude for the entire group. Acceleration and deceleration profiles show a monotonically increasing relation as a function of amplitude, in general agreement with past work. We also observed that whereas the acceleration duration across amplitudes is closely matched (one-way ANOVA, $F = 0.15, P = 0.69$), the deceleration duration is significantly less for primary saccades, compared with their corresponding no-step counterparts (one-way ANOVA, $F = 9.24, P = 0.0024$). The ratio of the acceleration duration to the deceleration duration is also significantly different (one-way ANOVA, $F = 4.96, P = 0.026$), suggesting that the primary saccade of a hypometric correction trial, on average, accelerated normally but decelerated more abruptly, compared with the primary saccade of the no-step hypometric trial. Taken together, these results favor the generation of primary saccades in hypometric corrections as a consequence of the STOP process interrupting the GO process during execution, leading to truncation of the erroneous first saccade.

Based on the earlier results, we modified the race model to account for midflight truncation by allowing STOP and GO processes to compete beyond the saccade planning stage into its execution stage (see Fig. 11A). Such a race model should predict a systematic relation between the frequency of hypometric corrections and the reprocessing time: the time from the onset of the second target until the end of the first saccade. At shorter TSDs, corresponding to longer reprocessing times, the STOP process, having an early start, is more likely to inhibit the GO process before saccade execution begins, producing successful responses (see Fig. 1B1) and almost no hypometric corrections. On the other hand, at longer TSDs, corresponding to shorter reprocessing times, the STOP process would be unable to interrupt the GO process during either the planning or the execution stage and would result in nonhypometric erroneous first saccades (see Fig. 1B2), whereas for certain intermediate TSDs the likelihood of the STOP process colliding with the GO process during saccade execution would be greater, increasing the frequency. Thus the frequency of hypometric corrections is expected to peak at intermediate reprocessing times but decrease not only at longer but also at shorter reprocessing times. Figure 11B shows the observed frequency as a function of reprocessing times for the nine typical subjects. The frequency distributions conform to the predictions of the extended race model, which further validates the hypothesis that hypometric corrections are generated as a consequence of
rupted and brought to a halt. In practice, we used the median of target reached completion when the first saccade was interrupted and brought to a halt. In practice, we used the median of the STOP process interrupting the first saccade during execution.

The architecture of the race model in Fig. 11A allows us to directly estimate the duration of the covert STOP process that inhibits the erroneous first saccade during execution. The STOP process that was initiated with appearance of the final target reached completion when the first saccade was interrupted and brought to a halt. In practice, we used the median of the STOP process interrupting the first saccade during execution.

In earlier studies of the classical double-step task (Becker and Jurgens 1979; Lisberger et al. 1975), the performance of the subjects was accounted for by assuming two underlying processes: a GO1 process activated by the first stimulus and a GO2 process by the second stimulus, with the resulting saccade being the outcome of a race between the two. Recently Ludwig et al. (2007) also attempted to model double-step data using a subtle modification of the GO1–GO2 model. Their model includes broadly tuned activation units whose activity increases if the stimulus is in their response field and they undergo a passive decay if the stimulus moves away (disappears) from the response field. However, it must be emphasized that their simulated double-step paradigm differs from ours in a crucial respect—in their paradigm when the target steps to a new location it disappears from its earlier location. Thus it makes sense and may suffice to account for reduction in activation of the GO1 units by passive decay. However, in our paradigm the first target stays on even after the second target appears (see Fig. 1). This leads to constant activation of the units, thus preventing passive decay. Thus an active inhibitory process is needed to decrease the activity of the GO1 units to a mutual inhibitory interaction between the GO1 and GO2 processes: a GO1 process activated by the first stimulus and a GO2 process by the second stimulus, with the resulting saccade being the outcome of a race between the two. Recently Ludwig et al. (2007) also attempted to model double-step data using a subtle modification of the GO1–GO2 model. Their model includes broadly tuned activation units whose activity increases if the stimulus is in their response field and they undergo a passive decay if the stimulus moves away (disappears) from the response field. However, it must be emphasized that their simulated double-step paradigm differs from ours in a crucial respect—in their paradigm when the target steps to a new location it disappears from its earlier location. Thus it makes sense and may suffice to account for reduction in activation of the GO1 units by passive decay. However, in our paradigm the first target stays on even after the second target appears (see Fig. 1). This leads to constant activation of the units, thus preventing passive decay. Thus an active inhibitory process is needed to decrease the activity of the GO1 units to allow the GO2 units to rise and reach the threshold. In a similar vein, a mutual inhibitory interaction between the GO1 and GO2 processes (lateral inhibition) is also unlikely to produce the

A question that arises at this point is whether this TSRT for inhibiting a saccade during execution is different from the TSRT for inhibiting a saccade before it began? In other words, do they represent two distinct sources of inhibitory control or a common inhibitory mechanism acting at central and peripheral levels? We tested this by plotting the two TSRTs against each other for all subjects (see Fig. 12) and found a significant correlation between the two (Pearson’s correlation $r = 0.77$, $P = 0.0008$), consistent with the mechanism of unitary inhibitory control manifesting at different levels.

**DISCUSSION**

In this study, we addressed for the first time how the brain controls multisaccade gaze shifts by testing the predictions of a race model that has been used to successfully describe performance in double-step tasks. In the process, we have shown that, first, a saccade can be inhibited not only centrally during its programming but also peripherally during its execution, by the operation of unitary inhibitory control that can account for subjects’ performance. Second, the intersaccadic interval in a multisaccade gaze shift is a ballistic stage not subject to voluntary control. To the best of our knowledge, this is the first clear demonstration that the brain implements a point of no return in sensorimotor decision making.

**Race model of double-step saccade performance**

In earlier studies of the classical double-step task (Becker and Jurgens 1979; Lisberger et al. 1975), the performance of the subjects was accounted for by assuming two underlying processes: a GO1 process activated by the first stimulus and a GO2 process by the second stimulus, with the resulting saccade being the outcome of a race between the two. Recently Ludwig et al. (2007) also attempted to model double-step data using a subtle modification of the GO1–GO2 model. Their model includes broadly tuned activation units whose activity increases if the stimulus is in their response field and they undergo a passive decay if the stimulus moves away (disappears) from the response field. However, it must be emphasized that their simulated double-step paradigm differs from ours in a crucial respect—in their paradigm when the target steps to a new location it disappears from its earlier location. Thus it makes sense and may suffice to account for reduction in activation of the GO1 units by passive decay. However, in our paradigm the first target stays on even after the second target appears (see Fig. 1). This leads to constant activation of the units, thus preventing passive decay. Thus an active inhibitory process is needed to decrease the activity of the GO1 units to allow the GO2 units to rise and reach the threshold. In a similar vein, a mutual inhibitory interaction between the GO1 and GO2 processes (lateral inhibition) is also unlikely to produce the
behavioral outcome that we observe. In a task like the redirect task, where the GO1 process is always given a head start, it will always be more activated than the GO2 process. Thus its inhibitory influence on the GO2 process will be more than vice versa. As a consequence such an interaction will never allow the expression of the GO2 process. Therefore in the absence of an active inhibitory process that interrupts the current motor program (the GO1 process) it is hard to understand how competing motor programs that begin later can ever get to be expressed at the behavioral level.

In contrast, our model that includes an inhibitory STOP process is congruent with evidence from other studies using more detailed simulations (Camalier et al. 2007; see also Verbruggen and Logan 2008) that point to the existence of a STOP process that starts following the appearance of the second target, which inhibits the GO1 process to allow the GO2 process to compensate for the target step. Thus redirecting saccades in double-step tasks is presumed to require an inhibitory process. The duration of this covert inhibitory process, the target step reaction time (Camalier et al. 2007; Murthy et al. 2009), can be quantified by adapting the theoretical framework developed to calculate the stop signal reaction time (Logan 1994). Although our estimates of TSRT (101 ms from simulations; 107 ms from conventional methods) may be a bit lower than the SSRT values estimated from some studies using versions of the stop signal paradigms (SSRT = 125–145 ms: Hanes and Carpenter 1999; SSRT = 128 ms: Assress and Carpenter 2001), they are nevertheless comparable to estimates from other studies (SSRT = 113 ms: Cabel et al. 2000; SSRT = 112 ms: Kornylo et al. 2003). Lower TSRT could be due to a higher than usual proportion of step trials in our task (50%) compared with earlier paradigms (e.g., 35%: Hanes and
Hierarchical control: unitary control at multiple levels

Consistent with previous work (Camalier et al. 2007), the current study explains behavior in a redirect task as arising from an interaction of GO1, GO2, and STOP processes, where the initial target initiates the GO1 process and the final target initiates the GO2 and the STOP processes. In this race model framework the STOP process may interrupt the GO process during the planning stage or sometimes, albeit infrequently, during the execution stage, leading to premature truncation of the first saccade. If this were true then the incidence of hypometric correction would be a function of the duration of the first saccade, which in turn is related directly to its amplitude. In fact earlier studies that have used smaller target eccentricities have reported few such hypometric responses (eccentricity = 15°: Colonius et al. 2001; Ozyurt et al. 2003; eccentricity = 16–20°: Walton and Gandhi 2006; eccentricity \( \equiv 10° \): Camalier et al. 2007) or none (e.g., in monkeys, eccentricity = 9°: Hanes and Carpenter 1999; Hanes and Schall 1995; in humans, eccentricity = 3–3.5°: Assress and Carpenter 2001; Kornylo et al. 2003), whereas we have observed relatively higher frequency of hypometric corrections (\( \sim 15% \)), despite a very generous amplitude cutoff (6°) that is likely to be due to the larger target eccentricity (\( \sim 30° \)) used in this study, which has allowed greater time for the STOP signal to interact with the saccade during its execution, thus lending support to the hypothesis that the STOP process can interrupt the GO process during saccade execution.

Our observation that such an interaction between the GO and STOP processes can also occur at the level of saccade execution implies that inhibitory control manifests at multiple levels in the oculomotor system. Previous studies that have implicated the superior colliculus (SC) fixation neurons during countermanding behavior (Paré and Hanes 2003) have shown the time course of reactivation (\( \sim 104 \) ms) of these neurons closely corresponds to and precedes (\( \sim 25 \) ms) the behavioral estimates of the cancellation times, the SSRT (analogous to TSRT). The modulation of frontal eye field (FEF) fixation neurons also occurs in the same timescale (Hanes et al. 1998). In our study the duration of the STOP process required to halt the primary saccade under preparation is more or less similar in duration (107 ms) to the fixation neurons of the FEF and SC. However, the duration of the STOP process required to halt the primary saccade in execution is significantly larger (152 ms). Thus for the latter form of control we speculate the involvement of neurons further downstream to the SC and FEF. Based on studies showing midflight saccade interruption by the stimulation of the brain stem omnipause neurons (OPNs; Keller 1974, 1977; Keller and Edelman 1994; Keller et al. 1996), we speculate that the reactivation of OPN may lead to inhibition of saccades during their execution. However, because this inference is indirect we cannot rule out the role of upstream neurons such as SC and FEF fixation neurons as well.

A conceptual issue that these data raise is whether inhibitory control occurring at multiple levels is equivalent to the brain implementing multiple independent STOP processes (Coxon et al. 2007; DeJong et al. 1995; van den Wildenberg and van der Molen 2004) or, alternatively, a unitary control mechanism that manifests at multiple levels. One line of evidence that would favor the former hypothesis is the observation that the TSRT to inhibit the saccade during execution is consistently greater (by \( \sim 44 \) ms) than the TSRT to inhibit the saccade before it begins. However, because the two sets of TSRTs were strongly correlated, this suggests a shared basis for control (Morein-Zamir et al. 2004; van Boxtel et al. 2001), which undermines the independent hypothesis. Taken together, these data seem to suggest the contribution of neurons that inhibit saccade production across different brain areas such as the frontal eye fields (Burman and Bruce 1997; Hanes et al. 1998; Izawa et al.
Ballistic stages in saccade programming: is there a point of no return?

Cognitive theorists have long since speculated that information processing stages underlying sensorimotor behaviors may be distinguished into controlled and ballistic stages (Bartlett 1958; Osman et al. 1986). Although controlled stages are thought to be under central control with respect to whether they ultimately lead to overt movement (Logan 1981), ballistic stages are believed to be inextricably linked to overt movement and thus cannot be stopped once they begin. The temporal boundary between controlled and ballistic stage is typically referred to as the point of no return (Bartlett 1958). Although it is intuitive to think that fast voluntary movements are difficult to modify or to cancel, especially prior to overt movement (e.g., Henry and Harrison 1961; Slater-Hammel 1960), experimental evidence for a point of no return has remained inconclusive (Cavina-Pratesi et al. 2004; DeJong et al. 1990; Logan et al. 1984; McGarry and Franks 1997; Osman et al. 1986, 1990; van Boxtel et al. 2001).

Kornylco et al. (2003) used a saccadic countermanding paradigm to look for evidence of the point of no return in the oculomotor system. In this study, the performance in a saccade countermanding task could be simulated only by assuming a ballistic stage of 28–60 ms for humans and 9–25 ms in monkeys for saccadic eye movements. In contrast, other studies (e.g., Colonius et al. 2001; Corneil et al. 1999; Walton and Gandhi 2006) have observed hypometric saccades during the execution of an erroneous first saccade, presumably due to midflight control, arguing against the notion of a ballistic stage. Although the above-cited studies have focused on single saccades using a countermanding paradigm, we have probed multisaccade gaze shifts using a redirect task in conjunction with the race model that provides a more rigorous test of the notion of a point of no return. Our finding that the proportion of erroneous first saccades violating the STOP2 cutoff could be interpreted as evidence of a possible ballistic stage. However, in contrast to the first saccades (both nonhypometric and hypometric), a vast majority of secondary saccades violated the cutoff defined by STOP2 by a surprisingly large extent (~114 ms in duration and ~78% in frequency). Further, unlike the first saccades, these violations were not restricted to the short TSDs. As a result, we argue that the programming of the secondary saccades reflects a ballistic stage not subject to voluntary control. We consider the following arguments that could be made against this hypothesis. One could argue that the secondary saccades were produced as a result of presenting the final target during the saccadic dead time (Ludwig et al. 2007), which is the point in time during saccadic preparation at which no new visual information can change the upcoming movement. Saccade 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). We can rule out this argument since there was more than adequate time (median latency across subjects ~215 ms) for visual information to modify the decision process. Alternately, one could suppose that inhibiting the secondary saccade could be more difficult and therefore the underlying race model could require a STOP process with a longer duration. In other words, one can assume that hypometric corrections and the hypometric errors are outcomes of a different race model. A number of reasons rule out this possibility. First, if hypometric errors are generated from a race model, they ought to be the shorter-latency secondary saccades than the corrective saccades of hypometric corrections. However, in 14/15 subjects this was not the case (P < 0.05, one-tail t-test). Second, even the higher TSRT, indicative of the peripheral STOP process, could not account for roughly 70% of the violations. Last, one could argue that hypometric errors represent instances when the subject did not initiate the STOP process. However, if this were true the proportion of violation should be similar for both the first saccade and the secondary saccade. However, because the proportion and the extent of violation are significantly greater for secondary saccades, despite using the same underlying race model, this possibility can be ruled out. Thus the most parsimonious explanation to account for the long latencies of the secondary saccades is if we assume that the intersaccadic interval in a multisaccade response is a ballistic stage.

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