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How to Stop and Change a Response: The Role of Goal Activation in Multitasking

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Multitasking was studied in the stop-change paradigm, in which the response for a primary GO1 task had to be stopped and replaced by a response for a secondary GO2 task on some trials. In 2 experiments, the delay between the stop signal and the change signal was manipulated to determine which task goals (GO1, GO2, or STOP) were involved in performance and to determine whether the goals were activated in series or in parallel. As the delay increased, the probability of responding on stop trials changed very little, but GO2 task reaction times decreased substantially. Such effects are consistent with both a nondeterministic serial model (in which the GO1 goal is replaced by the STOP goal, which is subsequently replaced by the GO2 goal) and a limited-capacity parallel model (in which stopping and GO2 processing occur concurrently) with a capacity-sharing proportion that resembles serial processing.

Keywords: response inhibition, executive control, goals, stop-signal paradigm, dual-task performance

Multitasking is a common psychological phenomenon in modern life. Every day, people switch from one task to another in response to changes in internal states or changes in the environment. Cognitive scientists have investigated multitasking in a variety of experimental paradigms, ranging from task-switching to dual-task procedures, to understand the executive control processes that underlie it (Logan, 1985a; Monsell, 1996). Each paradigm requires subjects to switch from one task to another, and theoretical analyses suggest that subjects do so by manipulating the goal representations that drive the subordinate processes involved in responding to the environment (Logan & Cowan, 1984; Logan & Gordon, 2001; Meyer & Kieras, 1997a, 1997b; Miller & Cohen, 2001). An important difference between the paradigms is the timing of the stimuli for the two tasks and the consequent temporal overlap in the underlying processes. Task-switching procedures (Jersild, 1927) present one stimulus on each trial and require switching tasks between trials, with several hundred milliseconds elapsing between the response to one stimulus and the appearance of the next. Thus, the processes for one task are finished before the processes for the next task begin. By contrast, dual-task procedures, including the psychological refractory period (PRP) paradigm (Telford, 1931; Welford, 1952), the stop-signal paradigm (Lappin & Eriksen, 1966; Logan & Cowan, 1984; Vince, 1948), and the stop-change paradigm (Logan, 1983; Logan & Burkell, 1986), present two stimuli on one trial at intervals so brief that the second stimulus often appears before the response to the first one is finished. This creates ample opportunity for temporal overlap in the underlying processes and raises the question of whether the processes are active in series or in parallel. The present study investigated multitasking in the stop-change paradigm, asking which goals are active when subjects must stop performing one task and change to another and whether the goals are active in series or in parallel.

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Multitask Paradigms: Processes and Task Goals

The PRP Paradigm

Dual-task performance is often studied in the PRP paradigm (for reviews, see Pashler, 1994; Welford, 1952). In this paradigm, two stimuli (S1 and S2) are presented in rapid succession on each trial and subjects are instructed to respond to each stimulus as quickly as possible. There are two overlapping processes on each trial: the GO1 process triggered by the presentation of S1 and the GO2 process triggered by the presentation of S2. The common finding is that GO2 processing is delayed when the delay between S1 and S2 is short, whereas GO1 processing is not influenced. This dual-task interference effect has been explained in terms of a structural response-selection bottleneck (Pashler, 1994), capacity sharing between the tasks (Navon & Miller, 2002; Tombu & Jolicœur, 2003), or strategic deferment of the second task (Logan & Gordon, 2001; Meyer & Kieras, 1997b).

Many theorists assume that the PRP paradigm involves two goals: the GO1 goal associated with Task 1 and the GO2 goal associated with Task 2. Whether the two goals can be activated at the same time is still debated, although most theories assume that serial processing occurs at least at certain stages (see, e.g., Byrne & Anderson, 2001; Logan & Gordon, 2001; Meyer & Kieras, 1997b; Pashler, 1994).

The Stop-Signal Paradigm

The stop-signal paradigm is a popular tool for investigating response inhibition (for reviews, see Boucher, Palmeri, Logan, & Schall, 2007; Logan, 1994). In this paradigm, subjects usually perform a choice reaction task (hereafter referred to as the *GO1 task*). On a random selection of the trials, a stop signal is presented that instructs subjects to withhold their response. To account for performance on stop-signal trials, Logan and Cowan (1984) proposed a horse-race model that assumes that two processes race against each other: a GO1 process that is triggered by the presentation of the GO1 stimulus and a STOP process that is triggered by the presentation of the stop signal. If the STOP process finishes before the GO1 process, then subjects inhibit their response (*signal-inhibit* trials). If the GO1 process finishes before the STOP process, then subjects fail to inhibit their response (*signal-respond* trials).

The race model assumes that two task goals are involved: a GO1 goal and a STOP goal. When a stop signal is presented, the GO1 goal is replaced by a STOP goal, and responses are suppressed by countermanding the orders to the subordinate systems. With no orders to support performance, processing in the subordinate systems stops relatively quickly, often within 200–300 ms of stop-signal presentation (Logan & Cowan, 1984).

The Stop-Change Paradigm

The stop-change paradigm (Logan, 1982, 1983) represents a "procedural bridge" between the PRP paradigm and the stop-signal paradigm (Logan & Burkell, 1986, p. 549). The paradigm is similar to the standard stop-signal paradigm in that subjects are instructed to stop their response for the GO1 task whenever a stop-change signal is presented. It is similar to the PRP paradigm in that subjects have to make a response for a secondary task (the GO2 task). These similarities raise the first question that was addressed in the present study: Does the stop-change paradigm require three goals or two? The three-goal account assumes that the stop-change paradigm requires all of the goals involved in the stop-signal and PRP paradigms: a GO1 goal, a STOP goal, and a GO2 goal. Subjects begin each trial by activating the GO1 goal. On stop-change trials they activate the STOP goal to stop the GO1 response and the GO2 goal to enable the GO2 response. The two-goal account assumes that the stop-change paradigm requires only the goals involved in the PRP paradigm: a GO1 goal and a GO2 goal. Subjects begin each trial by activating the GO1 goal. On stop-change trials, they activate the GO2 goal, which stops the GO1 response and enables the GO2 response. If the three-goal account is necessary to account for the results (and it is), then that raises the second question that was addressed in the present study: Are the STOP and GO2 goals activated in series or in parallel? That is, do subjects stop the GO1 response before they begin to respond to the GO2 stimulus, or do they begin responding to the GO2 stimulus as they are stopping their response to the GO1 stimulus?

Recently, Camalier et al. (2007) conducted a study that bears on these issues in a task that is similar to the stop-change paradigm. In their study, subjects had to make an eye movement to a target, but on some trials, the location of the target changed before the initial eye movement was made and subjects had to make an eye movement to the new location. Camalier et al. distinguished between three models of task performance. The GO-GO model assumes a race between two GO processes: the GO1 process, triggered by the presentation of the initial target, and the GO2 process, triggered by the changed target (i.e., a two-goal account). The GO-STOP-GO model assumes that a STOP process cancels GO1 processing, and GO2 processing starts when the STOP process has finished (i.e., a three-goal account in which STOP and GO2 processing occur serially). The GO-GO+STOP model also assumes that a STOP process cancels GO1 processing, but GO2 processing starts together with the STOP process (i.e., a three-goal account in which STOP and GO2 processing occur in parallel). Camalier et al. fit the three models to the data of both humans and macaque monkeys. The three-goal models that included a STOP process fit the data better than the two-goal model without it. However, the serial and parallel STOP models fit the data equally well, and the experiment included no manipulation that discriminated between the two STOP models.[1]

The results of Camalier et al. (2007) suggest that a three-goal account involving a STOP goal is needed to cancel and replace an eye movement. However, Logan and Irwin (2000) and Boucher, Stuphorn, Logan, Schall, and Palmeri (2007) found important differences between stopping eye movements and hand movements, so it is not clear whether the conclusions of Camalier et al. generalize to stopping and changing hand movements, which are more commonly used in PRP, stop-signal, and stop-change paradigms. Therefore, in the present study, we asked whether a three-goal account involving a STOP goal is also needed to stop manual movements in the stop-change paradigm, testing the GO-GO, GO-STOP-GO, and GO-GO+STOP models proposed by Camalier et al. More importantly, our study went beyond that of Camalier et al. by including a manipulation of the delay between the stop signal and the change signal that allowed us to distinguish more clearly between the different models.

The Present Study

We conducted two experiments using variations of the stop-change paradigm. In both experiments, the GO1 task involved judging whether a target (a filled circle) appeared above or below a reference point (see Figure 1). The visual stop signal was a change in the color of a rectangle surrounding the display (from white to red) and the auditory change signal was the word *high*, *middle*, or *low*. We manipulated the delay between the stop signal and the change signal (*stop-change delay*; SCD) to distinguish between different models of stop-change performance. In Experiment 1, the GO2 task involved making a new judgment about the position of the target with respect to a new reference point specified by the change signal (see Figure 1A). In Experiment 2, the GO2 task involved making a separate response to the change signal, reporting its identity with a keypress.

[1] Camalier et al. (2007) distinguished between the GO-STOP-GO and GO-GO+STOP models on plausibility grounds. They observed that the predicted GO2 RTs were much faster than the predicted GO1 RTs for the GO-STOP-GO model, and they argued that there is no reason to assume why this would be the case. Because of this, Camalier et al. claimed that the GO-GO+STOP model offered a better account of their data.

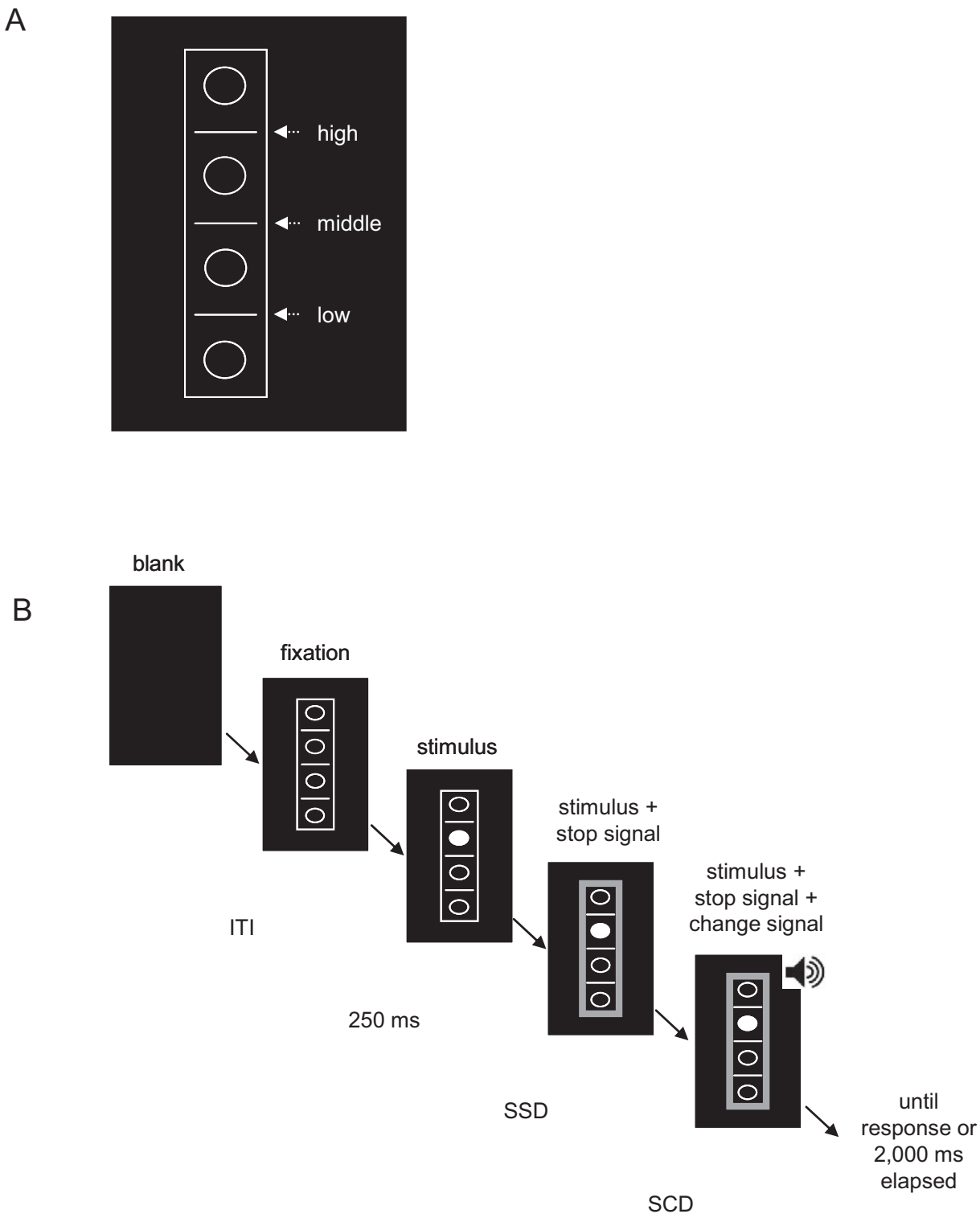

Figure 1. A: The stimulus display. Circles represent possible target locations, and lines represent reference points. The words on the right indicate the mapping of the auditory change signals to the reference points. The words did not appear onscreen during the experiment. B: The sequence of events (depicted from left to right) of a stop-change trial. The red stop signal used in the experiment is represented by a thick gray stop signal. The auditory change signal used in the experiment is represented by a speaker icon. ITI = intertrial interval; SSD = stop-signal delay; SCD = stop-change delay.

We tested five different models of stop-change performance; the models and their respective predictions (discussed below) are depicted in Figure 2. The models are built around three questions. First, is there a STOP goal involved in cancelling the GO1 response in the stop-change paradigm? Second, if there is a STOP goal involved, are the STOP and GO2 goals activated in series or in parallel? Third, if the STOP and GO2 goals are activated in parallel, do STOP and GO2 processing share capacity?

Is a STOP Goal Involved?

To address the first question, we distinguished between a (two-goal) GO1-GO2 model (see Figure 2, Model 1) and four (three-goal) STOP models. The GO1-GO2 model assumes that only two GO goals are involved in the stop-change paradigm (a GO1 goal and a GO2 goal) and that stopping and changing a response is accomplished by replacing the GO1 goal with the GO2 goal. By contrast, the STOP models assume that three goals are involved: a GO1 goal, a STOP goal, and a GO2 goal. According to these models, stopping and changing a response is accomplished by activating the STOP goal to cancel the GO1 response and activating the GO2 goal to enable the GO2 response.

Are STOP and GO2 Goals Activated in Series or in Parallel?

To address the second question, we distinguished between two serial versions and two parallel versions of the three-goal model. The serial models (GO1-STOP-GO2; see Figure 2, Models 2 and 3) assume that GO2 processing starts after STOP processing finishes, whereas the parallel models (GO1-GO2+STOP; see Figure 2, Models 4 and 5) assume that GO2 processing starts when STOP processing starts.

Do STOP and GO2 Processing Share Capacity?

To address the third question, we distinguished between two parallel models: a GO1-GO2+STOP model in which the STOP and GO2 processes do not share capacity (see Figure 2, Model 4) and a GO1-GO2+STOP model in which the STOP and GO2 processes do share capacity (see Figure 2, Model 5).

Models of Stop-Change Performance

GO1-GO2 Model

The GO1-GO2 model (see Figure 2, Model 1) assumes that two goals are sufficient for stop-change performance, just as two goals are sufficient for stop-signal and PRP performance. Subjects simply replace the GO1 goal with the GO2 goal. This would cancel support for the subordinate processes underlying the GO1 task and result in the inhibition of the GO1 response (Logan & Cowan, 1984). In other words, the GO1-GO2 model assumes that stopping and changing a response can be accomplished by a single act of control.

The GO1-GO2 model predicts that the probability of making the GO1 response given a stop-change signal, *p*(respond|signal), will be influenced by SCD, as depicted in Figure 3. Given stop-signal delay (SSD) and stop-signal reaction time (SSRT; this is the covert latency of the STOP process), *p*(respond|signal) is equal to the area to the left of Line A (SSD + SSRT) when SCD = 0 ms. When SCD > 0 ms and stopping is delayed until the change signal is presented (i.e., until GO2 processing can start), *p*(respond|signal) is equal to the area to the left of Line B (SSD + SCD + SSRT). Thus, *p*(respond|signal) will increase as SCD increases. However, the GO1-GO2 model predicts that GO2 RTs (i.e., the time interval between the change signal and the registration of the GO2 response) will not vary with SCD because GO2 processing starts when the change signal is presented. Note that the GO1-GO2 model assumes that subjects strategically defer the STOP process until all task information is available so that stopping and GO2 processing can be accomplished in a single act.

GO1-STOP-GO2 Model

The serial GO1-STOP-GO2 models (see Figure 2, Models 2 and 3) assumes that three goals are required for stop-change performance (GO1, STOP, and GO2 goals) but the STOP and GO2 goals are not active at the same time (i.e., they are active in series, not in parallel). These models predict that *p*(respond|signal) will not be influenced by SCD because stopping can start as soon as the stop signal is presented. They predict that GO2 RTs will be influenced by SCD because the STOP process has to finish before GO2 processing can start (see Figure 4A). At short SCDs, stopping may not be finished before the change signal is presented, thereby delaying GO2 processing. At longer SCDs, stopping is more likely to be finished before the change signal is presented, so GO2 processing can begin immediately (see Figure 4A). Consequently, the GO1-STOP-GO2 models predict that GO2 RTs will decrease as SCD increases.

The analysis so far has followed the common practice in the PRP literature (e.g., Pashler, 1994) of assuming that the process durations are constant (i.e., that the process durations are deterministic) to make it easier to derive the models' predictions. However, PRP researchers acknowledge that process durations are unlikely to be deterministic, and Schwarz and Ischebeck (2001) showed that stochastic variation in process duration flattens the slope of the function relating RT of the second process to the interval between the first and second stimulus. Following the analyses of Schwarz and Ischebeck, we distinguished between deterministic (see Figure 2, Model 2) and nondeterministic (i.e., stochastic; see Figure 2, Model 3) variants of the GO1-STOP-GO2 model. The deterministic variant assumes that stopping and GO2 processing are strictly deterministic (i.e., there is no variability in SSRT and GO2 RT), whereas the nondeterministic variant assumes that stopping and GO2 processing are stochastic (i.e., there is variability in both SSRT and GO2 RT). As shown in Figure 2, the deterministic GO1-STOP-GO2 model predicts a linear decrease in GO2 RT with a slope of −1, whereas the nondeterministic GO1-STOP-GO2 model predicts a monotonic decrease in GO2 RT with a (local) slope that is less than −1 (Schwarz & Ischebeck, 2001). In the nondeterministic version, the slope reflects the probability that the STOP process has not finished before the change signal is presented. If the STOP process has not finished, the slope is −1; if the STOP process has finished, the slope is 0. Stochastic variation in stopping and GO2 processing will result in a mixture of trials in which the GO1 response is not inhibited before the change signal is presented (which produces a slope of −1) and trials in which the GO1 response is inhibited before the change signal is presented (which produces a slope of 0).

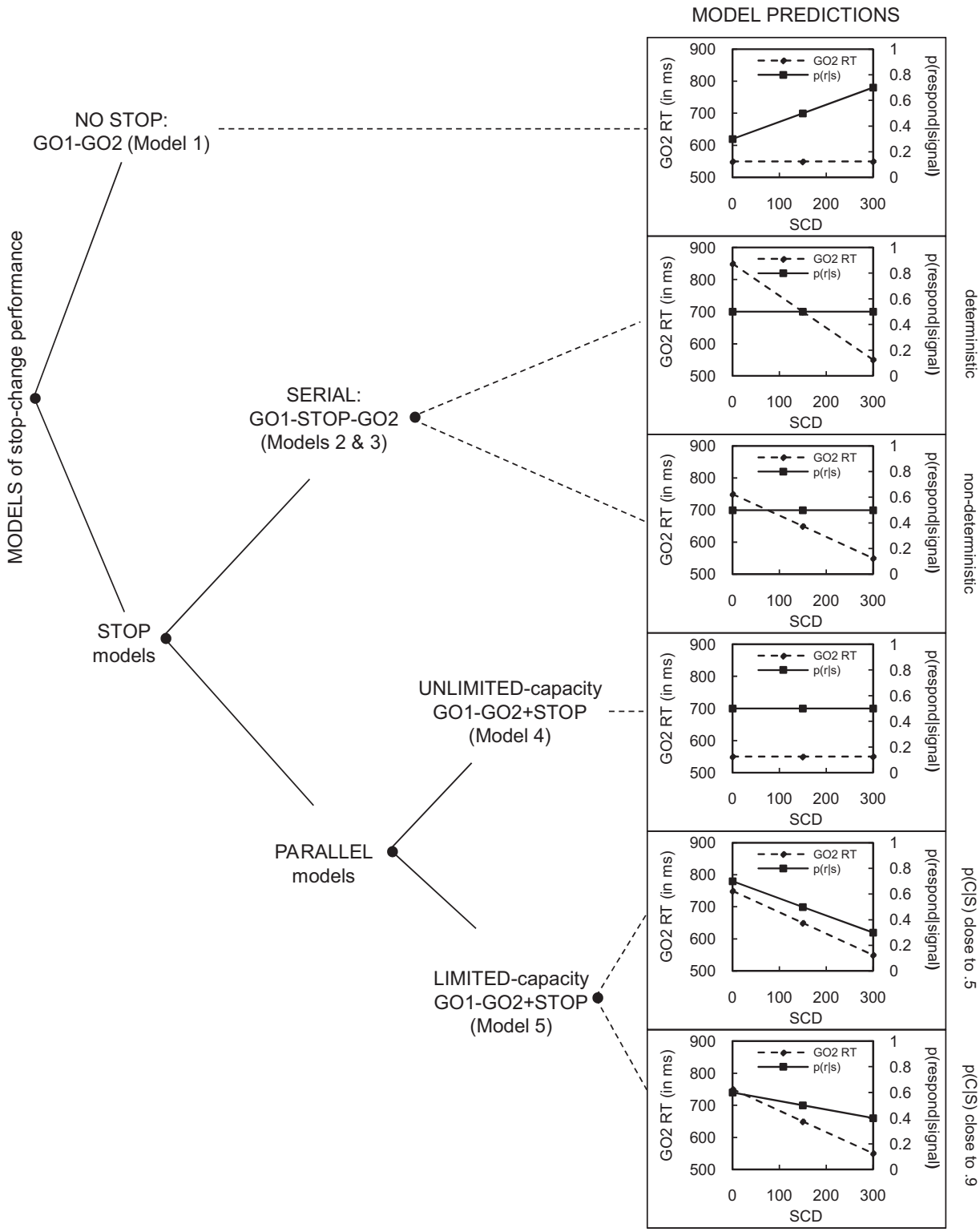

Figure 2. The different models and their predictions for probability of responding, $p(r|s)$, and GO2 reaction time (RT) as a function of stop-change delay (SCD: 0, 150, or 300 ms). For the limited-capacity model, $p(c|s)$ is the proportion of capacity allocated to the STOP process. See text for further details.

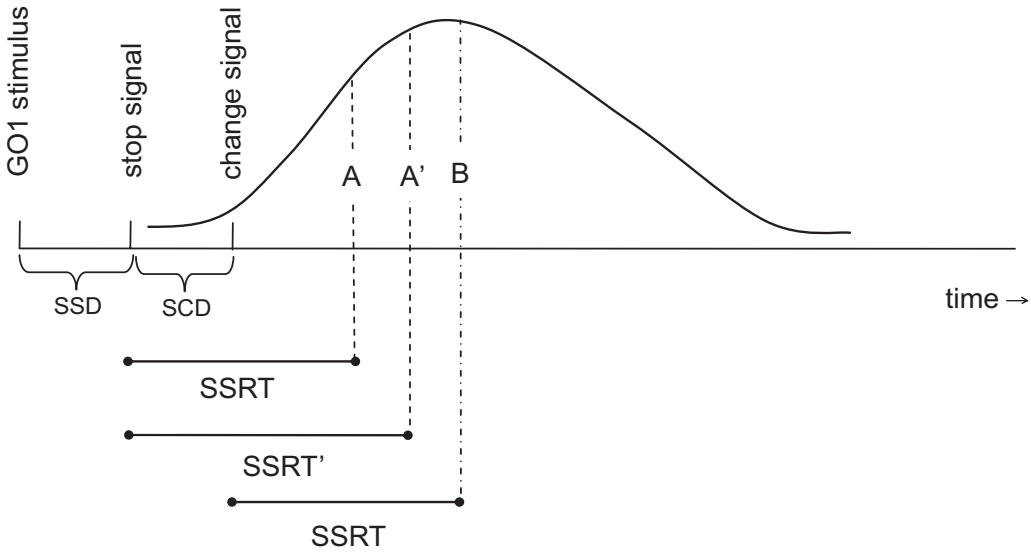

Figure 3. Illustration of the predicted probabilities of responding, *p*(respond|signal), based on the horse-race model (Logan & Cowan, 1984), given the distribution of GO1 reaction times (GO1 RT) and the stop-signal reaction time (SSRT). The *p*(respond|signal) is represented by the area under the curve to the left of each dashed line, which increases if SSRT is prolonged (compare Lines A and A′) or if subjects do not stop until the change signal is presented (compare Lines A and B).

GO1-GO2+STOP Models

The parallel GO1-GO2+STOP models also assume that three goals are required for performance in the stop-change paradigm: a GO1 goal, a GO2 goal, and a STOP goal. However, these models assume that the STOP and GO2 goals can be active in parallel; the STOP goal does not have to stop GO1 processing before GO2 processing can begin. The assumption that STOP and GO2 goals are active in parallel raises the question of whether they share capacity. To answer this question, we distinguished between two GO1-GO2+STOP models. The unlimited-capacity GO1-GO2+STOP model assumes that stopping and GO2 processing occur in parallel and that capacity is unlimited. This model is consistent with the hypothesis that there is no dual-task interference between response inhibition and GO processing (Logan & Burkell, 1986; Logan, Cowan, & Davis, 1984). Both STOP and GO2 processing start when the relevant signal is presented and, therefore, the model predicts that SCD will have no effect on *p*(respond|signal) or GO2 RT (see Figure 2, Model 4).

The limited-capacity GO1-GO2+STOP model (see Figure 2, Model 5) assumes that stopping and GO2 processing occur in parallel, but they share capacity, so both processes will be slowed down (see Figure 4B). When the STOP process is slowed, *p*(respond|signal) will increase. This effect is depicted in Figure 3. The *p*(respond|signal) is the probability that GO1 RT is faster than SSD + SSRT, which can be estimated as the area of the GO1 RT distribution curve to the left of the point representing SSD + SSRT (i.e., the area to the left of Line A in the figure). When capacity sharing prolongs SSRT (resulting in SSRT′), *p*(respond|signal) is higher because a greater proportion of the GO1 RT distribution is less than SSD + SSRT′ (i.e., the area to the left of Line A′ in the figure). Thus, *p*(respond|signal) increases as SSRT increases.

These effects of capacity sharing on the STOP and GO2 processes are depicted in Figure 4B. The limited-capacity GO1-GO2+STOP model predicts that both *p*(respond|signal) and GO2 RTs will increase when stopping and GO2 processing share capacity. When SCD $>$ 0 ms, stopping does not have to share capacity with GO2 processing until the change signal is presented. Therefore, SSRT will decrease when SCD $>$ 0 ms (note how the duration of the STOP process decreases as SCD increases in Figure 4B). However, the effects of capacity sharing on *p*(respond|signal) also depend on the proportion of capacity allocated to the STOP process. The larger the proportion, the shorter SSRT will be (see Figure 4C). The effect of SCD will also be smaller when the proportion of capacity allocated to the STOP process increases. This becomes clear when one compares Figure 4C with Figure 4B. When the proportion is .5, SSRT for SCD = 0 ms is substantially longer than SSRT for SCD = 300 ms (at SCD = 300 ms, all capacity can be allocated to the STOP process). However, when the proportion is .9, SSRT for SCD = 0 ms is only slightly longer than the SSRT for SCD = 300 ms.

The limited-capacity GO1-GO2+STOP model also assumes that GO2 RTs are influenced by SCD. Once stopping has finished, all capacity can be allocated to GO2 processing. At longer SCDs, stopping is more likely to be finished by the time the change signal is presented, such that GO2 processing no longer has to share capacity with the STOP process. Thus, GO2 RT will also decrease with increasing SCD (note how the duration of the GO2 process decreases as SCD increases in Figure 4B).

Whereas GO2 RT is influenced by SCD, it is not influenced by the proportion of capacity allocated to the STOP process (Navon & Miller, 2002; Tombu & Jolicœur, 2003). This can be seen in Figure 4C. In the top panel of Figure 4C, the proportion of capacity allocated to the STOP process is .5, and stopping and GO2 pro-

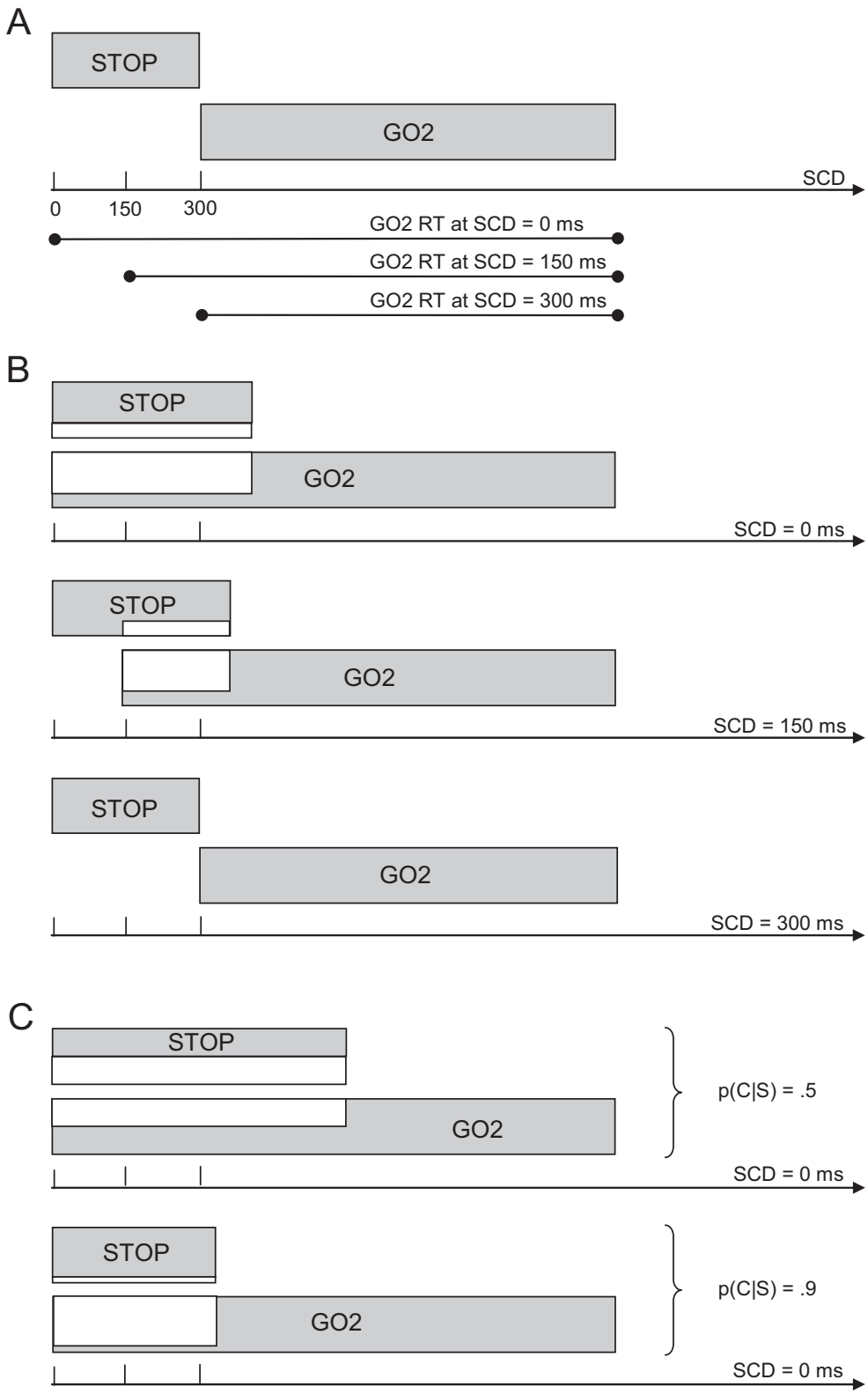

Figure 4. Stage diagrams for the STOP and GO2 processes as a function of stop-change delay (SCD) for the GO-STOP-GO models (A) and the limited-capacity GO-GO+STOP model (B). The length of each box represents the duration of each stage. White areas represent shared capacity. See text for further details. Stage diagrams for the STOP and GO2 processes as a function of the proportion of capacity allocated to the STOP process, *p*(cls), for SCD = 0 ms (C).

cessing have to share capacity until the STOP process is finished. However, when the proportion of capacity allocated to the STOP process is .9 (the bottom panel of Figure 4C), the duration of STOP processing decreases and all capacity can be allocated to GO2 processing sooner. Thus, the sooner the STOP process finishes, the sooner all capacity can be allocated to GO2 processing. As a result, GO2 processing is not influenced by the proportion of capacity allocated to the STOP process (note that the duration of the GO2 process is not influenced by the proportion of capacity allocated to the STOP process in Figure 4C). Because GO2 processing is not influenced by the capacity-sharing ratio, limited-capacity models typically predict the same slopes as serial models (Navon & Miller, 2002). Thus, the slopes predicted by the limited-capacity GO1-GO2+STOP model are similar to the slopes predicted by the GO1-STOP-GO2 models (see Figure 2). At this point, we will assume that slopes between −1 and 0 (i.e., the range of slopes predicted by the nondeterministic GO1-GO2+STOP model) are consistent with the limited-capacity GO1-GO2+STOP model, but we will return to this issue in the General Discussion when we discuss the models in greater detail.

We do not distinguish between deterministic and nondeterministic parallel versions of the GO1-GO2+STOP model. For the unlimited-capacity GO1-GO2+STOP model, the expected slopes are 0 regardless of whether the process durations are deterministic or nondeterministic. Similarly, for the limited-capacity GO1-GO2+STOP model, the expected slopes are smaller than −1 regardless of whether the process durations are deterministic or nondeterministic.

Experiment 1

In Experiment 1, we tested the five models by manipulating SCD and observing whether the resultant patterns of *p*(respond|signal) and GO2 RT were consistent with any of the model predictions in Figure 2. Both the GO1 and GO2 tasks required subjects to respond to the position of a target relative to a reference point. The reference point for the GO1 task was always the middle line in the display (see Figure 1A). The reference point for the GO2 task could be any of the lines, as indicated by the change signal.

We interpret data as consistent with a model's predictions if the observed patterns (i.e., increase, decrease, or no change) of *p*(respond|signal) and GO2 RT across SCD match the predicted patterns. Whether and where the lines for *p*(respond|signal) and GO2 RT cross are not important because each variable is associated with a different *y*-axis in our presentation of the data, and crossover can be manipulated simply by changing the scales.

Method

Subjects. Twenty undergraduate students from Vanderbilt University participated for course credit. All subjects had normal or corrected-to-normal vision and were naive as to the purpose of the experiment. One subject was replaced for having deviant error rates (2.5 *SD*s above the mean) in certain conditions.

Apparatus and stimuli. The experiment was run on a Pentium 4 PC running Tscope (Stevens, Lammertyn, Verbruggen, & Vandierendonck, 2006), and the stimuli were presented on a 21-in. (53.3-cm) cathode ray tube monitor. The GO1 task was to judge whether a white filled circle was above or below a reference point. For the GO1 task, subjects responded by pressing the *P* key (for "above") or the *L* key (for "below") of a QWERTY keyboard with the middle and index fingers of the right hand, respectively (*GO1 responses*). The GO2 task was a new judgment about the position of the target with respect to a new reference point specified by the change signal. For the GO2 task, subjects pressed the *E* key (for "above") or the *F* key (for "below") with the middle and index fingers of the left hand, respectively, on stop-change trials (*GO2 responses*). The trial events and screen setup are depicted in Figure 1. The targets were four vertically arranged circles (8 mm diameter), and the reference points were three horizontal lines (1 × 8 mm). The distance between the edge of a circle and a reference point was 12 mm. All stimuli were presented on a black background and viewed at a distance of about 60 cm. The stimuli and reference points always appeared in a white rectangle (20 × 96 mm, line thickness = 1 mm).

On some trials, the white rectangle was replaced by a red rectangle of the same size (but with line thickness = 2 mm), informing the subjects that they had to stop their response (see Figure 1B). After a variable delay, a loud and clear auditory change signal—indicating the reference point for the GO2 task—was presented through speakers positioned to the left and right of the screen. On the one hand, presenting the two signals in different modalities would reduce the potential for perceptual interference between the signals within the visual system (e.g., from competition for the same resources; Treisman & Davies, 1973). On the other hand, presenting signals in different modalities could incur a cost from shifting attention between modalities (Spence, Nicholls, & Driver, 2001; Turatto, Benso, Galfano, Gamberini, & Umiltà, 2002; but see Alais, Moronne, & Burr, 2006). However, we used familiar words as auditory cues whose conventional meanings specified the relevant reference points (using the same terms we used in the initial instructions), on the hypothesis that this would counteract any costs incurred from switching attention between modalities. Three different words served as change signals: *high*, *middle*, and *low*. These words were recorded in a female voice and subsequently manipulated with Audacity software (Version 1.2; http://audacity.sourceforge.net). After recording, we manipulated the sound files in three steps. First, we applied the built-in noise removal procedure. Second, we manipulated the duration of each word, setting it to 200 ms for all three words. This was done with the procedure for changing tempo without changing pitch. Third, on the basis of visual inspection of the amplitudes, we adjusted the volume of each word such that all three words sounded approximately equally loud. During the experiment, the volume of the words was 80 dB.

Procedure. Instructions were read by the subjects and (if necessary) explained orally by the experimenter. Instructions emphasized both accuracy and speed. All trials began with the presentation of the four unfilled circles and the three reference points in the center of the screen (see Figure 1A). After 250 ms, one of the four circles was filled (the GO1 stimulus) and required a response based on its position. When no stop signal was presented, the reference point was always the middle line and the stimulus required a right-handed GO1 response. The stimulus and reference points remained on the screen until subjects responded or until 2,000 ms had elapsed. The intertrial interval was 500 ms (see Figure 1B).

On one third of the trials, a stop signal (i.e., the red rectangle) was presented. Signal trials could be preceded by no-signal or signal trials. On signal trials, subjects were instructed to stop their right-handed GO1 response and execute a left-handed GO2 response instead. They were told that the reference point could change for the GO2 response and that it was signaled by a word presented through the speakers. This word was presented after a variable SCD: 0, 150, or 300 ms. When the word was *high*, the upper line became the reference point; when the word was *middle*, the middle line remained the reference point; and when the word was *low*, the lower line became the reference point (see Figure 1A). The three words occurred with equal probability.

The SSD was initially set to 250 ms and was continuously adjusted according to a tracking procedure to obtain a probability of stopping of .50. Separate tracking procedures were used for each reference point. Each time a subject responded to the stimulus in the presence of a stop signal, SSD decreased by 50 ms. When inhibition succeeded, SSD increased by 50 ms. Subjects were informed about the tracking procedure, and they were told not to let the stop task interfere with the GO1 task and not to wait for the stop signal. Furthermore, it was explained that on some trials it would be easy to stop and on other trials it would be more difficult or impossible to stop because the stop signal would be presented near response execution.

The experiment started with one practice block of 36 trials. During the practice phase, subjects received immediate feedback about their responses. When subjects made an error on a no-signal trial, the word *wrong* appeared. If they did not respond in time (i.e., before 2,000 ms had elapsed), the words *too slow* were presented. When subjects responded in the presence of a stop signal, the sentence *Try to stop your right-handed response* appeared. If the response with the left hand was erroneous, *Wrong left-handed response* was presented. The feedback remained in the center of the screen for 750 ms. If mean error percentage was above 10% or mean GO1 RT was greater than 1,000 ms, subjects received another practice block. The experimental phase consisted of 12 blocks of 108 trials. At the end of each block, the number of GO1 errors made during the block, the mean GO1 RT, and the probability of stopping were displayed and subjects had to pause for 15 s. If GO1 accuracy was too low (i.e., mean error percentage was above 10%), a message was displayed urging the subjects to make fewer errors. When the GO1 RT exceeded 1,000 ms or the mean probability of stopping the GO1 response across SCDs was above .7, faster responding was encouraged. Finally, when the mean probability of stopping across SCDs was below .3, subjects were encouraged to stop more responses. These messages were displayed at the bottom of the screen between blocks. It was our hope that this feedback would discourage waiting for the stop signal and encourage fast and accurate GO1 responses. No GO2 task information was provided as part of the feedback. The whole experiment lasted approximately 1 hr.

Results and Discussion

Mean GO1 RTs were calculated after removal of GO1 errors. Similarly, mean GO2 RTs were calculated after removal of GO2 errors. For signal-inhibit trials (i.e., trials on which the inhibition of the GO1 response succeeded), we did not know whether the inhibited response would have been correct. Therefore, we included all signal-respond trials (i.e., trials on which the inhibition of the GO1 response failed), regardless of whether the GO1 response was correct. Outlying RTs (i.e., RTs longer than 2.5 *SD*s above the mean for each trial type) were discarded from data analysis. This trimming procedure resulted in a data reduction of 2.5%.

GO1 performance. We analyzed GO1 performance on no-signal and signal-respond trials. Consistent with the race model (Logan & Cowan, 1984), GO1 RTs were shorter for signal-respond trials (443 ms; $SE = 19$ ms) than for no-signal trials (496 ms; $SE = 29$ ms), $F(1, 19) = 12.1$, $MSE = 2{,}260$, $p < .01$, $\eta_p^2 = .39$. The percentage of GO1 choice errors was comparable for signal-respond trials (2.7%; $SE = 0.8$ %) and no-signal trials (3.0%; $SE = 0.5$%). GO1 data were not analyzed further.

Stopping performance. The relevant means for stopping performance are presented in Table 1 and are depicted in the left panel of Figure 5. Mean SSD was 255 ms, and there were no differences in SSD across reference points, $F(2, 38) < 1$. On the basis of the assumptions of the horse-race model, SSRT can be calculated by subtracting mean SSD from the untrimmed mean GO1 RT (Logan, 1994; Logan, Schachar, & Tannock, 1997). Mean SSRT was 298 ms, and SSRT did not differ across reference points, $F(2, 38) < 1$. SSRT analyses as a function of SCD appear in the Appendix.

We analyzed the *p*(respond|signal) by means of a repeated measures analysis of variance (ANOVA), with reference point (high, middle, low) and SCD (0, 150, 300 ms) as within-subjects factors. In this analysis (and all other analyses that will follow), planned comparisons were conducted using the relevant error terms from the omnibus ANOVA. The *p*(respond|signal) approximated .50 for the three reference points, with no difference between them, $F(2, 38) = 1.3$, $MSE = 3.4$, $p > .29$, $\eta_p^2 = .06$. There was a main effect of SCD, $F(2, 38) = 17.6$, $MSE = 74.2$, $p < .001$, $\eta_p^2 = .48$. Planned comparisons showed that *p*(respond|signal) was lower for SCD = 0 ms (.48; $SE = .016$) than for SCD = 150 ms (.55; $SE = .018$), $F(1, 38) = 20.5$, $p < .001$, $\eta_p^2 = .53$, and *p*(respond|signal) for SCD = 150 ms did not differ significantly from *p*(respond|signal) for SCD = 300 ms (.56; $SE = .017$), $F(1, 38) = 1.1$, $p > .29$, $\eta_p^2 = .07$. The interaction between reference point and SCD was nonsignificant, $F(4, 74) < 1$.

In sum, *p*(respond|signal) increased slightly with increasing SCD. Only two models predicted an effect of SCD on *p*(respond|signal): the two-goal GO1-GO2 model (see Figure 2, Model 1) and the limited-capacity version of the three-goal parallel GO1-GO2+STOP model (see Figure 2, Model 5). However, the limited-capacity GO1-GO2+STOP model predicted an effect of

Table 1
Mean Probability of Responding (p[respond|signal]), Stop-Signal Delay (SSD), and Stop-Signal Reaction Time (SSRT) for Each Reference Point in Experiment 1 (with Standard Errors in Parentheses)

	Reference point		
Measure	High	Middle	Low
p(respond|signal)	.530 (.015)	.526 (.013)	.530 (.015)
SSD	256 (30)	253 (30)	256 (33)
SSRT	297 (12)	300 (12)	297 (12)

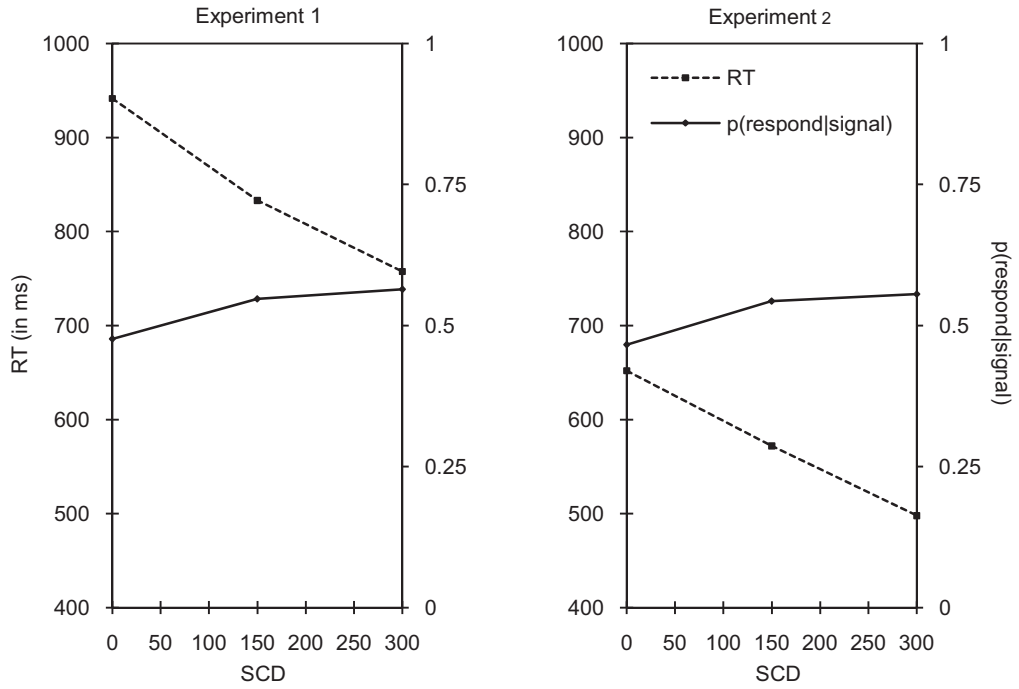

Figure 5. Mean GO2 reaction times (RTs; left *y*-axis) and probabilities of responding (*p*[respond|signal]; right *y*-axis) as a function of SCD (in ms) in Experiment 1 (left panel) and Experiment 2 (right panel). The dotted lines represent the GO2 RTs, and the solid lines represent the *p*(respond|signal).

SCD in the opposite direction, so the data are inconsistent with that model. The magnitude of the observed effect is inconsistent with the predictions of the GO1-GO2 model. This model predicted a large, monotonic increase of *p*(respond|signal) with increasing SCD,[2] but we observed only a small difference between SCD = 0 and 150 ms and no difference between SCD = 150 and 300 ms.

The relatively flat *p*(respond|signal) pattern is more consistent with both versions of the serial GO1-STOP-GO2 model (see Figure 2, Models 2 and 3) and the unlimited-capacity version of the parallel GO1-GO2+STOP model (see Figure 2, Model 4), which predicted no SCD effect on *p*(respond|signal). Note that when the proportion of capacity allocated to the STOP process is large (see Figure 2, Model 5, bottom panel), the limited-capacity version of the parallel GO1-GO2+STOP model also predicts virtually no effect of SCD on *p*(respond|signal). The observed small difference in *p*(respond|signal) between SCD = 0 ms and the two longer SCDs could be interpreted in terms of intersensory facilitation, as it has previously been demonstrated that redundant targets in two modalities are processed faster than a single target in one modality (J. Miller, 1982; Schröger & Widmann, 1998). Consequently, subjects may be faster at detecting the stop signal when the change signal and the stop signal are presented together, resulting in a lower *p*(respond|signal) because the STOP process can start racing sooner.

GO2 task performance. Mean GO2 RTs and mean error data appear in Table 2 as a function of SCD, reference point, and whether stopping of the GO1 response succeeded (signal-inhibit trials) or failed (signal-respond trials). GO2 RTs collapsed across reference points of signal-inhibit trials also appear as a function of SCD in the left panel of Figure 5. GO2 RTs following a signal-respond trial are not plotted because the model's predictions plotted in Figure 2 are based on the assumption that stopping is successful. For signal-respond trials, GO2 responding is not dependent on the finishing time of the STOP process but on the finishing time of GO1 processing.

We analyzed GO2 RTs by means of a 3 (reference point: high, middle, low) × 3 (SCD: 0, 150, 300 ms) × 2 (STOP: signal-respond vs. signal-inhibit) repeated measures ANOVA. Only the main effects reached significance (all other *F*s < 1). GO2 RTs were longer when the inhibition of the GO1 response succeeded, $F(1, 19) = 12.2$, $MSE = 26{,}495$, $p < .01$, $\eta_p^2 = .39$. GO2 RTs also became longer as SCD decreased, $F(2, 38) = 144.2$, $MSE = 4{,}737$, $p < .001$, $\eta_p^2 = .88$. The slope of the observed SCD function was

[2] For the GO1-GO2 model, if subjects do not stop until the change signal is presented, *p*(respond|signal) should increase monotonically with increasing SCD. Because we used a tracking procedure and SCD = 150 ms is the middle SCD, *p*(respond|signal) should only approximate .50 at this specific SCD. On the basis of the horse-race model (see Figure 3), we derived the predicted *p*(respond|signal) for SCD = 0 ms by subtracting 150 ms from the median GO1 RT (Md) and estimating *p*(respond|signal) for SCD = 0 ms as equal to the area of the GO1 RT curve that is to the left of this value. Following the same logic, *p*(respond|signal) for SCD = 300 ms is equal to the area to the left of Md + 150. Following this procedure, we estimated the *p*(respond|signal) values predicted by the GO1-GO2 model. In Experiment 1, the predicted *p*(respond|signal) values were 0.08, 0.5, and 0.79, for SCD = 0, 150, and 300 ms, respectively. The predicted *p*(respond|signal) values for SCD = 0 and 300 ms deviate substantially from the observed values, indicating that the GO1-GO2 model does not adequately explain the slight increase in *p*(respond|signal) that we observed.

Table 2
Mean GO2 Reaction Time (RT) and Error Percentage as a Function of Stop-Change Delay (SCD; in ms), Trial Type (Signal-Inhibit or Signal-Respond), and Reference Point (High, Middle, or Low) in Experiments 1 and 2 (With Standard Errors in Parentheses)

	RT (ms)			Error (%)		
Experiment and trial type	SCD 0	SCD 150	SCD 300	SCD 0	SCD 150	SCD 300
Experiment 1						
Signal-inhibit						
High	953 (41)	842 (47)	781 (43)	8.1 (1.6)	9.6 (1.9)	10.7 (1.7)
Middle	930 (42)	819 (45)	737 (46)	1.8 (0.6)	1.0 (0.5)	3.3 (0.8)
Low	954 (46)	852 (41)	775 (51)	10.3 (2.0)	12.4 (1.4)	12.8 (2.0)
Signal-respond						
High	887 (39)	776 (49)	674 (45)	15.0 (1.9)	14.9 (1.8)	15.8 (1.8)
Middle	841 (48)	753 (43)	672 (52)	3.2 (1.0)	5.3 (1.2)	3.6 (1.0)
Low	884 (45)	797 (44)	695 (53)	14.0 (2.4)	13.1 (1.8)	14.5 (1.5)
Experiment 2						
Signal-inhibit	652 (24)	572 (25)	498 (22)	2.9 (0.6)	2.8 (0.9)	7.9 (1.1)
Signal-respond	706 (31)	610 (33)	514 (25)	4.1 (1.3)	5.4 (1.3)	9.5 (2.0)

−.61. Planned comparisons showed that the observed GO2 RTs were shorter for SCD = 150 ms than for SCD = 0 ms, $F(1, 38) = 86.2$, $p < .001$, $\eta_p^2 = .84$, and GO2 RTs were shorter for SCD = 300 ms than for SCD = 150 ms, $F(1, 38) = 58.8$, $p < .001$, $\eta_p^2 = .74$. These results are consistent with the nondeterministic version of the serial GO1-STOP-GO2 model (see Figure 2, Model 3) and the limited-capacity version of the GO1-GO2+STOP model (see Figure 2, Model 5). These two models assumed that GO2 RTs should decrease with increasing SCD and that the slope of the SCD function should be less than −1.

Note that there was also a main effect of reference point, $F(2, 38) = 5.16$, $MSE = 7{,}470$, $p < .05$, $\eta_p^2 = .21$. Planned comparisons showed that GO2 RTs of trials with the middle line as reference point were generally shorter than GO2 RTs with the upper line, $F(1, 38) = 5.7$, $p < .05$, $\eta_p^2 = .17$, or lower line, $F(1, 38) = 9.3$, $p < .01$, $\eta_p^2 = .34$, as reference point. The latter two did not differ from each other, $F(1, 38) < 1$. This finding is consistent with recent findings of Schneider and Logan (2007), who observed a reference-point switch cost in a task that required left/right judgments of spatial targets.

For the error data, there were main effects of stopping, $F(1, 19) = 8.3$, $MSE = 117.0$, $p < .01$, $\eta_p^2 = .30$, and reference point, $F(1, 38) = 55.7$, $MSE = 65.8$, $p < .001$, $\eta_p^2 = .75$. The main effect of stopping suggests that response-correspondence effects were less pronounced when the GO1 response was successfully stopped. This finding is consistent with all models as they all predict that activation of the GO1 response should be lower for signal-inhibit trials than for signal-respond trials, regardless of whether a STOP goal is involved. The interaction between stopping and reference point also reached significance, $F(2, 38) = 3.6$, $MSE = 39.3$, $p < .05$, $\eta_p^2 = .16$. No other effects were significant.

Discussion

Both *p*(respond|signal) and GO2 RTs must be taken into consideration when evaluating the models. The *p*(respond|signal) data are consistent with both versions of the serial GO1-STOP-GO2 model (see Figure 2, Models 2 and 3), the unlimited-capacity GO1-GO2+STOP model (see Figure 2, Model 4), and the limited-capacity GO1-GO2+STOP model when the proportion of capacity allocated to the STOP process is large (see Figure 2, Model 5). The GO2 RTs are consistent with the nondeterministic version of the GO1-STOP-GO2 model (see Figure 2, Model 3) and the limited-capacity GO1-GO2+STOP model (see Figure 2, Model 5). When both data patterns are considered together (see the left panel of Figure 5), they are only consistent with the nondeterministic version of the GO1-STOP-GO2 model (Model 3) and the limited-capacity GO1-GO2+STOP model (see Figure 2, Model 5) when the proportion of capacity allocated to the STOP process is large.

To answer the first question the experiment was designed to address, the data suggest that a STOP goal is involved in stopping a response in the stop-change paradigm. This is consistent with the study of Camalier et al. (2007) in which a variant of the stop-change paradigm was used. To answer the second question the experiment was designed to address, the data suggest that the STOP and GO2 goal were activated serially or that parallel processing with limited capacity mimicked serial processing. We conducted Experiment 2 to provide converging evidence for these conclusions.

Note that we cannot exclude the possibility that shifting attention from the visual modality (the target and the stop signal) to the auditory modality (the reference point cue) also influenced GO2 processing (e.g., Spence et al., 2001; Turatto et al., 2002; but see Alais et al., 2006). However, shifting attention between the two modalities occurs for every SCD so the effect of attention shifting may be additive to the effect of SCD.

Experiment 2

The results of Experiment 1 support the nondeterministic version of the serial GO1-STOP-GO2 model and the limited-capacity version of the parallel GO1-GO2+STOP model and suggest that a STOP goal is involved in the stop-change paradigm. In Experiment 2, we sought to replicate these findings with a different procedure. In Experiment 1, we used the same stimulus for the GO1 and GO2 tasks. While some versions of the PRP procedure use overlapping stimuli and tasks (e.g., Hommel, 1998), it is more common to use different stimuli for the two tasks (see Pashler, 1994). Consequently, we replicated Experiment 1 with nonoverlapping stimuli to provide a conceptual replication of Experiment 1 and to assess

the predictions in a procedure that is more like the typical PRP and stop-change procedures.

In Experiment 2, subjects had to respond to the position of the visual stimulus for the GO1 task but not for the GO2 task. Instead, the GO2 task required subjects to report the identity of the auditory word. We used the same SCD manipulation as in Experiment 1. If using the same stimulus for the GO1 and GO2 tasks produced the SCD effect in Experiment 1, we should see a different effect in Experiment 2.

Method

Subjects. Twenty undergraduate students from Vanderbilt University participated for course credit or monetary compensation. All subjects had normal or corrected-to-normal vision and were naive as to the purpose of the experiment. None of the subjects had participated in Experiment 1.

Apparatus, stimuli, and procedure. Only differences from Experiment 1 are discussed. For the GO2 task, the words *high* or *low* were presented through the speakers after a variable SCD (0, 150, or 300 ms), and subjects had to respond to the identity of the word. When the word was *high*, they pressed the *E* key with the middle finger of the left hand; when the word was *low*, they pressed the *F* key with the index finger of the left hand.

The experiment started with a practice block of 32 trials. If necessary, subjects received another practice block. The experiment consisted of 9 blocks of 72 trials. The experiment lasted approximately 30 min.

Results and Discussion

Mean GO1 RTs were calculated after removal of GO1 errors. Similarly, mean GO2 RTs were calculated after removal of GO2 errors. We used the same trimming procedure as in Experiment 1, resulting in a data reduction of 2.4%.

GO1 performance. Once again, GO1 RTs were shorter for signal-respond trials (433 ms; $SE = 20$ ms) than for no-signal trials (503 ms; $SE = 24$ ms), $F(1, 19) = 97.1$, $MSE = 513$, $p < .001$, $\eta_p^2 = .84$. The percentage of GO1 choice errors was comparable for signal-respond trials (2.2%; $SE = 0.5$ %) and no-signal trials (2.6%; $SE = 0.4$%), $F(1, 19) < 1$.

Stopping performance. Only one tracking procedure was used. Probability of stopping was .521 ($SE = .01$). The average SSD was 260 ms ($SE = 29.7$ ms), and the average SSRT was 260 ms ($SE = 9.3$ ms). SSRTs as a function of SCD appear in the Appendix.

We analyzed the effect of SCD on *p*(respond|signal) by means of a repeated measures ANOVA, with SCD (0, 150, or 300 ms) as a single within-subjects factor. The probabilities of responding are depicted in the right panel of Figure 5. We found a main effect of SCD, $F(2, 38) = 9.5$, $MSE = .005$, $p < .001$, $\eta_p^2 = .33$. Planned comparisons showed that *p*(respond|signal) was lower for SCD = 0 ms (.47; $SE = .013$) than for SCD = 150 ms (.54; $SE = .016$), $F(1, 38) = 12.0$, $p < .001$, $\eta_p^2 = .42$, and *p*(respond|signal) for SCD = 150 ms did not differ significantly from *p*(respond|signal) for SCD = 300 ms (.56; $SE = .20$), $F(1, 38) < 1$. These results replicate the findings of Experiment 1. The two-goal GO1-GO2 model (see Figure 2, Model 1) predicted much larger differences than we observed.[3] Once again, the relatively flat *p*(respond|signal) pattern is more consistent with both versions of the serial GO1-STOP-GO2 model (see Figure 2, Models 2 and 3), the unlimited-capacity GO1-GO2+STOP model (see Figure 2, Model 4), and with the limited-capacity GO1-GO2-STOP model (see Figure 2, Model 5) when the proportion of capacity allocated to the STOP process is large (see Figure 2). In the latter case, the limited-capacity GO1-GO2-STOP model predicts virtually no effect of SCD on *p*(respond|signal).

GO2 task performance. We analyzed GO2 RTs by means of a 3 (SCD: 0, 150, or 300 ms) × 2 (STOP: signal-respond vs. signal-inhibit) repeated measures ANOVA. Mean GO2 RTs and mean error data appear in Table 2 as a function of SCD and whether stopping of the GO1 response succeeded (signal-inhibit trials) or failed (signal-respond trials). Mean GO2 RTs of signal-inhibit trials also appear as a function of SCD in the right panel of Figure 5. There was a main effect of SCD, $F(2, 38) = 347.6$, $MSE = 858$, $p < .001$, $\eta_p^2 = .95$. The slope of the observed SCD function was −.51. Planned comparisons showed that observed GO2 RTs were shorter for SCD = 150 ms than for SCD = 0 ms, $F(1, 38) = 180.2$, $p < .001$, $\eta_p^2 = .96$, and GO2 RTs were shorter for SCD = 300 ms than for SCD = 150 ms, $F(1, 38) = 167.4$, $p < .001$, $\eta_p^2 = .86$. These results are consistent with the nondeterministic version of the GO1-STOP-GO2 model (see Figure 2, Model 3) and the limited-capacity GO1-GO2+STOP model (see Figure 2, Model 5).

Contrary to Experiment 1, GO2 RTs for signal-inhibit trials were shorter than GO2 RTs for signal-respond trials, $F(1, 19) = 5.6$, $MSE = 6{,}987$, $p < .05$, $\eta_p^2 = .23$. The interaction between SCD and stopping just failed to reach significance, $F(2, 38) = 3.2$, $MSE = 1{,}106$, $p = .052$, $\eta_p^2 = .14$. As can be seen in Table 2, the difference between GO2 RTs for signal-inhibit trials and GO2 RTs for signal-respond trials became smaller as SCD increased.

For the error data, there was also an effect of SCD, $F(2, 38) = 23.6$, $MSE = 13.5$, $p < .001$, $\eta_p^2 = .55$. Planned comparisons revealed that there was no difference between SCD = 0 ms and SCD = 150 ms, $F(1, 38) < 1$. However, subjects made more errors for SCD = 300 than for SCD = 150 ms, $F(1, 38) = 30.7$, $p < .001$, $\eta_p^2 = .60$. This might suggest a speed–accuracy trade-off for SCD = 300 ms. The main effect of stopping was nonsignificant, $F(1, 19) = 1.4$, $MSE = 69.6$, $p > .24$, $\eta_p^2 = .06$. The interaction between SCD and stopping was also nonsignificant, $F(1, 19) < 1$.

Discussion. We observed the same SCD effects in Experiment 2 as in Experiment 1, even though stimulus overlap was not a factor in Experiment 2. The *p*(respond|signal) data are consistent with both versions of the serial GO1-STOP-GO2 model (see Figure 2, Models 2 and 3), the unlimited-capacity GO1-GO2+STOP model (see Figure 2, Model 4), and the limited-capacity GO1-GO2+STOP model when the proportion of capacity allocated to the STOP process is large (see Figure 2, Model 5). The GO2 RTs are consistent with the nondeterministic version of the GO1-STOP-GO2 model and the limited-capacity GO1-GO2+STOP model. When both data patterns are considered to-

[3] We used the same procedure as in Experiment 1 to estimate *p*(respond|signal) values predicted by the GO1-GO2 model. The predicted *p*(respond|signal) values were 0.0, 0.5, and 0.82, for SCD = 0, 150, and 300 ms, respectively. The predicted *p*(respond|signal) values for SCD = 0 and 300 ms deviate substantially from the observed values, replicating the findings of Experiment 1.

gether (see the right panel of Figure 5), we find that they are only consistent with the nondeterministic version of the GO1-STOP-GO2 model and the limited-capacity GO1-GO2+STOP model when the proportion of capacity allocated to the STOP process is large.

In sum, the results of Experiments 1 and 2 converge on the same answers to the questions that motivated this study: They suggest that a STOP goal is involved in cancelling the GO1 response and that STOP and GO2 processing occur serially or in parallel with a capacity-sharing proportion that mimics serial processing. Experiment 1 used the same stimuli for the GO1 and GO2 tasks (like Hommel, 1998), whereas Experiment 2 used different stimuli (like most PRP and stop-change studies). Despite this methodological difference, we observed similar SCD effects, which suggests that the observed SCD effects are not due to a stimulus overlap.

However, stimulus overlap could explain two differences between Experiment 1 and 2. First, in Experiment 2, GO2 RTs were shorter for signal-inhibit trials than for signal-respond trials (see also Logan & Burkell, 1986), whereas the opposite was observed in Experiment 1. However, this slowing on signal-inhibit trials in Experiment 1 could be due to temporary suppression of stimulus processing (Logan, 1983, 1985b). Second, SSRTs were longer in Experiment 1 than in Experiment 2. One could speculate that response inhibition needed to be more selective in Experiment 1 than in Experiment 2 because of the stimulus (and task) overlap. Logan and Burkell (1986) have argued that the stop-change paradigm involves a slow but selective mode of inhibition, whereas the standard stop-signal paradigm involves a fast but nonselective mode of inhibition (see also Band & van Boxtel, 1999; De Jong, Coles, & Logan, 1995). On the basis of this distinction, it could be hypothesized that a more selective but slower mode of inhibition is used in Experiment 1, explaining the observed SSRT differences.

General Discussion

The purpose of the present study was to investigate multitasking in the stop-change paradigm (Logan, 1983; Logan & Burkell, 1986). We conducted two experiments involving a manipulation of SCD to distinguish between five models of stop-change performance. The models were built around three questions: First, is a STOP goal involved in cancelling the GO1 response? Second, if a STOP goal is involved, are the STOP and GO2 goals activated in series or in parallel? Third, if the STOP and GO2 goals are activated in parallel, do STOP and GO2 processing share capacity?

Is a STOP Goal Involved?

To address the first question, we distinguished between a (two-goal) GO1-GO2 model and four (three-goal) STOP models. The GO1-GO2 model assumes that stopping the GO1 response is accomplished by replacing the GO1 goal with the GO2 goal, whereas the STOP models assume that stopping the GO1 response is accomplished by replacing the GO1 goal with the STOP goal.

Considered together, the *p*(respond|signal) and GO2 RT data of both experiments are inconsistent with the GO1-GO2 model. This model predicted large effects of SCD on *p*(respond|signal) and no effect of SCD on GO2 RTs (see Figure 2, Model 1). As can be seen in Figure 5, the observed *p*(respond|signal) and GO2 RT data are inconsistent with these predictions, suggesting that a STOP goal is involved (see below). Recently, Camalier et al. (2007) arrived at a similar conclusion. They investigated which task goals were involved in stopping and replacing an eye movement. They compared models with and without a STOP goal and found that the two models that involved a STOP goal fit the data better than the model without a STOP goal. In the present study, we replicated this finding in the stop-change paradigm with manual responses. Therefore, the results of Camalier et al. and the present study suggest that a STOP goal is involved in cancelling eye and hand movements, even though there are some important differences between the two (see, e.g., Boucher, Stuphorn, et al., 2007; Logan & Irwin, 2000).

In sum, the answer to the first question is as follows: Yes, a STOP goal is involved in cancelling the GO1 response in the stop-change paradigm.

Are the STOP and GO2 Goals Activated in Series or in Parallel?

To address the second question, we distinguished between two versions of the serial GO1-STOP-GO2 model (deterministic vs. nondeterministic processing) and two versions of the parallel GO1-GO2+STOP model (unlimited vs. limited capacity). The serial models assume that GO2 processing starts after the STOP process has finished, whereas the parallel models assume that GO2 processing can start before the STOP process has finished.

The *p*(respond|signal) and GO2 RT data of both experiments are consistent with the nondeterministic version of the GO1-STOP-GO2 model (see Figure 2, Model 3) and with the limited-capacity version of the parallel GO1-GO2+STOP model (see Figure 2, Model 5) when the proportion of capacity allocated to the STOP process is large. Thus, answering the second question requires discussing both models in more detail.

The nondeterministic GO1-STOP-GO2 model. The nondeterministic GO1-STOP-GO2 model assumes that subjects first replace the GO1 goal with the STOP goal and, upon inhibition of the GO1 response, replace the STOP goal with the GO2 goal. This serial processing is advantageous because it avoids the order-control problems that result from having the STOP and GO2 goals active at the same time (Logan & Gordon, 2001). With parallel processing, the GO2 goal may become active before the STOP goal, and subsequent activation of the STOP goal may replace the GO2 goal (i.e., the STOP process may stop the GO2 response instead of the GO1 response). This would suspend or delay GO2 processing.

The model also assumes there is trial-to-trial variability in stopping and GO2 processing. Schwarz and Ischebeck (2001) demonstrated that stochastic variation in the durations of two successive tasks (or in our case, stopping and GO2 processing) will flatten the slope of the delay function. More specifically, Schwarz and Ischebeck showed that the local slope at a given delay reflects the probability that the first process has not finished. If it has not finished, the slope is -1; if it has finished, the slope is 0. Trial-to-trial variability results in a probability of the first process finishing that falls between 0 and 1, which produces a probability mixture of slopes of -1 and 0 that results in an overall slope between -1 and 0. Thus, the observed local slope at a given delay reflects the probability that the first process has finished by that delay.

We applied this idea to our data to estimate the probability that the STOP process had finished by a particular SCD. We calculated separate local SCD slopes for both experiments. In Experiment 1, the slopes of the observed SCD function are −.72 (SCD = 0 ms to SCD = 150 ms) and −.50 (SCD = 150 ms to SCD = 300 ms). These data are consistent with the idea that the STOP process was finished on 28% of the trials by SCD = 150 ms and on 50% of the trials by SCD = 300 ms. In Experiment 2, we found that the slopes of the observed SCD function were −.53 (SCD = 0 ms to SCD = 150 ms) and −.49 (SCD = 150 ms to SCD = 300 ms), suggesting that the STOP process was finished on 47% of the trials by SCD = 150 ms and on 51% of the trials by SCD = 300 ms. However, there are two problems with these estimated probabilities.

First, the values of the slopes depend on the density of the SCDs sampled in the experiment, so we may be under- or overestimating the local slopes by having only three SCDs. Second, the SCDs were separated by 150 ms and the longest SCD (300 ms) was longer than the mean SSRT in Experiment 1 (298 ms) and in Experiment 2 (260 ms). Given that slopes of −1 are typically observed only at very short stimulus onset asynchronies (SOAs) and they tend to flatten at longer SOAs, the slopes of the present study may have been less than −1 because we used at least one relatively long SCD (300 ms).

The limited-capacity GO1-GO2+STOP model. According to this model, GO2 processing occurs before the GO1 response is stopped, such that the STOP and GO2 goals are simultaneously active. To avoid the order-control problem noted above, this parallel processing must involve a selective STOP process that cancels the GO1 response without disabling GO2 processing. This selectivity assumption is consistent with the recurrent finding that SSRT is longer in the stop-change paradigm than in the standard stop-signal paradigm (De Jong et al., 1995; Logan & Burkell, 1986). On the basis of these results, Logan and Burkell (1986) distinguished between local (slow but selective) and global (fast but nonselective) modes of inhibition to account for differences in SSRT between the stop-change and the standard stop-signal paradigms. The idea is that subjects take longer to stop a response in the stop-change paradigm because they have to use a local inhibitory mode to stop the GO1 response without disabling the GO2 response, whereas in the standard stop-signal paradigm, the fast but nonselective mode can be used to stop all responses (see also Band & Van Boxtel, 1999; De Jong et al., 1995).

The GO1-GO2+STOP model also assumes that the STOP and GO2 processes share limited capacity, so processing can occur in parallel. However, limited-capacity sharing models of the PRP procedure suggest that dual-task data can only be accounted for if 90% or more of the capacity is first allocated to one task and then allocated to the other (see Navon & Miller, 2002; Tombu & Jolicœur, 2003, 2005). Thus, parallel processing with limited capacity can mimic serial processing, in which 100% of the capacity is first allocated to one task and then to the other (see also Logan, 2002; Townsend & Ashby, 1983; Townsend & Wenger, 2004).

The results of Experiments 1 and 2 suggest that capacity sharing in the stop-change paradigm could only occur if the proportion allocated to the STOP process was large. The limited-capacity GO1-GO2+STOP model predicted very small effects of SCD on *p*(respond|signal) when the sharing proportion was large (see Figure 2, Model 5, and Figure 4C). In addition, the small effects of capacity sharing may have been counteracted by intersensory facilitation (see above; J. Miller, 1982; Schröger & Widmann, 1998). Thus, the *p*(respond|signal) data may be consistent with the limited-capacity GO1-GO2+STOP model but only when almost all capacity is first allocated to the STOP process.

The GO2 RTs are also consistent with the limited-capacity GO1-GO2+STOP model. Capacity-sharing models are typically compared with deterministic serial models (Navon & Miller, 2002; Tombu & Jolicœur, 2003), and Navon and Miller (2002) demonstrated that both types of models predict the same slopes for the RT2-SOA curve. We suggest that stochastic variation in the durations of stopping and GO2 processing will also flatten the slope of the delay function when they share capacity. However, further investigation of nondeterministic capacity-sharing models is needed to determine whether our speculation is valid.

A final remark concerns why a high sharing proportion might be advantageous in the stop-change paradigm. As can be seen in Figures 4B and 4C, allocating capacity to stopping decreases *p*(respond|signal) without affecting GO2 RT. These strategic considerations would encourage subjects to use a high sharing proportion or to process the STOP and GO2 goals in series, which suggests that our effects are due to strategic limitations (Logan & Gordon, 2001; Meyer & Kieras, 1997a), not structural limitations (i.e., a central-processing bottleneck; Pashler, 1994).

In sum, the results of Experiments 1 and 2 are consistent with the nondeterministic version of the serial model (see Figure 2, Model 3) and the limited-capacity version of the parallel model with almost all capacity allocated initially to the STOP process (see Figure 2, Model 5). Thus, the answer to the second question is as follows: STOP and GO2 processing occur serially or in parallel with a capacity-sharing ratio that mimics serial processing.

Do STOP and GO2 Processing Share Capacity?

By answering our second question, we have also answered our third question: If parallel processing occurs, then GO2 processing has to share capacity with STOP processing. As can be seen in Figure 5, the data are inconsistent with the unlimited-capacity version of the parallel GO1-GO2+STOP model (see Figure 2, Model 4), but they are consistent with a limited-capacity version of the model in which the proportion of capacity allocated to the STOP process is large.

The Relation Between the Stop-Change Paradigm and Other Multitask Paradigms

In the introduction, we described the PRP paradigm, the stop-signal paradigm, and the task-switching paradigm that involve multitasking. Many theoretical accounts of performance in these paradigms are based on the idea that goal representations are manipulated when switching from one task to another (Gilbert & Shallice, 2002; Logan & Cowan, 1984; Logan & Gordon, 2001; Meyer & Kieras, 1997a, 1997b; E. K. Miller & Cohen, 2001; Rubinstein, Meyer, & Evans, 2001). In the remainder of this article, we focus on the relation between the stop-change paradigm and these other multitask paradigms.

The PRP paradigm. The stop-change paradigm has previously been compared with the PRP paradigm (Hübner & Druey, 2006; Logan & Burkell, 1986) to investigate the nature of the dual-task interference effect. Logan and Burkell (1986) demonstrated that in

their stop-change condition, GO1 response selection had no effect on GO2 task performance on signal-inhibit trials, whereas dual-task interference (i.e., the PRP effect) was found in their dual-task condition and on signal-respond trials in their stop-change condition (see also Logan, 1985b). These findings suggest that successfully inhibiting the GO1 response helps to avoid the dual-task interference caused by GO1 processing. However, the results of the present study suggest that STOP processing influences GO2 processing.

On the basis of our findings, we suggest that the occurrence of dual-task interference between two processes depends on the relevance of the task goals associated with the processes. In the PRP paradigm, the GO2 stimulus often appears before the GO1 response has been executed. When this happens, both GO goals are relevant simultaneously and GO1 processing has to be in an advanced phase before some stages of GO2 processing can start (Byrne & Anderson, 2001; Logan & Gordon, 2001; Meyer & Kieras, 1997b; Pashler, 1994). In the stop-change paradigm, the stop-change signal also often appears before the GO1 response has been executed. However, stopping can start the moment a stop-change signal is presented. That is, the GO1 goal becomes irrelevant and it can be replaced immediately by the STOP goal, regardless of the state of GO1 processing. Consequently, no dual-task interference from the GO1 response is expected in the stop-change paradigm. A different pattern emerges for GO2 processing. When the stop-change signal is presented, the STOP and GO2 goals are both relevant. Analogous to the PRP paradigm, STOP processing has to be in an advanced phase before GO2 processing can start, consistent with the conclusions drawn from the results of the present study.

In sum, we hypothesize that dual-task interference in the PRP paradigm and the stop-change paradigm depends on the relevance of task goals. There should be no dual-task interference between two processes when one goal is no longer relevant. However, there should be dual-task interference in both paradigms when two task goals are relevant and the processes associated with those goals overlap in time.

The stop-signal paradigm. Previous studies of the stop-signal paradigm have shown that STOP processing is hardly influenced by GO1 processing (Logan & Burkell, 1986; Logan & Cowan, 1984; Logan et al., 1984). The results of the present study also suggest that STOP processing is not influenced by GO2 processing: STOP and GO2 processing appeared to occur serially or, if they occur in parallel, they do so in such a way that almost all capacity is allocated to the STOP process. As we argued before, we do not assume that this dual-task interference effect reflects structural limitations. Instead, we assume that serial or close-to-serial processing occurs because of strategic limitations. That is, allocating all capacity to the STOP process is beneficial because it will decrease SSRT and the probability of responding to the GO1 task.

As we discussed above, different inhibitory mechanisms may be involved in the stop-signal and stop-change paradigms (Band & van Boxtel, 1999; De Jong et al., 1995; Logan & Burkell, 1986). However, the present findings suggest that some of the underlying processes are similar. In both paradigms, stopping is accomplished by replacing the GO1 goal with the STOP goal, and stopping is not influenced by GO1 (or GO2) processing because subjects allocate all of their capacity to the STOP process.

The task-switching paradigm. In the preceding sections, we focused on multitask paradigms in which there is temporal overlap in the underlying processes. Another paradigm that is used to study multitasking—but in which there is no temporal overlap—is the task-switching paradigm (Jersild, 1927).

Several studies have demonstrated a relation between response inhibition in the stop-signal paradigm and the task-switching paradigm. Miyake et al. (2000) showed this by means of latent-variable analyses, whereas Aron and colleagues found remarkable overlap in brain regions such as the right inferior frontal cortex (Aron, Monsell, Sahakian, & Robbins, 2004; Aron, Robbins, & Poldrack, 2004). Furthermore, several authors have hypothesized that switching between tasks involves inhibition of the irrelevant task sets (Arbuthnott & Frank, 2000; Mayr, Diedrichsen, Ivry, & Keele, 2006; Mayr & Keele, 2000) or elements of the irrelevant task sets (such as category-response rules; Schuch & Koch, 2003). In addition, Aron and colleagues (Aron, Monsell, et al., 2004; Aron, Robbins, & Poldrack, 2004) proposed that suppression of inappropriate responses is crucial when switching between tasks. Therefore, the relation between response inhibition and task switching may be the common requirement of inhibiting responses or task sets (Aron, Monsell, et al., 2004; Aron, Robbins, & Poldrack, 2004; Miyake et al., 2000; Verbruggen, Liefooghe, Szmalec, & Vandierendonck, 2005; Verbruggen, Liefooghe, & Vandierendonck, 2006).

However, the relation between response inhibition and task switching may be the common requirement of changing task goals. Stopping a response is accomplished by replacing the GO1 goal with the STOP goal. Similarly, Logan and Gordon (2001) argued that task switching is accomplished by changing the task goals first and then reconfiguring task-set parameters. Rubinstein et al. (2001) also argued that subjects switch tasks by changing the task goals first and then activating the appropriate category–response rules. Therefore, the act of changing task goals appears to underlie both response inhibition and task switching. Miyake et al. (2000) suggested that maintaining task goals could produce moderate correlations between different measures of executive processes. We concur with this idea, suggesting that changing task goals can at least partly explain the relation between response inhibition and task switching and, by extension, relations among all multitask paradigms.

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Appendix

SSRT as a Function of SCD

We also analyzed SSRT as a function of SCD. In Experiment 1, we collapsed across reference points because our initial analyses showed no differences between them.

Because we did not use separate tracking procedures for each SCD, we used the integration method (Logan & Cowan, 1984) to calculate SSRT as a function of SCD. The no-signal RTs are rank-ordered, then the *n*th RT is selected, where *n* is obtained by multiplying the number of RTs in the distribution by the probability of responding at a given delay. To estimate SSRT, SSD is subtracted from the *n*th RT. This process is repeated for each SSD for each subject. The results are then averaged across SSDs.

When using the tracking procedure, some SSDs will occur more often than other SSDs. To obtain reliable SSRT estimates, we selected for each subject every SSD that occurred 10 times or more and we calculated SSRT for each selected SSD. For each subject, we then calculated the average of the SSRTs. Note that in Experiment 2, there were less stop-signal trials; therefore, we selected for each subject every SSD that occurred five times or more.

In Experiment 1, we found a significant effect of SCD on SSRT, $F(2, 38) = 11.8$, $MSE = 449$, $p < .001$, $\eta_p^2 = .38$. Consistent with the p(respond|signal) data, we found that the mean SSRT for SCD = 0 ms (255 ms; $SE = 13$ ms) was shorter than the mean SSRT for SCD = 150 ms (271 ms; $SE = 13$ ms), which was shorter than the mean SSRT for SCD = 300 ms (288 ms; $SE = 16$ ms).

We observed a similar pattern of results in Experiment 2. SSRT decreased significantly with increasing SCD, $F(2, 38) = 5.3$, $MSE = 2{,}958$, $p < .01$, $\eta_p^2 = .22$. The mean SSRT for SCD = 0 ms (226 ms; $SE = 9$ ms) was shorter than the mean SSRT for SCD = 150 ms (268 ms; $SE = 16$ ms), which was shorter than the mean SSRT for SCD = 300 ms (279 ms; $SE = 25$ ms).

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