Research Report

SOURCES OF DUAL-TASK INTERFERENCE:
Evidence From Human Electrophysiology

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Abstract—*When an individual attempts to perform two tasks at the same time, the tasks often interfere with each other. This interference has been studied for several decades with the psychological refractory period paradigm, in which two targets that require independent responses are presented on each trial, separated by a variable delay period; interference typically takes the form of increased response times for the second target at short interstimulus delays. The present study used electrophysiological recordings to determine whether a specific index of perception and categorization (the P3 wave) is delayed in the same manner as response times. Although response times for the second target were found to be greatly delayed at short interstimulus intervals, the P3 wave was not substantially delayed. This finding indicates that there was minimal interference during target identification and categorization and that the prolongation of response times in this paradigm primarily reflects a delay in a relatively late process, such as response selection.*

The psychological refractory period (PRP) paradigm is extremely useful for studying dual-task interference because it is a very simple paradigm and yet yields extremely robust interference effects (see review by Pashler, 1994). Most notably, when the stimulus onset asynchrony (SOA) between the first target (T1) and the second target (T2) is short, the response time for the second target (RT2) increases sharply. Under appropriate conditions, equivalent effects are observed independent of the modality of the stimulus or the form of the response, which suggests that the delays in RT2 are caused by interference between "central" rather than "peripheral" processes (Pashler, 1989; Pashler & O'Brien, 1993).

Following from the early theory of Welford (1952), Pashler and his colleagues have proposed a model in which interference occurs at two separate stages of processing in the PRP paradigm (Pashler, 1989, 1994; Pashler & Johnston, 1989). The main source of the delays in RT2 at short T1-T2 SOAs is proposed to be a "bottleneck" at the stage of response selection, which causes the selection of the T2 response to be postponed until after the T1 response has been selected. In addition, limitations in perceptual processing capacity may cause some slowing in the identification of T2 when it is being identified concurrently with T1, although this perceptual interference effect is relatively small under typical experimental conditions. Thus, this model proposes strong interference at the stage of response selection and somewhat weaker interference at the stage of target identification.

Although this model of dual-task interference has been supported by a number of studies, several alternative models have been proposed (e.g., De Jong, 1993; Logan & Burkell, 1986; Meyer & Kieras, 1997). This lack of agreement is caused, in part, by the fact that behavioral response times (RTs) reflect contributions from virtually every stage of processing, and it is therefore difficult to attribute a change in RT to a change in a specific stage of processing without the use of potentially questionable assumptions. To avoid this limitation of behavioral response measurements, the present study used event-related potential (ERP) recordings to examine the causes of dual-task interference. ERPs are particularly well suited for addressing this issue because they provide a continuous measure of processing between a stimulus and a response, allowing the effects of a given experimental manipulation to be isolated to a specific stage of processing.

A previous ERP study of the PRP paradigm focused on the lateralized readiness potential (LRP), an index of the response selection process (Osman & Moore, 1993). Both RT2 and the LRP were found to be delayed at short T1-T2 SOAs, which indicates that the slowing of RT2 is caused by a delay at or before the stage of response selection. There are many cognitive processes that precede response selection, however, and the observed delay in LRP onset could have been caused by a delay in any of these processes. The purpose of the present study was to determine the locus of interference more precisely by examining the P3 wave, which can be used to distinguish between response selection and earlier stages, such as target categorization.

Under typical conditions, the amplitude of the P3 wave is greater for improbable targets than for probable targets (Johnson, 1986). Consider, for example, an observer who views a sequence of names and must indicate whether each stimulus is a male name or a female name. If any particular name is presented only once, but 10% of the total are male names and 90% are female names, then a larger P3 wave will be elicited by the male names than by the female names. Thus, the P3 wave is sensitive to the probability of a task-defined category. Logically, therefore, the P3 cannot be elicited until the observer has determined the task-defined category of the stimulus (e.g., whether it falls into the male name category or female name category). Several studies have supported and extended this logic by showing that the latency of the P3 wave is increased when the difficulty of identifying or categorizing the targets is increased (e.g., Kutas, McCarthy, & Donchin, 1977; Magliero, Bashore, Coles, & Donchin, 1984). In addition, the amplitude of the P3 wave is decreased when perceptual processing resources are diverted away from the P3-eliciting stimuli in dual-task experiments (Isreal, Wickens, Chesney, & Donchin, 1980). In contrast, manipulations of the difficulty of response selection have little or no effect on P3 amplitude or latency (Kramer, Wickens, & Donchin, 1983; Magliero et al., 1984). Thus, P3 latency can be used as a relatively pure measure of the amount of time required to perceive and categorize a stimulus, and P3 amplitude can be used as a relatively pure measure of the availability of cognitive processing resources for accomplishing target perception and categorization. Because the model of Pashler (1994) states that RT2 slowing in the PRP paradigm is primarily caused by a postponement in response selection, this model would predict that—unlike RT—P3 latency should not be increased at short T1-T2 SOAs. In addition, this model would predict a modest reduction in P3 amplitude at short SOAs due to a reduction in the processing resources that are available for identifying and categorizing T2.

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EXPERIMENT 1

In this experiment, T1 was a red or green box and T2 was the letter *X* or the letter *O*; the participants were required to make two button-press responses on each trial, one to indicate the color of T1 and one to indicate the form of T2. As shown in Figure 1, the procedure differed
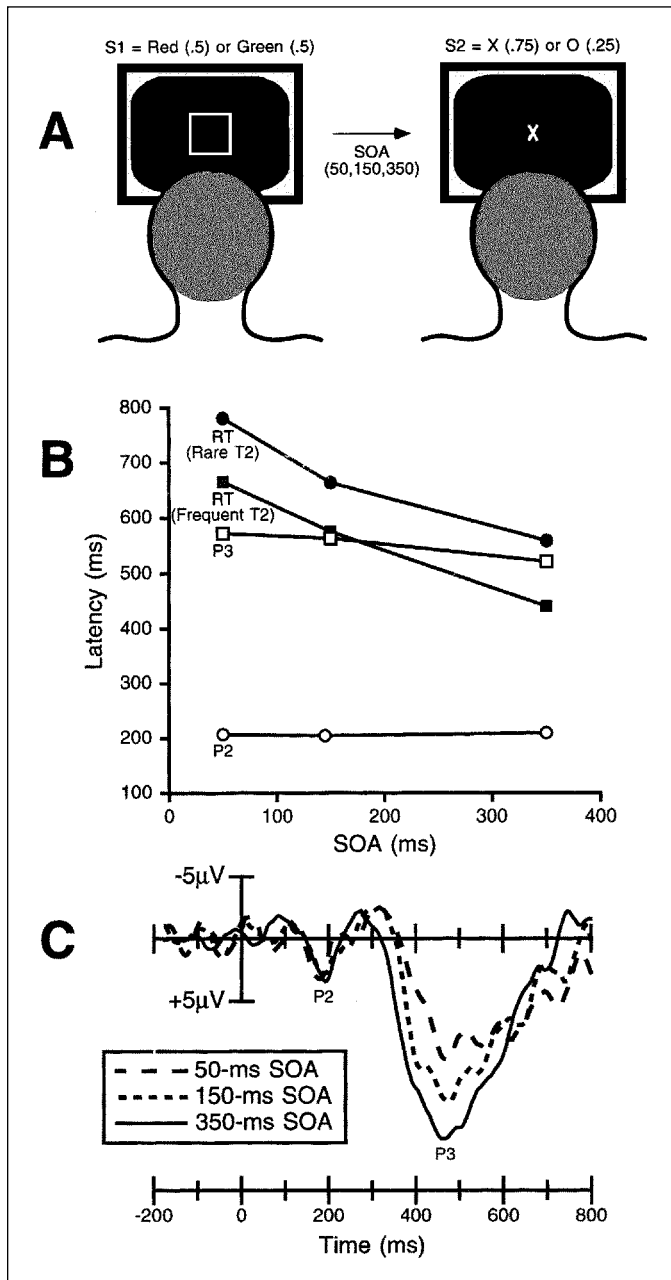

Fig. 1. Stimuli and results for Experiment 1. Shown in (a) are examples of the two stimuli (S1 and S2) and their probabilities. Response times (RTs) and event-related potential latencies, averaged across participants, are shown in (b). Shown in (c) are difference waveforms (infrequent minus frequent) of event-related potentials at the midline parietal electrode site, averaged across participants. SOA = stimulus onset asynchrony.

slightly from the typical PRP procedure in that one of the two T2 alternatives was frequent (*p* = .75) and the other was infrequent (*p* = .25). This probability manipulation played two important roles. First, an infrequent T2 was necessary to elicit a robust P3 component. Second, the probability difference made it possible to solve a problem that occurs in ERP recordings with short SOAs. Specifically, when two stimuli are presented in close succession, the ERP elicited by the first stimulus will overlap the ERP elicited by the second stimulus, making it difficult to isolate the electrophysiological response elicited by each stimulus. This problem was overcome by computing difference waves in which the ERP waveform elicited by the frequent T2 alternative was subtracted from the ERP waveform elicited by the infrequent T2 alternative. This procedure subtracts away any ERP activity that is the same for these two trial types, including the overlapping T1 activity. The resulting difference wave reflects only those ERP components that are sensitive to T2 probability (primarily the P2, N2, and P3 waves). This procedure has been used in several previous ERP studies and is analogous to the use of subtraction images in functional neuroimaging studies.

Method

The stimuli and procedure are illustrated in Figure 1a. On each trial, a red or green outline box (T1, 3.9° × 3.9°) was presented on a black background for 50 ms, followed at an SOA of 50, 150, or 350 ms by a 50-ms presentation of a white *X* or a white *O* (T2, 0.4° × 0.4°). For half of the participants, *X* was frequent (*p* = .75) and *O* was infrequent (*p* = .25); the probabilities were reversed for the remaining participants. The red and green T1 alternatives were equiprobable.

ERPs were recorded using standard electrode locations (F3, Fz, F4, C3, Cz, C4, P3, Pz, P4) and standard recording procedures (see Luck & Hillyard, 1990). The participants were 12 neurologically normal undergraduates who gave informed consent. All ERP measurements were obtained from the infrequent-minus-frequent difference waves. So that the ERP and RT data could be compared directly, behavioral response latency was quantified as the time point that bisected the RT frequency distribution (i.e., the median RT), and P3 latency was quantified as the time point that bisected the area under the P3 wave.[1] A time window of 300 ms to 800 ms was used for this P3 latency measure and also for measuring mean P3 amplitude. The P2 wave was quantified in an analogous manner with a time window of 150 ms to 250 ms. Each of these measures was subjected to a two-way analysis of variance (ANOVA) with factors of SOA and electrode site, accompanied by the Greenhouse-Geisser epsilon correction for nonsphericity.

Results and Discussion

As shown in Figure 1b, RT2 was strongly affected by the T1-T2 SOA, with a decrease of approximately 220 ms between the 50-ms SOA and the 350-ms SOA. This is the typical PRP effect. In addition, RT2 was approximately 100 ms longer for the infrequent T2 than for the frequent T2.

As shown in Figure 1c, the infrequent-minus-frequent ERP difference waveforms consisted primarily of a P2 wave and a P3 wave. The amplitude of the P3 wave was significantly reduced at shorter SOAs compared with longer SOAs, leading to a significant SOA main effect

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1. Comparable results were obtained with traditional mean RT and peak P3 latency measures.

in the P3 amplitude analysis, $F(2, 22) = 7.21$, $p < .005$. There was also a slight decrease in P3 latency at longer SOAs, leading to a significant main effect of SOA in the P3 latency analysis, $F(2, 22) = 5.33$, $p < .05$. However, as can be seen in Figure 1b, the decline in P3 latency between the shortest and longest SOAs was only 51 ms, less than 25% of the size of the effect of SOA on RT. In addition, neither the amplitude nor the latency of the P2 wave was significantly affected by SOA.

The finding of no significant effect of SOA on P2 latency and only a small effect of SOA on P3 latency is consistent with the hypothesis that the delays in RT2 at short T1-T2 SOAs are caused primarily by interference at a relatively late stage of processing, after T2 has been perceived and categorized. However, the decrease in P3 amplitude at short SOAs indicates that there was a reduction in the cognitive resources available for identifying and categorizing T2 at short SOAs,[2] and this is also the most likely explanation of the slight effect of SOA on P3 latency. In contrast, there was no effect of SOA on P2 amplitude or latency, even though the P2 component was sensitive to T2 probability and therefore must have followed T2 discrimination. Thus, the reduction in P3 amplitude probably reflects interference at a fairly late stage, after the initial identification of T2. These results generally support the model of Pashler (1994), with the primary interference effect occurring at a late stage of processing and a more modest interference effect occurring at a somewhat earlier—but still fairly late—stage of processing. Similar conclusions were reached by Hoffman, Houck, MacMillan, Simons, and Oatman (1985), who also used the P3 wave to examine dual-task interference.

These conclusions are based on the finding that P3 latency was much less affected by SOA than was RT. However, although previous studies have shown that increases in perceptual difficulty lead to comparable changes in RT and P3 latency (Kutas et al., 1977; Luck & Hillyard, 1990), it is important to demonstrate that the small size of the P3 latency effect in the present experiment was not due to a lack of sensitivity or power. This issue was addressed in Experiment 2.

EXPERIMENT 2

T2 perceptual difficulty was manipulated directly in Experiment 2 so that the sensitivity of P3 latency to changes in perceptual difficulty could be assessed. Specifically, T2 was either very bright or very dim, and P3 latency was expected to be longer for dim targets than for bright targets.

The model of Pashler (1994) makes additional predictions about interactions between SOA and brightness, as illustrated in Figure 2. At long SOAs, the increased amount of time required to identify the dim T2 compared with the bright T2 should lead directly to a substantial increase in RT2. At short SOAs, however, response selection for T2 is

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2. Because the P3 wave was measured from infrequent-minus-frequent difference waves, it is possible that the apparent reduction in P3 amplitude at short SOAs was caused by an increase in P3 amplitude on frequent-target trials rather than a decrease in P3 amplitude on infrequent-target trials. This possibility was ruled out by an additional experiment in which the Adjar (adjacent response) filter technique (Woldorff, 1988), rather than difference waves, was used to eliminate the problem of overlapping ERP waveforms. This technique made it possible to assess the P3 wave separately for frequent and infrequent targets. The findings were consistent with the results of Experiment 1: Significant decreases in P3 amplitude were observed at short SOAs for both frequent and infrequent targets in this experiment, and SOA had only small effects on P3 latency.
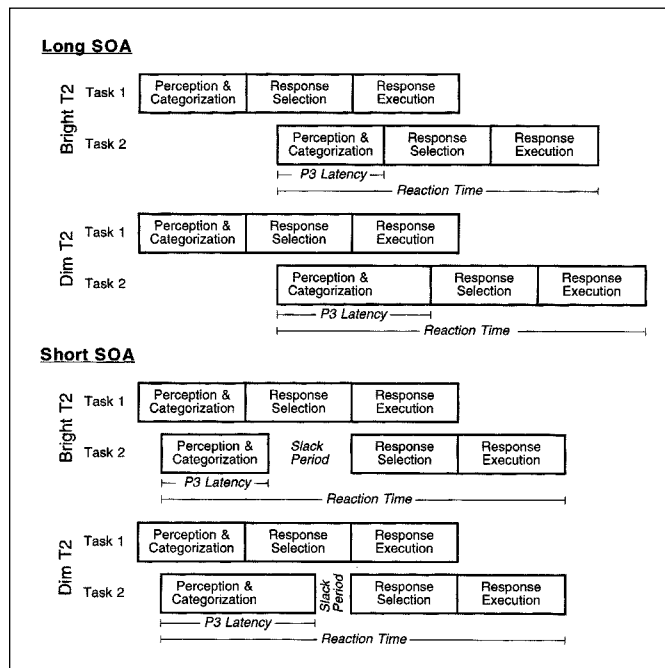

Fig. 2. Postponement model of dual-task interference, in which the response selection stage for the second task cannot begin until response selection for the first task has been completed (adapted from Pashler & Johnston, 1989). Decreases in the brightness of the second target (T2) increase the amount of time required to perceive and categorize T2. At long stimulus onset asynchronies (SOAs), this increase leads to a delay in the onset of response selection and therefore an increase in response time for the second target (RT2). At short SOAs, however, response selection for T2 is postponed until after response selection for the first target has been completed, leading to a period of cognitive slack that can be used to cushion the effects of increases in the time required for T2 identification. As a result, the effect of T2 brightness on RT2 is reduced at short SOAs. However, because P3 latency reflects processes that occur before the slack period, the effects of T2 brightness on P3 latency are equal at short and long SOAs.

postponed, leaving a period of "cognitive slack" between the categorization and response selection processes. Because increases in the amount of time required to perceive T2 are pushed into this slack period, there should be little or no effect of T2 brightness on RT2 at short SOAs. This pattern of RT2 results has been observed in several previous experiments (e.g., De Jong, 1993; McCann & Johnston, 1992; Pashler & Johnston, 1989). A different set of predictions can be made for the P3 wave, however. Specifically, because the cognitive slack at short SOAs follows the stage of processing indexed by P3 latency, the effects of T2 brightness on P3 latency should be the same at short and long SOAs. Thus, Pashler's model predicts an interaction between T2 brightness and SOA for RT2, but no interaction for P3 latency.

Method

Twelve new individuals participated in this experiment. The stimuli and procedures were identical to those in Experiment 1, except that T2

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was bright (35.27 cd/m^2) on 50% of the trials and dim (1.58 cd/m^2) on the remaining trials.

Results and Discussion

The results of Experiment 2 are summarized in Figure 3. As in Experiment 1, RT2 decreased as the SOA increased and was longer for the infrequent T2 than for the frequent T2. In addition, RT2 was longer when T2 was dim than when T2 was bright, and this effect was larger at long SOAs than at short SOAs. This interaction replicates previous studies (e.g., Pashler & Johnston, 1989) and supports the hypothesis that the delay in RT2 at short SOAs reflects a postponement in a relatively late process, such as response selection.

As in Experiment 1, there was a significant effect of SOA on P3 latency, $F(1, 11) = 5.32$, $p < .05$, but this effect was substantially

Fig. 3. Results for the second target (T2) in Experiment 2. Shown in (a) are response times (RTs) and P3 latencies, averaged across participants. Shown in (b) are difference waveforms (infrequent minus frequent) of event-related potentials, recorded at the midline parietal electrode site and averaged across participants and stimulus onset asynchronies (SOAs).

smaller than the effect of SOA on RT2 (see Fig. 3a). In addition, P3 latency was delayed by approximately 60 ms when T2 was dim compared with when T2 was bright, yielding a significant main effect of T2 brightness, $F(1, 11) = 7.41$, $p < .005$ (see Fig. 3b). This effect was even larger than the effect of T2 brightness on RT2, which was approximately a 30-ms difference at the 350-ms SOA.[3] This large and highly significant effect of T2 brightness on P3 latency demonstrates that the P3 wave is highly sensitive to changes in the amount of time required to perceive and categorize a stimulus. Thus, the small size of the effect of SOA on P3 latency cannot be explained by a lack of power or sensitivity, and instead provides strong evidence that the slowing of RT2 at short SOAs is primarily due to a delay in a process that occurs after the target has been perceived and categorized, such as response selection.

Although T2 brightness strongly influenced P3 latency, the interaction between T2 brightness and SOA did not approach significance, $F(2, 22) = 1.05$, $p > .35$. This lack of interaction contrasts with a significant Brightness × SOA interaction for RT2, $F(2, 22) = 6.06$, $p < .01$. This dissociation between P3 latency and RT2 was directly predicted by the model of Pashler (1994), as illustrated in Figure 2.

As in Experiment 1, P3 amplitude was somewhat smaller at shorter SOAs than at longer SOAs, $F(2, 22) = 9.24$, $p < .002$. Again, this result provides evidence for some modest interference between the two tasks at an intermediate stage of processing.

Although the P2 wave was not as large in Experiment 2 as it was in Experiment 1, P2 latency was clearly longer for dim targets than for bright targets, $F(1, 11) = 13.47$, $p < .005$. In addition, there was no effect of SOA on P2 amplitude or latency, nor did T2 brightness interact with SOA.

GENERAL DISCUSSION

Prior research has indicated that dual-task interference in the PRP paradigm occurs after the early sensory stage and before the motor execution stage (Osman & Moore, 1993; Pashler & Johnston, 1989), and the results of the present study provide converging electrophysiological evidence for this conclusion. More important, the present results narrow the range of possible sources of interference by demonstrating that the delays in RT2 at short T1-T2 SOAs are primarily due to a delay in a process that follows the identification and categorization of T2, such as response selection.

These results also indicate that some interference is present at a somewhat earlier—but still fairly late—stage of processing. Specifically, there was no effect of SOA on the amplitude or latency of the P2 component, but the P3 component was slightly smaller and slightly delayed at short SOAs. Thus, the participants were initially able to identify T2 without any interference (yielding an unsuppressed P2 wave), but some interference was present at a later stage (yielding a partially suppressed P3 wave). The leading theory of the P3 wave proposes that it reflects a process associated with the updating of working
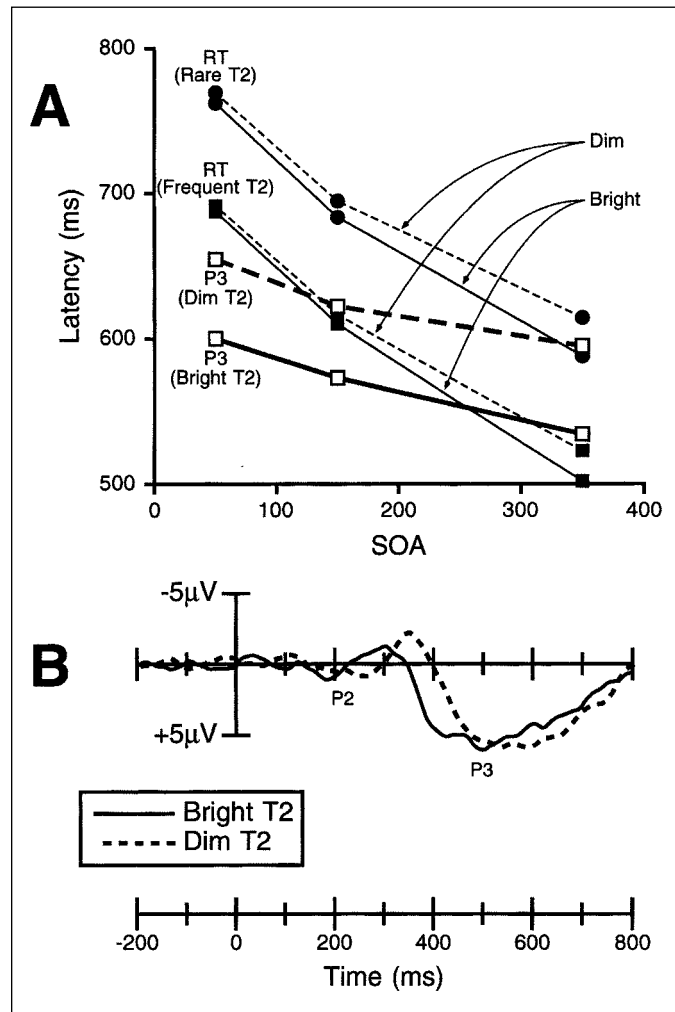
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3. The most likely explanation for the larger effect of T2 brightness on P3 latency than on RT2 is that some of the effect of T2 brightness on the duration of T2 identification fell into the cognitive slack period even at the longest SOA, thereby decreasing the apparent effect of T2 brightness on RT2 without influencing the effect of T2 brightness on P3 latency. At a sufficiently long SOA, the P3 and RT2 effects would be expected to be identical.

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memory (Donchin, 1981), and the present results therefore suggest that this process is somewhat impaired at short SOAs in the PRP paradigm.

This conclusion dovetails nicely with recent studies of the *attentional blink* phenomenon (see review by Shapiro & Raymond, 1994). The attentional blink is an impairment in accuracy for identifying a masked T2 at short SOAs, and this impairment is analogous to the increased RTs observed at short SOAs in the PRP paradigm. Speeded responses are not required in the attentional blink paradigm, and so the impairment in T2 accuracy at short SOAs cannot be attributed to interference at the stage of response selection; the attentional blink may therefore reflect an exaggerated version of the P3 suppression effect observed in the present study. Indeed, ERP experiments have indicated that perceptual processing is unimpaired during the attentional blink, but the P3 wave is completely suppressed (Luck, Vogel, & Shapiro, 1996; Vogel, Luck, & Shapiro, in press). Together, these results indicate that severe dual-task interference can arise either at a late stage (i.e., at the stage of response selection in the PRP paradigm) or at a somewhat earlier stage (i.e., at the stage of working memory encoding in the attentional blink paradigm), depending on the nature of the stimuli and task.

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