



Cuing and stimulus probability effects on the P3 and the AB

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Abstract

Two experiments were conducted to investigate the relation between the attentional blink (AB), a deficit in reporting the second of two targets when it occurs 200–500 ms after the first, and the P3 component of the event-related potential. Consistent with the view that the AB reflects a limited ability to consolidate information in working memory and that the P3 reflects working memory updating, increasing the amplitude of the P3 elicited by a first target (T1) by varying T1 probability (Experiment 1) or T1 cue validity (Experiment 2) led to an increase of the AB. Overall, the P3 elicited by T1 was greater when T2 was not identified than when it was. However, the correlation between P3 and AB magnitude across participants was not significant, leaving open the question of how direct the relationship between the P3 and the AB is.

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1. Introduction

In the past decade, the attentional blink (AB) paradigm has been used effectively to study the temporal dynamics of visual attention. Defined as a deficit in reporting the

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second of two targets presented in rapid serial visual presentation (RSVP) when that target occurs 200–500 ms after the first one (e.g., Raymond, Shapiro, & Arnell, 1992), the AB has been attributed to a temporary lack of attentional capacity needed to consolidate relevant information in working memory (e.g., Jolicoeur & Dell'Aqua, 1998). Support for this hypothesis has been provided by both behavioral (e.g., Martens, Wolters, & van Raamsdonk, 2002; Shapiro, Driver, Ward, & Sorensen, 1997) and EEG (e.g., Kranczoch, Debener, & Engel, 2003; Luck, Vogel, & Shapiro, 1996; Rolke, Heil, Streb, & Hennighausen, 2001; Vogel, Luck, & Shapiro, 1998) studies of the AB. Most importantly, in EEG studies, the P3 component of the event-related potential (ERP) commonly associated with the updating of working memory (e.g., Donchin, 1981; Donchin & Coles, 1988) is absent or suppressed for targets that are not identified due to an AB, whereas perceptual (N1, P1) and semantic ERP components (N400) evoked by the second target are unaffected by the AB (e.g., Vogel et al., 1998; Kranczoch et al., 2003; Rolke et al., 2001).

Whereas the presence of the P3 is assumed to reflect updating in working memory, the amplitude of the P3 has been assumed to reflect demands on attentional resources (e.g., Kramer & Spinks, 1991; Sirevaag, Kramer, Coles, & Donchin, 1989; Wickens, Kramer, Vanasse, & Donchin, 1983). Thus, the lack of a P3 component for unidentified targets suggests that processing resources were not available or not called into action. In other words, the task of identifying T1 seems to tie up attentional resources that are needed for the task of identifying T2. If this assumption is correct, it follows that the larger the amplitude of the P3 that is elicited by T1, the fewer resources will be available for the processing of T2.

Fell, Klaver, Elger, and Fernández (2002) have also suggested that the magnitude of the P3 elicited by T1 is related to the AB. In particular, they hypothesize that the P3 evoked by T1 might suppress the early gamma response to T2 that is needed for the initialization of the P3 and memory consolidation for T2. The early evoked gamma response is known to occur 100 ms post-target (Hermann & Mecklinger, 2000, 2001), and should therefore coincide with the T1-related P3 when T2 is presented at a lag of 200–500 ms (typically the interval during which the AB occurs). Given that slow positive potentials such as the P3 have been associated with decreased cortical excitability (Elbert & Rockstroh, 1987), a process such as the early gamma response might indeed be suppressed, preventing successful consolidation of T2.

Using whole-head magnetoencephalography (MEG), Shapiro, Schmitz, Martens, Hommel, and Schnitzler (2006) recently showed that the probability of missing the second target in an AB task can indeed be predicted from the amount of attentional resources devoted to processing the first target, as measured by the corresponding neural activation. In other words, they found a significant positive correlation between an individual's level of activation in response to T1 and the magnitude of the AB found for that individual. Both the latency and region of brain responses found in this MEG study coincided with the latency and presumed generator sites of the P3.

Additional evidence supporting a link between the amplitude of the P3 elicited by T1 and the magnitude of the AB has been provided by McArthur, Budd, and Michie (1999), who showed that increasing task difficulty (by increasing the number of possible targets) for T1 increased the magnitude of both the AB and the P3 evoked by T1. McArthur et al. established a link between the P3 evoked by T1 and the AB by comparing the P3 evoked in a single-target (T1 only) condition with the AB in a dual-target (T1 and T2) condition. In the experiments reported here, we, too, vary the amplitude of the P3 to the first target experimentally and examine the effects of this manipulation on the magnitude of the AB.

However, we also recorded ERPs on dual-target trials in order to allow a direct comparison between trials in which an AB did or did not occur. The technique of averaging blink and non-blink trials separately has effectively been used before to show that the T2-related P3 is suppressed on blink trials (e.g., Kranczoch et al., 2003; Rolke et al., 2001; Kranczoch, Debener, Schwarzbach, Goebel, & Engel, 2005; Shapiro et al., 2006). Here we look at the P3 evoked by T1 to see whether the amplitude of the T1-related P3 is linked to the chance that T2 can be successfully reported. In contrast to Shapiro et al., we systematically manipulated the magnitude of the T1-related P3 in order to determine whether there were corresponding changes in the magnitude of the AB.

Two established methods of manipulating P3 magnitude were employed in the present study, one based on stimulus probability and one based on cue validity. In Experiment 1 we varied the probability of T1 by presenting a T1 that was a frequent (occurring on 75% of the trials) or infrequent (occurring on 25% of the trials) letter,¹ and in Experiment 2 cue validity was manipulated by presenting a cue which validly indicated the identity of the forthcoming T1 on 75% of the trials.

2. Experiment 1

It is well established that an infrequently presented target induces a greater P3 than a frequently presented target (e.g., Kok, 2001; Öhman, 1979). In this experiment, we examined whether applying this frequency manipulation to T1 also produces a greater P3 in our task of identifying letters in a stream of digit distractors and if this is accompanied by a greater AB. T1 was either a frequent (occurring in 75% of the trials) or infrequent (occurring in 25% of the trials) letter target. If the processing required to consolidate T2 is interfered with the P3 elicited by T1, the average magnitude of the AB should be greater when T1 is infrequent.

2.1. Method

2.1.1. Participants

Twenty-four undergraduate students from the University of Groningen, aged 18–40 (mean age 20.6, 17 females), participated in the experiment for course credit. Informed consent was obtained prior to the start of the experiment. All participants reported that they were right-handed, had normal or corrected-to-normal vision, and had no history of neurological problems. Participants with corrected vision wore glasses instead of contact lenses in an effort to reduce the frequency of eye blinks.

2.1.2. Stimuli and apparatus

The generation of stimuli and the collection of responses were controlled using E-prime 1.0 software (Schneider, Eschman, & Zuccolotto, 2002) running under Windows 98 on a PC with a 1.2GHz processor. Stimuli were digits (excluding 1 and 0) and consonants

¹ After running the first experiment, we learned that Crebolder, Jolicoeur, and McIlwaine (2002, Experiment 5) had conducted a similar experiment, showing that decreasing the relative probability of T1 in AB task increased the magnitude of the AB. However, as the studies by Crebolder et al. (2002) contained only four trials per participant and lag in the lowest T1 probability condition and no EEG was measured, we present Experiment 1 as a replication and extension of their study.

(excluding ‘M’, ‘Q’, ‘W’, and ‘Z’), subtending 0.2° by 0.2° of visual angle at a viewing distance of approximately 75 cm. The stimuli were presented in black (4 cd/m^2) on a white background (140 cd/m^2), presented in 10-point Courier New font, on a 15-in. monitor at a refresh rate of 75 Hz.

2.1.3. Procedure

The experiment consisted of a practice block of 32 trials, and four experimental blocks of 192 trials each. Thus, 48 infrequent T1 trials per lag and 144 frequent T1 trials per lag were presented. At the start of each block, four additional warm-up trials were provided which were not included in the analyses. A short break was given after each block, and a long break (30 min) was given halfway through the experiment.

Participants pressed the space bar to initiate a trial. After 100 ms, a fixation cross was presented in the middle of the screen for 650 ms. After a 100-ms blank interval, an RSVP stream was presented consisting of 18 stimuli. Each item in the RSVP stream was presented for 80 ms at the center of the screen. On 75% of the trials, two of the 18 characters were letters (T1 and T2), and the other characters were digits. On 25% of the trials only one target letter was present in the stream to provide a baseline of single-target performance. Digit distractors were randomly selected with the constraint that no single digit was presented twice in succession. Participants were instructed to identify any target letters among the digits in the stream. T1 randomly appeared as the fourth, fifth, or sixth item in the stream and was always an ‘N’ or an ‘R’. For half of the participants, T1 was the letter ‘N’ in 25% of the trials (infrequent T1 condition), and the letter ‘R’ in 75% of the trials (frequent T1 condition). For the other half of the participants, ‘N’ was the target on 75% of the trials and ‘R’ on 25% of trials. T2 was presented as the first, fourth, or tenth item following T1 (lag 1, 4, or 10, respectively). These specific lags were chosen on the basis of the literature and previous work in our laboratory. T2 is likely to be “blinked” (i.e., not identified) at lag 4, whereas at lag 1 and 10 little or no reduction in T2 accuracy is usually observed. T2 could be any consonant letter except ‘N’, ‘R’, ‘M’, ‘Q’, ‘W’, and ‘Z’, and it was always followed by at least two distractors. Both frequent and infrequent T1 trials were distributed equally across T2 lags.

Following the presentation of the RSVP stream, participants were asked to type the letters they had seen using the keyboard. Participants were encouraged to press the keys in the order in which letters had been presented, but responses were accepted and counted correct in either order. If a letter was not seen, the space bar was to be pressed instead. Participants were seated in a sound attenuated and dimly lit testing room, and they completed the task in one session, lasting approximately 90 min (excluding the long break).

2.1.4. EEG recording

EEG activity was recorded from tin electrodes mounted on an elastic electro cap organized according to the international 10/20 system. Electrode impedance was reduced to below $5 \text{ k}\Omega$. The signal was referenced against electrodes on the earlobes, and an electrode on the sternum was used as a common reference. The scalp electrodes used were FP1, FP2, F7, F3, FZ, F4, F8, FC5, FC1, FC2, FC6, T7, C3, CZ, C4, T8, CP5, CPZ, CP6, P7, P3, PZ, P4, P8, PO3, POZ, PO4, O1, OZ, O2, PO9, and PO10. In the final analysis, only the signal from the midline parietal site (electrode Pz) was used, where the P3 was largest and is typically reported. The other electrodes were used to obtain a clear scalp distribution of the various components to ensure the accurate detection of the P3 waveform.

The horizontal EOG was recorded from tin electrodes attached approximately 1–2 cm to the left and right of the outside corner of each eye. The vertical EOG was recorded from tin electrodes attached approximately 3 cm below the left eye and 1 cm above the brow of the left eye. The EEG and EOG were amplified using a physiological amplifier system (PAS) with a bandpass of 0.016–200 Hz. This signal was sampled with a frequency of 1 kHz. Later, the sample frequency was digitally reduced to 250 Hz by the data acquisition processor (DAP). To avoid aliasing effects the signal was first passed through a 60-Hz low-pass filter.

2.1.5. EEG data analysis

Trials containing movement artifacts, ocular artifacts, or amplifier saturation were removed from the analysis (a total of 14.2% of the trials, ranging from 0.3% to 63.1%, $SD = 21.8$, of the trials per participant²). The data were analyzed using the BrainVision Analyzer software. The ERPs were time locked to T1 with a length of 1000 ms, and calculated relative to a 200-ms pre-target baseline, yielding a total length of 1200 ms. Only trials on which T1 was accurately identified were included in the computation of the ERPs. The average amplitude of the P3 response to T1 was measured by taking the peak amplitude of the average waveforms by visual inspection for each individual subject. This was done to reduce between-subjects variance caused by individual differences in P3 latency. As an alternative way to estimate the P3 amplitude, specific windows were defined (based on visual inspection of the grand average) from which the mean ERP signal was calculated by averaging the voltage of each individual data point within the specified window.

A specific goal of the current experiment was to study the relation between the T1 probability effect and the amplitude of the P3. Therefore, seven participants for whom a clear P3 component could not be established were excluded from the EEG analyses, together with six additional participants whose behavioral data lacked a clear effect of T1 frequency on T2 (defined as T2|T1 accuracy at lag 4 being at least 5% lower in the infrequent than in the frequent T1 condition), thereby restricting the EEG analyses to data from 11 participants. Across conditions, ERP analyses were based on an average of 38 (lag 4 trials in which T1 was a correctly identified infrequent target) to 347 trials (trials in which T1 was a correctly identified frequent target).

2.2. Behavioral results

Percent correct for each target was computed for each lag as a function of T1 frequency. An initial analysis of variance (ANOVA) with frequency (frequent or infrequent T1) and lag (1, 4, or 10) as within-subjects factors conducted on percentage correct T2 identification given correct report of T1 revealed no significant effect of T1 frequency, $F(1, 23) = 2.24$, $MSE = 128.91$, $p = .148$, a significant effect of lag, $F(2, 46) = 29.27$, $MSE = 179.96$, $p < .001$, and a marginally significant T1 frequency \times lag interaction, $F(2, 46) = 2.56$, $MSE = 34.99$, $p = .089$. In contrast, a behavioral pilot experiment with 32 participants had revealed significant effects of T1 frequency on T2|T1 performance, $F(1, 31) = 4.52$, $MSE = 93.17$, $p = .04$, lag, $F(2, 62) = 39.67$, $MSE = 169.88$, $p < .001$, and a marginally significant T1

² For only one participant, more than 50% of the trials were lost due to artifacts. As the participant showed good performance for T1, a clear AB effect, a probability effect, and a P3 component, she was included in the analyses.

Table 1

Mean percentage correct report of T1 and T2 given the correct report of T1 in Experiment 1, as a function of lag and T1 frequency (standard deviation in parentheses)

Measure	Condition	Lag		
		1	4	10
T1	Frequent T1	64.1 (30.97)	75.1 (25.05)	75.2 (24.05)
	Infrequent T1	80.7 (18.84)	90.7 (9.74)	89.8 (3.29)
T2 T1	Frequent T1	83.1 (9.93)	64.5 (14.63)	75.2 (17.55)
	Infrequent T1	78.0 (12.93)	50.6 (13.81)	71.2 (20.41)

frequency \times lag interaction, $F(2, 62) = 2.72$, $MSE = 53.58$, $p = .073$, which is in line with the results reported by Crebolder et al. (2002, Experiment 5).

Given previous findings of a significant T1 frequency effect on T2 performance and our goal of studying the relationship between the T1 probability effect and the amplitude of the P3, we restricted the EEG analyses to those 11 participants for whom a clear P3 component and a clear effect of T1 frequency on T2 (defined as T2|T1 accuracy at lag 4 being at least 5% lower in the infrequent than in the frequent T1 condition) could be established. A repeated measures ANOVA with frequency (frequent or infrequent T1) and lag (1, 4, or 10) as within-subjects factors conducted on percentage correct T1 identification (see Table 1) for these participants revealed a significant effect of lag, $F(2, 20) = 15.34$, $MSE = 50.83$, $p < .001$, but no significant effect of T1 frequency, $F(1, 10) = 2.40$, $MSE = 1679.45$, $p = .15$, and no significant T1 frequency \times lag interaction, $F(2, 20) = .09$, $MSE = 65.23$, $p = .92$. When only one target was presented, T1 accuracy was 78.8% when it was a frequently presented letter, and 92.1% when it was an infrequently presented letter. This difference was, however, not significant, $t(10) = 1.75$, $SE = 7.59$, $p = .11$. The lack of a significant frequency effect for T1—despite the differences in mean T1 performance in the frequent and infrequent conditions—was probably due to the variability in performance for the letter ‘R’. For unknown reasons, participants found it more difficult to identify an ‘R’ when it was a frequently presented letter (49.2%) than when it was an infrequently presented letter (78.9%), whereas performance for the letter ‘N’ was comparable across conditions (90.1% and 96.8% when presented as a frequent or infrequent letter, respectively).

Performance for T2 on trials for which T1 was reported correctly is shown in Table 1. A repeated measures ANOVA with T1 frequency and lag as within-subjects factors showed significant effects of T1 frequency, $F(1, 10) = 17.05$, $MSE = 56.86$, $p = .002$, lag, $F(2, 20) = 14.15$, $MSE = 215.68$, $p < .001$, and a significant T1 frequency \times lag interaction, $F(2, 20) = 3.82$, $MSE = 42.36$, $p = .04$. The interaction reflects that the AB was more pronounced when T1 was infrequent compared to when a frequent T1 was presented.

2.3. Electrophysiological results

Fig. 1 shows the ERPs time locked to the presentation of T1 and includes data from single- and dual-target trials, the latter averaged across all lags. The mean amplitude of the P3 in response to T1 was 36.8% greater when an infrequent T1 was presented than when a frequent T1 was presented, $t(10) = 3.11$, $SE = .77$, $p = .006$ (one-tailed). As an alternative method of calculating the mean amplitude of the P3 evoked by T1, the voltage of each data

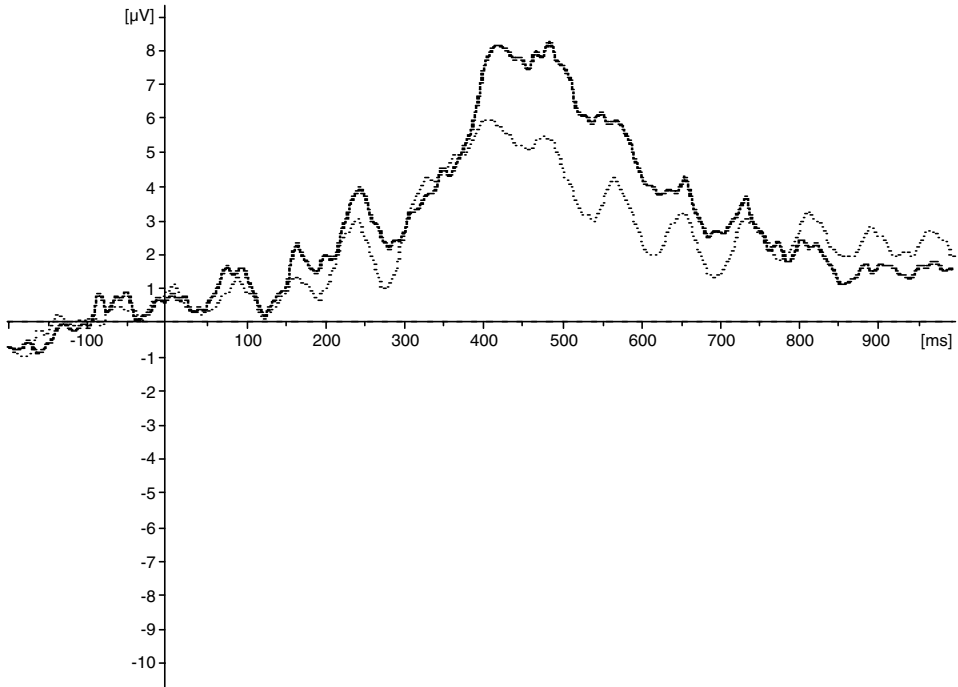


Fig. 1. Grand averages of the mean activation at Pz as a function of time in the infrequent T1 condition (solid line) and in the frequent T1 condition (dotted line) of Experiment 1. ERPs were time locked to T1 presentation and include single- as well as dual-target data across lags.

point was taken within a window from 370 to 540 ms post-target, based on visual inspection of the grand average, and averaged together for each condition. With this method, the P3 was found to be 44.6% greater in response to an infrequent T1, $t(10) = 4.08$, $SE = .54$, $p = .001$ (one-tailed). The mean amplitude of the ERP signal evoked by T2 was calculated by taking the voltage of each data point within a window of 720–996 ms post-T1 onset for the frequent and infrequent T1 conditions at lag 4. As expected, a significant effect of T1 manipulation on the T2-related P3 was found, $t(10) = 2.06$, $SE = .79$, $p = .03$ (one-tailed), with the ERP amplitude being 33.3% smaller in the infrequent T1 condition.

We also compared the ERPs of ‘blink trials’ (i.e., trials in the lag 4 condition in which T1 was correctly identified and T2 was not correctly identified) with ERPs from ‘no blink trials’ (i.e., trials in the lag 4 condition in which both T1 and T2 were correctly identified). Fig. 2 shows the ERPs related to the presentation and identification of both T1 and T2 in blink (solid line) versus no blink (dotted line) trials. In blink trials, the average peak amplitude of the P3 component in response to T1 was 14.0% greater than the average peak amplitude in no blink trials. This difference was not significant, $t(10) = 1.48$, $SE = .64$, $p = .085$ (one-tailed). However, when the mean amplitude of the P3 evoked by T1 was calculated by averaging the voltage of each data point within a window from 400 to 520 ms post-target (based on visual inspection of the grand average) for each condition, the resulting difference of 23.2% was found to be significant, $t(10) = 1.83$, $SE = .66$, $p = .048$ (one-tailed).

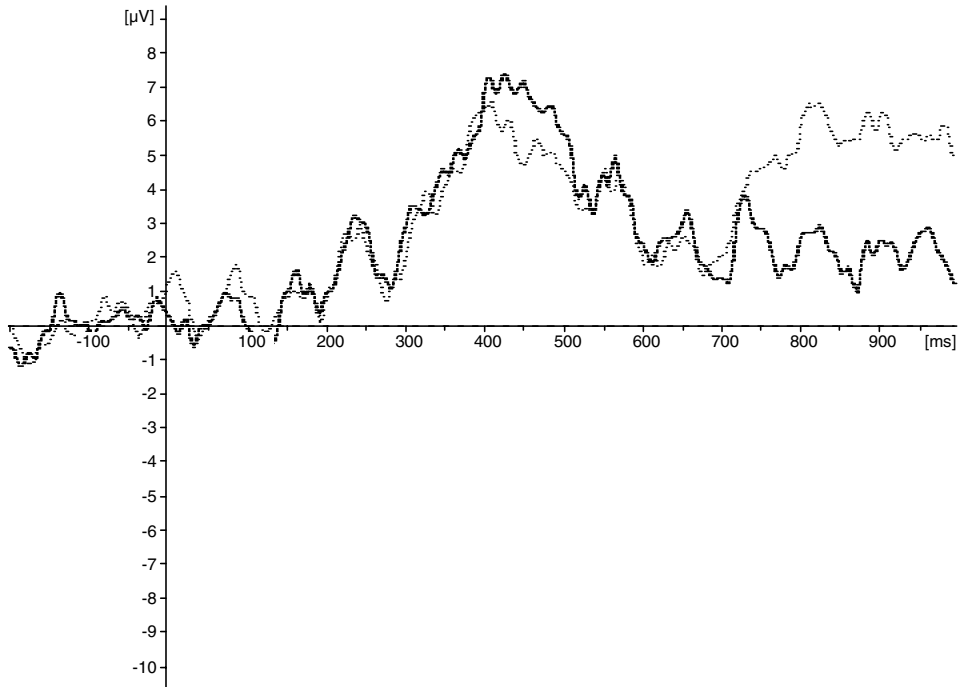


Fig. 2. Grand averages of the mean activation at Pz as a function of time for trials during which an AB occurred ('blink trials'; solid line) versus trials during which an AB did not occur ('no blink trials', dotted line) in Experiment 1. ERPs were time locked to T1 presentation and are limited to dual-target trials at lag 4.

Finally, we addressed the question of whether individuals who show a greater P3 response also show a greater blink. The difference between the mean P3 peak amplitude elicited across lags by T1 in the frequent and infrequent T1 conditions and the difference in the AB magnitude³ in frequent versus infrequent conditions were calculated for each individual. A Pearson product moment correlation computed on these differences was not significant, $r = .22$, $p = .259$. The correlation between peak P3 amplitude in response to T1 and the magnitude of the AB, both averaged across conditions, also was non-significant, $r = .10$, $p = .388$. The results were similar when the mean ERP amplitude within a window from 370 to 540 ms post-target was taken instead of the peak amplitude of the P3 elicited by T1.

The T2-related P3 was also calculated by taking the mean ERP amplitude within a window of 730–996 ms post-T1 onset. As can be seen in Fig. 2, the T2-related P3 amplitude was reduced by 59.5% when an AB occurred relative to when an AB did not occur, $t(10) = 3.09$, $SE = 1.05$, $p = .006$ (one-tailed).

2.4. Discussion

An AB occurred as indicated by worse T2 performance at lag 4 than at the other lags. In addition, a significant interaction was found between T1 frequency and lag, such that the

³ AB magnitude was defined as the $\left(\frac{((\text{the mean of T2|T1 accuracy at lag 1 and 10}) - (\text{T2|T1 accuracy at lag 4}))}{(\text{the mean of T2|T1 accuracy at lag 1 and 10})} \right) * 100$.

AB was greater when an infrequent T1 was presented. In other words, T1 frequency affects T2 accuracy specifically during the interval for which the availability of attentional resources is presumed to be low and an AB is most likely to occur (i.e., approximately 300 ms after the onset of T1).

Little T2-related activation seemed to be present at Pz when T2 was not successfully identified, compared to trials when T2 was successfully identified, which is in line with the findings reported by Krancioch et al. (2003), Rolke et al. (2001) and Vogel et al. (1998). More importantly, an increased T1-related P3 amplitude and a reduced T2-related P3 amplitude were found on blink trials compared to no blink trials, supporting previous MEG findings reported by Shapiro et al. (2006) and extending the findings reported by McArthur et al. (1999). We have no neurophysiological explanation why we were unable to find a significant correlation between P3 amplitude elicited by T1 and the magnitude of the AB at an individual level. The lack of such a correlation is, however, in line with McArthur et al.'s failure to find a significant correlation at an individual level. The fact that Shapiro et al. (2006) did find a significant positive correlation between the neural response to T1 and the magnitude of the AB might be due to the use of a different technique (MEG instead of EEG) in combination with source modeling, which might have provided a better signal-to-noise ratio than in the EEG studies in which no source modeling was applied.

3. Experiment 2

Experiment 1 showed that T1 frequency affects both the magnitude of the P3 elicited by T1 in an AB task and the magnitude of the AB. In Experiment 2, a cue with 75% validity was presented at the start of each trial to indicate the likely identity of T1 in the subsequent RSVP stream. It was expected that a greater P3 would be elicited by T1 when it was invalidly rather than validly cued, resulting in a stronger AB on invalid cue than on valid cue trials.

3.1. Method

3.1.1. Participants

Seventeen undergraduate students from the population described in Experiment 1, aged 18–30 (mean age 21.1, 12 females), participated in the experiment. One participant who had suffered from epilepsy as a child was included after determining that the EEG data showed no abnormalities.

3.1.2. Stimuli and apparatus

The same software and hardware was used as in Experiment 1. The only exceptions were that targets could be any consonant letter except 'Q' and 'V', and that stimuli were presented in 10-point Courier font, subtending 0.2° by 0.3° of visual angle.

3.1.3. Procedure

The experiment consisted of a practice block of 16 trials, and four experimental blocks of 192 trials each. Thus, 48 invalid cue trials per lag and 144 valid cue trials per lag were presented. At the start of each experimental block, four additional "warm-up" trials were given that were not included in the analyses. Participants initiated each trial by pressing the spacebar. After a 100-ms delay, a fixation cross was displayed, accompanied by a letter cue

for 1500 ms. The letter cue matched the identity of T1 in 75% of the trials (valid cue condition) and was different from T1 in 25% of the trials (invalid cue condition). The fixation cross was centered on the screen and the letter cue was located 0.6° of visual angle above the cross. The fixation cross remained on the screen for an additional 1000 ms following the offset of the cue. After a 200-ms delay, the RSVP stream consisting of 16 stimuli was presented. Each item in the RSVP stream was presented for 93 ms at the center of the screen; this presentation time was chosen on the basis of performance in a pilot study. On 75% of the trials, two of the 16 characters were letters (T1 and T2), and the other characters were digits. On 25% of the trials only one target letter was present in the stream to provide a baseline of single-target performance. Digit distractors were randomly selected with the constraint that no single digit was presented twice in succession. T1 randomly appeared as the fourth, fifth, or the sixth item in the stream. On dual-target trials, T2 was the first, third, or eighth item following T1 (lag 1, 3, or 8, respectively) and was always followed by at least two distractors. Both validly and invalidly cued trials were distributed equally across T2 lags. The experiment was conducted in one session, lasting approximately 90 min (excluding the long break). Responses were made and scored as in Experiment 1.

3.1.4. EEG recording

The data were recorded and analysed as in Experiment 1, except that ERPs were time locked to T1 with a length of 1200 instead of 1000 ms.

3.1.5. Data analysis

Analyses were restricted to 15 participants for whom a clear P3 component could be established. All participants showed the behavioral effect of cue validity such that T2|T1 performance at lag 3 was always numerically smaller in the invalid cue than in the valid cue condition. Trials containing movement artifacts, ocular artifacts, or amplifier saturation were removed from the analysis (a total of 10.6% of the trials, ranging from .6% to 39.9% [SD = 11.8] of the trials per participant). Across conditions, ERP analyses were based on 37 (lag 3 trials in which T1 was a correctly identified target following an invalid T1 cue) to 347 trials (trials in which T1 was a correctly identified target following a valid T1 cue) on average per participant.

3.2. Behavioral results

Percent correct for each target was computed for each lag (see Table 2). A repeated measures analysis of variance (ANOVA) with cue validity (valid or invalid) and lag (1, 3, or 8)

Table 2

Mean percentage correct report of T1 and T2 given the correct report of T1 in Experiment 2, as a function of lag and T1 cue validity (standard deviation in parentheses)

Measure	Condition	Lag		
		1	3	8
T1	Valid cue	84.8 (10.36)	92.0 (5.06)	93.2 (4.86)
	Invalid cue	74.2 (13.03)	86.9 (10.61)	90.0 (6.50)
T2 T1	Valid cue	88.8 (11.48)	75.7 (13.97)	88.3 (7.53)
	Invalid cue	82.8 (13.53)	59.3 (16.63)	82.1 (13.69)

as within-subjects factors was conducted on the percentage of correct T1 identifications. Significant effects of cue validity, $F(1,14)=12.76$, $MSE=70.23$, $p=.003$, and lag $F(2,28)=24.34$, $MSE=51.91$, $p<.001$, were found, as well as a significant cue validity \times lag interaction, $F(2,28)=5.41$, $MSE=20.31$, $p=.01$. In the single-target condition, T1 accuracy was 94.2% correct when a valid T1 cue was given, and 92.1% when an invalid T1 cue was given. This difference was not significant, $t(14)=1.47$, $SE=1.45$, $p=.16$.

Performance for T2, given that T1 was reported correctly, is shown in Table 2. A repeated measures ANOVA with cue validity and lag as within-subjects factors showed significant effects of T1 cue validity, $F(1,14)=27.20$, $MSE=74.72$, $p<.001$, lag, $F(2,28)=21.73$, $MSE=150.29$, $p<.001$, and a significant cue validity \times lag interaction, $F(2,28)=7.27$, $MSE=36.70$, $p=.003$.

3.3. Electrophysiological results

Fig. 3 shows the ERPs time locked to the presentation of T1 and includes data from single- and dual-target trials, the latter averaged across all lags. The mean amplitude of the P3 in response to T1 was 20.9% greater when the T1 cue was invalid than when it was valid, $t(14)=5.06$, $SE=.32$, $p<.001$ (one-tailed). Using the alternative method to calculate the amplitude of the P3 evoked by T1 (described in Experiment 1), the voltage of each data point was taken within a window from 408 to 560 ms post-target (based on visual

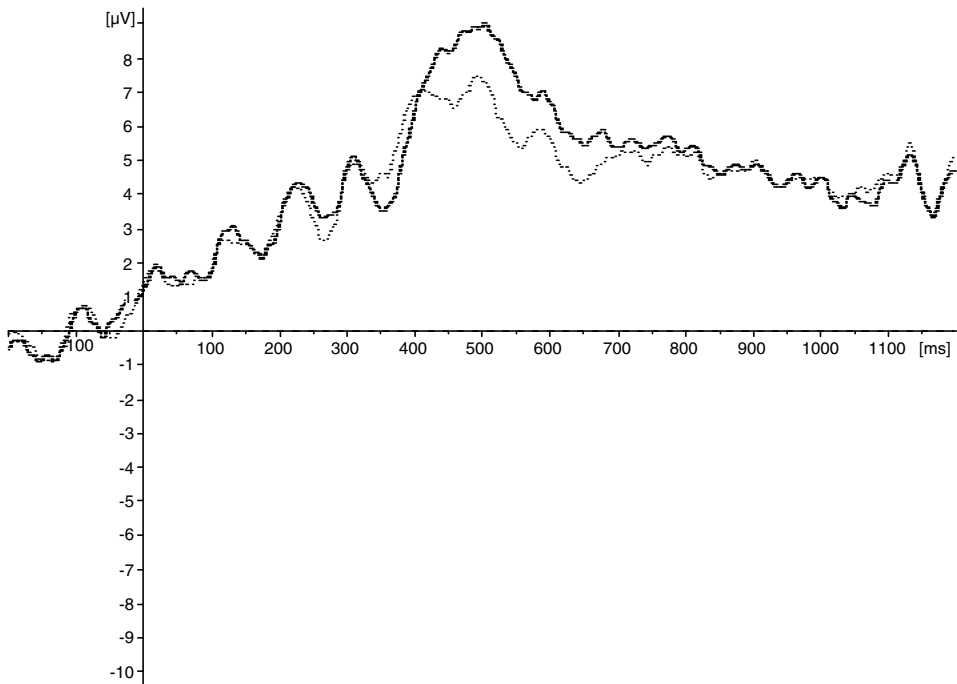


Fig. 3. Grand averages of the mean activation at Pz as a function of time in the invalid T1 cue condition (solid line) and in the valid T1 cue condition (dotted line) of Experiment 2. ERPs were time locked to T1 presentation and include single- as well as dual-target data across lags.

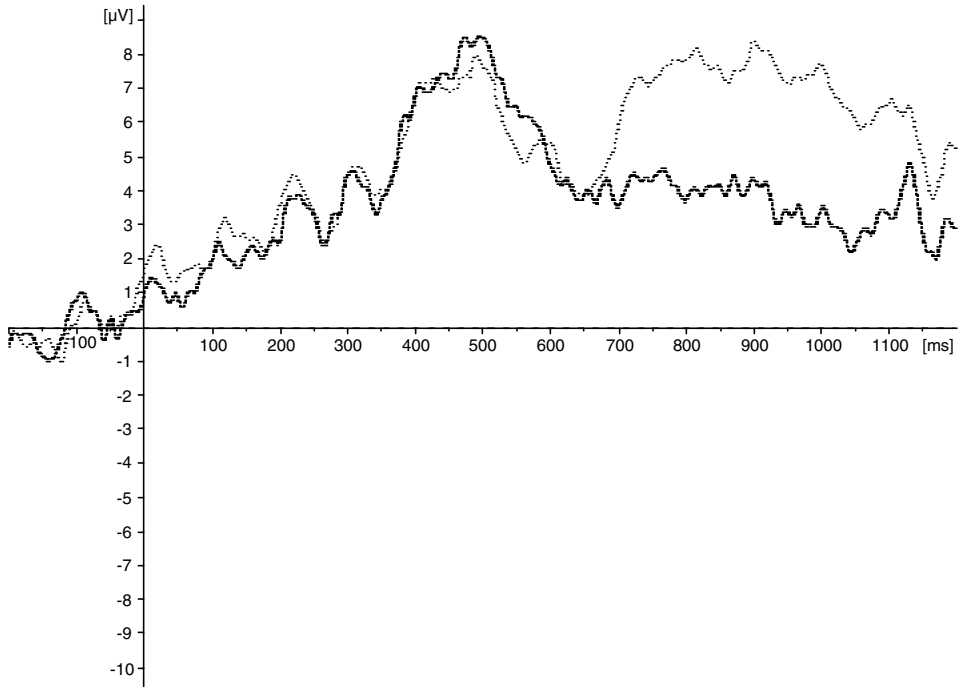


Fig. 4. Grand averages of the mean activation at Pz as a function of time for trials during which an AB occurred ('blink trials'; solid line) versus trials during which an AB did not occur ('no blink trials', dotted line) in Experiment 2. ERPs were time locked to T1 presentation and are limited to dual-target trials at lag 3.

inspection of the grand average), and averaged together for each condition. With this method, the P3 was found to be 23.3% greater when an invalid cue was presented, $t(14)=4.10$, $SE=.38$, $p<.001$ (one-tailed). The mean amplitude of the ERP signal evoked by T2 was calculated by taking the voltage of each data point within a window of 688–1024ms post-T1 onset for the lag 3 valid and invalid T1 cue conditions. Although the T2-related mean P3 amplitude was numerically smaller (10.3%) in the invalid T1 cue condition, the difference was not significant, $t(14)=1.17$, $SE=.59$, $p=.13$ (one-tailed).

Fig. 4 shows the ERPs associated with blink (solid line) versus no blink (dotted line) trials. The amplitude of the P3 component in response to T1 (when successfully identified) was 7.2% larger on blink trials than on no blink trials, $t(14)=1.76$, $SE=.36$, $p=.050$ (one-tailed). When the amplitude of the P3 evoked by T1 was calculated by taking the mean ERP amplitude within a window from 432 to 584ms post-target for each condition, the resulting difference of 12.1% was found to be marginally significant, $t(14)=1.63$, $SE=.48$, $p=.062$ (one-tailed). The same correlational analyses were carried out as in Experiment 1, but again none of them produced a significant result, $r=-.34$ to $.025$, $p>.10$.

T2-related activity, shown in Fig. 4, was estimated by calculating the mean P3 amplitude within a window of 652–1156ms post-T1 onset. The T2-related mean P3 amplitude was reduced by 46.4% when an AB occurred relative to when an AB did not occur, $t(14)=3.54$, $SE=.90$, $p=.002$ (one-tailed).

3.4. Discussion

As in Experiment 1, an AB occurred as indicated by worse performance for T2 at lag 3 than at the other lags. A significant interaction between T1 condition and lag was also found: The AB was larger when T1 was unexpected (following an invalid cue) rather than expected (following a valid cue). This suggests that, as with T1 frequency in Experiment 1, cue validity specifically affected T2 accuracy during the interval in which the availability of attentional resources is low and an AB is likely to occur (i.e., approximately 300 ms after the onset of T1). Interestingly, infrequent T1s were identified more accurately than frequent T1s, whereas unexpected (invalidly cued) T1s were identified less accurately than expected (validly cued) T1s, thus dissociating T1 identification performance from the AB effect.

The electrophysiological results were similar to those found in Experiment 1. Little T2-related activation seemed to be present at Pz when T2 was not successfully identified, compared to trials when T2 was successfully identified. In addition, an increased T1-related P3 amplitude and a reduced T2-related P3 amplitude was again found on blink trials compared to no blink trials. As in Experiment 1 and in the study reported by [McArthur et al. \(1999\)](#), a significant correlation between P3 amplitude elicited by T1 and the magnitude of the AB was not found at an individual level.

4. General discussion

Two experiments in which the magnitude of the P3 evoked by a first target was manipulated showed a relation between the amplitude of the P3 elicited by T1 and the probability of identification of a second target presented in RSVP. In Experiment 1, P3 amplitude was manipulated by varying the frequency of T1. When T1 was an infrequently presented letter, participants were more likely to miss T2 (i.e., an AB occurred more often) than when T1 was a frequently presented letter. In Experiment 2, T1 was cued and cue validity was manipulated. Both P3 amplitude and AB magnitude were greater when T1 was preceded by an invalid cue than by a valid cue. Moreover, in both Experiments 1 and 2, a comparison between blink trials (T2 not identified) and no blink trials (T2 identified) revealed that the amplitude of the P3 response to T1 was significantly larger on trials in which T2 could not be reported.

The results of the present study replicate and extend [McArthur et al.'s \(1999\)](#) finding of a relationship between the P3 and the AB. [McArthur et al.](#) manipulated the amplitude of the P3 by increasing the response set size of T1 and concluded that the increase in T1 “difficulty” affected the magnitude of both the P3 and the AB. However, the probability of T1, not the difficulty of identifying it, seems to be the critical factor. In our Experiment 1, T1 accuracy was higher for infrequent than for frequent T1s, whereas in Experiment 2, T1 accuracy was lower for invalidly cued than for validly cued T1s.⁴ In both the infrequent T1 and the invalid T1 cue conditions, an increase in AB (and P3) magnitude was observed, showing that it is not T1 difficulty that determines the likelihood of an AB, as was suggested by [McArthur et al.](#) Rather, it appears to be the amount of uncertainty about the

⁴ Two pilot experiments (without EEG measurements), one with frequent and infrequent T1s and 32 participants, and the other with valid and invalid T1 cues and 18 participants, showed a similar pattern of behavioral results.

identity of T1 that is experienced by the participant that influences not only the amplitude of the P3, but also the magnitude of the AB.

An important aspect of the present results is the general pattern of an interaction between the T1 manipulations and lag on T2 identification performance. That is, manipulating the subjective probability of T1 specifically affected T2 accuracy during the interval in which an AB is most likely to occur (200–500 ms after the onset of T1) with little or no effect at shorter or longer T1–T2 intervals. The present results support the idea that the amplitude of the P3 associated with T1 reflects the amount of attention or resources that are allocated to process and consolidate T1. When the amplitude of the P3 is greater, fewer attentional resources are available for a period between 200 and 500 ms for the processing and consolidation of T2, resulting in an AB (see, e.g., Shapiro et al. (2006)). Possibly, the early evoked T2 gamma response is suppressed by the T1-related P3 component which has been found to play a role in the allocation of attention to a selected object and, thus, in successful target identification (Fell et al., 2002; Hermann & Mecklinger, 2000).

To conclude, we found converging evidence that there is a link between the amplitude of the P3 generated by an identified target, and the temporary failure to report a subsequent target presented approximately 300 ms after the onset of the first target. When the P3 amplitude in response to T1 is larger, an AB is more likely to occur. Although the relationship between the P3 and the AB may prove to be an indirect one, the finding that an enhanced neural response to one target can have a marked carry-over effect on the consolidation of a subsequent target is intriguing and might help to elucidate why and how the AB occurs.

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