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Research Report

Short-term consolidation of visual patterns interferes with visuo-spatial attention: Converging evidence from human electrophysiology

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ARTICLE INFO

Article history:
Accepted 4 September 2007
Available online 14 September 2007

Keywords:
Spatial attention
N2pc
P3
Attentional blink

ABSTRACT

In order to investigate the interplay between visuo-spatial attention and central attention, we varied the relative probability (25% vs. 75%) of the responses to lateralized targets in an attentional blink paradigm. When the first target was associated with a less probable response, we observed a larger attentional blink, that is, a general reduction in accuracy for the second target. The efficiency of deployment of spatial attention to the second target was also reduced as a function of the response frequency for the first target. Both the N2pc, an event-related potential (ERP) associated with the deployment of attention in visual space, and the SPCN (sustained posterior contralateral negativity), an ERP associated with the maintenance of information in visual short-term memory, time-locked to T2 were significantly reduced when the first target was associated with a less frequent response. Furthermore, the P3 ERP to T2 was abolished when the response to T1 was rare but not when it was frequent. The results show that the association of T1 to either a rare or frequent response causes significant interference with the deployment of visual spatial attention to T2, and with the short-term consolidation of T2 into visual short-term memory.

1. General introduction

The attentional blink (AB) is a phenomenon typically observed when two briefly presented and masked visual targets, each requiring some form of delayed response, are presented at short stimulus-onset asynchronies (SOAs). The AB consists in a lower probability of correct report of the second target (T2) while the first target (T1) is generally reported successfully (Raymond et al., 1992). A generally accepted view by researchers in this domain is that the AB arises from limitations at a post-perceptual stage of processing of T2. Such limitations would prevent a modality-dependent, perceptual representation of T2 from being consolidated in visual short-term memory (VSTM; Chun and Potter, 1995; Jolicœur, 1998; Shapiro et al., 1994; Vogel et al., 1998).

The idea of a post-perceptual locus of the AB has received solid support in studies using the event-related potential (ERP) technique. Vogel et al. (1998), for instance, used ERPs to monitor various aspects of the processing of T2 during the AB interval. In a series of rapid serial visual presentation (RSVP) designs, several factors tapping distinct stages of T2 processing were examined as potential causes of the AB. The results documented the preservation of the T2-locked ERP components up to and including the N400 (e.g., P1 and N1 components) at short SOAs,

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doi:10.1016/j.brainres.2007.09.004

suggesting that the AB did not interfere with stages of T2 processing that included the generation of a semantic code for T2 (see also Potter et al., 2005; Rolke et al., 2001; Visser et al., 2005). In contrast, Vogel et al. (1998) found that the amplitude of the T2-locked P3 component was markedly reduced at short SOAs (during the AB), with the P3 component returning to a normal amplitude when T2 was displayed outside the AB (see also Dell'Acqua et al., 2003a,b; Kranczioch et al., 2003), or when T1 could be ignored (Sessa et al., 2007).

The nature of the functional processes reflected in the P3 component is not clear as of yet. Several researchers tend to regard the P3 as related to the decision on how to classify and respond to an eliciting stimulus (e.g., Squires et al., 1973, 1977; Verleger et al., 2005). Others maintain that the P3 reflects access of the eliciting stimulus to global mental workspace, with this access making the event conscious and therefore reportable (Dehaene et al., 2003; Koivisto and Revonsuo, 2003; Sergent et al., 2005). Probably the most influential and popular line of thinking is that linking the P3 to updating of working memory (Donchin and Coles, 1988; Johnson, 1993; Polich and Criado, 2006). Suppression of P3 in the AB would be interpreted by the first view as mere reflection of the fact that no decision could be reached about T2, by the second view as reflecting the failure of T2 to enter global mental workspace, and by the third view as reflecting the failure of T2 to be encoded in visual working memory. For the present purposes, it is important to note that all notions mentioned above are convergent with the idea that the P3 is a direct reflection of functional activity occurring once a visual stimulus has already been processed at a sensory/perceptual stage. In this particular perspective, the notions about the P3 are also convergent with AB models positing a post-perceptual 'bottleneck' along the flow of processing leading to encode T2 in VSTM (Chun and Potter, 1995; Jolicoeur and Dell'Acqua, 1998). There are, however, slight differences among the P3 models as to what exactly would be the cause of the P3 modulation observed during the AB. While indeed the former two notions imply that P3 suppression reflects the consequences of such bottleneck, the memory-updating notion implies that P3 suppression reflects just this bottleneck. Specifically, when the consolidation stage hypothesized to transfer target information in VSTM is occupied with processing T1, consolidation of T2 is postponed, and T2 remains temporarily in a stand-by state during which it is vulnerable to corruption by items trailing T2 in the RSVP stream (Chun and Potter, 1995; Dell'Acqua et al., 2003a,b; Giesbrecht and Di Lollo, 1998; Jolicœur, 1999a).

A further important application of the ERP technique in the AB context has been useful to illuminate the impact of the AB on the efficiency of the control of visual spatial attention. The logic behind these studies was that of presenting a lateralized T2 during the AB, and tracking the displacement of spatial attention to T2 through the monitoring of a component of the ERP to T2 labeled N2pc. The N2pc consists in a greater negativity, starting about 180 ms after the onset of a visual display, over the posterior hemisphere contralateral to an attended visual target, relative to analogous regions of the hemisphere ipsilateral to the target (Eimer, 1996; Luck and Hillyard, 1994; Woodman and Luck, 2003). In general, a reduction in the amplitude of the N2pc indicates either an absolute reduction in attention allocation, or a reduction in the differential allocation of visual spatial attention to a target (relative to attention allocated to the opposite visual field), or both.

Jolicœur et al. (2006a,b) used two distinct experimental manipulations in order to measure the N2pc to a T2 that was subject to the AB influence. The authors varied the SOA between a centrally displayed T1 and a lateralized T2. At an SOA of 200 ms, a reduction of the amplitude of the N2pc was observed relative to a control condition in which T2 was presented at an SOA of 800 ms. A second manipulation involved the task performed on a centrally displayed T1 under conditions in which the SOA between T1 and the lateralized T2 was fixed at 200 ms. On half of the trials, the subjects had to encode T1 for delayed report, whereas T1 could be ignored in the remaining trials. This was achieved by dividing the experiment into two blocks, one during which the subject was instructed to report both targets, the other in which the subject was instruct to report only the last target. When T1 had to be reported, a reduction of the amplitude of the N2pc was observed relative to a control condition in which T1 could be ignored. Based on these results, Jolicœur et al. (2006a,b) concluded that the consolidation of T1 was likely to interfere with the deployment of spatial attention to the position occupied by T2, with the consequent difficulty to encode T2 in VSTM.

The conclusions of Jolicœur et al. (2006a,b) could, however, be questioned based on details of the design that allowed for different interpretations of the results. Specifically, T1 was presented centrally, whereas T2 was lateralized. One could argue that processing of T1 demanded an initial state of attention focused at the center of the screen, where T1 was displayed throughout the experiment. If this were so, it is reasonable to assume that allocating attention to a lateralized T2 required a disengagement of visual spatial attention from the center of the screen and a shift to the location of T2. In this optic, the N2pc was reduced during the AB because there was less time, at the short SOA, to allow spatial attention to disengage from the position occupied by T1 and shift to the lateralized T2 relative to a condition with a long delay between T1 and T2.

An alternative explanation can also be raised for the second control condition used by Jolicœur et al. (2006a,b). It is of note that trials in which T1 was to be encoded and trials in which T1 could be ignored were blocked. The criticism in this case would be that in blocks of trials where the subject knew in advance that T1 could be ignored, subjects were likely not to engage at all their visual spatial attention on the centrally displayed T1, in contrast with the blocks of trials where T1 had to be reported.

An attempt to solve these problems was made by Dell'Acqua et al. (2006) by using two synchronized RSVP streams of letters, one on each side of a central fixation point. In the condition of most interest in the present context, T1 consisted of a pair of simultaneously presented digits, each embedded in one of the lateralized RSVP streams. The task was to determine if the digits were the same or different. T1 could also consist of a pair of "=" signs. In those trials, subjects were instructed to ignore T1. This condition was designed to address one of the concerns mentioned previously, namely the potential discrepancy in attentional state demanded by processing T1 and T2. The subjects did not know in advance if T1 was to be processed deeply (digits) or not ("=" signs), so they presumably expanded their spatial attention so as to cover

both positions occupied by the two lateralized RSVP steams on every trial. T2, which was displayed at an SOA fixed at 250 ms, consisted of a pair of squares that each had a small gap on one of the sides. These squares were followed by masking patterns. The squares were displayed one in green and one in red, and subjects were instructed to attend selectively to only one of the squares based on a specific color in order to be able to locate the gap position (responding 'top, bottom, left or right' with no speed pressure at the end of the trial). Consistent with the results obtained by Jolicœur et al. (2006a,b), the results of Dell'Acqua et al. (2006) showed a marked reduction both in accuracy of T2 report and in the amplitude of the N2pc to T2 in trials in which the T1 digits had to be encoded relative to trials in which the T1 "=" signs could be ignored.

Although the results of Dell'Acqua et al. (2006) helped to rule out a different initial attentional state in the processing of T1 and T2 as a potential explanation of the reduction in the N2pc amplitude found when the post-perceptual processing load was increased (i.e., under AB conditions), their interpretation is, however, not devoid of problems. The pair of "=" signs had some critical differences with the digit pairs used for T1. These stimuli were neither letters nor digits, so they were presumably easier to identify based on low-level features. The "=" signs were present on half of the trials, so they were more likely to appear than any of the digits in the context of the experiment. Furthermore, a T1 made of "=" signs was consistently composed of two identical stimuli whereas a T1 composed of digits was only an identical pair on half of the T1-digit trials. In this vein, a potential criticism might be that the ERP effects described by Dell'Acqua et al. (2006) could not be interpreted solely as reflections of a limit at post-perceptual stage of processing, because the critical task differences for T1 were confounded with variations at a sensory/perceptual stage of processing T1. To complicate further the interpretation of Dell'Acqua et al.'s (2006) results, it must be noted that the task on T1 and the task on T2 were substantially different, namely, T1 was a go/no-go task based on alphanumeric visual stimuli that had to be classified as same or different on some trials and the task on T2 was a gap localization task performed on simple geometric shapes. An additional doubt may thus arise as to the influence of task switching effects that some researchers view as different from AB effects proper (e.g., Potter et al., 1998).

The present work avoided these difficulties by counterbalancing the same physical stimuli across all experimental conditions and by using the same task for Task1 and Task2. The key manipulation was the relative probability of the response associated with different (but highly similar) T1 and T2 stimuli, and this was done based on two basic considerations. Firstly, we capitalized on the earlier work of Crebolder et al. (2002), who showed that the AB is exacerbated (lower probability of report of T2) when T1 is less likely to occur relative to the AB observed for more frequent T1 stimuli. By using equivalent stimuli in all conditions (squares with a gap, differing only in the location of the gap), we ensured that different T1 stimuli required the same degree of visual processing. We used a single SOA in all conditions so the time between the disengagement of spatial attention on T1 and the appearance of T2 was always the same. Secondly, we made use of a well-established property of the P3, namely that the amplitude of the P3 is increased for stimuli associated with less probable classifications. Monitoring of the P3 in the present empirical context was used purposely to provide electrophysiological evidence that the frequency manipulation implemented in the present design influenced post-perceptual processing mechanisms.

We used a two-event AB paradigm in which T1 and T2 were each followed by masks, but in which there were no other stimuli, as in the work of Duncan et al. (1994). In order to remove the possibility of any task-switching between T1 and T2, we used the same stimuli for T1 and T2, and the task associated with T1 and T2 was the same. Targets were empty squares with a gap on one side, followed by a mask. Four gap locations (top, bottom, left, and right) were used, each having 25% probability of occurrence. The subject had to report the location of the gap in the square. As is typical in experiments using the AB paradigm, the response was not speeded and it was performed at the end of the trials. Two responses key were used, one for one of the gap position (e.g., top) and the other for the remaining three gap positions (i.e., not top). Consequently, each visual stimulus had the same probability of occurrence, but they were categorized in two response categories, one with 25% probability and the others with 75% of probability. This allowed us to isolate T1-locked and T2-locked P3 activity purely related to the frequency manipulation, by subtracting the ERP time-locked to a stimulus associated with a frequent response from the ERP time-locked to the stimulus associated with an infrequent response (see Dell'Acqua et al., 2003a,b, 2005; Vogel et al., 1998).

We first conducted a behavioral AB experiment that included a manipulation of SOA, to replicate the results of Crebolder et al. (2002), namely a larger AB when T1 is assigned to a lower probability category, in a design appropriate to elicit an N2pc. We will then use a single SOA in Experiment 2, which included ERP recordings and measured the N2pc, SPCN, and P3.

2. Results of Experiment 1

The proportion of correct responses to T1 and T2 were first submitted to a four-way ANOVA (Block order of the orientation of the first squares × SOA × Frequency of T1 × Frequency of T2). The block order of the orientation of the first squares had neither a main effect nor interaction with any of the others variables. Consequently, the data were collapsed across this variable. The staircase procedure successfully maintained the accuracy of response for T2 for the long SOA between the prescribed boundaries (the average accuracy for T2 was 76.3%). The average duration of T2 was 134.4 ms.

2.1. *Accuracy for T1*

The mean accuracy for T1 is listed in Table 1. Mean accuracy was higher at the long SOA (76.54%) than at the short SOA (69.4%), $F(1,15)=28.924$, MSE = .0056, $p<.0001$. At the short SOA, the accuracy of T1 was lower when T1 was rare (reduction of 5.1% from T1 frequent to T1 rare), $F(1,15)=7.420$, MSE = .0057, $p<.02$, which was not the case at the long SOA (augmentation of 1% from T1 frequent to T1 rare), $F(1,15)=.336$, MSE = .0047, $p>.5$. This led to a significant T1 frequency × SOA interaction, $F(1,15)=4.874$, MSE = .0062, $p<.04$.

Table 1 – Success rate (%) for T1, for Experiments 1 and 2, for each experimental condition

Exp.	SOA	T1F		T1R	
		T2F	T2R	T2F	T2R
1	250	80.3	63.6	80.1	53.5
1	850	81.9	70.2	82.3	71.8
2	350	97.9	96.2	90.0	92.3

Note. T1F means T1 frequent, T1R means T1 rare; T2F means T2 frequent, T2R means T2 rare.

Furthermore, when T2 was rare, the accuracy for T1 was reduced (from 81.1% when T2 was frequent to 64.7% when T2 was rare), $F(1,15)=11.035$, MSE=.0777, $p<.005$. The accuracy of T1 at the short SOA was reduced when T2 was rare (reduction of 21.7% from T2 frequent to T2 rare), $F(1,15)=16.257$, MSE=.0461, $p<.001$, and at the long SOA (reduction of 11.1%) from T2 frequent to T2 rare), $F(1,15)=4.886$, MSE=.0403, $p<.05$. The effect was stronger at short SOA than at long SOA, which lead to a significant SOA×T2 frequency interaction, $F(1,15)=10.238$, MSE=.0087, $p<.006$. Clearly, both the temporal distance between T1 and T2 and the frequency of the response associated to T2 influenced the accuracy of T1.

2.2. *Accuracy for T2*

The accuracy rate for T2 is shown in Fig. 1. Only the trials with a correct response for T1 were considered here. Generally, the accuracy for rare T2 was lower than the accuracy for frequent T2, $F(1,15)=12.10$, MSE=.077, $p<.004$. This difference was larger when T1 was rare than when T1 was frequent, $F(1,15)=4.78$, MSE=.0648, $p<.045$. Furthermore, this interaction (frequency of T1×frequency of T2) was larger at short SOA than at long SOA, $F(1,15)=8.08$, MSE=.0111, $p<.02$. Further analysis indicated that this interaction (frequency of T1×frequency of T2) was significant at short SOA $F(1,15)=8.87$, MSE=.041, $p<.009$, but not at long SOA $F(1,15)=.95$, MSE=.03, $p>.34$. Note that T2 accuracy was lowest for rare T2s when T1 was rare and the SOA was short, and accuracy for frequent T2s was highest when T1 was rare and SOA was short.

2.3. *Effect of the repetition of the stimulus*

It could be argued that the AB effect in these data is confounded with a repetition-suppression (or repetition blindness) effect. Indeed, when both targets are rare, both targets are also identical. This is not the case for the frequent target, where there are three possible stimuli. In order to verify if a repetition-blindness effect was occurring here, we ran an analysis using only the trials were the two targets were of the frequent category. The repetition or the non-repetition of the target had no effect on the accuracy of either T1, $F(1,15)=3.003$, MSE=.0021, $p>.1$, or T2 $F(1,15)=.0523$, MSE=.0013, $p>.8$.

3. Discussion of Experiment 1

The higher accuracy for frequent T2s at short SOA when T1 was rare may appear counterintuitive, at first glance. However, this was the expected result if processing a rare T1 increases the AB. In absence of knowledge of the correct response, the subjects were more likely to guess using the frequent response rather than the rare response. This strategy would decrease accuracy for infrequent T2s but increase accuracy for frequent T2s. This is exactly what we observed, particularly when the T1-T2 SOA was short, indicating that decreasing SOA increased the probability of guessing (that is, increased the AB). We conclude that our experimental design was successful in creating an AB that was modulated by the probability of the categorization assigned to T1.

The accuracy for T1 was reduced when T2 was rare, and this effect was stronger when the SOA was short. Furthermore, the accuracy of T1 was reduced at short SOA (when T1 was rare). These results indicated that the processing of T1 was influenced by the processing of T2. This result could be explained by a central capacity sharing model (Navon and Miller, 2002; Tombu and Jolicœur, 2003; Shapiro et al., 2006), if we assume that when the difficulty of T2 processing was increased (rare T2), more capacity would be dedicated to T2 than when T2 processing was easier (frequent T2). Consequently, less capacity would be available for T1 processing, leading to errors in the report of T1.

4. Introduction to Experiment 2

We modified the experimental design developed in Experiment 1 so we could measure the N2pc in the ERP to T2 during the task, and the frequency-related P3 differential response in the ERP to each target. This enabled us to explore the effects of a central load created by processing T1 on the deployment of

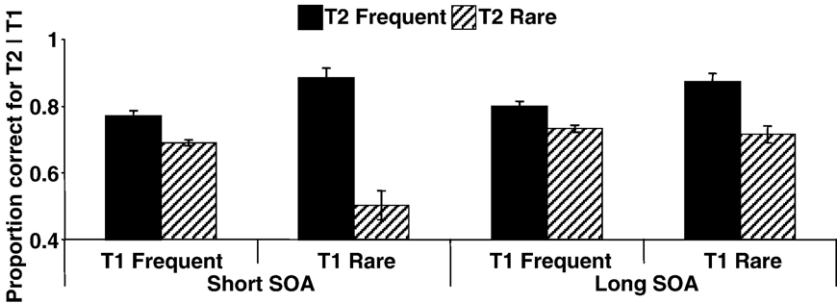

Fig. 1 – Success rate in the report of T2 in Experiment 1, for the trials where T1 was successfully reported. The error bars represent one standard deviation.

visual spatial attention to T2, and to monitor concurrently whether the frequency manipulation had the desired effect at a central stage of processing or at different stages. The results of Experiment 1 showed that the same patterns of results were obtained regardless of the order of presentation of T1 and T2 along the vertical and horizontal midlines (i.e., vertical-horizontal vs. horizontal-vertical). Given that order did not matter, we fixed the orientation of T1 to the vertical midline and of T2 to the horizontal midline, in order to evoke an N2pc to T2 in every trial. Stimuli presented on the vertical midline cannot produce an N2pc. The N2pc requires that attention be deployed to the left or to the right visual field, which, in turn, produces a left–right lateralized interhemispheric ERP difference. In order to maximize our ability to detect changes in the N2pc to T2, as a function of differences in the response probability associated with T1, we presented T2 lateralized to the left or right in every trial. This required that T1 be presented on the vertical midline on every trial. As shown in Experiment 1, however, the behavioral results were equivalent for the two types of trials (T1 on the vertical midline with T2 on the horizontal midline vs. T1 on the horizontal midline with T2 on the vertical midline), and thus the AB does not depend on this difference, allowing us to concentrate on the case in which T2 was on the horizontal midline.

We also fixed the SOA at 350 ms. The use of a fixed SOA and of stimuli having identical visual characteristic and occurrence probability ensured that the visuo-spatial characteristics of T1 in the high central attention load condition (when T1 is in the rare response category) were equivalent to those in the low central attention load condition (when T1 is in the frequent response category). Consequently, any effects of T1 processing on the visuo-spatial treatment of T2 would be attributed to the difference in central attention load created by the categorization of T1. In particular, an effect of the probability manipulation for T1 on the N2pc elicited by T2 could not be due to visual capture by T1, *per se*, given that the same stimuli and SOA were used for the two T1 response probability levels. If visual spatial attention was captured for a longer time by T1 in the low-probability condition, it would have to be the result of the categorization assigned to T1 and the subsequent increase in difficulty in updating working memory for T1 associated with a rare categorization. We assume these effects reflect central attentional capacity limitations, as demonstrated by Crebolder et al. (2002) for similar ranges of probability levels. Therefore, consistently with evidence produced in prior work (Dell'Acqua et al., 2003a,b, 2005; Vogel et al., 1998), we predicted that increases in central load would be reflected in enhanced P3 activity elicited by a T1 associated with an infrequent response relative to a T1 associated with a frequent response. Furthermore, we expected that the P3 to T2 would be significantly reduced under conditions leading to a larger AB (namely, when T1 was associated with the infrequent response).

5. Results of Experiment 2

The staircase procedure for T2 converged to an average presentation time of 77.8 ms for T2. Fifteen subjects out of 16 reached the minimal T2 duration (50 ms) during the recording. Consequently, the staircase did not succeed to keep the mean accuracy for T2 when T1 and T2 were frequent below 87% (the mean accuracy was 92.4% in this case).

5.1. *Behavior*

The mean accuracy for T1 in each condition is listed in Table 1. Accuracy for T1 decreased when T1 was rare (91.2%) compared to when T1 was frequent (96.7%), $F(1,15)=13.46$, MSE = .00358, $p<.003$. However, this difference was stronger when T2 was frequent (decrease of 7.1% between frequent T1 and rare T1) than when T2 was rare (decrease of 3.9% between frequent T1 and rare T1), $F(1,15)=6.399$, MSE = .0006, $p<.024$.

The accuracy for T2 is shown in Fig. 2. Generally, we found the same pattern of results as in the short SOA condition of Experiment 1. The accuracy for T2 was reduced both when T1 was rare, $F(1,15)=20.17$, MSE = .003, $p<.0005$, and when T2 was rare $F(1,15)=45.98$, MSE = .02, $p<.0001$. More importantly, the accuracy for T2 decreased more as a function of the frequency of T2 when T1 was rare than when T1 was frequent, leading to a significant interaction between those two factors, $F(1,15)=15.44$, MSE = .008, $p<.002$.

Subsequent analysis of the accuracy for T2 indicated that when T1 was rare, the accuracy for T2 was dramatically reduced when T2 was rare compared to when T1 was frequent, $F(1,15)=18.32$, MSE = .0098, $p<.0007$. However, when T1 was frequent, the accuracy for T2 was slightly increased when T2 was rare compared to when T2 was frequent, $F(1,15)=3.93$, MSE = .0012, $p<.066$.

5.2. *T2-locked ERP: N2pc*

The N2pc waveforms are shown in Fig. 3. These waveforms were computed from the following recording site, O1, O2, PO3, PO4, PO7, PO8, P5, P6, P7, and P8, by subtracting the waveform measured at the electrode over the ipsilateral hemisphere (ipsilateral to the target) from the corresponding electrode over the contralateral hemisphere (contralateral to the target), see Experimental procedures for details. The frequency manipulation led to different numbers of trials across conditions. Therefore, using a two-way ANOVA with frequency of T1 and frequency of T2 as a factor would have led to Np2c waveforms computed from only 100 trials (50 with T2 on the left and 50 with T2 on the right), before artifact rejection. This

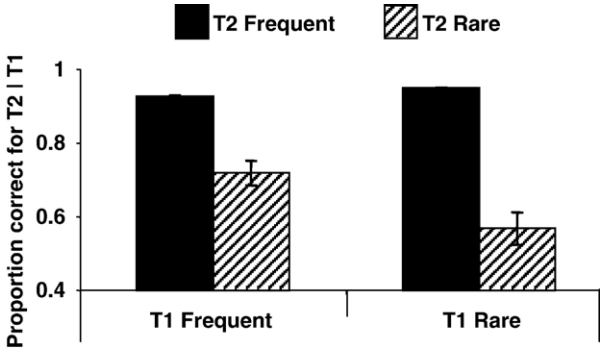

Fig. 2 – Success rate in the report of T2 in Experiment 2, for the trials where T1 was successfully reported. The error bars represent one standard deviation.

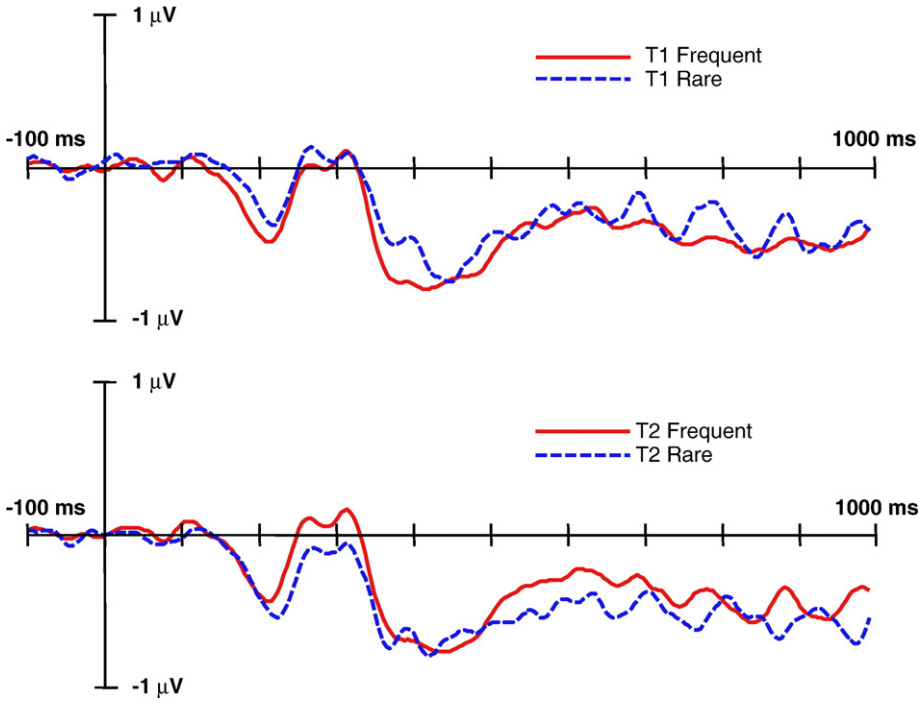

Fig. 3 – N2pc and SPCN subtraction waveforms, calculated from the pooled O1, PO3, PO7, P5, P7, and pooled O2, PO4, PO8, P6, and P8 responses. The top panel shows the N2pc and SPCN to T2 when T1 was rare and when T1 was frequent. The bottom panel shows the N2pc and SPCN to T2 when T2 was rare and when T2 was frequent.

is insufficient to give a stable N2pc. Therefore, we computed two separated one-way ANOVAs, one where the waveforms were averaged according to the frequency of T1 and one where they were averaged according to the frequency of T2. This averaging method allowed us to compute the N2pc and the SPCN with at least 400 trials (200 with T2 on the left and 200 with T2 on the right) prior to artifact rejection.

The amplitude of the N2pc component was measured by computing the mean amplitude in a 150–300 ms time window. The N2pc to T2 was reduced when T1 was rare, compared to when T1 was frequent, $F(1,15)=5.31$, $MSE=.0898$, $p<.036$. Interestingly, the frequency of T2 itself had the opposite effects on the N2pc, $F(1,15)=3.98$, $MSE=.123$, $p>.064$. Although only marginally significant, this last results indicated that the different number of trials used to calculate the T1 rare N2pc could not explain the reduction of the N2pc in this case, because the T2 rare condition (which is calculated with 400 trials) produced a larger N2pc than the T2 frequent condition (which is calculated with 1200 trials prior to artifact rejection).

There was some suggestion of latency differences across conditions. To measure the latency of the onset and of the offset of the N2pc component, we used a jackknife procedure (Miller et al., 1998). Instead of measuring the latency of the component on each of the 16 subject waveforms, 16 averages of all subjects except one (each subject is excluded once) were computed. These waveforms are more stable than the individual subject waveforms, allowing for a more stable estimate of the latency measure. The latency of each of theses average waveforms was then measured by taking the point where half of the amplitude of the peak was reached. These latencies were then submitted to an ANOVA, and the F values were corrected appropriately (see Miller et al. (1998) for the derivation of the appropriate correction). The onset of the N2pc was delayed by 16 ms in the T1-rare conditions compared to the T1-frequent conditions. This difference was marginally significant, $F(1,15)=3.15$, $p<.097$. However, the onset of the N2pc components was not delayed when T2 was rare, compared to when T2 was frequent, $F(1,15)=.56$, $p>.45$. The offset of the N2pc followed the opposite pattern. The offset of the N2pc component was not affected by the frequency of T1, $F(1,15)=.05$, $p>.8$, but it was delayed by 13 ms when T2 was rare, $F(1,15)=3.27$, $p<.091$. In brief, although the latency differences did not reach the traditional significance threshold, there was a suggestion that the N2pc ended later when the current target was rare and that it started later when the preceding target was rare.

5.3. *T2-locked ERP: SPCN*

The amplitude of the SPCN was measured from the same difference waveforms used to estimate the N2pc, but with a 350–600 time window. As for the N2pc, the amplitude of the SPCN to T2 was reduced when T1 was rare, compared to when T1 was frequent, $F(1,15)=4.52$, $MSE=.497$, $p<.05$. Furthermore, the frequency of T2 did not influence the SPCN, $F(1,15)=1.39$, $MSE=.0928$, $p>.25$.

5.4. *T1-locked and T2-locked ERP: P3*

The P3 waveforms at Oz, Pz, and Cz are shown in Fig. 4. Analyses were performed at several electrode sites with similar results. We report here the results for electrode Pz, which was an electrode near the peak amplitude of the P3

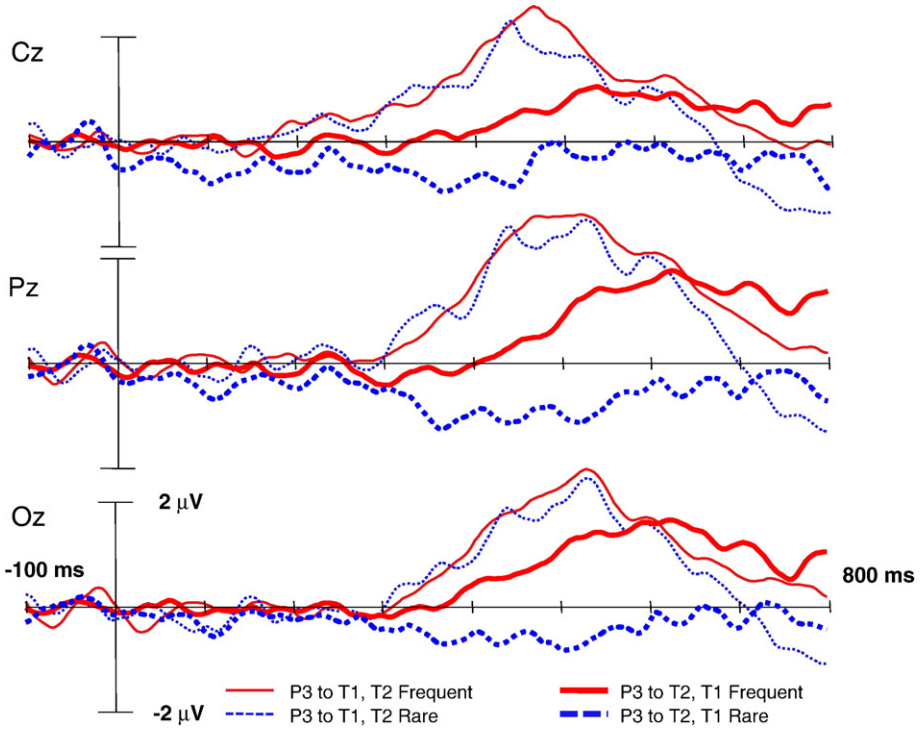

Fig. 4 – P3 subtraction waveforms (infrequent–frequent), for electrodes Cz, Pz, and Oz, for T1 (as a function of the frequency of T2) and for T2 (as a function of the frequency of T1). See text for further details.

wave. Each of these waveforms was computed by subtracting the ERP to the frequent target from the ERP to the rare target. For each target (T1, T2), two P3 waveforms were computed, averaging over the frequency of the other target. The amplitude of the P3 was measured with a 300–650 ms time window. It can be observed that the P3 to T1 was not influenced by the frequency of T2, $F(1,15)=.23$, MSE = .589, $p>.64$. However, the P3 to T2 was strongly reduced in amplitude when T1 was rare, $F(1,15)=28.08$, MSE = .637, $p<.0001$. In fact, no P3 was visible for T2 when T1 was rare, but a negativity was observed (average amplitude of −.827 μV; $t(15)=-3.678$, $p<.002$).

Furthermore, the P3 to T2, when T1 was frequent, was reduced compared to the combined P3 to T1, $F(1,15)=25.72$, MSE = .384, $p<.0001$. The latency of the P3 seemed also delayed in this case. We used a jackknife procedure adapted from Miller et al. (1998) to evaluate the difference in latency of the P3 to T1 and the P3 to T2. Here, the latency of the P3 components was then measured on theses waveforms by measuring the point where half of the area of the P3 was reached (using a 0–1000 ms time window). Everything else was the same as the preceding latency analysis. This latency measure, also referred as fractional area measure, is very stable and resistant to noise (Luck, 2005). There was a significant latency difference between the P3 to T1 and the P3 to T2, $F(1,15)=52.14$, $p<.0001$.

6. General discussion

The goal of this study was to determine whether central capacity limitations implicated in the AB phenomenon can impair the ability to deploy visual spatial attention. We wanted to examine this issue under conditions that avoided possible differences in visual capture by T1, differences in initial attentional state, and differences in task, across Task1 and Task2, in the various experimental conditions. To achieve these conditions, we capitalized on an earlier finding, reported by Crebolder et al. (2002), in which a larger AB was caused by processing a less frequent T1 stimulus. In Experiment 1, we extended the work of Crebolder et al. by showing that a similar probability effect on the AB can be observed with stimuli that are equiprobable (all stimuli had a probability of .25), but for which the associated categorization was either frequent (.75) or infrequent (.25). This extension of the Crebolder et al. (2002) results allowed us to use the effect of T1 frequency on the AB while nonetheless presenting stimuli that were equiprobable, and in all other ways completely equivalent. This was an important feature that enabled us to examine ERP effects, in Experiment 2, that were not compromised by stimulus confounds.

In Experiment 2, we found that the probability of the response associated with T1 both influenced accuracy of report of T2, and modulated the amplitude of the N2pc to the second target. A higher central load created by the need to process a T1 stimulus associated with a less frequent categorization reduced the amplitude of the N2pc and of the SPCN, and abolished the P3 to T2. In our view, the central processing load required to categorize T1 reduced the efficiency of the spatial selection of T2, leading to a smaller N2pc, and consequently to a weaker representation of T2 in VSTM, leading to a smaller SPCN (Dell'Acqua et al., 2006; Jolicœur et al., 2006a,b). It appears that this degraded representation was also insufficient to create a stable representation in (non-visual) working memory, resulting in the abolition of the P3, and reduced behavioral accuracy for T2.

The patterns of behavioral results for both Experiment 1 and Experiment 2 indicated that the processing of T2 was

impaired when T1 was associated with a less frequent categorization, but only at short SOA (250 ms for Experiment 1 and 350 ms for Experiment 2). In both case, the accuracy of T2 was reduced by a higher central load, but only when T2 was rare. When T2 was frequent, accuracy was increased by the higher central load. However, it is clear that this increase in accuracy resulted from a bias to guess the frequent response when subjects had not perceived T2.

The presence of an N2pc to T2 in both T1-frequent and T1-rare conditions suggests that the subjects successfully used the selection cue (the color) to identify which of the two stimuli was the target. This was also the case in Jolicœur et al. (2006a), in a control AB experiment where the second task was to identify the side of the masked colored target in the presence of a masked colored distractor. The success rate for the localization of T2 was very high even in the conditions where an AB is usually observed for the identification of T2, indicating that the subjects were able to detect the color of the targets even when they were unable to identify the target.

The reduction of the N2pc in the T1-rare condition, however, indicated a dependency of the deployment of spatial attention on the available processing capacity of central attentional mechanisms. Previous results showed more dramatic effects of the AB on spatial attention (Dell'Acqua et al., 2006; Jolicœur et al., 2006a,b). The smaller magnitude of the reduction of the N2pc in the current paradigm could be explained in several ways. First, the effect size of the frequency of T1 in Crebolder et al. (2002) was small, especially at a ratio of 4:9, which is close to the 1:3 ratio we have here. Furthermore, the 1:3 ratio here was manipulated independently for both T1 and T2, leading to at least a rare target on 43.45% of the trials. This combined ratio could have increased the perceived frequency of the rare target and perhaps reduced the overall impact of the frequency manipulation for T1. Second, the control condition here, the T1-frequent condition, was expected to cause a central load by itself because of the short SOA between T1 and T2. This expectation was confirmed by the amplitude reduction and the delay of the P3 to T2 compared to the P3 to T1 (see Fig. 4; presumably T1 suffers very little AB interference, and so the P3 to T1 provided a measure of the P3 in the absence of AB interference). Consequently, it could be assumed that the N2pc in the T1 frequent conditions is already affected by the central load caused by a frequent T1.

The SPCN to T2 was clearly visible in both the T1-frequent and T1-rare conditions. Indeed, some visual information entered in VSTM following the presentation of T2. According to Vogel and Machizawa (2004), a greater amplitude of the SPCN signifies more items in VSTM. Robitaille and Jolicœur (2006) found evidence that the SPCN was of higher amplitude when lateralized stimuli were masked, suggesting that the mask might act as a second item in VSTM. In the present Experiment 2, the VSTM was probably loaded by a combination of items consisting of the target stimulus and the mask. The less efficient spatial selection in the T1-frequent conditions would have lead to a weaker representation of T2 in VSTM, but to an equivalent representation of the mask, which would explain the small reduction of the amplitude of the SPCN.

The finding that the N2pc to T2 was increased when T2 was associated with a low-frequency response could be seen as conflicting with our initial claim that we created a design in which the visual processing of all the targets was equivalent. Indeed, if the spatial deployment of attention to T2 was increased when T2 required the rare response, we might also assume that the spatial deployment of attention to T1 was affected by the frequency of T1. One could argue that the reduction of the N2pc to T2 when T1 was rare was caused by this greater deployment of attention to T1 in this case. Indeed, if spatial attention was more engaged or engaged for a longer period of time on a rare T1, there may have been less time to redeploy spatial attention toward T2, and thus the N2pc to T2 would be reduced is this case. However, we consider this result, the increase in the amplitude of the N2pc to a target when the response associated to it was rare, as supporting our primary hypothesis, namely that the deployment of spatial attention depends on central attention. Consider first that all four possible stimulus squares were equivalent in term of visual features and in term of probability of occurrence (each had a probability of .25). Furthermore, the square considered as the rare target for a specific subject was not the same for another subject—the stimulus which was the rare target was counterbalanced across subjects. These considerations suggest to us that the probability effects are very unlikely to reflect influences at sensory or perceptual levels of processing.

Interestingly, we did not find any modulation of the infrequent minus frequent difference wave (Fig. 4, used to estimate the P3) prior to 300 ms, which indicated that the early visual components (P1, N1, N2) were equivalent for the rare-response stimulus and for the frequent-response stimuli. However, the deployment of attention toward a rare-response target (T2) produced a larger N2pc to T2. The analysis of the latency of the N2pc components suggested that spatial attention may have dwelled longer on a rare target than on a frequent target. Previous evidence suggests that the locus of frequency effects (in this range of frequencies) is at or after the bottleneck in the psychological refractory period paradigm (Crebolder et al., 2002), that is, at a post-perceptual, central, stage of processing. In Experiment 2, spatial attention may have dwelled longer on T1 when T1 was rare, which could be the cause of the reduction of the N2pc to T2. Indeed, the reduction of the N2pc to T2 when T1 was rare was accompanied by a delay in the onset of the N2pc. However, this inability to disengage earlier from T1 was caused by the central attentional load created by T1, not by the visual processing of T1, per se (because all T1 stimuli were perceptually equivalent). In sum, according to this interpretation, our results indicate that the deployment of visual spatial attention depends on central attention.

The P3 to T2 was abolished when T1 was rare compared to when T1 was frequent. This results mirror the one obtained by Vogel et al. (1998), by Vogel and Luck (2002), and by Dell'Acqua et al. (2003a,b, 2005). The P3 has been argued to reflect a process that updates the contents of working memory (Donchin and Coles, 1988). On this view, the reduced P3 amplitude for T2 when T1 was associated with a rare response suggests that subjects had a greater difficulty to create a representation of the second target in working memory. These results provide strong converging evidence for our interpretation that a T1 stimulus associated with a less-frequent categorization produced a larger AB. The delay in the onset latency of the P3 at short SOA (for the frequent T1 condition),

compared with the onset latency of the P3 for T1 itself, provides further evidence that processing T1 causes a delay in the categorization of T2, as suggested by models of the AB that ascribe an important role to the central processing of T1 by capacity limited mechanisms (Chun and Potter, 1995; Jolicœur, 1999a,b; Sessa et al., 2007; Vogel and Luck, 2002).

It is worth pointing out that, over and above the crucial findings concerning the inverse correlation between central load and visuo-spatial processing efficiency, the present P3 results corroborate and extend in an important way previous results obtained by monitoring the ERP and the magnetoencephalographic (MEG) reflections of both targets displayed using the RSVP technique. Kessler et al. (2005a,b), and Shapiro et al. (2006) for instance, have recently shown an increasing trade-off in the magnitude of M3 responses elicited by T1 and T2 (the MEG equivalent of the P3 ERP component observed with electrophysiological recordings) as the temporal interval between their onsets was reduced. Incidentally, the overlap of M3 peak responses was observed in regions held to be of interest for identification processes (e.g., infero-temporal regions), but not in regions more likely involved in sequencing (e.g., fronto-parietal regions). In an attempt to establish a quantitative link between the P3 to T1 and AB magnitude estimated behaviorally as the percentage of correct T2 identification, McArthur et al. (1999) found that increased P3 responses to T1 were associated with increased AB effect magnitude. One of the most extensive and recent examination of the relationship between P3 responses to targets and AB magnitude is probably that carried out by Martens et al. (2006), whose focus was on the difference at the individual level between blinkers (i.e., people particularly prone to 'blink' under RSVP condition) and non-blinkers (i.e., people with small or no AB effect). The ERP recording technique in the work of Martens and colleagues included online monitoring of the ERP response during the entire RSVP stream presentation, so as to capture the ERP reflection of T1 and T2 across the different time intervals separating their onsets. The results of interest for the present discussion were those mirroring the MEG results of Kessler and colleagues. That is, the results revealed the tendency of P3 responses to T2 to be partially suppressed and postponed in blinkers relative to non-blinkers, thus providing the ERP equivalent of the trade-off between P3 responses to T1 and T2 that the MEG results described above had illuminated. The extension provided by the present investigation stays in the method used to isolate P3 activity in the ERP elicited by targets under the present circumstances. Contrary to the work just described, in which this technique was not adopted, we used a subtraction method that was specifically designed to isolate P3 activity that was associated to the systematic manipulation of the frequency of the response to a target class. This method, apart from enabling a more accurate individuation of the portion of the ERP specifically responsive to the variable manipulated, allowed us to 'clean' the P3 response from the potentially spurious cumulative EEG activity that is certainly generated when more than one stimulus is displayed to subjects. Our experimental design allowed us to isolate that portion of the P3 that was specifically related to the frequency of the classification of the targets, independently of the frequency of presentation of individual stimuli, pointing to a central locus of processing, as we have discussed at length before. With these considerations in mind, we interpret the P3 results as a parallel, independent test that the frequency manipulation adopted herein affected central processing stages, and this suggests strongly that the attenuation of the N2pc to T2 observed in the results is a relatively 'pure' reflection of engaging, in close temporal contiguity, mechanisms devoted to the control of central attention and mechanisms devoted to the control of visuo-spatial attention.

7. Experimental procedures

7.1. *Experiment 1*

7.1.1. *Subjects*

Sixteen volunteers, 14 women, aged between 19 and 26 (average 22), were paid for their voluntary participation. They reported no neurological problems, normal or corrected-to-normal vision, and normal color vision. We obtained informed consent from each subject at the beginning of the experiment.

7.1.2. *Stimuli*

The stimuli were presented on a 15-inch color cathode-ray tube (CRT) driven by a microcomputer running MEL 2.01 software at 60 HZ in 640×480 pixel mode. The stimuli were outline squares, subtending 1.2° of visual angle, .2° thick, shown in pink or green. Each squares contained a gap (.2° thick), in the middle of one of the sides. The squares were always presented in pairs (one in left visual field, one in right visual field), each square centered 3.6° off-center. A fixation point (.2°) was present at the center of the display. Counterbalancing (explained below) ensured that the small difference in luminance across the green and pink stimuli could not have influenced the results.

7.1.3. *Procedure*

The sequence of events in each trial is illustrated in Fig. 5. Each trial began with two symbols at the center of the display, indicating (in reading order) if the answers for the last trial were correct (+) or incorrect (–). The subject started each trial by pressing the space bar on a standard computer keyboard. The + or – sign was then replaced by a small fixation point. The fixation point remained alone on the screen for 500 ms. The first stimulus pair then appeared and consisted of two squares, one pink and one green, presented 3.6° from the center of the screen. For half of the experiment, the first pair of stimuli was on the vertical midline. For the second half of the experiment, the first pair of stimuli was presented on the horizontal midline. This condition was blocked, that is, all vertical first trials were presented in one a part of the experiment, and all the horizontal first trials were presented in another part of the experiment. The order of the block was counterbalanced across subject. The squares remained on the screen for 100 ms and were immediately followed by a mask. The mask consisted of a ticker square (.4°) having a gap on each four sides, presented for 33 ms.

Either 250 or 850 ms after the onset of the first display, a second display appeared. This display also contained a pink and a green square, at 3.6° from the center of the screen. The square in this display was always orthogonally located relative to the

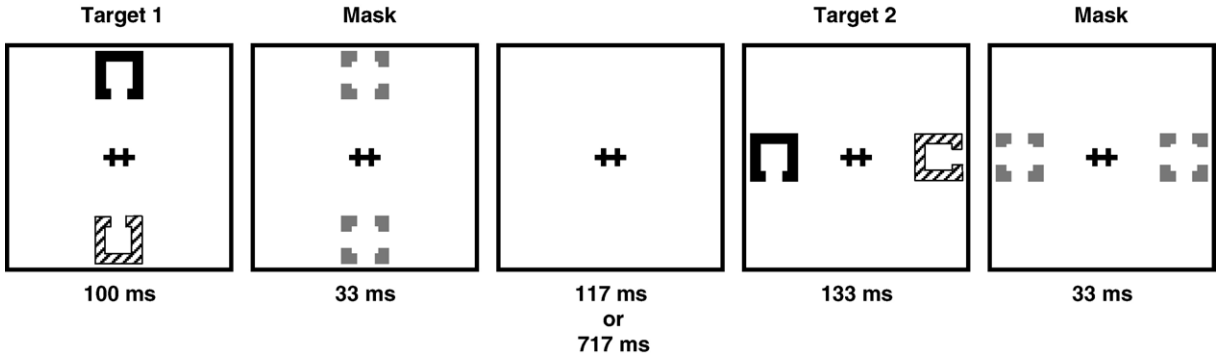

Fig. 5 – Sequence of events in each trial. The actual target and distractor were pink and green (equiluminant), followed by a grey mask.

first one, so when the first set of squares was presented on the vertical midline, the second set of squares was presented on the horizontal midline, and conversely when the first was on the horizontal, the second was on the vertical. The second display was also masked. The duration of the second target was adjusted with a staircase procedure, to ensure accuracy between 70 and 87%, for the second target, when the SOA was 850 ms. The duration of T2 was adjusted after each block (i.e., after the 32 practice trials at the beginning of each part and after the 128 trials of each of the two experimental blocks of each experiment part). At each adjustment, the number of video frames (16.7 ms each) for T2 was increased by one if the accuracy was lower than 70% and was decreased by 1 if the accuracy was higher than 87%.

For half of the subjects, the task was to report the location of the gap in the pink squares. For the other half, the task was to report the location of the gap in the green squares. Both responses had to be entered in order of target presentation at the end of the trial, using the keyboard. Subjects were aware that the reaction times were not recorded and they were instructed to focus on maximizing accuracy.

Each subject received a response-key mapping at the beginning of the experiment. The response-key mapping was identical for the first and the second response. The task was to determine the location of the gap in the target square and to respond as a function of this location (e.g., left vs. not-left). Given that each gap position was equally likely to occur, there was no frequency manipulation for the stimuli themselves, but only for the responses produced by the subject. One response was more frequent (e.g., not-left, 75% of trials) than the other (e.g., left, 25% of trials). The gap location mappings (4 different ones) were counterbalanced across subjects. Each subject executed two blocks of 256 trials (512 trials total), in which trials with different SOAs, T1 gap locations, and T2 gap locations were presented in a randomized order.

7.2. *Experiment 2*

7.2.1. *Subjects*

Of the 23 subjects who were tested, 7 were excluded form the analyses because they had an insufficient number of trials after the artifacts rejection procedures. The remaining 16 subjects (12 women) were between 18 and 26 years of age (average 20.6), had a normal or corrected to normal vision, normal color vision, and declared having no neurological disease.

7.2.2. *Stimuli*

The stimuli were displayed on a 17-inch computer screen located 57 cm in front of the subject. The stimuli were identical to those in the first experiment, except that their size was 13% bigger due to a larger screen. To ensure an equal initial response of the brain to the colored stimuli, the luminance of the stimuli was equated. The luminance and chromaticity were measured with a Minolta CS-100 chromameter. The luminance of the green was 19.7 cd/m^2 (x=.292, y=.550) CIE (x, y) chromaticity coordinates (Wyszecki and Stiles, 1982); that of the pink was 18.5 cd/m^2 (x=.386, y=.279), the fixation point was 30.8 cd/m^2 (x=.280, y=.302), and the background was .10 cd/m^2 (x=.449, y=.442).

7.2.3. *Procedure*

The procedure was almost identical to the one used in the first experiment except for the following: the duration of T1 was 133 ms, the SOA was fixed at 350 ms, and T1 was always vertical and T2 horizontal. Each subjects had 32 practice trials followed by 25 blocks of 64 trials, for a total of 1600 experimental trials. For this experiment, the staircase procedure adjusted the duration of T2 in order to achieve an accuracy between 80% and 87%, for the conditions where T1 and T2 were both frequent.

7.2.4. *ERP recording and analysis*

The recordings were made with a BioSemi Active-two system, with 70 active Ag–AgCl scalp electrodes, 64 of which were positioned using the extended International 10–20 system (see Pivik et al., 1993), two were at the mastoids, and 4 for the electrooculogram, in an electrically shielded and dimly lit room. The EEG was algebraically re-referenced to the average of the left and right mastoids. The electrooculogram (EOG) was recorded with active Ag–AgCl electrodes placed at the left and right canthi and above and below the left eye. HEOG was obtained by subtracting the signal at the left electrode from the signal recorded at the right electrode. VEOG was obtained by subtracting the signal at the electrode above the left eye from the signal at the electrode below the left eye. The signals were amplified, low-pass filtered with a cut-off frequency of 67 Hz, and digitized at 256 Hz during the recording. They were filtered again, during post-recording analysis, using a Butterworth zero-phase high-pass filter with a cut-off frequency of .05 Hz and then a Gaussian low-pass filter with a cut-off frequency of 30 Hz. Eye blinks and eye movements were detected using an automated function.

Trials with eye blinks and eye movement were removed during post-recording analysis. Furthermore, if an electrode contained a recording artifact during a trial, this trial was also removed from the analysis. As a further precaution to ensure that subjects did not move their eyes in the direction of the target (despite screening individual trials on the basis of the HEOG), separate average HEOG curves were computed for left-target trials and for right-target trials. Any residual tendency of the subject to move their eyes toward the target, for the trials included in the analysis, would produce systematic deviations in these average HEOG waveforms. For each subject, we averaged all the left trials and all the right trials. The maximal amplitude of the difference between the HEOG to the left trials and the HEOG to the right trials for any given subject was 2.3 μV. This indicated that on average, the eye moved less than .15° toward the target for this subject (Lins et al., 1993).

For the N2pc and the SPCN, average waveforms were computed at each scalp electrode site for each condition with a 100 ms pre-stimulus baseline and a 1000 ms post-stimulus period relative to the onset of T2. The epochs were baseline corrected based on the mean activity during the 100 ms pre-stimulus period, for each electrode site. The average ipsilateral and contralateral waveforms were computed for all lateralized posterior electrode pairs. However, given that the results were similar across several sites, and that we are more interested in differences between conditions rather than differences between electrode sites, we pooled the O1, PO3, PO7, P5, and P7 electrodes together and O2, PO4, PO8, P6, and P8 together. We first computed an average ipsilateral waveform by averaging the waveform for the left pool of electrodes for trials in which the target was on the left with the waveform for the right pool of electrodes for trials in which the target was on the right. Similarly, we computed an average contralateral waveform by averaging the right-sided response to left targets with the left-sided response to right targets. These waveforms were then subtracted (contralateral – ipsilateral) to produce an N2pc/SPCN difference waveform for each condition.

For the P3, a 100 ms pre-stimulus baseline and an 800 ms post-stimulus window were computed. For each target, four waveforms were averaged, according to the frequency of both target response category. The data were collapsed across T2 on the left and T2 on the right trials. The P3 to T1 were calculated by subtracting the frequent T1 waveform from the rare T1 waveform. The P3 to T2 were calculated by subtracting the frequent T2 waveform form the rare T2 waveform.

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