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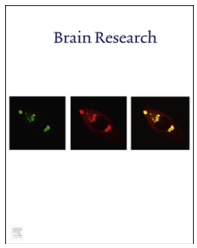

Research Report

The attentional blink freezes spatial attention allocation to targets, not distractors: Evidence from human electrophysiology

Vincent Jetté Pomerleau[a], *Ulysse Fortier-Gauthier*[a], *Isabelle Corriveau*[a], *John J. McDonald*[b], *Roberto Dell'Acqua*[c], *Pierre Jolicœur*[a,*]

[a]*Université de Montréal, Montréal, Québec, Canada*
[b]*Simon Fraser University, Burnaby, British Colombia, Canada*
[c]*University of Padova, Padova, Italy*

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ABSTRACT

Previous work found a significant reduction of the amplitude of the N2pc ERP component during the attentional blink in response to lateral visual targets, suggesting that the allocation of attention to visual targets is impaired during the attentional blink. Recent theorizing on the processes reflected by the N2pc suggests the possibility of distinct sets of neural mechanisms underlying its generation, one responsible for target activation, and one for distractor inhibition. To disentangle whether either or both of these mechanisms are impaired during the attentional blink, an RSVP sequence of circles, equidistant from fixation was used. The first target frame (T1) contained the same repeated target colour circle and target whereas the second target frame (T2) contained a distractor colour singleton as well as a target colour singleton. Only the target or only the distractor was presented at a lateral position; the other singleton was presented on the vertical midline so as not to elicit any event-related lateralization. Impaired T2 report accuracy at a short stimulus-onset asynchrony (SOA) was accompanied by a significant delay of the N2pc to lateral T2 targets when compared to a long SOA condition. No such delay was found when the lateralized stimulus was a distractor, suggesting that the attentional blink impacts attention allocation to targets, not distractors. We also observed a lateralized component earlier than the N2pc, a posterior contralateral positivity (Ppc) that did not depend on T1–T2 SOA and that was elicited by both lateral targets and distractors. We conclude that, contrary to N2pc, the Ppc likely reflects activity of bottom-up mechanisms responding unselectively to asymmetrical visual displays.

Abbreviations: AB, Attentional blink; NT, Target negativity; PD, Distractor positivity; Ppc, Posterior contralateral positivity; RSVP, Rapid serial visual presentation; SOA, Stimulus-onset asynchrony

*Correspondence to: Département de Psychologie, Université de Montréal, C.P. 6128, succursale Centre-ville Montréal, QC, Canada H3C 3J7. Fax: +1 514 343 6511.
E-mail addresses: vincent.jette.pomerleau@umontreal.ca (V.J. Pomerleau), pierre.jolicoeur@umontreal.ca (P. Jolicœur).

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1. Introduction

The attentional blink (AB) is a well-known behavioural consequence of the limitations of central attention (Jolicoeur, 1999). The AB can be observed in a dual-task paradigm in which two target stimuli are presented in rapid succession, typically within a rapid serial visual presentation (RSVP) of nontargets (Raymond et al., 1992; Shapiro et al., 1997). The AB is characterised by a reduced performance in the task related to the second target stimulus (T2) compared to the performance related to the first target (T1) (Cousineau et al., 2006). The AB is generally largest when the SOA between T1 and T2 is about 200–300 ms, although several papers report a significant AB at SOAs longer than 500 ms (Arnell and Jolicoeur, 1999; Jolicoeur, 1999; Ouimet and Jolicoeur, 2007; Raymond, 2003; Visser et al., 1999). Thus, the AB is not tied to a specific time window but rather is a function of many tasks and display parameters, including the rate of presentation and the nature of the task associated with the first target (Jolicoeur, 1999; Ouimet and Jolicoeur, 2007).

At least five classes of models have been proposed to account for the AB and other attentional perturbations in temporal domain (for a detailed review of the theories, see e.g., Dux & Marois, 2009): the limited capacity model (Dell'Acqua et al., 2009); the temporary loss of control model (Kawahara et al., 2006); the boost and bounce theory of temporal attention (Olivers and Meeter, 2008); the ACT-R based model (Taatgen et al., 2009), and the episodic simultaneous type serial token model (Wyble et al., 2009). Although these models differ in the details of their explanation, and even though it is sometimes possible to observe an AB in the absence of masking, all agree that presenting T1 and then a masking distractor creates conditions that foster the AB.

Physiological processes related to visual-spatial attention and limitations of attention (e.g., the AB) can be evaluated with event-related potentials (ERPs). One ERP component in particular, the N2pc, has been associated with the deployment of visual spatial attention (Luck and Hillyard, 1994a, 1994b; Woodman and Luck, 2003). The N2pc is a lateralized ERP component that can be observed by subtracting the electrical potentials measured at electrode sites ipsilateral to a lateral attended object from the electrical potentials measured at electrode sites contralateral to that object. The N2pc is observed over the posterior scalp, with a peak amplitude typically observed at or near electrodes PO7/PO8. As its name suggests, the latency of the N2pc is in the N2 time range, which occurs at about 180–280 ms following the onset of an attended stimulus (Brisson and Jolicoeur, 2007; Luck and Hillyard, 1994a, 1994b; Robitaille and Jolicoeur, 2006). Luck and Hillyard, 1994a, 1994b argued that the N2pc reflects spatial filtering of distractors, whereas Eimer (1996) and, more recently, (Mazza et al., 2009a, 2009b) argued that the N2pc reflects target enhancement. Another ERP component of interest is the sustained posterior contralateral negativity (SPCN), reflecting maintenance in visual short-term memory selection and individual differences in storage capacity (Jolicoeur et al., 2008; Klaver et al., 1999; Vogel et al., 2005).

Several studies have examined the deployment of visual spatial attention in RSVP tasks and showed a decrease in the amplitude of the N2pc as a result of AB interference (Dell'Acqua et al., 2006; Jolicoeur et al., 2006a, 2006b. In the experiment of Jolicoeur et al. (2006b), coloured digits (one in left visual field and one in right visual field) were shown within an RSVP stream of white letters. The task for the second target was to report the identity of a digit shown in a particular colour. A reduction in the amplitude of the N2pc was found when the N2pc-eliciting stimulus was presented during the AB, when a short temporal interval (200 ms) separated the two targets (Jolicoeur et al., 2006a). This electrophysiological effect was congruent with a decrease in report accuracy for the second task in the AB condition. Latency effects were mentioned in the paper but were not significant. In a subsequent study, however, the N2pc was delayed during the AB relative to a non-AB condition (Zhang et al., 2009). Zhang and colleagues presented a distractor (D1) at various SOAs before T1. The authors observed an inhibitory effect of D1 on T2 for a period of about 300 ms when they shared semantic properties. This effect was reflected in the accuracy of the identification to the T2 and in a delay of the N2pc. Taken together, the results of these various studies provide evidence for interactions between the mechanisms required to encode T1 and to deploy visual spatial attention to T2. The encoding of representations in visual short-term memory for stimuli presented in an RSVP sequence has also been of some interest in previous AB studies (Jolicoeur et al., 2006a, 2006b). These studies showed a clear decrease of the SPCN when T2 lagged T1 by a short SOA. Such a decrease was associated with poorer encoding of targets, as observed in the presence of an AB.

It had proven difficult to determine whether AB reflects interference with target processing or distractor suppression because the spatial relationship between these objects had usually been constant. For example, in Jolicoeur et al.'s (2006a, 2006b) studies, the lateral T2 target was always accompanied by a distractor item on the opposite side of fixation. In this case, the target-related N2pc could reflect a greater negativity contralateral to the target or a greater positivity contralateral to the distractor.

The purpose of the present research was to disentangle the influences of AB interference on target- and distractor-related processing. To this end, we recorded EEG during an AB task and measured lateralized ERP negativities associated with target selection – the aforementioned N2pc and SPCN components – and a lateralized ERP positivity that has been associated with distractor suppression. Although the N2pc is usually described as a negativity contralateral to the attended target, a recent study provided evidence and argued for a two-component hypothesis of the N2pc (Hickey et al., 2009). One of the suggested sub-components of the N2pc would be a target-related negativity (N_T). The N_T, presumably representing the processing specific to the target during attentional deployment, was isolated by presenting a single salient distractor on the vertical midline and a single target to the left or right of fixation. This approach relied on the fact that selection of stimuli on the vertical midline creates a constant pattern of hemispheric activation that produces no lateralization as a function of other lateral stimuli (Woodman and Luck, 2003). Because only the target was lateralized, the observed N_T, which occurred in the time range of the N2pc, could be linked unambiguously to the target (Hickey et al., 2009; Woodman

and Luck, 2003). This negative going component was observed over the contralateral side of the target. It peaked between 175 and 325 ms post-stimulus and had an amplitude between −1.5 and −2.25 mV, bearing a close similarity with the N2pc. A similar procedure was used to isolate lateralized activity related to the distractor by positioning the target on the vertical midline and the distractor laterally. This procedure indicated a different sub-component, the P_D, for "distractor positivity." As suggested by the name, the P_D was a positivity contralateral to the side of the distractor (Carlisle and Woodman, 2011; Hickey et al., 2009; Sawaki and Luck, 2011).

Few experiments have studied attention using procedures to isolate target-related activity from distractor-related activity (Hickey et al., 2009; Hilimire et al., 2011; Sawaki and Luck, 2010). The evidence that target and distractor processing may be subserved by dissociable neural sources is appealing and motivates further studies to enhance our understanding of the mechanisms of visuo-spatial attentional deployment and their interactions with distinct types of attention. In the present work, our focus is on the interplay between mechanisms controlling the allocation of attention in the spatial domain – as reflected by the N2pc – and mechanisms controlling the allocation of attention in the temporal domain, using a two-target detection task while subjects are exposed to the rapid serial visual presentation (RSVP) of spatially overlapping visual stimuli. The purpose of the present research was to examine the influences of the AB paradigm on the possible target-related processing ($N2pc/N_T$) and distractor-related processing (P_D). Examining dual-task interference from the AB paradigm on each of these aspects of visual-spatial attention will increase our understanding of the processes underlying the deployment of the visual-spatial attention.

We expected that the AB would likely have an effect on target-related processing contributing to the N2pc (i.e., N_T), because this appears to be the larger contributor to the N2pc. In addition, we wished to discover whether the AB would also interfere with possible distractor suppression, as might be revealed by the P_D. A diminution of amplitude of one or both sub-components under AB conditions would imply a less effective deployment of attention to enhance target processing and/or an attenuation of distractor interference. An increase in latency of one or both sub-components would imply a reduced efficiency in attentional selection, likely leading to less efficient downstream processing. Both of these results could contribute to the observed behavioural AB effect. We also expected the SPCN component to be present only for lateralized targets, on the assumption that distractors would be filtered out from processing prior to entry into visual short-term memory. An attenuated, or completely suppressed, SPCN for lateral distractors, in the presence of a clear SPCN for lateral targets, would provide converging evidence for effective suppression of distractors from downstream processing.

2. Results

2.1. Behaviour

The mean accuracy in each condition is listed in Table 1. The overall mean accuracy rates were 90% (S.E.=10.0) for Task 1 and 72% (S.E.=16.0) for Task 2. For Task 1, an analysis of variance (ANOVA) revealed a significant effect of Trial Type (no-T1, short-SOA, long-SOA), $F(2, 34)=15.66$, $p<.0001$, $\eta^2=.48$. No-T1 trials had a significantly higher Task 1 accuracy than short or long SOA trials, which did not significantly differ. As seen in Table 1, the mean accuracy rates on the short-SOA and long-SOA trials were lower than on the no-T1 trials in Task 1. For Task 2, we also submitted the mean accuracy for each condition to a repeated-measure ANOVA with Trial Type as the within-subjects factor, $F(2, 34)=14.71$, $p<.0001$, $\eta^2=.46$. A post-hoc Tukey ($q(3,34)=3.46$)[1] confirmed a significant difference between the short-SOA ($M=.67$) and the long-SOA ($M=.74$) and between the short-SOA and no-T1conditions ($M=.74$). As can be seen in Table 1, accuracy was lower on short-SOA trials than on the long-SOA trials and the no-T1 trials, reflecting the expected AB effect. That AB effect was furthermore reflected in this set of data through a significant contrast that compared the mean accuracy for the report-T1, Short-SOA condition with the average of the two control condition, $F(1, 17)=25.74$, $p<.0001$, $\eta^2=.60$ (i.e., the contrast weights were 1, −2, 1, for the no-T1, short-SOA, and long-SOA conditions, respectively). This pattern of means was expected and the main ANOVA and contrast analysis confirmed the presence of an AB.

Table 1 – Mean accuracy in per cent and standard deviation (in parentheses) for the control condition (no-T1), the short-SOA condition, and the long-SOA condition, for each task.

	Task 1	Task 2
No-T1	98 (2)	75 (16)
Short SOA	86 (15)	67 (18)
Long SOA	86 (15)	75 (15)

The effect of the position of the target (midline or lateral) was also evaluated. Table 3 shows T2 results for all trial types depending on target position (midline or lateral). A repeated measure ANOVA with lateralization (midline or lateral target) and trial type, we found no effect of lateralization ($F(1, 17)=1.32$, $p>.26$) nor interaction ($F(1, 17)=2.06$, $p>.16$). As expected, the trial type effect was highly significant ($F(2, 34)=14.71$, $p<.0001$). Thus the ERP analyses for T2 were based on approximately equal numbers of T2-seen trials for both midline and lateral targets.

2.1.1. Electrophysiology ($N2pc: N_T/P_D$)

Fig. 2 presents grand-averaged event-related lateralizations (ERLs) obtained by subtracting ipsilateral ERP waveforms from contralateral ERP waveforms, for electrode pair PO7 and PO8, separately for T2 displays containing a lateral target or a lateral distractor. These averages were time-locked to the onset of T2 and included a 200-ms pre-stimulus baseline and extended 700 ms after T2 onset.

Analyses of mean amplitudes were performed on the average voltage at PO7/PO8 (where the N2pc was largest) in a window starting 30 ms before and extending to 30 ms after

[1]For individual t-tests comparing means for every post-hoc Tukey, the reader is referred to Table 2.

Table 2 – Paired samples *t* tests ($t=17$) comparing means also tested with Tukey tests throughout the paper.

	Behavioural results	N2pc amplitude 170–270 ms window results	N2pc latency results
No-T1 vs Short SOA	4.24, $p<.005$	−2.24, $p=.038$*	−4.13, $p<.001$**
No-T1 vs Long SOA	−.01, $p>.99$	.91, $p>.37$	−1.03, $p>.30$**
Short SOA vs Long SOA	−4.91, $p<.001$	3.07, $p<.01$	3.32, $p<.005$**

* Although this result seems contradictory with our reported results, one must remember that repeated *t*-tests are slightly more lenient than Tukey tests. In this case, the Tukey showed a close to significant difference between the two conditions. This result does however fit with that of the contrast analysis (pp. 10–11).

** The use of the *t*-tests with the jackknife required some correction to the *t* value. This correction compensated for the artificial deflation of the error variance associated with sets of jackknife waveforms ($t'=t/(n-1)$); (Ulrich and Miller, 2001).

the grand average waveform peak for each condition of lateral stimulus target (for the N2pc) and of lateral distractor-related activity (see Table 4). A 3 × 2 ANOVA with Trial Type (no-T1, short-SOA, long-SOA) and lateral singleton (target, distractor) revealed a main effect of lateral singleton, $F(1, 17)=18.65$, $p<.0005$, reflecting a larger contralateral negativity for lateral targets ($M=-3.07\ \mu V$) than for lateral distractors ($M=-.90\ \mu V$). Two subsequent ANOVAs evaluated mean amplitude differences across trial type, independently for each lateral stimulus condition. Neither one found significant amplitude differences across conditions (lateral target N2pc: $F(2, 34)=1.71$, $p>.19$; lateral distractor N2pc: $F(2, 34)=.55$, $p>.58$). Because no trial type effect reached significance for either component, data from no-T1, short-SOA, and long-SOA trials were averaged together for each of the two lateral stimulus condition. These averaged components reflected the activity related to a lateral distractor or a lateral target, independently of trial type. In each case, the contralateral negativity was significantly different from 0, $t(17)=5.86$, $p<.0001$, for lateral targets, and $t(17)=4.30$, $p<.0005$, for lateral distractors (see Table 5 for individual condition *t*-tests).

A different window was also chosen for further analyses on the N_T/P_D amplitudes (170–270 ms). This wider window was chosen based on the overall activity of both lateral distractor and target. A 3 × 2 ANOVA with Trial Type (no-T1, short-SOA, long-SOA) and lateral singleton (target, distractor) revealed an interaction effect between trial type and lateral singleton $F(2, 34)=3.44$, $p<.05$. To explore that interaction, two subsequent ANOVAs evaluated mean amplitude differences across trial type, independently for each lateral stimulus condition. Significant differences were found between the conditions of the lateral target only (lateral target N2pc: $F(2, 34)=4.95$, $p<.013$; lateral distractor N2pc: $F(2, 34)=.71$, $p>.45$). A post-hoc Tukey ($q(3,34)=3.46$)[2] revealed a significant difference between the short-SOA ($M=-1.60\ \mu V$, SD=.56) and long-SOA ($M=-2.80\ \mu V$, SD=.58) conditions for the lateral target, the short-SOA N2pc/N_T being the least negative component. Neither the long SOA nor the short SOA differed significantly from the No-T1 condition ($M=-2.42\ \mu V$, SD=.56). Interestingly, in this time window, the mean voltage of the N2pc was smaller for the short-SOA condition than the average voltage of both the no-T1 and long-SOA conditions, as shown by a significant contrast, $F(1, 17)=10.04$, $p<.006$ (contrast weights were 1, −2, 1, for the no-T1, short-SOA, and long-SOA conditions, respectively). Note that these amplitude effects would be explained, in whole or in part, by latency differences. This possibility is explored in the next paragraph.

Table 3 – Mean accuracy in Task 2, per cent correct and standard deviation (in parentheses), for the control condition (no-T1), the short-SOA condition, and the long-SOA condition, for midline and lateral targets.

	Midline target	Lateral target
No-T1	75 (16)	74 (16)
Short SOA	67 (17)	67 (20)
Long SOA	76 (15)	73 (17)

We examined the latency of the onset of lateralized activity across experimental conditions. These analyses used repeated-measures ANOVAs combined with the jackknife method (Kiesel et al., 2008; Miller et al., 1998). The use of the repeated measure ANOVA with the jackknife required some correction to the *F* value. This correction compensated for the artificial deflation of the error variance associated with sets of jackknife waveforms ($F'=F/(n-1)^2$; (Ulrich and Miller, 2001). We measured the latency at which jackknife curves crossed a threshold set at a percentage of 50% of maximum amplitude between 150 and 300 ms. The search for the threshold started at 150 ms post stimulus. No significant SOA differences were found for the distractor related activity (Fig. 2B) ($F'(2, 34)=1.81$, $p>.15$). The N2pc (N_T) however showed significant differences between SOAs (Fig. 2A) ($F'(2, 34)=9.39$, $p<.0001$). Post-hoc (Tukey) ($q(3,34)=3.46$)[3] tests revealed a significant difference between long (M=191.29 ms, SD=2.32) and short SOA (M=209.71 ms, SD=2.50) and between no-T1 (M=186.35 ms, SD=1.70) and short SOA conditions. Tukey post-hoc testing was also corrected for the reduced variance in jackknife curves ($H'=H^*(n-1)$) (Ulrich and Miller, 2001). Condition specific voltage maps shown in Fig. 3a were produced to examine the scalp distribution of lateralized activity in the most important experimental conditions. The distributions were quite similar, overall, all showing a typical N2pc-like peak near PO7/PO8, as expected from previous research (e.g., Jolicoeur et al., 2008).

2.1.2. *Electrophysiology (SPCN)*

We assessed the mean amplitude of the SPCN at electrodes PO7/PO8, as we did for the N2pc. For that purpose, the time

[2]For individual *t*-tests comparing means for every post-hoc Tukey, the reader is referred to Table 2.

[3]For individual *t*-tests comparing means for every post-hoc Tukey, the reader is referred to Table 2.

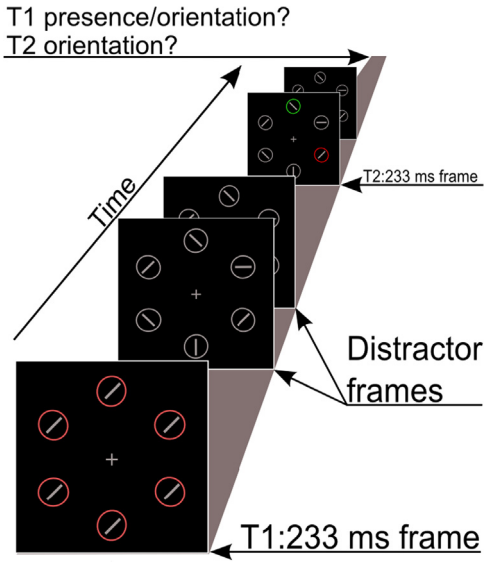

Fig. 1 – Illustration of a rapid serial visual presentation for a short SOA trial (associated with the attentional blink). The arrow represents the passage of time. Illustrated is a trial with red as a target colour. Circles in T1 thus appeared in desaturated red. Two frames (466 ms) later T2 was presented, with (in this case), a lateralized target (red) and a distractor (green) on the midline. Actual trials had five to nine frames before T1 and two to six frames after T2. The first visual frame, not visible here, displayed a fixation cross. A representation of a long SOA condition would contain five instead of two visual frames between the two targets, and a representation of a no-T1 condition would have only T2.

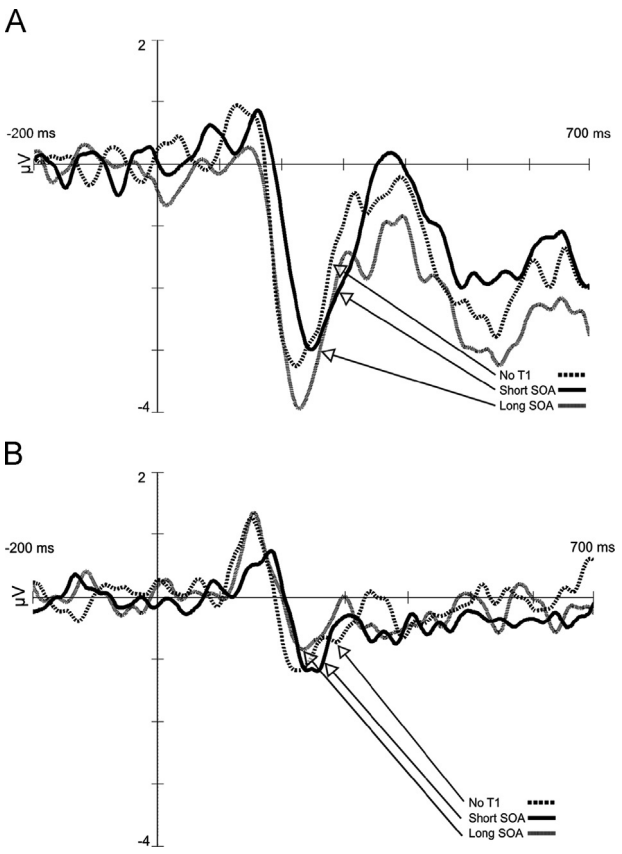

Fig. 2 – Grand average of event-related lateralization waveforms (contralateral minus ipsilateral) at PO7/PO8 time-locked to the onset of T2 in each condition. Tick marks on the time axis represent 100 ms increments. (A) Trials in which the target was lateral. (B) Trials in which the distractor was lateral.

window for the lateral target and lateral distractor SPCN waves extended from 500 to 570 ms. A 3 × 2 ANOVA with Trial Type (no-T1, short-SOA, long-SOA) and lateral singleton (target, distractor) revealed a main effect of lateral singleton, $F(1, 17) = 16.84$, $p < .0008$, reflecting a larger contralateral negativity for lateral targets ($M = -2.46\ \mu V$) than for lateral distractors ($M = -.17\ \mu V$). Two subsequent ANOVAs evaluated mean amplitude differences across trial type, independently for each lateral stimulus conditions. A marginally significant difference was found for lateral targets, but no effect was significant for lateralized distractors (lateral targets: $F(2, 34) = 3.13$, $p = .056$; lateral distractors: $F(2,34) = .52$, $p > .60$). The marginal difference, as indicated by the means, would suggest that the long-SOA (−3.06) differed from the short-SOA (−1.82).[4] All three conditions of the lateralized target were significantly different from zero (no-T1: $t(17) = -5.39$, $p < .0001$; short-SOA: $t(17) = -3.31$, $p < .005$; long-SOA: $t(17) = -4.84$, $p < .0002$). Because no trial type effect reached significance for lateralized distractors, data from no-T1, short-SOA, and long-SOA trials were averaged together for that condition. That averaged component reflected the activity related to the encoding of a lateral distractor, independently of trial type and did not differ from 0 $t(17) = -.90$, $p > .35$. As it was observed in the behavioural and the N2pc/N_T data, a contrast evaluating the difference between the short-SOA condition and the mean of the two control conditions was significant and reflected the larger SPCN related to lateralized targets

Table 4 – Time windows for N2pc/N_T and N2pc/P_D in milliseconds, post stimulus.

	N2pc/Targets	N2pc/Distractor
No-T1	200–260	200–260
Short SOA	220–280	220–280
Long SOA	200–260	215–275

Table 5 – *T*-values of *t*-tests against zero for lateralized activity related to targets (N2pc/ N_T) and distractors (N2pc/P_D).

	N2pc/Target	N2pc/ Distractor
No-T1	$t(17) = -5.02$, $p < .001$	$t(17) = -4.07$ $p < .005$
Short SOA	$t(17) = -4.63$, $p < .001$	$t(17) = -3.26$, $p < .01$
Long SOA	$t(17) = -6.01$, $p < .005$	$t(17) = -2.36$, $p < .05$

[4]Subsequent two-tailed *t*-tests revealed that only the short and long-SOA SPCN significantly differed ($t(17) = 2.77$, $p < .015$), while the short and no-T1 ($t(17) = -1.59$, $p > .10$) as well as the long and no-T1 ($t(17) = .95$, $p > .35$) conditions did not differ in terms of mean voltage.

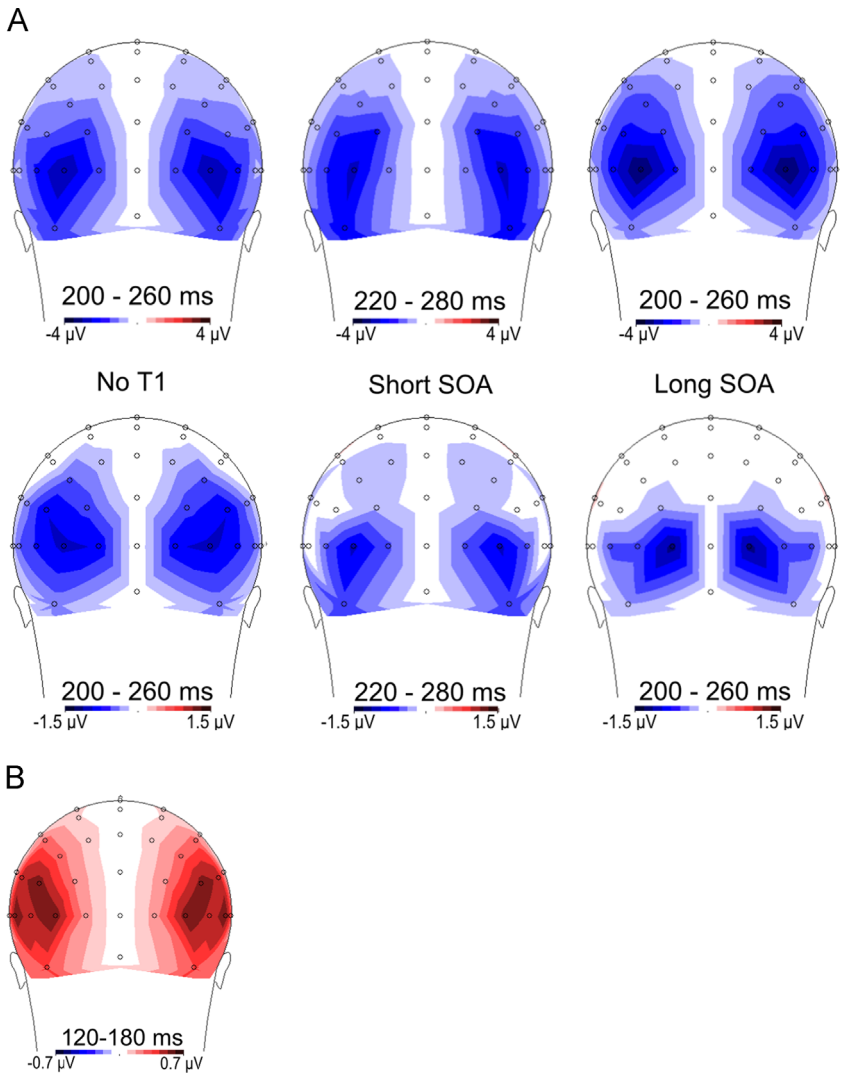

Fig. 3 – Grand average scalp voltage distributions. (A) N2pc: top row shows the distributions for the lateralized target trials for each condition (no-T1, short-SOA, long-SOA); bottom row shows the distributions for lateralized distractor trials, for each condition. (B) Scalp voltage distribution for the Ppc, averaging across all conditions.

$F(1, 17)=8.51$, $p<.01$ (contrast weights of 1, −2, 1, for the no-T1, short-SOA, and long-SOA conditions, respectively). That result indicates the presence of an AB on the amplitude of the SPCN consistent with a reduced probability of transfer of T2 into visual short-term memory in the short-SOA condition compared with the mean of the two control conditions. This difference, expected from earlier AB experiments, is also evident when illustrated through a voltage distribution map of the SPCN for the no-T1, short, and long-SOA trials with a lateralized target (see Fig. 4) (Dell'Acqua et al., 2006; Jolicoeur et al., 2006a, 2006b).

2.1.3. *Electrophysiology (Ppc)*

Analyses also examined a positive posterior contralateral (Ppc) component observed just prior to the N2pc both for lateral target and lateral distractor trials. Grand averages of both components can be seen in Fig. 2. The mean amplitude of the Ppc for each subject and condition in a window of 30 ms around the peak of each component (see Table 6) was submitted to a 3×2 ANOVA (Condition × lateralized item (target or distractor)). The ANOVA revealed no significant effect of lateral item ($F(1, 17)=1.63$, $p>.20$), SOA condition ($F(2, 34)=1.40$, $p>.25$), or interaction of the two ($F(2, 34)=1.71$, $p>.15$). For that reason, subsequent analyses of Ppc amplitude did not consider which item was lateral or SOA conditions, and the six different components were averaged together.

The existence of the Ppc component was assessed by a one sample *t*-test versus 0 on the component average amplitude. The Ppc ($t(17)=5.56$, $p<.0001$) was clearly different from zero. The Ppc was therefore unlikely to be noise (and this was further corroborated by the fact that the positive deflection was visible by eye in all 6 experimental conditions, as can be seen in Fig. 2). Although the component exists, it was not different in amplitude across distractor or a target lateralization, or depending on whether the presentation was at short SOA or long SOA relative to T1, or even when there was no-T1. Because no significant differences were found on

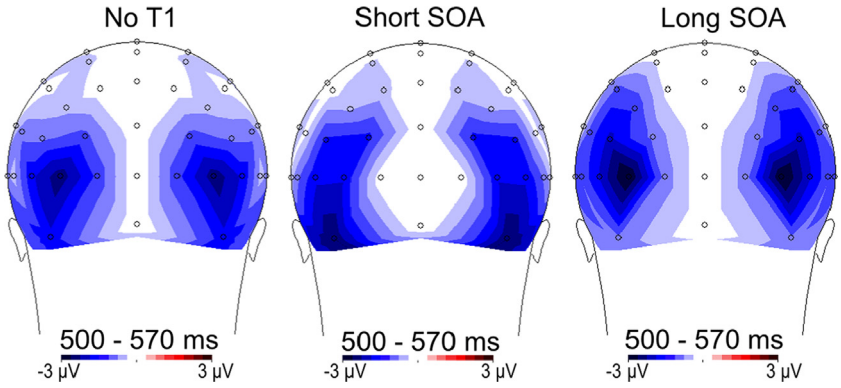

Fig. 4 – Grand average scalp voltage distributions of the SPCN for the lateralized target's conditions (no-T1, short-SOA, long-SOA).

Table 6 – Time windows for each Ppc component in milliseconds, post stimulus.

	$N2pc/N_T$	$N2pc/P_D$
No-T1	100–160	120–180
Short SOA	130–190	130–190
Long SOA	120–180	120–180

amplitudes on conditions or lateralized item and visual inspection of the individual distributions did not reveal systematic differences, only one voltage distribution map for the Ppc component is shown here, based on the average waveform computed over all conditions (see Fig. 3b). The distribution is clearly very posterior and has some similarity with the N2pc distribution, but with a sign reversal, and a slightly more superior and anterior extent.

3. Discussion

We studied how the AB interacts with the control of the deployment of visual spatial attention to a second stimulus (T2) by manipulating the presence/absence of a preceding target (T1) or the delay between T1 and T2. We isolated target-related and distractor-related processing by placing either the target or the distractor laterally in displays that contained two salient coloured stimuli (one target, one distractor). The item that was not lateralized was presented on the vertical midline, effectively nulling out contributions of this stimulus to spatially-driven event-related lateralized activity. Three major findings resulted from the experiment. Firstly, we found a clear delay of target-related spatial processing in the AB (Fig. 2, Panel A, short-SOA condition). Secondly, lateral distractors were associated with a small but significant contralateral negativity that was not significantly affected by the AB. Thirdly, we found an earlier positivity contralateral to the salient lateral stimulus (the Ppc), whether that stimulus was a target or distractor, in every condition (no-T1, short-SOA, long-SOA). We discuss each of these findings in turn in the following paragraphs.

Behavioural results indicated a significant decrease in task 2 accuracy for the short-SOA condition relative to the other two conditions as shown in an omnibus AVOVA, and with a significant difference in a contrast that compared the AB condition with mean of the two control conditions. The significant difference between the no-T1 condition and the other conditions for Task 1 probably reflects a difference between condition difficulties for that task. This difference in difficulty does not seem to affect Task 2 performance, as the long-SOA and no-T1 condition did not differ.[5]

In addition to a decrease in accuracy for T2, the AB delayed the onset of the N2pc for lateral targets, as shown in Fig. 2. We note that the rate of stimulus presentation in our RSVP streams was slower than in many previous AB studies, and that the shortest tested SOA between T1 and T2 was 466 ms. Despite this relatively long delay, we found a clear AB in accuracy and a delay of the N2pc for targets, suggesting that the effects were due to processing limitations required to process T1 (i.e., to encode and store the orientation of T1 for later report), rather than effects at the level of the spatial reorienting of visual-spatial attention, which appear to take place on a shorter time scale (on the order of 100–200 ms), based on effects confined to lag-1 sparing in several AB experiments with a switch in spatial location across targets; see Visser et al (1999). Thus, one of the main findings of the present study was that spatial attention specifically related to target processing could be delayed as a result of the AB. This in turn suggests that previous results showing similar effects on the N2pc, but in the context of displays that confounded target-related with distractor-related processing, likely contained a significant proportion of target-related interference

[5]The NO-T1 condition had some differences with the two other conditions, as this condition needed no precise answer for line orientation. This situation created a bias in which if a participant were to press the space bar in response to all trials in the experiment, accuracy in the no-T1 condition would be 100% correct. Doing so would however result in a 0% accuracy in T1 for the short and long SOA conditions. Upon reviewing individual performance, such a situation did not happen. Performance for T1 for the short and long SOA conditions varied between 40 and 98%, with a mean of 86% in both conditions (see Table 1 for T1 performance). Moreover, erroneous T1-present trials where participants pressed the space bar represented an average of .35% of participant's answers and 6 participants did not make such a mistake throughout the experiment.

(Dell'Acqua et al., 2006; Jolicoeur et al., 2006a, 2006b; Robitaille et al., 2007; Zhang et al., 2009).

An unexpected result was that, for the short-SOA condition, the amplitude of the N2pc in the lateral-target condition was not significantly smaller than in the no-T1 or long-SOA conditions. Previous work on the AB and the N2pc found a systematic decrease in the amplitude of the N2pc when the SOA between target stimuli was short (Dell'Acqua et al., 2006; Jolicoeur et al., 2006a, 2006b). The only amplitude effect that was found was between the short and long SOA in a time window that could reflect differences in latency rather than amplitude *per se*. It is possible that previous work contained some amplitude effects as well as some latency effects.

Interestingly, instead of the typical amplitude differences we found a stronger latency effect in the present work (although hints of delays on the N2pc onset can be gleaned from previous papers, they were not tested; e.g., Jolicoeur et al., 2006a). One possibility is that previous displays pitted a lateralized target with a lateralized distractor positioned symmetrically across the vertical midline. It is possible that attention was partially deployed to the distractor under high AB load (reflecting a form of loss of control over the deployment of attention). Attention deployed to the distractor would be associated with an N2pc relative to the distractor, which would be subtracted from the N2pc to the target, leading to a reduced amplitude. In the present design, a loss of selection specificity and attention deployed to the distractor would result in some attention deployed to the vertical midline (when the target was lateralized), which would neither add nor subtract from the N2pc to the target. Perhaps present conditions allowed us to see more clearly a processing delay that may have been present in earlier work, but partially masked by concurrent lateral distractor competition. Alternatively, our visual presentation could partially explain this finding. Indeed, an unexpected effect of our longer SOA could have been to give participants more time to deploy their attention to the target. If that would be the case then subjects could proceed to deploy their attention to T2 in the short SOA condition slightly later, thus allowing a better encoding of T2 and a better performance.

It should also be acknowledged that suppression of the N2pc towards a lateral target has been observed for conditions in which the opposing distractor is unchanged; that is, as a consequence of working memory load alone (Akyurek et al., 2010). As such, although our visual display does differ from that of many previous papers, the opposing-distractor account probably cannot, by itself, fully explain our observations.

Sometimes, latency effect in ERPs correlate well with RT effects in behaviour and sometimes this correlation is near zero (Verleger, 1997). In the AB paradigm, Jolicoeur, his colleagues, and others have argued and provided strong evidence that a longer period of central processing of T1 causes a delay of processing of T2, leading to a loss of report accuracy for T2 when T2 is masked effectively (Giesbrecht and Di Lollo, 1998; Jolicoeur, 1999; Jolicoeur and Dell'Acqua, 1998; Ptito et al., 2008; Sessa et al., 2007; Vogel and Luck, 2002). A delay in the onset of processing of T2 likely results in a loss of information in the representation of T2 that is exacerbated by the mask, leading to a loss of report accuracy. Results such as those of Vogel and Luck (2002) or Ptito et al. (2008) provided ERP evidence that corroborated previous psychophysical evidence for a postponement of certain aspects of T2 processing during the AB, and the present results provide evidence that one locus of such postponement can occur at the level of the deployment of visual spatial attention to T2.

In order to study how the AB affects the deployment of visual spatial attention to lateral targets or distractors, we embedded the T2 frame in an RSVP sequence in which the T1 frame contained a repeated colour cue that matched the cue for the target in T2, and a repeated line orientation required for the response. Given that the location of the target in the T2 frame could be at any of the six possible circle locations in these displays, spatial attention presumably had to be maintained in a diffused state at the onset of each trial. Attention would presumably be focused on the location of the target stimulus some time after the presentation of T2. We wished to avoid the need for attention to focus on a specific location in the T1 frame, which is why we presented the information required for the T1 response in all 6 locations. Sequential location effects across T1 and T2 in the AB tend to be confined to T1–T2 SOAs on the order of 100–200 ms (Visser et al., 1999). By using a longer SOA (466 ms) in our experiment, we aimed to be well outside the range of purely spatial interactions across T1 and T2. Given that the magnitude of the AB is affected both by the rate of presentation and by the SOA between T1 and T2, we expected that the present conditions would produce a smaller AB. As expected, our AB effect was relatively small, but it was nonetheless, clearly significant and sufficient to cause a significant delay of the N2pc to lateral targets. This effect on the N2pc is unlikely to be due to a general non-specific dual-task load because the paradigm we used required subjects to remain prepared to process both T1 and T2 in all trials. The SOA effect on the latency of the N2pc to lateral targets thus most likely reflects AB interference on the deployment of visual spatial attention to the target. Furthermore, because there was a long delay between T1 and T2, the source of this interference is unlikely to be linked to spatial aspects of T1 processing. Rather, the evidence suggests a more central locus of interference in which encoding and processing of T1 somehow interfered with spatial processing of lateral targets. This suggests some overlap between mechanisms that encode and store a representation of T1 and those that control the deployment of visual-spatial attention to T2.

Our experiment also isolated lateralized activity specifically related to distractors. In these trials, the distractor was lateralized while the target was on the vertical midline. Based on the results and arguments of Hickey et al. (2009), we expected to observe a positivity contralateral to the lateral distractor (P_D), and we wished to discover whether this component would be reduced in amplitude, delayed, or both by the AB associated with concurrent processing of T1. However, we did not find any distractor positivity that could correspond with the P_D in the present experiment. Rather, we found a negative component with a polarity, a latency, and a scalp distribution similar to the N2pc. Importantly, the N2pc to distractors was significantly smaller than that for lateral targets. Clearly, attention was preferentially directed to targets, but our results also suggest strongly that attention was

sometimes erroneously deployed to the distractor on some trials (leading to a small N2pc when averaged over all lateral-distractor trials). Under present stimulus and task conditions, attention was more likely to be deployed to the lateral distractor than in the Hickey et al. (2009) study, which was based on a single-task paradigm. Such loss of attentional selectivity could mask a possible small P_D that may have been present. Alternatively, maintaining an attention filter to exclude a distractor from further processing may be particularly difficult under dual-task load and the multiple simultaneous RSVP sequences used in the present experiment. Thus, it is possible that the P_D was simply abolished under our conditions. Research on attentional capture suggests that attention is sometimes deployed to lateral distractors and that this is accompanied by a contralateral negativity, namely an N2pc, as we found here (Hickey et al., 2006; Leblanc et al., 2008; McDonald et al., 2013). In particular, when the features (colour and shape) of stimuli vary randomly from trial to trial, a salient lateral distractor was found to elicit an N2pc on the slowest half of trials; on the fastest half of trials, the same distractor elicited no N2pc but a late P_D instead (McDonald et al., 2013). Even though we used the same target colour and distractor colour for the entire test session (colour was counterbalanced over subjects, which should have facilitated setting up target-selection and distractor-suppression filters), and used colours that were equiluminant with each other and with the grey distractor stimuli, it is evident that attention was nonetheless captured by the lateral distractor on a fraction of the trials, resulting in a small N2pc. Interestingly, when we examined the amplitude of the small N2pc to lateral distractors, the amplitude was slightly (but not significantly) larger in the short-SOA condition than in the long-SOA condition. This could be consistent with an increase in involuntary capture of attention by the lateral distractor under conditions of high central load (i.e., during the AB). This loss of control may have been exacerbated by reduced distractor suppression. Additional research will be needed to determine whether a P_D can be elicited with the displays used in the present work.

The analyses for the SPCN yielded important additional results. The most important was the presence of a significantly larger SPCN for lateral targets than for lateral distractors. In fact, the SPCN for lateral distractors did not differ from 0. These results suggest that only targets were encoded and maintained in visual short-term memory. Although lateral distractors elicited an N2pc, suggesting that attention was deployed at the distractor location, this initial attentional deployment did not result in further processing to the level of visual short-term memory. Thus, an effective filter for selecting targets and suppressing distractors was in place, but this filter was visible in the electrophysiological results mainly in biasing attention to the target and away from distractors, although not with complete effectiveness (resulting in a small residual N2pc), and in preventing representations of distractors from entering visual short-term memory (where any residual distractor processing was no longer statistically detectable). Another interesting result was the larger SPCN for lateral targets presented in the long-SOA conditions compared with the SPCN for the short-SOA condition (see footnote, p.12). These results suggest that, following a delayed attentional deployment, targets in the report-T1, short-SOA condition, were less likely to enter VSTM, resulting in a reduced likelihood of overt report. These results suggest that one contribution to the AB effect in the present paradigm was likely due to a relatively early interference at the level of visual spatial attention to T2.

Although highly statistically significant, the magnitude of the AB effects in this experiment were small. This likely reflects the long SOA between T1 and T2, required to minimize effects of a purely spatial nature. The small AB effect across the conditions could explain why the differences in the SPCN amplitude were also relatively small. This is the most probable account of the absence of a significant difference between the SPCN for the no-T1 and the two other conditions. On *a priori* considerations, the SPCN for the short-SOA condition was predicted to be smaller than for the mean of the two control conditions. This result was confirmed by a significant contrast testing this specific predicted pattern (see Jolicoeur et al., 2006b, pp. 419).

Importantly, the present dual-task conditions required to observe an AB were comparable in many ways to those used in previous studies of AB effects on the N2pc (Dell'Acqua et al., 2006; Jolicoeur et al., 2006a, 2006b). One difference is that earlier studies tended to produce larger AB effects. It is all the more remarkable that the present results produced no clear evidence for a P_D component when the lateral item was a distractor. One might hypothesize that present conditions, perhaps related to a significant dual-task load, disturbed the distractor-suppression mechanisms postulated to underlie the P_D. Importantly, if the present dual-task conditions were sufficient to disturb the mechanisms leading to a P_D, then earlier studies probably also abolished the P_D (particularly considering they typically had larger AB effects). Consequently, the present results support the view that AB interference on the N2pc in the present and previous studies was mediated primarily via interference on target-specific visual-spatial attention mechanisms.

One factor that needs to be considered in future research is the particular target-distractor distance used in the study given that this factor can affect the amplitude of the N2pc (Hilimire et al., 2009, 2010) and could influence the P_D. In particular, one might expect that greater target-distractor proximity would be associated with greater distractor interference on target processing, and increase the need for distractor suppression, perhaps leading to a larger P_D. Thus, another possible reason for the absence of a P_D in the present results might be the relatively large distance between target and distractor in our displays. These considerations, although pertinent in the understanding of visual-spatial attention, exceed the scope of this study, but would be a useful focus for future work.

The third major set of findings in the present study concerned the Ppc. The Ppc was a positivity contralateral to the lateralized salient stimulus just prior to the N2pc, whether that stimulus was a target or a distractor. Neither the amplitude nor the latency of the Ppc were significantly influenced by our experimental manipulations. The Ppc did not depend on the status of the stimulus as a target or a distractor, nor did it vary across the conditions that created the AB (no-T1, short-SOA, long-SOA), as can be seen in Fig. 2. The target and distractor related Ppc, are therefore assumed

to reflect brain activity related to the presentation of a salient coloured stimulus that was not balanced by an equivalent stimulus in the other visual field. Although our displays were balanced in terms of total luminance (a saliently coloured stimulus was balanced by an equiluminant grey stimulus on the other side), it appears that the lateralized coloured stimulus triggered an additional neuronal response that was not created by the luminance-matched grey distractor. The colour-unbalanced visual presentation thus enabled us to observe a brain response that likely reflects the relative salience of the lateralized coloured item, perhaps via activity of cells responding differently to the chromatic properties of the stimulus. An important result was that this response was equivalent in all conditions. Thus, it did not depend on the status of the stimulus as target or distractor, nor on the attentional load differences created by the dual-task conditions that lead to the AB. The relative impenetrability of our manipulations on the mechanisms responsible for the Ppc suggest that these mechanisms are of a more bottom-up nature than those that produced later ERPs, such as the N2pc.

Finally, we consider the left-right colour imbalance created by the presentation of a single lateral coloured stimulus and implications for the interpretation of effects on components like the Ppc, the N2pc, and the SPCN. It is possible that the Ppc reflects differential lateralized P1 and N1 response that depends on the low-level physical characteristics of the stimuli (in this case, colour), and that equating luminance does not control for all relevant brain responses leading to differences in lateralized ERPs (e.g., Fortier-Gauthier et al., 2013; Pomerleau, et al., 2014; Woodman and Luck, 2003, pp. 126, 128). In the present context, the presence of the Ppc, *per se*, could well reflect such a low-level sensory effect (see Luck and Hillyard, 1994a, 1994b). Importantly, however, modulations lateralized ERPs by experimental manipulations, were not confounded by differences in sensory input because such inputs were equated across conditions. Thus, we find it interesting that the Ppc was not strongly affected by target-distractor status or the AB, whereas the N2pc and the SPCN were both strongly affected by these manipulations. The modulation of the N2pc by target/distractor status of the lateral stimulus shows that attention can have strong influences on lateralized brain activity, over and above possible sensory imbalances due to local colour differences. We note that counterbalancing across subjects ensures that the target-distractor difference in the N2pc time window was not confounded by an association with a particular colour. The strong modulation of the following SPCN wave, which was present for lateral targets and virtually eliminated for lateral distractors, also could not have arisen because of a low-level stimulus confound. In this context, we find it interesting that the Ppc, which preceded the N2pc, was apparently not influenced by the target-distractor status of the lateral item, nor by the AB manipulation, providing boundary conditions for the latency of attention effects under present stimulus and attentional conditions.

4. Conclusion

According to extant models, the AB could reflect either capacity limitations at relatively late stages of processing, the presence of inhibitory feedback, an overexertion of cognitive control, or capacity limitations of working memory encoding mechanisms, all of which affect relatively late stages of T2 processing (Jolicoeur, 1999; Jolicoeur and Dell'Acqua, 1998; Olivers and Meeter, 2008; Taatgen et al., 2009; Wyble et al., 2009). Such models must be augmented, however, to include an overlap between mechanisms producing the AB with those that control the deployment of visual spatial attention, as argued by Jolicoeur et al., 2006a, 2006b, Dell'Acqua et al. (2006), and Robitaille et al. (2007). The present results suggest that demonstrations of AB interference on the deployment of visual spatial attention reflect primarily interference on mechanisms of target-selection and encoding, rather than mechanisms of distractor suppression (see also Corriveau et al., 2012).

5. Experimental procedure

5.1. *Participants*

A total of 28 subjects participated in the study. After verification that lateral eye movements towards the target did not take place, 10 participants were removed. The 18 remaining participants (11 females and 7 males) had a mean age of 22.2 years (*S.E.* = 2.2, range 19–27). All participants were neurologically normal undergraduates at Université de Montréal and had normal colour vision, and either normal or corrected-to-normal visual acuity.

5.2. *Stimuli*

Each visual search frame consisted of six coloured or grey circles (1° radius), displayed at equidistant positions (90° and 270° or at 45°, 135°, 225°, or 315°) around an imaginary circle with a 3° radius from fixation (14.1 cd/m^2) (see Fig. 1). Each circle in the frame contained a grey bar (1.2° long by 2 pixels wide) that could be randomly displayed at one of four possible orientations, that is, horizontal, vertical, tilted to the left, or to the right. The orientation of the bar in a given circle position changed from frame to frame in the RSVP stream. All visual-search frames were presented on a black background.

Three types of frames were presented: Filler frames, T1 target frames, and T2 target frames. Filler frames were presented between, before, and after target frames. They consisted of six grey circles (14.1 cd/m^2), displayed as described above. T1 frames were physically identical to filler frames, with two exceptions. First, all circles were uniformly coloured in pink (14.1 cd/m^2) or desaturated green (14.5 cd/m^2), counterbalanced across subjects (see below). Second, all lines inside the circles composing the T1 frame had the same orientation. T2 frames were also physically identical to a filler frame, with one exception. In T2 frames, two circles were in a unique colour, either red (13.2 cd/m^2) or green (14.7 cd/m^2). For every T2 frame, one of these two coloured circles was on the vertical meridian (90° or 270°) whereas the other was lateral, in the right or the left visual hemifield. The central item could be presented either at a 12 o'clock with a lateral

item just below the centerline or at 6 o'clock with a lateral item just above the centre line.

5.3. Procedure

The colours for T2 targets and T2 distractors (red or green) were counterbalanced between subjects. The colour for T1 circles was determined according to target colour. If green was assigned as the target colour, all circles in T1 were a desaturated green colour. If the target colour was red, circles in T1 were pink. Target-distractor proximity was constant throughout the experiment. The distance between coloured circles was always of two circles. That is, there was always exactly one intervening grey item between the two coloured items, as illustrated in Fig. 1.

A practice block of 24 trials preceded the experiment. The actual experiment consisted of 672 experimental trials divided into eight blocks. Each trial contained 14–16 visual search frames presented sequentially without blank inter-stimulus intervals (ISIs), forming a rapid serial visual presentation (RSVP) stream, as illustrated in Fig. 1. Each visual search frame was shown for 233 ms.

The experimental design had three types of trials, presented an equal number of times, for every participant: (1) No-T1 trials, on which the T1 frame was omitted from the sequence; (2) short-SOA trials, on which the SOA between T1 and T2 was 466 ms (two frames, or lag2); (3) long-SOA trials, on which the SOA between T1 and T2 was 1165 ms (five frames, or lag5). The no-T1 trials served as a control that allowed us to measure the accuracy and the efficiency of visual spatial processing in Task 2 in the absence of T1. This type of control condition equates for preparation because participants do not know when T1 will, or will not, be presented, and hence they must prepare for both T1 and T2. When T1 is not presented, however, central attention mechanisms are not engaged by T1 and are thus available for processing T2. Given that these trials occur with high frequency (on 33.3% of the trials), they are not unusual, unexpected, or surprising. The long-SOA trials provide a second control in which T1 was presented, but sufficient time has elapsed to ensure that encoding of T1 is completed by the time T2 is presented (e.g., Jolicoeur, 1999). A reduction of Task 2 accuracy was thus expected on the short-SOA trials relative to no-T1 trials and long-SOA trials, and the latter two trial types were expected to produce similar results. Thus, on *a priori* grounds based on numerous published studies, we expected to compare performance, and electrophysiological results, for the short-SOA condition (AB, experimental condition) with the average of the two control conditions using contrast analyses (contrast weights of 1, −2, 1, for the no-T1, short-SOA, and long-SOA conditions, respectively).

Pressing the spacebar initiated a trial, which removed the feedback from the previous trial and displayed a fixation cross for 500±100 ms. A random number of frames (from five to nine) preceded the T1 frame. For the first task, participant had to identify the orientation of the bar common to all circles in the T1 frame through the characters "x," "c," "v," and "b" on a keyboard. These letters respectively represented the orientations tilted to the left, vertical, horizontal, or tilted to the right. Alternatively, in the absence of a T1 frame (in the no-T1 trials), participants were asked to press the spacebar. For the second task, the participant identified the orientation within one target circle in the T2 frame. Responses were made with a standard keyboard using characters "n," "m," "," and "." for the bars tilted to the left, vertical, horizontal, or tilted to the right. Two to six frames followed T2, ending the RSVP stream.

5.4. Electrophysiological recording and analyses

A BioSemi Active Two system (BioSemi Inc., Amsterdam, The Netherlands) was used for the recording of the EEG signal with 64 active Ag/AgCl electrodes mounted on an elastic cap and re-referenced to the algebraic mean of right and left mastoid signals. Electrodes were placed according to the 10–10 system (Sharbrough et al., 1991) 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 the voltage difference between electrodes placed lateral to the external canthi, which was used to measure horizontal eye movements. The vertical electrooculogram (VEOG), recorded the voltage difference between two electrodes placed above and below the left eye, was used to detect eye-blinks. Signals were recorded at a sampling frequency of 512 Hz from DC to 134 Hz. A bandpass filter of .05–30 Hz was applied during post-recording processing. Trials with an HEOG difference larger than 25 μV on a 200 ms interval suggested lateral eye movements toward the lateral stimulus, and were rejected. Trials with a variation of VEOG larger than 50 μV over a 200 ms interval were flagged as blinks and were removed. Trials with other types of artifacts (i.e., variation of more than 100 μV over an interval of 50 ms for a specific electrode) were also removed.

Our criterion for lateral eye movements resulted in the rejection of several participants, but this procedure was important for two reasons. The first one is theoretical. In order to assess solely attentional deployment, we needed to ensure that participants did not move their eyes towards the target, only their attention. The second reason was to avoid a confounding variable in our data analysis. Lateral eye movements towards a target could cause a contralateral negativity through volume conduction. A significant lateral eye movement could, through volume conduction, add to the observed N2pc and thus complicate the interpretation of the results. To confirm the necessity of our artefact-rejection measures, we also tried a more liberal approach, allowing a maximum of 40 μV over a 200 ms period. That criterion resulted in the loss of almost all participants when we later checked for the mean lateralized voltage at HEOG electrodes, which was over the 8 μV criterion, consistent with an eye movement of more than 1/2 of a degree toward the lateral stimulus. Indeed, our criterion allowed a remaining mean activity of a maximum of 8 μV between 200 and 700 ms post-stimulus per condition. That is to say that, after the removal of trials with a 25 μV difference on a 200 ms interval, the subtraction between the mean HEOG for eye movements to the left and the mean HEOG for eye movements to the right did not exceed 8 μV in

the remaining trial of every condition. Hence the mean remaining lateral movements were minimal (at most of 1/2 degrees for all conditions).

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