A psychological refractory period in access to visual short-term memory and the deployment of visual–spatial attention: Multitasking processing deficits revealed by event-related potentials

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Abstract
In this psychological refractory period (PRP) experiment, a tone (T1) was presented, followed by a visual target (T2) embedded in a bilateral display, and a speeded response was required for each target. The T1–T2 stimulus onset asynchrony (SOA) was 300, 650, or 1000 ms. Mean response time to T2 increased as SOA was reduced, replicating the well-known PRP effect. Importantly, the N2pc component of the event-related potential was progressively attenuated as SOA was reduced, and the onset latency of the sustained posterior contralateral negativity (SPCN) that follows the N2pc was progressively lengthened. Conditional analysis based on Task1 difficulty corroborated the analyses based on effects of SOA. The results suggest that central processing leading to the PRP effect interferes with the deployment of visual–spatial attention (as indexed by the N2pc) and delays encoding into visual short-term memory (as indexed by the SPCN onset latency).

Descriptors: Cross-modal PRP paradigm, Dual-task interference, Central attention, Visual–spatial attention, Visual short-term memory, N2pc, SPCN

Attentional limitations in multiple task situations have been studied extensively using the psychological refractory period (PRP) paradigm. In the PRP paradigm, two distinct targets, T1 and T2, are presented sequentially, and a speeded response is required for each target. The processing overlap between Task1 and Task2 usually is manipulated by varying the temporal interval between the onset of the two targets (i.e., the T1–T2 stimulus onset asynchrony, or SOA). Even with very simple stimuli and associated tasks, the PRP paradigm yields robust interference effects, reflected mostly by an increase in mean response time to the second target (RT2) as SOA is reduced (i.e., as task overlap is increased).

Several researchers have proposed that the lengthening of RT2 (i.e., the PRP effect) is caused by a structural “bottleneck” at the stage of response selection and decision making (e.g., McCann & Johnston, 1992; Pashler, 1994; Pashler & Johnston, 1989; Welford, 1952). Specifically, the central bottleneck model postulates that, under appropriate conditions (e.g., when sensory modalities are not overloaded and when responses do not require the same output), sensory-perceptual processes and response execution processes can operate in parallel, without significant interference, for multiple targets. However, central processes such as response selection and decision making can only operate sequentially, on one target at a time. Therefore, under high task overlap conditions (e.g., at short SOAs), response selection to T2 is postponed until central mechanisms have finished selecting the response to T1. This postponement leads to a longer waiting period as SOA is shortened, which would explain the lengthening of RT2.

Some aspects of this model have been criticized. For example, some have argued that the central bottleneck is strategic in nature and not structural (e.g., Meyer & Kieras, 1997). Others have demonstrated that central capacity sharing models predict all the hallmark effects of the PRP paradigm and therefore are viable alternatives to the central all-or-none bottleneck model (e.g., Navon & Miller, 2002; Tombu & Jolicœur, 2003). However, the claim that interference in the PRP paradigm occurs at a relatively late, central locus of processing is accepted by virtually all models of dual-task interference, although recent electrophysiological evidence suggests that it may start to occur prior to response selection. Indeed, SOA effects on P3 latency have been observed (Arnell, Helion, Hurdelbrink, & Pasieka, 2004; Dell’Acqua, Jolicœur, Vespignani, & Toffanin, 2005; Luck, 1998), and this effect has been positively correlated with the behavioral PRP effect.
Another attentional phenomenon that has been studied extensively (often referred to as visual–spatial attention) involves our ability to deploy attention to specific locations (and/or items) in the visual field without moving our eyes. Spatial cuing studies have demonstrated that performance is improved to stimuli that appear at an attended location (e.g., Posner, 1980). It is also postulated that visual–spatial attention must be deployed on individual items in a search array in order to identify a predefined target among multiple distractors, at least when the distractors and target share similar features (Duncan & Humphreys, 1989; Treisman & Gelade, 1980; Woodman & Luck, 2003b).

It has been argued that the central attentional mechanisms that underlie the PRP effect are distinct, and therefore independent, of the mechanisms involved in deploying visual–spatial attention. In an elegant chronometric study using identical critical stimuli, task, and method (i.e., the locus-of-slack logic: Jolicœur, Dell’Acqua, & Crebolder, 2001; McCann & Johnston, 1992; Pashler & Johnston, 1989; Schweickert, 1980), Johnston, McCann, and Remington (1995) demonstrated an attention restriction before the stage of letter identification in a spatial cuing paradigm, but after letter identification in the PRP paradigm. The critical stimuli were either the letter A or the letter H, and the duration of the letter identification stage was manipulated by presenting the letters either normally or distorted. The critical task was a two-alternative speeded discrimination as to the identity of T2, which was masked, there was a critical time period for visual–spatial attention to be deployed to T2 before the mask terminated sensory-perceptual processing of the items in the visual display. Following this logic, reducing SOA should result in poorer report of T2 if both types of attention share at least some mechanisms (because of the increased postponement of the deployment of visual–spatial attention as SOA was reduced), whereas no SOA effect on report accuracy for T2 should be observed if the two types of attention are distinct. Results showed a significant reduction in T2 accuracy between the shortest (50 ms) and longest (650 ms) SOA when color was used as the selection index (Experiment 7: 4.7%; p < .005) and when attention had to be deployed on the opposite side of a peripheral onset cue (Experiment 6: 5.1%; p < .001). However, because these SOA effects on T2 accuracy were much smaller than a 30% effect found when the peripheral onset cue itself was delayed (Experiment 2), Pashler concluded that the observed SOA effect on T2 accuracy could not be caused by a lengthy period of central postponement of the deployment of visual–spatial attention. Pashler explicitly acknowledged, however, that he could not, with the behavioral methods used in his study, offer an alternative explanation of the observed significant SOA effects on accuracy in Task2.

Brisson and Jolicœur (in press) recorded event-related potentials (ERPs) in addition to behavioral measures to shed new light on this important issue. The ERP component of interest in their study is called N2pc (N2 posterior contralateral; Eimer, 1996; Luck, Girelli, McDermott, & Ford, 1997; Luck & Hillyard, 1994; Woodman & Luck, 2003b). The N2pc is a lateralized ERP component that is maximal at posterior electrode sites contralateral to an attended item and is isolated by subtracting activity at ipsilateral electrode sites from the corresponding activity at contralateral electrode sites (e.g., PO7/PO8). Although the N2pc onset latency could vary with the difficulty of target localization (Washer, 2005), it typically starts about 180 ms after target onset and lasts about 100 ms. Luck and colleagues, who were the first to study this component meticulously in visual search tasks, suggested that the N2pc reflected distractor suppression processes (Luck & Hillyard, 1994; Luck et al., 1997). Others, who have used bilateral displays with only one distractor, have argued that the N2pc reflected target enhancement processes (e.g., Eimer, 1996). Nonetheless, even if there is still an ongoing debate on the specific processes that underlie the N2pc, it is widely accepted that it is a valid index of covert visual–spatial attention in light of several results reviewed by Woodman and Luck (2003b).

Brisson and Jolicœur (in press) measured the N2pc elicited by a lateralized visual target (defined by color) under different concurrent central load conditions, manipulated using a modified PRP paradigm similar to that used by Pashler (1991). A smaller N2pc was observed in high concurrent central load conditions both when central load was manipulated by varying the SOA (100 ms or 1500 ms; Experiment 1) and in a fixed 100-ms SOA PRP paradigm in which Task1 difficulty was manipulated (four-alternative discrimination vs. simple reaction time; Experiment 2).
The attenuation of the N2pc in this previous study provided strong evidence that concurrent central processing does, in fact, interfere with some aspect of the deployment of visual–spatial attention. However, because T2 appeared more than 800 ms after mean RT1 in the long SOA of Experiment 1, it is possible that N2pc modulations resulted from differential Task2 preparation after trial initiation. Furthermore, differential pretrial preparatory states could have accounted for N2pc modulations in Experiment 2, where attentional load conditions varied across blocks. According to this task preparation hypothesis, participants were more prepared for Task1 and less prepared for Task2 in the high load condition than in the low load condition. As a consequence, visual–spatial attention would have been deployed on a distractor item opposite to the target, or not deployed at all, on a portion of trials, leading to an attenuation of the N2pc. Although this argument does not contradict the claim that concurrent central processing interfered with the control of visual–spatial attention, because optimal preparation for Task2 could not be maintained concurrently with processing required for Task1, it does imply a different kind of interference than that bottleneck or capacity sharing that is postulated to be responsible for the behavioral PRP effect (Pashler, 1994; Tombu & Jolicœur, 2003).

The primary goal of the present study was to determine whether the N2pc attenuation observed in Brisson and Jolicœur (in press) arose because of central postponement (or central capacity sharing) as opposed to task preparation. To minimize the possibility of differential task preparation, three randomly presented SOAs, separated by only 350 ms, were chosen (i.e., SOAs of 300, 650, or 1000 ms). Because SOA conditions were randomly presented, it was impossible for subjects to know which condition would be presented, and therefore it was impossible for them to prepare differentially for each condition before trial initiation. Also, the posttrial task preparation hypothesis holds only if there is enough time between response to T1 and onset of T2 to increase Task2 preparation in a long SOA condition compared to a shorter SOA condition. By choosing a difficult four-alternative discrimination Task1 that should produce long RT2s (on the order of 700 ms; see Brisson & Jolicœur, in press) and by separating SOAs by only 350 ms, we considerably reduce this possibility. Furthermore, because the two shortest SOAs were chosen so that T2 would be presented usually before response to T1, it is highly unlikely that an attenuation of the N2pc between these two SOAs would be due to differential task preparation. Therefore, a progressive attenuation of the N2pc as SOA is reduced in the present study would provide compelling evidence that central postponement (or capacity sharing) interferes with the deployment of visual–spatial attention in absence of differential task preparation.

In addition, dual-task interference associated with task overlap was also manipulated within SOA conditions by mapping four tone (T1) frequencies arrayed from low to high to four response keys arrayed from left to right. It has been demonstrated that in these situations, it is harder to respond to the middle frequency tones than to the highest and lowest frequency tones. This difficulty effect, reflected by longer mean response times and lower accuracy to the middle frequencies than to the highest and lowest frequencies, was associated with a stage of processing that is likely in the central PRP bottleneck (see Jolicœur, 1999b; Van Selst & Johnston, 1996). Taking advantage of this built-in Task1 difficulty manipulation, the trials in which the tones of the middle frequencies were presented were included in the hard-Task1 condition, whereas the trials in which the tones of the highest and lowest frequencies were presented were included in the easy-Task1 condition. An attenuation of the N2pc in the hard-Task1 condition compared to the easy-Task1 condition would provide further evidence against the task preparation hypothesis, because the sequence of events (i.e., SOA) in both Task1 difficulty conditions was identical, rendering differences in task preparation based on perceived task intervals impossible.

A second important goal of this study was to discover if the sustained posterior contralateral negativity (SPCN) that follows the N2pc is affected by dual-task interference associated with task overlap. As is the case for the N2pc, the SPCN is thought to index visual activity, because it arises at electrode sites contralateral to the to-be-memorized visual items, which links the activity to the location of the task-relevant items in the visual field and has a posterior scalp distribution, which is consistent with activity in the extrastriate visual cortex (McCollough, Machizawa, & Vogel, 2007). Specifically, the SPCN is thought to reflect visual short-term memory (VSTM) activity (Dell’Acqua, Sessa, Jolicœur, & Robitaille, 2006; Jolicœur, Sessa, Dell’Acqua, & Robitaille, 2006a, 2006b; Klaver, Talsma, Wijers, Heinz, & Mulder, 1999; McCollough et al., 2007; Robitaille & Jolicœur, 2006; Vogel & Machizawa, 2004). Indeed, it has been shown that the amplitude of the SPCN increases as the number of to-be-remembered items in the visual display increases, but only up to the participants’ VSTM capacity, and that it is a sustained response throughout the retention period (McCollough et al., 2007; Vogel & Machizawa, 2004). Furthermore, it has been found that the SPCN duration was correlated with reaction time (RT) in tasks that required a speeded response (Robitaille & Jolicœur, 2006). It was argued that the conditions that produced the longer RT most likely required the participants to maintain the visual trace in VSTM for a longer period, and therefore that the time course of the SPCN tracks the duration the visual trace must be held in VSTM (Prime, Chénier, & Jolicœur, 2006).

Because the SPCN reflects neural activity specifically related to the maintenance of information in VSTM, it is possible, by measuring the onset latency of the SPCN, to evaluate whether central attentional mechanisms underlying the PRP effect interfere with transfer into VSTM. If this is the case, it would provide the first demonstration that early visual memory processes are delayed by an overlapping speeded auditory task.

Methods

Participants
Twenty-four undergraduate students from the Université de Montréal participated in this experiment for financial compensation. Eight participants had to be excluded because less than 50% of trials in at least one SOA condition remained after artifact rejection (see below). Thus 16 participants (9 women), aged 20–27 years (mean age of 21.4 years) remained in the sample. All participants were neurologically intact and reported having normal hearing and normal or corrected-to-normal visual acuity and color vision.

Stimuli
Participants sat in a dimly lit, electrically shielded room, facing a computer screen, at a viewing distance of 57 cm. On each trial, a 100-ms tone (T1), emitted simultaneously by two loudspeakers...
that were placed on each side of the computer screen, was followed by a 133-ms visual display that contained the second target (T2; see Figure 1). The T1–T2 SOA was 300, 650, or 1000 ms. T1 could be at one of four frequencies (randomly presented from trial to trial: 200 Hz [68 dB], 430 Hz [60 dB], 926 Hz [60 dB], or 2000 Hz [56 dB]). The visual display contained four colored squares (two on each side of fixation), each with a gap in one side (different for each square). T2 was a red square (x = .382, y = .275; CIE [x, y] chromaticity coordinates; Wyszecki & Stiles, 1982) among green distractors (x = .277, y = .506) for half of the participants and a green square among red distractors for the other half. Both colors were equiluminant (26.3 cd/m²) to equate low sensory responses and were presented on a dark-gray background (0.25 cd/m²). All squares subtended a visual angle of 1° × 1° and the gaps were 0.33°. The center of the squares nearest to fixation was 1.5° below and 3.5° to the left or right of fixation. The center of the far squares was 3° below and 5° to the left or right of fixation. T2 appeared randomly in each of the four possible positions.

Procedure

After hearing the four tones presented from low to high frequency five times, participants performed one practice block of 64 trials (16 single-Task1 trials, 16 single-Task2 trials, and 32 dual-task trials) followed by 12 experimental blocks of 64 trials.

Each trial was initiated by pressing the “N” and “V” keys simultaneously with the right and left index fingers, respectively. Feedback from the preceding trial disappeared and a fixation point simultaneously appeared at the center of the computer screen, which was visible throughout the remainder of the trial. Five hundred milliseconds later, a tone (T1) was presented (all tone frequencies were randomly presented equally often in each block), followed at an SOA of 300, 650, or 1000 ms, by a visual display that contained T2 (all SOAs were randomly presented equally often in each block).

Two separate four-choice speeded responses were required on each trial. The first response was to the pitch of T1 and the second response was to the location of the gap in T2. Responses to T1 were made with fingers of the right hand (adjacent response keys arrayed from left to right, “N,” “M,” “,”, and “.” for the 200, 430, 926, and 2000 Hz tones, respectively) and responses to T2 were made with the fingers of the left hand (response keys were “Z,” “X,” “C,” and “V” for left, bottom, up, and right gaps, respectively). Instructions emphasized the importance of responding as quickly and accurately as possible to T1 as soon as T1 was presented and of responding as quickly and accurately as possible to T2 as soon as T2 was presented.

Trials ended with the simultaneous disappearance of the fixation point and appearance of the visual feedback, 1250 to 1750 ms after the response to T2. Immediately to the left of the center of the screen, a “+” or “−” indicated a correct or incorrect response to T1, respectively. Immediately to the right of the fixation point a “+” or “−” indicated a correct or incorrect response to T2. Participants were instructed to maintain central eye fixation throughout the trial and blink only when the feedback was on the screen.

**EEG Recording and Analysis**

The EEG was recorded from 64 active Ag/AgCl electrodes (Biosemi Active Two system) mounted on an elastic cap and referenced to the average of the left and right mastoids. Electrodes were placed according to the extended International 10/20 system at Fp1, Fpz, Fp2, AF7, AF3, AFz, AF4, AF8, F7, F5, F3, F1, Fz, F2, F4, F6, F8, FT7, FC5, FC3, FC1, FCz, FC2, FC4, FC6, FT8, T7, C5, C3, C1, Cz, C2, C4, C6, T8, TP7, CP5, CP3, CP1, CPz, CP2, CP4, CP6, TP8, P9, P7, P5, P3, P1, Pz, P2, P4, P6, P8, P10, PO7, PO3, POz, PO4, PO8, O1, Oz, O2, and Iz sites. The horizontal electrooculogram (HEOG), recorded as the voltage difference between electrodes placed lateral to the external canthi, was used to measure horizontal eye movements. The vertical electrooculogram (VEOG), recorded as the voltage difference between two electrodes placed above and below the left eye, was used to detect eyeblinks. A bandpass filter of 0.01–40 Hz was applied and the EEG and EOG signals, digitized at 256 Hz, were averaged off-line.

Trials with eyeblinks (VEOG >80 μV), large horizontal eye movements (HEOG >30 μV), and/or artifacts at electrode sites of interest (i.e., >80 μV at O1, O2, PO7, PO8, P7, and/or P8 electrode sites) were rejected. Eight participants were excluded because more than 50% of trials were rejected in at least one experimental condition. Of the remaining 16 participants, an average of 84% of 300-ms SOA trials, 82% of 650-ms SOA trials, and 80% of 1000-ms SOA trials remained after artifact rejection. None of these participants had residual eye movements that deviated more than 0.2° (i.e., HEOG >3.2 μV) toward T2 after rejection criteria were applied1 (see Luck, 2005).

The EEG was averaged starting 200 ms prior to T2 onset and ending 600 ms after T2 onset and baseline corrected based on the 200-ms pretarget period. The ipsilateral waveform (average of left-sided electrode with left visual field target and right-sided electrode with right visual field target) and contralateral waveform (average of left-sided electrode with right visual field target and right-sided electrode with left visual field target) time-locked to T2 for all SOA conditions at O1/O2, PO7/PO8, and P7/P8 electrode sites were computed separately. To isolate the N2pc and the SPCN from overlapping activity that was not lateralized with respect to the side of T2 (i.e., Task1 stimulus, preparation, and response activity, as well as Task2 preparation and response activity), the N2pc and SPCN were quantified following the subtraction of the ipsilateral waveforms from the contralateral

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1The HEOG criteria was lowered to 25 μV for three participants and to 15 μV for one more participant so that the residual HEOG would be less than 3.2 μV.
waveforms. Separate waveforms were computed also for the easy-Task1 (lowest and highest tone frequencies) and hard-
Task1 (middle tone frequencies) conditions. To maintain an
adequate number of trials per waveform, all SOAs were collapsed
to compute the waveforms for the easy- and hard-Task1
conditions.

N2pc measurements (mean amplitude during the 180–260-ms
post-visual-display time window) and SPCN measurements
(mean amplitude during the 340–420-ms and 500–600-ms post-
visual-display time window) were obtained from the subtraction
waveforms. SPCN onset latency measurements were also calcu-
lated using a jackknife method (Miller, Patterson, & Ulrich,
1998; Ulrich & Miller, 2001). With the jackknife method, n grand
average waveforms are computed with n − 1 participants (a
different participant is removed for each waveform). Latency mea-
sures are obtained for each of these n grand average waveforms,
and the values are submitted to a conventional analysis of
variance (ANOVA), but for which the F values must be adjusted
according to

\[ F_{\text{adjusted}} = F / (n-1)^2 \]

(see Ulrich & Miller, 2001, for a general proof of this adjust-
ment).

Behavioral data (mean percent accurate responses and RT for
both Task1 and Task2) and electrophysiological measures were
both submitted to two separate repeated measures ANOVAs:
one in which SOA condition (300 ms, 650 ms, or 1000 ms) was
treated as a within-subject factor and another in which Task1
difficulty condition was treated as a within-subject factor. Electro-
pose position (O1/O2, P7/P8, or P7/P8) was included as an
additional within-subject factor in the analysis performed on the
electrophysiological data.

Results

Behavioral results

Only trials with correct responses to both T1 and T2 were included
in the RT analyses, and outliers were excluded using the
method described in Van Selst and Jolicœur (1994). RT and ac-
curacy for each SOA and each Task1 difficulty condition is pre-
tented in Table 1. Mean Task1 accuracy increased as SOA
increased, \( F(2,30) = 4.38, p < .022 \). Mean RT1 also increased
with increasing SOA, \( F(2,30) = 6.99, p < .004 \). This (slight)
speed–accuracy trade-off pattern was most likely caused by T2
onset precipitating T1 response before processing of the tone was
complete in a portion of short SOA trials.\(^2\) As expected, mean
Task1 accuracy was lower in the hard-Task1 condition than in the
easy-Task1 condition, \( F(1,15) = 109.2, p < .001 \), and mean RT1
was respectively longer, \( F(1,15) = 118.7, p < .001 \).

As is typically observed in PRP studies where T2 is not
masked, there were no SOA effect on Task2 accuracy, \( F < 1 \),
although there was a small but reliable Task1 difficulty effect,

\(^2\) A subset of participants also appeared to have grouped their
responses. That is, they seemed to have waited to select responses to T2
before emitting a response to T1 on a portion of trials. However, it is
known that grouping does not influence Task2 performance (see Pashler
& Johnston, 1989), which is of more direct concern for the present study.
Therefore, these participants were not excluded from further analyses.
Response grouping, however, increases mean RT1 as SOA is increased
because these subjects waited for the presentation of T2 before producing
the response to T1, and T2 is presented at increasingly long delays, relative
to T1 as SOA is increased.

Table 1. Mean Accuracy (percent correct) and RT (ms) to T1 and
Task2 for Each SOA (ms) Condition and Each Task1 Condition

<table>
<thead>
<tr>
<th>Experimental Conditions</th>
<th>RT1</th>
<th>ACC1</th>
<th>RT2</th>
<th>ACC2</th>
</tr>
</thead>
<tbody>
<tr>
<td>300 ms SOA</td>
<td>663  (45)</td>
<td>82.7 (2.2)</td>
<td>898 (53)</td>
<td>92.4 (2.3)</td>
</tr>
<tr>
<td>650 ms SOA</td>
<td>692  (51)</td>
<td>84.8 (2.0)</td>
<td>697 (38)</td>
<td>92.7 (2.3)</td>
</tr>
<tr>
<td>1000 ms SOA</td>
<td>754  (72)</td>
<td>85.5 (1.9)</td>
<td>658 (31)</td>
<td>92.2 (2.3)</td>
</tr>
<tr>
<td>Hard-Task1</td>
<td>807  (58)</td>
<td>74.3 (2.6)</td>
<td>817 (51)</td>
<td>91.3 (2.3)</td>
</tr>
<tr>
<td>Easy-Task1</td>
<td>633  (56)</td>
<td>94.3 (1.6)</td>
<td>706 (36)</td>
<td>93.5 (2.3)</td>
</tr>
</tbody>
</table>

RT1 = reaction time in Task1; RT2 = reaction time in Task2;
ACC1 = accuracy for Task1; ACC2 = accuracy for Task2.

Standard error of the mean in parentheses.

Electrophysiological Results

N2pc. Ipsilateral and contralateral waveforms are shown in
Figure 2 as a function of electrode positions and SOA, and the
corresponding contralateral minus ipsilateral subtraction wave-
forms are presented in Figure 3. The scalp distribution of the
electric potentials for the N2pc difference wave is presented in the
left panel of Figure 4. The N2pc and SPCN scalp distributions
were computed with the collapsed data of the two longest SOA
conditions, where the components were largest. They are sym-
metrical about the midline because they were calculated on the
basis of the contralateral minus ipsilateral difference waves used
to calculate the N2pc (and SPCN). This was done specifically to
avoid systematic left–right hemispheric asymmetries due, among
other things, to the fixed manual responses associated to Task1
and Task2. The scalp distribution of the N2pc is similar to pre-
viously published N2pc distributions (see Hopf et al., 2000;
Robitaille & Jolicœur, 2006).

The analyses performed on the N2pc, which is the first large
negative deflection in the subtraction waveforms, revealed a
progressive attenuation of N2pc mean amplitude as SOA was
reduced, reflected by a main effect of SOA, \( F(2,30) = 6.43, p < .015 \).\(^3\)
No main effect of electrode position, \( F(2,30) = 2.62, p > .10 \), or Electrode Position × SOA interaction, \( F(4,60) = 2.23, p > .10 \), were found. Furthermore, when the data from the
longest SOA were removed in a separate analysis, the effect of
SOA on N2pc mean amplitude was still significant between the
two shortest SOAs, \( F(1,15) = 19.3, p < .001 \).

Contralateral minus ipsilateral subtraction waveforms as a
function of electrode position and Task1 difficulty are presented
in Figure 5. N2pc mean amplitude was reduced in the hard-Task1
condition relative to the easy-Task1 condition, \( F(1,15) = 18.3, p < .001 \). No main effect of electrode position, \( F(2,30) = 2.55, p > .10 \), was observed, although an Electrode Position × Task1

\(^3\) For the electrophysiological analysis, a Greenhouse–Geisser correc-
tion was used for the estimation of F statistics associated with more than
one degree of freedom in the numerator.
Difficulty interaction, $F(2,30) = 5.50, p < .01$, revealed a more reliable Task difficulty effect at P7/P8 and PO7/PO8 electrode sites than at O1/O2 electrode sites.

SPCN. The SPCN is the second large negative deflection in the subtraction waveforms (see Figure 3). The scalp distribution of the electric potentials for the SPCN difference wave is presented in the right panel of Figure 4. The posterior distribution of the SPCN indicates activity in the visual cortex. Whereas the SOA effect on the N2pc was mainly reflected by an attenuation of the component, the SOA effect on the SPCN seems to be mainly reflected by an increase of the onset latency of the component as SOA was reduced (see Figure 3). To assess if the increase in the SPCN latency was significant, we proceeded in two steps. First, we calculated the mean amplitude in the 340–420-ms post-visual-display time window. The analyses revealed a main effect of SOA in this time window, $F(2,30) = 6.85, p < .005$, which remained when the longest SOA was removed, $F(1,15) = 7.68, p < .014$. As for the N2pc analyses, no main effect of electrode position, $F(2,30) = 1.26, p > .29$, or Electrode Position × SOA interaction, $F(4,60) = 1.20, p > .31$, were observed. Then, to provide further evidence that the observed amplitude reduction in this time window was caused by an increase in the SPCN latency, an additional 10-Hz low-pass filter was applied to the subtracted waveforms.
waveforms, and the time at which the pooled subtracted waveform reached 0.4 µV, starting at 300 ms after the visual display, was measured using the jackknife method (Miller et al., 1998; Ulrich & Miller, 2001). This analysis revealed a main effect of SOA on SPCN latency, $F(2,30) = 8.53, p < .001$. The main effect of SOA was marginally significant in a separate analysis in which the longest SOA was removed, $F(1,15) = 4.18, p = .06$.

The Task$_1$ difficulty effect on the SPCN mean amplitude in the 340–420-ms post-visual-display time window was also significant, $F(1,15) = 7.99, p < .013$. No main effect of electrode position, $F(2,30) = 1.21, p > .31$, or Electrode Position $\times$ Task$_1$ Difficulty interaction, $F(4,60) = 1.20, p > .31$, were observed. Furthermore, as was the case with the SOA analysis, the jackknife method revealed a main effect of Task$_1$ difficulty on SPCN latency, $F(1,15) = 4.65, p < .05$.

When analysing the SPCN mean amplitude in the later 500–600-ms post-visual-display time window, no main effect of experimental condition, $F < 1$ for both SOA and Task$_1$ difficulty, or any other effect was observed, which suggests that a stable VSTM representation was eventually achieved in all conditions (corroborated by the high T$_2$ report accuracy in all conditions).

**Discussion**

Two important results obtained in this study strongly suggest that central processes underlying dual-task interference in the cross-modal PRP paradigm can interfere with early sensory-specific processes. First, the N2pc was progressively attenuated as task overlap increased (i.e., as SOA decreased and as Task$_1$ difficulty within SOAs increased) between a demanding speeded auditory task and a speeded visual task that required the deployment of visual–spatial attention. Second, the onset latency of the SPCN, reflecting encoding into VSTM, following the N2pc, was progressively delayed as SOA was shortened and as Task$_1$ difficulty increased.

We assume that the N2pc reflects the successful deployment of spatial attention to the lateralized visual target (Brisson & Jolicœur, in press; Dell’Acqua et al., 2006; Eimer, 1996; Jolicœur et al., 2006a, 2006b; Luck & Hillyard, 1994; Woodman & Luck, 2003b). The attenuation of N2pc caused by concurrent central processing suggests that the deployment of visual–spatial attention, or the control of this process, suffered significant central interference. An attenuation of the N2pc when subjects attempted to deploy visual–spatial attention while they also...
performed a capacity demanding speeded auditory choice task was first observed by Brisson and Jolicœur (in press). It has been demonstrated in this previous study that the N2pc attenuation could not be caused by a PRP-induced failure of color perception. Furthermore, they argued that their results are unlikely to reflect cross-modal spatial capture (see McDonald & Ward, 2000) associated with the location of the source of the sound, because the tones, presented with a pair of speakers behind the monitor, did not appear to come from a well-localized point in space, but rather filled a large volume in the room, as in the present experiment. They also pointed out that any existing spatial capture would have been equivalent in their Experiment 2, where an N2pc modulation was observed in an experiment with a fixed 100-ms SOA. Experiment 2 also confirmed that the N2pc attenuation could not be due to ERP overlapping activity obscuring the N2pc. Indeed, sensory activity overlap was identical between the easy simple reaction time and hard four-alternative discrimination Task1 conditions. Moreover, greater Task1 motor overlap in the N2pc time range would have been expected in the simple reaction time Task1 condition, where mean RT1 was shorter than in the four-alternative discrimination Task1 condition. If overlapping activity obscured the N2pc, this should have led to opposite results than those observed. Finally, in Experiment 1, where central load conditions (SOA of 100 ms vs. 1500 ms) were randomly presented, the differential attenuation of the N2pc was obtained in absence of any possible differential pretrial preparatory state. Therefore, the N2pc modulation had to be due to the different concurrent central processing demands in Task1. However, because T2 appeared more than 800 ms after mean RT1 in the long SOA condition of Experiment 1, it is possible that N2pc modulations in this earlier work resulted from differential Task2 preparation after trial initiation, which implies a different kind of interference than the bottleneck or capacity sharing that is postulated to be responsible for the behavioral PRP effect.

The progressive attenuation of the N2pc as SOA decreased in this study, however, provides compelling evidence that differential Task2 preparation is not the underlying cause of the observed N2pc modulation. Indeed, a differential pretrial preparatory state was impossible because SOA conditions were randomly intermixed in each block of trials. Furthermore, the three SOAs were chosen so that the interval between the response to T1 and T2 onset would be too short to allow a dynamic shift in task preparation from Task1 to Task2. T2 onset occurred before mean RT1 in the two shortest SOA conditions and only 246 ms after mean RT1 in the longest, 1000 ms, SOA condition. It would be very improbable that participants would be able to modify their processing strategy while they were still executing the first task. Moreover, mean RT2 was about 200 ms longer in the 300-ms SOA condition than in the 650-ms condition, which means that at least 200 ms of the 350-ms difference in SOA between the two shortest SOA conditions was likely solely occupied in selecting the response to T1, which would leave only a 150-ms difference between the two shortest SOAs to modify processing strategies. Furthermore, Task1 performance also argues against differential preparation across SOAs. Indeed, differences in Task1 performance across SOAs seem to indicate the presence of a slight speed–accuracy trade-off most probably caused by T2 onset precipitating T1 response rather than differences in task preparation. Finally, the N2pc was also attenuated in a hard-Task1 condition relative to an easy-Task1 condition. Here, dual-task interference associated with task overlap was manipulated by varying Task1 difficulty at each SOA, thereby making it impossible for subjects to adapt their task preparation depending on perceived T1–T2 SOA. Moreover, the combination of N2pc attenuation both with decreasing SOA and within SOAs (as a function of Task1 dif-

**Figure 4.** Scalp distribution of the electrical potentials measured during the N2pc (180–260 ms) and SPCN (500–600 ms) post-T2-onset time windows. The scalp distributions were computed with the collapsed data of the two longest SOA conditions, where the components were largest, and were calculated on the basis of the contralateral minus ipsilateral difference waves used to calculate the N2pc and SPCN and are thus symmetrical about the midline.
difficulty) show convincingly that ERP component overlap cannot be the cause of the N2pc attenuation. This is because decreasing SOA must increase overlap, whereas increasing Task1 difficulty at a given SOA must decrease overlap. Yet, both these manipulations have the same effect on N2pc amplitude, which provides new empirical evidence to support the theoretical assumption that N2pc amplitude reductions is not caused by component overlap. In combination, the evidence indicates that the all-or-none or capacity sharing bottleneck that is postulated to be responsible for the behavioral PRP effect is also responsible for the progressive N2pc attenuation as SOA was shortened (which was also significant between the two shortest SOAs) and as Task1 difficulty increased.

Although the N2pc mean amplitude was progressively attenuated as task overlap increased, there was no such effect on the ultimate amplitude of the SPCN. Rather, the onset latency of the SPCN was progressively lengthened as task overlap increased (i.e., as SOA was decreased and as Task1 difficulty increased within SOAs). The different patterns of N2pc and SPCN modulations provide further evidence that the N2pc and SPCN components index different processes with different functions. Whereas the N2pc reflects visual–spatial attention processes, we assume that the SPCN reflects activity specifically related to retention in VSTM (Dell’Acqua et al., 2006; Jolicœur et al., 2006a, 2006b; Klaver et al., 1999; McCollough et al., 2007; Robitaille & Jolicœur, 2006; Vogel & Machizawa, 2004). The progressive lengthening of the SPCN onset latency, therefore, suggests that transfer into VSTM was delayed by concurrent central processing responsible for the PRP effect. It is likely that the PRP effect is dominated by central postponement, because the 65-ms increase in SPCN latency between the shortest (300 ms) and longest (1000 ms) SOAs accounts for only about 27% of the 240-ms RT2 effect. Nevertheless, the substantial increase in the latency of the SPCN observed here is an important result, because it is the first demonstration that early visual encoding processing (e.g., consolidation in VSTM) can be delayed by a demanding concurrent speeded auditory task. Although delayed, the SPCN finally reached similar amplitudes across task overlap conditions (i.e., SOA and Task1 difficulty conditions), suggesting that a stable VSTM representation could eventually be achieved in all conditions, which is consistent with the high accuracy of report of T2 in all conditions.

Figure 5. Contralateral minus ipsilateral difference waves time-locked to T2 onset at P7/P8, PO7/PO8, and O1/O2 for the hard-Task1 and easy-Task1 conditions and the pooled response over these three electrode pairs for both Task1 difficulty conditions.
Recent studies have demonstrated that the N2pc was also attenuated in the attentional blink (AB) paradigm (Dell’Acqua et al., 2006; Jolicœur et al., 2006a, 2006b). In the AB paradigm, accuracy of report for some aspect of a masked T2, such as target identity, suffers when T2 is presented at a short SOA following a T1 that must also be processed. The similarity of the interaction of central load on the N2pc mean amplitude when using PRP and AB paradigms provides more evidence in support of the central interference theory (Jolicœur, 1998, 1999a) that postulates that response selection (assumed to be an important locus of the PRP effect) and short-term consolidation (postulated to be an important locus of the AB effect) have some overlap at the level of limited central mechanisms.

Although the N2pc modulations are similar when using the PRP and AB paradigms, interesting differences can be observed for the SPCN. In the AB paradigm, where dual-task interference is reflected by a decrease in T2 report accuracy as SOA is reduced, the SPCN is also sharply attenuated (Dell’Acqua et al., 2006; Jolicœur et al., 2006a, 2006b). In the PRP paradigm, where dual-task interference is usually reflected by an increase in RT2 without any effect on T2 report accuracy, the SPCN onset latency is lengthened, but finally reaches a similar amplitude in all SOA conditions. In a recent study, Woodman and Luck (2003a) demonstrated that delayed-offset four dot masking (also called object-substitution masking; Di Lollo, Enns, & Rensink, 2000), which reduces report accuracy of the masked item, does not attenuate the N2pc, but seems to have a large effect on the SPCN. As in the previously mentioned AB experiments, the SPCN amplitude seemed to follow closely report accuracy. The pattern of results in this experiment are complimentary to those observed in Woodman and Luck (2003a) in that the N2pc was attenuated, whereas both the late portion of the SPCN (500–600 ms) and T2 accuracy were unaffected by SOA. This double dissociation between N2pc amplitude and report accuracy suggests that conscious report is not directly correlated to a successful allocation of visual–spatial attention. On the other hand, the amplitude of the SPCN, and therefore VSTM activity, seems to be a good predictor of conscious report (Dell’Acqua et al., 2006; Jolicœur et al., 2006a, 2006b). The present results show that encoding a visual representation in a format that supports conscious report is delayed significantly by cross-modal multitasking.

REFERENCES


4The SPCN was not analyzed in Woodman and Luck (2003a), but is visible in the presented figures.


(Received June 13, 2006; Accepted December 11, 2006)