Journal of Experimental Psychology:
Human Perception and Performance
2013, Vol. 39, No. 4, 941–946

0096-1523/13/$12.00 DOI: 10.1037/a0033049

OBSERVATION

Stop Before You Leap: Changing Eye and Hand Movements Requires Stopping

Patrick G. Bissett and Gordon D. Logan
Vanderbilt University

The search–step paradigm addresses the processes involved in changing movement plans, usually saccadic eye-movements. Subjects move their eyes to a target (T1) among distractors, but when the target steps to a new location (T2), subjects are instructed to move their eyes directly from fixation to the new location. We ask whether moving to T2 requires a separate stop process that inhibits the movement to T1. It need not. The movement plan for the second response may inhibit the first response. To distinguish these hypotheses, we decoupled the offset of T1 from the onset of T2. If the second movement is sufficient to inhibit the first, then the probability of responding to T1 should depend only on T2 onset. If a separate stop process is required, then the probability of responding to T1 should depend only on T1 offset, which acts as a stop signal. We tested these hypotheses in manual and saccadic search–step tasks and found that the probability of responding to T1 depended most strongly on T1 offset, supporting the hypothesis that changing from one movement plan to another involves a separate stop process that inhibits the first plan.

Keywords: cognitive control, eye movements, inhibition, race model, search–step task

The ability to change one's mind is a hallmark of cognitive control. It is addressed in a variety of tasks that require changes in movement plans, including the double-step task (Becker & Jürgens, 1979), the stop-signal task (Logan & Cowan, 1984), the stop–change task (Logan & Burkell, 1986), and the search–step task (Camalier et al., 2007; Murthy, Ray, Shorter, Schall, & Thompson, 2009; Murthy, Thompson & Schall, 2001). An important question in each of these tasks is whether changing from one movement plan to another requires a separate act of control that inhibits the first movement. That act of control is sufficient but it might not be necessary. The second movement plan may directly inhibit the first without involving a separate stop process. We report two experiments designed to distinguish these hypotheses in the search–step task.

The search–step task involves presenting a target (T1) among distractors and requires subjects to execute a movement to the location of the target. On a random subset of trials, the first target changes to a distractor and one of the distractors changes to a target (T2). When this happens, subjects are instructed to move directly to T2 without moving to T1. The probability of moving to T1 increases with target step delay, which is the interval between the onset of T1 and the offset of T1 and onset of T2. Note that T1 offset and T2 onset are simultaneous in typical search–step tasks. Our experiments separate these events.

Search–step performance has been explained in terms of a race model, in which the processes responding to T1 (Go1 processes) race against the processes responding to T2 (Go2 processes). Computational modeling suggests that the race must include a separate stop process as a third runner (Camalier et al., 2007; Ramakrishnan, Sureshbabu & Murthy, 2012). If the stop process finishes before Go1, subjects inhibit Go1 and move directly to T2; if the stop process finishes after Go1, subjects move to T1 before moving to T2. However, these conclusions require strong assumptions about the nature of the underlying processes. Camalier et al. (2007) assumed independence between Go1, Go2, and stop processes and assumed that performance depended only on the finishing times of the processes (cf. Logan & Cowan, 1984). Ramakrishnan et al. (2012) assumed that Go1, Go2, and stop were specific stochastic accumulators with specific inhibitory interactions (cf. Boucher, Palmeri, Logan & Schall, 2007).

We adopted a different strategy for determining whether search–step performance requires a separate stop process. We introduced a delay between T1 offset and T2 onset (*T2 delay*), decoupling the processes that respond to T1 offset (the stop process) from the processes that respond to T2 onset (Go2; see Verbruggen, Schneider & Logan, 2008). We manipulated T1 offset time (*T1 duration*) and T2 delay orthogonally. If search–step performance does not require a separate stop process (if programming Go2 is sufficient to inhibit Go1), then the probability of responding to T1—

This article was published Online First May 13, 2013.

Patrick G. Bissett and Gordon D. Logan, Department of Psychology, Vanderbilt University.

This research was supported by grant number BCS-0957074 from the National Science Foundation and grant number R01-EY021833-01 from the National Eye Institute.

Correspondence concerning this article should be addressed to Patrick G. Bissett or Gordon D. Logan, Department of Psychology, Vanderbilt University, Nashville, TN 37240. E-mail: patrick.g.bissett@vanderbilt.edu or gordon.logan@vanderbilt.edu

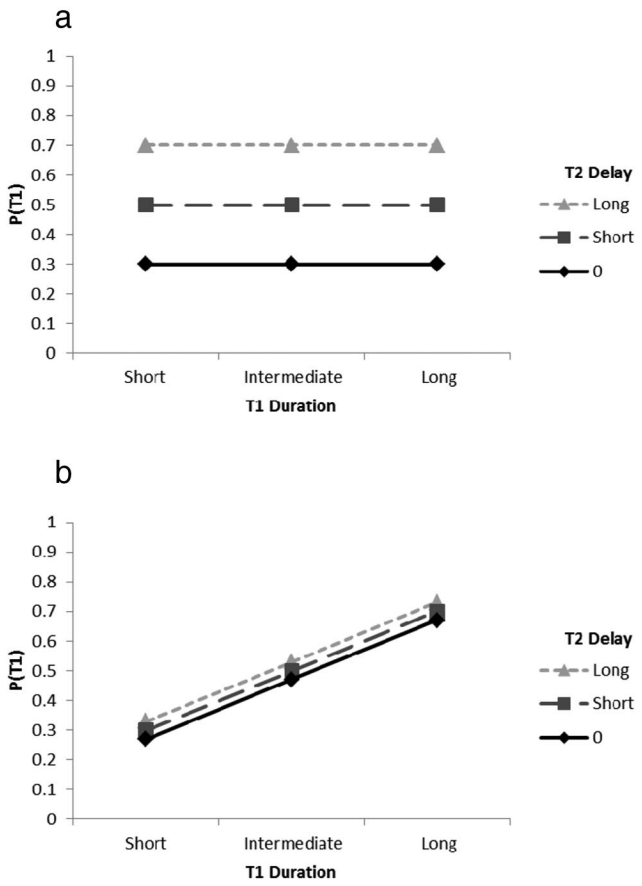

Figure 1. Predicted probability of responding first to T1 (P[T1]) across varying T1 duration and T2 delay if a separate stop process is not (a) or is (b) necessary to explain search–step performance.

P(T1)—should depend only on T2 delay (see Figure 1a). If search–step performance involves a separate stop process triggered by T1 offset and no inhibition from Go2, then P(T1) should depend only on T1 duration (see Figure 1b).

This manipulation was instantiated in two search–step experiments. Experiment 1 involved manual responding; Experiment 2 involved saccadic eye movements. Our goal was to reach general conclusions about the role of a stop process in changing movement plans by building a procedural bridge between manual tasks that are often run in single sessions with many subjects, like the stop–signal task (Logan & Cowan, 1984) and the stop–change task (Logan & Burkell, 1986) and eye-movement tasks that are often run in many sessions with fewer subjects, like the double-step task (Becker & Jürgens, 1979), saccadic stop–signal task (Hanes & Schall, 1995), and the search–step task (Murthy et al., 2001).

Experiment 1

Method

Subjects. Forty-eight naïve subjects (24 in Group A; 24 in B) were recruited from the Nashville community and were compensated $12 for a single 1-hr session. All subjects had normal or corrected-to-normal vision. We replaced three subjects for no-step accuracy below .85.

Apparatus and stimuli. The experiment was run on a PC running E-Prime 1 (pstnet.com). There were eight 2° squares arranged in a circle with the center of each square 7° from a central fixation point. Half of subjects looked for a green square among red distractors, and the other half looked for a red square among green distractors. Luminance for green squares was 50 cd/m^2, red squares 15 cd/m^2, and the white background was 80 cd/m^2. Subjects responded with the number pad at the lower right corner of the full desktop keyboard. They responded with the 1, 2, 4, and 5 keys, according to the spatially compatible location on the screen. Going clockwise from vertical, subjects responded 5 to the first two square locations, 2 to the next two, 1 to the next two, and 4 to the last two.

Procedure. Each trial began with a 500-ms fixation display, followed by the square array. The array displayed for 1000 ms, and then was replaced by a 850-ms blank screen intertrial interval (ITI).

A step occurred on 1/3 of all trials. A step involved a change of T1 from the target to the distractor color, and then a change of T2 from the distractor to the target color after T2 delay. Steps were equally likely (1/7) to each of the seven distractor locations. Steps within the same quadrant were excluded from analyses (1/7 of step trials) as Go1 and Go2 require the same response. Subjects were instructed to respond once according to the quadrant of the final step location, but to correct their error (by pressing an additional key corresponding to the T2 quadrant) if they responded to Go1. T1 duration and T2 delay were each chosen randomly from a set of three values. T1 durations were 100 ms, 300 ms, and 500 ms for Group A, and 200 ms, 300 ms, and 400 ms for Group B. For both groups, T2 delays were 0 ms (like a typical search–step task), 100 ms, and 200 ms, measured from the offset of T1. These values were chosen to sample a wide range of P(T1). The array was displayed for 1000 ms to match no-step trials.

Subjects were instructed to respond as quickly and accurately as possible when squares appeared, and to try to respond to only the final location on step trials. Subjects were given 32 trials of practice. The main task included 5 blocks of 216 trials. Between blocks, subjects were rested and were given feedback on their mean no-step reaction time (RT) and combined step and no-step accuracy from the previous block. No feedback was given between trials.

Results and Discussion

Mean correct no-step RT was 511 ms in Group A and 470 ms in Group B. There was no effect of search color on RT, $t(46) = .667$, $p > .5$, so we collapsed across search color for all analyses. Mean no-step accuracy was .98 in Group A and .97 in Group B. P(T1) was calculated by first excluding trials in which no response was made and trials in which the first response was to neither the T1 or T2 quadrant (<3% for both groups), then calculating the probability that the first response was to T1. RTs to T1 on step trials (469ms for Group A; 435 ms for Group B) were faster than RTs on no-step trials, (both ps $< .001$). This would be expected from an independent race between Go1 and a stop process, initiated by either T1 offset or T2 onset (Logan & Cowan, 1984).

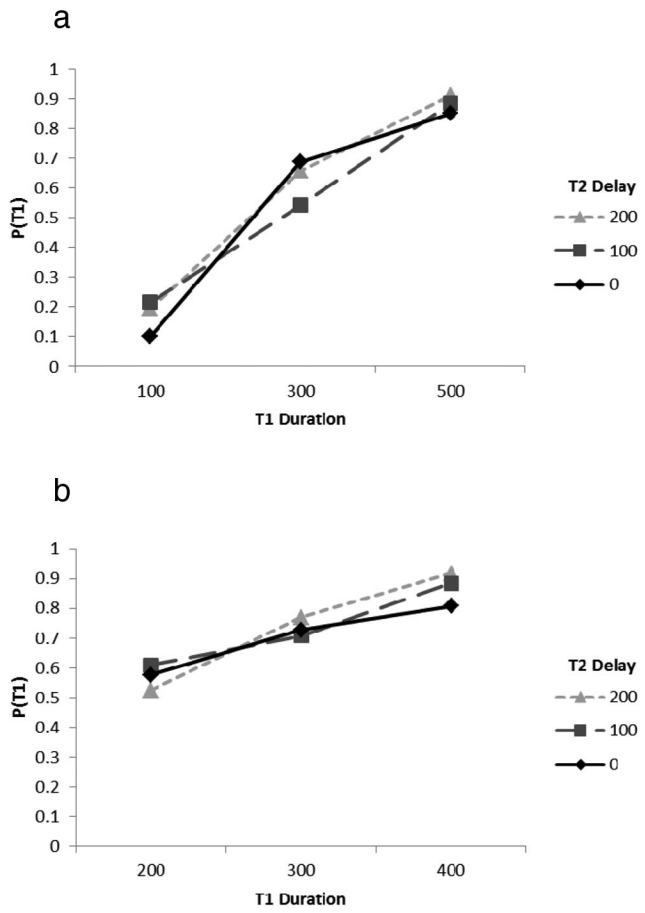

Figure 2. Experiment 1 Group A (a) and B (b) probability of the first response going to T1 (P[T1]) across T1 duration and T2 delay.

To determine whether search–step performance required a separate stop process, we examined the effects of T1 duration and T2 delay on P(T1) (see Figure 2) with separate 3 (T1 duration: 100, 300, and 500 ms in Group A; 200, 300, and 400 ms in Group B) × 3 (T2 delay: 0, 100, and 200 ms) within-subject analyses of variance (ANOVAs). There was a main effect of T1 duration on P(T1) for both Group A, $F(2, 46) = 167.77$, $MSE = .056$, $p < .001$, and Group B, $F(2, 46) = 33.23$, $MSE = .049$, $p < .001$. Using a $p = .05$, the Fisher's LSD for T1 duration were .08 in Group A and .07 in Group B, so all pairwise differences in P(T1) were significant (Group A Means: .20, .75, and .96; Group B Means: .57, .73, and .87). This suggests that the offset of T1 acted as a potent stop signal.

T2 delay also had a significant effect on P(T1) for Group A, $F(2, 46) = 8.58$, $MSE = .0047$, $p < .001$, and for Group B, $F(2, 46) = 4.84$, $MSE = .0052$, $p < .05$. Fisher's LSD for T2 delay was .02 in both Group A and Group B. For Group A, P(T1) at 200 ms T2 delay ($M = .59$) differed from P(T1) at 100 ms ($M = .55$) and at 0 ($M = .54$), but 100 ms and 0 did not differ. For Group B, P(T1) at 0 T2 delay ($M = .70$) differed from P(T1) at 100 ms ($M = .73$) and at 200 ms ($M = .74$), but 100 ms and 200 ms did not differ.

To test whether T1 duration or T2 delay had a larger effect on P(T1), we computed the change in P(T1) separately across T1 duration and T2 delay (i.e., linear regression slope showing change in probability over change in time in ms). We showed that P(T1) increased much more with T1 duration than it did with T2 delay for both Group A (mean regression slope = .0018 vs. .0002), $t(23) = 11.67$, $p < .001$ and Group B (mean regression slope = .0015 vs. .0002), $t(23) = 5.60$, $p < .001$. The large increase in P(T1) with T1 duration and the small, inconsistent increase in P(T1) with T2 delay suggests that T1 offset is the primary determinant of inhibiting responses to T1, but T2 may play a secondary role. We also observed a significant interaction of T1 duration and T2 delay in Group A, $F(4, 92) = 19.34$, $MSE = .0054$, $p < .001$ and Group B, $F(4, 92) = 13.45$, $MSE = .0044$, $p < .001$. We will discuss this interaction in the Discussion.

Experiment 2

Method

Subjects. Three naïve subjects were recruited from the Nashville community and were compensated $12 for 11 1-hr sessions ($132 total). All subjects had normal or corrected-to-normal vision.

Apparatus and stimuli. The apparatus matched Experiment 1 with the following exceptions: The experiment was run on a PC running E-Prime 1 (pstnet.com) connected to a PC running the Eyelink 2000 (sr-research.com). The red and green squares were luminance matched at 30 cd/m^2, and the white background was 80 cd/m^2. Saccades were registered if above a velocity threshold of 30°/s (and remained above the threshold for 4ms) or an acceleration threshold of 8000°/s/s. The minimum motion threshold was .1°.

Procedure. The procedure matched Experiment 1 with the following exceptions: Subjects pressed the spacebar to begin each trial, which initiated drift correction and began a 300-ms fixation period before the target array appeared for 1000 ms, followed by the 850-ms blank-screen ITI.

T1 duration and T2 delays were tailored to each subject based upon their Session 1 and 2 no-step RT, to sample a wide range of P(T1). T1 durations were 15%, 30%, and 45% of no-step RT, and T2 delays were 0, 15%, and 30% (see Figure 3 for subject-specific values), rounded to the nearest monitor refresh (100 Hz refresh rate). The results include the final 10 sessions for subject 2 and the final 9 sessions for subjects 1 and 3, as these have the same timing parameters within subjects.

Subjects were instructed to look promptly at T1, but to look directly at T2 on step trials. Subjects were instructed to look at T2 even if they looked at T1 first on step trials. After instructions, subjects were given 24 trials of practice. The main task included 5 blocks of 96 trials per session. At the end of each block, subjects were given rest but no feedback.

Results and Discussion

Mean correct no-step RTs were stable across sessions (see Figure 4), with a mean of 193 ms across subjects. To assess saccade accuracy, we assigned each saccade to the nearest target square. We then removed trials in which the end point of the first saccade was less than .5 or more than 1.5 the distance from central fixation to target (1.6% of trials), drift correction failed (.7% of

trials), or the end point of the first saccade was closest to a distractor (.6% of trial). For each subject, step-trial RTs to T1 were faster than no-step RTs (S1 218 vs. 208 ms, S2 173 vs. 170 ms, S3 189 vs. 180 ms, all $ps < .001$).

We examined the effect of T1 duration and T2 delay on P(T1) with a 3 (T1 duration: short, intermediate, long) × 3 (T2 delay: 0, short, long) within-subject ANOVA. There were significant main effects of both T1 duration $F(2, 4) = 74.15$, $MSE = .017$, $p < .001$, and T2 delay, $F(2, 4) = 25.01$, $MSE = .005$, $p < .01$, but no interaction, $F(4, 8) < 1$.[1] The two main effects replicate Experiment 1, suggesting that both T1 offset and T2 onset improve step performance. The data from each subject are presented in Figure 3.

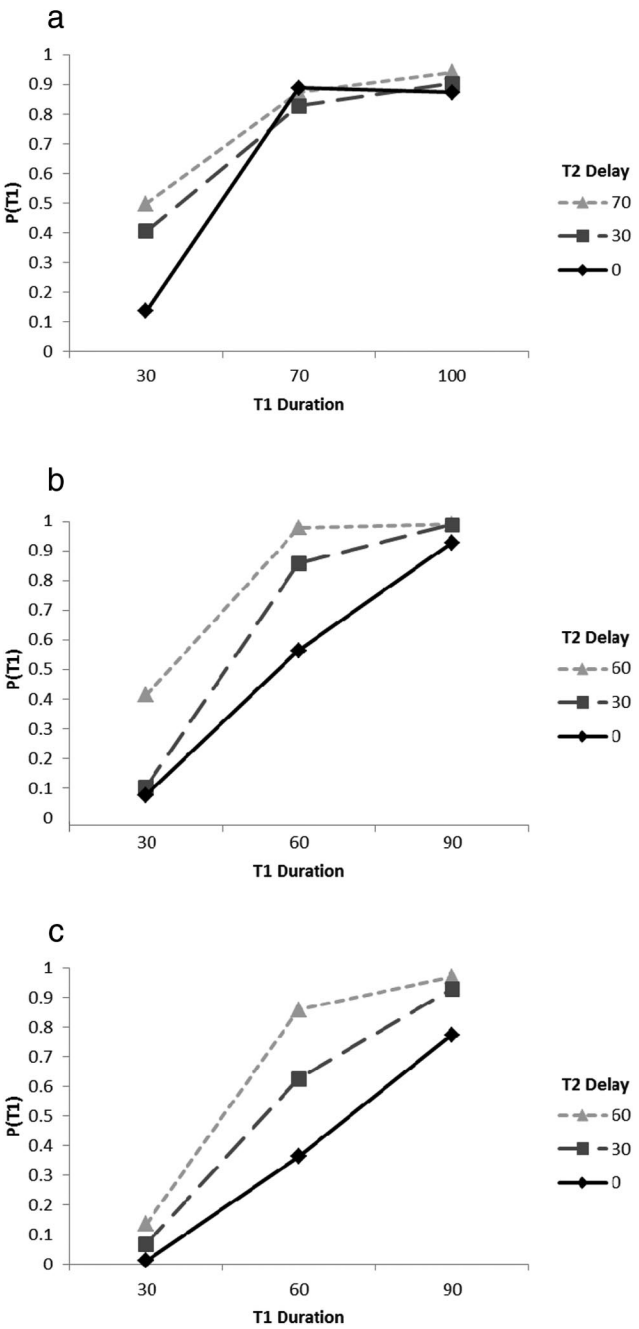

Figure 3. Experiment 2 subject 1 (a), 2 (b), and 3 (c) probability of the first saccade going to T1 (P[T1]) across T1 duration and T2 delay.

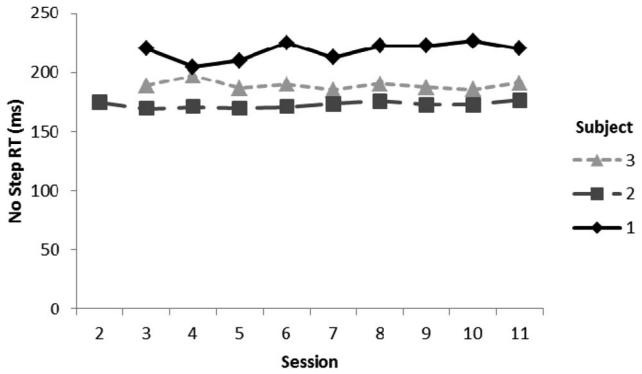

Figure 4. Experiment 2 no-step trial RT separated by subject and by session.

Fisher's LSD for T1 delay was .17, so the short T1 duration ($M = .21$) differed from the intermediate ($M = .76$) and long ($M = .92$) T1 durations, but intermediate and long T1 duration were not quite significantly different. This null pairwise effect may be the result of both intermediate and long T1 durations being at ceiling in certain subjects and conditions (e.g., Subject 1). Fisher's LSD for T2 delay was .09, so all pairwise differences in P(T1) were significant (M 0 = .51, M short = .63, M long = .74). These results suggest that both T1 offset and T2 onset affected stopping ability.

We computed the linear regression of P(T1) against T1 duration and T2 delay, as in Experiment 1. The slope for P(T1) versus T1 duration was larger ($M = .012$) than the slope for P(T1) versus T2 delay ($M = .004$), $t(2) = 2.76$, $p = .01$. As in Experiment 1, increasing T1 duration had a larger effect on P(T1) than increasing T2 delay. Thus, T1 offset was the primary stop signal, but T2 onset also contributed to stopping T1.

General Discussion

We asked whether search–step performance involves a separate stop process by manipulating T1 duration and T2 delay, decoupling T1 offset from T2 onset (Verbruggen et al., 2008). If T1 offset initiates a separate stop process, then T1 duration should be the primary determinant of P(T1). If the Go2 response is sufficient to inhibit the Go1 response, then T2 onset should be the primary determinant of P(T1). Our results suggested T1 duration was the primary determinant of P(T1). In both manual search step (Experiment 1) and saccadic search–step (Experiment 2), T1 duration had stronger effects on P(T1) than T2 onset. These results suggest that a separate stop process is necessary to explain search–step performance.

This work attempts to form a bridge between behavioral paradigms using hands to examine stopping and changing plans, like the stop–signal task (Logan & Cowan, 1984) and the stop-change

[1] We also ran ANOVAs for each subject separately, with each trial as an independent sample within each subject. This led to similar conclusions, with large main effects of T1 duration and T2 delay in each subject.

task (Logan & Burkell, 1986), and paradigms using eyes to examine stopping and changing plans, like the double-step and search–step tasks (Camalier et al., 2007). The same conclusions apply to both effector systems: When changing plans, a separate stop process inhibits the first movement, allowing the second movement to proceed. This suggests that the control signal to both the musculoskeletal system and the oculomotor system may come from the same source. Human neuroscientific research suggests that this source may be a fronto-basal ganglia network (Aron, Behrens, Smith, Frank, & Poldrack, 2007; Chambers, Garavan, & Bellgrove, 2009; Stinear, Coxon, & Byblow, 2009), possibly including the right inferior frontal gyrus (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Swann et al., 2009), the supplementary motor area (Stuphorn & Schall, 2006; Sharp et al., 2010), and the basal ganglia, including the striatum (Vink et al., 2005) and the subthalamic nucleus (Aron & Poldrack, 2006).

The effect of T2 delay on P(T1) was smaller than the effect of T1 duration on P(T1), but T2 delay did affect P(T1) in both experiments. T2 onset may have acted as a second stop signal, speeding inhibition, and decreasing P(T1). Cavina-Pratesi, Bricolo, Prior, and Marzi (2001) showed that a stop signal made of two dots produced faster stop–signal RT than a stop signal made of one dot. Alternatively, the Go2 process may also inhibit Go1. Two sources of inhibition, one from T1 offset and a second from T2 onset, could result in smaller P(T1) than one source. Distinguishing these explanations would require a model that is beyond the scope of this study.

The effect of T2 delay appeared to be larger with eyes than with hands. The slopes comparing P(T1) with T1 duration and T2 delay were larger with eyes than hands because eye RTs are faster, so the slopes cannot be compared directly across Experiment 1 and 2. To account for the larger slopes in eyes, we compared the slopes proportionally, dividing the T2 delay slope by the T1 duration slope. In Experiment 1 with hands, these ratios were .12 and .11 in Groups A and B, respectively. In Experiment 2 with eyes, this ratio was .32. This suggests that T2 delay influenced stopping eye movements more than stopping hand movements. This greater effect of T2 delay may arise from constraints in eye movements. The eyes can only be in one location at a time, but each finger can initiate a different response. This may produce greater competition between alternative eye movements, resulting in greater inhibition from new commands (e.g., Go2) on older commands (e.g., Go1).

We observed an interaction of T1 duration and T2 delay on P(T1) in Experiment 1. The pattern of results is not consistent across groups, and we do not have an explanation for it, but the pattern of results does not compromise our main conclusions. If we reanalyze Experiment 2 using ANOVA within each subject, making use of the power of the many observations across sessions, the interaction is present in each of the three subjects (all $ps < .01$). The interaction in Experiment 2 appears to arise from ceiling and floor effects. When P(T1) is off ceiling ($<.8$) and floor ($>.2$), it increases consistently with T1 duration and T2 delay. This suggests that the interaction may not be present if we chose a small range of T1 durations and T2 delays that did not produce P(T1) values at ceiling or floor.

A reviewer suggested that subjects may delay their T1 response by a fixed amount on every trial to decrease the P(T1) on step trials. This is possible, and increases in RT when signals to stop or change are introduced (Chikazoe et al., 2009), are cued as relevant (Verbruggen & Logan, 2009), or increase in probability (Bissett & Logan, 2011; Logan & Burkell, 1986) are well documented. Delays in RT do not compromise the race model (Logan & Cowan, 1984). The race model addresses the finishing times of the stop and go processes, and the logic is the same whether or not the go process is prolonged by a delay in RT. If the T1 response is delayed long enough, P(T1) could become zero across all T1 durations or T2 delays, which would undermine our analysis of the effect of T1 duration and T2 delay on P(T1). We chose T1 durations and T2 delays to minimize this possibility, and our results show that we succeeded. P(T1) varied greatly with T1 duration and T2 delay (see Figures 2 and 3). Thus, delayed T1 responses do not undermine our conclusions.

Conclusions

We tested the necessity of an independent stop process in the search–step task by decoupling the offset of T1 from the onset of T2, separating the stop signal from the second go signal (Verbruggen et al., 2008). We found that T1 offset had a strong effect on P(T1) for both hands and eyes, suggesting that search–step performance involves a separate stop process as well as separate go processes responding to T1 and T2. T2 onset also improved step performance, suggesting that it may act as a secondary stop signal or that the Go2 response it instigates may inhibit Go1. These conclusions corroborate conclusions drawn from computational modeling (Camalier et al., 2007; Ramakrishnan et al., 2012) and show the power of experimental manipulations for distinguishing between models.

References

Aron, A. R., Behrens, T. E., Smith, S., Frank, M. J., & Poldrack, R. A. (2007). Triangulating a cognitive control network using diffusion-weighted magnetic resonance imaging (MRI) and functional MRI. *The Journal of Neuroscience, 27,* 3743–3752. doi:10.1523/JNEUROSCI.0519-07.2007

Aron, A. R., Fletcher, P. C., Bullmore, E. T., Sahakian, B. J., & Robbins, T. W. (2003). Stop–signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature Neuroscience, 6,* 115–116. doi:10.1038/nn1003

Aron, A. R., & Poldrack, R. A. (2006). Cortical and subcortical contributions to stop signal response inhibition: Role of the subthalamic nucleus. *The Journal of Neuroscience, 26,* 2424–2433. doi:10.1523/JNEUROSCI.4682-05.2006

Becker, W., & Jürgens, R. (1979). An analysis of the saccadic system by means of double step stimuli. *Vision Research, 19,* 967–983. doi:10.1016/0042-6989(79)90222-0

Bissett, P. G., & Logan, G. D. (2011). Balancing cognitive demands: Control adjustments in the stop–signal paradigm. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 37,* 392–404. doi:10.1037/a0021800

Boucher, L., Palmeri, T. J., Logan, G. D., & Schall, J. D. (2007). Inhibitory control in mind and brain: An interactive race model of countermanding saccades. *Psychological Review, 114,* 376–397. doi:10.1037/0033-295X.114.2.376

Camalier, C. R., Gotler, A., Murthy, A., Thompson, K. G., Logan, G. D., Palmeri, T. J., & Schall, J. D. (2007). Dynamics of saccade target selection: Race model analysis of double step and search step saccade production in human and macaque. *Vision Research, 47,* 2187–2211. doi:10.1016/j.visres.2007.04.021

Cavina-Pratesi, C., Bricolo, E., Prior, M., & Marzi, C. A. (2001). Redundancy gain in the stop–signal paradigm: Implications for the locus of

coactivation in simple reaction time. *Journal of Experimental Psychology: Human Perception and Performance, 27,* 932–941. doi:10.1037/0096-1523.27.4.932

Chambers, C. D., Garavan, H., & Bellgrove, M. A. (2009). Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neuroscience and Biobehavioral Reviews, 33,* 631–646.

Chikazoe, J., Jimura, K., Hirose, S., Yamashita, K-I., Miyashita, Y., & Konishi, S. (2009). Preparation to inhibit a response complements response inhibition during performance of a stop–signal task. *The Journal of Neuroscience, 29,* 15870–15877. doi:10.1523/JNEUROSCI.3645-09.2009

Hanes, D. P., & Schall, J. D. (1995). Countermanding saccades in macaque. *Visual Neuroscience, 12,* 929–937. doi:10.1017/S0952523800009482

Logan, G. D., & Burkell, J. (1986). Dependence and independence in responding to double stimulation: A comparison of stop, change, and dual-task paradigms. *Journal of Experimental Psychology: Human Perception and Performance, 12,* 549–563. doi:10.1037/0096-1523.12.4.549

Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review, 91,* 295–327. doi:10.1037/0033-295X.91.3.295

Murthy, A., Ray, S., Shorter, S. M., Schall, J. D., & Thompson, K. G. (2009). Neural control of visual search by frontal eye field: Effects of unexpected target displacement on visual selection and saccade preparation. *Journal of Neurophysiology, 101,* 2485–2506. doi:10.1152/jn.90824.2008

Murthy, A., Thompson, K. G., & Schall, J. D. (2001). Dynamic dissociation of visual selection from saccade programming in frontal eye field. *Journal of Neurophysiology, 86,* 2634–2637.

Ramakrishnan, A., Sureshbabu, R., & Murthy, A. (2012). Understanding how the brain changes its mind: Microstimulation in the macaque frontal eye field reveals how saccade plans are changed. *The Journal of Neuroscience, 32,* 4457–4472. doi:10.1523/JNEUROSCI.3668-11.2012

Sharp, D. J., Bonnelle, V., De Boissezon, X., Beckmann, C. F., James, S. G., Patel, M. C., & Mehta, M. A. (2010). Distinct frontal system for response inhibition, attentional capture, and error processing. *Proceedings of the National Academy of Sciences, USA, 93,* 8694–8698.

Stinear, C. M., Coxon, J. P., & Byblow, W. D. (2009). Primary motor cortex and movement preparation: Where stop meets go. *Neuroscience and Biobehavioral Reviews, 33,* 662–673. doi:10.1016/j.neubiorev.2008.08.013

Stuphorn, V., & Schall, J. D. (2006). Executive control of countermanding saccades by the supplementary eye field. *Nature Neuroscience, 9,* 925–931. doi:10.1038/nn1714

Swann, N., Tandon, N., Canolty, R., Ellmore, T. M., McEvoy, L. K., Dreyer, S., . . . Aron, A. R. (2009). Intracranial EEG reveals a time- and frequency-specific role for the right inferior frontal gyrus and primary motor cortex in stopping initiated responses. *The Journal of Neuroscience, 29,* 12675–12685. doi:10.1523/JNEUROSCI.3359-09.2009

Verbruggen, F., & Logan, G. D. (2009). Proactive adjustments of response strategies in the stop–signal paradigm. *Journal of Experimental Psychology: Human Perception and Performance, 35,* 835–854. doi:10.1037/a0012726

Verbruggen, F., Schneider, D. W., & Logan, G. D. (2008). How to stop and change a response: The role of goal activation in multitasking. *Journal of Experimental Psychology: Human Perception and Performance, 34,* 1212–1228. doi:10.1037/0096-1523.34.5.1212

Vink, M., Kahn, R. S., Raemaekers, M., van den Heuvel, M., Boersma, M., & Ramsey, N. F. (2005). Function of striatum beyond inhibition and executive of motor responses. *Human Brain Mapping, 25,* 336–344. doi:10.1002/hbm.20111

Received June 25, 2012
Revision received March 22, 2013
Accepted April 12, 2013 ■