

Canceling of Pursuit and Saccadic Eye Movements in Humans and Monkeys

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Kornlyo, Krista, Natalie Dill, Melissa Saenz, and Richard J. Krauzlis. Canceling of pursuit and saccadic eye movements in humans and monkeys. *J Neurophysiol* 89: 2984–2999, 2003; 10.1152/jn.00859.2002. The countermanding paradigm provides a useful tool for examining the mechanisms responsible for canceling eye movements. The key feature of this paradigm is that, on a minority of trials, a stop signal is introduced some time after the appearance of the target, indicating that the subject should cancel the incipient eye movement. If the delay in giving the stop signal is too long, subjects fail to cancel the eye movement to the target stimulus. By modeling this performance as a race between a *go* process triggered by the appearance of the target and a *stop* process triggered by the appearance of the stop signal, it is possible to estimate the processing interval associated with canceling the movement. We have now used this paradigm to analyze the canceling of pursuit and saccades. For pursuit, we obtained consistent estimates of the stop process regardless of our technique or assumptions—it took 50–60 ms to cancel pursuit in both humans and monkeys. For saccades, we found different values depending on our assumptions. When we assumed that saccade preparation was under inhibitory control up until movement onset, we found that saccades took longer to cancel (humans: ~110, monkeys: ~80 ms) than pursuit. However, when we assumed that saccade preparation includes a final “ballistic” interval not under inhibitory control, we found that the same rapid stop process that accounted for our pursuit results could also account for the canceling of saccades. We favor this second interpretation because canceling pursuit or saccades amounts to maintaining a state of fixation, and it is more parsimonious to assume that this involves a single inhibitory process associated with the fixation system, rather than two separate inhibitory processes depending on which type of eye movement will not be made. From our behavioral data, we estimate that this ballistic interval has a duration of 9–25 ms in monkeys, consistent with the known physiology of the final motor pathways for saccades, although we obtained longer values in humans (28–60 ms). Finally, we examined the effect of trial sequence during the countermanding task and found that pursuit and saccade latencies tended to be longer if the previous trial contained a stop signal than if it did not; these increases occurred regardless of whether the preceding trial was associated with the same or different type of eye movement. Together, these results suggest that a common inhibitory mechanism regulates the initiation of pursuit and saccades.

INTRODUCTION

Humans and monkeys use a combination of smooth-pursuit and saccadic eye movements to visually track objects in their environment. The motor properties of pursuit and saccades are quite distinct—pursuit is a continuous slow movement that smoothly rotates the eyes to follow moving targets, whereas

saccades are high-velocity movements that quickly direct the eyes toward the target. Despite these differences in output, the mechanisms for initiating pursuit and saccades are similar in that both movements require a break from fixation and involve the selection of a target. The release of fixation appears to involve signals that are common to the two eye movements. The extinction of a fixated stimulus before the appearance of a visual target (the “gap paradigm”) decreases the latencies of pursuit (Knox 1996; Krauzlis and Miles 1996a,c; Merrison and Carpenter 1995) and saccades (Fischer and Boch 1983; Fischer and Ramsperger 1984; Fischer et al. 1984; Krauzlis and Miles 1996a,c; Saslow 1967), and both movements exhibit the same dependence on “gap” duration. These findings have led to the suggestion that shared inputs, acting through different motor output mechanisms, control the release of fixation for pursuit and saccades (Krauzlis and Miles 1996c).

Likewise, the process of selecting a visual target is similar but not identical for pursuit and saccades. The latencies of saccades are increased when subjects must search the visual field for a target among additional distracting stimuli (Findlay 1997; Ottes et al. 1985; Williams 1967); similar increases in pursuit latency occur when subjects must choose between two stimuli moving in opposite directions (Ferrera and Lisberger 1995; Krauzlis et al. 1999) and also in the presence of stationary distractors (Knox 2001). However, selection by the two systems is not strictly yoked. Human and monkey subjects sometimes incorrectly begin pursuit in the direction of a “distractor” stimulus before changing their pursuit direction and making their initial saccade correctly toward the target stimulus (Krauzlis and Dill 2002; Krauzlis et al. 1999). The initiation of pursuit and saccades therefore appears to involve common neural signals that are read out in different ways for the two movements, perhaps reflecting differences in the desired tradeoffs between speed and accuracy (Krauzlis and Dill 2002).

For saccades, the mechanisms responsible for *canceling* eye movements have been studied with the “countermanding” paradigm (Hanes and Carpenter 1999; Hanes and Schall 1995). On the majority of trials in this task, subjects rapidly make a saccade to an eccentric target stimulus that appears just as the fixated stimulus is extinguished. However, on a minority of the trials, a stop signal (e.g., reappearance of the fixation spot) is given after the appearance of the target, indicating that the subject should maintain fixation and that the saccade to the target should be canceled. If the delay in giving the stop signal is too long, subjects fail to cancel the eye movement to the

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peripheral stimulus. By comparing this critical delay to the normal latency of saccades, it is possible to estimate the processing interval required to cancel the saccadic eye movement—this interval is referred to as the “stop-signal reaction time.” For visual stop signals, the stop-signal reaction time for saccades has values of ~ 80 ms in monkeys (Hanes and Schall 1995) and 100–150 ms in humans (Asrress and Carpenter 2001; Cabel et al. 2000; Hanes and Carpenter 1999).

Performance in the countermanding task has been modeled as a race between two processes—a go process triggered by the target stimulus and a stop process triggered by the stop signal (Logan et al. 1984; Osman et al. 1986). The behavioral outcome is then determined by whether the go process reaches its threshold before the stop process (response is initiated) or vice versa (response is cancelled). This race model can account for the findings in a variety of tasks requiring inhibition of responses (Logan and Cowan 1984). In a version of the race model applied specifically to saccades (Hanes and Carpenter 1999), the activation levels of the two processes are assumed to increase linearly over time but at a rate that varies randomly from trial to trial. The stochastic nature of these rates of increase can account for the randomness in the ability to cancel saccades after stop signals are given at different delays and can also reproduce the distribution of latencies observed when subjects fail to cancel saccades (Asrress and Carpenter 2001; Colonus et al. 2001; Hanes and Carpenter 1999).

To examine the mechanisms responsible for canceling pursuit, we have now used the countermanding paradigm with pursuit stimuli in both human and monkey subjects. To compare the findings from pursuit with those from saccades, subjects performed a saccade version of the countermanding task on separate trials. Using now-standard techniques for calculating the stop-signal reaction time, as well as a new method based on maximum likelihood estimation, we found that the race model could account for our results. Based on the outcome of these experiments and analyses, we argue that pursuit and saccades are probably regulated by a common inhibitory mechanism and that differences in the timing of cancelled pursuit and saccades are most likely due to differences in their motor output pathways.

METHODS

Subjects

We recorded eye movements from two adult rhesus monkeys (*Macaca mulatta*), 5 yr old, and four human subjects (*Homo sapiens*), 16–37 yr old. All experimental protocols for the monkeys were approved by the Institute Animal Care and Use Committee and complied with Public Health Service Policy on the humane care and use of laboratory animals. The monkeys were under the care of the Institute veterinarian. All experimental procedures for use with human subjects were reviewed and approved by the Institutional Review Board and each subject gave informed consent. Because the 16-year-old was underage, her parents also provided signed informed consent. Except for the methods used to record eye movements, the experimental design was nearly identical for monkey and human subjects.

Eye-movement recording

For the monkey subjects, we recorded eye movements using the scleral search coil technique. Under isoflurane anesthesia and aseptic conditions, we attached a head-holder using dental acrylic and tita-

nium screws. The head-hold allowed us to fix the head in the standard stereotaxic position during experiments. During the same surgery, we also implanted a search coil around each eye (Judge et al. 1980). The coils were used to monitor eye position with the electromagnetic induction technique (Fuchs and Robinson 1966). The AC voltages induced in the search coils were routed to a phase detector circuit that provided separate DC voltage outputs proportional to horizontal and vertical eye position (Riverbend Instruments). The outputs were low-pass filtered (6-pole Bessel, -3 dB at 180 Hz) and then sampled at 1 kHz (A/D converter: ComputerBoards). The coil output voltages were calibrated with respect to eye position by having the animal fixate small spot stimuli at known eccentricities. During the experimental sessions, monkeys sat in a standard primate chair and viewed stimuli presented on a video monitor (Eizo FX-E7, 120 Hz refresh rate) located 40 cm in front of the animal.

For the human subjects, we recorded eye movements using an infrared video-based eye-tracker system (Iscan, RK-726). The eye tracker reported the horizontal and vertical positions of the pupil with 12-bit resolution using a proprietary algorithm that computes the centroid of the pupil at 240 Hz. We calibrated the output from the eye tracker by recording the raw digital values as subjects fixated a set of known locations three times in a pseudorandom sequence. In the current experiments, we focused our analysis on the horizontal component of eye movements because the stimuli were located exclusively along the horizontal meridian. We used the mean values during 500-ms fixation intervals at each location to generate a smooth function (using cubic spline interpolation) for converting raw tracker values to horizontal eye position. The measurement noise caused by the eye tracker was $\sim 0.05^\circ$ (the SDs of the measurements from these 500-ms intervals), and the accuracy of the eye tracker measurements was $\sim 0.10^\circ$ (the SD of the mean values over these 500-ms intervals obtained with repeated fixations). During the experimental sessions, subjects used a bite bar to minimize measurement errors due to head movements and viewed stimuli presented on a video monitor (Eizo FX-E7) at a viewing distance of 41 cm.

Behavioral paradigms

At the beginning of each experimental trial, subjects fixated a small spot stimulus (0.3° for humans, 0.2° for monkeys) that appeared at the center of the display (Fig. 1). At the end of the fixation period, which had a randomized duration of 500–1,000 ms, the central spot was extinguished and a second small spot stimulus appeared at an eccentricity of $3\text{--}3.5^\circ$ and slightly above (0.3°) the horizontal meridian. Both spots had luminances of ~ 80 cd/m² and were presented on a uniform gray background (luminance, ~ 30 cd/m²) in a dimly illuminated room (ambient luminance, <0.1 cd/m²). On pursuit trials, this second spot stimulus moved horizontally toward and through the center of the display at a constant speed ($15^\circ/\text{s}$ for monkeys and $10^\circ/\text{s}$ for humans); the small vertical offset was used so that the moving spot would not occlude the central spot as it passed by the center of the screen (Fig. 1C). On saccade trials, the eccentric stimulus remained at its starting location. For the monkey subjects, pursuit and saccade trials were randomly interleaved. For the human subjects, pursuit and saccade trials were presented in randomly interleaved blocks, to reduce the occurrence of corrective saccades during pursuit. On the majority of trials (“go trials,” 75%), the second stimulus was presented by itself for 1,200 ms, after which the trial ended. However, on a minority of trials (“stop trials,” 25%), the central spot reappeared after a delay (the stop-signal delay) and was displayed together with the second stimulus for the remainder of the trial (Fig. 1, B and D). We chose a fraction of 25% stop trials, and stop-signal delays ranging from 0 to 300 ms (in 25-ms increments), to match the values typically used in previous studies using the countermanding paradigm (Asrress and Carpenter 2001; Cabel et al. 2000; Hanes and Carpenter 1999; Hanes and Schall 1995). For each of the human subjects, data were collected in 8 experimental sessions, each session lasting ~ 1 h. For

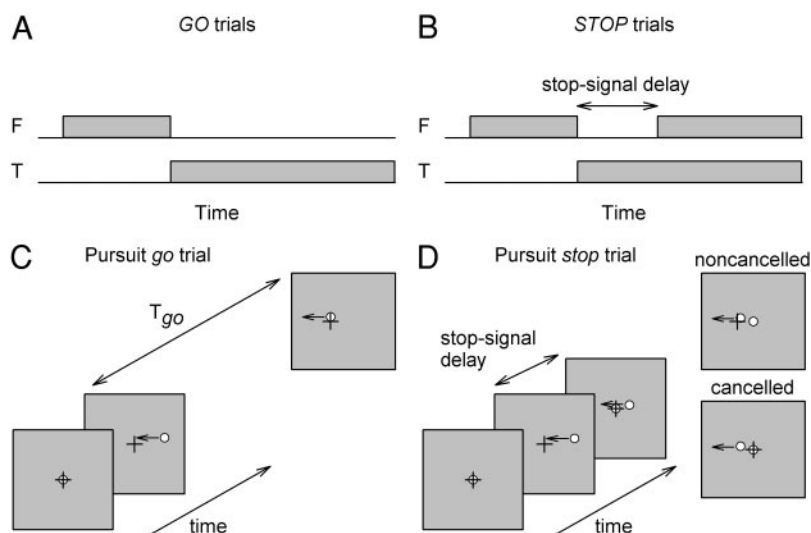


FIG. 1. Schematic diagrams of the experimental conditions. *A*: on go trials, subjects initially fixated a central target (F) that was extinguished at the same time that an eccentric target (T) appeared. *B*: on stop trials, the central target reappeared after a variable interval, the “stop-signal delay,” indicating that the subject should cancel the eye movement to the target. *C*: on pursuit go trials, the target stimulus moved at a constant speed toward and past the center of the screen, and the starting location was offset to eliminate the need for catch-up saccades. The small cross indicates the subject’s eye position. *D*: on pursuit stop trials, the central target reappeared at a variable delay after the appearance of the moving stimulus. On “non-cancelled” stop trials, subjects failed to cancel the pursuit movement and followed the moving stimulus. On “cancelled” stop trials, subjects successfully canceled pursuit and maintained fixation.

the monkey subjects, data were collected in 14 (*monkey A*) and 12 (*monkey W*) sessions, each session lasting ~ 3 h.

Monkey subjects had been previously trained to make saccades to visual stimuli in exchange for a juice reward. During the fixation period, they were required to remain within 1.7° of the central target, otherwise the fixation spot was extinguished and the paradigm reverted to the fixation period after a 2,500-ms time-out. After the fixation period, the behavioral rules depended on whether or not the fixation spot reappeared. On go trials, the monkey was provided a 450-ms grace period after the appearance of the eccentric stimulus and was required to be within 3° of the stimulus for the last 750 ms of the trial. The length of the grace period was determined empirically based on the latency distribution of typical $3\text{--}3.5^\circ$ saccades—a shorter grace period would have penalized the monkey inappropriately on some fraction of go trials. On stop trials, the monkey was given an 800-ms grace period starting at fixation point offset, but he was required to be within 1.7° of the fixation spot during the last 100–300 ms of the trial. This longer grace period was used so that correct performance was contingent only on whether the monkey followed the behavioral rules by the end of the trial and not on whether or not the monkey made a saccade to the eccentric stimulus. This reduced the impetus for waiting that might otherwise have affected our data. The monkey was given a liquid reward at the end of each correctly performed trial. Human subjects were verbally instructed to track the eccentric target when it appeared, except for trials on which the fixation spot reappeared. On stop trials with shorter stop-signal delays, both monkey and human subjects were able to cancel or withhold tracking eye movements to the eccentric stimulus (“cancelled trials”). However, on stop trials with longer stop-signal delays, subjects tended to initially track the eccentric target before making a centering saccade back to the fixation spot (“noncancelled trials”).

Data collection and analysis

The presentation of stimuli, and the acquisition, display, and storage of data were controlled by a personal computer using the Tempo software package (Reflective Computing). A second personal computer, equipped with a high-speed graphics card (Cambridge Research Systems VSG2/3) and VisionWorks software (Swift et al. 1997) acted as a server device for presenting the visual stimuli and received instructions from the Tempo computer via its serial and parallel ports. All eye-movement data, and events related to the onset of stimuli, were stored on disk during the experiment, and later transferred to a FreeBSD Linux-based system for subsequent off-line analysis. An interactive analysis program was used to filter, display, and make measurements from the data. Signals encoding horizontal eye velocity

were obtained by applying a finite impulse response (FIR) filter (-3 dB at 54 Hz for monkeys) to the calibrated horizontal eye-position signals. Signals encoding eye acceleration were then obtained by applying the same FIR filter to the signals encoding velocity.

We detected the occurrence of saccades by applying a set of amplitude criteria to the eye-velocity and eye-acceleration signals as described previously (Krauzlis and Miles 1996b). This algorithm permitted us to detect saccades with amplitudes as small as $\sim 0.15^\circ$ measured with the eye coil and $\sim 0.3^\circ$ measured with the eye tracker. For the saccade stop condition, we classified trials as noncancelled if we detected a saccade $>0.9^\circ$ for monkeys and 0.5° for humans within 1,000 ms of the appearance of the eccentric stimulus; otherwise, we classified trials as cancelled. The amplitude criteria of 0.9 and 0.5° were determined by comparing the amplitudes of targeting saccades that occurred on saccade go trials to the amplitudes of corrective saccades that occurred during fixation—we selected the criterion that maximized the number of saccades correctly identified as targeting saccades and that also minimized the number of corrective saccades erroneously identified as targeting saccades. For saccade go and noncancelled trials, we measured saccadic latency with respect to the appearance of the eccentric stimulus and also the saccadic amplitude and peak velocity.

The onset of pursuit was estimated using a variant of an algorithm described previously (Krauzlis and Miles 1996b). In this previous technique, the variance associated with a “baseline” interval was used to detect the beginning of a “response” interval. A linear regression of the response interval as a function of time was used to determine when the response intersected the baseline—this point in time was defined as the latency of pursuit. The extrapolation used in this method makes the latency estimates sensitive to noise in the response interval, and this problem is worse with the somewhat higher noise associated with video-based methods of eye-movement recording. We therefore constrained the response interval to immediately follow, and be continuous with, the baseline interval, forming a “hinge.” The baseline interval had a duration of 100 ms and the response interval had a duration of 75 ms, and we tested possible hinge placements ranging from ± 40 ms from an initial subjective estimate of pursuit latency. For each of these hinge placements, the slope of the response interval was determined by linear regression, and we measured the mean squared error between the data and the model (baseline plus response intervals). The hinge placement that provided the best fit was defined as the latency of pursuit.

For the pursuit stop condition, we classified trials as noncancelled if we detected a saccade back to the fixation spot after the appearance of the eccentric stimulus; otherwise, we classified trials as cancelled. The logic of this method is based on the observation that when

subjects failed to cancel their eye movement on pursuit stop trials, they always made a centering saccade back to the fixation spot. Thus rather than measuring the occurrence of pursuit directly, we detected the occurrence of pursuit indirectly by measuring the centering saccades required after even a short-lived pursuit eye movement. We classified pursuit stop trials as noncancelled if we detected a centering saccade $>0.3^\circ$ for monkeys and $0.35\text{--}0.4^\circ$ for humans within 1,000 ms of the appearance of the eccentric stimulus; otherwise, we classified trials as cancelled. We evaluated other possible methods, such as testing for significant changes in eye velocity at different intervals after the appearance of the moving stimulus. However, because the timing and duration of the pursuit response was variable, these approaches did not provide robust results, especially for the shortest stop signal delays for which the pursuit responses were often quite short-lived. For pursuit go and noncancelled trials, we measured pursuit latency with respect to the appearance of the eccentric stimulus. We also measured the eye displacement and average eye velocity at different time points after stimulus appearance to compare the dynamics of pursuit in different experimental conditions.

RESULTS

Canceling pursuit and saccades

The traces in Fig. 2 show examples of pursuit eye-velocity records from *monkey W*. On pursuit go trials (Fig. 2A), eye velocity increased from zero (---) after the offset of the fixation spot and matched target speed ($15^\circ/\text{s}$) within a few hundred milliseconds. For this trial, we estimated the onset of pursuit as occurring at 121 ms after the appearance of the target (\downarrow). On some pursuit stop trials, eye velocity did not deviate from zero (Fig. 2B), indicating that the subject successfully maintained fixation. This trial, on which the fixation spot reappeared after 50 ms (the stop-signal delay), was therefore classified as a pursuit cancelled trial. On other pursuit stop trials, eye velocity did deviate from zero (Fig. 2C), and we detected this by the occurrence of the corrective saccade made back to fixation (\rightarrow), which in this case reached a peak velocity of $46^\circ/\text{s}$ and had an amplitude of 0.42° . This trial, which also had a stop-signal delay of 50 ms, was therefore classified as a pursuit noncancelled trial, and we estimated the onset of pursuit as occurring at 124 ms (\downarrow).

Across all of our subjects, delaying the reappearance of the fixation spot on stop trials increased the probability of generating pursuit and saccadic eye movements. For the shortest stop-signal delays, subjects almost always cancelled the eye movement (cancelled trials), whereas for longest stop-signal delays, subjects almost always failed to cancel the eye movement (noncancelled trials). This relationship is summarized for each of the subjects by the graphs in Fig. 3, which plot the probability of a pursuit (○) or saccadic (□) eye movement as a function of the stop-signal delay. Superimposed on the data are logistic functions fit to the data for pursuit (—) and saccades (---). These functions have been previously referred to as “inhibition functions” (Logan and Cowan 1984) or “response functions” (Osman et al. 1986); the parameters for these fitted functions are summarized in Table 1. For three subjects (*R*, *S*, and *W*), the inhibition functions for pursuit lie to the left of those for saccades, whereas the reverse is true for one subject (*G*), and the remaining two subjects showed little difference (*H* and *A*). In contrast to the variable pattern found for the inhibition functions, the latency of eye movements on go trials was consistently longer for saccades than for pursuit. As shown in

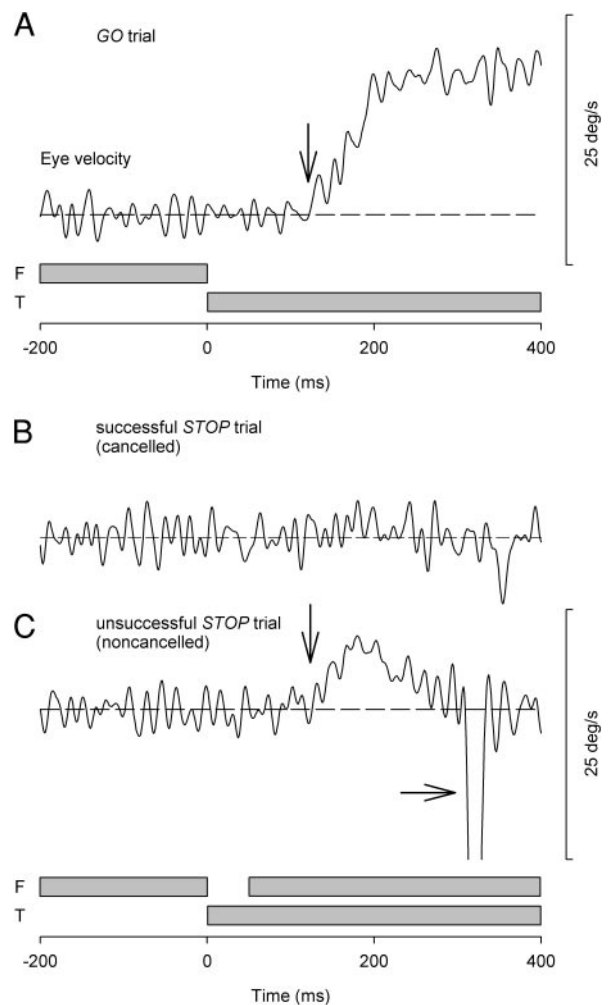


FIG. 2. Sample eye velocity traces on pursuit trials. A: on this pursuit go trial, the subject followed the stimulus moving at $15^\circ/\text{s}$ to the right. The estimated pursuit latency on this trial was 121 ms, indicated by the arrow. The horizontal dashed line indicates $0^\circ/\text{s}$. The trace is aligned on the onset of the target stimulus (T), which coincided with the offset of the central fixated stimulus (F). B: on this pursuit stop trial, the subject successfully canceled pursuit and maintained fixation as indicated by eye velocity remaining near $0^\circ/\text{s}$. C: on this pursuit stop trial, the subject failed to cancel pursuit and briefly followed the moving stimulus. We detected the occurrence of pursuit by the corrective saccade made by the subject back to the central fixation stimulus (\rightarrow). The estimated latency on this trial was 124 ms (\downarrow). The stop-signal delay for the trials depicted in B and C was 50 ms, as indicated by the reappearance of the central stimulus (F) shortly after the appearance of the target (T). Data are from *monkey W*.

Fig. 4, the cumulative latency distributions for saccades (---) all lie to the right of those for pursuit (—). As summarized for each of the six subjects in Table 1, the mean latencies of saccades were 20–120 ms longer than the latencies of pursuit; these differences were highly significant for each subject (2-tailed *t*-test, $P < 0.001$).

Estimating the stop-signal reaction time for pursuit and saccades

Based on the inhibition functions and latency distributions for pursuit and saccades, we estimated the timing of the inhibitory processes that cancelled the incipient pursuit and saccadic eye movement commands. These estimates were based on the

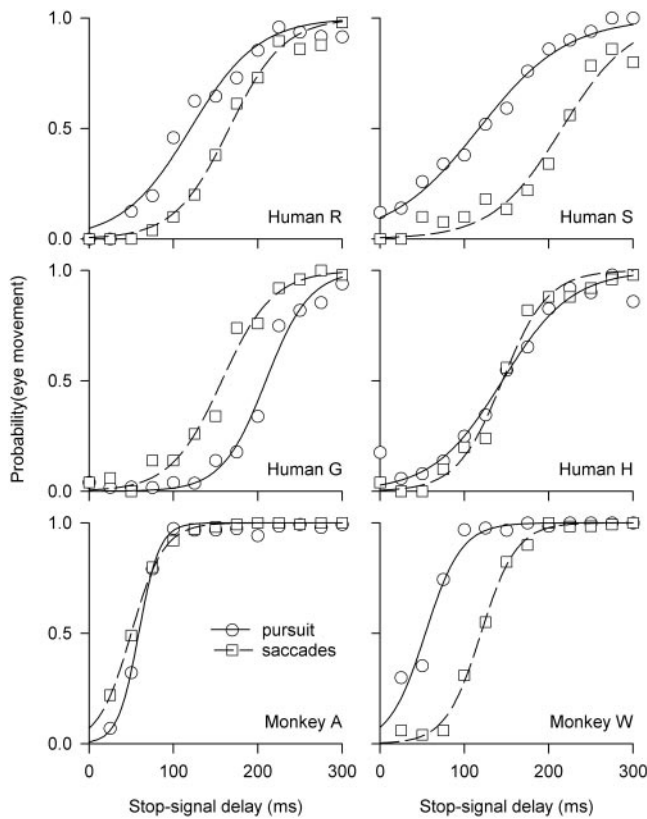


FIG. 3. Inhibition functions for pursuit and saccades. The observed probability of an eye movement response on stop trials is plotted as a function of stop-signal delay for each of the 6 subjects. \circ , pursuit data; \square , saccade data. The smooth curves are logistic functions fit to the pursuit (—) and saccade (---) data, described by the equation: $y = 1 / (1 + e^{-(x-a)/b})$. The r^2 value associated with each fitted curve was >0.97 .

assumption that response preparation involves a race between a go process (also referred to as the controlled process) and a stop process (also referred to as the inhibition process) (Logan and Cowan 1984). The go process is initiated by the appearance of the eccentric stimulus and can potentially trigger a

TABLE 1. Summary of inhibition functions and latencies for pursuit and saccade

Inhibition Functions	Pursuit			Saccades		
	a	b	n	a	b	n
Subject R (human)	120	40	346	166	33	279
Subject S (human)	117	52	307	216	41	206
Subject G (human)	210	26	206	157	30	310
Subject H (human)	145	41	277	145	27	329
Subject A (monkey)	59	12	1394	50	19	1547
Subject W (monkey)	54	21	1231	120	22	982
Latencies	Mean	n	Mean	n		
Subject R (human)	168 ± 31	1355	280 ± 60	1873		
Subject S (human)	196 ± 33	1429	312 ± 57	1936		
Subject G (human)	250 ± 31	903	271 ± 53	1847		
Subject H (human)	195 ± 32	1310	254 ± 55	1942		
Subject A (monkey)	115 ± 15	5666	136 ± 25	5942		
Subject W (monkey)	127 ± 16	4851	197 ± 30	5334		

For the inhibition functions, the table lists the fitted parameters for the logistic equation (a and b) and the sample size (n) for each subject. For the latencies, the table lists the means ± SD and sample size (n) for each subject.

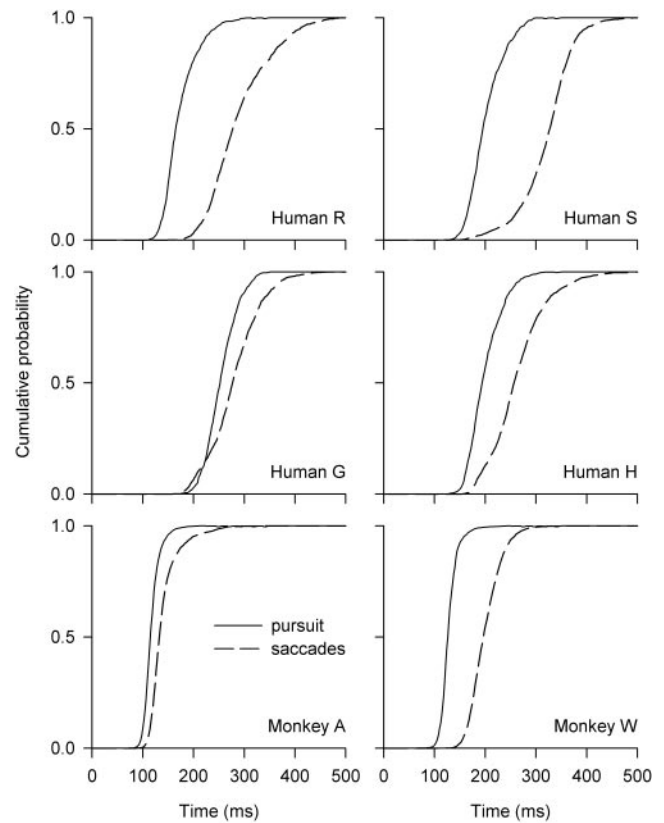


FIG. 4. Cumulative latency distributions for pursuit and saccades. The observed cumulative probability of an eye movement response on go trials is plotted as a function of time for pursuit (—) and saccades (---) for each of the 6 subjects.

response after a variable amount of time, which is designated by the random variable T_{go} . The stop process is initiated by the reappearance of the fixation spot and requires an amount of time, the stop-signal reaction time, to cancel any incipient movement. Thus the probability of an eye-movement response on stop trials (the value of the inhibition function) is given by the equation: inhibition function (stop-signal delay) = $P((T_{go} - \text{stop-signal reaction time}) < \text{stop-signal delay})$.

This relationship is shown graphically in Fig. 5. In the absence of the stop process, the go process is initiated by the onset of the target and completed after a variable amount of time (T_{go}), producing a distribution of latencies for the completed movements (Fig. 5A). If a stop-signal is provided (at a particular stop-signal delay), the stop process finishes after the stop-signal reaction time, and the eye movements that might have occurred after this point in time are cancelled. Conversely, the eye movements triggered before this point in time are not cancelled, and these can be measured at a range of stop-signal delays to compute the inhibition function (Fig. 5B). If the stop-signal reaction time was a constant, the inhibition function would take the form of a cumulative probability that was simply a time-shifted version of the cumulative latency distribution. Thus even though the stop-signal reaction time cannot be directly measured, its value can be estimated by comparing the inhibition function to the distribution of latencies.

We estimated the stop-signal reaction time using three different methods described previously (Hanes and Carpenter 1999; Hanes and Schall 1995). First, we used a method based

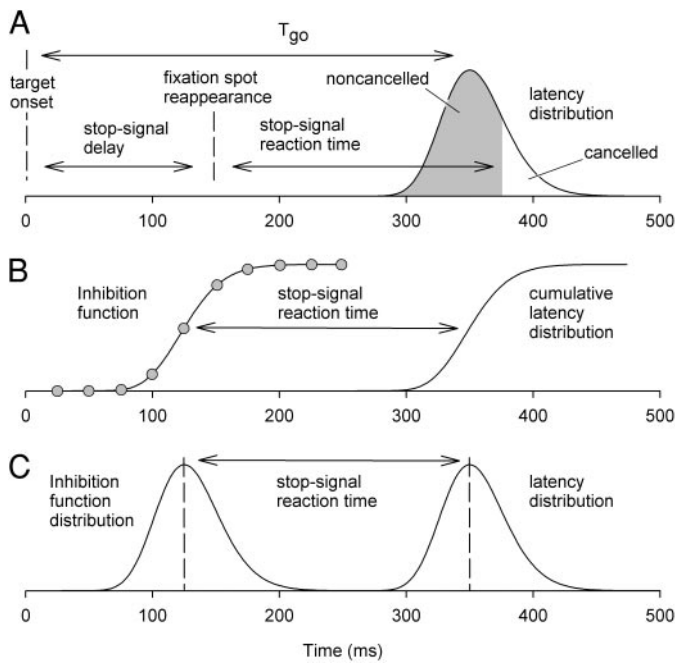


FIG. 5. Estimating the stop-signal reaction time for pursuit and saccades. *A*: schematic diagram showing the temporal relationship between the go and stop processes. The go process elapses after a variable amount of time, T_{go} , resulting in the observed distribution of latencies on go trials. The stop process begins after the experimentally controlled stop-signal delay, and elapses after an interval defined as the stop-signal reaction time. In this schematic diagram, the stop-signal reaction time is assumed to have a fixed value. On stop trials, responses with latencies shorter than the sum of the stop-signal delay and the stop-signal reaction time are noncancelled, whereas those associated with longer latencies are cancelled. *B*: the probability of observing a response on stop trials, described by the “inhibition function,” increases as a function of stop-signal delay because a larger fraction of responses are noncancelled as the stop-signal delay is increased. The temporal distance between the inhibition function and the cumulative latency distribution provides an estimate of the stop-signal reaction time. *C*: the temporal distance between the mean or median of the inhibition function and the mean of the observed latency distribution provides another estimate of the stop-signal reaction time.

on integrating the latency distributions. For each measured value on the inhibition function, we determined the point on the cumulative latency function (Fig. 5*B*) with the same value. The difference in time between this point on the cumulative latency function and the stop-signal delay provides an estimate of the stop-signal reaction time. This method provided a stop-signal reaction time for each stop-signal delay, based on the temporal separation between the inhibition functions and the cumulative latency functions. However, because this method is affected by irregularities in the tails of the inhibition functions, we calculated the average stop-signal reaction time after omitting data from the tails of the functions. We did this by including only those stop-signal delays for which the probability of making an eye movement was between 10 and 90%, as was done previously by Hanes and Schall (1995).

The other two methods for calculating the stop-signal reaction time treat the inhibition function as though it was a cumulative distribution (Hanes and Schall 1995; Logan and Cowan 1984), analogous to the distribution of eye movement latencies (Fig. 4). We converted the inhibition functions from each subject to probability distributions and determined the mean response time from each of the distributions. For cases in which the inhibition function did not start at 0 or terminate at

1.0, it was necessary to rescale the function by the difference between its maximum and minimum values (Colonius et al. 2001; Hanes and Carpenter 1999). Because the inhibition functions we measured were symmetrical, we also measured the median of the inhibition function (Hanes and Schall 1995; Logan and Cowan 1984), which was simply the time value (stop-signal delay) at which the function equaled 0.5. The difference between the mean eye movement latency and the mean or median response time provided additional estimates of the stop-signal reaction time (Fig. 5*C*).

The results from these three methods for estimating the stop-signal reaction time are reported in Table 2. For pursuit, the average stop-signal reaction times were 61 ms for the two monkeys and 54 ms for humans. For saccades, the average stop-signal reaction times were 80 ms for monkeys and 112 ms for humans. Thus on average using these methods, the stop-signal reaction times for pursuit were shorter than those for saccades by 19 ms for the two monkeys and 58 ms for the four humans.

Estimating the stop-signal reaction time using maximum likelihood techniques

The preceding analyses provide estimates of the average stop-signal reaction time but do not provide estimates of its variance. If the stop process also has a variable rate, as assumed in the complete version of the race model (Logan and Cowan 1984) as well as in diffusion models (Ratcliff et al. 1999), neglecting this variance may lead to a biased estimate of the stop-signal reaction time. In general, noncancelled trials will occur when the go rate is faster than the stop rate. However, if the stop process has a variable rate, some fraction of the go rates can be faster than some fraction of the stop rates, even if the mean stop rate is appreciably faster than the mean go rate (e.g., Fig. 6*A*). Using conventional techniques for estimating the stop-signal reaction time (Fig. 5), we would attribute the occurrence of these noncancelled trials to a stop-signal reaction

TABLE 2. Summary of stop-signal reaction times estimated using three standard methods described previously

	Integration Method	Median Method	Mean Method	Average of 3 Methods
Pursuit SSRTs				
Subject R (human)	48	47	42	46
Subject S (human)	86	79	68	77
Subject G (human)	42	41	44	42
Subject H (human)	49	50	49	49
Subject A (monkey)	55	57	54	55
Subject W (monkey)	68	73	59	67
Average across subjects				
Humans	56	54	51	54 ± 16
Monkeys	61	65	56	61 ± 8
Saccade SSRTs				
Subject R (human)	121	109	108	113
Subject S (human)	107	114	115	112
Subject G (human)	121	96	111	110
Subject H (human)	116	114	114	115
Subject A (monkey)	85	86	74	82
Subject W (monkey)	81	77	76	78
Average across subjects				
Humans	116	108	112	112 ± 2
Monkeys	83	82	75	80 ± 3

Values are means ± SD. SSRTs, stop-signal reaction time.

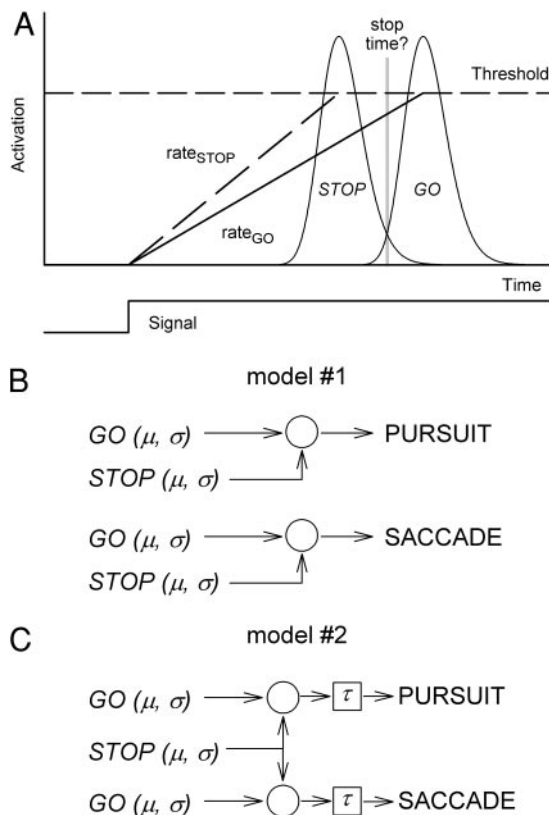


FIG. 6. Estimating the stop-signal reaction time assuming that the stop and go processes both have variable rates. *A*: schematic diagram showing the temporal relationship of the stop and go processes. Variability in the rates for the 2 processes (rate_{STOP}, rate_{GO}) results in 2 temporal probability distributions (STOP, GO). Gray vertical line labeled “stop time?” indicates approximate estimate of stop-signal reaction time if the stop process is assumed to have a constant rate. *B*: extension of the race model assuming that pursuit and saccadic systems have independent stop processes as well as independent go processes. The Gaussian distribution of rates for each process is described by its mean (μ) and SD (σ). *C*: version of the race model assuming that pursuit and saccades share a single stop process, but that the final motor pathways contain an interval (τ) that is not under inhibitory control.

time (vertical line labeled “stop time?” in Fig. 6*A*) that was later than the actual mean stop time (indicated by distribution labeled “STOP” in Fig. 6*A*). Thus variance in the stop rate might be misinterpreted as a lower stop rate, leading to an overestimate of the stop-signal reaction time.

We developed an analysis technique for estimating the variable rates for the go and stop processes, assuming that the rates for both processes are drawn from a Gaussian distribution with mean μ and SD σ . Previous studies have used Monte Carlo simulations to estimate these parameters (Colonius et al. 2001; Hanes and Carpenter 1999). However, because the variable rates in the race model give rise to probability distributions of movement latencies and stop times, it is also possible to estimate their values somewhat more directly using maximum likelihood techniques. Estimating the rates for the go process was straightforward. The reciprocal of the observed latencies (i.e., the rates) on go trials forms a Gaussian distribution as described previously (Carpenter and Williams 1995). For example, the skewed latency distribution labeled GO in Fig. 7*A* corresponds to the Gaussian rate distribution, also labeled GO, in Fig. 7*B*. We directly obtained the maximum likelihood estimates for the Gaussian description (μ, σ) of this rate

distribution using the “mle” function in the Matlab Statistics Toolbox (The Mathworks).

Estimating the rates for the stop process was a more complicated procedure and involved determining which stop rate distribution provided the best match to the observed proportion of noncancelled trials. In brief, we used a nonlinear minimization technique (Nelder and Mead 1965) to determine the most likely parameters for the stop rate distribution, given the previously determined go rate distribution. To illustrate the steps of this procedure, Fig. 7*B* shows a candidate stop rate distribution (labeled STOP) that corresponds to the skewed stop latency distribution in Fig. 7*A*. To calculate the frequency of noncancelled trials predicted by this candidate stop distribution, we converted the go and stop rate distributions in Fig. 7*B* to cumulative distributions, inverting the sign of the stop distribution to indicate its inhibitory effect on eye movements (Fig. 7*C*). The point of intersection between the two cumulative distributions (indicated in Fig. 7*C*, left arrow) indicates the expected proportion of noncancelled trials, given these particular go and stop rate distributions. By testing a variety of candidate stop rate distributions, it is possible to determine the parameters for the stop rate distribution that minimize the difference between the expected and observed proportion of noncancelled trials.

Unfortunately, this procedure applies only to the case in which no stop-signal delay is imposed. To extend this approach to the more general case in which the stop process starts after the go process, it is necessary to take into account the effects of time delays on the shape of the stop rate distribution. To do this, we converted the stop rate distribution (Fig. 7*B*) to a latency distribution (Fig. 7*A*), added the particular stop-signal delay, and then re-converted the latency distribution back to a rate distribution. As might be expected, the effect of this transformation is to shift the stop distribution toward progressively lower rates as the stop-signal delay is increased (Fig. 7*D*, dashed line). Thus delaying the onset of the stop process is similar to having a slower stop process with no delay. However, the imposed delays do not simply translate the distribution uniformly toward lower rates because delaying the onset of the process has a disproportionately larger effect on higher rates.

The points of intersection in Fig. 7*D* provide the predicted frequency of noncancelled trials for the full set of stop-signal delays, given this candidate stop rate distribution. We compared these predicted frequencies to the observed frequency of noncancelled trials described by the inhibition functions (Fig. 3). For each stop-signal delay, we computed the likelihood of observing the particular number of noncancelled trials, given the frequency predicted by the intersection of the go and stop rate distributions, based on the binomial probability distribution. We defined a cost function by taking the negative log of each likelihood value and summing these values across stop-signal delays; this step converted the set of small likelihood values into a single positive number that was smaller for candidate stop rate distributions that provided better matches to the data. Using the Nelder-Mead simplex method (i.e., the “fminsearch” function in Matlab), we then determined the parameters of the stop rate distribution (μ, σ) that minimized this cost function. By minimizing the negative log of likelihood, this method therefore provided estimates of the stop rate

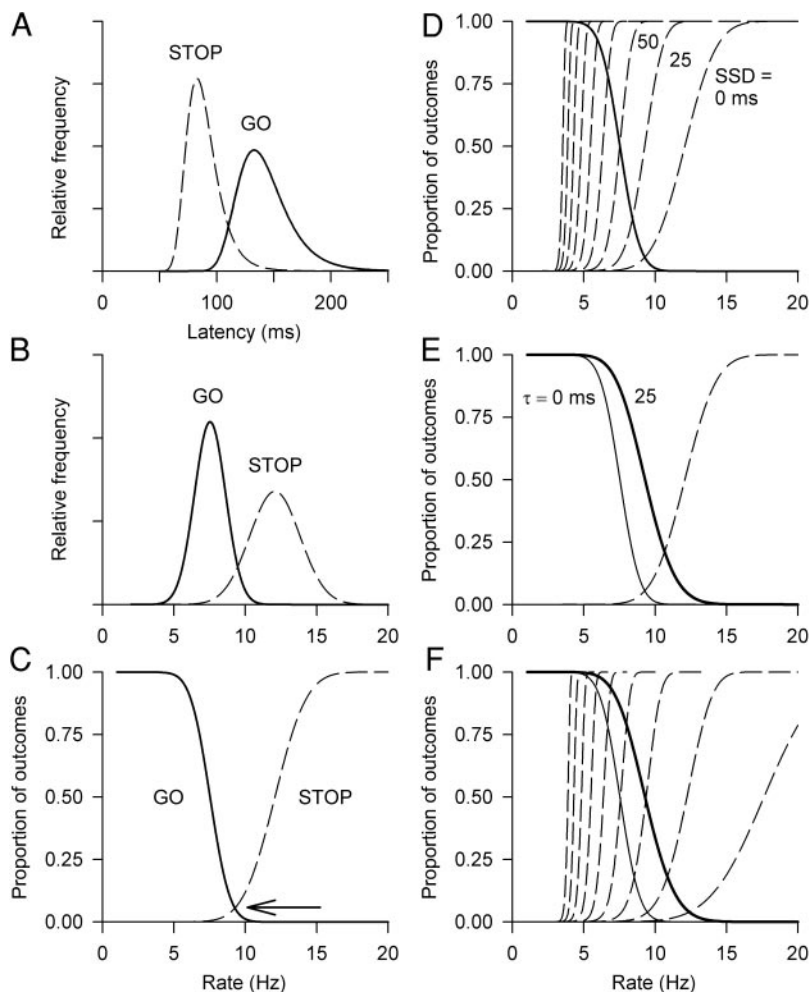


FIG. 7. Graphical representation of our new method for estimating the stop-signal reaction time. *A*: the slightly skewed GO distribution (solid line) was obtained from the measured latencies of pursuit or saccades. The similarly skewed STOP distribution (dashed line) was posited to test how well it might explain the frequency of noncancelled trials. *B*: the GO and STOP distributions plotted as rate (inverse of latency) form nonskewed Gaussian distributions. *C*: the GO and STOP rate distributions plotted as cumulative probability functions. The point of intersection between the 2 functions indicates the predicted frequency of noncancelled trials, indicated by the horizontal arrow. *D*: family of STOP rate distributions (dashed lines) obtained by adding stop-signal delays from 0 to 200 ms, in 25-ms increments. GO distribution (solid line) is same as in *C*. *E*: introducing a terminal ballistic interval ($\tau = 25$ ms) shifts the effective GO distribution toward higher rates (solid line) compared with the original distribution ($\tau = 0$ ms, solid line). STOP distribution (dashed line) is same as in *C*. *F*: a 2nd family of STOP rate distributions (dashed lines), obtained by adding stop-signal delays as in *D*, but starting with an initial STOP distribution shifted toward higher rates. Note that the points of intersection between the STOP distributions and the shifted GO distribution (thick solid line) in this panel predict approximately the same frequency of noncancelled trials for each stop-signal delay as predicted in *D*.

distribution that maximized the likelihood of observing the experimentally obtained inhibition functions.

We first simulated the race model using a simple extension to include both pursuit and saccade data. In this version of the model (Fig. 6*B*, *model 1*), we simply assumed that the pursuit and saccadic systems each had their own *go* process (with parameters μ and σ) and their own stop process (also with parameters μ and σ), resulting in an eight-parameter model. For each subject, we then used the technique described in the preceding text to obtain maximum likelihood estimates of the parameters describing the go and stop rate distributions. As shown in Fig. 8, the frequency of noncancelled trials for pursuit (○) and saccades (□) predicted by this version of the race model (*model 1*) provided a good match to the observed inhibition functions (— and - - -); the predicted and observed values were not significantly different for any of the six subjects (Kolmogorov-Smirnov test, $P > 0.05$). The parameters for these fits are provided in Table 3 (GO trials, and STOP trials: *model 1*). It should be noted that the rates of the go and stop processes listed in Table 3 are lower than those found in previous Monte Carlo simulations of the race model (Colonius et al. 2001; Hanes and Carpenter 1999). This difference is due to the fact that, in contrast to these previous simulations, our model did not include an extra parameter accounting for delays in visual processing but instead incorporated all delays into the two rate parameters.

We then simulated a second version of the race model (Fig. 6*C*, *model 2*) that also contained eight parameters. In this second model, we assumed that the pursuit and saccadic systems again had their own go process (with parameters μ and σ) but were inhibited by a common stop process (also with parameters μ and σ). In addition, we assumed that the pathways for both pursuit and saccades contained a terminal interval (with duration τ ms) that was not subject to inhibitory control, which has been previously referred to as a ballistic interval (Osman et al. 1986). The distributions plotted in Fig. 7*E* show how the introduction of a ballistic interval changes the probability distribution associated with the go process—it shifts it toward higher rates. This would be expected because, from the viewpoint of the stop process, reducing the portion of the total reaction time that is under inhibitory control is equivalent to reducing the overall latency. Introduction of a ballistic interval trades off with changes in the speed of the stop process. For example, Fig. 7*F* depicts a go process with a 25-ms ballistic interval and a stop process that is speedier (has a higher rate distribution) than that depicted in Fig. 7*D*. However, the predicted frequency of noncancelled trials shows the same relationship to stop-signal delay as the go and stop processes depicted in Fig. 7*D*. Thus increasing the rate of the stop process can offset the effect of increasing the ballistic interval.

Using the same techniques described in the preceding text, we obtained maximum likelihood estimates of the parameters

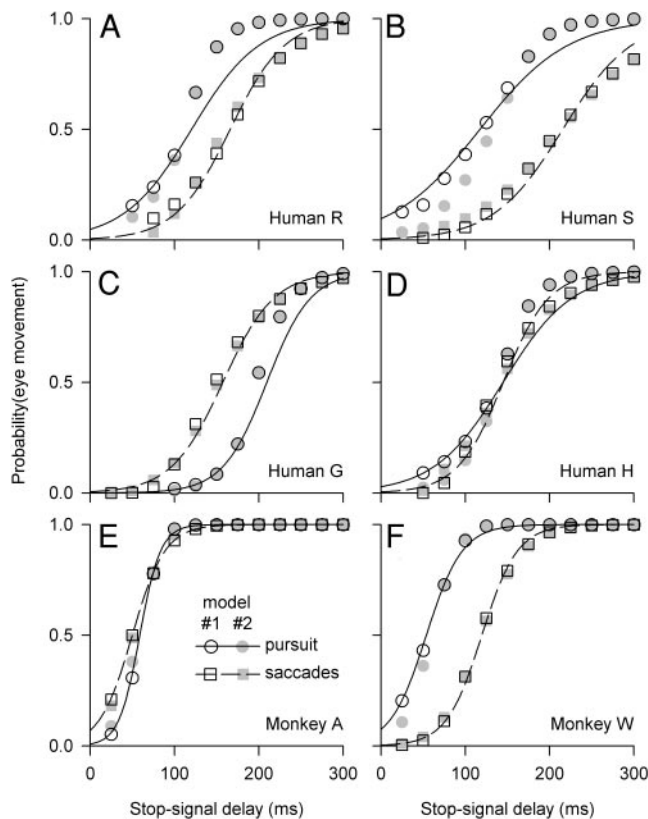


FIG. 8. Comparison of simulated and observed inhibition functions for pursuit and saccades. The observed probability of an eye movement response on stop trials is plotted as a function of stop-signal delay for pursuit (\circ , \ominus) and saccades (\square , \boxminus). \circ and \square , the results from *model 1* (Fig. 6B); \ominus and \boxminus , the results from *model 2* (Fig. 6C). Smooth functions (— and - - -) are duplicated from Fig. 3.

for *model 2*. As shown in Fig. 8, the frequency of noncancelled trials for pursuit (\ominus) and saccades (\boxminus) predicted by this version of the race model (*model 2*) also provided a good match to the observed inhibition functions (— and - - -). The predicted and observed values were not significantly different for any of the six subjects (Kolmogorov-Smirnov test, $P > 0.05$). The parameters for these fits are also provided in Table 3 (GO trials and STOP trials: *model 2*). For all six subjects, the maximum likelihood estimate of the ballistic interval for pursuit was 0 ms, meaning that this model ended up possessing one less parameter than *model 1*. We evaluated the two models by applying a log-likelihood ratio test, based on the likelihoods associated with the two models' predictions about noncancelled trials. For each pair of predictions, we subtracted the log likelihood of *model 2*'s prediction from the log likelihood of *model 1*'s prediction, multiplied this difference by 2 so that the result would conform to a χ^2 distribution, and then applied a standard χ^2 statistic on the resulting value, assuming 1 df (*model 1* had one additional parameter). We found no significant differences between the two models for any of their predictions about noncancelled trials (Fig. 8, 140 pair wise comparisons, $P > 0.05$).

These simulations of the race model provide additional estimates of the stop-signal reaction times for pursuit and saccades that are summarized in Table 4. The results from *model 1* involve assumptions that are similar to those used in conventional estimates of the stop-signal reaction time (in

particular, movements can be inhibited up until their reaction time). Accordingly, the estimates from *model 1* are similar to those we obtained using traditional methods. For pursuit, the average stop-signal reaction times were 61 ± 11 ms for the two monkeys and 51 ± 15 ms for humans. For saccades, the average stop-signal reaction times were 79 ± 6 ms for monkeys and 104 ± 11 ms for humans. The lower estimate of the saccade stop-signal reaction time for humans is likely the result of treating the stop time as a random variable for reasons described in the preceding text. The results from *model 2* provide an alternative interpretation based on the assumption of a common inhibitory process. For pursuit and saccades, the average stop-signal reaction times were 61.0 ± 6 ms for monkeys and 48.3 ± 8 ms for humans; the average ballistic intervals were 0 ms for pursuit for all subjects, 48 ± 14 ms for saccades in humans and 17 ± 11 ms for saccades in monkeys. Thus depending on one's assumptions, the race model provides at least two distinct interpretations for our observations about canceling pursuit and saccades.

Tests of the race model

The race model makes several predictions that we tested against our data. The race model assumes that the responses on noncancelled trials come from the same distribution as *go* trials, except that the stop process eliminates the upper tail (Fig. 5A). Consequently, the mean latency on noncancelled trials should be shorter than the mean latency on *go* trials, at least for shorter stop-signal delays. This prediction was confirmed by our data. As shown in Fig. 9, the latency of pursuit on noncancelled stop trials (\bullet , \circ) was almost always lower than the latency on *go* trials (- - -), and this difference was usually significant for the shorter stop-signal delays (\blacksquare , Kruskal-Wallis, $P < 0.05$). Similarly, the latency of saccades

TABLE 3. Maximum likelihood estimates of the parameters for two versions of the race model applied to our pursuit and saccade data

Go Trials	μ_{GO_PURS}	σ_{GO_PURS}	μ_{GO_SACC}	σ_{GO_SACC}
Subject R (human)	6.15	1.01	3.73	0.73
Subject S (human)	5.24	0.84	3.34	0.76
Subject G (human)	4.06	0.51	3.83	0.74
Subject H (human)	5.27	0.81	4.11	0.84
Subject A (monkey)	8.79	1.03	7.53	1.09
Subject W (monkey)	8.02	0.93	5.20	0.75
Stop Trials: <i>model 1</i>	μ_{STOP_PURS}	σ_{STOP_PURS}	μ_{STOP_SACC}	σ_{STOP_SACC}
Subject R (human)	21.04	9.71	9.16	0.56
Subject S (human)	14.14	6.97	11.27	1.88
Subject G (human)	28.61	8.81	8.85	0.08
Subject H (human)	20.02	8.05	9.47	0.01
Subject A (monkey)	18.88	2.90	12.08	1.76
Subject W (monkey)	14.51	3.94	13.36	1.71
Stop Trials: <i>model 2</i>	μ_{STOP}	σ_{STOP}	τ_{BAL_SACC}	τ_{BAL_PURS}
Subject R (human)	21.32	7.57	52.3	0.0
Subject S (human)	16.97	5.64	27.6	0.0
Subject G (human)	24.52	5.92	59.8	0.0
Subject H (human)	21.69	5.71	50.9	0.0
Subject A (monkey)	17.62	2.98	25.2	0.0
Subject W (monkey)	15.30	2.75	8.5	0.0

Rate parameters (μ , σ) have units of hertz; ballistic interval parameters (τ) have units of milliseconds.

TABLE 4. Estimates of the go and stop intervals based on our maximum likelihood estimates of the race model parameters

	Go Process	Stop Process		Ballistic Interval (model 2)
		Model 1	Model 2	
Pursuit				
Subject R (human)	163	48	47	0
Subject S (human)	191	71	59	0
Subject G (human)	246	35	41	0
Subject H (human)	190	50	46	0
Subject A (monkey)	114	53	57	0
Subject W (monkey)	125	69	65	0
Average across subjects				
Humans	197.5 ± 34.8	51.0 ± 14.9	48.3 ± 7.6	0
Monkeys	119.5 ± 7.9	61.0 ± 11.3	61.0 ± 5.7	0
Saccades				
Subject R (human)	268	109		52
Subject S (human)	299	89		28
Subject G (human)	261	113		60
Subject H (human)	243	106		51
Subject A (monkey)	133	83		25
Subject W (monkey)	192	75		9
Average across subjects				
Humans	267.8 ± 23.3	104.3 ± 10.6		47.8 ± 13.8
Monkeys	162.5 ± 41.7	79.0 ± 5.7		17.0 ± 11.3

Values are means ± SD. All values have units of milliseconds. Duration of the go and stop processes were obtained by taking the reciprocal of the mean rates (μ) reported in Table 3. One set of stop values is reported for *model 2* (3rd column) because this model included only one stop process that was applied to both pursuit and saccades.

on noncancelled stop trials (□, ■) was also typically lower than that on go trials (···). Second, as the stop-signal delay is increased, the stop process eliminates less of the upper tail of the latency distribution, resulting in longer latencies. As a result, the latency on noncancelled trials should be longer for longer stop signal delays. This prediction was also generally borne out by our data as indicated by the mostly monotonic increases in pursuit and saccade latency that occurred as the stop-signal delay was increased. Third, based on the observed latencies of pursuit and saccades on *go* trials, we calculated the latencies predicted by the race model for each stop-signal delay. We calculated the predicted latencies by taking the average of the go trial latencies that lie to the left of the stop-signal finish line (defined as the sum of the stop-signal delay and the average of the 3 traditional estimates of the stop-signal reaction time, shown in Table 2). The predicted latencies for pursuit (—) and saccades (- - -) were generally similar to the observed latencies on noncancelled trials except for some cases of shorter stop-signal delays for which there were fewer trials.

The race model assumes that the stop and go processes are independent, implying that the outcome produced by a completed go process should not be altered by the presence or absence of a concurrent stop process. One test of this prediction is to compare the metrics of the movements generated on go trials and noncancelled trials (Hanes and Schall 1995). For saccades, we documented the metrics by measuring the amplitudes and peak velocities of the saccades from the two types of trials for each subject, for a total of 12 measurements (6 subjects × 2 trial types). For pursuit, we similarly made measurements of amplitude and velocity, but because of the continuous nature of pursuit, we made measurements at several fixed points in time for each subject (from 125 to 425 ms for humans, from 75 to 375 ms for monkeys, both in 25-ms increments), for a total of 156 measurements (6 subjects × 2

trial types × 13 time points). These measurements are compared in Fig. 10, which plots the values obtained from noncancelled trials against the values obtained from go trials. The data lie mostly along the line of unity slope (- - -), indicating that the amplitudes (Fig. 10, *A* and *B*) and velocities (Fig. 10, *C* and *D*) were very similar during the two types of trials. For the amplitude measurements, there were no significant differences for any of the subjects (○); for the velocity measurements, there was a significant difference for seven of the measurements (●), all of which came from *monkey A*. Overall, these data indicate that the presence or absence of a stop signal generally had little effect on the metrics of eye movements, although there was a clear effect on pursuit for one subject.

Effects of trial sequence

We found evidence of an interaction between go and stop trials in another aspect of our data—the latencies of pursuit and saccades were affected by the sequence of trials during the experiment. The graphs in Fig. 11 compare the latencies from *go* trials for pursuit (Fig. 11, *A* and *C*) and saccades (Fig. 11, *B* and *D*) when the previous trial was either a stop trial or another go trial. The data are shown separately for humans (*top*) and monkeys (*bottom*) and are further divided according to whether the previous trial was either a pursuit or a saccade trial. Latencies tended to be longer on trials after stop signals (labeled stop) than on trials after go signals (labeled go), as indicated by the higher values for the group means (■). We tested the significance of this effect by submitting the pursuit and saccade latencies to a three-way ANOVA (factors: subject, type of preceding trial—stop or go, and type of preceding eye movement—pursuit or saccade), followed by a Tukey test. For both pursuit and saccades, the increases in latency after stop trials were significant ($P = 0.002$ for pursuit, $P = 0.007$ for saccades). The average increase in latency after stop trials was

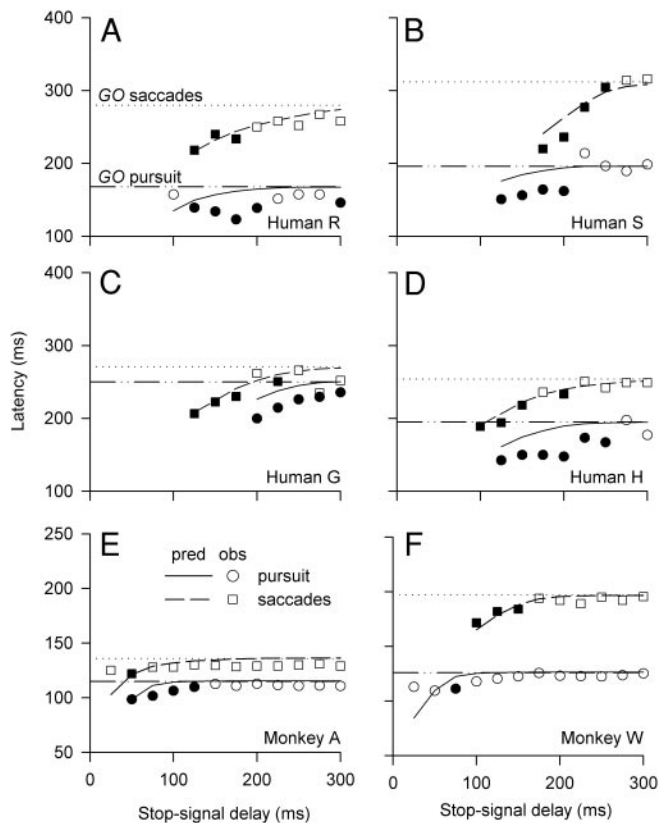


FIG. 9. Comparison of actual and predicted latencies on go trials and noncancelled stop trials. Each panel shows data and predicted latencies for one subject. \cdots , the average latency on saccade go trials; $-\cdots-$, the average latency on pursuit go trials. The actual median latencies observed on noncancelled stop trials for each stop-signal delay for pursuit (\circ and \bullet) and saccades (\square and \blacksquare) were lower than the latencies on go trials. \bullet and \blacksquare , data points for which this difference reached statistical significance (Kruskal-Wallis, $P < 0.05$). Each point represents the average latency from ≥ 10 trials; conditions with fewer observations were excluded from analysis. The actual latencies were also similar to those predicted latencies at each stop-signal delay for pursuit ($-$) and saccades ($-\cdots-$).

27 ± 6 ms (95% CI) for pursuit and 46 ± 6 ms (95% CI) for saccades. However, these increases in latency after stop trials did not require that the previous trial involve the same type of eye movement—for both pursuit and saccade latencies, the type of preceding eye movement had no effect ($P = 0.860$ for pursuit, $P = 0.926$ for saccades). We also tested the significance of these effects on a subject-by-subject basis by performing one-way ANOVAs on the individual pursuit and saccade latencies. These tests indicated a significant increase in latency (ANOVA, $P < 0.05$) after stop trials for most, but not all of the subjects (indicated by \bullet). For example, pursuit stop trials caused significant increases in the latency of saccades on subsequent go trials for three of the four human subjects (Fig. 11B, pursuit, \bullet), and for both monkeys (Fig. 11D, pursuit, \bullet). This finding again indicates that the effect of trial sequence tended to transfer between the two types of eye movements.

DISCUSSION

Canceling pursuit and saccadic eye movements

We have used the countermanding paradigm to investigate the mechanisms responsible for inhibiting the generation of

pursuit eye movements. By comparing the probability of canceling pursuit on stop trials to the latencies obtained on go trials, we have estimated the processing time required to countermand the incipient eye movement, referred to as the stop-signal reaction time (SSRT) (Logan and Cowan 1984). Using techniques described previously for studies of countermanding saccades (Hanes and Carpenter 1999; Hanes and Schall 1995), we estimate that it takes 50–60 ms to cancel pursuit eye movements (Table 2), and we found slightly shorter values for humans (54 ± 16 ms) than for monkeys (61 ± 8 ms). Using a new method using maximum likelihood estimation, we found similar estimates for the SSRT (Table 4) and again found slightly shorter times for humans (51.0 ± 14.9 or 48.3 ± 7.6 ms, depending on assumptions) than for monkeys (61.0 ± 11.3 or 61.0 ± 5.7 ms). By comparison, the measured latency of pursuit on go trials in these same subjects was 175 ± 50 ms (range: 115–250 ms), and we found longer latencies for the four human subjects (202 ± 35) than for the two monkey subjects (121 ± 8). Thus the estimated times required to cancel pursuit were shorter and less variable than the times required to initiate pursuit.

To compare the mechanisms for canceling pursuit and saccades, we also measured the SSRT for saccades in the same subjects. When we assumed that saccades are under inhibitory control up until their onset, using conventional techniques we

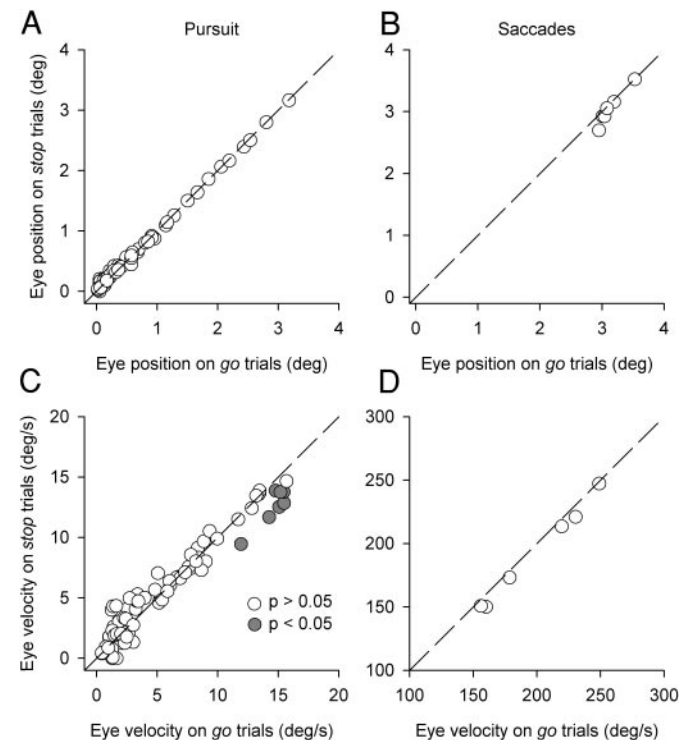


FIG. 10. Comparison of the metrics of pursuit and saccades on go vs. stop trials. A: the average eye positions at fixed time points after the onset of the eccentric target on pursuit stop trials are plotted against the same measurements obtained from pursuit go trials. The graph plots 78 pairs of measurements (6 subjects \times 13 time points). B: the average amplitude of saccades on stop trials is plotted against the average amplitude of saccades on go trials for each of the 6 subjects. C: the average eye velocities at fixed time points after the onset of the target on pursuit stop trials are plotted against the same measurements obtained from pursuit go trials (78 pairs of measurements). D: the average peak velocity of saccades on stop trials is plotted against the average peak velocity of saccades on go trials, for each of the 6 subjects. \bullet , significant differences (Kruskal-Wallis, $P < 0.05$).

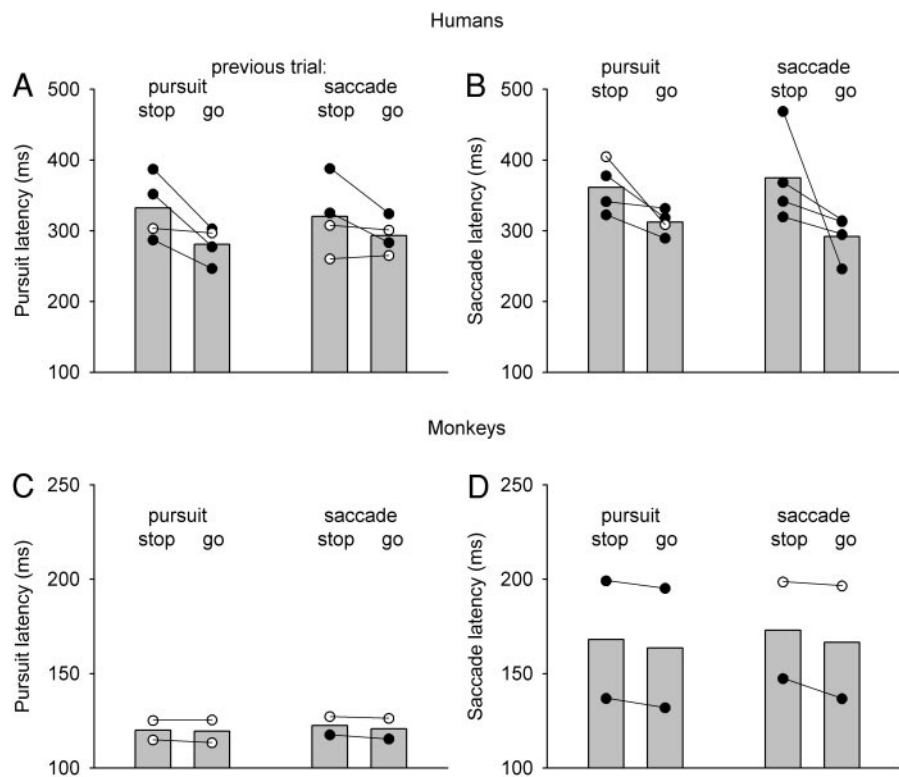


FIG. 11. Effect of trial sequence on the latencies of pursuit and saccades. *A* and *C*: latencies of pursuit on go trials are divided into 4 categories based on whether the previous trial was a pursuit stop trial, a pursuit go trial, a saccade stop trial, or a saccade go trial. Bars show group means, circles show data from individual subjects. *B* and *D*: latencies of saccades on go trials are similarly divided into four categories based on whether the previous trial was a pursuit stop trial, a pursuit go trial, a saccade stop trial, or a saccade go trial. Data from humans (*A* and *B*) and monkeys (*C* and *D*) are shown in separate plots. ●, significant differences (Tukey test, $P < 0.05$) between pairs of stop and go data.

found SSRTs that were longer than those for pursuit (Table 2) and somewhat longer in humans (112 ± 2 ms) than in monkeys (80 ± 3 ms). Using maximum likelihood estimation (Table 4, *model 1*), we similarly found values of 104.3 ± 10.6 ms for humans and 79.0 ± 5.7 ms for monkeys. These results are in good agreement with the previous estimates of 100–150 ms in human subjects (Asrress and Carpenter 2001; Cabel et al. 2000; Hanes and Carpenter 1999) and 70–90 ms in monkeys (Hanes and Schall 1995). However, when we assumed that saccades are under inhibitory control only up until a point of no return (Osman et al. 1986), we found that the canceling of saccades could be accounted for by the same rapid stop process that accounted for our pursuit results, providing estimates of the SSRT of 48.3 ± 7.6 ms in humans and 61.0 ± 5.7 ms in monkeys (Table 4, *model 2*). In this case, the sum of the SSRT and the ballistic interval were ~ 96 ms for humans and ~ 78 ms for monkeys, again in good agreement with previous estimates of the SSRT for saccades. Our results therefore suggest that a common inhibitory process could exert control over both pursuit and saccades and provide a reinterpretation of previous estimates of the SSRT for saccades.

Are our estimates of the SSRT too short?

As in previous studies, our estimates of the SSRT depended on the temporal separation between the distribution of noncancelled movements on stop trials (the inhibition function) and the distribution of normal latencies. The value obtained for the SSRT therefore depends on the sensitivity of the measurements of noncancelled movements—higher sensitivity shifts the inhibition functions toward earlier times and longer SSRT estimates, whereas lower sensitivity shifts the inhibition functions toward later times and shorter SSRT estimates. For saccades, there is little ambiguity in detecting noncancelled movements

because even small saccades involve velocities that are easily detected. The same is not true for noncancelled pursuit because subjects often only briefly pursued the moving stimulus before detecting their mistake and making a saccade back to the central fixation spot. In these cases, the noncancelled pursuit often did not achieve very high speeds. Therefore we detected noncancelled pursuit based on the centering saccades back to the fixation spot that occurred after even short-lived pursuit eye movements and used a very sensitive criterion for detecting these saccades (see METHODS). The short pursuit SSRT we found is therefore unlikely to be due to our method of detecting noncancelled pursuit because using some other, less sensitive method would have resulted in even shorter estimates.

Are our estimates of the SSRT physiologically plausible? Although short, our estimates of 50–60 ms lie within the range of behavioral reaction times for smooth eye movements. Ocular following responses, the smooth eye movements evoked by the motion of a large visual stimulus, have a latency of ~ 52 ms (Miles et al. 1986), and some fraction of this reaction time is presumably due to efferent motor delays that would not be relevant for canceling pursuit. Even faster visually guided responses were found in a study by Kimmig et al. (1992), who found that presenting a full-field light flash during maintained pursuit caused an abrupt slowing of pursuit eye velocity after only ~ 41 ms. To our knowledge, these effects involving the inhibition of pursuit are the fastest visually guided responses reported in primates, and demonstrate that eye movements can be inhibited in less time than our current estimates of the SSRT.

Do pursuit and saccades have separate inhibitory control or different ballistic intervals?

One of the advantages of the new method we describe for estimating the parameters for the go and stop processes is

that it shows explicitly how delaying the stop process (e.g., by imposing a stop-signal delay) shifts the stop distribution toward lower rates and how these shifts, in turn, can predict the frequency of noncancelled movements described by the inhibition function. Conversely, the method illustrates how the inclusion of a terminal ballistic interval (Osman et al. 1986) shifts the go distribution toward higher rates; the presence of the ballistic interval means that the go process accounts for only a fraction of the latency, and hence the go rates are higher than expected based on the total reaction time. Perhaps most significantly, the method also shows that these components of the model provide similar degrees of freedom, such that a particular inhibition function can result from different combinations of stop rates and ballistic intervals. As a result, we identified two very different mechanisms that can account for the differences in the canceling of pursuit and saccades. First, extending the standard race model to both types of eye movements, we tested an eight-parameter model with separate stop processes for pursuit and saccades and no ballistic interval (*model 1*, Fig. 6B). As shown in Fig. 8, this model provided a good match to the observed inhibition functions for pursuit and saccades, suggesting that pursuit and saccades might be subject to two independent inhibitory processes. Second, we tested another eight-parameter model that contained a single stop process for both pursuit and saccades but that included ballistic intervals in the pathways for pursuit and saccades (*model 2*, Fig. 6C). This model also matched the observed inhibition functions (Fig. 8). The maximum likelihood estimate of the ballistic interval for saccades was 28–60 ms for the human subjects and 9–25 ms for the monkey subjects. In contrast, the estimate of the ballistic interval for pursuit was 0 ms for every subject, effectively reducing the number of parameters associated with this model to seven. The success of this second, simpler model provides support for an alternative mechanism in which a common inhibitory process regulates pursuit and saccades, but the saccade pathways include an extra delay beyond a point of no return (Osman et al. 1986). Although the ballistic interval featured in this second model is unusual in that it has not been included in previous descriptions of canceled eye movements, it has been included in more general descriptions of inhibitory control (Osman et al. 1986), and we argue in the following text that it provides a more parsimonious explanation of our pursuit and saccade data.

At first blush, one might not be surprised to find it took less time to cancel pursuit than saccades because pursuit latencies are typically shorter than saccade latencies. Indeed, using methods that did not include a ballistic interval, our estimates of the SSRT for pursuit were shorter than that for saccades (Tables 2 and 4, *model 1*). However, we think that this analogy between latency and SSRT is specious for two reasons. First, previous studies of countermanding have shown that the go process responsible for starting movements is independent of the stop process responsible for canceling movements (Hanes and Carpenter 1999; Hanes and Schall 1995). Thus there is little reason to expect that the speed of the stop process (i.e., the SSRT) should be related to the speed of the go process (i.e., latency).

Second, canceling pursuit is not necessarily due to processing in the pursuit pathways but could result from re-activation

of the fixation system; consequently, the latency of pursuit could be irrelevant. The distinction between pursuit and fixation has been shown in several experiments demonstrating that visual inputs for eye movements have markedly different effects during pursuit than during fixation (Goldreich et al. 1992; Krauzlis and Miles 1996d; Morris and Lisberger 1987; Schwartz and Lisberger 1994). Similarly, stopping pursuit requires a transition from pursuit back to fixation (Krauzlis and Miles 1996d). Canceling pursuit is therefore different from initiating, or even stopping, pursuit because the successfully canceled pursuit movement involves maintaining fixation and by definition requires no activity from the pursuit system. In contrast to the time required to initiate pursuit and saccades, it might therefore be expected that the time required to cancel the two movements should be identical because canceling either type of eye movement involves maintaining a state of active fixation.

Unlike the problematic status of separate inhibitory processes for pursuit and saccades, there is evidence that the final pathways for saccades include a point of no return. Omnipause neurons in the reticular formation appear to act as the final inhibitory gate for saccades, regulating the triggering of saccades by determining when saccade burst neurons are allowed to fire (Scudder et al. 1988; Strassman et al. 1986a,b). Omnipause neurons exhibit tonic activity except immediately before saccades; this pause occurs 10–20 ms before the onset of saccades (Cohen and Henn 1972; Everling et al. 1998; Evinger et al. 1982; Keller 1974; Luschei and Fuchs 1972). Concomitant with this pause, burst activity from saccade-related neurons provides the motor command to the extraocular muscle motor neurons. The burst by neurons in the superior colliculus precedes the onset of saccades by ~20 ms (Munoz and Wurtz 1995; Sparks 1978); the burst in the paramedian pontine reticular formation precedes saccades by 5–15 ms (Scudder et al. 1988; Strassman et al. 1986a,b). Although it is possible to artificially stop saccades with electrical microstimulation in the region of the omnipause neurons (Keller and Edelman 1994; Keller et al. 1996), under natural conditions it does not appear possible to halt a saccade once the omnipause neurons have paused firing and the saccade-related burst begins. Thus the pathways for saccades appear to include a point of no return placed 5–20 ms before saccade onset, in good agreement with our estimates of the saccadic ballistic interval for our two monkey subjects (9 and 25 ms).

In contrast to saccades, pursuit appears to be controlled by neurons in the cerebellum and brain stem that provide a continuous and smoothly modulated eye motor command (Keller 1974; Krauzlis and Lisberger 1994; Lisberger and Fuchs 1978; Lisberger et al. 1994; Stone and Lisberger 1990; Suzuki and Keller 1988). Although it is unclear whether inhibitory influences on pursuit can be introduced as late as the ocular motor neurons, we know of no basis for identifying a point of no return in the pathways for pursuit. In fact, it has recently been shown that some omnipause neurons decrease their activity during pursuit as well as during saccades (Missal and Keller 2002). However, in contrast to the complete cessation of activity associated with saccades, these pursuit-related decreases are smaller and appear to be graded with smooth eye velocity. These observations provide direct support for the idea of a common inhibitory mechanism for pursuit and saccades and also

suggest that the motor pathways for pursuit may not include a point of no return, unlike the motor pathways for saccades. Accordingly, from our modeling we estimated the duration of the pursuit ballistic interval to be 0 ms for all of our subjects. We therefore conclude that the known properties of pursuit and saccades provide a physiological basis for assuming that the saccade pathways contain a terminal ballistic interval of 5–20 ms, whereas the pursuit pathways contain either a very short ballistic interval (<5 ms) or none at all.

Our estimates of the saccadic ballistic interval for our two monkey subjects (9 and 25 ms) are in good agreement with these physiological data about the final motor pathways for saccades. However, our estimates of the ballistic interval from the human subjects tended to be longer (28–60 ms). Does this discrepancy indicate a species difference in the motor pathways for saccades? Although we know of no evidence to contradict this interpretation, we view this as unlikely because the final motor pathways for eye movements are probably similar across primate species. Instead, we think this discrepancy may have resulted from a methodological difference in the trial structure for monkeys and humans. For the monkey subjects, pursuit and saccade trials were randomly interleaved, whereas for the human subjects, pursuit and saccade trials were presented in randomly interleaved blocks so as to reduce the occurrence of intrusive saccades during pursuit. We suspect that this blocking of trials may have allowed the human subjects to adopt different psychomotor sets during the two blocks, resulting in a slight difference in stop rates for the two types of trials that we then attributed to the duration of the saccadic ballistic interval.

In conclusion, although we cannot rule out the possibility that there are two separate inhibitory processes for pursuit and saccades, both driven by the same stop signal but demonstrating different temporal properties, this view seems less parsimonious. Based on the available data, we therefore favor the view that the canceling of pursuit and saccades involves a single inhibitory process associated with the fixation system and that the saccade pathways contain a longer ballistic interval than the pursuit pathways. Finally, although we have used the term “ballistic” to describe the portion of the motor pathways beyond the point of no return, we recognize that the trajectory of saccades is actively controlled. Results from experiments using double-step stimuli (Becker and Jürgens 1979; Zee et al. 1976) support the idea that the dynamics of saccades can be changed at any stage of processing (Leigh and Zee 1999). Thus our use of the term ballistic refers specifically to the process of determining whether or not to initiate an eye movement, and not to the on-line control that governs the metrics of eye movements.

Implications of the ballistic interval for saccades

If our reasoning about the ballistic interval for saccades is correct, then previous reports have likely overestimated the time interval associated with canceling saccades. Thus the previous estimates of 100–150 ms in human subjects (Asrress and Carpenter 2001; Cabel et al. 2000; Hanes and Carpenter 1999) and 70–90 ms in monkeys (Hanes and Schall 1995), based on assuming that the entire saccadic reaction time was under inhibitory control, should be short-

ened by 5–20 ms to take into account the portion of the reaction time that lies beyond the point of no return. This reinterpretation suggests that the minimum time required to cancel a saccade is actually closer to 50–70 ms in monkeys, similar to the time interval estimated from our current results. This line of reasoning also has implications for the interpretation of physiological data (e.g., Hanes and Pare 1998; Hanes et al. 1998). If the interval under inhibitory control was 5–20 ms shorter than previously estimated, then saccade-related neurons would need to exert their effect, not coincident with the conventional estimate of the stop-signal reaction time, but ≥ 5 ms earlier, to be eligible to cancel the incipient saccade. Conversely, neurons that exhibit a difference 5–20 ms before the conventional SSRT might actually lie at, or close to, the point of no return for saccades. For example, burst and buildup neurons in the superior colliculus may fall into this category (Hanes and Pare 1998).

Common inhibitory control of pursuit and saccades

The idea of common inhibitory control of pursuit and saccades found additional support in the interactions we found across trials. In particular, the latencies on go trials tended to be longer if the previous trial was a stop trial than if it were another go trial (Fig. 11). Similar effects of trial-type history have been observed in previous studies of saccades using the countermanding paradigm (Cabel et al. 2000; Schall and Taylor 1988). Because our study involved both pursuit and saccade, we were able to establish that these effects occur regardless of whether the preceding trial was associated with the same or different type of eye movement. Based on the transfer of these effects of trial sequence across the two eye movements, we conclude that they are due to changes at a site or sites that are common to pursuit and saccades. It is known that expectations can influence the control of eye movements, producing pursuit and saccades that anticipate changes in the visual stimulus (Barnes and Asselman 1991; Bronstein and Kennard 1987; Findlay 1981; Kowler 1989; Kowler and Steinman 1979a,b). For pursuit, prior information about the likely direction of an upcoming motion stimulus can bias both perceptual judgments and pursuit, suggesting an effect at relatively early stages of visual processing (Krauzlis and Adler 2001). Similar effects of visual attention could alter performance during the countermanding task because the presence or absence of a stop signal on one trial could bias visual-motor processing on subsequent trials. Although the exact mechanisms are unknown, it will be interesting to test whether the race model can be extended to incorporate these types of effects. For example, rather than assuming that the stop process always starts with zero activity, it might start with elevated or depressed activity based on prior information about the likelihood of needing to maintain fixation, similar to the way that prior information has been hypothesized to interact with the go process for saccades (Carpenter and Williams 1995). If a common inhibitory mechanism exercised veto control over both pursuit and saccades, this would help ensure that the probabilities of initiating pursuit and saccades were based on the same interpretation of sensory-motor events.

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