BRIEF REPORT

Spatial attention freezes during the attention blink

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Abstract

A variant of the rapid serial visual presentation paradigm was used to display sequentially two lateral sequences of stimuli, one to the left and one to the right of fixation, embedding two pairs of target stimuli, T1 and T2. T1 was composed of a pair of alphanumeric characters, and subjects had either to ignore T1 or to encode T1 for a delayed response. T2 was a lateral square of a prespecified color. The square had a small gap in one side, and the task for this stimulus was to report which side had the gap. When subjects were required to ignore T1, the T2-locked ERP produced a clear N2pc, that is, a greater negativity at electrode sites contralateral to the position occupied by T2. This N2pc was followed by a sustained posterior contralateral negativity (SPCN). When subjects were required to monitor T1 in addition to T2, both the N2pc and the SPCN components amplitude depended on the difficulty of the task associated with T1. If T1 was composed of digits that had to be encoded for a delayed same/different judgment, both the N2pc and the SPCN components were entirely suppressed. Although attenuated, such components were present when T1 was composed of a pair of symbols that subjects could disregard. The results suggest that a set of mechanisms subserving the allocation of attention in the spatial domain, resulting in the N2pc, suffer significant interference from concurrent cognitive operations required to encode information into visual short-term memory.

Descriptors: N2PC, Attentional blink, Control of spatial attention

In this article we examine the interactions between the control mechanisms subserving spatial attention and central attentional mechanisms. By central attention mechanisms, we mean mechanisms that operate at a postperceptual stage of processing and encompass response selection (Pashler, 1994), memory retrieval (Carrier & Pashler, 1995), short-term consolidation (Jolicœur & Dell’Acqua, 1998), and mental rotation (Band & Miller, 1997; Ruthruff, Miller, & Lachmann, 1995; Van Selst & Jolicœur, 1994), and that have been shown to impose large capacity demands when performed concurrently with other operations. Interactions between spatial and central attention mechanisms are suggested by the exacerbated attentional blink (Raymond, Shapiro, & Arnell, 1992) shown by neglect patients compared to neurological controls (e.g., Husain, Shapiro, Martin, & Kennard, 1997). These patients, in addition to reporting a difficulty in moving their attention to objects in the contralesional visual hemifield, tend often to miss the second of two sequential objects even when both are displayed at fixation and when more than 1200 ms elapses between their onsets.

Analogous indications come from behavioral tests in normals. When subjects are instructed to monitor a centrally displayed rapid serial visual presentation (RSVP) of characters followed by a visual array of eccentric Gabor patches, the efficiency of detecting a pop-out target (i.e., a misoriented patch) in this array is reduced during the attentional blink interval (Joseph, Chun, & Nakayama, 1997). Other research has shown that increases in central attention load during the attentional blink can lead to an increase in the degree to which spatially distributed distractors impact the processing of target information, arguing for interference in the control of spatial attention as a reflection of a perturbation at the level of central attention (Jiang & Chun, 2001). Recently, the above evidence has also been integrated with work showing that memory for spatial positions in a visual array can be disrupted during the attentional blink interval, as witnessed by the sizable reduction of visual marking effects when the positional markers are previewed during the attentional blink interval (Olivers, 2004). The behavioral tests carried out so far, however, leave open several possibilities concerning the specific type of interaction between spatial and central attention mechanisms. For example, in Joseph et al.’s work (1997), it is possible that attention could be deployed to the pop-out Gabor patch, but that this event could not be consolidated in visual short-term memory for later report, an explanation that potentially applies also to Olivers’ (2004) results. In Jiang and Chun’s (2001) study, it is possible that interference took place at late stages of processing (e.g., stimulus classification) rather than at the level of spatial attention.

Jolicœur, Sessa, Dell’Acqua, and Robitaille (2006) focused on interactions between central attention and visual spatial attention using the event-related potential (ERP) technique. In
their study, an attention shift was required to one of two lateral targets displayed during the attentional blink interval, and this attention shift was tracked by measuring the N2pc ERP component as subjects performed the task. The N2pc component (N2 posterior contralateral) is a lateralized ERP characterized by a greater negativity at posterior sites contralateral to the visual hemifield in which a to-be-processed target is displayed (Luck & Hillyard, 1994). As the term suggests, the N2pc component has a posterior scalp distribution, and it is estimated by computing the difference between the contralateral voltage and the ipsilateral voltage at corresponding symmetric electrode sites (e.g., P7/P8). The onset of the N2pc is usually at about 170–180 ms poststimulus, with a peak often between 220 and 250 ms and with potential latency variations owing to the difficulty in target localization (e.g., Wascher, 2005). This component seems to arise primarily from lateral portions of the extrastriate and inferior-temporal visual areas, with a possible contribution of posterior parietal areas (e.g., Hopf et al., 2000). Luck and his colleagues have provided evidence linking the N2pc to mechanisms of visual spatial attention. Several results led them to emphasize the potential role of distractor suppression in the generation of the N2pc (e.g., Luck & Hillyard, 1994). Others have argued that the N2pc may reflect processes of target enhancement rather than distractor suppression (e.g., Eimer, 1996).

Jolicœur et al. (2006) found an attenuation of the N2pc during the attentional blink, suggesting an influence on the N2pc originating relatively late in the flow of processing necessary to encode information in visual short-term memory (see also Cebolinder, Jolicœur, & McIwaine, 2002; Dell’Acqua, Jolicœur, Pesciarelli, Job, & Palomba, 2003; Jolicœur & Dell’Acqua, 1998). Interestingly, the N2pc suppression was accompanied by an attenuation of a lateralized component that often follows the N2pc, the sustained posterior contralateral negativity (SPCN). Strong evidence suggests that the SPCN component reflects neural activity specifically related to the maintenance of information in visual short-term memory (e.g., Klaver, Talsma, Wijers, Heinze, & Mulder, 1999; Vogel & Machizawa, 2004). In this vein, Jolicœur et al. (2006) argued that a failure to encode visual information in their lateralized attentional blink task was due to a failure to allocate attention to the spatial position occupied by target information, causing the N2pc attenuation, which in turn was reflected by the absence of information in visual short-term memory, causing the SPCN attenuation.

In the Jolicœur et al. (2006) paradigm, T1 was presented at fixation whereas T2 (i.e., the eccentric target) was presented 3° to the left or right of fixation. One interpretation of these results was that spatial attention could not disengage from the position in which T1 was encoded during the consolidation of T1 in visual short-term memory. To rule out this possibility, we used a variant of the original paradigm that was designed to discourage subjects from focusing their attention to any particular spatial positions prior to T2. We used two laterally displayed and synchronized RSVP streams of stimuli, in which T1 was composed of two simultaneous alphanumeric characters, one in each RSVP stream. T1 was then followed by the same pair of stimuli as those used in our earlier work, that is, two colored squares each having a small gap in one side whose position had to be reported later, with no speed pressure. To maximize the number of trials contributing to the ERP generation, the stimulus onset asynchrony (SOA) between T1 and T2 was fixed at 250 ms, an SOA at which the attentional blink normally peaks (e.g., Chun & Potter, 1995).

Method

Participants

The participants were 30 undergraduate students (15 women and 15 men) of the University of Padova who participated for course credit. All participants reported normal or corrected-to-normal vision, normal color vision, and no history of prior neurological disorders. The mean age was 22 years.

Stimuli

The stimuli (see Figure 1) were light gray alphanumeric characters (all capital letters, except B, O, I, and the digits 2 to 8) and red or green squares, displayed on the dark gray background of a cathode-ray tube computer monitor. The luminance of the light gray, red, and green colors was adjusted using a Minolta luminance meter to be the same (25 cd/m²). The luminance of the dark gray of the background was 8 cd/m². The stimuli were presented in two synchronized RSVP sequences that were 3° to the left and to the right of fixation. Participants sat at a distance of approximately 70 cm from the monitor. All characters subtended 1° in height and 0.8° in width, and the sides of the squares were 1°.

Procedure

Each trial began with a centrally presented plus sign. Participants initiated the trial sequence by pressing the spacebar on the computer keyboard. A fixed interval of 800 ms elapsed prior to the beginning of two concurrent RSVP sequences of stimuli. A variable number of letters (two to nine) preceded the onset of T1, which was composed of a pair of digits on 50% of trials. In the other trials, the digits were replaced with “=” signs. When T1 was composed of digits, the digits were the same on 50% of trials (i.e., 25% of total) or different in the other trials. T1 was then followed by a pair of letters, masking the T1 characters. After a blank interval of 50 ms, a frame including two squares, one green and one red, was displayed. Each of these squares had a small gap in one randomly chosen side. T2 was the red square for half of the participants, and the green one for the others. The pair of red/green squares was followed by a pair of light gray squares with a gap in all four sides, to mask the gap-location information in the colored squares. Following the masking squares, an interval of 800 ms preceded the presentation of one question or two suc-
The subjects were assigned at random to one of two groups of equal size (N = 15). Subjects in the dual-task group monitored the two concurrent RSVP streams of alphanumeric characters for both T1 and T2. As illustrated in Figure 1, the T2 frame was always presented at the end of the RSVP sequence, and was immediately followed by pattern masks. When T1 was a pair of digits, participants had to decide whether they were the same or different. They reported their decision at the end of the trial, without speed pressure, by pressing one of two keys ("C" for same and "V" for different). If T1 was a pair of " = " signs, they pressed the space bar. These responses were produced using the left hand. In addition, participants had to determine whether the T2 square had a gap in the top, bottom, left, or right side. They used the right hand and pressed one among the "2," "4," "6," and "8" keys on the numeric keypad to report the position of the gap using a spatially compatible mapping. Participants in the single-task group were instructed to ignore all stimuli except the T2 square and to report the position of the gap as just described. Each participant performed one block of 24 practice trials, and eight blocks of 48 experimental trials.

**EEG/ERP**

Using a head-cap with tin electrodes, EEG activity was recorded continuously from the Fp1, Fp2, Fz, F3, F4, F7, F8, C3, C4, Cz, P3, P4, Pz, O1, O2, T7, T8, P7, and P8 sites (see Pivik et al., 1993), referenced to the left earlobe. Horizontal EOG (HEOG) was recorded bipolarly from electrodes positioned on the outer canthi of both eyes. Vertical EOG (VEOG) was recorded bipo larly from two electrodes, above and below the left eye. EEG, HEOG, and VEOG activities were amplified, filtered using a bandpass of 0.01–80 Hz, and digitized at a sampling rate of 250 Hz. Impedance at each electrode was maintained below 5 kΩ. The EEG was re-referenced off-line to the average of the left and right earlobes and segmented into 1200-ms epochs starting from 200 ms prior to the onset of T2. Trials associated with a HEOG exceeding ± 30 μV were discarded from analysis. Trials associated with eyeblinks or any other artifact (electrode activity exceeding ± 80 μV) were also discarded from analysis. The above criteria led to the rejection of 17% of trials. Signal-averaged HEOG was used as control for possible eye movements toward T2. The difference between left and right HEOG electrodes was averaged separately for trials in which T2 was displayed to the right and to the left of the central fixation point. A maximum deflection of less than 3 μV was observed in any given participant, suggesting that the average eye position did not deviate by more than 0.2° toward T2 during the ERP epoch (for a review, see Luck, 2005). Lateralized activity was monitored at posterior sites (i.e., O1/O2, P7/P8, P3/P4). For each electrode pair, the ERP contralateral to T2 was calculated by averaging the ERP generated at the left-sided electrode when T2 was displayed in the right visual hemifield and the ERP generated at the right-sided electrode when T2 was displayed in the left visual hemifield. The ERP ipsilateral to T2 was calculated with an analogous algorithm, by averaging the ERPs at the complementary sites.

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1Note that the present algorithm for the generation of T2-locked contralateral and ipsilateral ERPs, in combination with the specific instructions on how to respond to the stimuli at the end of each trial (i.e., to always use the right hand to respond to T2), exclude motor-related activity from the possible modulatory sources of EEG activity at N2pc-related sites of interest. Although motor-related activity would be systematically related to the hand of response (e.g., the lateralized readiness potential), such activity would not systematically vary as a function of the side of presentation of the target. Furthermore, the responses in the task were not speeded and occurred about 1.5 s or more after the end of the RSVP stream, whereas the N2pc occurred within 300 ms of the presentation of T2.
task condition compared to the single-task condition. Furthermore, there was a clear tendency of the P3 component to be relatively attenuated under conditions in which T2 was missed more often (i.e., in T1 digit trials of the dual-task condition; see Vogel, Luck, & Shapiro, 1998). The negative deflection at about 900 ms post-T2 onset was probably a reflection of the onset of the written questions on the screen prompting subjects to enter a response to the target stimuli. Focusing more narrowly on the pattern investigated in the present context, the N2pc was larger in the single-task condition than in the dual-task condition, \( F(1,28) = 5.6, p < .02 \). The N2pc was also larger with T1 ‘’=’’ signs than with T1 digits, \( F(1,28) = 7.5, p < .01 \). Most importantly, however, the two factors interacted significantly, \( F(1,28) = 6.8, p < .02 \), providing statistical support to what is evident in Figure 2, namely, that the N2pc was completely suppressed in the dual-task condition when T1 consisted of digits, but remained present when T1 consisted of ‘’=’’ signs. When T1 consisted of ‘’=’’ signs, the N2pc was significantly attenuated in the dual-task condition relative to the single-task condition, \( t(13) = 2.3, p < .05 \). In the dual-task condition, separate \( t \) tests revealed null lateralized T2-locked activity following T1 digits, \( t < 1 \). In contrast, the N2pc amplitude was significantly different from zero in the dual-task condition following T1 ‘’=’’ signs, \( t(14) = 3.6, p < .003 \). Finally, the N2pc was equally large for the two T1 stimulus types in the single-task group, \( t < 1 \), showing that differences in the dual-task group could not be attributed to differences in stimulus materials in the different T1 conditions.

For the SPCN, the patterns of results were strikingly similar to those for the N2pc. The amplitude of the SPCN was larger in the single-task condition than in the dual-task condition, \( F(1,28) = 9.2, p < .005 \), and larger with T1 ‘’=’’ signs than with T1 digits, \( F(1,28) = 18.1, p < .001 \). Again, the two factors interacted, \( F(1,28) = 4.362, p < .05 \). Separate \( t \) tests carried out in the dual-task condition revealed null lateralized T2-locked activity following T1 digits, \( t < 1 \). In contrast, the N2pc amplitude was significantly different from zero in the dual-task condition following T1 ‘’=’’ signs, \( t(13) = 2.839, p < .015 \). The amplitude of the SPCN did not differ across T1 conditions (digits vs. ‘’=’’

Figure 2. Results of the experiment. Top: ERP results based on activity recorded from the P7/P8 sites. The grand-average ERPs were generated by including all artifact-free epochs, including both trials with correct and incorrect behavioral performance (see text for details). Left: Contralateral and ipsilateral ERPs to T2 in the single-task condition. Right: Contralateral and ipsilateral ERPs to T2 in the dual-task condition. Bottom: Behavioral performance. Proportion of correct responses to T2 in each condition.

Figure 3. ERP results. Difference (contralateral minus ipsilateral) waveforms at selected electrode pairs, time-locked to the onset of T2, as a function of the different conditions of the present experiments. Data are plotted as a function of task (single-task vs. dual-task) and as a function of T1 stimuli (high-load digits vs. low-load ‘’=’’ signs).
Discussion

The present results were clear-cut. Under single-task conditions, the requirement to shift attention to a target item indicated by color produced clear N2pc and SPCN components in the ERP time-locked to T2 onset. Under dual-task conditions, the N2pc and the SPCN components were still apparent, though attenuated, when T1 was composed of to-be-disregarded “=” signs. Most importantly, however, both the N2pc and SPCN components were entirely suppressed when T1 was composed of digits that had to be encoded for a delayed same/different judgment. Thus, the N2pc and SPCN followed closely the patterns observed in the behavioral results, which exhibited a classic attentional blink pattern reflecting worse accuracy for T2 under increased processing loads associated with the T1 task. These behavioral results dovetailed nicely with previous research showing reduced costs associated with the processing of T1 stimuli that can be discarded (the “=” signs; see Jolicœur & Dell’Acqua, 2000).

Most importantly, the present results have implications for our understanding of the mechanisms that control the deployment of spatial attention mechanisms leading to the N2pc. In the single-task condition, subjects presumably prepared to shift attention to the left or right in order to process the lateralized T2 target square. An optimal strategy would be to adopt an initially diffused attentional state capable of monitoring both possible locations for T2 and shifting attention to the correct location upon detection of the target color. The large N2pc responses observed in the single-task condition confirm that subjects were able to do so.

The dual-task condition in this paradigm was designed also to encourage observers to adopt an initially diffused state of attention, due to the need to monitor both RSVP streams for T1. Indeed, observers had to monitor exactly the same locations required to select the T2 square. The fact that N2pc and SPCN were increasingly reduced under increasing processing demands at these same spatial locations prior to the presentation of T2 suggests that the task demands associated with processing T1 prevented a shift from an initially diffused attentional state to the more focused state required for the gap localization task associated with T2 (given that identical stimuli when T1 was not associated with a processing task produced much better behavioral performance for T2 and larger N2pc amplitudes).

We propose that processing a bilateral T1 impeded the more focused deployment of visual spatial attention to T2. The results therefore suggest that the mechanisms required to encode and process visual information, which have been argued to engage limited-capacity central processing mechanisms (e.g., Jolicœur & Dell’Acqua, 1998), overlap with those that control the deployment of visuo-spatial attention.

Our favored interpretation of the SPCN component is that it likely reflects neural activity associated with maintenance of information in visual short-term memory (Jolicœur, Sessa, Dell’Acqua, and Robitaille, 2005; Jolicœur et al. 2006; Klaver et al., 1999; McCollough, Machizawa, & Vogel, 2006; Vogel & Machizawa, 2004). Several results allow us to associate the SPCN to this specific process. First, the amplitude of the SPCN increases as the number of items to be held in visual-short term memory increases, but only up to the number of items that a given subject can reliably remember. That is, the SPCN grows in amplitude as items to be remembered are added to the display, up to the span of visual short-term memory. The duration of the component in the experiments of Vogel and his colleagues is directly tied to the duration of the retention interval. We have observed that the duration of the SPCN in experiments that are not explicitly designed as memory experiments varies with response time when the task requires a speeded response, with a longer component duration found in conditions that cause longer response times (Robitaille & Jolicœur, 2006). A task analysis for these latter studies supports the view that conditions that produced longer response times also required a longer duration of processing of the visual memory trace encoded from a briefly presented stimulus. The very posterior and contralateral scalp distribution of the SPCN links the neural activity to the location of the visual stimuli to be encoded and processed in a way that is consistent with ongoing activity in extrastriate visual cortex with possible involvement of posterior parietal cortex (McCollough et al., 2006). Thus, we interpret the concomitant modulations of the SPCN as suggestive that a visual representation of T2 could not be transferred to visual short-term memory when spatial attention could not be focused on T2. In this view, the results provide support for the idea that transfer to visual short-term memory is a crucial processing step in the attentional blink paradigm (as opposed to transfer to a more verbal or abstract form of short-term memory, which would not be expected to produce posterior brain activity strongly lateralized as a function of target position), as well as in other types of paradigm (e.g., Eimer & Mazza, 2005).

We acknowledge, however, that we cannot rule out another interpretation of the SPCN as a continuation of the N2pc, although we cannot think of a reason why subjects would want to keep spatial attention focused at the former location of T2 once T2 was masked and removed from the screen. With these considerations and other work on the SPCN reviewed above in mind, our preferred interpretation for the SPCN in the present work remains an indication of the involvement of visual short-term memory, in which case both spatial attention (N2pc) and visual short-term memory (SPCN) are affected by the attentional blink.

We turn now to the partial or total attenuation of the N2pc and SPCN observed in the dual-task condition. Was this attenuation produced by a graded reduction in the amplitude of the components or rather the result of a mixture of trials in which the components had a full amplitude and trials in which the components were fully attenuated? In the former case, one might interpret the results as a reduction in the efficiency of the allocation of attention to T2 on all trials, whereas in the latter (especially in the case of trials with T1 composed of “=” signs) we would conceptualize the result as a probability mixture of success and failure in the allocation of attention. The results of the analysis produced by dividing the trials into two bins based on success in the task on T2 is reported in Figure 4. It must be anticipated that the substantial reduction of trials in the cells of the analysis prevented us from obtaining statistically significant results (which is not entirely unexpected, given that we designed the experiment to have sufficient power to measure differences across conditions on the basis of all trials in each condition).

However, with all due caution, Figure 4 inspires a series of considerations concerning the point presently at stake. Succinctly, when T1 was composed of “=” signs, there was evidence of increased contralateral negativity whether or not T2 was correctly responded to. On the other hand, when T1 was composed
of digits, increased contralateral negativity seemed to characterize more consistently the trials associated with the correct report of T2 than trials in which T2 was missed. Speculatively, an interpretation of this pattern may be that T1-related cognitive load had an impact on the ability of subjects to move their attention to the to-be-attended location that was radical. With an increased cognitive load (i.e., with T1 digits), the allocation of attention was likely to be impacted on most trials, apart from the few in which attention moved successfully and T2 was detected, as suggested by evidence of increased N2pc amplitude in trials associated with a correct T2 relative to trials in which T2 was missed. With a cognitive load relatively attenuated (as when T1 was composed of ‘‘=’’ signs) instead, attention movement efficiency was increased, as is witnessed by an N2pc component that was generally larger in this condition compared to the condition with T1 digits. Interestingly, the N2pc does not appear to be modulated by the success in reporting T2, and this brings to the fore the argument of the complex relationship between the N2pc component and conscious report. Compare, for instance, the present results with the results of Woodman and Luck (2003), who presented two lateralized geometric shapes that were masked with four dots (e.g., Dell’Acqua, Pascali, Jolicœur, & Sessa, 2003; Di Lollo, Enns, & Rensink, 2000). The instructions given to subjects in Woodman and Luck’s experiment were to report, with no speed pressure at the end of each trial, whether either masked shape was a triangle. The EEG was recorded during the experiment, and a method analogous to that used in the present context was adopted to isolate the N2pc component timed-locked to the onset of the pair of shapes. The behavioral results indicated that subjects were basically at chance in the report task. Nevertheless, when a triangle was one of the two shapes, an N2pc (more negativity contralateral to the triangle) was clearly detected. These results are crucial to establish the relative independence of the target-locked N2pc component from the target explicit report. Furthermore, this allows us to argue with even more confidence that, had the attentional blink exerted a null influence on the control of spatial attention, our design would have been nonetheless sensitive to N2pc variations (and, possibly, to SPCN variations) even on trials associated with no explicit report of T2.

One line of evidence that, over and above the considerations included in the previous paragraph, may be raised to speculate about the sources of the intermediate result obtained with ‘‘=’’ signs in dual-task trials is the recent evidence produced by Sergent, Baille, and Dehaene (2005), who integrated the standard behavioral variable monitored in RSVP designs (i.e., success in reporting target information) with a procedure aimed at estimating the subjective visibility of targets embedded in RSVP streams. The logic in this study was to compare the binary outcome associated with the first type of dependent variable (T2 reported vs. T2 not reported) with a more continuous (on a 100-point scale) estimate of the “visibility” of T2 in the attentional blink. Interestingly, the rate of subjective visibility of T2 and the rate of report were almost perfectly correlated. The rate of visibility was bimodally distributed, and, more importantly, the modes coincided with the extremes of the scale of visibility, suggesting that the attentional blink produced in the majority of cases a dichotomous outcome: T2 was either seen and reported or T2 was lost radically, as the subjective rating of visibility suggested. If one incorporates the results obtained by Sergent et al. (2005) into the theoretical framework we are currently developing, probably the most viable interpretation of the reduction of N2pc amplitude under easy (‘‘=’’ signs) dual-task conditions is that of a combination of fully sized N2pc and SPCN components when T2 was correctly reported and suppressed N2pc and SPCN component when T2 was missed.

REFERENCES


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