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A startling acoustic stimulus interferes with upcoming motor preparation: Evidence for a startle refractory period

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Abstract

When a startling acoustic stimulus (SAS) is presented in a simple reaction time (RT) task, response latency is significantly shortened. The present study used a SAS in a psychological refractory period (PRP) paradigm to determine if a shortened RT1 latency would be propagated to RT2. Participants performed a simple RT task with an auditory stimulus (S1) requiring a vocal response (R1), followed by a visual stimulus (S2) requiring a key-lift response (R2). The two stimuli were separated by a variable stimulus onset asynchrony (SOA), and a typical PRP effect was found. When S1 was replaced with a 124 dB SAS, R1 onset was decreased by 40-50 ms; however, rather than the predicted propagation of a shortened RT, significantly longer responses were found for RT2 on startle trials at short SOAs. Furthermore, the 100 ms SOA condition exhibited reduced peak EMG for R2 on startle trials, as compared to non-startle trials. These results are attributed to the startling stimulus temporarily interfering with cognitive processing, delaying and altering the execution of the second response. In addition to this "startle refractory period," results also indicated that RT1 latencies were significantly lengthened for trials that immediately followed a startle trial, providing evidence for longer-term effects of the startling stimulus.

Keywords: psychological refractory period, dual-task performance, response preparation, startle reflex

1. Introduction

A common technique used over the past century to examine people's ability to perform multiple activities concurrently is the psychological refractory period paradigm (Telford, 1931), in which participants are required to identify and respond to two stimuli (S1 and S2) which are separated in time. Typically, as the time interval between the two stimuli (stimulus onset asynchrony; SOA) shortens, the reaction time (RT) to respond to the first stimulus (RT1) is unaffected, while the response latency to the second stimulus (RT2) is increased. The delay in RT2 is known as the *psychological refractory period* (PRP) and is thought to be indicative of the cost associated with processing two stimulus-response streams simultaneously (see Lien & Proctor, 2002; Pashler, 1994; 1998 for reviews).

Explanations offered for a delayed RT2 in PRP tasks can typically be divided into capacity sharing or "bottleneck" models (Pashler, 1994). Capacity theories assume that processing resources are shared among tasks and thus when multiple tasks are performed there is less resource available for each task, leading to impaired performance (Kahneman, 1973). Conversely, bottleneck theories posit that certain processing stages cannot be performed in parallel and thus processing multiple stimuli reaches a rate-limiting stage at some point whereby only one item can be processed at a time. Although the location of the bottleneck is still debated, considerable evidence exists suggesting that stimulus perception can occur in parallel and therefore is unlikely to contribute to the bottleneck (Pashler, 1994). While some research has provided support for a response selection bottleneck (e.g., Karlin & Kestenbaum, 1968; Smith, 1969), a PRP effect also occurs in a simple RT paradigm where response selection is minimal, indicating the

bottleneck may involve the response production stage (Bratzke, Rolke, & Ulrich, 2009; Maslovat, et al., 2013). It is also possible that a bottleneck occurs at multiple stages or that a central bottleneck affects both response selection and movement production (De Jong, 1993; Pashler, 1994).

In order to examine the PRP effect and which stage of processing is affected, the bottleneck theory offers a number of testable predictions. One such prediction is that any modification to task 1 that changes the central processing time required (up to or including the bottleneck stage), should have an equal effect on both RT1 and RT2 (Pashler, 1994). That is, at short SOAs, any RT change of task 1 should be *propagated* to task 2 (see Figure 3, middle panel), whereas propagation effects would not be predicted at long SOAs as there is no overlap in processing (Miller & Reynolds, 2003). Propagation effects have been confirmed by manipulating response selection variables such as number of response alternatives (Karlin & Kestenbaum, 1968; Smith, 1969), as well as response production variables such as sequence length (Bratzke, et al., 2008) or movement amplitude (Bratzke, et al., 2009; Ulrich, et al., 2006). In these experiments, increasing the time required to process task 1 resulted in similar magnitude increases for both RT1 and RT2 at short SOAs, consistent with the predictions of the bottleneck theory. Additionally, other research has reduced the response latency of RT1 through increased temporal predictability (Bausenhart, Rolke, Hackley, & Ulrich, 2006) or practice (Ruthruff, Johnston, Van Selst, Whitsell, & Remington, 2003), resulting in a similar decrease in RT2 at short SOAs.

The purpose of the current study was to examine response propagation effects in a PRP paradigm by reducing task 1 latency through the use of a startling acoustic stimulus

(SAS). When a SAS is presented in a simple RT task, RT is significantly shortened as the SAS acts as an involuntary trigger of the prepared response, bypassing response selection processes and shortening stimulus detection and response initiation stages (see Carlsen, Maslovat, & Franks, 2012; Valls-Solé, Kumru, & Kofler, 2008 for reviews). Specifically, it is thought that the SAS activates subcortical brain structures via connections between the cochlear nucleus and reticular formation, leading to both a reflexive startle response as well as involuntary activation leading to the initiation of a prepared response (provided a sufficient level of advance preparation of the movement; see Carlsen, et al., 2012 for more details). As the pathways and processes associated with the startle-mediated release of a response are faster than voluntary response initiation, responses to the SAS are significantly shortened as compared to non-startle trials (e.g., muscle activation onset <80 ms; Valls-Solé, Rothwell, Goulart, Cossu, & Munoz, 1999).

In the current study, participants performed two simple RT tasks in a PRP paradigm, in which they were required to respond to an auditory stimulus (S1) with a vocal response (R1), which was followed by a visual stimulus (S2) requiring a key-lift movement (R2). On selected trials, S1 was replaced with a SAS, with the expectation that this would shorten RT1 latency in the range of 40-60 ms, as has been previously shown for a vocal response (Stevenson, et al., 2014). Of primary interest was whether the RT "savings" associated with startle trials would propagate to RT2 for short SOAs, as predicted by the central bottleneck model. As both responses were known in advance, any propagation effects would be attributed to a shortened response execution stage of R1, leading to a similar reduction in the latency of R2. Although this logic is similar to previous work examining propagation effects, the use of a SAS provides unique benefits,

as the SAS is considered to act via a separate and involuntary response initiation pathway, thus bypassing any response initiation bottleneck (Bratzke, et al., 2009; De Jong, 1993). Indeed, a SAS has been successfully used in a dual-task paradigm to assess the attentional demands of a continuous task (Begeman, Kumru, Leenders, & Valls-Sole, 2007), as well as in a PRP paradigm as a probe to determine the preparation level of the second response (Maslovat, et al., 2013).

2. Methods

2.1 Participants

Data were collected from 17 right-handed volunteers with no sensory or motor dysfunctions. However, five participants were excluded due to a lack of activation in the sternocleidomastoid (SCM) muscle within 120 ms following a SAS (a reliable indicator of a startle response; see Carlsen, Maslovat, Lam, Chua, & Franks, 2011 for inclusion criteria) on all four startle trials in the single-task vocal RT block (see Section 2.2 Experimental Design). Thus, data are presented from twelve participants (7 male, 5 female; M = 24.8 yrs, SD = 6.1 yrs). All participants signed an informed consent form and were naïve to the hypothesis under investigation. This study was approved by the University of British Columbia ethics committee and was conducted in accordance with the ethical guidelines set forth by the Declaration of Helsinki.

2.2 Apparatus, Task, and Experimental Design

Participants sat in a height-adjustable chair in front of a table with a 22-inch computer monitor (Acer X233W, 1152 x 864 pixels, 75 Hz refresh) placed on it. Participants placed the right hand on a telegraph key (E.F. Johnson Speed-X, Model 114-300) located on the table that required 2 N of force to close (i.e., simply resting the hand

on the switch was sufficient to close it). A microphone (Sennheiser, MKH 416-P48) was placed in front of the participant, below the monitor to capture vocal responses.

To determine baseline performance, participants began by performing 20 trials of each of the two required responses in a single-task situation. All trials began with the word "Ready!" presented on the computer screen, followed by a variable foreperiod of 2500-3500 ms. For the first block of trials, participants were instructed to respond to an auditory stimulus by vocalizing the word "TAT" as quickly as possible. The auditory stimulus consisted of a non-startling tone on 16 trials (82 +/-2 dB, 40 ms, 1000 Hz) and a startling tone on 4 trials (124 +/-2 dB, 40 ms, 1000 Hz, <1 ms rise time). Startle trials were interspersed pseudorandomly such that the first trial was never a startle trial and there were never two consecutive startle trials. Acoustic signals were generated by a customized computer program and were amplified and presented via a loudspeaker placed behind the head of the participant. Acoustic stimulus intensity was measured at a distance of 30 cm from the loudspeaker (approximately the distance to the ears of the participant) using a sound level meter (Cirrus Research model CR:252B; "A"-weighted decibel scale, impulse response mode). In the second block of trials, participants were instructed to respond to the presentation of a green circle (10 cm diameter) in the middle of the computer screen by lifting their right hand off the telegraph key as quickly as possible. During the single-task testing blocks, RT was presented on the screen for five seconds following each trial with a monetary reward of CDN $0.05 per trial for RTs below 250 ms.

Following the single-task trials, participants were informed that they would be performing both the vocal response and key-lift in a dual-task situation, and that they

should give equal priority to performing each task as quickly as possible. The auditory stimulus (S1) was always presented first and required a vocal response of "TAT" (R1), followed by the visual stimulus (S2) requiring a right hand key-lift response (R2). A practice block of 20 trials was conducted, with SOAs of 100 ms (10 trials), 200 ms (4 trials), 500 ms (2 trials), 1000 ms (2 trials), and 1500 ms (2 trials) randomly presented. A high proportion of short SOA trials were used, as propagation effects are only expected for these conditions. Following the practice block, participants performed 5 blocks of 25 test trials whereby 20 trials involved the same distribution of SOAs as the practice trials, but one additional trial was presented at each SOA where the 124 dB SAS was presented in place of the normal 82 dB auditory stimulus (S1) (i.e., 5 startle trials per test block, 25 startle trials total). Startle trials were interspersed pseudorandomly within each block in a similar manner to the single-task testing condition. During the dual-task testing blocks, RT for each task was presented simultaneously on the screen for seven seconds following each trial with a monetary bonus of CDN $0.05 per task (i.e., up to $0.10 per trial) for fast RTs (<250 ms for RT1, <300 ms for RT2). Participants were instructed to try and maximize their reward bonus by minimizing total RT and thus receiving the reward bonus for both responses. Participants were allowed a rest period of approximately one minute in between blocks and the testing session lasted approximately one hour.

2.3 Recording Equipment

Surface EMG data were collected from the muscle bellies of the right extensor carpi radialis longus (ECR - agonist), and right and left sternocleidomastoid (SCM – used as a startle indicator only) using preamplified surface electrodes connected via shielded cabling to an external amplifier system (Delsys Model DS-80). Recording sites were

prepared and cleansed in order to decrease electrical impedance. The electrodes were oriented parallel to the muscle fibers, and then attached using double sided adhesive strips. A grounding electrode was placed on the left ulnar styloid process. EMG onsets were defined as the first point where the rectified and filtered (25 Hz low pass elliptical filter) EMG activity first reached a sustained value of two standard deviations above baseline levels (mean EMG activity 100 ms prior to S1), with EMG offsets determined in a similar manner. EMG onset and offset points were determined using a custom LabVIEW® (National Instruments Inc.) program and then visually confirmed and manually adjusted (if necessary) to compensate for any errors due to the strictness of the algorithm.

Displacement RT of key lift-off was monitored using the contact switch of the telegraph key, while vocal responses were collected using the microphone placed in front of the participant. Voice onset and offset was determined in an identical manner to EMG, whereas displacement onset for the key-lift task was determined by the time at which switch contact was broken. A customized LabView® computer program controlled stimulus and feedback presentation, and initiated data collection (National Instruments, PC-MIO-16E-1) at a rate of 1 kHz for 3 s, starting 500 ms prior to the presentation of the S1 “go” signal.

2.4 Data Reduction

The first block of dual-task trials was not analyzed as this block was considered practice and did not include a SAS. Before analyzing the results of the experimental blocks (1980 total trials across participants), we discarded 46 trials (2.3 %) in which an error occurred (most often due to a telegraph key not being fully depressed at the start of

the trial), 14 trials (0.8 %) in which a response occurred prior to the stimulus (i.e., anticipation), 17 trials (1.1%) in which a slow (>500 ms) vocal response (R1) occurred , and 16 trials in which the participant did not show any SCM activation within the first 120 ms for a startle trial (i.e., lack of startle indicator). Of the remaining 1887 trials, we discarded an additional 93 trials (4.9 %) in which the two responses occurred less than 100 ms apart, as these trials may represent a "grouped" response which may introduce unwanted effects (see Miller & Ulrich, 2008; Ulrich & Miller, 2008 for more details). Overall, our analysis included 1794 of the 1980 total trials (90.6 %).

2.5 Dependent Measures & Analyses

Primary dependent measures included voice onset (RT1) and key-lift displacement onset (RT2). To confirm that processing time for R1 (vocal response) was not different between the single-task condition and all SOA conditions in the dual-task paradigm, we analyzed RT1 via a 2 Stimulus (non-startle, startle) x 6 Condition (single-task, 100 SOA, 200 SOA, 500 SOA, 1000 SOA, 1500 SOA) repeated measures analysis of variance (ANOVA). To confirm a typical PRP effect for the key-lift task (R2), we examined RT2 for non-startle trials using a one-way, 6 factor (Condition: single-task, 100 SOA, 200 SOA, 500 SOA, 1000 SOA, 1500 SOA), repeated measures ANOVA. To determine the effects of the SOA and startling stimulus on performance of the key-lift task (R2), RT2 was analyzed using a 2 Stimulus (non-startle, startle) x 5 SOA (100 SOA, 200 SOA, 500 SOA, 1000 SOA, 1500 SOA) repeated-measures ANOVA.

We were also interested in whether the performance characteristics of the vocal and key-press response were affected by either the intensity of S1 or SOA condition. Thus, we measured the vocal response duration as well as ECR (agonist) duration and

peak amplitude (defined as maximal rectified EMG amplitude between onset and offset) for the key-lift task. Voice duration was analyzed via a 2 Stimulus (non-startle, startle) x 6 Condition (single-task, 100 SOA, 200 SOA, 500 SOA, 1000 SOA, 1500 SOA) repeated measures ANOVA, whereas ECR duration and peak amplitude were analyzed using a 2 Stimulus (non-startle, startle) x 5 SOA (100 SOA, 200 SOA, 500 SOA, 1000 SOA, 1500 SOA) repeated-measures ANOVA.

Greenhouse-Geisser corrected degrees of freedom were used to adjust for violations of sphericity if necessary. Uncorrected degrees of freedom are reported, with the corrected *p* values. Partial eta squared (η_p^2) values are reported as a measure of effect size. The alpha level for the entire experiment was set at .05, and where appropriate, significant results were examined via Tukey's honestly significant difference (HSD) test to determine the locus of the differences.

3. Results

3.1 Response Latencies

As expected, analysis of vocal responses showed that RT1 latencies were significantly shorter on startle trials (M = 172 ms, 95% CI [153.5, 190.1]) compared to non-startle trials (M = 216 ms, 95% CI [193.3, 238.2]), as confirmed by a main effect of stimulus, $F(1, 11) = 136.56$, $p < .001$, $\eta_p^2 = .93$ (Figure 1A). Analysis of RT1 also yielded a significant main effect of condition, $F(5, 55) = 7.75$, $p = .004$, $\eta_p^2 = .41$ which post-hoc testing confirmed was due to a significantly longer RT1 when performed as a single-task compared to all conditions of the dual-task paradigm, which were not significantly different to each other. This effect has been shown previously and has been attributed to practice effects when the single-task paradigm is performed prior to the dual-task trials

(Maslovat, et al., 2013). To further confirm this main effect of condition was the result of practice effects, we performed an additional post-hoc analysis of RT1 (collapsed across condition) using a 2 Stimulus (non-startle, startle) x 6 Block (Single-Task, Block 1, Block 2, Block 3, Block 4, Block 5) repeated-measures ANOVA. This analysis produced both a main effect of stimulus, $F(1, 11) = 121.92$, $p < .001$, $\eta_p^2 = .92$ and a main effect of block, $F(5, 55) = 12.29$, $p < .001$, $\eta_p^2 = .53$, in which RT1 significantly decreased as the experiment progressed in a linear manner, $F(1, 11) = 19.37$, $p = .001$, $\eta_p^2 = .64$ (Figure 1B). Although a practice effect was present for RT1, the lack of difference in vocal response latency between SOAs during the dual-task task indicates that the first response was processed in a similar manner throughout the dual-task portion of the experiment.

(INSERT FIGURE 1 ABOUT HERE)

Analysis of the key-lift task (RT2) on non-startle trials showed a main effect of condition, $F(5, 55) = 120.31$, $p < .001$, $\eta_p^2 = .92$. This represents a typical PRP effect in which RT2 latency significantly decreased with increasing SOA, reaching single-task key-lift latencies at long SOAs (Figure 2). Post-hoc tests indicated that RT2 was significantly longer at SOAs of 100 ms (M = 343 ms, 95% CI [316.5, 370.2]), 200 ms (M = 283 ms, 95% CI [260.7, 306.0]), and 500 ms (M = 244 ms, 95% CI [225.1, 263.0]), as compared to the single task RT2 (M = 196 ms, 95% CI [182.4, 209.9]; shown as a solid black line in Figure 2).

(INSERT FIGURE 2 ABOUT HERE)

Our primary research question was whether the RT1 “savings” during startle trials would be inherited by RT2, as would be predicted by the central bottleneck theory. However, in contrast to our predictions, startle trials resulted in *longer* RT2 values at

short SOAs (Figure 2). Analysis of RT2 confirmed both a main effect of stimulus, $F(1, 11) = 14.54$, $p = .003$, $\eta_p^2 = .57$, and SOA, $F(4, 44) = 80.03$, $p < .001$, $\eta_p^2 = .88$, which were superseded by a significant Stimulus x SOA interaction, $F(4, 44) = 3.98$, $p = .024$, $\eta_p^2 = .27$. Post hoc analysis of this interaction revealed that startle resulted in significantly longer RT2 values compared to non-startle trials at short SOAs of 100 ms (startle *M* = 397 ms, 95% CI [346.0, 447.0], non-startle *M* = 343ms, 95% CI [316.5, 370.2]) and 200 ms (startle *M* = 319 ms, 95% CI [276.3, 360.8], non-startle *M* = 283ms, 95% CI [260.7, 306.0]).

Note that as opposed to the shortened RT1 latencies in startle trials being propagated to RT2, RT2 latencies were in fact *delayed* on startle trials at short SOAs (see Figure 3 for a schematic). Thus, to determine the effects of the SAS on RT2, it is necessary to add the RT1 savings to the RT2 delay (Figure 4). These additive effects at short SOAs can be considered a "*startle refractory period*" in which using a SAS to trigger task 1 at an earlier latency results in a delay in initiating the second response. The startle refractory period appears to be short in duration as no significant RT2 delay was observed at longer SOAs (500 ms or greater). Although there are still RT1 savings associated with long SOAs, these savings would not be predicted to be propagated to RT2 due to the first response having passed through the central bottleneck.

(INSERT FIGURE 3 & 4 ABOUT HERE)

Contrary to our prediction, reducing the latency of the first response via presentation of a SAS resulted in a delayed second response, which we attributed to a startle refractory period. Although these effects had vanished by the 500 ms SOA, we were interested in whether eliciting a startle reflex had a more lasting effect, which would

be demonstrated by a change in performance on the subsequent trial. To examine this possibility we performed a post-hoc analysis of RT1 latency, irrespective of SOA condition, using a paired sample *t*-test comparing the non-startle trial prior to and following each startle trial in both the single-task and dual-task conditions. This ensured we compared trials at a similar time in the experiment, although trials were omitted if a startle trial was the last trial of a block (as there was no comparable post-startle trial), or if the non-startle trial prior to a startle trial happened to also follow a startle trial (as startle trials could be two trials apart). This analysis showed that post-startle trials were performed with significantly longer latencies, as compared to pre-startle trials in both the single-task condition, t(11) = -2.22, $p = 0.048$ (pre-startle $M = 228$ ms, post-startle $M = 259$ ms), and dual-task condition, t(11) = -2.64, $p = 0.023$ (pre-startle $M = 209$ ms, post-startle $M = 222$ ms).

3.2 Response Characteristics

Analysis of the voice duration (R1) showed that startle trials resulted in a significantly longer vocal response ($M = 171$ ms, 95% CI [142.5, 198.6]) compared to non-startle trials ($M = 156$ ms, 95% CI [133.6, 177.9]), as confirmed by a main effect of stimulus, $F(1, 11) = 7.73$, $p = .018$, $\eta_p^2 = .41$. No effects were found for condition, $F(5, 55) = 3.50$, $p = .061$, $\eta_p^2 = .24$, or Stimulus x Condition interaction, $F(5, 55) = 0.60$, $p = .561$, $\eta_p^2 = .05$. Although the main effect of condition approached significance ($p = .061$), examination of mean values indicated that this trend was primarily due to a longer duration on single task trials ($M = 177$ ms) as compared to all other SOA conditions (100 ms SOA, $M = 159$ ms; 200 ms SOA, $M = 158$ ms; 500 ms SOA, $M = 163$ ms; 1000 ms SOA, $M = 162$ ms; 1500 ms SOA, $M = 160$ ms). Consistent with the results of the RT1

analysis, the lack of difference in voice duration confirms that the first response was produced in a similar manner during the dual-task testing conditions.

Analysis of the duration of the agonist EMG (R2) showed no effects of stimulus, $F(1, 11) = 0.69$, $p = .424$, $\eta_p^2 = .06$, SOA, $F(4, 44) = 2.86$, $p = .098$, $\eta_p^2 = .21$, or Stimulus x SOA interaction, $F(4, 44) = 1.01$, $p = .345$, $\eta_p^2 = .09$. However, while analysis of peak agonist EMG produced no main effects of stimulus, $F(1, 11) = 0.19$, $p = .674$, $\eta_p^2 = .02$, or SOA, $F(4, 44) = 2.43$, $p = .125$, $\eta_p^2 = .18$, there was a significant Stimulus x SOA interaction, $F(4, 44) = 6.17$, $p = .002$, $\eta_p^2 = .36$. Post hoc analysis of this interaction confirmed the only statistically different value was a significantly lowered peak agonist EMG on startle trials for the 100 ms SOA ($M = 0.851$ mV, 95% CI [0.466, 1.236]) compared to non-startle trials ($M = 1.013$ mV, 95% CI [0.628, 1.398]).

3.3 Other Considerations

One possible confound in this experiment is that the reflexive response to a SAS typically includes a blink reflex, resulting from activation in the orbicularis oculi (OOc) muscle at a latency of 35-40 ms following the SAS, with a duration of 30-150 ms (Blumenthal, et al., 2005; Brown, et al., 1991). This reflexive response to the SAS may have resulted in participants' eyes being closed when the visual stimulus (S2) was presented at short SOAs. To examine this possibility, we recorded EMG activity from the left OOc for one participant and recorded their responses using a video camera (Casio EX-F1 Exilim Digital Camera, recorded at 30 fps, image size of 512 x 384 Pixels). This participant showed robust OOc activation during all startle trials with an average onset latency of 50 ms and offset latency of 77ms; however, video recording showed the participant's eyes closed from 66-165 ms (± 33ms due to camera speed limitations)

following the SAS. Thus, for the 100 ms SOA condition, it is likely that the participant's eyes were closed when the visual stimulus was presented, which may partially explain the RT2 delay. However, the auditory blink reflex was completed prior to the visual stimulus in the 200 ms SOA condition and thus the RT2 delay at longer SOAs was not contaminated by the reflexive activation in the OOc.

4. Discussion

The purpose of the current study was to examine RT propagation effects through the use of a SAS in a PRP paradigm. On non-startle trials, participants performed the vocal response at a similar latency (Figure 1A) and with a consistent duration for all SOAs, confirming the first response was processed in a similar manner throughout the dual-task portion of the experiment. Additionally, non-startle trials showed a typical PRP effect in which shorter SOAs resulted in longer RT2 latencies, while longer SOAs resulted in latencies similar to the single-task condition (Figure 2). By replacing S1 with a startling stimulus, we were able to trigger the prepared vocal response and reduce RT1 by an average of approximately 45 ms (Figure 1A). Of primary interest was whether the reduction in RT1 on startle trials would propagate to RT2, as predicted by the central bottleneck model. In contrast to our prediction, startle trials produced significantly longer RT2 values for the 100 ms and 200 ms SOA (Figure 2). Thus, rather than propagation effects, it appears that a SAS produces a "startle refractory period" that results in a delay in the preparation and/or execution of upcoming responses (Figure 3). Further evidence for a transient startle refractory period is provided by significantly reduced peak agonist EMG activation on startle trials for the second response at the 100 ms SOA. Thus, at

short SOAs, the startling stimulus not only delayed the key-lift response but also reduced the amount of peak muscle activation produced by the participant.

The length of the startle refractory period can be estimated at short SOAs by considering both the RT1 savings from the early triggering of the first response and the observed RT2 delay (Figure 4). While the confound of the auditory blink reflex does not allow us to accurately measure the latency of RT2 at the 100 ms SOA, data from the 200 ms SOA condition can provide an approximation of the startle refractory period. Even with the RT1 savings of 40 ms, RT2 was delayed by an additional 35 ms, meaning that the second response occurred 75 ms later than would be expected without interference and with propagation effects. Note that this startle refractory period appears to be independent to the psychological refractory period as no differences were found between startle and non-startle trials at the 500 ms SOA, yet there was still a delay in RT2, relative to single task control values (i.e. PRP effect).

One explanation for the short-term performance decrements may relate to motor cortex suppression as a number of studies have shown that a startle-evoked activation of reticulo-cortical projections can transiently (~50 ms) inhibit the motor cortex (Furubayashi, et al., 2000; Kuhn, Sharott, Trottenberg, Kupsch, & Brown, 2004). Similarly, it has been shown that the use of a SAS during a choice RT task can cause cognitive interference and give rise to more movement production errors (Carlsen, Chua, Inglis, Sanderson, & Franks, 2004). For the current study, neural activation models (Hanes & Schall, 1996; see also Carlsen et al., 2012; Maslovat, Hodges, Chua, & Franks, 2011) predict that the amount of time required to prepare and initiate a movement is dependent upon the activation level of the cortex. If the SAS causes temporary inhibition

of the motor cortex, it would be predicted that response latency of task 2 in a PRP paradigm would also be transiently delayed at short SOAs, consistent with the reported results.

In addition to the short-term effect of the SAS on RT2, there also appeared to be a longer-term effect on reduced motor preparation as RT1 latencies were significantly lengthened for trials that immediately followed a startle trial. This effect was present in both single-task and dual-task conditions, suggesting that this result was not related to the preparation of multiple responses but rather an effect of the startling stimulus on subsequent performance. These results are in line with early studies involving the effects of a startling stimulus on task performance, as researchers were concerned about possible adverse effects of sonic booms on pilots. Although RTs were often facilitated by the SAS, transient performance decrements were found for pursuit tracking (Thackray & Touchstone, 1970; Thackray, Touchstone, & Jones, 1972) and cognitive tasks such as mental arithmetic (Vlasak, 1969), which lasted as long as 20-30 seconds. Whereas the aforementioned startle refractory period may involve short-term inhibition of the motor cortex, the longer-term performance decrements may relate to the excitation in the sympathetic nervous system caused by the acoustic startle reflex (Eder, Elam, & Wallin, 2009), which likely requires a longer time frame to return to pre-startle levels.

Although we believe the results of the current study provide strong evidence that the presentation of a startling stimulus interferes with motor preparation at both a short (~75 ms) and long (10-15 s) time frame, we did not directly measure motor cortex or sympathetic nervous system activation. Thus, it is worthwhile to consider other possibilities for the reported results. One such possibility is that detection of S2 was

affected by a phenomenon known as “attentional blink” (Raymond, Shapiro, & Arnell, 1992), in which the second of two target visual stimuli is less likely to be detected when it appears in close temporal proximity to the first (see Dux & Marois, 2009 for a review). More recent work has shown a similar effect with a cross-modal paradigm in which the first stimulus is auditory followed by a visual second stimulus (similar to the current methods), and attributed the attentional blink to a similar cortical bottleneck as implicated in the PRP phenomenon (Marti, Sigman, & Dehaene, 2012).

While we cannot definitively rule out any effects of attentional blink in the current study, a number of findings suggest that this is not a sufficient explanation for our reported results. First, attentional blink paradigms usually present rapid multiple visual stimuli which are flashed briefly on the screen, with the second target stimulus occurring at some point in the sequence following the initial target stimulus. Conversely, the current study employed a single visual stimulus that remained on the screen from initial presentation until the end of the trial, requiring much less stimulus recognition processing which may be responsible for the cortical bottleneck. Second, one peculiarity of the attentional blink effect is that exhibits what is known as “lag-1 sparing,” meaning that if the second target stimulus is presented immediately following the first target stimulus (rather than later in the sequence), detection is not negatively affected (Hommel & Akyurek, 2005). In the current study, the stimulus following S1 was always the visual “go” signal, which would thus be unlikely to be affected by the attentional blink. Third, any effects of attentional blink would be present on all trials, yet our results show clear effects of the SAS presentation on RT2 latency and peak EMG at the short SOA condition, as well as delayed RT in the trial following a startle. Thus we believe the

reported results are more likely to be attributed to effects of the startling stimulus, rather than other confounding factors such as the attentional blink.

In summary, by implementing a startling acoustic stimulus in a psychological refractory period paradigm, we have provided novel evidence that a SAS interferes with motor preparation of subsequent actions. This interference results in reduced preparation in the short-term (~75 ms following the SAS), which we attribute to cortical suppression and in the long-term (10-15 s following the SAS), which we attribute to recovery from excitation of the sympathetic nervous system.

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Figure Captions

Figure 1. Mean verbal reaction time (RT1, with error bars representing 95% confidence intervals) for various SOA intervals (top panel, A) and blocks (bottom panel, B), separated by stimulus type (startle and non-startle trials). In the top panel, a single asterisk (*) represent a main effect of stimulus, while a double asterisk (**) represent longer RT1 in the single-task condition. In the bottom panel, the double asterisk (**) represents a main effect of block, with decreasing RT1 with practice.

Figure 2. Mean key-lift reaction time (RT2, with error bars representing 95% confidence intervals) for various SOA intervals, separated by stimulus type (startle and non-startle), as compared to single-task performance (solid black line). Non-startle trials showed a typical PRP effect in which shorter SOAs (100 ms, 200 ms and 500 ms) resulted in significantly longer (**) RT2 latencies. In contrast to the predicted propagation effect, significantly longer (*) RT2 latencies were found for startle trials at the 100 ms and 200 ms SOA conditions.

Figure 3. Schematic of predicted versus actual results. In the baseline (top) condition, stimuli (S) are separated by a stimulus onset asynchrony (SOA). The shaded portion represents the bottleneck portion of the task, which cannot start for task 2 until completed for task 1. This results in a psychological refractory period (PRP) in which the second response (R) has a delayed reaction time (RT). The current experiment replaced S1 with a startling acoustic stimulus (SAS), resulting in a reduced RT1. The prediction of propagation effects (middle panel) is that the reduction in RT1 is inherited by RT2. However, actual results (bottom panel) showed an increase in RT2, which we attribute to a startle refractory period (SRP).

Figure 4. Mean Reaction time (RT) differences between startle and non-startle trials for various SOA intervals (significant differences are illustrated with an asterisk). Black bars represent RT1 “savings” due to shorter latency verbal RT on startle trials while grey bars represent RT2 delay due to longer latency key-lift RT on startle trials. These effects are shown as cumulative as RT1 savings on startle trials were predicted to be propagated to RT2 but instead RT2 values were longer for startle trials.

588 Figure 1
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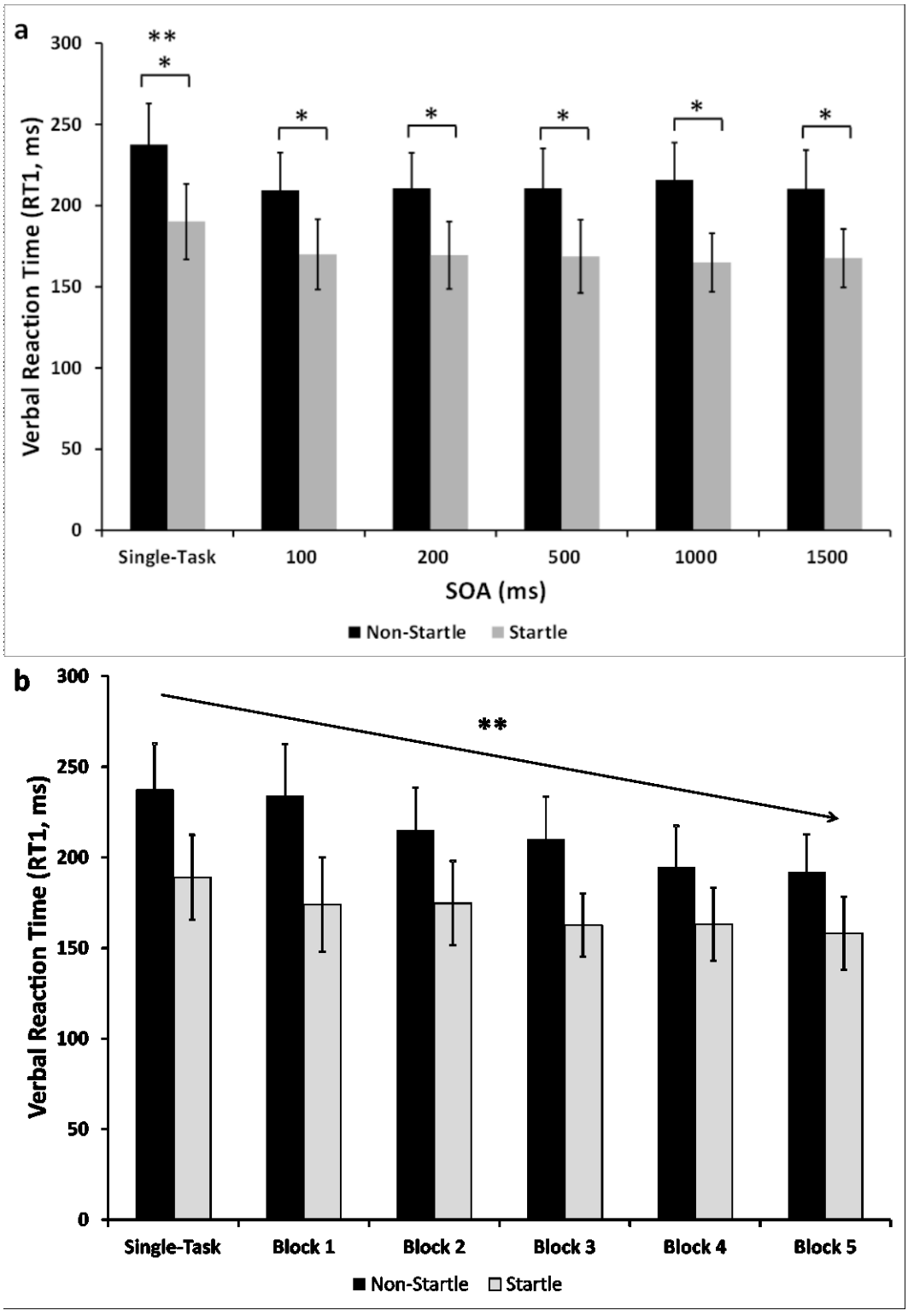

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592 Figure 2
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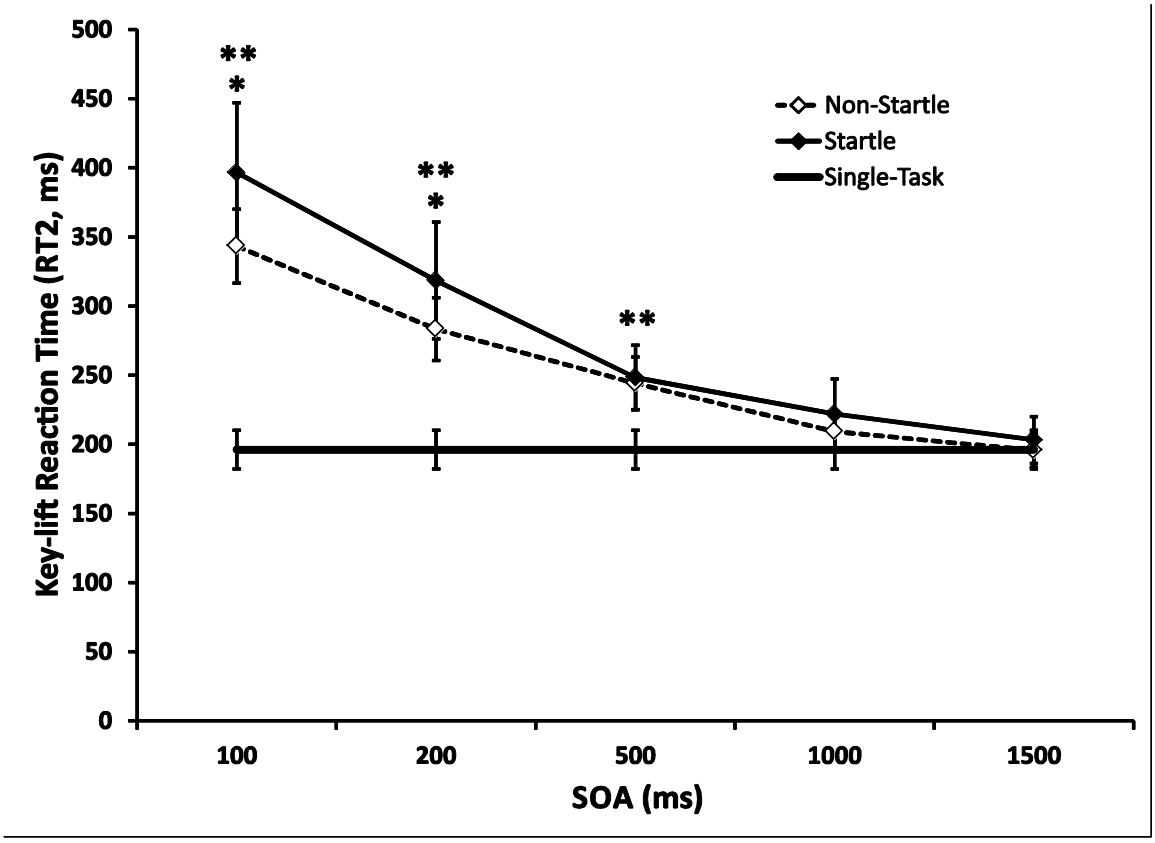

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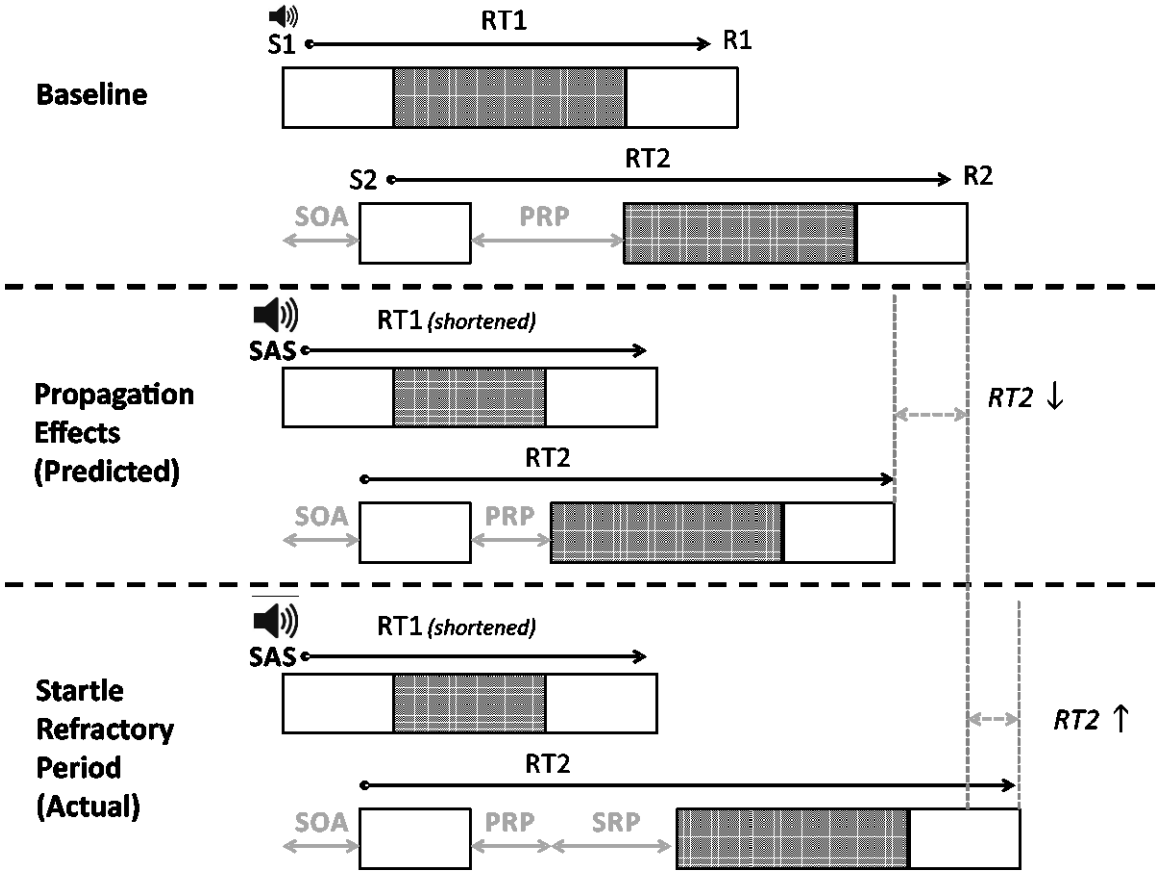
Figure 3


Baseline
S1
RT1
R1
S2
RT2
R2
SOA
PRP
Propagation Effects (Predicted)
SAS
RT1 (shortened)
RT2 ↓
RT2
SOA
PRP
Startle Refractory Period (Actual)
SAS
RT1 (shortened)
RT2 ↑
RT2
SOA
PRP
SRP

602 Figure 4
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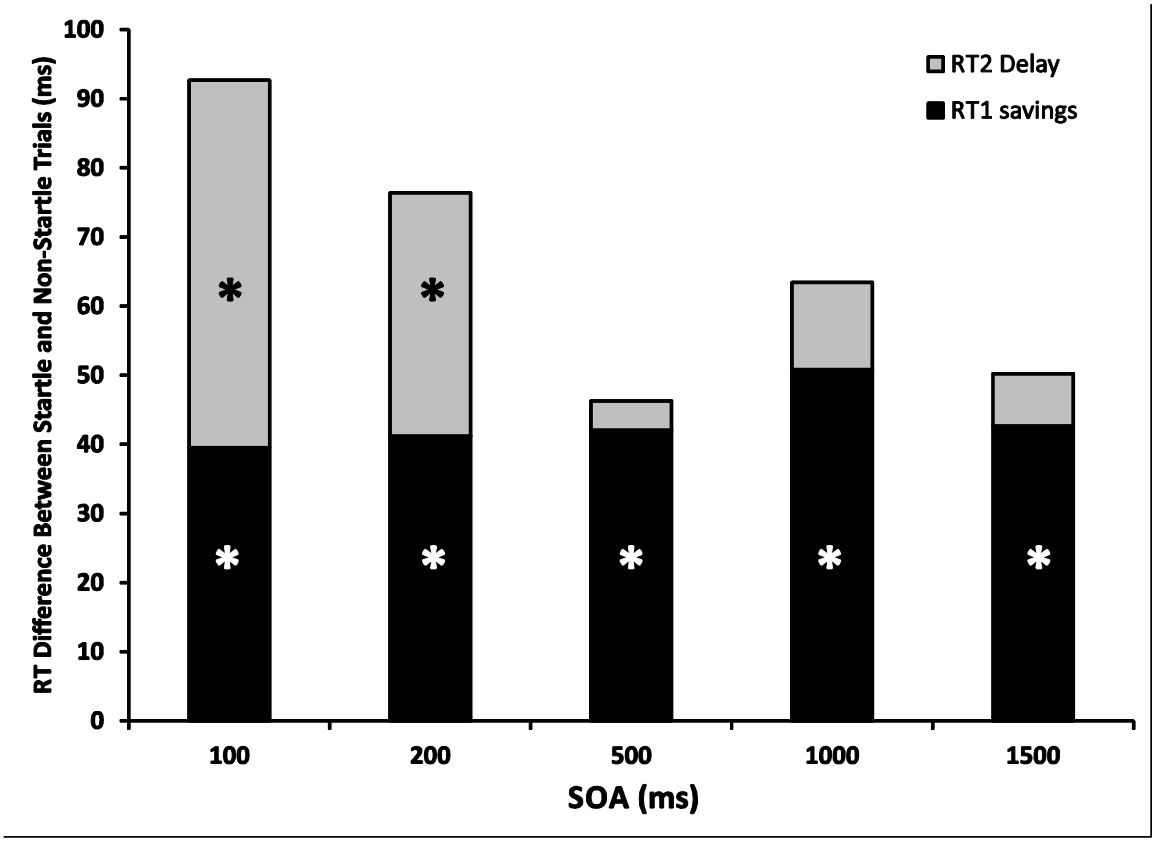

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