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On the control of visual spatial attention: evidence from human electrophysiology

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Abstract We used electrophysiological methods to track the deployment of visual spatial attention while observers were engaged in concurrent central attentional processing, using a variant of the attentional blink paradigm. Two visual targets (T_1 , T_2) were presented at a stimulus onset asynchrony of either 200 ms or 800 ms. T_1 was a white digit among white letters presented on a dark background using rapid serial visual presentation at fixation. T_2 was another digit that was presented to the left or right of fixation simultaneously with a distractor digit in the opposite visual field, each followed by a pattern mask. In each T_2 display, one digit was red and one was green. Half of the subjects reported the red digit and ignored the green one, whereas the other half reported the green digit and ignored the red one. T_1 and T_2 were reported in one block of trials, and only T_2 in another block (order counterbalanced across subjects). Accuracy of report of T_2 was lower at short SOA than at long SOA when both T_1 and T_2 were reported, but was similar across SOA when only T_2 was reported. The electrophysiological results focused on the N2pc component, which was used as an index of the locus of spatial attention. N2pc was reduced in amplitude when subjects reported T_1 , and particularly so at the short SOA. The results suggest that attention to T_1 interfered with the deployment of visual spatial attention to T_2 .

Introduction

The goal of the present work was to study capacity limitations in the mechanisms involved in the control of visual spatial attention. Attentional selection is thought to be necessary because capacity limitations in later stages of processing make it impossible to process all of the information available in the visual array (Pinker, 1984; Treisma & Gelade, 1980; Sperling, 1960). A subset of the information can be selected based on spatial position, per se, and/or on a search for particular attributes of the stimuli themselves (Posner, 1980; Sperling, 1960; Von Wright, 1972).

The selection of a visual target for further processing is often associated with an electrophysiological response, often called the N2pc, that has been studied extensively by several researchers, and most particularly by Luck and his colleagues (e.g. Eimer, 1996; Girelli & Luck, 1997; Hopf, Boelsman, Schoenfeld, Heinze, & Luck, 2002; Jolicœur, Sessa, Dell'Acqua, & Robitaille, 2005; Luck, Girelli, McDermott, & Ford, 1997; Luck & Hillyard, 1994; Woodman & Luck, 2003). The N2pc event-related potential (ERP) is a lateralized response characterized by a greater negativity over the hemisphere contralateral to the visual field of a target relative to the response over the hemisphere ipsilateral to the target. The N2pc difference waveform, computed by subtracting the ipsilateral from the contralateral responses of corresponding electrode pairs (e.g. T5, T6; O1, O2; Jasper, 1958) for lateralized visual targets, is usually found from about 180 to 280 ms post target onset and is maximal at posterior electrode sites (near the midpoint between O1 and T5, or O2 and T6; see, e.g. Luck & Hillyard, 1994).

We used the N2pc as a moment-to-moment index of the deployment of visual spatial attention (Woodman & Luck, 2003), in the context of an attentional blink paradigm. This combination of different paradigms allowed us to study the relationship between the mechanisms that mediate visual spatial attention and

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those that mediate central attentional operations. By central attention we mean operations that are postperceptual 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.

A dissociation between these mechanisms has sometimes been proposed (Johnston, McCann, & Remington, 1995). In this view, early spatial selection mechanisms act as a gate or filter through which selected information can flow to later central mechanisms and by which information not selected is not processed further. Later, central mechanisms, receive the selected information and perform different forms of processing (such as the selection of an appropriate response, and/or encoding the information into memory). These mechanisms have sometimes been hypothesized to operate sequentially and essentially independently (Johnston et al., 1995). The locus of spatial attention was influenced, in Johnston et al. (1995), by means of a peripheral flash of light. This methodology likely attracted attention exogenously, and this might account for the apparent independence between spatial and central attention suggested by the results of Johnston et al. (1995). In our work, we focused on a form of selection that was more likely to involve endogenous control of attention. We will discuss this issue at greater length in the Discussion.

Other work suggests that spatial selection and later control mechanisms may interact. The degree to which a target involuntarily captures attention appears to depend on attentional control settings selected by the observer. For example, an observer expecting to detect a uniquely coloured target (e.g. red) presented in a rapid sequence of stimuli in other colours, at fixation, will be significantly distracted (attention capture) by a stimulus presented in the periphery if that stimulus matches the colour of the target (e.g. red) but not if the stimulus is in another colour (e.g. green; Folk, Leber, & Egeth, 2002), a result which we replicated and extended in our laboratory (Leblanc & Jolicœur, 2005). Such results demonstrate that attention control settings can exert a top-down influence on the degree to which bottom-up signals can capture spatial attention.

In addition, other research has shown that increases in central attentional load can lead to an increase in the degree to which distracting stimuli interfere with processing of a target (de Fockert, Rees, Frith, & Lavie, 2001; Jiang & Chun, 2001). These authors have argued that spatial selection is impaired when central attention is engaged on a concurrent task. This conclusion regarding spatial attention is somewhat indirect, however, because intruding information from distractors could result from a loss of control at other, later, stages of processing (e.g. response selection).

In the present work we sought to provide a more direct test of the dependence of the control of spatial attention on central attentional mechanisms. We used the N2pc to index where and when subjects allocated spatial attention while they performed a task requiring concurrent central processing known to cause an attentional blink (Jolicœur, 1999a; Vogel, Luck, & Shapiro, 1998).

We modified the attentional blink procedure as illustrated in Fig. 1. In the initial portion of each trial, subjects viewed a sequence of distractor letters at fixation presented at a rate of 10 items per second. One of the letters in the sequence was replaced by a digit, and this was the first target, or T_1 . The degree of central processing involvement was manipulated by instructing subjects either to encode and remember this digit, or to ignore it. The second target, or T_2 , was presented following T_1 after either one additional item in the central stream (lag 2, at a stimulus onset asynchrony, or SOA, of 200 ms, illustrated in Fig. 1) or after seven additional items (lag 8, SOA of 800 ms). T_2 was also a digit and it was presented either to the left or right of fixation, and was either red (for half of the subjects) or green (for the others). Another digit was presented concurrently with T_2 on the other side of fixation, and in the other colour (e.g. in green if the target was red). The two digits in the T_2 time frame were followed by a bilateral mask (the letter W) to ensure that we would observe an attentional blink (Giesbrecht & Di Lollo, 1998; Jolicœur, 1999b).

The display containing T_2 also contained a lateral distractor (another digit, positioned symmetrically about fixation, relative to T_2) for two reasons. The first was to equate the low-level sensory response of the T_2 display across the left and right hemispheres. Had we

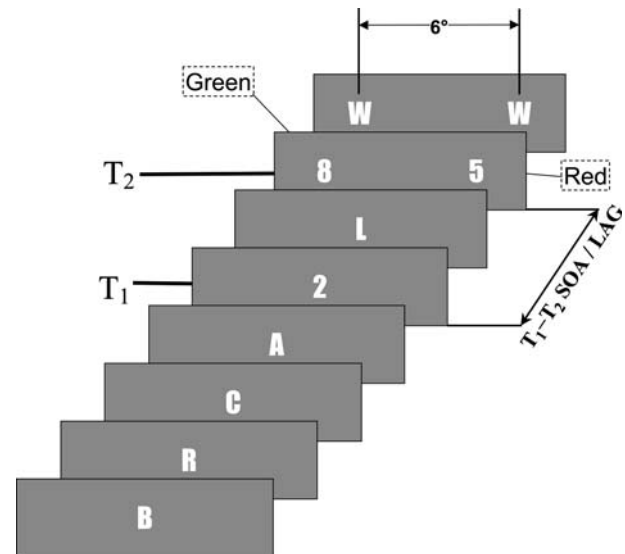


Fig. 1 Modified attentional blink paradigm used in the experiment. T_1 was a white digit presented at the centre, followed by 1 distractor. T_2 was red (half of the subjects) or green, presented 3° to the left or right of fixation, and followed by a pattern mask. T_1 was followed by 1 letter at fixation in the lag 2 condition (shown here) and by 7 letters in the lag 8 condition (not shown)

presented T_2 by itself (without a concurrent distractor), then large interhemispheric differences would have been observed at posterior electrode sites because of the structure of the visual system. Stimuli presented in the left visual field project initially only to the right hemisphere, whereas stimuli in the right visual field project initially only to the left hemisphere, in primary visual cortex (e.g. Coren, Ward, & Enns, 1994; Zeki, 1993). ERPs to a single stimulus in the left or right visual field would thus have produced large interhemispheric differences (e.g. Bayard, Gosselin, Robert, & Lassonde, 2004; Luck & Hillyard, 1994), making it more difficult to distinguish attentional effects from purely low-level sensory effects. The presence of an equivalent equi-luminant form in each hemisphere produces a balanced electrophysiological response of early visual cortex across the two hemispheres (counterbalancing eliminates any small residual differences). Differences in ERPs across the hemispheres for such displays can only arise as a function of differential processing due to attentional selection of one of the two stimuli (see Luck & Hillyard, 1994; Woodman & Luck, 2003, for further discussion).

The second reason to use a digit as a lateral distractor was to increase the probability that subjects would deploy visual spatial attention to T_2 by making the use of a late-selection strategy less useful. In order to give the correct response, subjects had to select the correct digit, namely the one in the target colour. If the display contained only one digit (say, paired with a letter), then subjects may have been able to perform the task without deploying visual spatial attention to the target location. They may have been able to use differences in semantic activation to perform the task (see Duncan, 1980, 1983). Although, prior work showed that an N2pc could be observed even when a single alphanumeric character is used with a nonletter distractor (e.g. Eimer, 1996), the presence of two digits in the display was meant to encourage subjects to use colour to guide visual spatial attention to one of them. Doing so would then engage differential processing at the location of T_2 , which should lead to a greater contralateral negativity at posterior electrode sites, thus producing an N2pc ERP.

The logic of the design was as follows. In the report- T_1 condition, encoding T_1 should occupy central mechanisms and this should produce the conditions required to observe an attentional blink (e.g. Jiang & Chun, 2001; Jolicœur, 1999a; Vogel et al., 1998). When T_2 is presented at lag 2, only a short time after T_1 , some aspect of the processing of T_2 suffers because a central mechanism or capacity is occupied by the ongoing processing of T_1 (Jolicœur, 1999a). When T_2 is presented at lag 8, and thus relatively long after T_1 , the encoding of T_1 should be completed and any interference on T_2 would likely be attributable to the load of maintaining a memory representation of T_1 until the end of the trial (Jolicœur & Dell'Acqua, 1998).

Trial blocks in which T_1 can be ignored provide a control condition with identical physical stimulation as in the report- T_1 condition, but without the processing

costs associated with the encoding and retention of a representation of T_1 (Jolicœur, 1999a; Raymond, Shapiro, & Arnell, 1992).

In the control condition (ignore- T_1), we expected to observe an N2pc both in the lag 2 and the lag 8 conditions, each of these conditions providing a baseline measure against which to compare the N2pc observed at the same lags, but when T_1 was reported rather than ignored. The amplitude (and/or latency) of the N2pc in the report- T_1 condition was thus of critical interest. If visual spatial attention can be deployed without interference from concurrent processing of T_1 , then the N2pc waveforms in the report- T_1 condition should be the same as that observed in the ignore- T_1 condition, and have the same lag effects as those in the ignore- T_1 trials. If, on the other hand, the attentional blink interferes with spatial attention, then the N2pc should be attenuated in report- T_1 trials, and particularly so at lag 2, when the attentional blink effect should be maximal. N2pc should return to 'normal' at lag 8, because processing T_2 should no longer be affected by concurrent processing of T_1 (Chun & Potter, 1995; Jolicœur, 1998, 1999a, b).

Another way to view the present experimental design is that it involves encoding T_1 , at fixation, and then requires a shift of the spatial locus of attention to a laterally presented T_2 . In number of previous behavioural experiments (e.g. reviewed in Visser, Bischof, & Di Lollo, 1999), it was found that the attentional blink is generally not influenced by changes in spatial location of T_2 relative to T_1 , beyond lag 1. That is, a change in spatial location across T_1 and T_2 has a large effect at lag 1 (indeed, reliably abolishing the lag-1 sparing effect), but produces results that are essentially the same as in performance without a change in spatial location beyond lag 1. Lag-1 sparing is said to occur when accuracy in Task₂ is significantly higher at lag 1 than at lag 2 (Visser et al., 1999). Based on this behavioural evidence, spatial capture by T_1 at fixation should not prevent the redeployment of attention to T_2 under with the present temporal parameters. This issue was explored further by Jolicœur et al. (2005). They presented T_1 in the same colour as the target colour for the T_2 . Under these conditions, a large AB was found even when instructions were to ignore T_1 (Folk et al., 2002), suggesting that the intention to process a peripheral target of a particular colour could trigger attentional capture by a target at fixation. In the present experiment T_1 was white and thus in the same colour as distractors, which prevented attentional capture on the basis of a similarity between T_1 and T_2 in terms of the colour used to select T_2 . This issue is discussed further in the Discussion section.

Method

Subjects

The subjects were 16 undergraduates at the Université de Montréal who participated for pay. All had normal or

corrected-to-normal vision, all reported having normal colour vision, and none suffered from neurological conditions known to affect electroencephalographic responses.

Stimuli

The targets, T_1 and T_2 , were the digits 2–9, presented among upper-case letters. The characters were about 1° of visual angle in height. Each character was presented for 100 ms, and each character was replaced by the next with no blank ISI. Figure 1 illustrates the sequence of events in each trial. Characters were presented at fixation during the initial portion of the trial, and 3° to the left and right of fixation when T_2 was presented, during the final portion of the trial. The background was black and the stimuli at fixation were light gray. T_2 was red for half of the subjects and green for the others. A distractor digit was presented in the visual field contralateral to T_2 , in the other colour (i.e., green if T_2 was red, or vice versa). The luminance of the red, green, and light gray were adjusted to be approximately equiluminant using a Minolta CS100 chromameter.

T_1 was followed by 1 letter when the lag between T_1 and T_2 was 2 and by 7 letters when the lag was 8, at the centre of the screen. This ensured that the stimulation events just before the onset of T_2 were equivalent across the two lag conditions (2 vs 8). In both cases, characters were presented for 100 ms each, until just before the onset of T_2 , which coincided with the offset of the last character in the central rapid serial visual presentation (RSVP) stream.

Procedure

Each trial was initiated by the subject by pressing the space bar. T_1 was always followed by at least one additional item in the central RSVP stream. Each subject performed two blocks of trials that had identical stimuli, but differed in terms of the task to be performed for T_1 . In the ignore- T_1 block, the instructions were to ignore T_1 and to report the identity of T_2 . In the report- T_1 block, the instructions were to report both the identity of T_1 and the identity of T_2 , in that order. Responses were not speeded and were made using the numeric keypad using the right hand, after the presentation sequence. Subjects were instructed, and trained, to respond using the numeric keypad without moving their eyes (i.e., without looking at their hand), in order to minimize EEG ocular artifacts. Responses were scored for accuracy and had to be in the correct order to be scored as correct.

For half of the subjects, T_2 was the red digit in the T_2 frame, and T_2 was the green digit for the others. T_2 was followed by a bilateral pattern mask (the letter W), for 100 ms.

The attend to T_1 versus ignore T_1 manipulation was performed within subjects, across different blocks of

trials (counterbalanced for order across subjects). Each subject performed 800 trials (384 experimental trials preceded by 16 practice trials in each block). T_2 was presented equally often to the left and right of fixation, at lags 2 and 8 relative to T_1 .

EEG was recorded continuously while 16 neurologically normal university students performed the attentional blink task, using a Neuroscan system with Ag/Ag–Cl electrodes positioned at the following sites, named using the International 10/20 system (Jasper, 1958): C3, C4, CP3, CP4, CPz, Cz, F3, F4, F7, F8, FC3, FC4, FCz, Fp1, Fp2, FT7, FT8, Fz, O1, O2, Oz, P3, P4, Pz, T3, T4, T5, T6, TP7, and TP8. The sampling rate was 250 Hz and signals were high-passed filtered at 0.05 Hz and low-passed filtered at 80 Hz and referenced to the average of left and right mastoids during the recording. Electrode impedance was kept below 5 K Ω . The electrooculogram was recorded using pair of electrodes located lateral to the left and right eyes, to monitor horizontal eye movements (HEOG), and above and below the left eye, to monitor vertical eye movements and eyeblinks (VEOG). One set of analyses was performed after artifact rejection performed by a trained medical electrophysiologist, who examined the EEG for each trial. Another set was performed following a semi-automated artifact rejection technique. Both analyses produced essentially the same results. Results reported in this article were based on the semi-automated procedure, which is more easily reproduced. Blinks and vertical eye movements were screened by taking the difference between the two VEOG electrodes and looking for voltage differences in excess of 100 mV in any 400 ms window and marking as bad the segment of EEG that included this window, plus 150 ms before and 250 ms after the window when the criterion was exceeded. Horizontal eye movements were screened by rejecting any portion of the continuous EEG in which the HEOG difference was less than -38 mV or greater than $+38$ mV including the portion 250 ms before and 250 ms after segments that exceeded the ± 38 mV criterion. On average, about 20% of the trials were rejected due to ocular artifacts. We assumed that ocular artifacts were constant across conditions, and behavioural and electrophysiological data were analyzed separately (i.e. we did not remove trials with ocular artefacts from behavioural analyses).

EEG was segmented based on the presentation of T_2 , with a 200 ms prestimulus baseline and a 500 ms post-stimulus period.

In addition, we used signal-averaged HEOG recordings as a further very sensitive control over possible eye movements toward T_2 . The difference between left and right HEOG electrodes was averaged separately for trials in which T_2 was to the left of fixation and for trials in which T_2 was to the right. Systematic investigations of this bipolar HEOG response have shown that the voltage difference is a linear function of the size of an eye movement over at least a 15° range, and that the voltage difference at electrodes located immediately adjacent to

the two eyes is approximately 16 μV per degree of eye movement (Hillyard & Galambos, 1970; Lins, Picton, Berg, & Scherg, 1993; McDonald & Ward, 1999; see Luck, 2005, for a review). We observed that the maximum deflection toward the target was less than 3 μV for any given subject, which means that the average eye position did not deviate more than 0.2° toward the target for any subject. The N2pc was measured at lateralized posterior electrodes (O1/O2, P3/P4, T5/T6) in a measurement window of 190–290 ms.

Results

Consider first the accuracy of report of the identity of T_2 , for each lag and each T_1 condition (ignore- T_1 vs report- T_1). These analyses were performed without conditionalizing performance in Task₂ on accuracy in Task₁. We performed such conditional analyses, for the report- T_1 condition, by including only trials in which a correct response was produced in Task₁, and found the results to be largely unchanged (mean accuracy was about 1% higher at both lags when we included only trials with a correct response in Task₁, but the lag effect remained highly significant). Because we could not conditionalize in this way in the ignore- T_1 condition, and because such conditional analyses differ very little from the unconditional analyses, we report the results without conditionalizing.

The accuracy of report of T_2 exhibited a classic attentional blink pattern, which can be seen in Fig. 2. Accuracy of T_2 report was lower in the report- T_1 condition than in the ignore- T_1 condition, $F(1, 15) = 20.84$, $\text{MSE} = 0.011994$, $P < 0.0004$, and particularly so at lag 2, producing an interaction between lag and Task₁ instructions (attend vs ignore), $F(1, 15) = 36.06$, $\text{MSE} = 0.002619$, $P < 0.0001$. The main effect of lag was also highly significant, $F(1, 15) = 44.64$, $\text{MSE} = 0.003172$, $P < 0.0001$.

The proportion of correct reports of T_1 (in the report- T_1 condition) was 0.84 at lag 2 and 0.90 at lag 8, suggesting some degree of competition for limited capacity at the shorter lag, $F(1, 15) = 22.65$, $\text{MSE} = 0.001337$, $P < 0.0003$ (Tombu & Jolicœur, 2003). The foregoing results were for Task₁ accuracy, not conditionalized on Task₂ accuracy. However, essentially the same results were observed when Task₁ performance was examined only for trials in which an accurate response was given in Task₂.

The ERP waveforms, time locked to the onset of T_2 , for ipsilateral and contralateral electrodes relative to the visual field of T_2 , at the site where the N2pc was maximal in our recordings (T5/T6), are shown in Fig. 3, for the ignore- T_1 lag 2 condition. The waveforms are complex because they reflect a superimposition of ERPs to a sequence of stimuli presented at 100 ms intervals immediately prior to the onset of T_2 , and from the mask (immediately after T_2). Because of the linear summation property of ERPs, however, the N2pc component can be

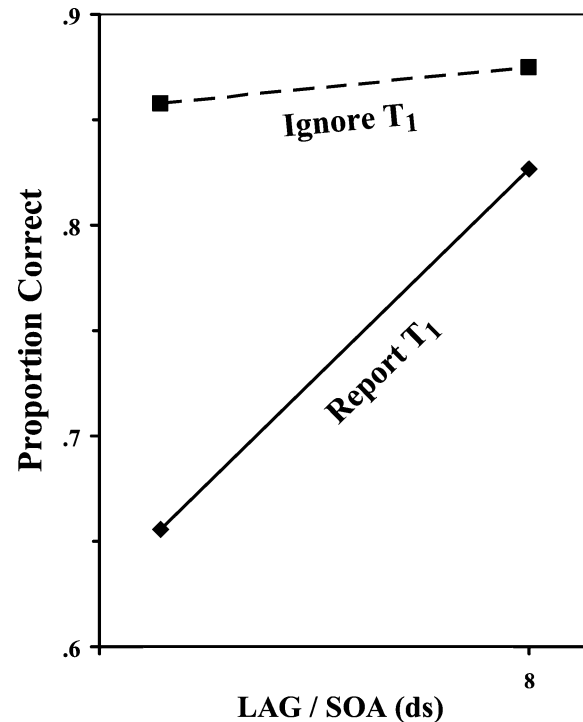


Fig. 2 Proportion correct report of T_2 , for each lag, in the ignore- T_1 and the report- T_1 conditions

extracted from these complex responses by taking the difference between the contralateral waveform and the ipsilateral waveform (Luck & Hillyard, 1994; Woodman & Luck, 2003). The N2pc difference waves are shown in Fig. 4, for the four conditions in the experiment, at T5/T6.

Analyses of ERPs were carried out without conditionalizing on accuracy of report of T_1 or of T_2 . We

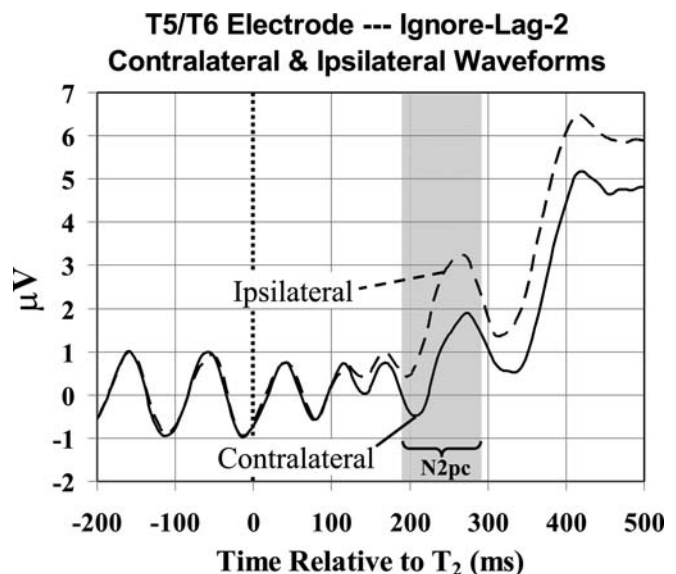


Fig. 3 Grand average event-related potentials for the ignore- T_1 , lag 2, condition, at electrode site T5/T6 (where the N2pc was largest). The shaded region shows the time window used to quantify the N2pc (see Fig. 4)

considered the experimental design as providing four conditions (the crossing of reporting vs ignoring T_1 and the two lags), and we were interested in describing the ERPs in each of these conditions, given that we knew that they also produced large differences in report accuracy for T_2 .

The N2pc was quantified by measuring the mean amplitude of the N2pc difference wave in a window of 190–290 ms (shaded region in Fig. 4). These mean amplitudes were estimated for each subject, for each condition, for each of the lateralized posterior electrodes in our montage (O1/O2, P3/P4, T5/T6). The mean amplitudes were submitted to ANOVA, with condition (ignore- T_1 vs report- T_1), lag (2 vs 8) considered as within-subjects factors. We performed both ANOVAs with electrode as a factor, and separate ANOVAs for each electrode site, with similar results in both cases.

Consider first the N2pc components observed in the ignore- T_1 conditions (Fig. 4). The N2pc responses in the ignore- T_1 conditions suggest that subjects were able to deploy spatial attention to T_2 , in the absence of concurrent processing of T_1 . Regardless of lag, there was a significant N2pc at all lateral posterior electrodes ($P < 0.007$ or better, at each electrode site for both lags), suggesting that subjects were able to deploy visual spatial attention to T_2 when T_1 could be ignored. These results are important because they show that the display conditions for T_2 induced a shift of spatial visual attention to the location of T_2 in the visual field, which could be tracked by the N2pc electrophysiological response, as expected based on prior work (Eimer, 1994; Luck & Hillyard, 1994; Woodman & Luck, 2003).

Figure 4 shows the N2pc waveforms and Fig. 5 shows the mean N2pc amplitudes measured at electrodes T5/T6 for each condition in the 190–290 ms temporal window. The same patterns of results were observed at O1/O2 and P3/P4. Separate ANOVAs at each of the three posterior lateral electrode sites, confirmed what can plainly be seen in Figs. 4 and 5, namely that the mean amplitude was lower in the report- T_1 condition than in the ignore- T_1 condition. The effect was found at each electrode site ($P < 0.02$ for O1/O2; $P < 0.0008$ for P3/P4; $P < 0.0007$ for T5/T6).

As can be seen in Figs. 4 and 5, lag affected the amplitude of the N2pc component differently for the report- T_1 condition and the ignore- T_1 condition. In the report- T_1 condition, reducing lag (increasing competition for central resources) caused a decrease in the mean amplitude of the N2pc response. In contrast, reducing lag had the opposite effect in the ignore- T_1 condition. This pattern of results produced a significant interaction between T_1 condition and lag for each or the three lateral posterior electrode pairs ($P < 0.015$ for O1/O2; $P < 0.025$ for P3/P4; $P < 0.006$ for T5/T6). In separate analyses of just the report- T_1 condition, we found a significant reduction in N2pc amplitude as lag was reduced for electrodes O1/O2 ($P < 0.031$) and T5/T6 ($P < 0.038$), but not for P3/P4 ($P > 0.27$), although the pattern of means was essentially the same for O1/O2 and P3/P4. The lag effect in the report- T_1 condition allows us to argue against the notion that attenuation of N2pc was caused by different physiological states or preparatory strategies induced by the different conditions because lag was manipulated from trial to trial. In additional work in our laboratory, we found an attenuation of N2pc with greater central encoding load in a paradigm in which

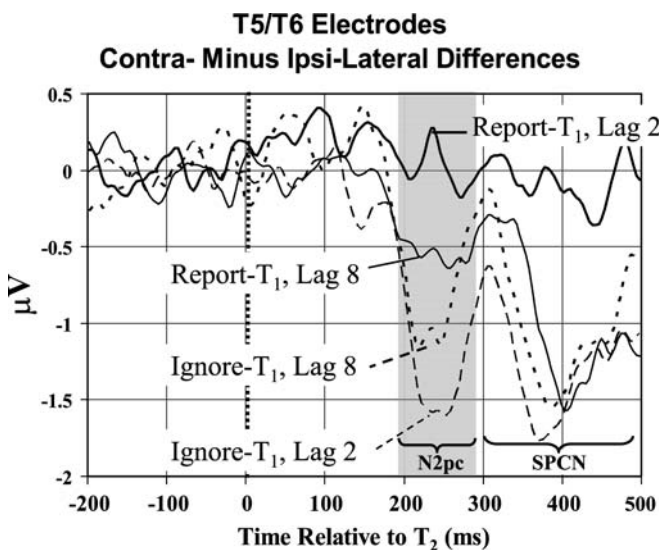


Fig. 4 Contralateral minus ipsilateral difference waves used to isolate the N2pc and the sustained posterior contralateral negativity (SPCN), at electrode site T5/T6, for the four conditions in the experiment. The shaded region shows the time window used to quantify the N2pc. The region from 300 to 420 ms was used to quantify the SPCN

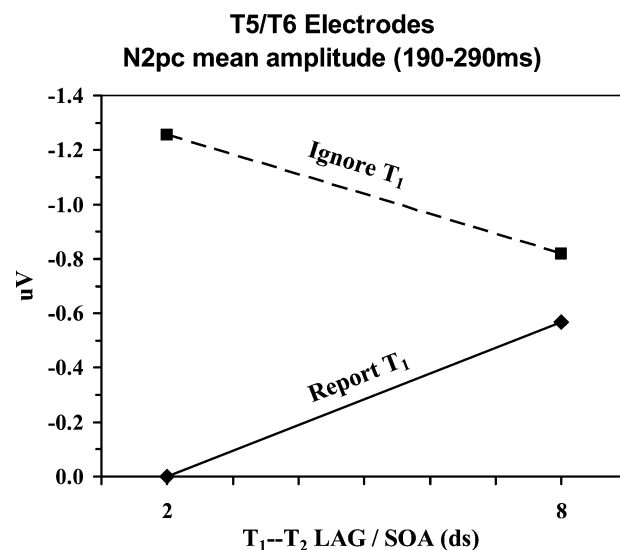


Fig. 5 Mean amplitude of the N2pc (in μV), between 190 and 290 ms, at electrode site T5/T6, for the report- T_1 and ignore- T_1 conditions, at each lag

encoding load varied from trial to trial. It is not clear why the amplitude of the N2pc increased with decreasing lag in the ignore- T_1 condition. This effect might be associated with the shorter RSVP streams in the lag 2 condition. Recall that the central stream ended one character after the presentation of T_1 , in the lag 2 condition, and 7 characters after T_1 in the lag 8 condition. T_2 was presented immediately following the last central stream item, and the entire presentation sequence ended after the post- T_2 masks. Consequently, the RSVP sequences were shorter for the lag 2 condition than for the lag 8 condition. Given that the presentation sequences were identical in the report- T_1 condition as in the ignore- T_1 condition, these two conditions can be compared directly. This makes the near-complete suppression of the N2pc in the report- T_1 , lag 2, condition all the more impressive.

One might wonder if the N2pc in the report- T_1 , lag 2, condition was delayed rather than attenuated. If the delay was several hundred milliseconds, it is possible that the present averaging window might have missed a later effect. We performed another analysis of the report- T_1 , lag 2, condition in which the ERP averaging window was extended from 500 to 1,000 ms post T_2 . We found no evidence of a delayed N2pc in the 500–1,000 ms interval. An inspection of the curves shown in Fig. 4 also suggests that the modulation effects on the N2pc were mainly ones of amplitude rather than latency. Consider, for example, the two ignore- T_1 and the report- T_1 , lag 8 conditions. Although, there is an obvious amplitude differences across these conditions, the three N2pc waves began at the same time (about 190 ms), and reached a minimum at about the same time (310 ms). There was no indication that the amplitude differences between these conditions were caused by increased variability in the latency of the components (across subjects, or across trials, within subjects).

Discussion

Overall, the patterns of results were clearcut: the N2pc was smaller in the report- T_1 condition than in the ignore- T_1 condition, and this difference was larger at the short T_1 – T_2 lag than at the long T_1 – T_2 lag. The N2pc provides an electrophysiological index of the moment-by-moment deployment of visual spatial attention. The sharply reduced N2pc response under high-central attentional load suggests that subjects were no longer able to deploy spatial attention to the side of the T_2 , during the attentional blink. The results suggest that the mechanisms that control the deployment of spatial visual attention are not independent of those that cause the attentional blink.

The results disconfirm the hypothesis that visual spatial selection constitutes an initial processing bottleneck that is independent from a later response-selection (or a more general central) bottleneck, at least when the spatial selection cue is colour. Evidence for indepen-

dence was found when attention was directed using a flash of light (Johnston et al., 1995). It is possible that such exogenous control would not be subject to the capacity limitations that we have discovered in our experiment. This hypothesis is currently under investigation in our laboratory. In the present experiment, selection was based on the colour of the target, and it is possible that this type of selection required attention. Interestingly, however, the colour differences we used were large, and previous research has shown that such colour differences are sufficient to produce pop-out in visual search (e.g. Bauer, Jolicœur, & Cowan, 1996; Nagy & Sanchez, 1990). Furthermore, colour singletons appear to draw spatial attention to their location when subjects maintain the intention to find targets of that colour (Folk, Leber, & Egeth, 2002; Leblanc & Jolicœur, 2005). In the latter case, the attentional capture effects appeared to take place automatically (once top-down attentional control settings were adopted by the subjects), given that the capture by the distractor always produced a decrement in report of the desired target. Apparently, however, the present conditions were sufficient to override such control settings, making it difficult for subjects to deploy their attention to the target location, when attention was engaged on T_1 .

The suppression of N2pc in the report- T_1 , lag 2, condition, could reflect a failure of processing at one or more of several possible stages of processing between the initial presentation of T_2 and the stage(s) at which spatial attention is deployed to the location of T_2 . For example, one may wonder whether the masks that immediately followed T_2 and the T_2 distractor, prevented the processing of the colour of these stimuli. If basic colour processing was affected by the masking manipulation we used, then it would not be surprising to find that subjects could not use colour to deploy spatial attention. If so, the suppression of N2pc would not reflect a failure of spatial attention, per se, but rather a failure of colour perception.

The results from the ignore- T_1 condition, however, show that the mere presence of the masks following T_2 did not impair colour perception sufficiently to abolish the N2pc, given that significant N2pc responses were observed when T_1 could be ignored. Furthermore, in a separate control experiment we used the same stimulus presentation sequences as in the main experiment (same number of trials, same block structure, etc.), but we asked nine observers to indicate the side of presentation of the T_2 target digit. The purpose of this control was to ensure that the colour information was readily perceivable, despite the presence of the masks trailing T_2 . When T_1 could be ignored, accuracy was 98.5% regardless of lag, demonstrating that the masks following T_2 had essentially no effect on the perceptibility of the selection cue for T_2 . Interestingly, we also found a small, but significant, decrement in accuracy of report of the side of presentation of T_2 when T_1 (digit) had to be reported, and an interaction with lag (accuracy was 96% at lag 8 and 92% at lag 2 when the identity of T_1 had to be

reported), providing converging evidence for an AB for colour (Ross & Joliceur, 1999). Importantly, this decrement was smaller than the corresponding lag effect in the report- T_1 condition in the main experiment, $F(1, 23) = 11.70$, $MSE = 0.0036$, $P < 0.002$; and combined with minimal effects of lag in the ignore conditions, produced a three-way interaction between experiments, T_1 condition, and lag, $F(1, 23) = 7.08$, $MSE = 0.0018$, $P < 0.015$ (reflecting a significantly larger AB in the main experiment than in the colour control experiment).

Although, we found a small attentional blink on subject's ability to report the side of presentation of T_2 , the effects were smaller than those in the main experiment, and were insufficient to explain them or the nearly complete suppression of the N2pc in the report- T_1 lag-2 condition. Furthermore, what is most critical is that simple masking did not impair performance when subjects merely reported the side of presentation of T_2 (when T_1 could be ignored), which means that colour information was correctly registered in early stages of visual processing and available for further processing. The sharply attenuated N2pc in the report- T_1 lag-2 condition implies that during the attentional blink the available colour information could not be used by the mechanisms that normally guide and engage visual spatial attention on T_2 . Hence, the control of visual spatial attention is not independent of the attentional blink. The small attentional blink observed in the control experiment could have resulted from a failure of spatial attention or from an impairment in the short-term consolidation of the information about side of presentation.

The complete suppression of the N2pc in the report- T_1 lag-2 condition in the presence of above-chance report accuracy for T_2 raises the issue of the relationship between N2pc, spatial attention, and report accuracy. Understanding this relationship, in turn, requires an interpretation of the N2pc itself. Luck and his colleagues have argued that the N2pc reflects a process of distractor suppression that is engaged in situations in which a target must be processed in the presence of visual distractors (see Luck et al., 1997; Luck & Hillyard, 1994). Eimer (1996), on the other hand, suggested that the N2pc likely reflects target enhancement associated with preferential processing of a target. One or both of these mechanisms would ultimately improve the signal-to-noise ratio of target processing, presumably leading to improved performance when attention could be deployed to the target. The precise nature of the underlying mechanisms that produce the N2pc are not critical in the present context, however. Whatever they are, it appears that the bottom-up signal generated by T_2 was sufficient to support above-chance performance, even if the target was not enhanced or the distractors were not suppressed by the mechanisms that generate the N2pc. Alternatively, some attention may have been deployed at both locations (generating no net lateralization, and hence no N2pc), perhaps enabling sufficient signal to support above-chance accuracy, but nonetheless much lower performance than when differ-

ential attention could be allocated to T_2 . Further research will be required to arrive at a more complete understanding of the relationships between N2pc, spatial attention, and behavioural performance.

Another interpretation of the attenuation of the N2pc in the report- T_1 condition is that observers may have found it more difficult to disengage from the central RSVP stream when relevant information was presented there. If so, the present work would show that observers could more easily redeploy their attention to the location of T_2 (one of two peripheral locations) when they did not need to monitor the central location for the presence of a target. The present results, per se, cannot completely rule out this interpretation. In anticipation of this possible objection, however, we used lag 2 as our short-lag condition, because prior work showed that effects of changing location between the presentation of T_1 and T_2 were confined to very short SOAs, namely lag 1, and were completely absent at lags 2 and beyond (see Visser, Bischoff, & Di Lollo, 1999, for a review). The results reviewed by Visser et al. (1999) on this matter suggest that spatial attention can readily disengage from the location at which T_1 was presented. If so, the present results demonstrate that central attentional load has a significant impact on the mechanisms that control spatial attention.

More importantly, additional work in our laboratory, using a very similar T_2 visual display as in the present experiment, found that the N2pc was attenuated when subjects performed a difficult speeded discrimination to a tone relative to when they made a simple RT response to the tone. The results showed that the attenuation of N2pc by a concurrent central load can be obtained in a paradigm in which the first task does not have the potential to tie up visual attention prior to the presentation of T_2 (e.g. Brisson & Joliceur, 2004).

Earlier work showing greater flanker interference under central attentional load suggested that control over spatial selection could be weakened by an increase in central load (de Fockert, Rees, Frith, & Lavie, 2001; Jiang & Chun, 2001). However, these results could also be explained by a loss of control at later stages of processing (i.e., consistent with late-selection accounts, such as response-selection, or selection for short-term consolidation; Joliceur & Dell'Acqua, 1998). The modulations of N2pc by central attentional load in the present and concurrent work in our laboratory provide direct electrophysiological evidence that visual spatial attention is dependent on central executive control. Studying these interactions further is likely to provide a powerful tool to study the neural and psychological mechanisms that underly the control of visual spatial attention.

SPCN, a new electrophysiological correlate of the AB phenomenon

The ERP results also revealed a new electrophysiological correlate of the AB. As can be seen in Fig. 4, in addition

to the N2pc, we observed what appeared to be a second component that was also characterized by a greater negativity contralateral to the target, but this component was later than the N2pc and was observed between 300 and 500 ms. This component was substantial for the two ignore- T_1 conditions and the report- T_1 , lag 8; and it was almost completely suppressed in the report- T_1 , lag 2, condition. The modulations of the amplitude of these ERP waveforms thus correlates very well with the pattern of results observed across conditions for the report accuracy of T_2 . We refer to this lateralized component as the SPCN, for sustained posterior contralateral negativity.

The onset of the SPCN, unlike the N2pc, appeared to have different latencies for the different conditions, with an earlier onset for the ignore- T_1 , lag 2, condition; an intermediate onset latency for the ignore- T_1 , lag 8 condition; and the longest onset latency for the report- T_1 , lag 8, condition. All three conditions had a similar peak amplitude, however, with perhaps a slightly higher peak for the ignore- T_1 , lag 2, condition. The SPCN had a very small amplitude and no clear onset for the report- T_1 , lag 2, condition.

We quantified the SPCN by measuring the mean amplitude, for each condition and each subject, in a time window of 300–420 ms. This measure captures both amplitude and latency differences across conditions in this time window.

We present here the analysis for the data at the T5/T6 electrode pair (similar results were found at O1/O2 and P3/P4). The mean amplitude was higher in the ignore- T_1 ($-1.3 \mu\text{V}$) condition than the report- T_1 condition ($-.45 \mu\text{V}$), $F(1,15) = 11.95$, $\text{MSE} = 0.961583$, $P < 0.004$. The main effect of lag was not significant, $F(1,15) = 1.08$, $\text{MSE} = 1.18$, $P > 0.31$. However, there was an interaction between T_1 condition (report vs ignore) and lag, $F(1,15) = 10.57$, $\text{MSE} = 0.419689$, $P < 0.0054$. As for the N2pc, there was an increase in the mean amplitude of the SPCN as lag was reduced, in the ignore- T_1 condition ($-1.42 \mu\text{V}$ at lag 2; $-1.18 \mu\text{V}$ at lag 8), but a sharp decrease in the amplitude of the SPCN as lag was reduced for the report- T_1 condition ($-0.05 \mu\text{V}$ at lag 2; $-0.86 \mu\text{V}$ at lag 8).

Vogel and Machizawa (2004) found a posterior contralateral response related to the maintenance of information in visual short-term memory (VSTM). Perhaps the present SPCN component reflects the same neural source as the delayed contralateral activity studied by Vogel and Machizawa (2004). In our modified AB paradigm, it is possible that information that could not be reported because of the AB also failed to be stored in VSTM. Information may be stored in VSTM only long enough to ensure a transfer to other memory systems (e.g., verbal STM). A failure to transfer the information into VSTM would cause processing failures later in the processing stream. We speculate that the onset of the SPCN in the present work may reflect the neural activity that mediates the loading of information into VSTM. This activity was delayed somewhat (in some conditions), but

not strongly attenuated (Fig. 4) in the three conditions in which subjects were able to report correctly the identity of T_2 . In the report- T_1 , lag 2, condition, however, in which report accuracy for T_2 was reduced (due to the AB), the SPCN was completely suppressed. This suppression of SPCN suggests that the transfer of T_2 to VSTM suffered from strong dual-task interference.

Vogel et al. (1998) and Dell'Acqua, Jolicœur, Pesciarelli, Job, and Palomba (2003) found that the P3 response to T_2 was completely suppressed during the AB, which they interpreted as an electrophysiological indicator that a representation of T_2 could not be transferred to STM. The attenuation of the SPCN caused by the AB in the present work could well be related the suppressed P3 response during the AB that was discovered by Vogel et al. (1998) and replicated and extended by Dell'Acqua et al. (2003). In this view, not only would the AB be associated with a failure to encode information in (perhaps an amodal, or a verbal) STM store (P3 suppression), but it would also be associated with a failure of encoding in VSTM (SPCN suppression).

Clearly, our present interpretation of the SPCN is speculative. Alternative interpretations include the possibility that what we are calling an SPCN is actually continued activity in the neural generator of the N2pc. Perhaps the N2pc has two subcomponents, and early wave and a late wave. One might imagine that the later component is due to latency jitter of the early wave. This is not very likely, however, because latency jitter would simply smear the N2pc, rather than produce what seems to be a well-defined initial component, a return to baseline, followed by a second clear component.

However, we openly admit that more work will be required to establish a stronger link between VSTM and SPCN in the present paradigm. Additional work in our laboratory is currently under way to test various counterinterpretations of the SPCN. Whatever neural activity caused the SPCN, it is clear that it was sharply attenuated by concurrent activity required to encode and remember T_1 , in the report- T_1 , lag 2, condition. This attenuation was not seen in the other three conditions, in which report accuracy for T_2 was quite high. The SPCN is thus a new electrophysiological correlate of the AB, and further study of this component may thus provide valuable information about the nature of the underlying mechanisms involved in the AB phenomenon.

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