Electrodermal responses to sources of dual-task interference

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Abstract There is a response selection bottleneck that is responsible for dual-task interference. How the response selection bottleneck operates was addressed in three dual-task experiments. The overlap between two tasks (as indexed by the stimulus onset asynchrony [SOA]) was systematically manipulated, and both reaction time and electrodermal activity were measured. In addition, each experiment also manipulated some aspect of the difficulty of either task. Both increasing task overlap by reducing SOA and increasing the difficulty of either task lengthened reaction times. Electrodermal response was strongly affected by task difficulty but was only weakly affected by SOA, and in a different manner from reaction time. A fourth experiment found that the subjectively perceived difficulty of a dual-task trial was affected both by task difficulty and by SOA, but in different ways than electrodermal activity. Overall, the results were not consistent with a response selection bottleneck that involves processes of voluntary, executive attention. Instead, the results converge with findings from neural network modeling to suggest that the delay of one task while another is being processed reflects the operation of a routing mechanism that can process only one stream of information for action at a time and of a passive, structural store that temporarily holds information for the delayed task. The results suggest that conventional blocked or event-related neuroimaging designs may be inadequate to identify the mechanism of operation of the response selection bottleneck.

Keywords Electrodermal activity · Electrodermal response · Skin-conductance response · Dual-task interference · Psychological refractory period

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Introduction

A widely held view is that humans are able to multitask effectively—that is, to successfully carry out two simultaneous tasks, such as conversing and driving an automobile. The empirical, scientific evidence clearly shows this to be false; performance on one or both tasks is degraded (in the instance of driving, see, e.g., Levy, Pashler, & Boer, 2006; Strayer & Drews, 2007). There are three principal questions to be asked about dual-task interference: What are the behavioral results of the interference? Where does the interference occur in the brain? How does the interference operate? The behavioral nature of the interference as seen in task reaction times (RTs) is well understood. Under most circumstances in which two tasks overlap, central processing (such as response selection) on one task must be halted until central processing on the other task is completed (see Pashler, 1994, 1998, for reviews). Concerning brain localization, studies using neuroimaging converge in implicating a neural substrate that is somehow involved with dual-task interference and that involves lateral and medial frontal mechanisms interacting with parietal mechanisms (for a review, see Marois & Ivanoff, 2005). Concerning how dual-task interference operates, we will first outline two competing explanations, and then we will use electrodermal activity (EDA) as the dependent measure to assess those explanations. Finally, we will consider the implications of the results for neuroanatomical models of dual-task processing.

The nature of dual-task interference: Behavioral studies

The experimental approach that gives the greatest analytical leverage in understanding dual-task interference is to present two distinct stimuli whose onsets are separated by a systematically manipulated stimulus onset

asynchrony (SOA) and that require two distinct, speeded responses. A large number of behavioral experiments have shown that under a very wide range of circumstances as the overlap between the first and second tasks increases—that is, as the SOA shortens—the RT to complete the second of the two tasks systematically lengthens (for reviews, see Pashler, 1994, 1998). This task 2 RT lengthening with decreasing SOA is called the *psychological refractory period (PRP) effect* (Vince, 1948; Welford, 1952), by analogy with the refractory period when a neuron is unresponsive to stimulation after an immediately preceding action potential (Marey, 1876). The results from such studies appear to be well fit by a model that posits a structural, passive bottleneck that limits access to a central response selection mechanism to one task at a time. This is often called the *response selection bottleneck model*. Because task 1 gains access to central processing first, task 1 RTs are unaffected by SOA. At short SOAs, central processing of task 2, however, must wait until that of task 1 is completed, so task 2 RTs will increase linearly as the SOA is decreased. At sufficiently long SOAs, the response for task 1 is completed before the stimulus for task 2 is presented, and task 2 RTs should be unaffected by SOA. The model predicts, and neuroscientific evidence confirms, that under most circumstances, both precentral processing (such as perceptual identification) and postcentral processing (such as response execution) for the two tasks can be carried out in parallel (Sigman & Dehaene, 2008). Consistent with the view that the central bottleneck reflects a structural limitation of the cognitive architecture, bottleneck interference can be found even after thousands of trials of dual-task training (e.g., Ruthruff, Johnston, & Van Selst, 2001; Van Selst, Ruthruff, & Johnston, 1999).

In contrast to the passive, immutable structural delay posited by the response selection bottleneck model, Meyer and Kieras (1997a, 1997b) proposed that the bottleneck is functional rather than structural and that it is under strategic, executive control. These authors suggested that under certain circumstances, the response selection bottleneck would not be put in place and, as a result, dual-task interference would not be observed. Consistent with this argument, there have been demonstrations with no observable dual-task interference, but only in some individuals and only under certain very specific conditions (Hazeltine, Teague, & Ivry, 2002; Maquestiaux, Laguë-Beauvais, Ruthruff, & Bherer, 2008; Ruthruff, Van Selst, Johnson, & Remington, 2006; Schumacher et al., 2001; but also see Anderson, Taatgen, & Byrne, 2005; Ruthruff, Johnston, Van Selst, Whitsell, & Remington, 2003; Tombu & Jolicoeur, 2004). Although the behavioral evidence favors the explanation of a passive, structural mechanism rather than an active, functional mechanism that is under

executive, cognitive control, converging evidence will be necessary to decide between the explanations.

Localization of dual-task interference: Neuroimaging studies

A number of fMRI studies of dual-task interference have been carried out using several different approaches (for a review and meta-analysis, see Marois & Ivanoff, 2005). Despite the variety of approaches, including new, time-resolved approaches reported more recently (Dux, Ivanoff, Asplund, & Marois, 2006; Sigman & DeHaene, 2008), the results converge on activations in the lateral frontal, medial frontal, premotor, and parietal cortex in high-overlap dual-task situations. Although different researchers describe it somewhat differently, a consensus model has emerged of how these areas might be involved in executive control of dual-task processing. In this consensus model, the lateral prefrontal cortex is optimized for rapid, adaptive control that is not restricted to one modality (Dosenbach, Fair, Cohen, Schlaggar, & Petersen, 2008; Dosenbach et al., 2007; Dosenbach et al., 2006; Marois, Larson, Chun, & Shima, 2006) and that is involved in the fast adaptation of response sets and coordination of selection of a processing stream for behavioral action (selection-for-action) in situations with interfering information (Collette et al., 2005; Koechlin, Basso, Pietrini, Panzer, & Grafman, 1999; Schubert & Szameitat, 2003; Szameteit, Schubert, Müller, & Von Cramon, 2002). Medial areas, including the anterior cingulate cortex (ACC), are optimized for stable set maintenance. That is, the ACC maintains and monitors associations between actions and their outcomes and manages the implementation of task sets particularly in situations of conflict (Dosenbach et al., 2008; Dosenbach et al., 2006; Fleck, Daselaar, Dobbins, & Cabeza, 2006; MacDonald, Cohen, Stenger, & Carter, 2000; Rowe, Hughes, Eckstein, & Owen, 2008). The lateral and medial prefrontal areas interact to exercise top-down control, biasing signals to parietal areas that load, transmit, or instantiate the required task set parameters (Dosenbach et al., 2008; Dosenbach et al., 2006; MacDonald et al., 2000; Sigman & DeHaene, 2006).

There is one important unresolved question from the neuroimaging results. The consensus model presumes that the dual-task situation increases demands for additional attentional control when the second task follows closely on the first (e.g., Erickson et al., 2005), consistent with the operation of an active, functional mechanism under executive control, such as that posited by Meyer and Kieras (1997a, 1997b). Some researchers, however, have found very few differences between the loci or levels of cortical activations in high- and low-task-overlap conditions (Jiang,

Saxe, & Kanwisher, 2004; Marois et al., 2006; Sigman & Dehaene, 2008). In contrast to claims that dual-task interference is due to active, executive control, they argued that the behavioral slowing is simply due to passive queuing as the second task waits for access to the response selection mechanism. In this view, the slowing is due not to increased executive activity, but simply to postponement, consistent with the interpretation that the bottleneck is passive and structural. If the response selection bottleneck is due to a passive mechanism, attempts to identify a unique cortical location dedicated to dual-task management using conventional blocked or event-related designs may not be successful.

How dual-task interference occurs: Electrodermal responses

In the present research, we used measures of EDA to address the question of whether the bottleneck is active or passive. If it is the result of active, controlled processes, its operation should result in autonomic activity and, thus, detectable electrodermal responses (EDRs). If it is the result of passive, reactive processes, there might be no additional autonomic activity and, thus, no EDR. EDA has been widely used from the earliest emergence of psychology as a science because phasic responses (EDRs) provide a sensitive index of sympathetic nervous system response to the processing of task-relevant stimuli (for a review, see Dawson, Schell, & Filion, 2007).

EDR reflects the demands of a task for controlled, effortful, executive processing of task-relevant stimuli. It has been widely confirmed that EDA responds to changes in task demands (e.g., Gendolla & Krüsken, 2001; Kohlisch & Schaefer, 1996; Naccache et al., 2005; Neumann, Lipp, & Siddle, 2002). Damasio (1994, 1999) has proposed that EDR reflects autonomic arousal that acts as a somatic marker for the level of demand on executive attention that similar tasks have elicited in the past. We note also that activations shared by the two tasks and those affected by task overlap in dual-task situations (Sigman & Dehaene, 2008) and those underlying change in EDA (Nagai, Critchley, Featherstone, Trimble, & Dolan, 2004) are proximally located in the premotor cortex, the insula, the cerebellum, and the lateral prefrontal cortex, although activations in these areas are seen in many tasks.

Factors that influence task 2 RT in the PRP procedure produce effects that are reliable and well explained by the response selection bottleneck model (for a review, see Pashler, 1994). Those factors include the difficulty of the precentral, central, and postcentral stages of the first task and of the second task and, most saliently, the SOA between the two tasks. In the first three experiments, we systematically explored the effects of manipulating SOA, as well as the effects of manipulating task difficulty factors that affect the executive demands of specific stages of processing, and we looked for

dependent changes in EDR, as well as in RT. If a manipulation increases the demands on controlled, executive processes, RT should increase, and EDR should increase. Specifically, if the response selection bottleneck is the result of active, effortful, controlled executive processing, EDR should be responsive to SOA, as well as to other manipulations of task difficulty. If, however, the response selection bottleneck involves passive, automatic, noncontrolled processing, EDR should be responsive to other manipulations of task demands but should not respond to SOA, because SOA does not necessitate executive control. The results should be informative about whether the response selection bottleneck operates actively or passively and, therefore, about constraints on neuroanatomical models of dual-task processing.

We are unaware of any previous studies that have collected EDR measures while presenting two RT tasks and systematically manipulating the SOA between the two tasks.

General method: Experiments 1–3

Participants

Because the participants were drawn from the same population and because there were no significant differences among experiments in demographic characteristics, we report the characteristics of the entire sample here. Two hundred sixty-three individuals volunteered to participate in these experiments, including 96 men and 167 women. Participants were undergraduate students recruited from introductory psychology courses and also via convenience sampling using word-of-mouth advertising. The participants ranged in age from 18 to 23 years, with an average age of 19.95 years ($SD = 1.19$ years). On a 10-point health-rating scale, with 10 indicating *excellent health*, the average rating was 8.56 ($SD = 1.04$). At the end of the procedure, far visual acuity was tested, and participants were asked whether or not they were red–green colorblind. Their Snellen visual acuity, measured at 6.1 m (20 ft), ranged from 20/15 to 20/50, with a mean of 20/20.89 ($SD = 5.69$). Two participants were excluded for self-reported color blindness. For compensation, the participants received either \$10.00 or extra course credit. The research was approved by a duly constituted Institutional Review Board, and all participants were treated within the ethical guidelines of the American Psychological Association.

Procedure

Participants provided informed consent. Before beginning the computer tasks, two BioPac Ag-AgCl foam-backed electrodes with a 0.5 % chloride gel (EL507) were placed on the palmar surface of the participant's left hand in order to measure his or her skin conductance responses. One

electrode was placed on the thenar eminence of the palm below the base of the thumb, while the other was placed on the hypothenar eminence below the base of the little finger. EDA measures were acquired using a BioPac Systems GSR transducer (SS3L) and acquisition unit (MP30). BioPac Lab Pro software (v3.6.6) was used for all data acquisition and preprocessing. Experimental tasks were presented using E-Prime software (Psychology Software Tools, Inc.) on a standard Intel/Windows computer. At the start of each block of an experiment, the EDA output was synchronized with the behavioral data output. For each trial, EDA was analyzed for a window from the onset of the task 1 stimulus until the start of the next trial. The window duration spanned 6 s in Experiments 1 and 2 and 8 s in Experiment 3. Preliminary testing indicated that this time between trials was sufficient for EDA to return to baseline. We measured EDA change with a 0.05-Hz high-pass filter. For each trial, we obtained the area under the skin conductance change curve during that period, measured in microSiemens (μS). The psychophysiological measure was then linked to the information about that trial in the experimental protocol, including the accuracy and latency of each response. The distributions of areas were strongly positively skewed, and consequently, a natural-log transformation was applied to correct for skew. Because the natural log is not defined for zero or negative values, this had the effect of excluding any trial on which there was not a positive deflection. We also measured the maximum conductance on each trial, but the maxima and areas were highly correlated (across the three experiments, the correlation ranged from .80 to .87). For simplicity, then, only the results of analyses for the log-transformed area are reported here.

Experiment 1

Experiment 1 was designed to determine the effect of the SOA between two simple, discrete tasks—a color judgment and a number judgment task—on EDA, as well as on RT. For this preliminary step, we explored a small set of SOAs—0 ms (simultaneous onset), 100, 200, and 1,000 ms—in a large number of participants.

Method

One hundred forty-five individuals from the pool described above participated in this experiment. Each trial began with a white fixation cross (+) presented in 36-point Courier New bold font, displayed in the center of a computer screen on a dark background. The approximate viewing distance was 46 cm. After 1,000 ms, the cross was replaced by the letter O, which was colored either red or green. Then, after an SOA of 0, 100, 200, or 1,000 ms, the colored circle was replaced by a single-digit number (1, 2, 3, 4, 5, 6, 7, 8, or 9),

with the color of the number the same as the color of the previously displayed circle (either red or green). This number remained on the screen for 500 ms. The participant's first task on each trial was to indicate the color (red or green) as quickly and accurately as possible, by pressing designated keys on the keyboard with the left index ("x") and second ("z") fingers. The participant's second task on each trial was to indicate whether the number was odd or even as quickly and accurately as possible, by pressing the period key with the right index finger or the slash key with the second finger. We allowed 3,000 ms for responding. The intertrial interval was adjusted so that the total time from the onset of the first stimulus on one trial to the onset of the first stimulus on the next was fixed at 6 s, the window for EDA collection. After 40 trials of practice with each of the two single tasks performed alone, participants were given 40 practice dual-task trials with accuracy feedback, followed by 240 experimental trials without feedback. Ad lib rest breaks were given every 60 trials. The order of trial types was randomly determined.

Results

For all analyses reported here, alpha was set at .05. Tests of sphericity were carried out, and, where significant, Greenhouse–Geisser adjusted probabilities are reported, although the adjusted degrees of freedom are not given.

RT An ANOVA was carried out on the RTs for both the first (color) and second (number) tasks for trials on which both responses were correct.¹ The SOA (0–1,000 ms) was a within-subjects factor. As is shown in the left panel of Fig. 1, there was a slight 120-ms reduction of task 1 RT from long to short SOAs, $F(3, 423) = 31.09$, $p < .001$, $\eta_p^2 = .18$, which may have occurred because the arrival of the task 2 stimulus on shorter SOA trials speeded the task 1 response. Alternatively, some individuals may have withheld their response to task 1 until task 2 was complete on some trials and emitted both responses at the same time, slowing task 1 RT. This absence of slowing of task 1 RTs at short SOAs is clearly inconsistent with the view that the two tasks are competing for resources (for further details, see Hartley & Little, 1999). Most important, task 2 RT increased significantly and monotonically, slowing by 409 ms from an SOA of 1,000 ms to an SOA of 0 ms, $F(3, 423) = 400.64$, $p < .001$, $\eta_p^2 = .74$.²

Area An equivalent ANOVA was carried out on the log-transformed measures of the area under the EDA change

¹ To economize, we do not include analyses of proportions of errors for any of the experiments we report. The average proportion of errors across Experiments 1–3 was .055.

² Partial eta-squared (η_p^2) is calculated in all of the analyses reported here as $SS_{\text{EFFECT}}/(SS_{\text{EFFECT}} + SS_{\text{ERROR(EFFECT)}})$.

Fig. 1 *Left panel:* Task 1 and task 2 reaction times in [Experiment 1](#) as a function of stimulus onset asynchrony (SOA). Bars showing standard errors are not visible at this resolution. *Right panel:* Log-transformed

area (in μ Siemens) under the electrodermal activity change curve as a function of SOA in [Experiment 1](#). Bars show standard errors

curve on each trial (in μ S). There was a significant effect of SOA, $F(3, 423) = 4.06$, $p = .007$, $\eta_p^2 = .03$, as can be seen in the right panel of [Fig. 1](#). Follow-up tests using the modified Bonferroni procedure showed that the log area at an SOA of 100 ms ($M = -1.63 \mu$ S, $SE = 0.09 \mu$ S) was greater than that at the other SOAs (0, $M = -1.70 \mu$ S, $SE = 0.09 \mu$ S; 200, $M = -1.72 \mu$ S, $SE = 0.09 \mu$ S; 1,000, $M = -1.74 \mu$ S, $SE = 0.09 \mu$ S), which did not differ. In contrast to task 2 RT, EDA did not respond monotonically to reduction in SOA.

Discussion

The results for RT replicated the well-established PRP effect. Whereas the RTs for task 1 were only moderately affected by SOA, the RTs for task 2 were slowed by 409 ms at the shortest SOAs relative to the longest. This pattern of results is what would be expected if central processing were devoted to task 1 until response selection was completed and only then could the central processor be directed to task 2. The electrodermal measure, the total area under the skin conductance curve, was affected by task overlap (SOA), but in a way quite different from task 2 RT. EDA was elevated only when the SOA separating task 1 and task 2 was 100 ms. With simultaneous onset—that is when the overlap was complete (SOA = 0) and when RT slowing was at its maximum—there was no sign of an EDR. Furthermore, the effect size for SOA was very much smaller for EDA than for task 2 RT, with the effect size for RT 25 times that for EDA. We will withhold further discussion of the SOA effect until more evidence has been presented.

Experiment 2

The response selection bottleneck model predicts that any manipulation that increases the difficulty of central processing for the first task in a dual-task situation will carry over, slowing the task 2 RT at short SOAs, but not at the longest

SOA. When task 1 is more difficult, the time required to complete task 1 central processing is lengthened, thus postponing the start of task 2 central processing. At sufficiently long SOAs, task 1 will have been completed prior to the arrival of the stimulus for task 2. In this case, the effect of the difficulty of task 1 will not carry over to task 2 performance. The resulting prediction is that the effect of manipulating the difficulty of task 1 will interact with the effect of SOA in affecting task 2 RT; task 2 will be slowed substantially at short SOAs by a more difficult task 1 but will be unaffected at long SOAs when task 1 processing is likely to have been completed, whether easy or difficult. This might be called the *overadditivity prediction* (cf. principle 1 in Pashler, 1994). We tested this prediction in [Experiment 2](#), which was nearly identical to [Experiment 1](#) except that the duration of the task 1 central stage was manipulated by adding a new, *difficult*, condition, requiring discrimination of four colors (red, green, blue, and yellow), in addition to the *easy* condition from [Experiment 1](#) with only two colors (red and green). Our intention was to replicate the finding that SOA affected the EDR only at short, nonzero SOAs, as well as to determine whether another manipulation of central stage difficulty would also affect the EDR.

Method

Forty individuals from the previously described pool participated. The number of SOAs used was increased from [Experiment 1](#) in order to explore the effects of SOA with a finer grain: 0-, 50-, 100-, 150-, 200-, 500-, or 1,000-ms SOA. Forty trials of practice, with feedback, were given in each of the single-task conditions, as well as in the difficult dual-task condition. Participants then completed 320 dual-task trials without feedback in each of the two conditions, easier (two-choice) and harder (four-choice), with the order counterbalanced across participants.

Results

RT ANOVAs were carried out on the RTs for task 1 (the color task) and on the RTs for task 2 (the number task) for trials on which both responses were correct. SOA (0–1,000 ms) and task 1 difficulty (easier, two choices; harder, four choices) were both within-subjects factors. For task 1 (color task) RT, there was a significant main effect of task 1 difficulty, $F(1, 39) = 80.40$, $p < .001$, $\eta_p^2 = .67$. There was also a significant main effect of SOA, $F(6, 234) = 3.36$, $p = .003$, $\eta_p^2 = .08$, but there was no interaction of the two, $F(6, 234) = 1.17$, $p = .323$, $\eta_p^2 = .03$. For task 2 (number task) RT, there were significant main effects of task 1 difficulty, $F(1, 39) = 99.61$, $p < .001$, $\eta_p^2 = .72$, and SOA, $F(6, 234) = 227.82$, $p < .001$, $\eta_p^2 = .85$. There was also a significant interaction of task 1 difficulty and SOA, $F(6, 234) = 6.08$, $p < .001$, $\eta_p^2 = .14$. Consistent with the overadditivity prediction, this effect carried over to the responses for task 2; the effect of task 1 difficulty on task 2 RT was greater at 0 ms ($M_D = 167$ ms) than at 1,000 ms ($M_D = 68$ ms). The results are shown in the left panel of Fig. 2.

Area An ANOVA on log-transformed areas showed a significant main effect of task 1 difficulty, $F(1, 39) = 9.71$, $p = .003$, $\eta_p^2 = .20$, with greater EDA response in the higher-difficulty four-choice condition ($M = -1.09$, $SE = 0.18$) than in the lower-difficulty two-choice condition ($M = -1.42$, $SE = 0.18$). Although the pattern of means, averaged over difficulty, was similar to that found in Experiment 1 (shown by the dotted line in the right panel of Fig. 2), the main effect of SOA was not significant, $F(6, 234) = 1.46$, $p = .191$, $\eta_p^2 = .04$. Unprotected t tests showed that the area was significantly greater for an SOA of 50 ms than for an SOA of 500 or 1,000 ms. There was no evidence of the interaction of task 1 difficulty and SOA that was seen in RT, $F(6, 234) = 0.51$, $p = .750$, $\eta_p^2 = .04$.

Discussion

Once again, we replicated the well-established SOA effect with RT, and as well, we demonstrated the overadditive

interaction effect predicted by the bottleneck model. RT to the second task was slowed as the overlap between the two tasks increased (i.e., as the SOA decreased), and this effect was exaggerated when the difficulty of the first task increased. In contrast, there was no significant relation between the area under the EDA curve and SOA in Experiment 2, although the pattern of lower response at the 0-ms SOA followed by elevated response at short, nonzero SOAs closely resembled that seen in Experiment 1. The very strong effect of task 1 difficulty on task 2 RTs ($\eta_p^2 = .72$) was reflected in a moderately strong effect on EDA ($\eta_p^2 = .20$). Most important, the overadditive effect of task 1 difficulty that was clearly evident in task 2 RTs with short SOAs was completely absent in EDA. A helpful tool in thinking about these results is additive factors logic (Sternberg, 1969; for more recent discussions and defenses, see Roberts & Sternberg, 1993; Sternberg, 1998). If two manipulations affect different stages in processing, they will have additive effects on the dependent variable being measured. If, however, they affect the same stage, they will have an interactive effect on the measured variable. In the response selection bottleneck model, both SOA and task 1 difficulty affect how long task 2 central processing will be delayed, and the result is an overadditive interaction effect on task 2 RT. This is precisely what was observed. For EDA, however, the effects were additive, indicating that task overlap and the difficulty of task 1 were affecting independent stages in the processes underlying autonomic activation.

Experiment 3

Experiment 3 tested two predictions of the response selection bottleneck model. The first is a strongly counterintuitive prediction that might be called the *underadditivity* prediction (cf. principle 3 in Pashler, 1994). According to this prediction, any manipulation increasing the difficulty of the precentral stages of task 2 (e.g., making the perceptual identification of the

Fig. 2 Left panel: Task 1 and task 2 reaction times in Experiment 2 as a function of task 1 difficulty and stimulus onset asynchrony (SOA). Bars show standard error. Right panel: Effect of task 1 difficulty and

stimulus onset asynchrony (SOA) on electrodermal activity (log-transformed area under the curve in μ Siemens) in Experiment 2. Bars show standard errors. Dotted line shows main effect of SOA

stimulus more difficult) will be “absorbed” in the bottleneck delay or *cognitive slack* (e.g., McCann & Johnston, 1992) allowed by the suspension of the processing of task 2 until central processing of task 1 is complete at short SOAs. This leads to the counterintuitive prediction that increasing the perceptual difficulty of task 2 will result in less slowing of task 2 RTs when there is high overlap with task 1 (i.e., at short SOAs with a long bottleneck delay) than when there is low overlap (i.e., at long SOAs when there is little or no bottleneck delay). The second prediction of the bottleneck model that was tested in Experiment 3 might be called the *additivity prediction* (cf. principle 4 in Pashler, 1994). Increasing the difficulty of the central processing for task 2 should slow responses to task 2, but because the effect of increased difficulty comes after the bottleneck, the additional time will simply be added on to the RT for task 2 at all SOAs.

For Experiment 3, we adapted a dual-task procedure developed by Sigman and DeHaene (2006) that allowed convenient manipulation of several factors in the same experimental session. The first task was to indicate whether a stimulus (a row of crosses) was red or green in color. The second task was to indicate whether a number was greater than or less than 45. In this number task, the difficulty of perceptual identification—a precentral stage—was manipulated by presenting the number either as digits or spelled as a word. Sigman and DeHaene (2006) found that RTs were significantly longer when the number was spelled as a word rather than presented as digits. The difficulty of task 2 response selection—a central stage—was manipulated by presenting numbers that were either relatively near to 45 or relatively far from 45. Sigman and DeHaene (2006) found that RTs were significantly longer for near numbers than for far numbers. Manipulating the duration of precentral and central stages of the second task allowed us to test the underadditivity and the additivity predictions of the response selection bottleneck model in the same experiment. Our intention was to determine whether these manipulations affected EDR in the same way as or differently than they affected RT.

Method

Forty individuals drawn from the population previously used participated in this experiment. Each trial began with a fixation stimulus, five black crosses (+++++) in 18-point Courier font and centered on the display, which was presented on a white screen for 1,000 ms. The color of the fixation stimulus then changed from white to red or green. The participant identified the color as red or green by pressing the “x” key with the left index finger or the “z” key with the second finger. After an SOA of 0, 50, 100, 200, 500, or 1,000 ms, the fixation stimulus was replaced by a number displayed in the same color. The numbers were presented either as digits or spelled

as words. The participant indicated whether the number was greater or less than 45 by a pressing the period key with the right index finger or the slash key with the second finger. Half of the numbers were greater than 45; half were less. Half of the numbers were close to 45 (38, 39, 41, 42 or 47, 49, 51, 52), and half were far from 45 (10, 11, 13, 14 or 77, 78, 81, 82). The number was erased after 1,000 ms. The time allotted for each response was 3,000 ms. After 40 practice trials on each single task and 40 on the dual task with feedback, participants completed 216 dual-task trials without feedback. The intertrial interval was 8 s.

Results

Because the design is fairly complex, we report only those specific analyses that address the hypotheses. To conserve space, we do not report ANOVAs of task 1 RT. We focus on the critical question of whether the predicted effects on task 2 RT were observed. Analyses were carried out on trials for which both responses were correct.

SOA The first test was of the overall effect of SOA. The effect of SOA on task 2 RT was significant, $F(5, 200) = 114.14$, $p < .001$, $\eta_p^2 = .74$, with mean RT increasing monotonically from 667 ms ($SE = 8$ ms) at the 1,000-ms SOA to 1,084 ms ($SE = 8$ ms) at the 0-ms SOA. The effect of SOA on the log area was also significant, $F(5, 200) = 6.96$, $p < .001$, $\eta_p^2 = .15$, as is shown in Fig. 3. The pattern was distinctly different from the monotonic increase of RTs with decreasing SOA. Follow-up tests showed that the EDR was greatest for 50 ms ($M = -1.30$, $SE = .10$), which was greater than that for the 100-ms SOA ($M = -1.39$, $SE = .10$), which was, in turn, greater than that for SOAs of 0, 500, and 1,000 ms, which did not differ ($M = -1.50$, $SE = .10$). Activation at the 200-ms SOA ($M = -1.42$, $SE = .10$) was intermediate and differed only from that at the 50-ms SOA.

Fig. 3 Log-transformed area (in μ Siemens) under the electrodermal activity change curve as a function of stimulus onset asynchrony (SOA) in Experiment 3. Bars show standard errors

Underadditivity prediction Experiment 3 tested the prediction that the effect of increasing the difficulty of precentral stages in Task 2, such as perceptual identification, could be absorbed into the cognitive slack time in which task 2 was held in abeyance. The signature of this prediction is a sub-additive interaction, with less RT slowing due to the more difficult condition at short SOAs than at long SOAs. We tested this prediction by comparing the effects of SOA in conditions in which the number was presented as digits with those in conditions in which the number was presented as words. ANOVA for task 2 RT showed a significant effect of the task 2 digit/word manipulation, $F(1, 40) = 8.37$, $p = .006$, $\eta_p^2 = .33$, with responses slower to words ($M = 861$ ms, $SE = 26$ ms) than to digits ($M = 816$ ms, $SE = 26$ ms). The interaction of SOA and task 2 difficulty was also significant, $F(5, 200) = 5.65$, $p < .001$, $\eta_p^2 = .12$. The mean difference between digit and word was smaller at the 0-ms SOA ($M_D = 28$ ms) than at the 1,000-ms SOA ($M_D = 63$ ms), indicating that this was a subadditive interaction, consistent with the absorption prediction. The descriptive statistics for task 2 RT are shown in the left panel of Fig. 4. For log area, the effect of the task 2 digit/word manipulation was also significant, $F(1, 40) = 9.21$, $p = .004$, $\eta_p^2 = .23$. Descriptive statistics for log area are shown in the right panel of Fig. 4. Unlike for RT, the interaction of SOA and difficulty was not significant, $F(5, 200) = 0.92$, $p = .469$, $\eta_p^2 = .02$.

Additivity prediction We tested this prediction by comparing conditions in which the number was far from 45 with those in which it was near 45. An ANOVA for task 2 RT showed a significant effect of the task 2 near/far manipulation, $F(1, 40) = 99.90$, $p < .001$, $\eta_p^2 = .71$, with RTs slower by 90 ms for near numbers ($M = 928$ ms, $SE = 26$ ms) than for far numbers ($M = 838$ ms, $SE = 26$ ms). The combination of SOA and difficulty was not additive as had been predicted; the interaction was significant, $F(5, 200) = 5.38$, $p < .001$, $\eta_p^2 = .12$. As can be seen in the left panel of Fig. 5, a narrowing of the near–far difference at the 100- and

500-ms SOAs caused the deviation from additivity. For log area, the effect of the task 2 near/far manipulation was significant, $F(1, 40) = 9.21$, $p = .004$, $\eta_p^2 = .19$, due to higher EDA for near numbers ($M = -1.40$ μ S, $SE = 0.06$ μ S) than for far numbers ($M = -1.47$ μ S, $SE = 0.06$ μ S). The interaction of difficulty and SOA shown in the right panel of Fig. 5, however, was nonsignificant, $F(5, 200) = 1.23$, $p = .296$, $\eta_p^2 = .03$.

Discussion

First, we once again found a pattern in which EDA did not respond when the two tasks appeared simultaneously (0-ms SOA) but then increased at short SOAs and fell back at longer SOAs. As in Experiment 2, the greatest response was at the 50-ms SOA, whereas the EDRs were comparable to those in Experiment 1 for the SOAs that had been used in that experiment.

Second, the data successfully confirmed the absorption prediction of the bottleneck model. The RT differences between the harder, word condition and the easier, digit condition were smaller at short SOAs than at long SOAs. The strong main effect of digits versus words on task 2 RT ($\eta_p^2 = .75$) was reflected in a modest effect on EDA ($\eta_p^2 = .12$). The underadditive interactive effect with SOA seen in RT was not seen in EDA; the effects of SOA and task 2 precentral or perceptual difficulty were additive. Once again, following additive factors logic, SOA and task 2 perceptual difficulty both affected the same stage for task 2 RT—presumably, task 2 central processing. However, SOA and task 2 perceptual difficulty must have affected independent stages in processes underlying autonomic response.

Third, we obtained somewhat mixed results for the additivity prediction. It had been predicted that increasing the difficulty of the central stages by presenting numbers near to the target, 45, rather than far would result in increased RTs, and this was found. It was further predicted that this increase would have the same effect at all SOAs,

Fig. 4 Left panel: Task 2 reaction times in Experiment 3 as a function of task 2 perceptual difficulty (words vs. digits) and stimulus onset asynchrony (SOA). Bars show standard errors. Right panel: Effect of

task 2 perceptual difficulty (words vs. digits) and SOA on electrodermal activity (area under the curve) in Experiment 3. Bars show standard errors

Fig. 5 *Left panel:* Task 2 reaction times in [Experiment 3](#) as a function of task 2 central stage difficulty (numbers near target vs. far from target) and stimulus onset asynchrony (SOA). Bars show standard

errors. *Right panel:* Effect of task 2 central stage difficulty (numbers near target vs. far from target) and SOA on electrodermal activity (area under the curve) in [Experiment 3](#). Bars show standard errors

resulting in an additive effect. The results deviated from additivity, but most important, they showed neither the signature reduction of difficulty effects at short SOAs characteristic of subadditivity nor the exaggeration characteristic of overadditivity. Physiological measures showed greater EDA for the more difficult near condition than for the less difficult far condition, but that difference was statistically additive with the effects of SOA.

Experiment 4

The results of the three previous experiments converge on the conclusion that the effects of the SOA (the overlap between the two tasks) and of the interaction of manipulations of task difficulty and SOA are not the same for EDA as they are for task 2 RTs. Most salient, evidence of interference in task 2 RTs is largest at the 0-ms SOA and then declines monotonically up to the longest SOA. In contrast, for EDA, evidence of interference comes only with SOAs of 50–100 ms, and activation is reliably lower at the 0-ms SOA. One possibility is that the EDRs reflect the subjective effort of various task combinations, whereas task 2 RTs reflect the timing of cognitive operations. Consistent with Damasio's (1994, 1999) assertion that elevated EDR serves as a somatic marker for the amount of executive attention demanded by a task, Bechara, Damasio, and Damasio (2000) found that EDR was related to judged task difficulty in a Stroop procedure, although in that case, both EDR and judged difficulty were related to RT. In this view, in the present procedure, either the simultaneous appearance of the two tasks (inducing simultaneous processing) or widely spaced appearances (inducing separate processing) may seem subjectively less demanding than a short offset. If this interpretation were correct, we reasoned that if we asked participants to judge the subjective difficulty of each trial, those judgments should show effects similar to those we had observed in EDA and different from those we had observed in task 2 RT. Corallo, Sackur, Dehaene, and Sigman (2008) and Marti, Sackur, Sigman, and Dehaene (2010) have

reported evidence consistent with this possibility. They asked individuals to estimate RTs in a PRP procedure. Estimated RTs, like actual RTs, were affected by both task 1 and task 2 difficulty; however, estimated RTs were unaffected by task overlap, whereas actual RTs were affected, consistent with the possibility that task overlap did not require executive operations and, so, did not result in conscious awareness. To the extent that perceived difficulty behaves as does estimated RT, we would expect to obtain similar results, contrary to what would be predicted if decreased SOA reflects increased subjective difficulty. To test this possibility, we essentially repeated [Experiment 3](#), but we added the additional step that, in [Experiment 4](#), the participants were asked to judge difficulty at the end of each trial, and we omitted the measurement of EDA.

Method

Thirty-six individuals drawn from the population previously used participated in this experiment. The experimental method was very similar to that in [Experiment 3](#), except that, this time, EDA was not measured. Instead, after either both responses had been given or the time had elapsed, the individual was prompted to rate the difficulty on a 10-point scale: *How difficult was this trial?* 9 = *among the very most difficult*; 1 = *among the very easiest*; 0 = *I made an error*. The other important change was that we administered the two tasks—the color judgment and the number judgment—in two different orders. In one block, the order of tasks was the same as in [Experiment 3](#), with the color task followed by the number task. In the other block, the task order was reversed: The number task was followed by the color task. The order of the blocks was counterbalanced across participants. In the reversed-order condition, the presentation of the number task first allowed us to test the overadditivity prediction that had been tested in [Experiment 2](#). Recall that the overadditivity prediction is that an increase in the difficulty of the task 1 central stage will increase the RT to task 2

at short SOAs, but not at long SOAs. Here, we manipulated task 1 central stage difficulty—whether the number was near to the target, 45, or far from the target—and examined the effect of the manipulation on the color task RT.

Results

Because the design is complex and to conserve space, we report only those specific analyses that address hypotheses equivalent to those of Experiments 1–3, and we report only measures of judged difficulty. The behavioral effects were established in Experiments 1, 2, and 3.

SOA The first test was of the overall effect of SOA, combined across task orders. For judged difficulty, there was a significant effect of SOA, $F(5, 175) = 8.09$, $p < .001$, $\eta_p^2 = .02$, as seen in Fig. 6. Judged difficulty was greater at the shortest SOAs than at the longest SOA.

Underadditivity prediction In Experiment 3, we tested the prediction that the effect of increasing the difficulty of precentral stages in task 2, such as perceptual identification, could be absorbed into the cognitive slack time in which task 2 was held in abeyance. We found a subadditive interaction in RT, such that reducing SOA had less effect in the more difficult condition than in the easier condition. We tested the underadditivity prediction in Experiment 4 in the situation with the number task second, comparing conditions in which the number was presented as digits (easy condition) with conditions in which the number was presented as words (difficult condition). For subjective difficulty, digits ($M = 3.24$, $SE = 0.05$) were judged easier than words ($M = 3.46$, $SE = 0.05$) although not significantly, $F(1, 35) = 3.38$, $p = .074$, $\eta_p^2 = .22$. The interaction of SOA and difficulty was not significant, $F(5, 175) = 0.50$.

Fig. 6 Effect of stimulus onset asynchrony (SOA) on mean judged difficulty (0 to 9 scale, with 9 being *most difficult*) in Experiment 4. Bars showing standard errors are not visible at this resolution

Additivity prediction Experiment 3 had also tested the prediction that the effects of increasing the difficulty of the central stage of task 2 would combine additively with the effects of SOA on task 2 RT. We tested this in Experiment 4 in the situation with the number task second, comparing conditions in which the number was far from 45 (easy condition) with those in which it was near 45 (hard condition). For judged difficulty, there was a significant effect of task 1 difficulty, $F(1, 35) = 14.24$, $p < .001$, $\eta_p^2 = .54$, with judged difficulty higher for near numbers ($M = 3.72$, $SE = 0.11$) than for far numbers ($M = 2.97$, $SE = 0.15$). The interaction of SOA and difficulty, shown in Fig. 7, was also significant, $F(5, 175) = 5.46$, $p < .001$, $\eta_p^2 = .31$. The simple main effect of SOA for near numbers was nonsignificant, whereas for far numbers, judged difficulty was higher at 50- and 100-ms SOAs than at 500- and 1,000-ms SOAs.

Overadditivity prediction Experiment 2 had tested the prediction that the effect of an increase in the difficulty of the task 1 central stage would result in an overadditive interaction with SOA on task 2 RT. We tested this again in Experiment 4, in the conditions with the number task first and the color task second, by comparing the case in which the number, now task 1, was far from 45 (the easy condition) with the case in which the number was near 45 (the hard condition). For judged difficulty, there was a significant effect of task 1 difficulty, $F(1, 35) = 27.68$, $p < .001$, $\eta_p^2 = .85$, with near numbers ($M = 4.34$, $SE = 0.08$) judged harder than far numbers ($M = 3.51$, $SE = 0.07$). The interaction of SOA and difficulty was not significant, $F(5, 175) = 0.62$.

Relations between judged difficulty and RT In addition to comparisons at the group level between effects on RT and effects on judged difficulty, we also explored the relationship at the level of individual trials. We treated each trial for each participant in each condition as a separate observation and regressed difficulty on task 1 RT + task 2 RT. The correlation

Fig. 7 Judged difficulty as a function of task 2 (number judgment task) central stage difficulty and stimulus onset asynchrony (SOA) in Experiment 4. Bars show standard errors

was significantly greater than zero, $R^2(7,829) = .238, p < .001$. This means that about 24 % of the variance in judged difficulty was accounted for by task 1 and task 2 RT. When SOA was added as an additional predictor, the change was minimal, $R^2 = .239$. The coefficient for SOA ($\beta = 0.03$) was small but significant, $t = 3.10, p = .002$. To place this finding in perspective, we returned to the earlier experiments and found the correlation between log area under the EDA change curve and task 1 RT + task 2 RT. In every case, they were close to zero: For Experiment 1, $r(28,627) = -.01$; for Experiment 2, $r(11,249) = .07$; and for Experiment 3, $r(8,638) = -.15$.

Discussion

The results of Experiment 4 were clear. Factors that influenced the size of the PRP effect in RT in Experiments 1–3 also affected the perceived difficulty of the task in Experiment 4, and those factors included SOA. The task was perceived to be more difficult when the two tasks overlapped than when they did not. The relationship was also seen in correlations at the level of individual participants, with conditions resulting in longer RTs also resulting in greater rated difficulty, with SOA making a small but significant contribution to overall difficulty. Difficulty judgments did not show the interactions of task difficulty manipulations with SOA found with RT. Judgments reflected awareness of manipulations of task difficulty but not how those manipulations interacted with SOA in RT. In this respect, the measure of judged difficulty converges with that of EDA. Judged difficulty, however, did not increase at short, nonzero SOAs, as was characteristic of EDA. It seems likely that EDA was not reflecting the subjective difficulty of the task.

General discussion

There is a central limitation called a *response selection bottleneck* that, in most cases, constrains the ability of people to simultaneously perform two overlapping tasks. This study examined how this central limitation functions, using electrodermal measures. Experiments 1–3 explored the effects on EDA of manipulations that had been shown to affect RT in dual-task procedures, varying the difficulty of both the first and second tasks, as well as the overlap between the tasks (the SOA). For task 2 RT, there was a very strong effect of SOA, with RTs increasing in lockstep by several hundreds of milliseconds with decreasing SOA. Manipulations of task 1 central stage difficulty and of task 2 perceptual stage difficulty interacted with SOA. By contrast, EDA showed a small response localized to SOAs of 50–100 ms, and that response was additive with the effects

of task difficulty. That is, manipulations that had interactive effects with SOA on RT (meaning that they affected the same processing stage) instead had additive effects with SOA on EDA (meaning that they affected different processing stages). Experiment 4 explored the effects of those manipulations on judgments of perceived difficulty. Judged difficulty was sensitive to the direct manipulations of task 1 or task 2 difficulty but was affected by SOA in ways distinctly different from either RT or EDA. The conclusion appears clear: There is a dissociation between the way RT or perceived task difficulty respond to increasing overlap between two tasks and the way EDA responds to task overlap. RT behaves as though there were a single, shared resource, presumably the all-or-none response selection bottleneck. EDA behaves as though manipulations that affect task difficulty drew on one pool of resources, whereas the effects of task overlap drew on a different resource pool.

Why was the EDR to SOA so weak? One possible explanation could be that the failure to detect a strong EDR to SOA is a result of the protocol that was used.³ If EDR habituates rapidly, we might not expect to see a strong response in experiments involving hundreds of trials. Nevertheless, the manipulations of task difficulty other than SOA did produce strong EDRs over hundreds of trials. Moreover, we did, in fact, see an effect of SOA, although it was small and was additive with other effects. In Experiment 2, the manipulation of task 1 difficulty was blocked, which should have produced even more rapid habituation to the manipulation of SOA, which was random within blocks; yet an effect was found. Another aspect of the protocol that might have affected the EDR is that the electrodes were attached to the left hand, which was also used for responding in task 1. Activity related to responding may have masked EDRs. Once again, if this artifact occurred, it did not mask EDRs to manipulations of task difficulty other than SOA. Those manipulations included some that should have affected task 1, in which a left-hand response (e.g., number of task 1 response alternatives in Experiment 2) was given, as well as others that should have affected task 2, in which a right-hand response was given (e.g., task 2 perceptual difficulty in Experiment 3).

A second possible explanation, that EDA is simply insensitive to task demands, can be rejected for the same reason as procedural or artifactual explanations. Not only have previous investigators found EDR to task-relevant aspects of the stimuli (see Dawson et al., 2007), but also we demonstrated substantial EDR to all other manipulations of the difficulty of either task, except for SOA in Experiments 2 and 3.

The third and most plausible explanation is the theoretically substantive one that managing dual-task overlap

³ We acknowledge an anonymous reviewer for suggesting this explanation and the next.

requires little or no executive attention, whereas factors that increase the difficulty of either of the two tasks do engage resource-demanding processes and, as a result, trigger autonomic responses. That is, as we framed the issue in the introduction, the response selection bottleneck is a passive, reactive, structural process, rather than an active, controlled, functional process involving executive attention, at least in relatively unpracticed individuals. SOA certainly does affect RT as though it were tapping a resource, so perhaps a more productive way to state this explanation for the results is that the management of dual-task overlap does involve resource-demanding operations, but it does not affect EDA as though it were tapping a resource.

If the operation of the bottleneck is passive and reactive, what implication does that have for neuroanatomical models of dual-task management? Zylberberg, Slezak, Roelfsma, Dehaene, and Sigman (2010) developed and tested a neurobiologically and neuroanatomically faithful model of serial processing in the brain that reproduces the major findings of the behavioral research on PRP and whose operation is consistent with our findings. The principal features of the model are sensory components that accumulate information about stimuli in both tasks, router networks that map sensory information onto responses, and task-setting networks that maintain the instructions for each set. Once sensory information consistent with one task is detected, that task set is activated, allowing the router to integrate sensory information for that task, leading to the emission of a response. Local lateral inhibition from the activated task set blocks activity in the other task set. The authors note that the same result could be obtained from lateral inhibition between the router networks for the two tasks. Once a response is activated, inhibitory feedback blocks the sensory, router, and response networks for the just-completed task, allowing the router for the other task to begin integration of sensory information from the second task. The information for task 2, which is said to be queued, is maintained for a period of time due to recurrent, subthreshold activation or resonance in the sensory networks. This recurrent activation is consistent with LaBerge's proposal that in thalamocortical columns, subthreshold activity is maintained by resonance in the apical dendrites (Kasevich & LaBerge, 2011; LaBerge, 2005, 2006; LaBerge & Kasevich, 2007).

In the model of Zylberberg et al. (2010), the operations of the router and task set networks would be the same for high-overlap trials as for low-overlap trials. Conventional approaches to fMRI might not detect any difference between the two types of trials (cf. Jiang et al., 2004; Marois et al., 2006). The critical difference is that the operation of the task 2 networks is time-shifted relative to the onset of the stimulus on high-overlap trials. Time-resolved fMRI using regressors reflecting the shift might be necessary, particularly to isolate areas involved with the task setting and router

components of the bottleneck (Dux et al., 2006; Sigman & Dehaene, 2008), because both the task set and router networks are active at both high and low overlap. This also argues for sampling more points in the overlap period than the coarse grain used in studies to date. The findings implicate a network involving the lateral prefrontal cortex and posterior parietal cortex in the inhibitory operation of the task set or router network. The queuing of task 2 sensory information may not be detectable if it is due to low-level resonance rather than spiking activity. Nevertheless, Sigman and Dehaene (2008) did find activations corresponding to the delay of task 2 in the bilateral medial visual cortex and intraparietal cortex, as well as in the motor cortex and SMA contralateral to the responding hand. In light of the operation of the Zylberberg et al. model, it is not surprising that EDA would be responsive to task difficulty but would not be responsive to SOA. Increases in task difficulty would be reflected in greater directed feed-forward activity in the networks, which may be interpreted as executive, attentional operations, whereas the bottleneck involves local feed-back inhibitory activity, activity that does not involve allocation of attention. In short, the present EDA results converge with the Zylberberg et al. model in implicating a passive, reactive bottleneck mechanism.

EDA shows no effect in the situation in which the interference reflected by RT is at a maximum—that is, when the tasks arrive simultaneously. After that, it shows a small effect, and only for short SOAs around 50–100 ms. To what is EDA responding? One interpretation is that it reflects low-level, early responses to the onset of closely spaced stimuli. VanRullen and Thorpe (2001) have shown that task-related information has begun to be extracted about 75 ms after stimulus presentation, well before the information begins to be correlated with the observer's behavior at about 150 ms poststimulus. This is precisely the region in which we found elevated EDA. Quite plausibly, then, the EDR reflects the point at which processing of task 1 is sufficiently far along for stimulus information from task 2 to be detected as needing additional, separate processing. When the onsets of stimuli for the two tasks are simultaneous, this may be registered as a single onset, and the filtering of the information into two stimulus sets proceeds from the outset. When the onset of the second task comes sufficiently late that higher level cognitive processing of the first task is already underway or even completed, the onset of the second task causes no special response. It is only when new stimulus information arrives just as processing of the first stimulus is reaching cognitive stages that an EDR is triggered. Certainly, this argument is speculative. What is not speculative is that EDA is not reflecting the same aspects of task overlap as are RTs.

Finally, we return to the question that motivated the research: How does the response selection bottleneck function?

Recall that Meyer and Kieras (1997a, 1997b) conceptualized the response selection bottleneck as the result of the strategic allocation of executive control. In this view, it is an optional, strategic decision to lock out central processing of task 2 until central processing of task 1 is complete. As we noted in the introduction, there have, in fact, been demonstrations of apparently bottleneck-free dual-task processing. From the perspective of our experiments, a strategic, optional, and ad hoc process that required extraordinary executive attention to suppress and that did not strongly affect EDA markers of task-relevant activity seems implausible. Such a demanding process would very likely generate an autonomic nervous system response that would be reflected in EDA. We suggest that it is more plausible that the psychological refractory period in dual-task processing is the result of the operation of a passive, structural mechanism that does not engage executive attention, consistent with the speculations of others on the basis of behavioral, neural network, and neuroimaging results.

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