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Countermanding saccades in macaque

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Abstract

A countermanding paradigm was utilized to investigate the regulation of saccade initiation. Two rhesus monkeys were instructed to generate a saccade to a peripheral target; however, on a fraction of trials after a delay, the monkeys were signaled to inhibit saccade initiation. With short delays between the presentation of the target and the signal to inhibit saccade generation, monkeys withheld saccades to the peripheral target. As the delay of the stop signal increased, monkeys increasingly failed to withhold the saccade. The hypothesis that the generation of the saccade is determined by a race between a go and a stop process provides three explicit means of estimating the covert latency of response to the stop signal. This latency, known as stop signal reaction time, was estimated to be on average 82 ms for both monkeys. Because the stop signal latency represents the time required to exert inhibitory control over saccade production, the countermanding paradigm will be useful for studying neural mechanisms that regulate saccade initiation.

Keywords: Saccade, Saccade latency, Eye movement, Oculomotor system, Monkey

Introduction

Neural circuits mediating inhibitory control over saccade production are being elucidated (Hikosaka & Wurtz, 1989; Munoz & Wurtz, 1992, 1993*a*,*b*). Nevertheless, how saccade production is regulated is not entirely understood even though numerous manipulations of saccade latency have been investigated (reviewed by Carpenter, 1988; Fischer & Weber, 1993). In a different field of inquiry another method, known as the countermanding paradigm, and associated theoretical construct have been developed to investigate the voluntary control of movement (Bartlett et al., 1961; Lappin & Eriksen, 1966; reviewed by Logan & Cowan, 1984; Osman et al., 1986). We implemented a version of the countermanding paradigm using rhesus monkeys to investigate the regulation of saccade initiation. A race model developed to explain performance in the countermanding paradigm was used to derive estimates of the covert latency of voluntary control over saccade production. The neural concomitants of responding to countermanding signals can now be investigated. In addition, the countermanding procedure may be a useful tool for diagnosing and investigating neurological disorders that impair the initiation or inhibition of saccadic eye movements including Parkinson's disease (Melvill Jones & DeJong, 1971; White et al., 1983; Teravainen & Calne, 1980), Huntington's disease (Leigh et al., 1983; Lasker et al., 1988), schizophrenia (Makert & Flechtner, 1989; Fukushima et al., 1990; Abel et al., 1992), Alzheimer's disease (Pirozzolo & Hansch, 1981; Hershey et al., 1983; Fletcher & Sharpe, 1986; Scinto et al., 1994), and AIDS (Currie et al., 1988).

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Methods

Subjects and surgery

Data were collected from two rhesus monkeys (*Macaca mulatta*). The animals were cared for in accordance with the National Institute of Health's Guide for the Care and Use of Laboratory Animals and the guidelines of the Vanderbilt Animal Care and Use Committee. All surgical procedures were carried out under aseptic conditions. Initially, the monkeys were tranquilized with ketamine (15 mg/kg) for intubation, catheterization, and cleaning. During surgery the monkeys were anesthetized with an N_2O/O_2 mixture and isoflurane (2-3%). ECG, rectal temperature, and respiration were monitored. Expired pCO_2 was maintained at approximately 4%. A scleral search coil (Judge et al., 1980) was implanted subconjunctively and a stainless-steel post was attached to the skull to restrain the head during testing.

Task

All trials began with the presentation of a central fixation spot (Fig. 1). Following a variable delay (250–350 ms), a target appeared at one of two locations on the horizontal meridian (6-deg eccentricity for Monkey C and 16 deg for Monkey B). Simultaneously, the fixation spot disappeared signaling the monkey to generate a saccade to the target. On 25% of the trials, the fixation spot reappeared after a delay referred to as *stop signal delay*. It is important to note that on these *Stop Signal* trials the target stimulus remained on. Stop signal delays ranged from 25–275 ms in 25- or 50-ms steps. During the 75% of trials in which the stop signal was not presented (referred to as *No Signal* trials), monkeys were rewarded for generating a single sac-

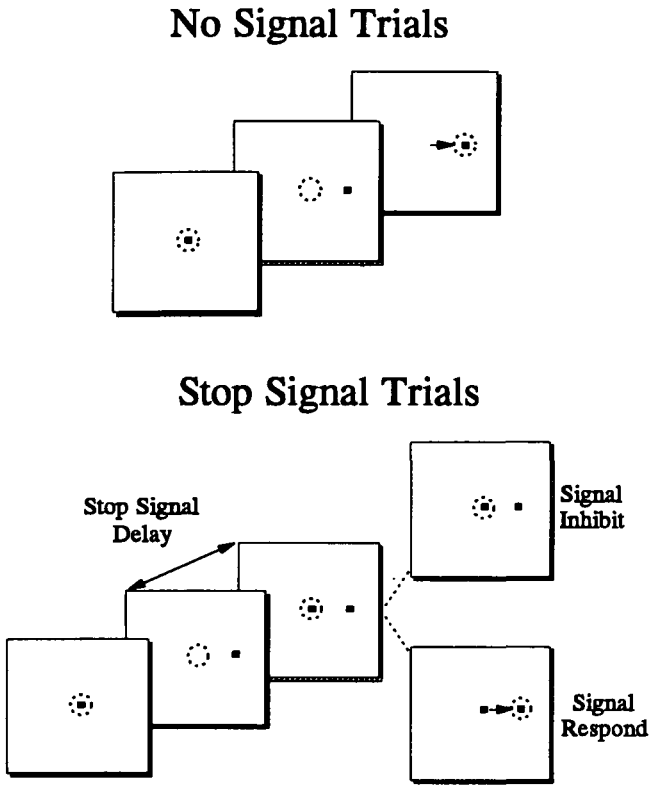

Fig. 1. Schematic representation of countermanding task showing *no signal* and *stop signal* trial types. The overlying panels show the temporal sequence of the visual displays. The dashed circle represents the monkey's current point of fixation. The arrow represents the saccade to the target. All trials begin with the presentation of a central fixation spot. After the fixation of this spot for a specified interval, the fixation spot disappears. Simultaneously, a target appears in the periphery. During *no signal* trials, the fixation spot remains off and the subject is rewarded for generating a single saccade to the target. During *stop signal* trials after the stop signal delay, the fixation spot reappears signaling the subject to withhold saccade generation. Two outcomes are possible on each *stop signal* trial. The subject can either generate a saccade to the target, *signal respond* trials, or the subject can successfully inhibit the saccade, *signal inhibit* trials.

cade to the peripheral target within 500 ms. During *stop signal* trials, monkeys were rewarded for maintaining fixation on the central spot for 400 ms (referred to as *Signal Inhibit* trials), or a time out period of 500 ms was imposed if the monkeys generated a saccade to the peripheral target (referred to as *Signal Respond* trials). By utilizing 25% *stop signal* trials and 75% *no signal* trials and a maximum permissible saccade latency of 500 ms on *no signal* trials, we ensured that the monkeys made a speeded response to the presentation of the target and did not adopt the strategy of postponing the saccade until they could determine if the stop signal was going to occur. Also, by imposing a 500-ms time out period during *signal respond* trials, we believe that the monkeys were not biased toward generating a saccade or withholding it.

Each animal was tested for approximately 3 h per day, 5 days per week. During testing, fruit juice was given as positive reinforcement. Monkeys' access to water in the home cage was controlled and monitored, fluids were supplemented as needed. Monkeys were seated in an enclosed chair within a magnetic field to monitor eye position using a scleral search coil. The stimuli were presented on a video monitor (Conrac 7241, 60 Hz interlaced) using computer-controlled raster graphics (Peritek VCH-Q, 512 × 512 resolution). The fixation spot was a yellow (CIE chromaticity coordinates $x = 0.411$, $y = 0.513$) square subtending 0.2 deg of visual angle and the target stimulus was a white square (CIE $x = 0.272$, $y = 0.272$) subtending from 0.3 deg to 1 deg of visual angle depending on its eccentricity and was 30 cd/m^2 on a 1 cd/m^2 background.

Data collection and analysis

The experiments were under computer control (PDP 11/83) which presented the stimuli, recorded the eye movements, and delivered the juice reward. Eye position was monitored with a scleral search coil (Robinson, 1963) sampled at 250 Hz and stored with event times on disk for offline analysis. Saccades were detected using a computer algorithm that searched first for significantly elevated velocity (≥30 deg/s). Saccade initiation and termination were then defined as the beginning and end of the monotonic change in eye position lasting 12 ms, before and after the high-velocity gaze shift. Based on the 250-Hz sampling rate, this method is accurate to within less than 4 ms.

Distributions of saccade amplitude and peak velocity generated in *no signal* trials and *signal respond* trials were compared using a nonparametric median test (Siegel & Castellan, 1988). Differences between the *signal respond* saccade latencies and *no signal* latencies were tested with a one-way analysis of variance (Sokal & Rohlf, 1981). A linear regression analysis (Sokal & Rohlf, 1981) was used to determine whether *signal respond* saccade latencies varied significantly as a function of increasing stop signal delay.

Results

A total of 2191 *no signal* trials and 656 *stop signal* trials were collected from monkey B during 12 sessions and 883 *no signal* trials and 358 *stop signal* trials from monkey C during 6 sessions. Stop signal delays ranged from 25 to 275 in 50-ms steps for monkey B and from 25 to 250 in 25-ms steps for monkey C.

Three explicit findings were determined from the raw data: the average (±S.E.M.) saccade latency on *no signal* trials (monkey B 214.8 ± 1.0 ms, monkey C 219.9 ± 1.4 ms), the average saccade latency on *signal respond* trials (monkey B 201.1 ± 2.1 ms, monkey C 211.9 ± 2.8 ms), and the probability of generating a saccade when a stop signal was given. Fig. 2 shows the probability of generating a saccade (*signal respond* trials) when a stop signal was given as a function of stop signal delay for each monkey. These inhibition functions show that following short stop signal delays, both monkeys successfully withheld saccades to the target but as the stop signal delay increased monkeys increasingly failed to withhold the saccade. The probability of generating a saccade at the 25-ms stop signal delay was 0.06 for monkey B and 0.12 for monkey C. At the longest stop signal delays tested, 275 ms for monkey B and 250 ms for monkey C, the probability of generating a saccade was 0.98 and 0.85, respectively. Note that for monkey C the probability of responding increases as a function of stop signal delay; however, the function is not monotonic. Therefore, to obtain a smooth representation of the inhibition function, a logistic equation

$$y = 1/\{1 + e^{[-(x-a)/b]}\}$$

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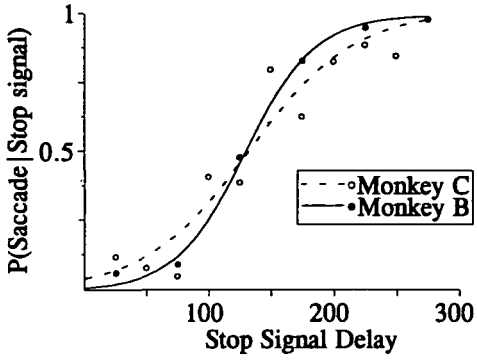

Fig. 2. Inhibition functions for both monkeys. The probability of generating a saccade given that a stop signal occurred is plotted as a function of the stop signal delay. The best-fit logistic equations are also shown.

was fit to the inhibition functions ($a = 129.27$, $b = 27.99$, $r^2 = 0.99$ for monkey B; $a = 132.60$, $b = 41.64$, $r^2 = 0.95$ for monkey C).

The most interesting issue under investigation is the duration required to inhibit the saccade being programmed, and thus maintain fixation on the central fixation spot. This duration, indicated by the latency of response to the presentation of the stop signal, is not explicit in the raw data. However, the application of a particular race model process provides a means of estimating the duration of this inhibitory process.

Based upon previous work (reviewed by Logan & Cowan, 1984), performance on the countermanding procedure was modeled as a race between a go and a stop process (Fig. 3). The go process generates a response following the presentation of the target stimulus that increases a subject's readiness to respond. This process includes both the release of fixation and programming the metrics of the saccade. The distribution of movement latencies during trials in which a stop signal is not presented (i.e. *no signal* trials) represents the outcome of the go process. The stop process inhibits movement in response to the presentation of the stop signal (i.e. the reappearance of the fixation spot). If the go process crosses its threshold before the stop process, the saccade will be generated (Fig. 3A). Alternatively, if the stop process crosses its threshold before the go process, the saccade will be inhibited (Fig. 3B). It is worth noting that the thresholds for the go and stop processes do not have to be, and most likely are not, at the same level. However, for ease of explanation the two thresholds are indicated as such in Fig. 3.

The version of the race model we implemented is based on the assumption that the go and stop processes are independent. Our data provided one possible test of the validity of this assumption. The amplitude and peak velocity of saccades generated on *no signal* trials, resulting from the go process alone, were compared to those in *signal respond* trials, in which the go and stop processes were competing in a "winner take all" race (Fig. 4). If the process of saccade generation was not independent of the process of inhibiting saccade generation, then one might expect reduced saccade velocity and/or hypometric saccades during *signal respond* trials. However, the distributions of saccade amplitude and peak velocity were not significantly different in *no signal* and *signal respond* trials ($P > 0.05$). Also, the scatter of points was similar for saccades produced in both trial conditions.

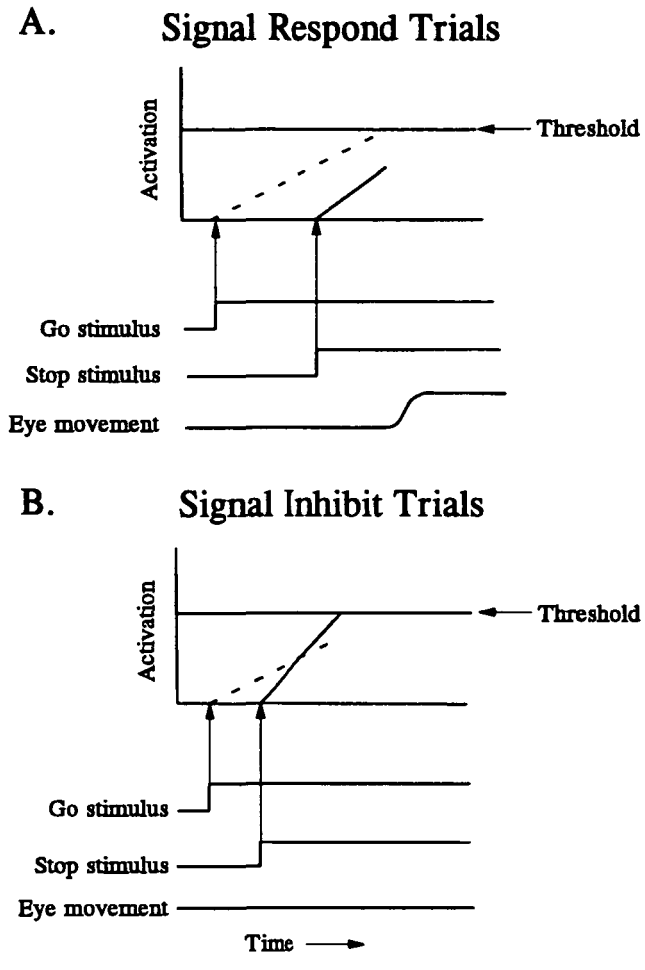

Fig. 3. Schematics of the two primary outcomes of the race model. Shown from top to bottom are plots of activation for go and stop processes as a function of time, go stimulus trace, stop stimulus trace, and eye movement trace. The upward deflection of the go and stop process traces signify the points in time when these stimuli are presented. The deflection in the eye movement trace signifies saccade onset. When the go stimulus is presented, the activation of this process begins to increase. Similarly, when the stop stimulus is presented, the activation of the stop process begins to increase. Whichever process reaches its threshold first determines whether a saccade will be generated. A: *Signal respond* trials in which the go process reaches its threshold before the stop process. B: *Signal inhibit* trials in which the stop process reaches its threshold before the go process.

The monotonically increasing inhibition function arises because increasing the stop signal delay postpones the onset of the stop process, thus increasing the probability that the go process will reach its threshold before the stop process reaches its threshold. This can be seen in Fig. 5 by comparing the top and bottom panels. In Fig. 5, the timing of two *stop signal* trials is superimposed on the *no signal* saccade latency distribution for monkey B. At a relatively short stop signal delay (Fig. 5A), the stop process more often reaches its threshold before the go process, resulting in a majority of *signal inhibit* trials. In Fig. 5B, the presentation of the stop signal has been delayed, which effectively eliminates more of the *no signal* saccade latency distribution, thereby increasing the probability of inadvertently responding and reducing the probability of inhibiting saccade

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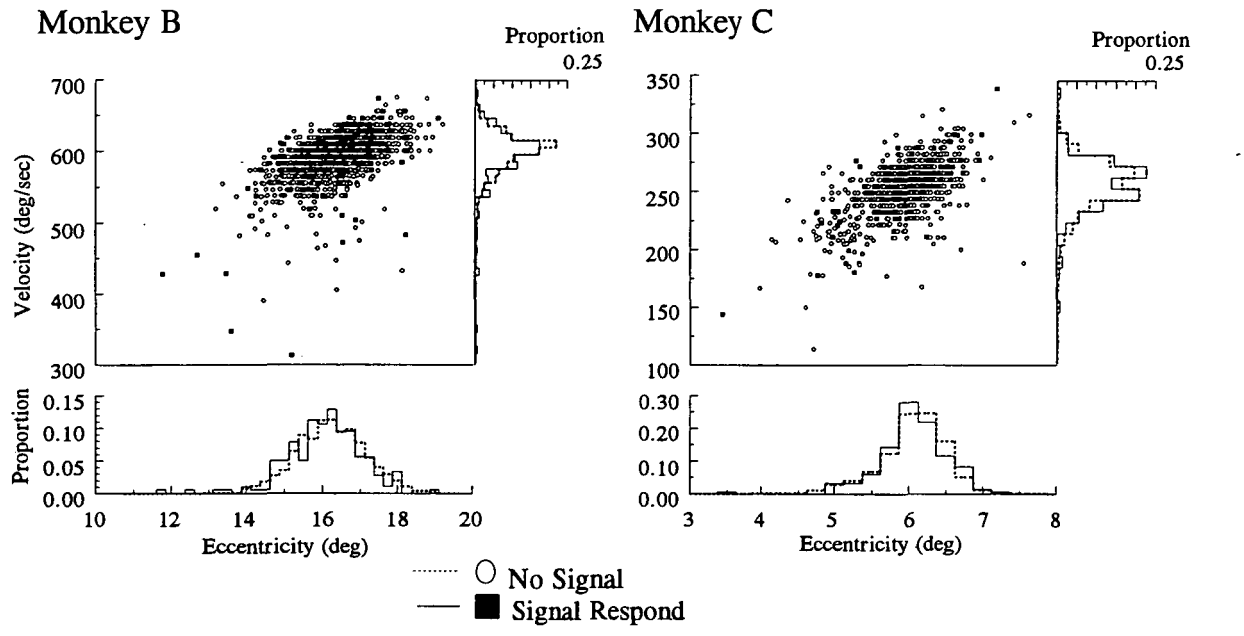

Fig. 4. Saccade dynamics for *no signal* and *signal respond* trials for both monkeys. Scatterplots and associated distributions of saccade amplitude and peak velocity are plotted. Distributions show the proportions of total trials in each bin. Binwidths are 10 deg/s for the velocity distributions and 0.25 deg for the eccentricity distributions.

generation. If one were to make plots similar to those in Fig. 5 for all of the stop signal delays, it is easy to see that if the stop signal delay occurred early enough, all saccades ought to be inhibited. Conversely, if the stop signal occurred late enough no saccades would be inhibited. Plotting the probability of responding at stop signal delays between these extremes produces the inhibition function.

The inhibition function is used in the context of the race model to estimate the latency of the inhibitory process that countermands saccade generation known as *stop signal reaction time*. The race model provides three methods for estimating the *stop signal reaction time*: one based on integration, another using the mean of the inhibition function, and the third using the median of the inhibition function.

The method of estimating the covert latency of response to the stop signal involving integration assumes that the duration from the onset of the stop process to its threshold is constant for a given stop signal delay. Initially, this assumption seems unwarranted as it is implausible that a physiological process would take a constant amount of time to execute. However, violation of this assumption does not substantially change the results of this particular analysis (Logan & Cowan, 1984; DeJong et al., 1990). With this assumption, as illustrated in Fig. 5A, the *stop signal reaction time* at each stop signal delay can be determined by integrating the *no signal* saccade latency distribution, beginning at zero, until the integral equals the proportion of *signal respond* trials at that stop signal delay. The time value at that location represents the finish line of the stop process. Thus, the time between the onset of the stop signal and this finish line represents the *stop signal reaction time* at this stop signal delay. The results of this method of analysis at the different stop signal delays are plotted in Fig. 6. *Stop signal reaction times* ranged from 127 to 61 ms for monkey B and 150 to 15 ms for monkey C. This method of estimating the average *stop signal reaction time* is very sensitive to the shapes of the *no signal* saccade latency distribution and the inhibition function. The estimates are unreliable at both tails of these distributions (i.e. very short and very long stop signal delays) and are most reliable at intermediate stop signal delays. Thus, we calculated the average (±S.E.M.) *stop signal reaction time* using three ranges of stop signal delays: all stop signal delays (monkey B 85 ± 9.2 ms, monkey C 83 ± 12.3 ms), stop signal delays during which the probability of responding given the occurrence of the stop signal was between 10 and 90% (monkey B 82 ± 1.0 ms, monkey C 67 ± 12.0 ms), and all stop signal delays except the shortest and longest stop signal delays tested (monkey B 80 ± 3.1 ms, monkey C 83 ± 9.1 ms). The estimated *stop signal reaction times* for monkey B were essentially the same using all three ranges of stop signal delays. For monkey C, the variability using the different ranges of stop signal delays is due to the variability in the inhibition function.

The two other methods for estimating the *stop signal reaction time* assume that the *stop signal reaction time* is a random variable. Logan and Cowan (1984) showed that the mean *stop signal reaction time* equals the difference between the mean of the saccade latency during *no signal* trials and the mean of the inhibition function. Logan and Cowan (1984) also showed that it is possible to use the median, instead of the mean, if the inhibition function is symmetrical. The median of the inhibition function is simply the stop signal delay at which the probability of responding given the presentation of a stop signal is 0.5. The mean of the inhibition function is determined by treating the inhibition function as a cumulative distribution and converting it to a probability density distribution. The mean of the inhibition function is simply the mean of this probability density distribution. Using the mean value of the inhibition function, the average *stop signal reaction time* was 79 ms for monkey B and 89 ms for monkey C. Using the median value of the inhi-

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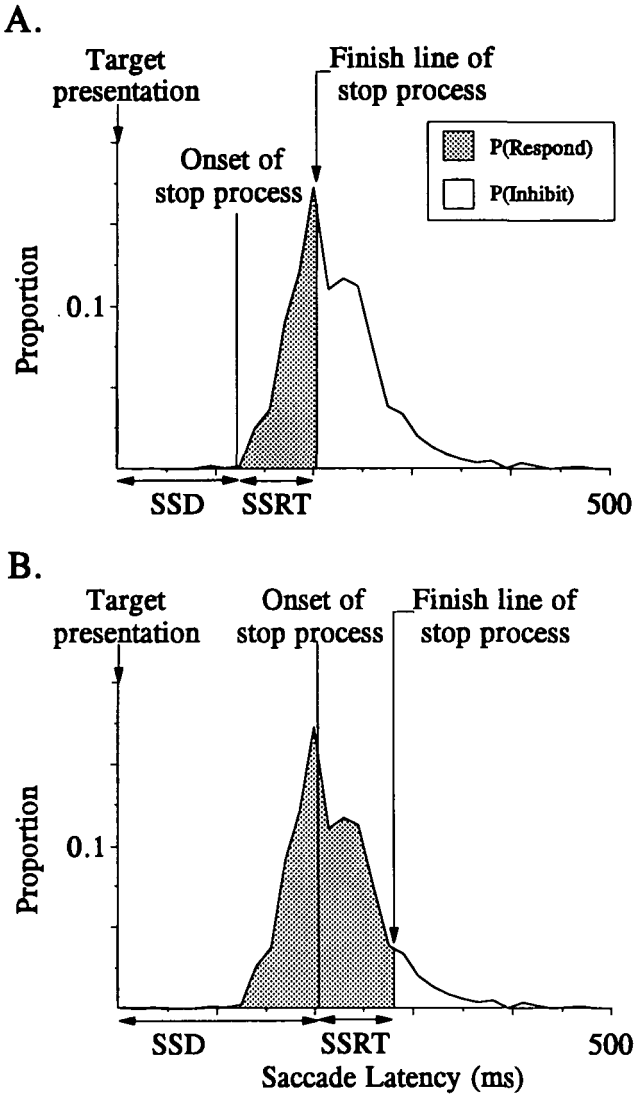

Fig. 5. Schematic representation of the predictions of the race model with a short stop signal delay (A) and a longer stop signal delay (B). The timing of two *stop signal* trials is superimposed on the *no signal* saccade latency distribution for monkey B. The comparison of A and B indicates how the probability of inhibiting a response, P (inhibit), and the probability of responding given a stop signal, P (respond), vary as a function of stop signal delay. SSD: stop signal delay; and SSRT: *stop signal reaction time*.

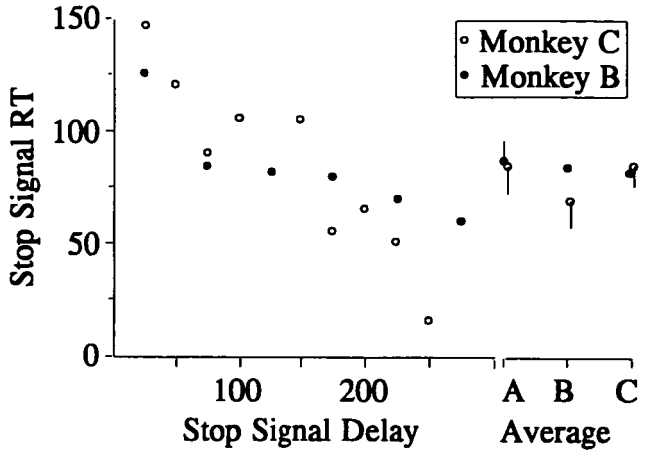

Fig. 6. Plot of *stop signal reaction time vs.* stop signal delay for both monkeys. *Stop signal reaction times* were determined by integrating the *no signal* saccade latency distribution until the integral equals the proportion of *signal respond* trials at that stop signal delay. The average *stop signal reaction time* using three ranges of stop signal delays is shown at the right: A—all stop signal delays, B—stop signal delays during which the probability of responding given the occurrence of the stop signal was between 10 and 90%, and C—all stop signal delays except the shortest and longest stop signal delays tested. The vertical bars at each data point indicate one standard error of the mean (S.E.M.). If no vertical bar is shown, the S.E.M. is less than the height of the data symbol.

bition function, the average *stop signal reaction time* was 84 ms for monkey B and 87 ms for monkey C. Table 1 shows the average *stop signal reaction time* using these three methods of estimation: one using the mean of the inhibition function, another using the median of the inhibition function, and the third based upon integration. Using all three methods, the average *stop signal reaction time* for both monkeys was 82 ms.

The latency of saccades that escape inhibition (*signal respond*) can be measured. An adequate model of performance in the countermanding paradigm should be able to predict the latency of these responses. Thus, three predictions that follow from the race model were tested (Fig. 7).

First, the average *signal respond* latencies should be less than the average *no signal* saccade latencies. *Signal respond* latencies come from the same distribution as the *no signal* latencies, except the countermanding process eliminates the upper tail (see Fig. 5). Thus, the mean of the left part of the distribution is less than the mean of the whole distribution. For both monkeys, the average *signal respond* saccade latency was significantly less than the average *no signal* saccade latency (monkey B: $df = 2548$, $F = 4.03$, $P < 0.05$; monkey C: $df = 1050$; $F = 5.72$, $P < 0.05$). The average (±S.E.M.) saccade latency on *signal respond* trials was 201.1 ± 2.1 ms for monkey B and 211.9 ± 2.8 ms for monkey C. The average saccade latency on *no sig-*

Table 1. *The average (±S.E.M.) stop signal reaction time using three methods of estimation are shown: one using the mean of the inhibition function, another using the median of the inhibition function, and the third based upon integration.*[a]

	Mean	Median	Integration		
			A	B	C
Monkey B	79	84	85 ± 9.2	82 ± 1.0	80 ± 3.1
Monkey C	89	87	83 ± 12.3	67 ± 12.0	83 ± 9.1

[a]The method of integration is very sensitive to the shape of the *no signal* saccade latency distribution and the inhibition function. Thus, the average *stop signal reaction time* using three ranges of stop signal delays is shown for the method of integration: A—all stop signal delays, B—stop signal delays during which the probability of responding given the occurrence of the stop signal was between 10 and 90%, and C—all stop signal delays except the shortest and longest stop signal delays tested. Times are measured in milliseconds.

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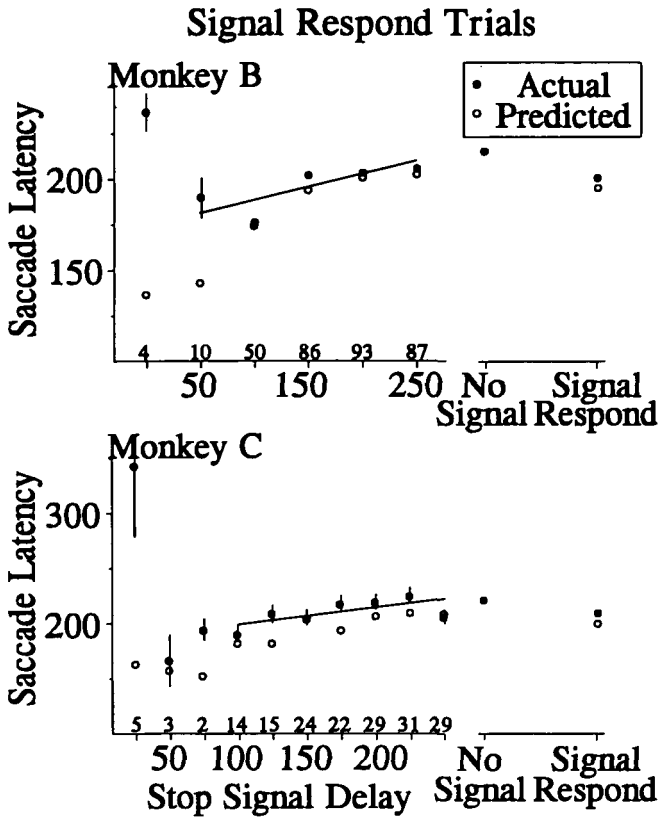

Fig. 7. Actual and predicted average saccade latencies on *signal respond* trials for both monkeys. The diagonal lines indicate a significant linear regression ($P < 0.01$). The vertical bars at each data point indicate one standard error of the mean (S.E.M.). If no vertical bar is shown the S.E.M. is less than the height of the data symbol. The actual average *no signal* saccade latency and the actual and predicted average *signal respond* saccade latency are shown at the right. The numbers above the abscissa indicate the number of *signal respond* trials at each stop signal delay.

nal trials was 214.8 ± 1.0 ms for monkey B and 219.9 ± 1.4 ms for monkey C.

Second, *signal respond* latencies should increase with stop signal delay. As stop signal delays increase slower go responses can reach their threshold before the stop process, resulting in *signal respond* trials. This prediction is commonly violated at short stop signal delays since so few *signal respond* trials occur at these delays (Logan & Cowan, 1984). Thus, the linear regression analysis did not include the *signal respond* trials that occurred at stop signal delays in which less than ten trials were produced. The regression analysis showed that the actual *signal respond* saccade latencies increased significantly with stop signal delay (monkey B: $df = 328$, $t = 3.95$, $b = 0.15$, $P < 0.01$; monkey C: $df = 162$, $t = 2.66$, $b = 0.14$, $P < 0.01$).

Third, the actual *signal respond* saccade latencies should be comparable to those predicted by the model. Predicted values at each stop signal delay were determined by averaging the *no signal* saccade latencies that lie to the left of the stop signal finish line (Fig. 5). Except for the shorter stop signal delays, where there were few *signal respond* trials, the average *signal respond* saccade latencies predicted by the race model are similar to the actual average latencies generated by the monkeys.

Discussion

This study represents an initial stage of our research aimed at elucidating the neural mechanisms underlying the regulation of saccade production. We have demonstrated that macaque monkeys can perform a saccade task that requires voluntary inhibitory control of gaze shifts. This study represents the first use of the countermanding paradigm, originally developed in studies of human behavior (Lappin & Eriksen, 1966; Logan, 1983; Logan et al., 1984; Logan & Cowan, 1984, DeJong et al., 1990; Osman et al., 1986), to investigate gaze control. Employing a race model of performance in the countermanding paradigm, we were able to estimate the time course of voluntary control over saccade production.

Relation to previous work

Many studies of manual choice and simple response time have used a race model to explain a subject's performance in the countermanding paradigm in humans (Logan, 1981, 1982, 1983; reviewed by Logan & Cowan, 1984; Osman et al., 1986; DeJong et al., 1990). One simplifying assumption of the race model is that the go and stop processes evolve independently. However, once one process reaches its threshold the other process is canceled by mechanisms that we currently do not understand. Several studies have provided evidence that is consistent with this assumption of independence (DeJong et al., 1990; Jennings et al., 1992). The results of the current study are also consistent with this assumption. We have shown that the saccades generated in *signal respond* trials when the stop process and the go process were both active were not slower than the saccades generated in *no signal* trials in which the go process operated alone. We take this as evidence for independence because if the go and stop processes were not independent, then the various inhibitory neural circuits regulating saccade initiation (see below) would not be entirely shut off in *signal respond* trials thereby reducing the drive on the saccade generating burst cells. This reduction would result in slower saccades.

We used three methods for estimating the covert latency of inhibitory control known as *stop signal reaction time*. First, the average *stop signal reaction time* was estimated at each stop signal delay by integrating the *no signal* saccade latency distribution until the integral equaled the probability of responding given the occurrence of the stop signal. This integration method is based upon the assumption that the duration of the effective stop process is constant at each stop signal delay. Initially, this assumption may seem unfounded since it is difficult to believe that a physiological process could take a constant amount of time to execute. However, using Monte Carlo simulations DeJong et al. (1990) showed that the effects of violating this assumption were quite small. Also, Logan and Cowan (1984) mathematically analyzed the consequences of this assumption and found that it introduced only small errors. Using this method, *stop signal reaction times* tended to decrease with increasing stop signal delay. This decline in *stop signal reaction time* is a common feature of most countermanding studies (reviewed by Logan & Cowan, 1984). This decrease is not generally consistent with the race model; however, the decrease may be a consequence of variability in the *stop signal reaction time* (Logan & Burkell, 1986). This method of estimating the average *stop signal reaction time* is very sensitive to the shape of the *no signal* saccade latency distribution and the inhibition

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function. Thus, the decline in *stop signal reaction time* as a function of increasing stop signal delay may also be due to this increased sensitivity at the tails of the inhibition function and the *no signal* saccade latency distribution. The two other methods of estimating the average *stop signal reaction time* assume that the *stop signal reaction time* is a random variable and estimate it as the difference between the average saccade latency on *no signal* trials and either the mean or median of the inhibition function.

Using these methods of estimation, we found the average *stop signal reaction time* to be 82 ms. Thus, if a saccade had not been generated within approximately 80 ms after the onset of the stop signal, saccade generation would be inhibited. In other words, once the stop signal was presented it took around 80 ms to exert inhibitory control over saccade programming. It is worth noting that the *stop signal reaction time* can be influenced by experimental factors such as fixation spot intensity and the percentage of *stop signal* trials. Using a variety of movement types and conditions in humans, other investigators have commonly found *stop signal reaction times* to range between 100 and 400 ms (e.g. Lappin & Eriksen, 1966; Logan, 1982, 1983; Zbrodoff & Logan, 1986). Specific possibilities may account for the significantly shorter stop signal latencies we found as compared to other investigators. First, the current study used rhesus monkeys as subjects while all other countermanding studies have used human subjects. Previous studies have noted faster reaction times for macaque monkeys than humans (reviewed by Fischer & Weber, 1993). Also, the current study is the first to use the countermanding task to investigate saccade generation. Finally, most previous human work has used choice reaction time tasks that are known to entail longer stop signal latencies (Logan et al., 1984).

Previous investigations of gaze control have presented subjects with two target steps to probe the time course of saccade programming (Westheimer, 1954; Wheeless et al., 1966; Komoda et al., 1973; Lisberger et al., 1975; Becker & Jurgens, 1979). In fact, a comparable race model of saccade generation was formulated by Becker and Jurgens (1979), but the specific predictions and analytical procedures were not developed. In one condition, known as pulse-return, of some of these double-step saccade studies, the target jumped to the peripheral location and then back to its original location. Superficially, this resembles the countermanding procedure. However, our task conditions were significantly different. In our study, the reappearance of the fixation spot on *stop signal* trials represented an imperative instruction signal rather than a second target for a saccade. In fact, unlike the double-step studies, the target stimulus remained on even when the fixation spot reappeared on *stop signal* trials.

These conditions create a situation quite different from the typical double-step task. This difference may be the basis for an apparent difference between saccade production in the countermanding task as compared to what was observed by Becker and Jurgens (1979). These authors observed that in the pulse-return condition some subjects made frequent saccades of intermediate amplitude. In our data, hypometric saccades were rare. Another possible explanation for the differing results of Becker and Jurgens' (1979) pulse-return condition and those of the current study involves the blocking of trials. Becker and Jurgens (1979) had 75% double-step trials within a block, while we had only 25% *stop signal* trials within a block. The subjects in Becker and Jurgens' (1979) experiment were in a condition in which they predominately had to track two target steps, while our subjects predominately had to track one target step. Thus, as Becker and Jurgens (1979) assert the subjects were programming two saccades in parallel. This parallel programming could account for the intermediate amplitude saccades in the pulse-return condition of Becker and Jurgens (1979) that were absent in the present study.

Implications for neural regulation of saccade initiation

Neural circuits mediating inhibitory control over saccade production have been identified. Recent work has identified a population of cells in the rostral superior colliculus that discharge during fixation and inhibit presaccadic burst cells in the superior colliculus (Munoz & Wurtz, 1993*a*,*b*). This population of neurons plays a central role in certain models of saccade generation (Optican et al., 1993). Presaccadic burst cells in the superior colliculus are tonically inhibited by GABAergic afferents from the substantia nigra pars reticulata (reviewed by Hikosaka & Wurtz, 1989). The nigrotectal inhibition is released by inhibitory influence from the oculomotor zone of the caudate nucleus (Hikosaka et al., 1993) which is innervated by the frontal eye field and supplementary eye field (Parthasarathy et al., 1992). The frontal eye field and supplementary eye field function in parallel in the production of purposive saccades (Bruce & Goldberg, 1985; Schall, 1991*a*,*b*; Schlag & Schlag-Rey, 1987; Hanes et al., 1995). Thus, *via* the basal ganglia pathway as well as through the direct projections to the superior colliculus, the two gaze control areas in the frontal cortex can exert regulatory control over saccade productions. In fact, recordings in supplementary eye field and the supplementary motor area have identified modulation of neural activity specifically related to withholding movements (Kurata & Tanji, 1985; Tanji & Kurata, 1985; Schall, 1991*a*).

A double-step task in which the target step was to a peripheral location and then back to the initial point of fixation (i.e. pulse-return) has been used as part of two neurophysiological studies of the superior colliculus (Mohler & Wurtz, 1976; Sparks, 1978). Both studies related the physiology of saccade-related cells in superior colliculus to saccade initiation. Mohler and Wurtz (1976) showed that the presaccadic activity in various types of saccade-related neurons within the superior colliculus, can occur even when a saccade is not generated. Subsequently, Sparks (1978) specifically analyzed neurons within the superior colliculus whose activity was tightly coupled to saccade initiation. These cells had a low-frequency prelude of activity followed by a burst of activity around 20 ms before saccade initiation. Sparks (1978) showed that on trials when a saccade to the peripheral target was inhibited the prelude of activity was observed. However, the saccade-related burst of activity was absent. Sparks' results suggest that the onset of bursting activity represents the trigger signal that initiates a saccade. Therefore, once the saccade-related burst neurons begin to burst a saccade is assured to occur. Although the studies of Mohler and Wurtz (1976) and Sparks (1978) implemented a simplified version of the countermanding task, they did not use variable stop signal delays, nor did they model the behavioral performance with the race model to estimate of the time required to exert inhibitory control over saccade production.

In conclusion, the countermanding procedure will provide a new opportunity to investigate how different classes of neurons in various visuomotor structures regulate saccade initia-

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tion. The central question will be to determine what patterns of neural modulation effectively predict performance and are correlated in time with the *stop signal reaction time*.

Note Added in Proof

We have now completed this experiment in a third monkey. A total of 1,021 no signal trials were collected under the same conditions. The average stop signal reaction time for this monkey was 86 ms.

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