

A “Gap effect” on Stop Signal Reaction Times in a human saccadic countermanding task

Scott A. Stevenson^{1,2}, James K. Elsley^{1,3} Brian D. Corneil¹⁻⁴

¹CIHR Group in Action and Perception, Graduate Program in Neuroscience²
Departments of Physiology & Pharmacology³ and Psychology⁴,
University of Western Ontario,
London, Ontario, Canada N6A 5C1

Running Head: A “Gap Effect” on SSRTs

Correspondence should be addressed to:

Brian D. Corneil, Centre for Brain and Mind,

Robarts Research Institute, London, Ontario, Canada N6A 5K8.

(519) 663-5777, ext 34132. FAX: (519) 663-3193. E-mail: bcorneil@uwo.ca

Words in abstract: 229

Number of pages: 37

Number of figures: 7

Number of Tables: 1

Acknowledgements: We thank Dr. S. Goonetilleke, and B. Chapman and M. Pace for comments on earlier versions of the manuscript. This work was supported by an operating grant from the Natural Sciences and Engineering Research Council (NSERC RGPIN-311680). SAS was supported by an Ontario Graduate Scholarship. JKE was supported by a NSERC PGS-M. BDC is a CIHR New Investigator.

ABSTRACT

The “gap effect” describes a phenomenon whereby saccadic reaction times are expedited by the removal of a visible fixation point prior to target presentation. Here, we investigated whether processes controlling saccade cancellation are also subjected to a gap effect. Human subjects performed a countermanding experiment which required them to try to cancel an impending saccade in the presence of an imperative visual stop signal, across different fixation conditions. We found that saccadic cancellation latencies, estimated via derivation of the stop signal reaction time (SSRT), were ~40 ms shorter on trials with a 200 ms gap between fixation point removal and target presentation compared to when the fixation point remained illuminated. Follow-up experiments confirmed that the reduction in SSRTs were primarily due to removal of a foveal fixation point (as opposed to a generalized warning effect), and persisted with an auditory stop signal that controlled for potential differences in stop signal saliency across different fixation conditions. Saccadic RTs exhibited a gap effect in all experiments, with reductions in RTs being due to both removal of a foveal fixation point and a generalized warning effect. Overall, our results demonstrate that processes controlling saccade cancellation can be expedited by a 200 ms gap. The simultaneous priming of both saccade cancellation and generation is of particular interest considering the mutually antagonistic relationship between the saccade fixation and generation networks in the oculomotor system.

INTRODUCTION

Foveal vision necessitates a selection process to help determine the relevance of potential saccade locations. Inherent to this selection process is the ability to withhold saccades to stimuli that become irrelevant or inappropriate in a changing behavioural context. The countermanding paradigm, which was initially applied to hand or limb movements (Logan and Cowan 1984; Logan 1994), permits study of such inhibitory control of movement. An oculomotor version of the countermanding task requires the subject try to cancel saccades to a peripheral target in the presence of an imperative stop signal (Hanes and Schall 1995). Performance in a countermanding trial has been conceptualized as a race between a GO process dictating saccade generation (initiated upon target presentation), and a STOP process dictating saccade suppression (initiated upon stop signal presentation). The performance on a given trial depends upon the outcome of this race, with a saccade being generated or withheld if the GO or STOP process wins the race, respectively (Fig. 1; (Logan and Cowan 1984; Logan 1994)). The countermanding paradigm has become increasingly popular for at least two reasons. First, analysis across multiple trials permits estimation of the duration of the STOP process through derivation of a metric called the stop-signal reaction time (SSRT), even though this process cannot be directly observed (Fig. 1B; (Logan and Cowan 1984; Logan 1994)). Second, it provides a formalized framework in which to interpret neural activity related to the immediate control of movement (Hanes and Schall 1996; Stuphorn et al. 2000; Paré and Hanes 2003; Curtis et al. 2005; Aron and Poldrack 2006; Brown et al. 2008; Emeric et al. 2008).

A number of manipulations influence the distribution of reaction times (i.e. the rate of completion of the GO process) in simple saccade tasks. The “gap effect” describes the generalized reduction in saccadic reaction times (RTs) observed when a central fixation point is removed prior to the presentation of a peripheral target. A maximal reduction in RTs occurs with a gap of ~200 ms (Saslow 1967;Fischer 1987;Dorris and Munoz 1995;Munoz et al. 2000). Multiple components contribute to the gap effect, including the benefit afforded by warning of impending target presentation (a “warning” component), and by disengaging fixation via removal of a foveal stimulus (a “foveal” component; (Ross and Ross 1980;Ross and Ross 1981;Juttner and Wolf 1992;Kingstone and Klein 1993;Reuter-Lorenz et al. 1995;Paré and Munoz 1996;Forbes and Klein 1996;Taylor et al. 1998;Fendrich et al. 1999;Pratt et al. 2000)).

Here, we investigate whether the introduction of a 200 ms gap prior to target presentation influences the SSRT (i.e., the rate of completion of the STOP process). Based on previous results in the literature, two outcomes seem possible. First, because of the assumed independence of the GO and STOP processes, one could predict that SSRTs will be unaffected by a 200 ms gap. In support of this, simultaneous target presentation and fixation point removal (i.e., a gap duration of 0 ms) expedites the duration of the GO process without influencing the duration of the STOP process (Morein-Zamir and Kingstone 2006). Second, a manipulation that decreases the duration of the GO process may increase the duration of the STOP process. This reasoning is based on the mutually antagonistic relationship between the saccade generation and saccade fixation networks throughout the oculomotor system (Munoz and Istvan 1998;Meredith and Ramoa 1998;Findlay and Walker 1999;Sparks 2002;Scudder et al. 2002) and on results which

suggest a moderate interaction between the GO and STOP processes (Ozyurt et al. 2003;Boucher et al. 2007a). In support of this alternative, recent results have reported that SSRTs for arm movements increase with the introduction of a 200 ms gap (Mirabella et al. 2008).

To study the influence of a 200 ms gap on saccade control, we had human subjects perform a variety of countermanding tasks. In the first experiment, the fixation point either remained illuminated during target presentation (an OVERLAP condition), or disappeared 200 ms prior to target presentation (a GAP condition). Contrary to both predictions laid out above, SSRTs were *shorter* in the GAP condition. A series of two follow-up experiments confirmed the consistency of this effect, and identified that this reduction was due more to the removal of a foveal visual stimulus rather than a generalized warning effect, and was present for both visual and auditory stop signals.

Portions of this manuscript have been published in abstract form (Stevenson et al. 2007).

METHODS

A total of 9 different subjects (Ages 22-35; 2 female) participated in at least one of three variants of the countermanding task after providing their informed written consent. Subjects reported no history of neurological or musculoskeletal disorders, and all had normal or corrected-to-normal vision. Experimental procedures were approved by the University Research Ethics Board for Health Science Research at the University of Western Ontario in accordance with the ethical standards established in the 1964 Declaration of Helsinki. Three subjects (*s1,s2* and *s3*) were the authors and hence were

knowledgeable about the specific goals of the experiment. The remaining subjects were naïve. Subjects were instructed beforehand on the nature of the countermanding task but were not given any feedback during the experiment. All subjects generated qualitatively similar trends in the data. Subjects were seated upright in a straight-back chair in a dark experimental room. The room was compartmentalized by a double layer of thick dark curtains that spanned from floor to ceiling, attenuating the residual illumination given off by the experimental equipment. The visual stimuli consisted of three tri-colour light-emitting diodes (LEDs; which could be illuminated red and/or green) embedded within boxes mounted on wooden stands positioned 1.2 m in front of the subject. All LEDs were elevated 1.2m off the ground to lie on the horizontal meridian from the subject's perspective. One LED was positioned directly in front of the subject to serve as the central fixation point (FP). Two target LEDs were fixed at a radial angle of 10° to the left or right of the FP. Two of the three variants of the countermanding task (see below) involved an auditory stimulus. The speaker was placed directly behind the subject, and consisted of a broadband burst (77 dB) of noise powered by a 5V TTL pulse.

Countermanding task

Subjects performed three variants of an oculomotor countermanding task. All three variants of the task required subjects to look to visual targets on *CONTROL* trials, and attempt to maintain fixation on *STOP* trials in the presence of a stop signal. *CONTROL* and *STOP* trials were intermixed, and within the *STOP* trials we varied the timing of presentation of the stop signal relative to the target (the stop-signal delay (SSD)). All aspects of the task were controlled by a customized LABVIEW program

downloaded onto a PXI box (National Instruments), which controlled the experiment at a rate of 1 kHz. Subjects performed a series of practice trials before the experimental data were collected. Subjects were instructed to look as quickly as possible to the presented target and to try not to move when the stop signal appeared.

Experiment 1

In the first experiment, we investigated the influence of a 200 ms gap on both the GO and STOP processes. To do this, we introduced two fixation conditions, GAP and OVERLAP, and investigated subject performance on both *CONTROL* and *STOP* trials. Within a block of 200 trials, all possible permutations of fixation condition (GAP vs. OVERLAP), trial type (*CONTROL* vs. *STOP*), and target direction (left vs. right) were randomly interleaved. Within the *STOP* trials, the possible SSDs were varied equally amongst six possible values, ranging between 0 to 250 ms in 50 ms steps. Six of the nine total subjects performed a series of 6 blocks of 200 trials each over two sequential days, with 30% of these trials being *STOP* trials.

All trial types started with the illumination of the central FP for an interval selected randomly between values of 1000, 1166, 1333, and 1500 ms. In the GAP condition, the FP was then extinguished for 200 ms, followed by target presentation to the left or right for 1000 ms. In the OVERLAP condition, the FP remained illuminated during target presentation (overall trial duration remained the same as in the GAP condition by adding an additional 200 ms to FP illumination; Fig. 2A). On *CONTROL* trials, subjects simply made a saccade to the target. On *STOP* trials in the GAP condition, the stop signal consisted of a re-illumination of the green central FP (recall that the

central FP is a tri-colour LED that can be either red and/or green; Fig. 2A). On *STOP* trials in the OVERLAP condition, the stop signal consisted of a colour change of the central FP from red to green (Fig 2A). The stop signal remained illuminated for the duration of the trial and trials were separated by an inter-trial interval of 500-1000 ms.

Experiment 2

In the second experiment, we introduced a third fixation condition to test the influence of a warning cue on subject performance. In this “AUDITORY” condition, an auditory stimulus accompanied FP presentation at the start of the trial, but was turned off 200 ms prior to target presentation (the FP remained illuminated for the entire trial; Fig. 2B). The OVERLAP and GAP conditions were run as in Experiment 1, and all three fixation conditions were interleaved with the different trial types and target locations. Within *STOP* trials, SSDs were varied over a range of 250 ms in 50 ms steps (6 SSDs total), with SSD ranging from either 0 to 250 ms or 100 to 350 ms for different subjects. Five of the nine subjects participated in this experiment, performing six blocks of 210 trials each. Within each block, 35% of trials were *STOP* trials.

Experiment 3

In the third experiment, we used an auditory cue as a stop signal to test the influence of a non-foveal stop signal. All aspects of this experiment were the same as in Experiment 1 (i.e., OVERLAP and GAP conditions); excepting the use of an auditory stop signal instead of a visual stop signal (Fig. 2C). Five of the nine subjects participated in this experiment, with the SSDs ranging between 0 to 200 ms in 40 ms steps for four

subjects, and from 0 to 250 in 50 ms steps for one subject. Within each block, 30% of the 200 trials were *STOP* trials, and subjects performed a total of six blocks over two sequential days.

Calculation of Stop Signal Reaction Times

The main goal of this experiment is to compare estimates of the duration of the *STOP* process (the stop-signal reaction time (SSRT)) across fixation conditions. Briefly, the SSRT is a derived parameter that estimates the amount of time required to cancel a planned movement. Here we used two measures of calculating the SSRT: the integration method and the mean method (Logan 1994; Hanes and Schall 1995). Calculating this parameter via the integration method requires both the inhibition function (see Fig. 3 for examples of inhibition functions) from *STOP* trials and the cumulative RT distribution functions (CDF) from *CONTROL* trials. The SSRT is estimated at each SSD by first finding the probability of making a saccade from the inhibition function for that SSD, then running the integral from zero to that probability in the *CONTROL* trial CDF. The SSRT is then estimated by subtracting the SSD from this value (Logan 1994). SSRTs were only calculated at SSDs where the probability of a saccade ranged between 0.1 and 0.9, to capture the linear portion of the inhibition function and cumulative RT distributions.

The mean method for estimating SSRTs assumes that the SSRT for a given subject will be the same regardless of the SSD. While this assumption seems unlikely to be true, violations of this assumption do not significantly affect the validity of the race model (Logan 1994). The mean method for calculating SSRTs simply takes the

difference between the mean saccadic reaction time and the mean of the inhibition function (Hanes and Schall 1995), using a rescaling factor as suggested by Logan (1994) since $P(\text{saccade})$ does not always range between 0 and 1.

Data collection and analysis

Bi-temporal DC electrooculography (EOG) was used to measure horizontal eye movements and signals were filtered and amplified with a P122 AC/DC preamplifier (Grass Instruments). Horizontal eye movements were filtered (100 Hz, low pass), amplified, and digitized at a rate of 1 kHz onto the PXI controller. Digitized data were then transferred to a PC computer and subsequent off-line analyses were performed using customized Matlab (the Mathworks) programs. Movement onsets and offsets were identified by an automarking program, which detected crossings of velocity thresholds ($50^\circ/\text{s}$; velocities were filtered with a low-pass Butterworth filter with $f_s/f_c < 17$). Eye movements were analyzed via a customized Matlab Graphical User Interface permitting the data analyst to check for errors and ensure consistency. Saccadic reaction times, inhibition functions and SSRTs were calculated offline and saved for further analyses. Trials where RTs were $< 80\text{ms}$ were classified as anticipatory and were excluded from analysis (Corneil and Munoz 1996). Trials with RTs $> 800\text{ms}$ were also excluded due to lack of subject alertness. Less than 1% of all trials were excluded with these two criteria. Reaction time and SSRT comparisons for Experiments 1 & 3 utilized paired t-tests while Experiment 2 (with three fixation conditions) utilized a one-way repeated measures ANOVA with a Bonferroni post-hoc correction test for multiple comparisons.

Differences in RTs and SSRTs across fixation conditions for individual subjects utilized two way t-tests.

RESULTS

Within each experiment, we will first present the results from *CONTROL* trials to confirm that subject performance was consistent with previous reports, and then describe subject performance on *STOP* trials.

Experiment 1

The six subjects participating in Experiment 1 generated a total of 4580 *CONTROL* trials, split equally across GAP and OVERLAP fixation conditions. We found a robust influence of fixation condition on the reaction times, with RTs being significantly shorter in the GAP condition across all subjects (Fig. 3A, Table 1; $P < 0.05$; paired t-test). The difference between RTs in the OVERLAP and GAP conditions (i.e. the gap effect) has been studied extensively (see (Munoz et al. 2000) for review) and in our subjects ranged from 6-105 ms (gap effect: 57 ± 32 (mean \pm s.d.) ms; the reduction in RTs was significant ($P < 0.05$, two-way t-test) in all six subjects).

Logically, it should become progressively more difficult to suppress a saccade for more delayed stop signals. Subject performance on *STOP* trials varied in this straightforward fashion depending on the SSD. For each subject, we constructed inhibition functions which describe the probability of generating a saccade as a function of SSD. Separate inhibition functions were constructed for data obtained from GAP (Fig.

3B) and OVERLAP conditions (Fig. 3C), and from these inhibition functions it is apparent that the probability of making saccades (i.e., “non-cancelled” trials) increased for progressively longer SSDs.

With the integration method, we observed a gap effect on SSRTs in five of our six subjects, in that the SSRTs for these five subjects were shorter in the GAP vs. OVERLAP condition. In the sixth subject, SSRTs were approximately equal across fixation conditions. Overall, the gap effect on SSRTs estimated by the integration method ranged from -3 to 68 ms (45 ± 27 ms; $P < 0.01$; paired t-test). Consistent with the integration method, we found a gap effect on SSRTs calculated via the mean method in all six subjects (38 ± 11 ms; range: 27 to 52 ms; $P < 0.01$; paired t-test).

The integration and mean methods yield equally valid estimates for SSRTs (Logan and Cowan 1984). Accordingly, we combined these estimates to yield an average SSRT for each subject for each fixation condition (Table 1; Fig 3D). Upon averaging, a consistent gap effect was observed in all subjects (42 ± 16 ms; range: 12 to 56 ms; paired t-test, $P < 0.05$). Together, these data suggest that completion of the STOP process can be expedited in the human countermanding task by a 200 ms gap.

Experiment 2

Previous studies have established that the reduction in RTs observed during the gap effect is due to both a warning component (as FP disappearance serves as a cue for impending target presentation) and a foveal component (as FP disappearance removes a visual stimulus from the fovea; see INTRODUCTION). Motivated by the surprising results from Experiment 1, Experiment 2 investigates whether the gap effect on SSRTs

can be similarly decomposed into a warning and foveal component. Accordingly, we added a third fixation condition, the AUDITORY condition, which provides a warning signal independent of a foveal component (Fig 2B). Any differences in RTs or SSRTs between OVERLAP and AUDITORY conditions would be due to the warning component since neither involves the loss of visual fixation and only the AUDITORY condition has a warning component. Conversely, any differences between AUDITORY and GAP conditions would be due to the foveal component since both conditions have warning components, while only the GAP condition involves the removal of a foveal stimulus.

A total of 5762 *CONTROL* trials, distributed equally amongst the OVERLAP, AUDITORY, and GAP conditions were analyzed in five subjects. As shown in Fig. 4A and Table 1, we again observed a significant gap effect across the sample (comparing RTs in OVERLAP and GAP condition; 81 ± 27 ms; range: 45 to 117 ms; significance assessed by ANOVAs on RTs across all 3 fixation conditions, followed by Bonferroni-corrected post-hoc t-tests; $P < 0.01$) and within all subjects (two way t-tests $P < 0.05$). This gap effect was composed of both a warning component (OVERLAP versus AUDITORY conditions) which ranged from 11 to 35 ms (20 ± 9 ms; $P < 0.05$), and a foveal component (AUDITORY versus GAP conditions) which ranged from 35 to 88 ms (62 ± 22 ms; $P < 0.01$).

As in Experiment 1, subjects had more non-cancelled saccades on *STOP* trials with longer SSDs. Inhibition functions for each subject were constructed for data obtained from GAP (Fig. 4B), AUDITORY (Fig. 4C) and OVERLAP (Fig. 4D) conditions. SSRT calculations yielded a significant gap effect (i.e., SSRTs were shorter

on GAP vs. OVERLAP conditions) for all subjects regardless of which method was used (integration method: 48 ± 5 ms, range: 43 to 55 ms ($P < 0.01$); mean method: 60 ± 32 ms, range: 36 to 116ms ($P < 0.05$)). We also found a significant foveal effect (AUDITORY vs. GAP conditions) on SSRTs for all subjects regardless of the method of SSRT calculation (integration method: 43 ± 10 ms, range: 29 to 54 ms ($P < 0.01$); mean method: 43 ± 9 ms, range: 29 to 52 ms ($P < 0.01$)). However, we did not observe a consistent warning effect (OVERLAP vs. AUDITORY) on SSRTs with either method (integration method: 5 ± 18 ms, range: -5 to 22 ms ($P = 0.35$); mean method: 17 ± 30 , range: -5 to 69 ms ($P = 0.27$)), with AUDITORY SSRTs being shorter in only three of the five subjects for each method.

Upon averaging SSRT estimates, a gap effect was observed for all five subjects (54 ± 14 ms; range: 44 to 79 ms ($P < 0.01$); Fig. 4E, Table 1). A foveal component was also observed in all subjects (43 ± 9 ms; range: 30 to 51 ms ($P < 0.01$); Fig. 4E, Table 1). However, we did not observe a significant warning component, even though modest reductions in SSRTs were observed in four of five subjects (11 ± 14 ms; range: -5 to 35 ms ($P = 0.17$); Fig. 4E, Table 1).

In summary, we found that the foveal and warning components each contribute significantly to the gap effect on RTs (Fig. 4A, Table 1), while only the foveal component contributes significantly to the gap effect on SSRTs (Fig. 4E, Table 1).

Experiment 3

Experiments 1 and 2 provide strong evidence for the priming of the STOP process in the GAP condition versus the OVERLAP condition. However, the reappearance of the FP as a stop signal in the GAP condition in both experiments could be perceived as a

more salient stop signal than the colour change in the OVERLAP condition. A potential difference in stop signal saliency could confound our results since SSRTs have been shown to be shorter for more intense stop signals (Hanes and Carpenter 1999). To avoid this potential confound, in Experiment 3 we presented a remote auditory stop signal in both GAP and OVERLAP conditions. Remote auditory stop signals have been used extensively in saccadic countermanding (Cabel et al. 2000; Colonius et al. 2001; Curtis et al. 2005; Aron and Poldrack 2006), and provide the advantage of being spatially displaced from the fovea, where the fixation point manipulation is taking place. While we could have used a remote visual stop signal (e.g., illumination of a background light), we felt that an auditory stop signal removed potential concerns regarding the use of a single modality. Accordingly, we believe that the saliency of the auditory stop signal was equal in the GAP and OVERLAP condition.

We analyzed a total of 5257 *CONTROL* trials over the entire sample. Consistent with previous findings, including Experiments 1 and 2, we found a significant gap effect on RTs both across our sample (87 ± 24 ms, range: 65 to 117 ms ($P < 0.005$); Fig. 5A, Table 1) and in all five subjects (all $P < 0.05$, two way t-tests). As in Experiments 1 & 2, subjects had more non-cancelled saccades on *STOP* trials with longer SSDs, regardless of fixation condition (Fig. 5B,C). Consistent with the findings from Experiment 1, we found a robust gap effect on SSRTs, with this reduction occurring in at least four of the five subjects using each method (integration method: 22 ± 15 ms, range: -2 to 40 ms ($P < 0.05$); mean method: 51 ± 23 ms, range: 24 to 86 ms ($P < 0.01$)).

Upon averaging SSRT estimates, a significant gap effect remained and this reduction occurred for all subjects (37 ± 9 ms; range 21 to 42 ms ($P < 0.001$); Fig. 5D,

Table 1). Overall, these data provide strong evidence that the STOP process can be primed by a 200 ms gap, regardless of stop signal saliency and modality.

Tests of the Race Model

One assumption of the Race Model is that the GO and STOP processes are stochastically independent. Therefore, the growth of the GO process should not affect the growth of the STOP process, and vice-versa. One test of such independence is to see how well reaction times of non-cancelled saccades can be predicted using control trial reaction times (Logan 1994). To do this, we compared non-cancelled *STOP* trials from a given SSD (≥ 15 non-cancelled saccades for a given subject) with their corresponding *CONTROL* trials whose reaction times were less than the sum of the subject's SSRT plus the given SSD (Fig. 6A). Over all three experiments, and all fixation conditions, we found that the representative non-cancelled portion of the *CONTROL* RT distribution predicted the actual RTs of non-cancelled saccades well (for simplicity, we present here the prediction based on the SSRT from the integration method, although the mean method produced equivalent results). In Experiment 1, the mean predicted RTs for the GAP and OVERLAP conditions exceeded the observed non-cancelled RTs by 2.9 and 0.7 ms respectively, with the differences on a per-SSD basis reaching significance in only 1/17 (GAP) and 2/13 (OVERLAP) comparisons. In Experiment 2, the mean predicted RTs for the GAP, AUDITORY, and OVERLAP conditions exceeded the observed non-cancelled RTs by 2.8, 0.3, and 1.5 ms respectively, with the differences on a per-SSD basis reaching significance in only 4/20 (GAP), 1/12 (AUDITORY) and 1/15 (OVERLAP) comparisons. In Experiment 3, the mean predicted RTs for the GAP and

OVERLAP conditions exceeded the observed non-cancelled RTs by 3.2 and 7.8 ms respectively, with the differences on a per-SSD basis reaching significance in only 6/25 (GAP) and 2/13 (OVERLAP) comparisons. Overall, these data are consistent with the assumption of independence of the GO and STOP processes.

Another test of the independence assumption in the race model is that the RTs for non-cancelled saccades should increase progressively for longer SSDs, as the delayed STOP process should eliminate less of the upper tail of the control RT distribution at longer SSDs (e.g., imagine how Fig. 6A would look for longer SSDs). Our observations validated this prediction for all three experiments, as the observed RT for non-cancelled saccades increased for longer SSDs for all fixation conditions in all three experiments (Fig. 6B-D). The change in RTs reached significance for the OVERLAP condition in Experiments 2 & 3 ($P < 0.05$; two way t-test; for inclusion, at least 3 subjects had to have at least 10 non-cancelled saccades at a given SSD). The change in RT approached significance for the other fixation conditions ($P = 0.07$ for OVERLAP and $P = 0.08$ for GAP conditions in Experiment 1, Fig. 6B; $P = 0.06$ for AUDITORY and $P = 0.13$ for GAP conditions in Experiment 2, Fig. 6C; $P = 0.08$ for GAP in Experiment 3; a one-way ANOVA was used when the inclusion criteria were met at more than 2 SSDs). These patterns are also generally consistent with the independence assumption of the GO and STOP processes.

DISCUSSION

We investigated the effect of introducing a 200 ms gap between the offset of a foveal fixation point and target presentation on subject performance in an oculomotor

countermanding task. Previous work has established that such a manipulation expedites reaction times to the target on a simple saccade task. This phenomenon has been termed the “gap effect”, and neural correlates have been observed in both the superior colliculus and frontal eye fields (Schall 1991;Dorris and Munoz 1995;Hanes and Schall 1995;Hanes et al. 1998;Opris et al. 2001;Paré and Hanes 2003). Here, we report a surprising finding that parallels the gap effect on reaction times: the introduction of a 200 ms gap also expedites the time required to cancel an impending saccade. Follow-up experiments confirmed the robustness of this effect, and also demonstrated that this effect is due primarily to the removal of a foveal visual stimulus, and is present for both visual and auditory stop signals. In this Discussion, we first contextualize our results in comparison to others in the literature, and then briefly speculate on possible underlying neural mechanisms.

Comparison to previous results

There is a rich literature in psychophysics regarding the gap effect on simple reaction time tasks. The effect was first reported by Saslow (1967), and subsequent experiments over the past 40 years have confirmed the consistency of this effect, and its presence in both human and animal studies (see (Munoz et al. 2000) for review). In general, our results from *CONTROL* trials are consistent with this literature in that we found a consistent gap effect in all three experiments, which averaged ~75 ms. The overall magnitude of this gap effect is somewhat larger than most other human studies (Munoz and Corneil 1995;Reuter-Lorenz et al. 1995;Pratt et al. 2000), which reported gap effects ranging from 30-60 ms. These differences could be subject related (note from

Table 1 that the gap effect varied considerably in different subjects) or perhaps related to the nature of the countermanding task. For example, it is known that RTs on *CONTROL* trials in the countermanding task are longer than in simple RT tasks (Lappin and Eriksen 1966). Therefore, if both *OVERLAP* and *GAP* RTs were lengthened by a similar percentage, due to the presence of infrequent *STOP* trials, it could lead to the larger gap effect seen in this study. Further, the SSRTs in the *GAP* condition, while shorter than in the *OVERLAP* condition, are consistent with previous results from our lab (Corneil and Elsley 2005). The SSRTs we observed in the *OVERLAP* conditions are generally consistent with those reported elsewhere (Morein-Zamir and Kingstone 2006).

The presence of an observable and consistent gap effect on SSRTs in all three experiments suggests that both the *GO* and *STOP* processes are primed by the introduction of a 200 ms gap. These results are consistent with a recent report using a double-step task, which demonstrated that both inhibitory and saccade preparatory processes could be primed in delayed- or memory-guided fixation conditions (Kapoor and Murthy 2008). While the magnitude of the gap effect on SSRTs was roughly comparable across all three variants of the task (~40 ms, see Table 1), the gap effect was proportionally smallest in Experiment 3 (e.g., *OVERLAP* vs. *GAP* SSRTs were reduced by 34, 36, and 19% in Experiments 1-3, respectively). This comparative result may attest in part to a perceptually more salient stop signal in the *GAP* condition in Experiments 1 and 2 (due to re-illumination of the fixation point; recall that the *STOP* signal in the *OVERLAP* condition consisted of a change in the color of the fixation point). However, the results from Experiment 3 demonstrate a persistent gap effect on SSRTs even when the presumed saliency of the stop signal is equated.

Experiment 2 confirmed that the gap effect on RTs can be decomposed into two components, one warning of impending target presentation and the other due to the loss of a foveal stimulus (Ross and Ross 1980; Ross and Ross 1981; Reuter-Lorenz et al. 1995). However, only the foveal component seems to be involved in the reduction of SSRTs with no discernible contribution from the warning component. This finding emphasizes distinctions between the processes dictating saccade generation and cancellation.

To our knowledge there are only two other reports which have examined the influences of manipulating the fixation point in countermanding paradigms. The most pertinent comparison of our work is to that of Morein-Zamir and Kingstone (2006), who examined human performance in an oculomotor countermanding task. The authors reported a negligible influence of the offset of a fixation point on SSRTs, despite a modest decrease in saccadic reaction times on *CONTROL* trials (which averaged ~18 ms). However, it is important to emphasize that Morein-Zamir and Kingstone studied the influence of simultaneous fixation point offset and target presentation (i.e., a gap of 0 ms), whereas we employed a gap of 200 ms. We suggest that the longer gap interval employed in our experiment is in a more optimal range to study the influences of fixation point manipulations on processes related to saccade cancellation, as reductions in saccadic RTs are greatest for gaps ranging between 200-400 ms (Juttner and Wolf 1992; Dorris and Munoz 1995; Munoz et al. 2000).

A similar mechanism cannot reconcile results reported for the countermanding of arm movements (Mirabella et al. 2008). This study reports that SSRTs for arm movements increase when a gap interval is increased from 0 ms to 212 ms, despite

decreases in the reaction times of arm movements on *CONTROL* trials. Our results attest to possible effector-related differences between the control of oculomotor versus limb-movement responses, consistent with previous reports which have suggested an independence between STOP processes for eye and hand movements (Logan and Irwin 2000;Boucher et al. 2007b). In light of these results and those reported by Mirabella and colleagues (2008), it would be particularly interesting to examine the magnitude and direction of gap effects on eye and hand SSRTs for combined eye-hand movements.

Underlying neural substrates

Based on the comparison of our results to those of Mirabella and colleagues (2008), we speculate that the implementation of the gap effect on SSRTs occurs downstream of general sensory or attentional processes, otherwise one would expect similar results for both eye and hand SSRTs. We therefore speculate that the priming of the STOP process we have observed is unique to the oculomotor system.

On the surface, the simultaneous priming of both the GO and STOP processes in the GAP condition would seem to support the assumption of independence common to many race model architectures. However, this assumption of independence runs counter to the mutually antagonistic relationship between saccade generation and saccade fixation mechanisms thought to exist in the oculomotor system. Within the SC and FEF, neural correlates of the gap effect (Dorris and Munoz 1995;Opris et al. 2001) and GO and STOP processes (Hanes et al. 1998;Paré and Hanes 2003;Brown et al. 2008) have been observed. At least within the SC, there is neurophysiological evidence for short-latency, mutually-antagonistic projections between saccade- and fixation-related neurons (Munoz

and Istvan 1998; Meredith and Ramoa 1998). Given that the introduction of a 200 ms gap increases saccade-related activity and decreases fixation-related activity, one would have expected that priming the GO process in the GAP condition should have slowed the STOP process, prolonging SSRTs. How can we reconcile our behavioural evidence for simultaneous priming of GO and STOP processes with known patterns of oculomotor activity?

A recent model of saccadic countermanding potentially resolves the paradox between the computational assumption of independence and neurophysiological activity within the oculomotor system (Boucher et al. 2007a). The crux of this model is that the GO and STOP processes remain independent for most of the processing, but then interact in a latter stage such that the STOP process potently inhibits the GO process. Unlike independent race model architectures, which remain agnostic about underlying patterns of neural activity, this interactive race model is consistent with both behaviour and profiles of oculomotor activity in a saccadic countermanding task.

We believe that our results can be explained within the context of this interactive race model. To illustrate this, consider first the idealized profiles of fixation-related activity recorded on cancelled and non-cancelled saccades in the OVERLAP condition [top part of Fig. 7A; activity profiles are based on those recorded from the SC by Paré and Hanes (2003; see their Fig. 7)]. Note how fixation-related activity remains fairly constant through the time of target and stop signal presentation, but then diverges sharply depending on ensuing behaviour, with activity increasing or decreasing depending on whether the saccade is cancelled or not, respectively. The timing of this divergence becomes particularly clear by taking the difference between these two profiles (lower part

of Fig. 7A). The key observation made by Paré and Hanes (2003) was that this divergence in activity, which they term the “neural estimate of SSRT” preceded behavioural estimates of SSRTs. This is a logical pre-requisite if fixation-related neurons are to play a causal role in saccade control.

We speculate similar profiles of fixation-related activity will be observed in the GAP condition, but with a few important differences. First, fixation-related activity would begin to decrease during the 200 ms gap [idealized profiles in the top part of Fig. 7B are based on those recorded by Dorris and colleagues (1995)], with this decrease continuing through target and stop-signal presentation. Because the subjects cannot anticipate trial type or the SSD, the decrease in fixation-related activity would be the same for both cancelled and non-cancelled trials. Second, we believe that the divergence in fixation-related activity on cancelled vs non-cancelled trials would be occurring *earlier* in the GAP condition compared to the OVERLAP condition (bottom part of Fig. 7B, 7C; as discussed below, we believe the signal dictating such divergence does not originate in the SC). This earlier divergence of fixation-related activity in the GAP vs. OVERLAP condition (Fig. 7C) would then explain our behavioural observations of priming of the STOP process in the GAP condition.

Although we have only depicted fixation-related activity in Fig. 7, we speculate that reciprocal profiles of activity would be observed on movement-related neurons (e.g., neural activity would increase during the GAP period, and increase further only on non-cancelled trials). In this regard, the asymmetric inhibition predicted by Boucher and colleagues (2007; that fixation-related neurons inhibit saccade-related neurons more than vice versa) is particularly important; otherwise the increase in movement-related activity

during the GAP period would prevent the subsequent increase in fixation-related activity for cancelled trials.

Within the context of our predicted profiles of activity, the pertinent question then becomes why the fixation-related neurons diverge in activity earlier in the GAP vs. OVERLAP condition. Although recordings of activity during saccadic countermanding have been made from a number of areas, to date only neurons within the FEF and SC have the appropriately timed divergence in activity to be involved in saccade control [i.e., whereby neural SSRTs precede behavioural SSRTs; (Hanes et al. 1998;Paré and Hanes 2003;Brown et al. 2008)]. A number of groups have speculated on the role of fronto-striatal networks in saccade cancellation (Paré and Hanes 2003;Aron and Poldrack 2006), but it remains to be determined whether these networks directly implement saccade control. However, recording studies throughout the basal ganglia have demonstrated changes in activity during a gap period preceding target presentation (Hikosaka and Wurtz 1983;Kobayashi et al. 2002). Thus, circuits through the basal ganglia could provide the means by which priming of the STOP process is implemented.

In conclusion, the STOP process can be primed in a human saccadic countermanding task by the introduction of a 200 ms gap. This robust priming is mainly driven by a foveal component and occurs regardless of stop signal saliency or modality. Further investigation is required to understand how, on a neural level, oculomotor areas manifest simultaneous priming of the GO and STOP processes.

Reference List

1. **Aron AR and Poldrack RA** Cortical and subcortical contributions to Stop signal response inhibition: role of the subthalamic nucleus. *J.Neurosci.* 26: 2424-2433, 2006.
2. **Boucher L, Palmeri TJ, Logan GD, and Schall JD** Inhibitory control in mind and brain: an interactive race model of countermanding saccades. *Psychol.Rev.* 114: 376-397, 2007a.
3. **Boucher L, Stuphorn V, Logan GD, Schall JD, and Palmeri TJ** Stopping eye and hand movements: are the processes independent? *Percept.Psychophys.* 69: 785-801, 2007b.
4. **Brown JW, Hanes DP, Schall JD, and Stuphorn V** Relation of frontal eye field activity to saccade initiation during a countermanding task. *Exp.Brain Res.* 2008.
5. **Cabel DW, Armstrong IT, Reingold E, and Munoz DP** Control of saccade initiation in a countermanding task using visual and auditory stop signals. *Exp.Brain Res.* 133: 431-441, 2000.
6. **Colonus H, Ozyurt J, and Arndt PA** Countermanding saccades with auditory stop signals: testing the race model. *Vision Res.* 41: 1951-1968, 2001.

7. **Corneil BD and Elsley JK** Countermanding eye-head gaze shifts in humans: marching orders are delivered to the head first. *J.Neurophysiol.* 94: 883-895, 2005.
8. **Corneil BD and Munoz DP** The influence of auditory and visual distractors on human orienting gaze shifts. *J.Neurosci.* 16: 8193-8207, 1996.
9. **Curtis CE, Cole MW, Rao VY, and D'Esposito M** Canceling planned action: an FMRI study of countermanding saccades. *Cereb.Cortex* 15: 1281-1289, 2005.
10. **Dorris MC and Munoz DP** A neural correlate for the gap effect on saccadic reaction times in monkey. *J.Neurophysiol.* 73: 2558-2562, 1995.
11. **Emeric EE, Brown JW, Leslie M, Pouget P, Stuphorn V, and Schall JD** Performance monitoring local field potentials in the medial frontal cortex of primates: anterior cingulate cortex. *J.Neurophysiol.* 99: 759-772, 2008.
12. **Fendrich R, Demirel S, and Danziger S** The oculomotor gap effect without a foveal fixation point. *Vision Res.* 39: 833-841, 1999.
13. **Findlay JM and Walker R** A model of saccade generation based on parallel processing and competitive inhibition. *Behav.Brain Sci.* 22: 661-674, 1999.
14. **Fischer B** The preparation of visually guided saccades. *Rev.Physiol Biochem.Pharmacol.* 106: 1-35, 1987.

15. **Forbes K and Klein RM** The magnitude of the fixation offset effect with endogenously and exogenously controlled saccades. *J.Cogn.Neurosci.* 8: 344-352, 1996.
16. **Hanes DP and Carpenter RH** Countermanding saccades in humans. *Vision Res.* 39: 2777-2791, 1999.
17. **Hanes DP, Patterson WF, and Schall JD** Role of frontal eye fields in countermanding saccades: visual, movement, and fixation activity. *J.Neurophysiol.* 79: 817-834, 1998.
18. **Hanes DP and Schall JD** Countermanding saccades in macaque. *Vis.Neurosci.* 12: 929-937, 1995.
19. **Hanes DP and Schall JD** Neural control of voluntary movement initiation. *Science* 274: 427-430, 1996.
20. **Hikosaka O and Wurtz RH** Visual and oculomotor functions of monkey substantia nigra pars reticulata. III. Memory-contingent visual and saccade responses. *J.Neurophysiol.* 49: 1268-1284, 1983.
21. **Juttner M and Wolf W** Occurrence of human express saccades depends on stimulus uncertainty and stimulus sequence. *Exp.Brain Res.* 89: 678-681, 1992.

22. **Kapoor V and Murthy A** Covert inhibition potentiates online control in a double-step task. *J.Vis.* 8: 20-16, 2008.
23. **Kingstone A and Klein RM** Visual offsets facilitate saccadic latency: does predisengagement of visuospatial attention mediate this gap effect?
J.Exp.Psychol.Hum.Percept.Perform. 19: 1251-1265, 1993.
24. **Kobayashi Y, Inoue Y, Yamamoto M, Isa T, and Aizawa H** Contribution of pedunculopontine tegmental nucleus neurons to performance of visually guided saccade tasks in monkeys. *J.Neurophysiol.* 88: 715-731, 2002.
25. **Lappin JS and Eriksen CW** Use of a delayed signal to stop a visual reaction-time response. *J.Exp.Psychol* 72: 805-811, 1966.
26. **Logan GD** On the ability to inhibit thought and action: a users' guide to the stop-signal paradigm. In Dagenbach D and Carr H eds. *Inhibitory processes in attention, memory and language*. Kent, Academic Press. 1994, 189-239.
27. **Logan GD and Cowan WB** On the ability to inhibit thought and action: a theory of an act of control. *Psychol Rev* 91: 295-327, 1984.
28. **Logan GD and Irwin DE** Don't look! Don't touch! Inhibitory control of eye and head movements. *Psychon.Bull.Rev.* 7: 107-112, 2000.

29. **Meredith MA and Ramoa AS** Intrinsic circuitry of the superior colliculus: pharmacophysiological identification of horizontally oriented inhibitory interneurons. *J.Neurophysiol.* 79: 1597-1602, 1998.
30. **Mirabella G, Pani P, and Ferraina S** The presence of visual gap affects the duration of stopping process. *Exp.Brain Res.* 2008.
31. **Morein-Zamir S and Kingstone A** Fixation offset and stop signal intensity effects on saccadic countermanding: a crossmodal investigation. *Exp.Brain Res.* 175: 453-462, 2006.
32. **Munoz DP and Corneil BD** Evidence for interactions between target selection and visual fixation for saccade generation in humans. *Exp.Brain Res.* 103: 168-173, 1995.
33. **Munoz DP, Dorris MC, Paré M, and Everling S** On your mark, get set: brainstem circuitry underlying saccadic initiation. *Can.J.Physiol Pharmacol.* 78: 934-944, 2000.
34. **Munoz DP and Istvan PJ** Lateral inhibitory interactions in the intermediate layers of the monkey superior colliculus. *J.Neurophysiol.* 79: 1193-1209, 1998.

35. **Opris I, Barborica A, and Ferrera VP** On the gap effect for saccades evoked by electrical microstimulation of frontal eye fields in monkeys. *Exp.Brain Res.* 138: 1-7, 2001.
36. **Ozyurt J, Colonius H, and Arndt PA** Countermanding saccades: evidence against independent processing of go and stop signals. *Percept.Psychophys.* 65: 420-428, 2003.
37. **Paré M and Hanes DP** Controlled movement processing: superior colliculus activity associated with countermanded saccades. *J.Neurosci.* 23: 6480-6489, 2003.
38. **Paré M and Munoz DP** Saccadic reaction time in the monkey: advanced preparation of oculomotor programs is primarily responsible for express saccade occurrence. *J.Neurophysiol.* 76: 3666-3681, 1996.
39. **Pratt J, Bekkering H, and Leung M** Estimating the components of the gap effect. *Exp.Brain Res.* 130: 258-263, 2000.
40. **Reuter-Lorenz PA, Oonk HM, Barnes LL, and Hughes HC** Effects of warning signals and fixation point offsets on the latencies of pro- versus antisaccades: implications for an interpretation of the gap effect. *Exp.Brain Res.* 103: 287-293, 1995.

41. **Ross LE and Ross SM** Saccade latency and warning signals: stimulus onset, offset, and change as warning events. *Percept.Psychophys.* 27: 251-257, 1980.
42. **Ross SM and Ross LE** Saccade latency and warning signals: effects of auditory and visual stimulus onset and offset. *Percept.Psychophys.* 29: 429-437, 1981.
43. **Saslow MG** Effects of components of displacement-step stimuli upon latency for saccadic eye movements. *J.Opt.Soc.Am.* 57: 1024-1029, 1967.
44. **Schall JD** Neuronal activity related to visually guided saccades in the frontal eye fields of rhesus monkeys: comparison with supplementary eye fields. *J.Neurophysiol.* 66: 559-579, 1991.
45. **Scudder CA, Kaneko CS, and Fuchs AF** The brainstem burst generator for saccadic eye movements: a modern synthesis. *Exp.Brain Res.* 142: 439-462, 2002.
46. **Sparks DL** The brainstem control of saccadic eye movements. *Nat.Rev.Neurosci.* 3: 952-964, 2002.
47. **Stevenson SA, Elsley JK, and Corneil BD.** A "Gap" effect on SSRTs: Priming of the stop signal with varying states of visual fixation in a saccadic countermanding task. *Soc.Neurosci.Abstr.* (719.4). 2007.

Ref Type: Abstract

48. **Stuphorn V, Taylor TL, and Schall JD** Performance monitoring by the supplementary eye field. *Nature* 408: 857-860, 2000.

49. **Taylor TL, Kingstone A, and Klein RM** The disappearance of foveal and non-foveal stimuli: Decomposing the gap effect. *Can.J.Exp.Psychol* 52: 192-199, 1998.

FIGURE LEGENDS

Figure 1 Schematic drawing of the Race Model where stochastically independent GO and STOP processes race towards a common threshold, with the outcome of this race determining which response is generated. The GO process begins with target onset while the STOP process begins with the onset of the STOP signal. The time between target and STOP signal presentation is the Stop Signal Delay (SSD), which can vary from trial-to-trial. *A.* The GO process starts sufficiently early to beat the STOP process to threshold, and hence a saccade is generated. *B.* The GO process begins, but the STOP process beats the GO process to threshold, resulting in the cancellation of a saccade. EOG traces are taken from one representative subject to contrast the two possible outcomes of the race model (X and Y axes represent 50 ms and 5° saccade amplitude).

Figure 2 Schematic drawing of STOP trials used in the three experiments, which composed 30-35% of all trials. *A.* Experiment 1 used two fixation conditions: GAP and OVERLAP. The GAP condition involves the disappearance of a central fixation point (FP) 200 ms prior to target onset while the FP remains illuminated for the duration of the trial in the OVERLAP condition. On *STOP* trials, after a given stop signal delay (SSD), the FP reappears as a green FP (GAP) or simply changes colour to green (OVERLAP). *B.* In Experiment 2, we introduced a third condition, the AUDITORY condition, where an auditory cue accompanied FP onset and was extinguished 200ms prior to target onset while the FP remains illuminated throughout the trial. On *STOP* trials, after a given SSD, the FP changes colour, similar to the OVERLAP condition. *C.* In Experiment 3, GAP

and OVERLAP conditions were used as in Experiment 1, but the stop signal consisted of a remote auditory tone.

Figure 3 Results from Experiment 1. *A.* Reaction time (RT) results for *CONTROL* trials for each subject (dashed lines) and the sample mean (squares and solid thick line) on OVERLAP and GAP conditions. A significant gap effect of ~50 ms was seen across the sample with a reduction in RTs observed in all six subjects. *B, C.* Inhibition functions for each subject in OVERLAP (*B*) and GAP (*C*) conditions, indicating the probability of making a saccade ($P(\text{saccade})$) at a given SSD. $P(\text{saccade})$ increased with increasing SSD. *D.* Mean SSRTs from combining the estimates from the Integration and Mean methods on OVERLAP and GAP conditions for each subject (dashed lines) and the sample mean (squares and solid thick line). A consistent gap effect of ~40 ms was seen across the sample, with reductions in SSRT occurring for all six subjects. * represent $P < 0.05$ using paired t-tests. Error bars represent standard error of mean.

Figure 4 Results from Experiment 2. Same format as Fig. 3. *A.* A significant gap effect of ~82 ms was seen across the sample, with RT reductions occurring in all five subjects. Significant warning (~20 ms) and foveal (~62 ms) effects were also observed for all five subjects. *B-D.* Inhibition functions for each subject in the various fixation conditions. *E.* SSRTs for the three fixation conditions. A significant gap effect of ~50 ms was observed. No significant warning effect is observed, but a significant foveal effect (~40 ms) was observed for all five subjects. * represent $P < 0.05$ using repeated measures ANOVA followed by Bonferroni post-hoc correction test for multiple comparisons.

Figure 5 Results from Experiment 3, using same format at Fig. 3. *A*. A significant gap effect of ~90 ms was observed across the sample *B,C*. Inhibition functions for the OVERLAP (*B*) and GAP (*C*) conditions. *D*. A significant gap effect on SSRTs of ~35 ms was observed.

Figure 6 Tests of the race model. *A*. Depiction of how the race model can be used to predict RTs of non-cancelled saccades at a given SSD. The RT distribution from control trials can be subdivided into a portion that would or would not have been cancelled, had the STOP signal been presented, by the sum of the predetermined SSRT and the given SSD. Those movements falling into the non-cancelled portion predict the RTs of non-cancelled movements at that SSD. *B-D*. Normalized RTs of non-cancelled saccades as a function of SSD in Experiment 1 (*B*), Experiment 2 (*C*), and Experiment 3 (*D*). Within each plot in *B-D*, mean RTs from all non-cancelled trials are normalized to the mean RT from CONTROL trials for that subject, and data was then pooled across all subjects. Consistent with the race model, RTs of non-cancelled movements increased for longer SSDs in all experiments. * denote significant observations using two-way *t*-tests, $P < 0.05$. The SSDs in *B-D* are denoted in rank order, since not all subjects had the same series of SSDs (e.g., see Fig. 4). In order to compare observations across subjects, SSDs were ordered from shortest to longest. Therefore SSDs 4,5 and 6 refer to the longest three SSDs for each subject).

Figure7 Sketch of predicted profiles of the activity of SC fixation-related neurons. The activity profiles are based on those recorded during a saccadic countermanding task (Paré and Hanes 2003), and during a saccade task with a 200 ms gap (Dorris and Munoz 1995). Top portions of A (for the OVERLAP condition) and B (for the GAP condition) show fixation-related activity on STOP trials, segregated based on whether the saccade was successfully cancelled (thick line) or not (thin line). Note how neural activity begins to decrease before target (T) presentation in B, consistent with Dorris and Munoz (1995). Bottom portions of A and B show the difference between these two curves, emphasizing that a neural estimate of SSRT runs from stop signal presentation until the divergence in neural activity. These difference curves are contrasted directly in C, emphasizing our prediction that the divergence in activity depending on performance occurs early in the GAP vs. OVERLAP condition.

Table 1. Reaction Times (RT) from *CONTROL* trials and Stop Signal Reaction Times (SSRT) estimated from *STOP* trials for all experiments and all subjects. Values in brackets denote standard deviations. SSRTs are the average of the estimates derived from the Integration and Mean methods. The subscripts *O*, *G*, and *A* denote OVERLAP, GAP, and AUDITORY fixation conditions respectively.

	Experiment 1				Experiment 2						Experiment 3			
	RT _O	RT _G	SSRT _O	SSRT _G	RT _O	RT _A	RT _G	SSRT _O	SSRT _A	SSRT _G	RT _O	RT _G	SSRT _O	SSRT _G
<i>S1</i>	261(38)	193(43)	119	78	289(44)	254(43)	172(44)	140	129	89				
<i>S2</i>	278(59)	214(40)	123	67	273(46)	256(61)	197(37)	139	144	93	296(59)	218(63)	180	142
<i>S3</i>					393(46)	372(68)	324(69)	164	129	85	337(71)	229(82)	220	178
<i>S4</i>	304(50)	252(42)	153	104	315(51)	304(54)	270(43)	151	141	107	323(57)	254(53)	176	155
<i>S5</i>	278(51)	173(42)	129	75	313(61)	301(39)	213(37)	156	151	105				
<i>S6</i>	234(73)	190(69)	109	71										
<i>S7</i>	253(41)	247(50)	95	83										
<i>S8</i>											378(85)	261(105)	216	175
<i>S9</i>											358(64)	293(66)	188	148
MEAN	268(24)	212(32)	121(20)	80(13)	317(46)	297(48)	235(61)	150(11)	139(10)	96(10)	338(32)	251(29)	196(21)	159(16)

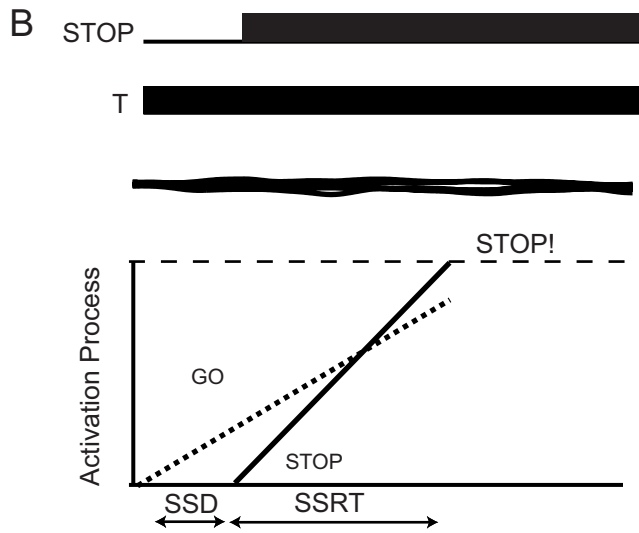
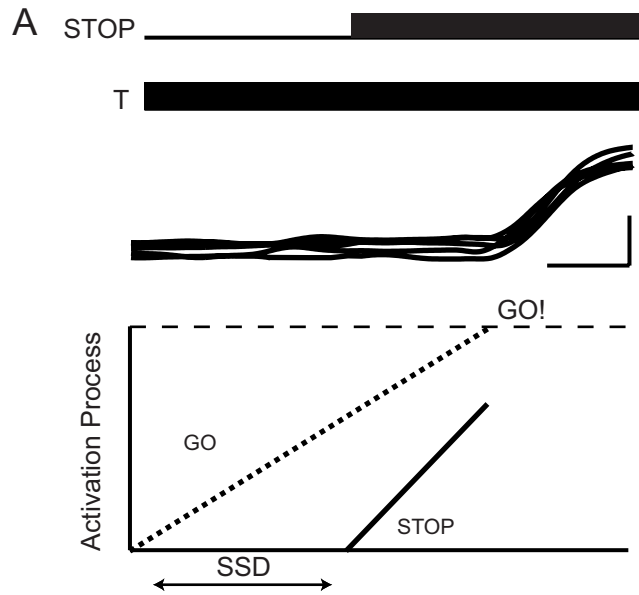
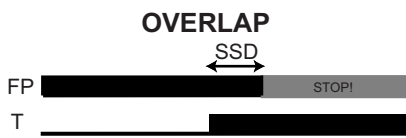
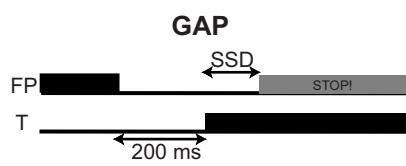
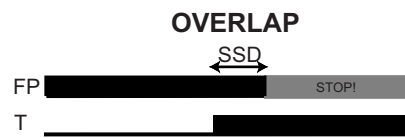
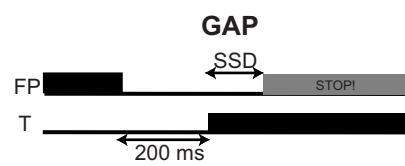


Figure 1

A Experiment 1



B Experiment 2



C Experiment 3

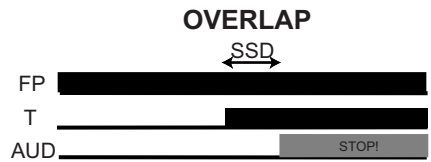
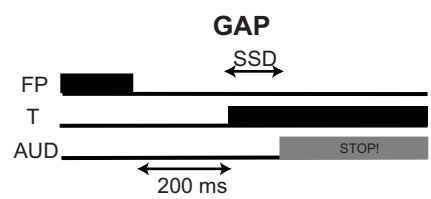


Figure 2

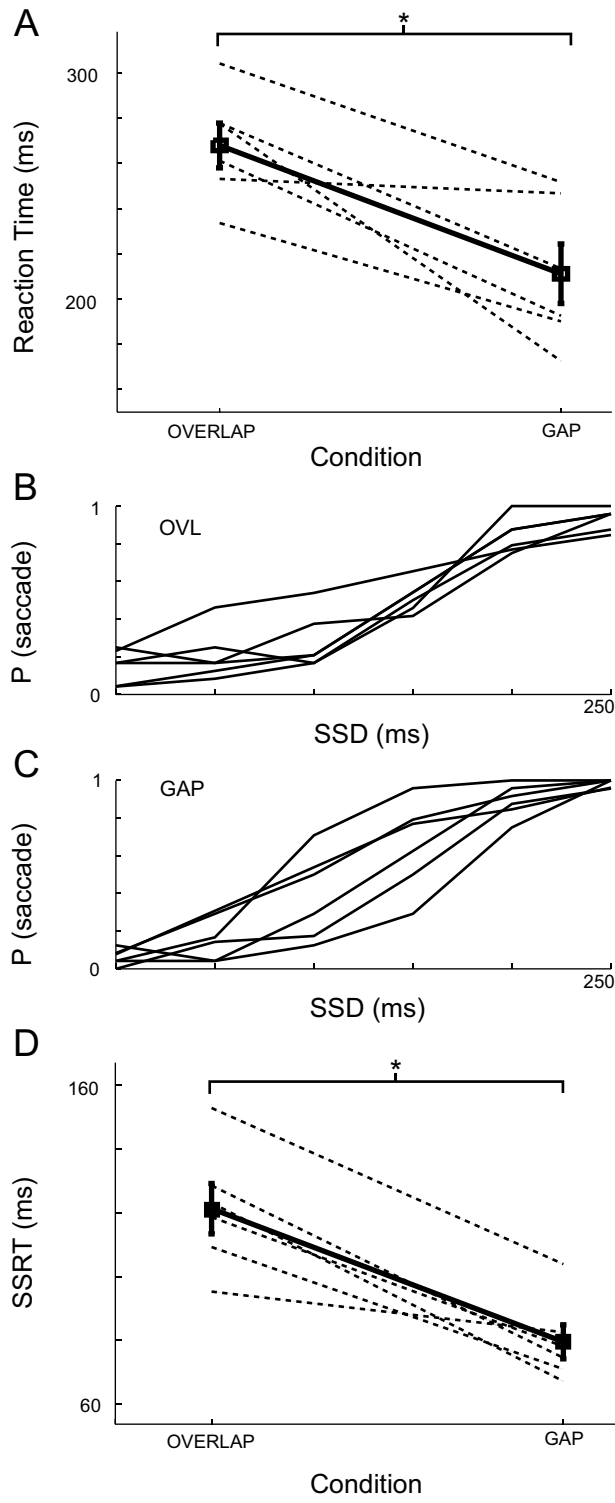


Figure 3

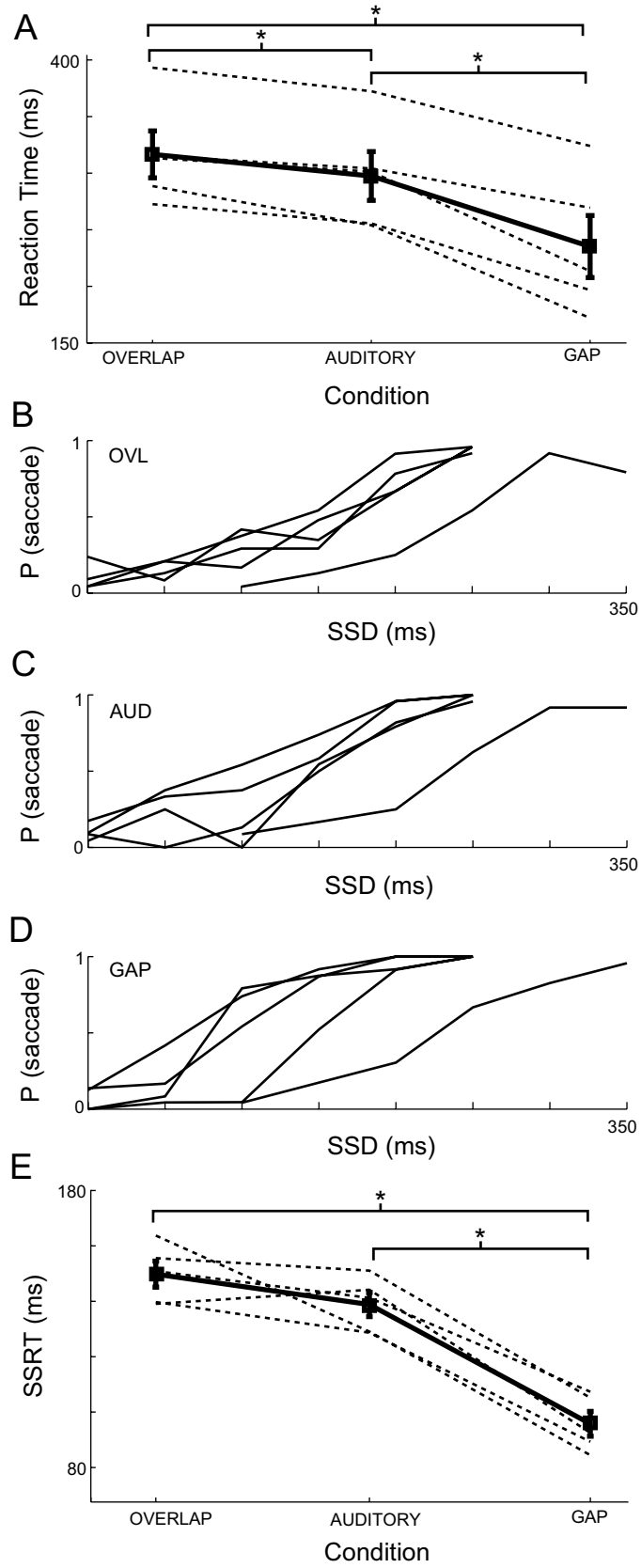


Figure 4

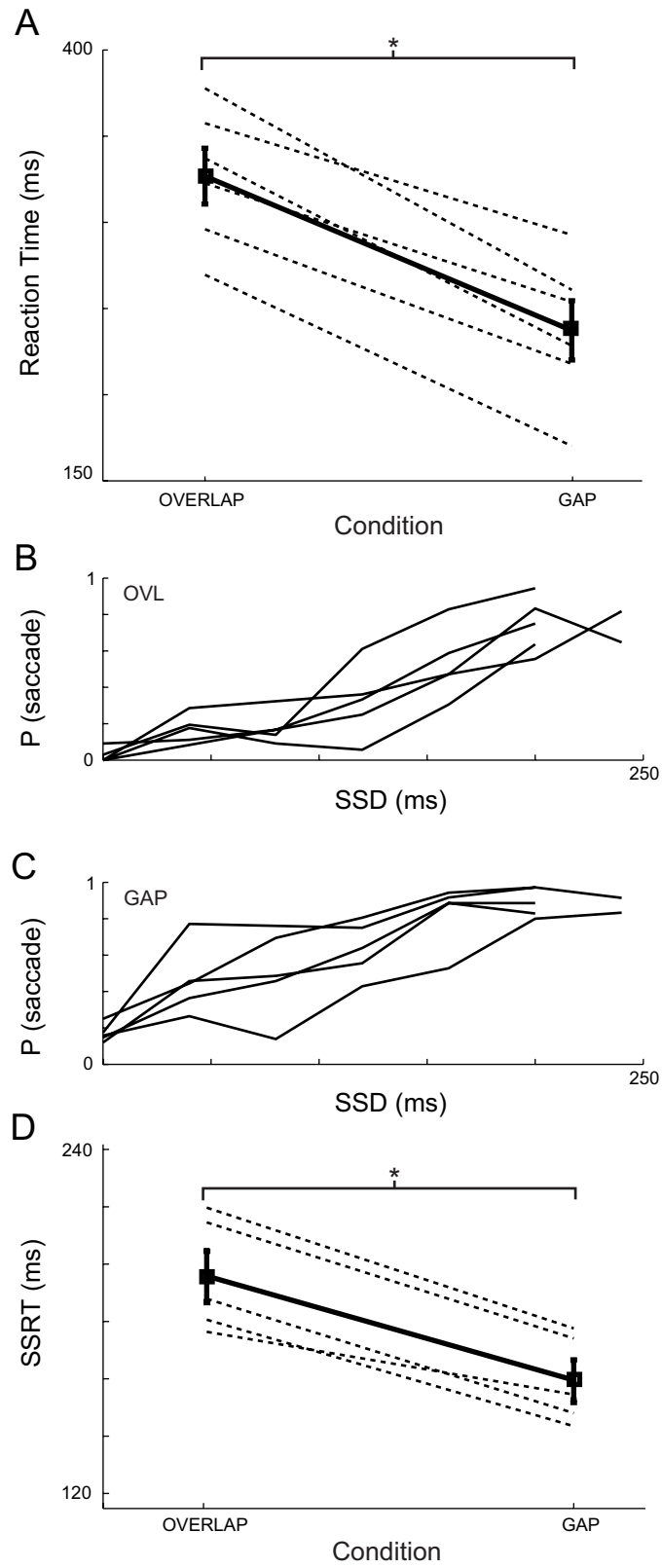


Figure 5

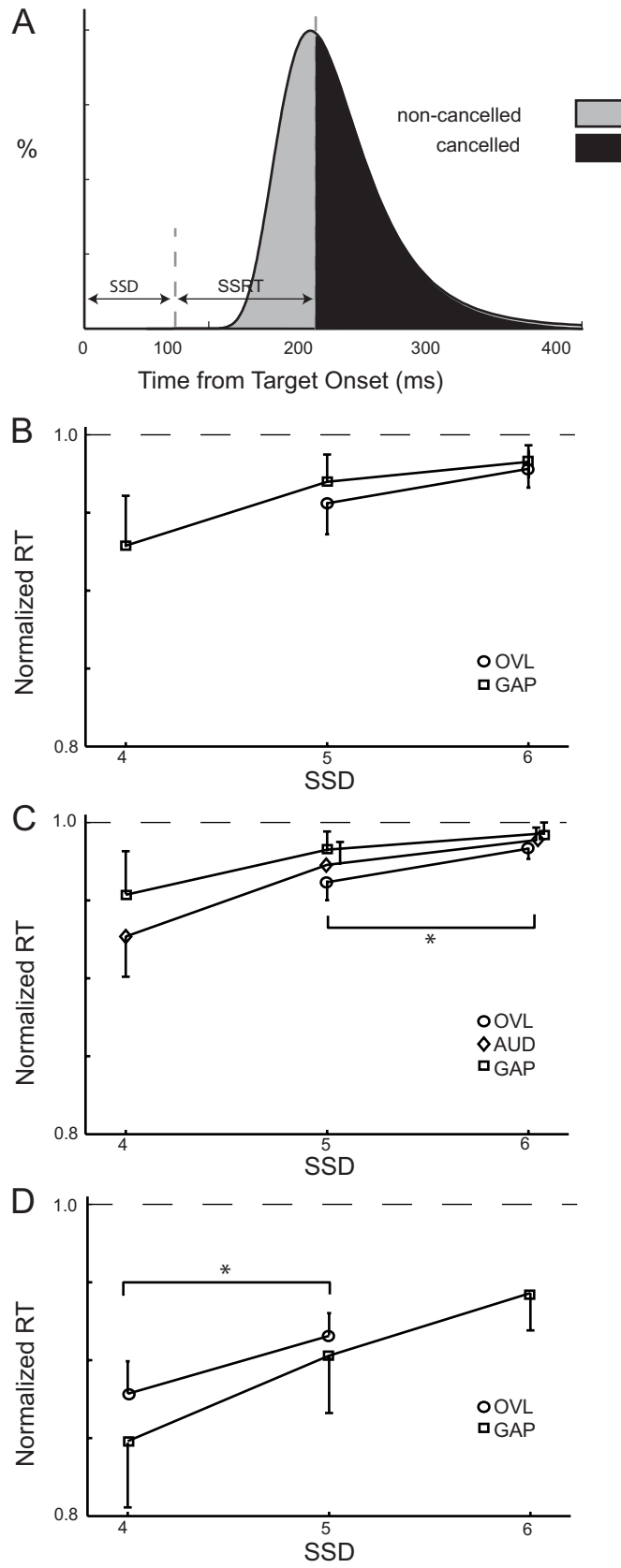


Figure 6

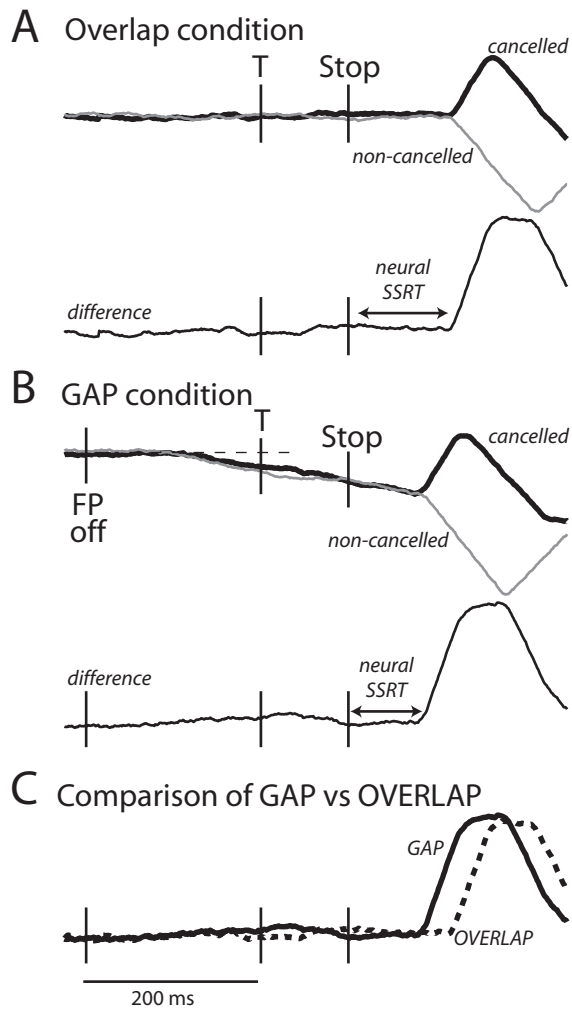


Figure 7