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P3 latency shifts in the attentional blink: Further evidence for second target processing postponement

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ABSTRACT

A rapid serial visual presentation technique was used to display sequentially two targets, T1 and T2, and monitor P3 amplitude and latency variations associated with the attentional blink (AB) effect. A red T1 digit was embedded on each trial in a sequence of black letters. T2 was either masked by a trailing stimulus or not masked. T1 had to be identified on a proportion of trials, or ignored in other trials. T2 was the black letter 'E' on 20% of the trials, or any other non-'E' black letter in the other 80% of the trials. A delayed 'E' detection task was required at the end of each trial. An AB was observed when T1 had to be reported and T2 was masked. The AB effect was associated with a sizable amplitude reduction of the P3 component time locked to T2 onset. When T2 was not masked, no AB or P3 amplitude variations were observed. When T1 had to be reported, a delayed P3 peak latency was observed at short compared to long T1–T2 intervals. No effect of T1–T2 interval was observed on the T2-locked P3 peak latency when T1 could be ignored. Taken together these findings provide converging evidence in support of temporal attention models bridging behavior and electrophysiology that postulate a direct link between the cause of the AB effect and the sources of both amplitude and latency variations in the T2-locked P3 component.

1. Introduction

When two target items, T1 and T2, are embedded in a rapid serial visual presentation (RSVP) stream of stimuli and displayed within less than 500 ms of each other, T2 is often missed. This phenomenon, known as attentional blink (AB; Raymond et al., 1992), has been the object of much investigation over the past two decades, and several models have been proposed to account for this striking limitation of the human visual information processing system. Although different under several aspects, extant AB models share a subset of assumptions concerning the potential causes of the AB effect. One such assumption is that the AB effect has certainly something to do with the cognitive resources taken up by processing T1. Indeed, when participants are instructed to ignore T1, the AB effect is substantially reduced.

The second assumption is that the AB effect is a 'late,' post-perceptual effect. Demonstrations in this direction are abundant, but one is particularly cogent. Vogel et al. (1998) used the event-related potential (ERP) technique to track the moment-by-moment reflections of the processing of T2 during the AB interval. In a series of RSVP designs, several factors tapping distinct stages of T2 processing were examined as potential causes of the AB. The results were consistent in showing the independence of T2-locked ERP components up to and including the N400 (e.g., P1 and N1 components) from the

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systematic manipulation of the interval elapsing between T1 and T2. This set of findings suggested strongly that, although missed during the AB, T2 could undergo significantly deep processing, up to the generation of a semantic code for T2 (see also Rolke et al., 2001; Visser et al., 2005). In another experiment included in the same series, Vogel et al. (1998) have however found that, during the AB (i.e., when T2 is likely to be missed), the amplitude of the T2-locked P3 component was markedly reduced. Furthermore, the P3 component regained normal amplitude when T2 was displayed outside the AB interval (i.e., when the interval between T1 and T2 was longer than 500 ms), or when T1 could be ignored (see also Dell'Acqua et al., 2003a,b; Kranczioch et al., 2003). Several hypotheses have been put forth regarding the nature of the functional processes reflected in the P3 component. One line of thinking regards P3 as related to the decision on how to classify and respond to an eliciting stimulus (e.g., Squires et al., 1973, 1977; Makeig et al., 2004; Verleger et al., 2005). Another line of thinking holds that P3 reflects access of the eliciting stimulus to global mental workspace, with this access making the event reportable (Dehaene et al., 2003; Koivisto and Revonsuo, 2003; Sergent et al., 2005). A third, influential line of thinking sees P3 strongly linked 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.

All three notions are compatible with the general proposal that the combination of a multitude of psychological events/processes, likely related to the simultaneous activation of many neural generators (e.g., see Johnson, 1993), produces a single observed long-duration positive wave (i.e., the P3 component; see Luck, 2005, for a discussion on observed ERP and latent components). Furthermore, all these notions are in line with functional models positing a structural 'bottleneck' along the flow of processing leading to encode the information vehicled by T2 in visual working memory (Chun and Potter, 1995; Jolicœur and Dell'Acqua, 1998) but while the former two notions imply that P3 suppression reflects the consequences of the bottleneck, the memory-updating notion implies that P3 suppression reflects just this bottleneck. Specifically, when the stage hypothesized to transfer target information into visual working memory, termed consolidation, is occupied with processing T1, consolidation of T2 is postponed, and T2 remains shortly in a stand-by state during which it is vulnerable to corruption by items trailing T2 in the RSVP stream. When T1 has finally been consolidated and transferred into visual working memory, consolidation mechanisms become available to process information following T1, yet no representation of T2 is left to be transferred in visual working memory (Dell'Acqua et al., 2003a,b). To note, when T2 is not masked, and therefore not subject to corruption by trailing items, an AB is typically not observed (Giesbrecht and Di Lollo, 1998).

Important to our study is that several lines of evidence have been provided from previous work, using the AB paradigm, that the P3 is elicited only by stimuli that have already reached working memory, and that the T2-locked P3 component is a good index for the completion of T2 consolidation process (e.g., Vogel et al., 1998; McArthur et al., 1999; Vogel and Luck, 2002; Dell'Acqua et al., 2003a,b; Martens et al., 2006).

On top of this evidence is the result by Vogel et al. (1998), reported above, which suggests that the P3 is as a clear marker that the AB occurs before or at least at the stage of working memory (and T2 is missed as a consequence of the failure of its consolidation process). Another line of evidence derives from the demonstration that, at a group level, the AB and the T2-locked P3 follow a very similar time course with a common onset about 200 ms after the presentation of the target, the reaching of the plateau at 300–400 ms and the return to baseline after 500–600 ms (McArthur et al., 1999). The strong relationship between P3 and AB ($r=-0.95$) suggests an association between these two phenomena and supports the hypothesis that the P3 is an optimal marker of T2 consolidation. Further evidence in favour of the P3 component as an index of consolidation of T2 have been recently provided by Martens et al. (2006). They compared event-related potentials during a typical AB task between participants who reported the AB deficit in identifying T2 ("blinkers") and those who did not ("nonblinkers"). The important result was that the T2-locked P3 peak was earlier for nonblinkers than for blinkers, and this corroborates the existence of a link between AB and P3 component even at the individual level.

The notion of consolidation postponement affecting T2 during the AB has recently received solid support from an electrophysiological study conducted by Vogel and Luck (2002). The authors monitored the T2-locked P3 component in a standard RSVP paradigm, under conditions in which T2 was either masked as in classical RSVP designs, or not masked, T2 being the last item in the RSVP stream. In their study, Vogel and Luck (2002) presented participants with RSVP streams composed of a variable number of black letters. T1 was a to-be-reported black digit, and T2 was the white letter 'E' that had to be detected among the letters trailing T1. A clear AB was observed when T2 was masked. The AB was accompanied by the suppression of the P3 component amplitude at short compared to long intervals between T1 and T2. In line with Giesbrecht and Di Lollo's (1998) findings, no AB was observed when T2 was not masked. However, the T2-locked P3 peak latency was substantially delayed at short compared to long T1–T2 intervals in this condition, and this was taken to dovetail nicely with the principle that consolidating T1 likely postponed the consolidation of T2 which, nonetheless, survived the AB, given the absence of items trailing T2 in the RSVP stream.

One crucial observation related to Vogel and Luck's (2002) study is that T2-locked P3 peak latency shifts were not estimated under conditions in which T1 could be ignored, that is, under the control condition normally implemented in standard AB designs to tell apart effects tied to T1 processing demands from effects of different origins. T1 had in fact to be identified on each trial of that study since it had to be reported at the end of each RSVP stream presentation. The lack of an appropriate control condition might motivate a number of potential objections concerning the original

interpretation of the findings, and open a space to the possibility that the P3 latency shifts observed by Vogel and Luck (2002) could be due to factors other than those subtended in the cause of the AB effect. One objection, for instance, might be that participants could not refrain from detecting the discontinuity in alphanumeric class generated by the onset of T1 (digit) among RSVP distractors (letters). Maybe this automatic and mandatory stimulus evaluation process which we argue to be independent from consolidation of T1 took time (e.g., Magliero et al., 1984), and this had an impact on the latency of a P3 triggered by stimuli presented shortly after this alphanumeric 'perturbation.' A second objection is that, on average, the absolute temporal distance between the beginning of a trial and the point in time corresponding to T2 onset was confounded with the interval elapsing between T1 and T2 in Vogel and Luck's (2002) study. In this perspective, one could argue that the T2-locked P3 peak latency was delayed at short compared to long T1–T2 intervals because, at short T1–T2 intervals, T2 was presented closer to the beginning of a trial relative to a T2 presented at long T1–T2 intervals. One should therefore consider the possibility that P3 latency shifts in this latter case could ensue from the fact that participants were less prepared to detect T2 when T2 was displayed shortly after the beginning of the trial compared to when a longer interval elapsed between the beginning of the trial and T2 onset, comparable to targets presented at a short interval when a long interval was expected in a study by Miniussi et al. (1999). A single-task condition can serve as a control condition to test whether involuntary evaluation of T1 and/or preparation processes are responsible for the P3 delay because such mandatory processes should be observed in the single-task condition as well.

The primary purpose of the present investigation was to establish a direct link between the cause of the AB effect and the cause of T2-locked P3 latency variations in a standard RSVP design. This demonstration would be of crucial importance for the argument that P3 latency variations in the AB paradigm are – unequivocally – reflections of one functional process (i.e., consolidation) hypothesized to be postponed due to attentional limitations, and not attributable to 'spurious' limitations, as the objections raised in the foregoing paragraph would lead one to suspect.

A secondary aim of the present investigation was to provide evidence converging with the original proposal made by Vogel et al. (1998) and Vogel and Luck (2002) that the AB effect is a consequence of a failure to encode T2 into visual working memory, as witnessed by the attenuation of T2-locked P3 amplitude found in their investigations. To this end, we used a variant of the seminal RSVP design that was devised specifically to explore P3 amplitude and latency variations in a context in which T2 masking (i.e., T2 masked vs. T2 not masked) and the load imposed by processing T1 (ignore T1 vs. encode T1) were orthogonally manipulated.

2. Results

Two participants were eliminated from the following analyses, one for problems related to the scoring of the behavioral performance and the other for an excessive number of trials contaminated by EEG artifacts.

2.1. *Behavior*

The mean proportion of correct responses to T1 in the dual-task condition was 0.96. An analysis of variance (ANOVA) with masking (T2 masked vs. T2 unmasked) and lag (3 vs. 7) as within-subject factors yielded no significant main effects or interactions (lowest $p > 0.16$).

A schematic representation of the identification rates of T2 is reported in Fig. 1. In the dual-task condition, the mean proportion of correct responses to T2 was calculated based on trials in which T1 was correctly identified. An ANOVA on the mean proportion of correct responses to T2 considering condition (single task vs. dual task), lag and T2 masking as within-subject factors yielded significant main effects of all factors, $F(1,15) = 22.86$, $p < 0.001$; $F(1,15) = 32.36$, $p < 0.0001$; and $F(1,15) = 12.97$, $p < 0.005$, respectively. All two-way interactions between condition and masking, $F(1, 15) = 26.86$, $p < 0.001$; condition and lag, $F(1,15) = 25.95$, $p < 0.001$; and T2 masking and lag, $F(1,15) = 35.77$, $p < 0.001$, were significant. The three-way interaction between condition, T2 masking and lag was also significant, $F(1,15) = 23.98$, $p < 0.001$. As is evident in Fig. 1, the likely source of this interaction was the AB effect that was entirely confined to the dual-task condition when T2 was masked, with a lower proportion of correct responses to T2 at lag 3 relative to that at lag 7, $F(1,15) = 35.75$, $p < 0.0001$. Such lag-dependent modulation in accuracy was absent in all remaining cases examined in the present experimental design (all Fs < 1).

2.2. *Electrophysiology*

A schematic representation of the electrophysiological results is reported in Fig. 2. Recording site (Cz vs. Pz) was included as an additional within-subject factor in the ANOVAs performed

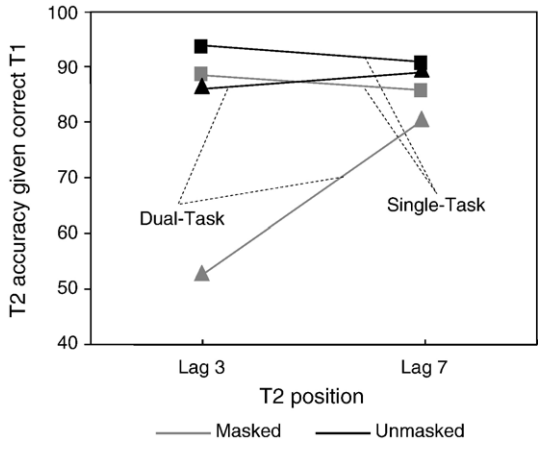

Fig. 1 – Behavioral results. Proportion of correct responses to T2, plotted as a function of task condition (dual task vs. single task), and as a function of T2 masking condition. The results in the T2 masked condition are reproduced in gray color, and the results from the T2 unmasked condition are reproduced in black color.

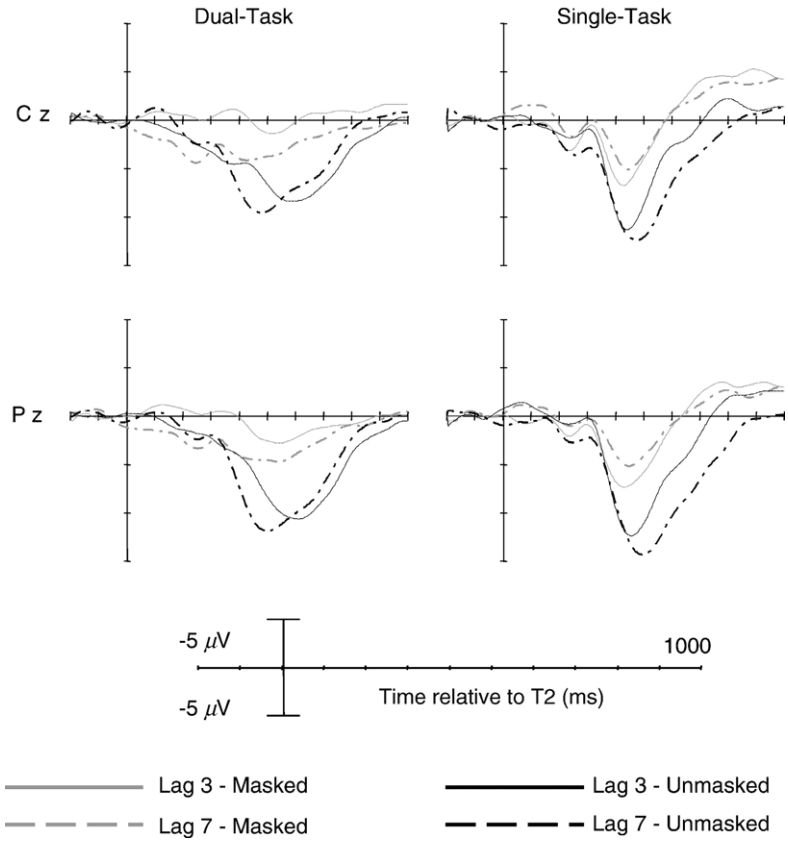

Fig. 2 – Electrophysiological results. Grand-average difference waveforms ('E'-minus-'non-E') recorded at electrode sites Cz and Pz, plotted separately for the dual-task condition (left panel) and for the single-task condition (right). In each panel, ERP functions are plotted as a function of lag, and as a function of T2 masking.

on P3 amplitude and latency values. The rationale for this factorial design was to provide a simple test for the P3 having the typical topography (Pz>Cz). P3 amplitude was larger in the single-task condition than in the dual-task condition, $F(1,15)=4.92$, $p<0.05$. P3 amplitude was larger when T2 was unmasked compared to when T2 was masked, $F(1,15)=21.45$, $p<0.001$. Furthermore, P3 amplitude was generally larger at Pz than at Cz, $F(1,15)=13.79$, $p<0.005$.

Of most interest for the issue of P3 suppression by the AB, a three-way interaction was obtained between condition, lag and T2 masking, $F(1,15)=7.02$, $p<0.03$, reflecting the fact that the effect of lag on P3 amplitudes differed between dual- and single-task conditions specifically in masked trials (condition×lag for masked trials, grey lines in Fig. 2: $F(1,15)=5.19$; $p<0.04$; for unmasked trials, black lines in Fig. 3: $F(1,15)=1.2$, n.s.) where P3 tended to be larger at lag 7 than at lag 3 in the dual task ($F(1,15)=3.2$, $p=0.09$), in contrast to the single task where a weak tendency pointed to the other direction, if anything ($F(1,15)=1.9$, $p=0.19$).

To investigate the main question under study, P3 peak latency values were analyzed on the basis of trials in which T2 was unmasked. P3 peak latency was generally delayed in the dual-task condition relative to the single-task condition, $F(1,15)=7.07$, $p<0.05$. The significant interaction between condition and lag, $F(1,15)=26.69$, $p<0.001$, lends statistical support to what is evident in Fig. 2, namely, P3 peak latency was substantially delayed (70 ms) at lag 3 relative to lag 7 in the dual-task condition, $F(1,15)=7.28$, $p<0.05$, whereas no such delay was observed in the single-task condition, $F=1$, with a mean difference of −14 ms. Indeed, as shown in Table 1, 75% of the participants (12/16) had later peaks with lag 3 than with lag 7 in the dual-task condition, compared to 37.5% in the single-task condition.

To make sure that same component is responsible for the delay between lag 3 and lag 7 (unmasked condition), we conducted a spatial PCA, as an additional analysis. Our goal was to rule out the possibility that this delay reflects a juxtaposition of different components/processes. The PCA analysis replaces the original electrodes by a smaller number of factors. These factors, "virtual electrodes," are a linear combination of the recording sites, and they try to preserve as much information given by these sites (see Spencer et al., 2001, for further details). We conducted only spatial PCA (and not temporal) because we were interested to know whether the P3 component would be identical (defined as having the same topography) across conditions, in spite of its latency shift which, if reliable, would produce a new temporal component by its very existence (Donchin and Heffley, 1978; Möcks, 1986). The data matrix input to the spatial PCA consisted of 19 recording sites by 3616 observations (16 subjects×2 lags×113 time points). Using the scree test, 3 spatial factors (SF) were extracted for Varimax rotation, accounting for 86.9% of the variance. After rotation, these factors accounted for 68.9%, 12.2% and

5.7% of variance. Factor loadings for each factor are displayed in Fig. 3 (top). The first factor constituted a composite of parieto-occipital recordings, the second factor of pre-frontal and frontal recordings and the third cluster specifically emphasized left-frontal recordings. Thus, the parieto-occipital composite was the most probable candidate for describing the P3 component. Because variance accounted for by the third factor was small, we did not analyze it any further. We then plotted the factors scores (the contribution of each factor to each observation) for the first and second factors, for each lag condition (averaged across participants).

As can be seen in Fig. 3 (bottom), within each SF, the factor scores were similar between the two conditions. Of particular importance, factor scores in the first spatial factor were delayed in lag 3 by 64 ms relative to lag 7 (as measured at their crossing of the *x* axis).

This difference is indeed similar to the P3 latency shift found for the Cz and Pz recording sites. Importantly, this spatial PCA gave no indication that the P3 delay reflects a juxtaposition of different components/processes. Rather, it seems that the same (and only one) component (or process) was responsible for this delay.

Table 1 – Individual participants' P3 latency values for lag 3 and lag 7 and differences in the single-task and dual-task conditions (when T2 was unmasked) averaged across recording sites (Cz and Pz)

Subject	Dual task			Single task		
	Lag 3	Lag 7	Difference	Lag 3	Lag 7	Difference
1	626	514	112	472	522	−50
2	620	514	106	450	474	−24
3	716	496	220	632	628	4
4	694	746	−52	432	530	−98
5	590	620	−30	448	500	−52
6	590	454	136	434	470	−36
7	500	466	34	442	420	22
8	420	430	−10	410	448	−38
9	710	584	126	550	528	22
10	534	498	36	448	450	−2
11	384	498	−114	494	598	−104
12	608	488	120	458	442	16
13	526	482	44	444	518	−74
14	656	398	258	726	502	224
15	534	424	110	418	416	2
16	620	594	26	446	476	−30

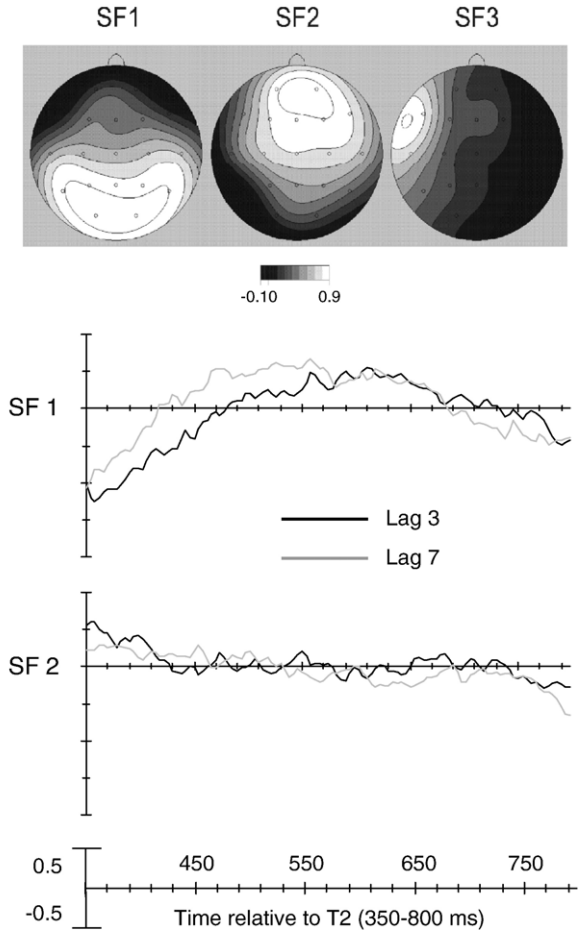

Fig. 3 – Top: Factor loadings of the three extracted spatial factors which accounted for 68.9%, 12.2% and 5.7% of the variance after rotation. Bottom: Grand average of spatial factor (SF) scores for lag 3 and lag 7 in the dual-task condition when T2 was unmasked for SF1 and SF2. The values of the factor scores (*y* axis) are unitless by definition.

3. Discussion

In a standard RSVP design, we manipulated T2 masking, lag and the cognitive load imposed by T1 processing in a fully factorial, within-subject design while also monitoring the T2-locked ERPs generated in the different conditions. By adopting the expedient of displaying a target stimulus, 'E,' with reduced frequency compared to stimuli belonging to a different category, non-'E' distractors, we managed to isolate frequency-related P3 activity not contaminated by activity associated with items preceding and following T2 within each RSVP streams.

Both the behavioral and the electrophysiological results were clear-cut. An AB effect was found only when T2 was followed by a masking stimulus, and only when participants had to identify T1 for later report. At the ERP level, the same experimental conditions brought about a substantial attenuation of P3 amplitude, thus replicating the original findings reported by Vogel et al. (1998). In particular, the suppression of P3 with masked T2 at lag 3 in our dual-task condition closely replicated the result reported by Vogel and Luck (2002).

It might be argued that the P3 result is trivial, mirroring the percentages of accuracy of T2 detection (see Fig. 1). After all, the waveshapes were averaged across trials in which T2 was reported and trials in which T2 was missed. Therefore, when assuming that P3 was evoked in hit-trials only, P3 is expected to be half as large in the average of the masked lag-3 AB condition, where detection rate was about 50%, compared to the unmasked conditions, where detection rate ranged between 90% and 100%. Yet P3 was more attenuated in that critical condition than to half the size. This suggests that even in trials where T2 was detected P3 was not as large as in unmasked trials. We have performed separate analyses of

missed versus correct T2 data for the lag 3 dual-task condition when T2 was masked and the results were clear-cut, demonstrating that P3 was elicited only when T2 was reported (see Fig. 4).

Of importance, even in these hit-trials of the lag 3 masked condition the P3 was not as large as in the unmasked condition. Of further relevance is the comparison between masked trials of the single-task condition and unmasked trials of the dual-task condition. Detection rate was nearly identical in both conditions (about 90%) but, as Fig. 2 shows, P3s were markedly smaller in masked trials (gray waveshapes in the right panel vs. black waveshapes in the left panel, $F(1,15)=7.9$, $p=0.01$).

This suppression of P3 in a condition with 90% detection rate is not compatible with accounts of P3 in terms of access to global workspace and in terms of access to working memory without making further assumptions because, after all, the stimulus was correctly reported and remembered. The obvious additional assumption to make, in the tradition of the early signal-detection studies (e.g., Squires et al., 1973) is to assume that P3 was reduced due to a lack of confidence in the participants' own decisions, perhaps for a sort of 'equivocation' of the signal (Ruchkin and Sutton, 1978; Johnson, 1986). This is actually the major assumption made by the notion that accounts for P3 suppression in the AB in terms of decision-related processes (cf. the Introduction). Thus, this latter notion appears to also provide a parsimonious account of the pattern of data obtained in the present experiment.

More important for the present purposes, however, were the ERP reflections of displaying an unmasked T2 while manipulating the load imposed by processing T1. Relative to the T2-locked P3 latency estimated at lag 7, the latency of the T2-locked P3 was delayed by 70 ms at lag 3 only when T1 had to be identified for later report. When T1 could be ignored, no such latency delay was observed.

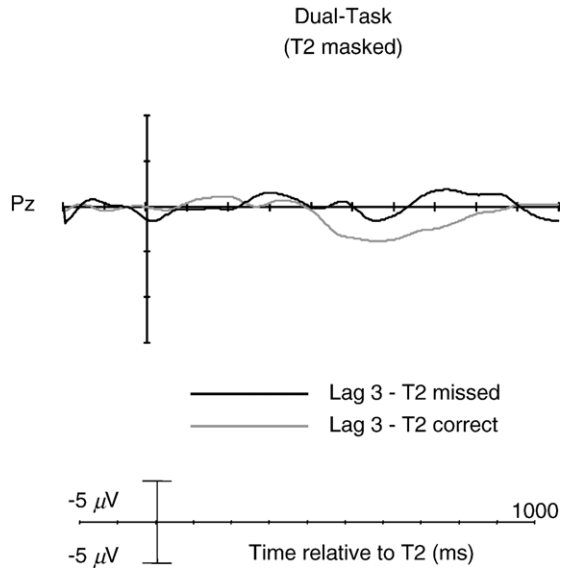

Fig. 4 – Grand-average difference waveforms ('E'-minus-'non-E') for the masked lag 3 dual-task condition recorded at electrode site Pz. Waveforms are plotted separately for the T2 hit-trials and the T2 missed trials.

The relevance of the present findings is two-fold. As far as P3 latency in RSVP designs is specifically concerned, the present demonstration helps constrain the possible causes of P3 latency shifts observed in previous work (i.e., Vogel and Luck, 2002). The fact that we did not observe any P3 latency shift in the single-task condition implies that uncontrolled variables as automatic detection of semantic (alphanumeric) deviations or differences between trial onset and T2 onset do not seem applicable in the present context as potential explanations of the P3 latency shifts originally described by Vogel and Luck (2002). At a more general level, it is of note that P3 amplitude and latency variations can both be couched as complementary emanations of a unique limitation underlying the AB effect, namely, T2 consolidation postponement. Of course, whether P3 activity in the present empirical context, like in previous contexts, should be best viewed as emerging from the consolidation process per se, or simply as a reflection of the consequences for selecting the response following the consolidation process, is clearly an issue that the present results leave open to future investigations. More in detail, both if P3 is conceived of in terms of access to global workspace and in terms of decision how to classify the stimulus, the delay of latency makes sense: The assumed processes of access to global workspace or of decision on how to classify the eliciting stimulus have to wait for some intervening process, which might well be the assumed process of consolidation. In any case, the great advantage of these ERP data, replicating Vogel and Luck's (2002) findings and confirming them by the additional single-task control, is that the existence of this intervening process can be seen in the waveshapes and therefore be inferred to possess psychological reality.

The present results concerning P3 latency bear a direct connection to an issue that has recently been debated in the AB field of studies, namely, that to get an estimate of the time taken to consolidate a single chunk of information into visual working memory. There are at least two divergent positions on this issue. One position holds that consolidating one item (in some cases, even 1 bit of information) may take several hundreds of milliseconds. Jolicœur and Dell'Acqua (1998), for instance, presented one or three masked characters as T1 that had to be identified for later report, followed at different stimulus onset asynchronies (SOAs) by a tone as T2 that required a speeded two-alternative forced choice response that had to be made manually through a button press. The reaction time to T2 (RT2) in this paradigm increased markedly as SOA was decreased, the more so as the number of characters in T1 was increased. Interestingly, the SOA between T1 and T2 before RT2 reached an asymptote was almost 700 ms (Experiment 4, p. 158), suggesting that, on a proportion of trials, the consolidation of the characters in T1, that was hypothesized to postpone response selection for T2, took that long to be finished. To note, the estimate extrapolated on the basis of Jolicœur and Dell'Acqua's (1998) results is not too distant from the temporal extension that, under normal circumstances, characterizes the AB effect. A different position was however suggested by recent findings by Vogel et al. (2006). In one of their experiments (Experiment 2), these authors used a change detection paradigm in which a memory array of colored objects was exposed for 100 ms, followed by a

masking pattern, with SOAs ranging from 117 to 317 ms. After a fixed delay of 1000 ms, a test array was displayed, and participants had to detect whether a change occurred in one of the colored items or not. By assuming that the onset of the masking pattern would terminate the consolidation of information in visual working memory, and regressing the SOA between the memory array and the mask on the K (Cowan, 2001) number of items held in memory, the authors arrived at an estimate of 50–60 ms for the time required to consolidate each single colored item in visual working memory. Although it can certainly be argued that there may be a number of differences between consolidating colored squares (i.e., the items used by Vogel et al., 2006) and alphanumeric characters, one thing that is immediately apparent is that this latter estimate is in the order of one tenth compared to that proposed by Jolicœur and Dell'Acqua (1998), and reflected in terms of effect duration by practically all AB studies published so far. There are a number of problems in estimating the consolidation duration by using a masking manipulation, as already pointed out by Jolicœur and Dell'Acqua (2000). Nonetheless, one observation that seems particularly pertinent in this context is that the overall P3 latency delay observed in the present empirical context, and reported also by Vogel and Luck (2002), is closer to the proposal of Vogel et al. (2006) than to that of Jolicœur and Dell'Acqua (1998). The delay in P3 latency produced by moving T2 position from lag 7 to lag 3 was in fact 70 ms. If one considers that lag 3 is the lag at which the AB effect usually peaks, 70 ms should in principle correspond to an estimate of the postponement to which T2 consolidation is subjected to when the AB is maximal. Even if one concedes that the alternative delay of 700 ms would be difficult to observe and highly unusual for a delay of P3 latency (Verleger, 1997) the present observed delay of 70 ms does lend some support to the idea that at least some first step of processing had to be postponed by an interval in the range of this 70-ms interval.

4. Experimental procedures

4.1. *Participants*

Eighteen students (12 females and 6 males) at the University of Padua, with a mean age of 22 years, participated in the experiment for course credit. All participants had normal or corrected-to-normal vision, and none had a history of prior neurological disorders.

4.2. *Stimuli and procedure*

The stimuli were light gray uppercase letters (all letters of the English alphabet, except B, I and O) and red digits (2–9) displayed on the black background of a 17-in. cathode ray tube monitor controlled by an IBM-clone and MEL 2 software. The luminance of the red and light gray colors (45 cd/m^2) was adjusted to be the same using a luminance meter. At a viewing distance of approximately 60 cm, each character subtended 1.0° of visual angle in height and width. Fig. 5 illustrates the sequence of events on each trial of the present experiment.

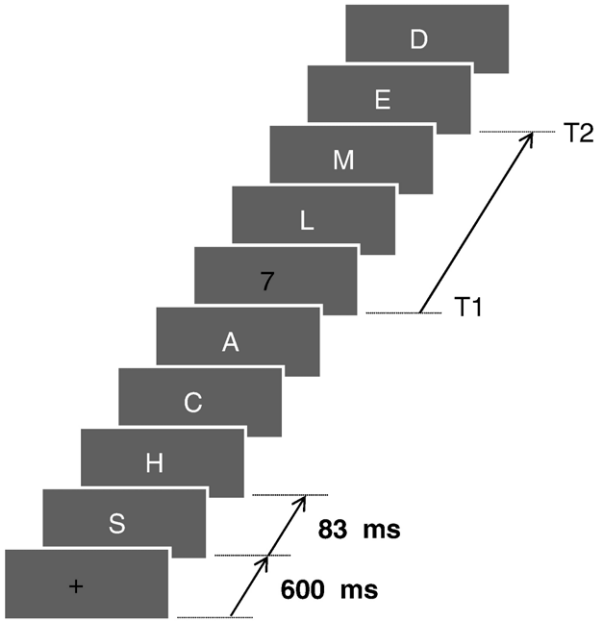

Fig. 5 – Schematic illustration of the RSVP paradigm used in the present experimental context. T1 was a red digit (black in the figure). T2 was either the third or seventh letter following T1 and was the letter 'E' on 20% of the trials, or any other randomly chosen letter on 80% of the trials. In the illustration, T2 is followed by a masking letter (T2 masked condition, see text for details). In a different condition implemented in the present experimental design, T2 was the last item of the stream (T2 unmasked condition).

The trial began with the presentation of a fixation point (i.e., '+') at the center of the screen. Participants were instructed to initiate the trial by pressing the spacebar of the computer keyboard. Upon the spacebar press, the fixation point disappeared and, after a fixed interval of 600 ms, the RSVP of characters began. Each character was displayed for 83 ms, with 0-ms inter-stimulus interval. There were 2 to 5 randomly selected letters prior to T1 (the red digit), and 2 to 8 letters following T1. T2 was the letter 'E' on 20% of the trials, or one randomly selected non-'E' letter on the remaining 80% of the trials. T2 was either the third or seventh letter following T1, corresponding to a T2 presentation occurring at lag 3 or lag 7, respectively.[1] In separate trials, T2 was either followed by one letter acting as a mask for T2, or T2 was the last item in the stream.

Each participant alternated between two different types of task. In the dual-task condition, participants were instructed to pay attention to both T1 and T2. At the end of each trial, participants were prompted by a question displayed in the lower part of the monitor to press one key of the numeric keypad of the keyboard to indicate the identity of T1, and to press the key 'E' of the keyboard if they saw an 'E' embedded in the sequence of letters following T1. If an 'E' was not seen, participants pressed the spacebar in order to move to the

[1] Lag 3 and lag 7 corresponded to T1–T2 onset asynchronies of 250 ms and 583 ms, respectively.

beginning of the next trial. In the single-task condition, participants were instructed to ignore T1, and only perform the 'E' detection task with T2. All responses had to be made with no speed pressure. The experiment was organized in 20 blocks of 40 trials each. Fourteen consecutive blocks were composed of dual-task trials, and 6 consecutive blocks were composed of single-task trials. Half of the participants began with the dual-task condition, and half with the single-task condition. In each block, the levels of lag (3 vs. 7) and T2 masking (T2 masked vs. T2 unmasked) were fully crossed and equiprobable.

4.3. *EEG/ERP*

EEG activity was recorded continuously with tin electrodes located at sites Fp1, Fp2, Fz, F3, F4, F7, F8, C3, C4, Cz, P3, P4, Pz, O1, O2, T7, T8, P7, P8 sites (see Pivik et al., 1993), referenced to the left earlobe. HEOG activity was recorded bipolarly from electrodes positioned on the outer canthi of both eyes. VEOG activity was recorded bipolarly from two electrodes, above and below the left eye. Impedance at each electrode site was maintained below 5 kΩ. EEG, HEOG and VEOG activities were amplified, filtered using bandpass of 0.01–80 Hz and digitized at a sampling rate of 250 Hz. The EEG was algebraically re-referenced offline to the average of the left and right earlobes and segmented into 1200-ms epochs starting from 200 ms prior to T2 onset. Single trials with ocular artifacts (exceeding 100 μV in VEOG or 80 μV in HEOG) and with other artifacts (zero lines, transients, large fluctuations, amplifier saturations) were excluded from analysis (9% total in the final sample of 16 participants, of these 6% ocular, 3% other artifacts). Separate average waveforms for each condition were then generated, and difference waves were constructed by subtracting the ERP waveforms elicited by the frequent T2 stimulus (non-'E') from the ERP waveforms elicited by the infrequent T2 stimulus ('E'). Amplitude and peak latency parameters of the P3 component were measured at Cz and Pz based on these difference waves following the elimination of high-frequency noise and alpha fluctuations using a low-pass filter at 5 Hz (phase-shift-free Butterworth filter; 24 dB/octave slope). This value of the low-pass filter was especially implemented to deal with the 12-Hz fluctuations in our data caused by the presentation rate (i.e., 1000/83.3–12 Hz). P3 peak latency in the individual ERPs was estimated in each condition as the point in time at which the largest positive value was detected within a 350- to 800-ms post-T2 interval. To estimate P3 amplitude in the individual ERPs, the P3 peak latency was first extrapolated from the grand-average waveform in each condition, and a temporal region of interest (tROI) of 100 ms was centered around each grand-mean P3 peak (i.e., −50 ms/+50 ms relative to the grand-mean P3 peak). The P3 amplitude was measured in this tROI for each participant and each condition.

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