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Post-Stop-Signal Slowing: Strategies Dominate Reflexes and Implicit Learning

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Control adjustments are necessary to balance competing cognitive demands. One task that is well-suited to explore control adjustments is the stop-signal paradigm, in which subjects must balance initiation and inhibition. One common adjustment in the stop-signal paradigm is post-stop-signal slowing. Existing models of sequential adjustments in the stop-signal paradigm suggest that post-stop-signal slowing may be based solely on the events of the previous trial, suggesting that post-stop-signal slowing is a reflexive byproduct of a stop signal. Alternatively, post-stop-signal slowing could be the result of implicit learning or strategic adjustment. The authors report three experiments that manipulated the probability of stop trial repetition and found that these contingencies eliminate, reverse, or greatly increase post-stop-signal slowing. When the contingency was not instructed or cued, modest adjustments of post-stop-signal slowing occurred, suggesting implicit learning. When the contingency was cued, performance adjustments occurred on the next trial, suggesting that strategies dominated post-stop-signal slowing. These results show that post-stop-signal slowing is not a reflexive byproduct of the stop signal. The large changes in strategy accompany large changes in task factors, suggesting that the modest post-stop-signal slowing usually observed may be a result of the relatively static task environment that does not encourage large strategic changes.

Keywords: cognitive control, inhibition, post-stop-signal slowing, strategy, stop-signal paradigm

Cognitive control is required to balance the competing demands of dynamic environments (Baddeley, 1996; Logan, 1985). One cornerstone of cognitive control research has been the investigation of performance adjustments, with examples like error correction and posterror slowing (Laming, 1968; Rabbitt, 1966) and conflict monitoring (Botvinick, Braver, Barch, Carter, Cohen, 2001) pervading the literature. One task that is well suited for investigating performance adjustments is the stop-signal paradigm (Lappin & Eriksen, 1966; Logan & Cowan, 1984), as every stop trial involves a competition between initiation and inhibition, and failures of inhibition are frequent and can be manipulated. In the stop-signal paradigm, there is a trade-off between a *go task* that requires a speeded choice response and a *stop task* that requires inhibition of the speeded choice response when an infrequent stop signal occurs. The stop-signal paradigm pits speed in the go task against caution in the stop task. Faster go responses are harder to inhibit, so subjects can increase their chances of stopping by slowing their go responses (Logan & Cowan, 1984).

Several studies have shown that subjects slow their go reaction times (RT) after stop signals (Bissett & Logan, 2011; Emeric et al., 2007; Rieger & Gauggel, 1999; Verbruggen, Logan, Liefooghe, & Vandierendonck, 2008). Existing hypotheses for post-stop-signal slowing suggest that the events on the stop trial are sufficient to yield post-stop-signal slowing, either because the stop signal shifts task priority (Bissett & Logan, 2011; Leotti & Wager, 2010; Liddle et al., 2009; Wong-Lin, Eckhoff, Holmes, & Cohen, 2010), triggers error correction (Rieger & Gauggel, 1999; Schachar et al., 2004; Verbruggen et al., 2008), produces response conflict (Emeric et al., 2007), or surprises the subjects and orients attention away from the task (Notebaert et al., 2009). These hypotheses suggest that only the events of the immediately preceding trial affect post-stop-signal slowing, as if it is a reflexive reaction to those events.

Our recent research suggests that events that extend beyond the immediately preceding trial can modulate post-stop-signal slowing (Bissett & Logan, 2011). We showed that greater experiment-wide probability of a stop signal and greater experiment-wide probability of stop failure increase post-stop-signal slowing. The experiment-wide effects that we observed could be attributable to implicit learning or strategies.

The purpose of the present research is to determine whether post-stop-signal slowing is a reflexive response to events on the immediately preceding trial, a result of implicit learning, or a strategic adjustment. With reflexive adjustments, the events of the immediately preceding stop trial are sufficient to produce post-stop-signal slowing. This appears to be the predominant view of post-stop-signal slowing. Rieger and Gauggel (1999) make this claim explicitly: "We assumed that an inhibitory aftereffect would always be observable after a stop signal occurred . . ." (page 511). With implicit learning, subjects slowly acquire knowledge about

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the conditions and manipulations of the experiment, and this knowledge impacts performance. Implicit learning can be separated from explicit strategies by asking subjects about their explicit knowledge (Abrahamse, Jimenez, Verwey, & Clegg, 2010; Cleeremans, Destrebecqz, & Boyer, 1998; Seger, 1994). Last, subjects may be implementing a strategy to adjust performance based upon task demands. Strategies involve an explicit choice between alternative ways of doing a task (Logan, 1985). Strategies are flexible and can be implemented immediately and proactively, based on instructions, cues, or explicit knowledge. The present experiments were designed to distinguish between these three alternative explanations for post-stop-signal adjustments.

In the traditional stop-signal paradigm, stop signals occur on a random subset of trials, and there is no strong manipulation of task goals that would suggest a need to adjust strategy. Within this environment, it is difficult to evaluate the three possible explanations for post-stop-signal slowing. To evaluate these explanations, we manipulated conditional stop-signal probabilities and cues to distinguish the effects of reflexive adjustments, implicit learning, and strategic adjustments.

In Experiment 1, we introduced a contingency in which stop signals never occurred on consecutive trials (i.e., the conditional probability of a stop trial given the previous trial was a stop trial was 0). If subjects reflexively slow in response to the events on the stop trial, post-stop-signal slowing should occur even when stop signals never repeat. If subjects implicitly learn this contingency or change their strategy in response to it, post-stop-signal slowing should be eliminated or reversed when stop signals never repeat. We investigated the effect of this contingency in two groups: an *instructed group* that was told about the contingency at the beginning of the experiment, and an *uninstructed group* that had to learn the contingency without instructions. A greater effect in the instructed group suggests an effect of strategy above and beyond the effect of learning.

In Experiment 2, we manipulated the conditional probability of stop trial repetition across blocks (80% vs. 20% repetition), while leaving the unconditional probability of a stop signal constant at .33. Subjects were cued about this change in conditional probability before each block. If subjects reflexively slow in response to the events on the stop trial, post-stop-signal slowing should be the same in the 80% and 20% repetition conditions. If subjects learn these contingencies implicitly or change their strategies in response to them, post-stop-signal slowing should be greater in the 80% condition than in the 20% condition.

In Experiment 3, we also manipulated the conditional probability of stop signal repetition (75% repetition or 17% repetition), but we changed this conditional probability every 21 trials. There were two groups: The *cued group* that was given a cue indicating the conditional probability at the beginning of each block, and the *uncued group* that was not cued. Differences in post-stop-signal slowing between groups suggest strategy, and differences in post-stop-signal slowing between 75% and 17% repetitions in the uncued group suggests learning. In addition, adjustments across condition in the very first trial after being cued suggest strategy, as implicit learning cannot occur before any stop signals have been presented. We had subjects report any knowledge of the task manipulations after completing the experiment to determine whether learning was explicit or implicit.

Experiment 1

Method

Subjects. Forty-eight subjects were recruited from the Nashville area and were compensated $24 for two 1-hr sessions on consecutive days. All subjects had normal or corrected-to-normal vision. Twenty-four subjects served in each group. We replaced two subjects whose probabilities of successful stopping fell outside the 95% confidence interval of 0.5.

Apparatus and stimuli. The experiment was run on a Pentium Dual-Core PC running E-Prime 1 (pstnet.com). The stimuli were presented on a 19-inch CRT monitor. The go task was to respond to a single black shape on a white background presented in the center of the screen. The shape was chosen from a set of four shapes: triangle, circle, square, or diamond. The height and width of each shape was 4 cm at the longest point. Two shapes were mapped onto each of two responses, and the mapping was counterbalanced across subjects. Subjects responded by pressing "Z" or "M" on a QWERTY keyboard with the left or right index finger, respectively. The stop signal was a 500-Hz tone (70dB, 100 ms) presented through closed headphones (Sennheiser eH 150).

Procedure. Each trial began with a 500-ms fixation display, followed by the go stimulus. The go stimulus remained on the screen for 850 ms. It was followed by a 1000-ms intertrial interval (ITI) in which the screen was blank.

The stop signal indicated to subjects that they should withhold their response on that trial. The delay between the presentation of the go stimulus and the stop signal (stop-signal delay, or SSD) was varied according to a tracking procedure designed to achieve a probability of responding of 0.5 (Levitt, 1971). The initial SSD was 250 ms. The tracking procedure increased SSD by 50 ms if subjects succeeded in inhibiting and decreased SSD by 50 ms if subjects failed to inhibit. If the probability of responding is 0.5, the race between going and stopping is tied, and stop signal RT (SSRT) can be estimated by subtracting mean SSD from mean RT from trials without a stop signal (Logan, Schachar, & Tannock, 1997).

For both contingent and noncontingent sessions, stop signals occurred on 25% of the trials. In the noncontingent session, stop signals occurred at random. In the contingent session, stop signals occurred at random but were never allowed to occur on successive trials. The order of contingency sessions was counterbalanced across subjects. In the instructed group, subjects were informed about the relevant contingency before each session. In the noninstructed group, subjects were not informed about the contingency in either session.

The experiment began with written and verbal instructions. Subjects were instructed to respond quickly and accurately to the shape and to do their best to withhold their response when stop signals occurred. Subjects were told not to wait for the stop signal. After these instructions, subjects in the instructed group were told about the contingency. Subjects in the uninstructed group were told nothing. After the instructions, subjects were given 24 trials of experimenter-supervised practice. After practice, subjects completed the main task, which included five blocks of 224 trials and lasted 1 hour. At the end of each block, subjects were given feedback on RT and accuracy.

Results and Discussion

We first evaluated the effect of the contingency on performance disregarding sequential effects. We computed four separate 2 (Contingency: control or contingent) × 2 (Instruction: instructed or uninstructed) analyses of variance (ANOVAs) for mean go RT on correct trials, go task accuracy, probability of response given a stop signal, and SSRT (calculated with the mean method, see Logan et al., 1997). There were no significant main effects or interactions in any of the ANOVAs (all *p*s $>$ .1), suggesting that nonsequential task performance was equivalent across conditions.

To investigate sequential effects, mean RTs from correct no-stop-signal trials were calculated for each cell of a 2 (Pre or Post: trial S-1 or trial S+1) × 2 (Contingency: control or contingent) × 2 (Instruction: instructed or uninstructed) experimental design, including only RTs from correct trials that were shorter than 1850 ms (shape presentation time plus ITI). Post-stop-signal slowing is observed if the RT on the trial after a stop signal (trial S+1) is longer than RT on the trial preceding a stop signal (trial S-1).We performed an ANOVA with this design in which Pre or Post and Contingency were within-subject and Instruction was between-subjects. The ANOVA results are presented in Table 1. Mean RTs across subjects appear in Figure 1a for the instructed group and Figure 1b for the uninstructed group. We focused on planned comparisons using the error terms from the ANOVA to evaluate our results.

In the control condition, we assessed whether our results replicated typical stop-signal paradigms. In the instructed group, post-stop-signal slowing was positive but not significant (*M*s = 511 and 517 ms for trials S-1 and S+1, respectively), $F(1, 46) < 1$. In the uninstructed group, there was significant post-stop-signal slowing (*M*s = 470 ms and 483 ms for trials S-1 and S+1, respectively), $F(1, 46) = 4.94$, $MSE = 436$, $p < .05$.

The central aim of this experiment was to investigate the effect of the contingency on post-stop-signal slowing. In the instructed group, subjects showed post-stop-signal speeding in the contingent condition (*M*s = 501 ms and 476 ms for trials S-1 and S+1, respectively), $F(1, 46) = 17.22$, $MSE = 436$, $p < .01$, suggesting that post-stop-signal slowing is not reflexive. This 25-ms speeding differed significantly from the 6-ms slowing in the control condition, $F(1, 46) = 25.97$, $MSE = 436$, $p < .01$. In the uninstructed group, subjects showed no post-stop-signal slowing in the contingent condition (*M*s = 461 ms and 458 ms for Trials S-1 and S+1, respectively), $F(1, 46) < 1$. This 3-ms speeding differed significantly from the 13-ms slowing in the control condition, $F(1, 46) = 7.51$, $MSE = 436$, $p < .01$. Additionally, the speeding in the contingent condition of the instructed group (25 ms) was greater than the speeding in the contingent condition of the uninstructed group (3 ms), $F(1, 46) = 13.19$, $MSE = 436$, $p < .01$.

Table 1
Summary Table for Analyses of Variance Performed on Experiment 1 Mean Reaction Times

Measure	*df*	*MSE*	*F*
Pre or Post	1, 46	537	.45
Pre or Post × Instruction	1, 46	537	4.89*
Instruction	1, 46	22041	2.40
Contingency	1, 46	7041	3.12
Contingency × Instruction	1, 46	7041	.14
Pre or Post × Contingency	1, 46	436	15.35**
Pre or Post × Contingency × Instruction	1, 46	436	1.39

Note. *df* = Degrees of freedom; *MSE* = Mean squared error.
* $p < .05$. ** $p < .01$.

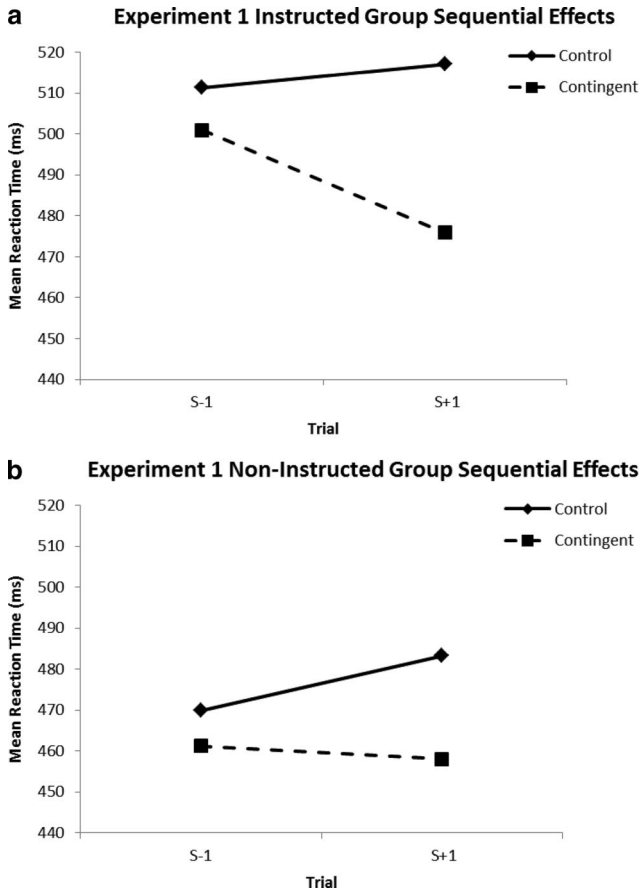

Figure 1. Experiment 1 instructed group (a) and uninstructed group (b) mean reaction time in the control and contingent conditions as a function of trial S − 1 and S+1 (Pre or Post).

Conclusions

These results show that the events on the immediately preceding stop-signal trial are not sufficient to produce post-stop-signal slowing. Subjects who were instructed that stop trials would not repeat showed post-stop-signal speeding. Uninstructed subjects showed no post-stop-signal slowing when stop trials did not repeat. Both groups of subjects showed changes in poststop signal performance in response to the contingency, suggesting that adjustments were not reflexive but rather were attributable to either implicit learning or a strategic adjustment. Greater slowing in the instructed group suggests that subjects may be explicitly adjusting strategy in response to the instructions.

Experiment 2

In this experiment, we compared post-stop-signal slowing in conditions in which stop signals repeated on 80% or 20% of the

stop trials, but the overall probability of a stop signal was held constant at .33. Existing research suggests that unconditional, experiment-wide increases in stop trial frequency increase post-stop-signal slowing (Bissett & Logan, 2011). Experiment 1 showed that conditional probabilities of 0 could eliminate (uncued group) or reverse (cued group) post-stop-signal slowing. Here, we investigated whether conditional probabilities much higher (80%) and somewhat lower (20%) than the experiment average rate (33%) affect post-stop-signal slowing. If subjects can adjust behavior in response to conditional probabilities, then slowing after a single stop trial should be substantial in the 80% repetition condition, in which stop signal repetitions are more frequent than average, and eliminated or reversed in the 20% condition, in which stop trial repetitions are less frequent than average. If subjects reflexively slow after stop signals, then post-stop-signal slowing values should be similar for the 80% and 20% repetition conditions. We investigated this contingency in two groups: In the *changed SSD group*, SSD changed after every stop trial, mirroring Experiment 1. In the *unchanged SSD group*, SSD changed only after isolated stop trials and after the first stop trial in a pair (i.e., SSD did not change contingent on the outcome of the second stop trial in a pair). The unchanged SSD procedure equated SSD for the first stop signal in a pair across the 80% and 20% repetition conditions.

Method

Subjects. Thirty-two subjects were recruited from the Nashville area and were compensated $18 for a single 90-min experimental session. All subjects had normal or corrected-to-normal vision. There were 16 subjects in each group. We replaced one subject whose probabilities of successful stopping fell outside the 95% confidence interval of .5 probability of stopping to a stop signal and two subjects for failure to comply with instructions.

Apparatus and stimuli. The apparatus and stimuli were the same as in Experiment 1, with one exception. Before each block, subjects were given a cue that said "In this block when a stop signal occurs, there is an 80% (or 20%) chance that it will repeat on the following trial. The overall probability of a stop signal on any trial is still 33%." The block did not start until they clicked past the cue.

Procedure. The basic trial structure and instructions for Experiment 2 were identical to Experiment 1 with the following exceptions: there was only a single experimental session, and our manipulation of stop trial repetition was within-session. On alternating blocks, subjects were cued as to whether stop trial would repeat on 80% of stop trials or 20% of stop trials. There were separate SSD tracking algorithm for the two repetition probabilities. Within each block, the overall probability of a stop signal was always held at 0.33, and there were never more than two stop signals in a row, so the probability of stop signal repetition given two stop signals have occurred was 0 for both block types. There were 16 blocks of 108 trials, totaling 1728 trials, 576 of which were stop-signal trials.

Results and Discussion

We first evaluated the effect of the repetition probability manipulation on performance disregarding sequential effects. We computed three separate 2 (Repetition Probability: 80% or 20%) $\times$ 2 (Group: changed or unchanged SSD) ANOVAs for mean go RT on correct trials, go task accuracy, and probability of response given a stop signal for single stop signals and the first stop signal in a pair. Repetition Probability was a within-subject factor, and Group was a between-subjects factor. There were no significant main effects or interactions in any of the ANOVAs (all $ps > .2$). Probability of a response given a stop signal differed between groups for the second stop signal in a pair because the tracking algorithm differed between groups, changing after every stop trial in the changed SSD group but changing only after the first stop trial in a pair in the unchanged SSD group.

To investigate the effect of repetition probability on post-stop-signal RT, we investigated three types of trials sequences that ended in the go trial of interest, which we will call GG, GSG, and GSSG. GG trials were go trials preceded by at least one go trial, GSG sequences were go trials preceded by one stop-signal trial and at least one go trial, and GSSG trials were go trials preceded by two stop-signal trials and then at least one go trial. GG trials are controls that provide a baseline for assessing post-stop-signal adjustments. GSG trials are critical, as these are the trials in which the expectation of a second stop trial may be violated, withholding an expected stop signal in the 80% condition or presenting an unexpected stop signal in the 20% condition, leading to increases and reductions in post-stop-signal slowing, respectively. If subjects slow reflexively after stop signals, the same post-stop-signal slowing should be observed in both conditions. GSSG trials are interesting because there can never be three consecutive stop trials, so if subjects learn this conditional probability, there should be no post-stop-signal slowing or maybe even post-stop-signal speeding. If subjects slow reflexively after stop signals, GSSG trials should be slower than GG trials. GSSG post-stop-signal adjustments may replicate the Experiment 1 contingency condition, as the conditional probability of a stop signal in both cases is 0. The effect of conditional probability may be weaker for the GSSG trials here than the contingency condition in Experiment 1. Here, the differences between the zero probability of three stop signals in a row and the .036 ($.33 \times .33 \times .33$) probability that subjects would expect if stop trials were entirely random is smaller than the difference in the Experiment 1 contingent condition between 0 and .0625 ($.25 \times .25$). The degree to which the contingency violates subjects' expectations of randomness may affect post-stop-signal slowing.

Mean RTs from correct no-stop-signal trials were calculated for each cell of a 2 (Repetition Probability: 80% or 20%) $\times$ 3 (Go Trial Sequence: GG, GSG, or GSSG) $\times$ 2 (Group: changed or unchanged SSD) experimental design, including only RTs from correct trials that were shorter than 1850 ms (shape presentation time plus ITI) and subjected to ANOVA in which Repetition Probability and Go Trial Sequence were within-subject and Group was between-subjects. The summary table for the ANOVA appears in Table 2. Mean RTs across subjects appear in Figure 2. We focused on planned comparisons using the error terms from the ANOVA to evaluate our results.

The results from both groups show that contingencies affect post-stop-signal slowing: when stop trial repetitions were frequent (80% repetition), subjects slowed considerably on trials after an isolated stop signal (GSG), anticipating the need to stop. For the changed SSD group, GSG RT (622 ms) was longer than GG RT

Table 2
Summary Table for Analyses of Variance Performed on Experiment 2 Mean Reaction Times

Measure	*df*	*MSE*	*F*
Repetition Probability	1, 30	926	48.07**
Repetition Probability × Group	1, 30	926	2.76
Go Trial Sequence	2, 60	4044	32.98**
Go Trial Sequence × Group	2, 60	4044	.28
Group	1, 30	105458	.01
Repetition Probability × Go Trial Sequence	2, 60	2401	39.99**
Repetition Probability × Go Trial Sequence × Group	2, 60	2401	4.10*

Note. *df* = Degrees of freedom; *MSE* = Mean squared error.
* $p < .05$. ** $p < .01$.

(488 ms), $F(1, 60) = 59.83$, $MSE = 2401$, $p < .01$; for the unchanged SSD group, GSG RT (584 ms) was longer than GG RT (496 ms), $F(1, 60) = 25.72$, $MSE = 2401$, $p < .01$. However, subjects did not slow after two consecutive stop signals (GSSG) in the 80% condition (changed SSD group $M = 482$ ms, $F(1, 60) < 1$; unchanged SSD group $M = 485$ ms, $F(1, 60) < 1$), replicating the Experiment 1 contingency results.

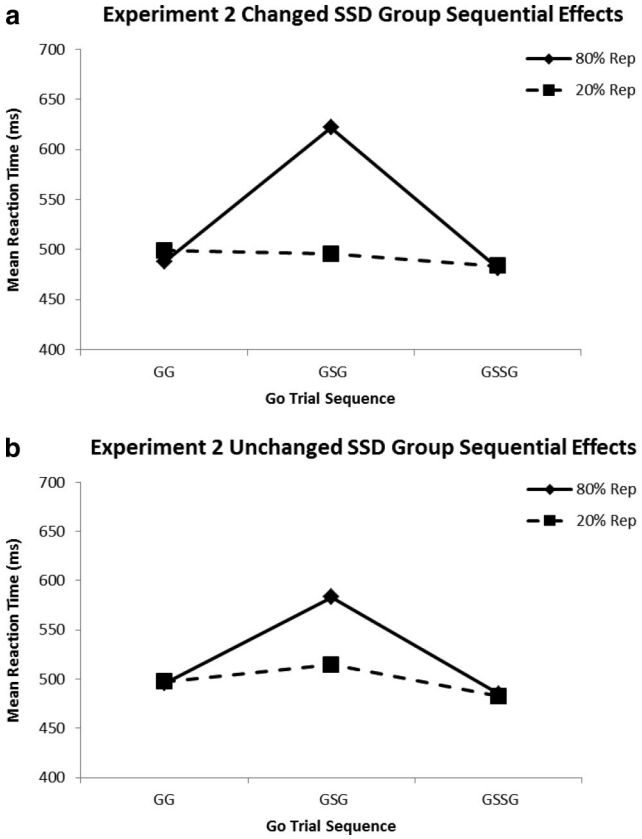

Figure 2. Experiment 2 changed SSD group (a) and unchanged SSD group (b) mean reaction time in the 80% and 20% stop trial repetition conditions separated by Go Trial Sequence, namely go trials preceded by at least one go trial (GG), go trials preceded by a single stop trial then at least one go trial (GSG), and go trials preceded by two stop trials then at least one go trial (GSSG).

Neither group slowed after stop signals in the 20% condition. For the changed SSD group, GSG RT (496 ms) did not differ from GG RT (499 ms), $F(1, 60) < 1$, and GSSG (484 ms) RT did not differ from GG RT (499 ms), $F(1, 60) < 1$. For the unchanged SSD group, GSG RT (515 ms) did not differ from GG RT (497 ms), $F(1, 60) = 1.00$, $MSE = 2401$, $p > .3$, and GSSG (483 ms) RT did not differ from GG RT (497 ms), $F(1, 60) < 1$. If subjects implement a strategy or implicitly learn the task contingencies, we might expect this elimination of post-stop-signal slowing in both GSG and GSSG trials because the probability of a stop trial after a single stop trial (.2) or after two stop trials (0) is lower than the average experiment-wide probability of a stop trial (.33).

We also investigated the effect of stop-signal repetition on the stop process. Using the integration method (Logan & Cowan, 1984), we calculated SSRT separately for the first stop signals (either in pairs or isolated) and second stop signal in pairs. We computed a 2 (Place in Pair: first or second) × 2 (Repetition Probability: 80% or 20%) × 2 (Group: changed or unchanged SSD) ANOVA for SSRT. There were no significant main effects or interactions in the ANOVA (all Fs < 1.2; see Table 3 for mean SSRTs), suggesting that the stop process is unaffected by Place in Pair or Repetition Probability.

Conclusions

These results replicate Experiment 1 by showing that stop-signal contingencies strongly influence post-stop-signal slowing. When subjects expect a stop signal to occur (80% repetition GSG trials), they show huge post-stop-signal slowing values, and when subjects do not expect a stop signal, post-stop-signal slowing is eliminated (20% repetition GSG trials, all GSSG trials).

Experiment 3

Experiments 1 and 2 showed that post-stop-signal slowing is not reflexive, as contingencies can reverse, eliminate, or greatly increase post-stop-signal slowing. An open question is whether the adjustments are a result of explicit strategies or implicit learning. The results of the first two experiments provided some preliminary suggestions, but Experiment 3 was aimed to answer this question more directly. Experiment 3 manipulated the probability of stop-signal repetition, but the block lengths were greatly reduced. Every 21 trials, blocks alternated between frequent repetitions (75% stop

Table 3
Experiment 2 Mean SSRT Summary Table

Condition	Mean SSRT (ms)
Changed SSD 80% repetition first SSRT	193
Changed SSD 80% repetition second SSRT	207
Changed SSD 20% repetition first SSRT	204
Changed SSD 20% repetition second SSRT	199
Unchanged SSD 80% repetition first SSRT	203
Unchanged SSD 80% repetition second SSRT	194
Unchanged SSD 20% repetition first SSRT	206
Unchanged SSD 20% repetition second SSRT	197

Note. SSRT = Stop-signal reaction time.

trial repetition) and infrequent repetition (approximately 17% stop trial repetition). Experiment 3 also manipulated the cuing of repetition probability between groups. The *cued group* was cued explicitly about repetition probability at the beginning of each block, but the *uncued group* was not. The cued group should benefit from strategic changes and, possibly, learning, whereas the uncued group could only benefit from learning. The only difference between the cued and uncued groups was the presence of the cues, so differences between them must be attributable to the cues and nothing else. To test for explicit learning, subjects were given a postexperiment debriefing that inquired about explicit knowledge gained throughout the session. If subjects have no knowledge of the repetition probability manipulation when asked at debriefing, we can conclude that any learning was implicit and that differences in post-stop-signal slowing between frequent and infrequent repetition blocks in the uncued group are attributable to implicit learning. Differences between the cued and the uncued condition suggest an effect of strategy above and beyond implicit learning. We also assessed the degree to which post-stop-signal slowing was present in the first trial in the block to further distinguish strategic adjustment from implicit learning. Strategic adjustment should occur on the first trial in a block, whereas implicit learning should require several trials to develop.

Method

Subjects. Thirty-two subjects were recruited from the Nashville area and were compensated $18 for a single 90-min experimental session. All subjects had normal or corrected-to-normal vision. There were 16 subjects in each group. We replaced one subject whose probabilities of successful stopping fell outside the 95% confidence interval of .5 probability of stopping to a stop signal, and one subjects for low go accuracy (<85%).

Apparatus and stimuli. The apparatus and stimuli were the same as in Experiment 2.

Procedure. The basic trial structure and instructions for Experiment 3 were identical to Experiment 2 with the following exceptions: blocks were only 21 trials long, and there were 80 blocks in total. SSD was tracked separately for the frequent and infrequent repetition blocks. Frequent repetition blocks had exactly three GSSG instances and one GSG instance, making the probability of stop trial repetition .75. Infrequent repetition blocks had exactly one GSSG instance and exactly five GSG instances, making the probability of a stop trial repetition approximately .17. Like Experiment 2, there were never more than two signals in a sequence, so the probability of stop signal repetition given two stop signals have occurred was 0 for both block types. Frequent and infrequent repetition blocks alternated every 21 trials. There were 80 blocks of 21 trials, totaling 1680 trials, 560 of which were stop-signal trials. SSD changed after every stop signal, mirroring Experiment 1 and the changed group from Experiment 2.

In the cued group, every 21 trials a cue of "frequent rep" or "infrequent rep" would be presented centrally for 500 ms for the final 500 ms of the 1000 ms ITI period. These cues told subjects whether they were entering a frequent repetition or infrequent repetition block of 21 trials. For the uncued group, there were never any cues, but the alternation between frequent and infrequent repetition blocks every 21 trials remained. All subjects were given a debriefing form at the end of the experiment asking whether they explicitly learned this (or any) contingency in the task.

Results and Discussion

We first evaluated the effect of the repetition probability manipulation on performance disregarding sequential effects. We computed three separate 2 (Repetition Probability: frequent or infrequent) × 2 (Cue: cued or uncued) ANOVAs for mean go RT for correct trials, go task accuracy, and probability of response given a stop signal. Repetition Probability was a within-subject factor, and Cue was a between-subjects factor. There were no significant main effects or interactions in any of the ANOVAs (all ps $>.25$).

Similar to Experiment 2, Mean RTs from no-stop-signal trials were calculated for each cell of a 2 (Repetition Probability: frequent or infrequent) × 3 (Go Trial Sequence: GG, GSG, or GSSG) × 2 (Cue: cued or uncued) experimental design, including only RTs from correct trials that were shorter than 1850 ms (shape presentation time plus ITI) and subjected to ANOVA in which Repetition Probability and Go Trial Sequence were within-subject and Cue was between-subjects. The summary table for the ANOVA appears in Table 4. Mean RTs across subjects appear in Figure 3. We focused on planned comparisons using the error terms from the ANOVA to evaluate our results.

The results from the cued group showed a strong effect of expectancy (see Figure 3a). In the frequent repetition blocks, RT was much slower on the unexpected GSG trials ($M = 593$ ms) than on the control GG trial sequence ($M = 481$ ms), $F(1, 60) = 193.11$, $MSE = 515$, $p < .01$. However, there was no significant slowing after two consecutive stop trials (GSSG $M = 491$ ms vs. GG $M = 481$ ms), $F(1, 60) = 1.28$, $MSE = 515$, $p > .25$. In the infrequent repetition blocks, when compared with control GG trials ($M = 485$ ms), there was no slowing after either a single stop trial (GSG $M = 495$ ms), $F(1, 60) = 1.59$, $MSE = 515$, $p > .2$, or after two consecutive stop trials (GSSG $M = 489$ ms), $F(1, 60) < 1$. This shows that slowing was only observed in the GSG sequence in the frequent repetition condition, the one condition that involves a strong expectation of a stop signal. This suggests that subjects implemented an explicit strategy that changed every 21 trials. To bolster this claim, in debriefing, all subjects had explicit

Table 4
Summary Table for Analyses of Variance Performed on Experiment 3 Mean Reaction Times

Measure	*df*	*MSE*	*F*
Repetition Probability	1, 30	852	12.08**
Repetition Probability × Cue	1, 30	852	16.72**
Go Trial Sequence	2, 60	748	28.17**
Go Trial Sequence × Cue	2, 60	748	21.64**
Cue	1, 30	89303	.02
Repetition Probability × Go Trial Sequence	2, 60	515	29.21**
Repetition Probability × Go Trial Sequence × Cue	2, 60	515	21.72**

Note. *df* = Degrees of freedom, *MSE* = Mean squared error.
* $p < .05$. ** $p < .01$.

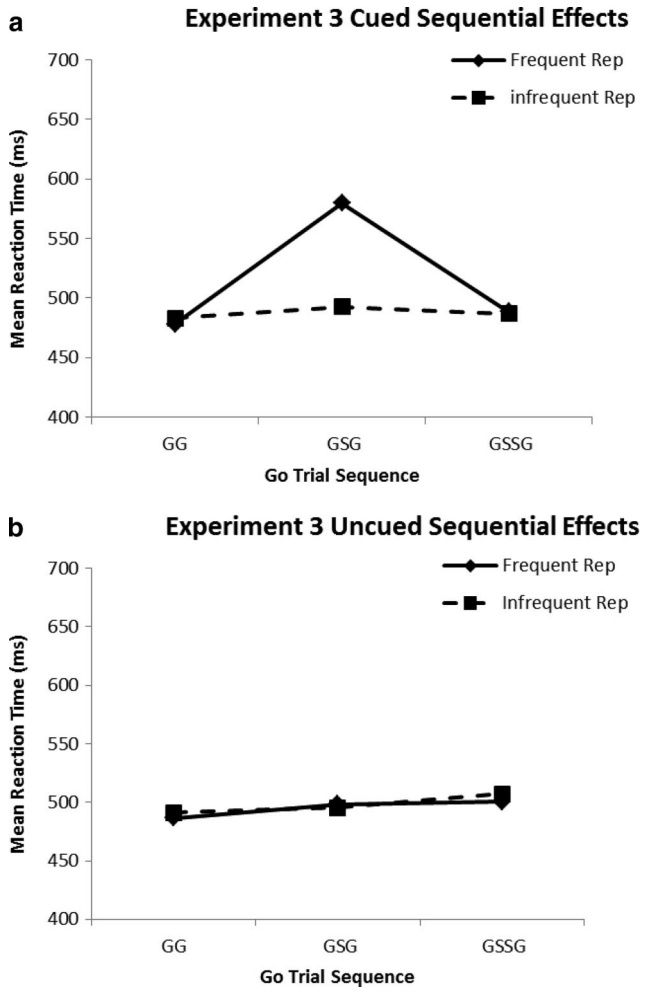

Figure 3. Experiment 3 cued (a) and uncued (b) mean reaction time in the frequent repetition and infrequent repetition conditions separated by go trials preceded by at least one go trial (GG), go trials preceded by a single stop trial then at least one go trial (GSG), and go trials preceded by two stop trials then at least one go trial (GSSG).

knowledge of the adjustment in repetition probability every 21 trials, which is not surprising because it was explicitly cued.

The results from the uncued group showed no effect of expectancy (see Figure 3b). In frequent repetition blocks, compared with the control GG trial sequence (M = 489 ms), the trend toward slowing after the unexpected single stop signals (GSG M = 503 ms), $F(1, 60) = 2.79$, $MSE = 515$, $p = .1$, was similar to the trend toward slowing after the more frequently occurring two consecutive stop signals (GSSG M = 504 ms), $F(1, 60) = 2.99$, $MSE = 515$, $p = .09$. In the infrequent repetition blocks, there was no significant slowing after a single stop signal (GSG M = 501 ms vs. GG M = 495 ms), $F(1, 60) < 1$, but there was a trend toward slowing after two consecutive stop signals (GSSG M = 509 ms vs. GG M = 495 ms), $F(1, 60) = 2.90$, $MSE = 515$, $p = .09$. There was very little difference in post-stop-signal slowing across repetition probability and number of preceding stop signals (GSG or GSSG), and these results mirror the modest post-stop-signal slowing usually observed in the stop-signal paradigm. Indeed, if we collapse across repetition probability and number of stop trials, and compare RT after at least one go trial (GG M = 489 ms) to RT after at least one stop trial (GSG or GSSG RT M = 500 ms), we observe statistically significant post-stop-signal slowing, $t(15) = 2.81$, $p < .05$. This suggests that subjects did not learn the contingency, and subjects acted similar to the way they do when stop trials are presented randomly. In debriefing, no subjects were able to make a correct statement about the contingency in the task, suggesting that any learning would have been implicit.

To distinguish strategies from implicit learning more clearly, we looked at how the post-stop-signal adjustments unfolded within blocks, examining each possible GSG stop sequence within a block. Strategic adjustments should occur immediately after the cue and persist throughout the block, whereas implicit learning adjustments should not be affected by the cue and may develop across the block. To equate number of stop signals in a block, which has been shown to affect post-stop-signal slowing (Bissett & Logan, 2011), we needed to use a different number of stop trial sequences across frequent and infrequent repetition blocks. Consequently, we compared the first four possible GSG instances in infrequent repetition blocks to the only four possible GSG instances within frequent repetition blocks. This resulted in a 4 (Place in Sequence: 1–4) × 2 (Repetition Probability: frequent or infrequent) × 2 (Cue: cued or uncued) ANOVA design of post-stop-signal slowing. For this analysis, we calculated GSG post-stop-signal slowing by subtracting go RT on the trial before a stop trial (the first G) from RT on the trial after a stop trial (the second G, see Bissett & Logan, 2011; Nelson, Boucher, Logan, Palmeri, & Schall, 2010). Positive values reflect post-stop-signal slowing, and negative values reflect post-stop-signal speeding. Figure 4a shows the results for the cued condition, and Figure 4b shows the results from the uncued condition. From this ANOVA, we tested learning effects with a linear contrast across the four possible GSG sequences (within each block) in each repetition and cueing condition, separately. Learning should result in increased slowing over the course of the block in the frequent repetition condition and decreased slowing (or speeding) in the infrequent repetition condition. Post-stop-signal slowing did not increase within blocks in either the cued or uncued frequent repetition condition, $F(1, 90) < 1$, and slowing did not decrease within blocks in either the cued or uncued infrequent repetition condition, $F(1, 90) < 1$. This suggests that there was no learning within blocks. Together, these results suggest that the differences we observe between conditions, namely, large GSG slowing in the frequent repetition cued condition and little or no slowing in the other conditions, are present within all stop trials within a block, suggesting that there is no effect of implicit learning within blocks. The cue is what dictates the adjustment of post-stop-signal slowing, suggesting strategies are paramount.

The previous analysis showed that there were no effects of implicit learning within blocks, but there could be implicit learning across blocks. Implicit learning of the task contingencies predicts greater slowing across blocks in the frequent repetition condition and reduced slowing or speeding in the infrequent repetition condition. To test this prediction, we separated the experiment in four sets of 10 blocks for each repetition probability and ran a 4 (Quarter of Experiment: 1–4) × 2

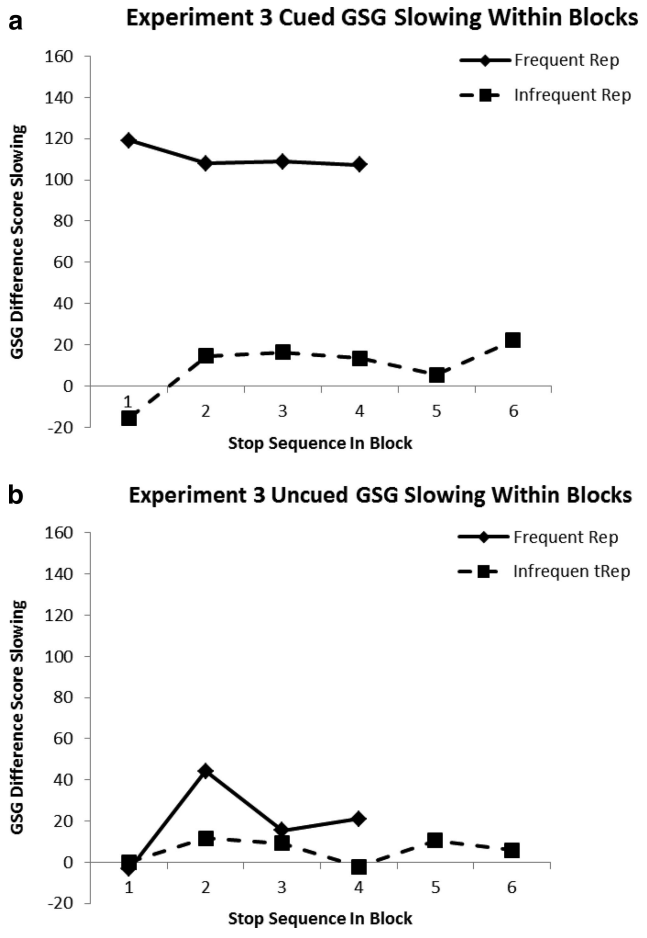

Figure 4. Experiment 3 cued (a) and uncued (b) post-stop-signal slowing (calculated as RT on the go trial after a single stop trial minus RT on the go trial preceding a trial stop trial) separated by Repetition Probability (frequent repetition vs. infrequent repetition) and where the GSG sequence occurs in the block, from sequence one through six.

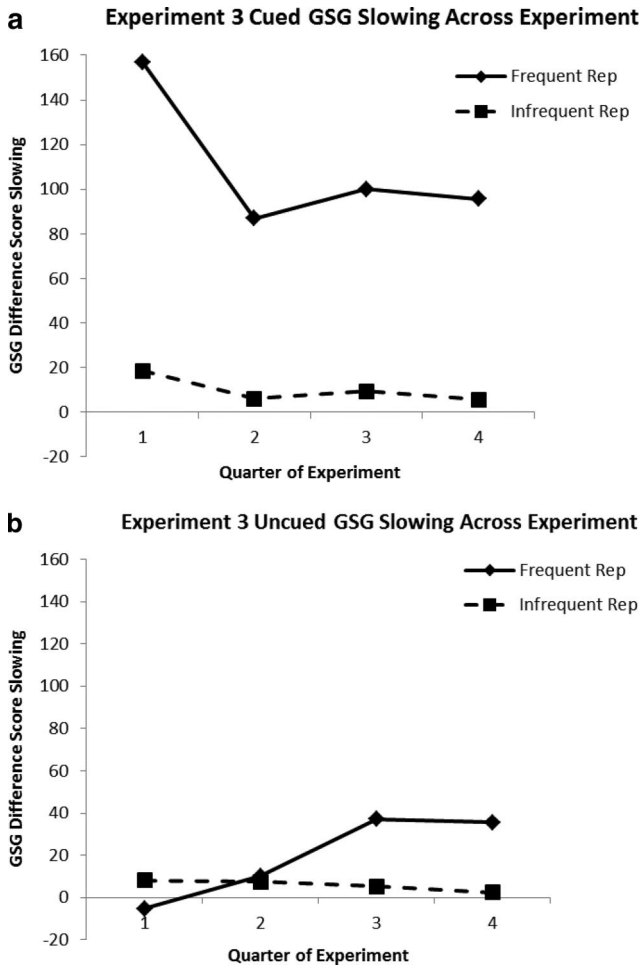

Figure 5. Experiment 3 cued (a) and uncued (b) post-stop-signal slowing (calculated as RT on the go trial after a single stop trial minus RT on the go trial preceding a single stop trial) separated by Repetition Probability (frequent repetition vs. infrequent repetition) and quarter of the experiment (1–4) that the GSG sequence occurred within.

(Repetition Probability: frequent or infrequent) × 2 (Cue: cued or uncued) ANOVA on post-stop-signal slowing difference scores. Figure 5a shows the results for the cued condition, and Figure 5b shows the results from the uncued condition. We calculated linear contrasts across the four quarters of the experiment in each Repetition Probability and Cue condition. Post-stop-signal slowing did not increase across blocks in the cued frequent repetition condition, $F(1, 90) < 1$, and slowing did not decrease across blocks in either the cued or uncued infrequent repetition condition, $F(1, 90) < 1$. However, slowing did increase across blocks in the uncued frequent repetition conditions, $F(1, 90) = 15.58$, $MSE = 1289$, $p < .01$, which suggests some learning occurred. No subjects in the uncued condition recognized the repetition probability manipulation in debriefing, so the learning in the uncued condition was implicit and not explicit.

We also investigated the effect of stop-signal repetition on the stop process, with the same procedure as in Experiment 2. We computed a 2 (Place in Pair: first or second) × 2 (Repetition Probability: frequent or infrequent) × 2 (Cue: cued or uncued) ANOVA on SSRT (Mean SSRT values presented in Table 5). Here we observed a significant main effect of Repetition Probability, $F(1, 30) = 6.03$, $MSE = 633$, $p < .05$, in which mean SSRT in the frequent repetition conditions ($M = 214$ ms) was faster than mean SSRT in the infre-

Table 5
Experiment 3 Mean SSRT Summary Table

Condition	Mean SSRT (ms)
Cued frequent repetition first SSRT	208
Cued frequent repetition second SSRT	187
Cued infrequent repetition first SSRT	218
Cued infrequent repetition second SSRT	217
Uncued frequent repetition first SSRT	230
Uncued frequent repetition second SSRT	230
Uncued infrequent repetition first SSRT	238
Uncued infrequent repetition second SSRT	225

Note. SSRT = Stop-signal reaction time.

quent repetition conditions ($M = 225$ ms). We also observed a significant interaction between Repetition Probability and Cue, $F(1, 30) = 4.31$, $MSE = 633$, $p < .05$, which suggests that the main effect of Repetition Probability may be driven by shorter SSRT in the cued condition. Finally, there was a significant main effect of Place in Pair, $F(1, 30) = 4.86$, $MSE = 537$, $p < .05$, in which the second SSRT in a pair is faster ($M = 215$ ms) than the first SSRT in a pair (224 ms). The three-way interaction of Repetition Probability, Place in Pair, and Cue was borderline significant, $F(1, 30) = 4.04$, $MSE = 478$, $p = .053$, and the main effect of cue trended toward significance $F(1, 30) = 2.99$, $MSE = 5834$, $p = .09$. These results suggest that cueing improves SSRT on frequent repetition blocks, and SSRT may speed if the previous trial is also a stop trial. These results were not observed in the similar analysis for Experiment 2, suggesting that additional research is necessary to clarify this result.

Conclusion

Experiment 3 showed that large adjustments in post-stop-signal slowing are present on the very first trial after the repetition probability cue, and these adjustments remain throughout the block and across the experiment. This suggests that strategic adjustments dominate the post-stop-signal slowing results presented here. Additionally, implicit learning appears to have a modest effect across the course of the 90-min session, increasing GSG slowing in frequent repetition blocks. This suggests that implicit learning plays a secondary role to strategic adjustments.

General Discussion

These three experiments showed that conditional probabilities have large effects on post-stop-signal slowing. In the contingent condition of Experiment 1, when stop trials never repeated, subjects either sped up (instructed group) or failed to slow down (uninstructed group) after stop signals. In Experiment 2, when stop trials repeated 80% of the time, subjects showed huge post-stop-signal slowing effects after a single stop trial but no post-stop-signal slowing after a single stop signal when stop signals repeated 20% of the time. These two experiments showed that post-stop-signal slowing is not reflexive consequence of the stop signal and is greatly influenced by strategies or implicit learning. Experiment 3 showed that these large adjustments occur immediately after a cue, suggesting that the large adjustments are strategic and not attributable to implicit learning. Subjects who were not cued gradually adjusted post-stop-signal slowing in accord with the contingencies over the course of the experiment, suggesting implicit learning can have some effect in long sessions. Together, these results show post-stop-signal adjustments are predominantly strategic, with implicit learning playing a smaller role.

Post-Stop-Signal Slowing Is Not Reflexive Here. Is It Ever Reflexive?

Existing hypotheses for sequential adjustments in the stop-signal paradigm are simple and well constrained, in that control adjustments are driven by the immediately preceding trial. Post-stop-signal slowing can be explained as a response to the erroneous (Rieger & Gauggel, 1999; Schachar et al., 2004; Verbruggen et al., 2008), conflicting (Emeric et al., 2007), or surprising (Notebaert et al., 2009) nature of the previous trial. In the present experiments, we did not reduce the number or salience of stop errors (signal-respond trials) or reduce conflict between competing responses. We also did not manipulate the overall probability of a stop trial, which should be the primary determinant of its surprise level. It is possible that surprise is driven by conditional probabilities, but this would require rather sophisticated calculations that may be conscious and strategic. Currently, the surprise hypothesis is explained as an orienting reflex, so strategic conditional probability computations do not seem consistent with this reflexive explanation. Consequently, we would expect no differences in post-stop-signal slowing in any of our experiments. However, our results showed profound differences that occurred before experiencing any stop trials, which suggests that strategies are the prime driver of post-stop-signal adjustments.

The simplest explanation is that all post-stop-signal adjustments are strategic. The observation that post-stop-signal slowing is usually consistent and modest may reflect the fact that the strategic balance between going and stopping does not need to change drastically. This conceptualization of the stop-signal task as a decision making task affected by probabilities, payoffs, and motivation is becoming more common (Bissett & Logan, 2011; Leotti & Wager, 2010; Liddle et al., 2009; Padmala & Pessoa, 2010; Shenoy, Rao, & Yu, 2010). Subjects may strategically adjust speed and accuracy in an attempt to optimize utility, and some of these researchers have begun to specify utility functions that could be optimized in this task environment (Liddle et al., 2009; Shenoy et al., 2010). From this perspective, the strong conditional probability manipulations in our experiments may have changed the value of fast and slow responses and subjects may have adjusted their performance to increase their expected gains. This same attempt to optimize utility should be present in the usual stop-signal paradigm, but the adjustments in reaction time may be smaller to match the more constant task environment.

This is not to say that errors, conflict, or surprise are not involved in the stop-signal task. Instead, it suggests that these factors on the stop trial do not reflexively drive behavioral adjustments. These factors may be registered, but the behavioral response to them is a strategic choice. Our previous research suggests that the dominant strategy in the typical stop signal paradigm is to slow go reaction time to a similar degrees after all stop signals, which we discuss as a goal priority shift from going to stopping (Bissett & Logan, 2011). However, errors, conflict, or surprise could lead to strategic adjustments in some situations. In fact, some researchers have concluded that stop errors (Rieger & Gauggel, 1999; Schachar et al., 2004; Verbruggen et al., 2008) or successful inhibition, described as conflict (Emeric et al., 2007), may be the primary drivers of post-stop-signal slowing. Some of these effects may have resulted from using an inappropriate baseline to assess post-stop-signal slowing (Bissett & Logan, 2011; Nelson et al., 2010), but an alternative explanation is they reflect differences in strategy driven by differences in experimental factors, instructions, or subjects. An interesting avenue for future research would be a meta-analysis of stop-signal studies that measure post-stop-signal slowing, to investigate whether different experimental factors, like stop-signal probability, SSDs being fixed or adjusted by tracking, and stop trial outcome, result in different post-stop-signal strategies.

We cannot rule out the possibility that reflexive post-stop-signal slowing always occurs but is enhanced or counteracted by strategy. To justify a more complex model that involves both reflexive and strategic adjustments, one would need to show that the reflexive component of post-stop-signal slowing is invariant across conditions while the strategic component varies. Similar approaches have successfully separated reflexive and strategic components of the Stroop task (Logan, 1980) and retrieval from episodic memory (Jacoby, 1991). Future research will be required to evaluate the plausibility of a two-process model of post-stop-signal slowing.

One special case of post-stop-signal slowing is the greater slowing that is observed if the go stimulus from a stop trial repeats in a go trial later in the experiment (Bissett & Logan, 2011; Verbruggen & Logan, 2008; Verbruggen et al., 2008). Verbruggen and Logan (2008) showed that stimulus repetition costs can occur if as many as 20 trials intervene between the first presentation and the second. This effect is probably better described as automatic than reflexive (Logan, 1988), but automatic processes are often regarded as reflexive (Moors & De Houwer, 2006). It is not clear how these memory effects are affected by strategies, but this would be an interesting line of future research.

Errors, Conflict, and Cognitive Control

The stop-signal paradigm is well-suited to investigate sequential control adjustments, as errors can be manipulated independently from the primary go task. But there is a long history of research on sequential control adjustments that has not centered on the stop-signal paradigm, with two of the most investigated theoretic frameworks being error monitoring (Laming, 1968; Rabbit, 1966) and conflict monitoring (Botvinick et al., 2001; Carter et al., 1998). Early error monitoring research suggested that subjects slowed their responses after errors by increasing the threshold for their response. This threshold adjustment traded speed for accuracy, strategically reducing the probability of committing future errors. But some studies suggest that accuracy does not always increase after errors (Hajcak & Simmons, 2008; Rabbitt & Rodgers, 1977). Other studies suggest that accuracy decreases after errors (Gehring, Goss, Coles, Meyer & Donchin, 1993) because of the persistence of the malfunctioning process from the preceding trial, which is a more reflexive explanation. Notebaert and colleagues (2009) suggest that errors slow RT because they are surprising, and surprising stimuli reflexively orient attention away from the main task. This evidence seems to leave open the question of whether post-error processing is automatic or reflexive. As is discussed in the previous section, greater slowing after failed inhibition has been observed in some studies (Rieger & Gauggel, 1999; Schachar et al., 2004; Verbruggen et al., 2008) but not in others (Bissett & Logan, 2011; Emeric et al., 2007), suggesting that errors may not be the primary determinant of post-stop-signal slowing. One explanation for why errors do not drive post-stop-signal strategies is that subjects may be less concerned with correcting errors because they are so frequent (Gehring et al., 1993). They often occur on half of stop-signal trials. It is also possible that post-error slowing occurs after errors of choice but not after errors of timing. In the stop-signal paradigm, failures to inhibit involve errors of timing (responding too quickly), not errors of choice.

Another explanation for post-error adjustments is that error trials involve conflict between competing responses (Botvinick, 2001; Carter et al., 1998). The conflict between competing responses recruits control processes, which result in a more cautious response mode that increases RT. This conflict framework is especially attractive because it explains how conflict is recruited in a variety of circumstances, not simply on error trials, and accounts for performance in other cognitive control tasks, like the Stroop and flanker task (Botvinick et al., 2001).

Schall and colleagues have applied the conflict framework to countermanding eye-movements in monkeys (Emeric et al., 2007; Ito, Stuphorn, Brown, & Schall, 2003; Stuphorn & Schall, 2006; Stuphorn, Taylor, & Schall, 2000). They identified conflict with the coactivation of movement-related neurons and fixation-related neurons and proposed that the greatest conflict occurs on successful inhibition trials, because both fixation and movement neurons are active, which is not the case on failed inhibition trials. This suggests that post-stop-signal slowing should be greater after successful than after failed inhibition, but this result is not generally found (Bissett & Logan, 2011; Rieger & Gauggel, 1999; Schachar et al., 2004; Verbruggen et al., 2008) and their own results that led them to this conclusion (Emeric et al., 2007) were probably an artifact of using inappropriate baselines (Nelson et al., 2010). This is strong evidence against the existing conceptualization of conflict in the stop-signal paradigm proposed by Schall and colleagues. It is possible that monkey results do not generalize to humans or eye-movement results do not generalize to keypress responses. Another possibility is that coactivation of fixation and movement neuron activity does not index response conflict. These neurons are coactive at the beginning and end of every saccade (Hanes, Patterson & Schall, 1998), which suggests that coactivation of fixation and movement neurons may be insufficiently specific to index conflict.

This is not to say that conflict is not involved in the stop-signal paradigm, as conflict may occur differently than proposed by Schall and colleagues. The race model (Logan & Cowan, 1984) assumes that the stop process and the go process are engaged on every stop-signal trial, and that engagement may produce conflict regardless of which process finishes first. This is consistent with the finding of similar post-stop-signal slowing after stop success and stop errors (Bissett & Logan, 2011). But as we discuss above, conflict from the immediately preceding trial does not drive post-stop-signal slowing reflexively, as the amount of conflict should be the same across stop trials, but we found huge differences in post-stop-signal slowing across conditional probabilities. Additional research will be necessary to provide alternative explanations of how conflict may act in the stop-signal paradigm.

Stop-Signal Paradigm and Speed-Accuracy Tradeoff

The stop-signal paradigm can be conceived as a speed–accuracy trade-off task (Fitts, 1966; Wickelgren, 1977), with go task speed pitted against accuracy. In the stop-signal paradigm, accuracy takes two forms: go task choice accuracy and stop task accuracy (or inhibition probability). Previous experiments have shown that RT increases with the probability of a stop trial, suggesting that go task speed is traded-off to increase stop accuracy (Bissett & Logan, 2011; Logan, 1981; Logan & Burkell, 1986; Verbruggen & Logan, 2009). The present experiments have shown that RT increases after stop trials in proportion to the conditional probability that a stop signal will occur on the next trial. This also suggests that go task speed is traded for stop-task accuracy. One point that we alluded

to earlier is that failures of inhibition are errors of timing and not errors of choice, as subjects tend to pick the correct keypress response but do so too quickly to correctly stop. Thus, the results from the stop-signal paradigm suggest that go-task speed can be traded off to reduce errors of timing. This result can be explained by the race model (Logan & Cowan, 1984): An increase in go task finishing time allows more time for the stop process to finish, and that increases the chance of successful inhibition. One challenge to this simple explanation is the tracking algorithm for SSD, which adjusts the starting time of stopping in relation to going to attempt to equate their finishing times. When subjects succeeded in stopping, their SSD increased by 50 ms, so to consistently succeed in stopping subjects must increase their go reaction time by more than 50 ms. Our data show that when subjects expect a stop signal (See GSG RTs in Figures 2a, 2b, and 3a), they slow more than 50 ms when compared with the baseline GG trials. As expected, this results in high successful inhibition rates when highly likely stop signals occur, even when SSD has just increased on the previous trial because of successful inhibition (Stopping rates after one successful stop in Experiment 2 changed group = 70%, Experiment 2 unchanged group = 68%, Experiment 3 cued group = 67%, all rates higher than mean inhibition rates in each experiment, $p < .01$). In a normal stop-signal paradigm without contingencies, post-stop-signal slowing is typically smaller and is not large enough to circumvent the tracking algorithm in this way.

Another way that subjects may trade going for stopping is by trading speed in the go task (RT) for speed in the stop task (SSRT). If subjects can trade go speed for stopping speed, one might expect SSRT to improve when subjects expect a stop signal, as in the second stop signal in pairs in the 80% repetition conditions in Experiment 2 and the cued frequent repetition condition in Experiment 3. The results here are mixed, with no improvement in SSRT on the second stop trial in pairs (compared with the first) in the 80% repetition condition of Experiment 2, but a planned comparison showed an improvement in SSRT in the second stop trials in pairs (compared with the first) in the cued frequent repetition condition of Experiment 3, $F(1, 30) = 7.38$, $MSE = 478$, $p < .05$. This suggests additional research to more directly test whether SSRT improves after stop signals, which would examine the degree to which speed in the go task can be traded for stop processing.

The Role of Implicit Learning in Post-Stop-Signal Slowing

The uninstructed condition in Experiments 1 and the uncued condition in Experiment 3 assessed whether subjects could learn the conditional probabilities and use them to adjust behavior. Post-stop-signal slowing was eliminated in the uninstructed contingent condition of Experiment 1, but without a debriefing questionnaire for that study we could not assess whether the learning was implicit or explicit. The uncued condition in Experiment 3, which included debriefing, was a better test of implicit learning, and we showed that subjects began to show greater GSG slowing in the frequent repetition condition than the infrequent repetition condition as the experiment progressed.

An interesting avenue for future research would be to extend the length of the uncued session. It is not clear from the current data (see Figure 5b) whether the difference between the frequent and infrequent repetition condition reached asymptote. In a longer experiment, this difference may become as large as the difference observed in the cued condition, suggesting that strategic changes are not stronger than changes based on implicit learning, but simply can be implemented more quickly. We speculate that this will not be the case, as Experiment 3 was already quite long (almost 1700 trials), and the effects of learning are usually strongest early in practice (Logan, 1988; Newell & Rosenbloom, 1981).

Proactive and Reactive Control: Two Sides of the Same Coin?

In the stop-signal literature, researchers often distinguish between proactive adjustments that precede stop signals (Chikazoe et al., 2009; Logan & Burkell, 1986; Verbruggen & Logan, 2009) and reactive adjustments that immediately follow stop signals (Emeric et al., 2007; Rieger & Gauggel, 1999; Verbruggen et al., 2008). Proactive adjustments are assumed to be strategic, based on subjects' estimates of stop signal probability (Bissett & Logan, 2011; Logan & Burkell, 1986) or on subjects' responses to cues indicating stop signal relevance (Chikazoe et al., 2009; Verbruggen & Logan, 2009). Reactive adjustments are often assumed to be reflexive, but they need not be, as strategic adjustments can occur as a reaction to an event such as a stop-signal trial. The present results resemble proactive effects in that they are strategic, but they resemble reactive adjustments in that they occurred immediately after stop signals. This suggests that the usual assumption that reactive adjustments are reflexive is not always valid, as both post-stop-signal and proactive adjustments are strategic and responsive to expectancy.

We have previously suggested that post-stop-signal slowing is the result of a shift in goal priority from going to stopping (Bissett & Logan, 2011). We suggest that the driver of this goal priority shift is the subjective probability of the next trial being a stop signal and the value of stopping and going. If this is the case, then goal priority can shift after a stop signal (reactive slowing) or before a stop signal (proactive slowing). This basic framework may be able to explain longer mean RT and greater post-stop-signal slowing when the probability of a stop signal is higher (Bissett & Logan, 2011) as well as the results in this article showing large post-stop-signal slowing when stop signals are likely (Experiments 2 and 3) and post-stop-signal speeding when stop signals are impossible (Experiment 1). Additional research is necessary to flesh out this account of control adjustments.

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