

Suppression of EEG Gamma Activity May Cause the Attentional Blink

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The attentional blink (AB) is an impairment of attention, which occurs when subjects have to report a target stimulus (T2) following a previous target (T1) with a short delay (up to 600 ms). Theories explaining the AB assume that processing of T2 is more vulnerable to decay or substitution, as long as attention is allocated to T1. Existing models of the AB, however, do not account for the fact that T2 detection accuracy reaches the minimum when T2 is presented after about 300 ms and not immediately following T1. Therefore, a new model is suggested, which is based on chronometrical considerations together with recent neurophysiological findings concerning the relation between the P3 event-related potential and the AB, the interaction between P3 and gamma oscillations, and the significance of the early evoked gamma band response. We hypothesize that suppression of the early gamma response to T2, accompanying the P3 related to T1, causes the AB. © 2002 Elsevier Science (USA)

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INTRODUCTION

The attentional blink (AB) is a transitory blind spot of attention, which occurs when two target stimuli are presented in rapid succession. In this case the second target often cannot be identified correctly. This impairment of attention was reported for the first time in 1987 by Broadbent and Broadbent and has been replicated in many studies since then (see, e.g., Shapiro et al., 1997). The AB is observed in so-called rapid serial visual presentation tasks (RSVP), where visual items are presented with a short delay at the same location. In each trial subjects are required to detect two targets within the stream of rapidly presented items. Typical interitem intervals in RSVP tasks are of the order of 100 ms. Time lags between the first target (T1) and the second target (T2) are randomly varied between trials. The items immediately following T1 and T2 serve as backward masks, i.e., they reduce the visibility of T1 and T2 (e.g., Enns & Di Lollo, 2000). Under these conditions the detection of T2 was found to be impaired when T2 is presented within 600 ms following T1 (Raymond et al., 1992).

AB experiments are evaluated by recording the proportion of correct responses to the second target under the premise that the response to the first target was correct. The AB interval is defined as the time range after T1 presentation, when T2 detection in the dual target condition is worse than in the single target condition (when T1 is ignored). One might expect that the detection accuracy for T2 decreases continuously

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with a decreasing time lag between T1 and T2. However, a very surprising aspect of the AB is the fact that this is not the case. T2 detection accuracy was reported to reach the lowest point for a T2–T1 lag of about 300 ms (see Fig. 1). Detection accuracy for T2 recovers not only for longer, but also for shorter time lags.

Two conditions are fundamentally necessary for the AB to occur. First of all, attention has to be focused on T1. No AB is observed in the control condition, where subjects are instructed to ignore T1 (Raymond et al., 1992). Moreover, T2 has to be masked by the following distractor item of the serial visual stream. It has been shown that absence of masking or masks being spatiotemporally superimposed on T2 instead of backward masking are not effective in producing the AB (Kawahara et al., 2001; Brehaut et al., 1999). If, on the other hand, the distractor item following T1 is omitted, the AB has been found to be reduced, but not completely eliminated (Seiffert & Di Lollo, 1997; Chun & Potter, 1995). In contrast to the findings for T2 masking, backward as well as superimposed masking of T1 has been shown to be effective (Brehaut et al., 1999).

AB experiments revealed that T2 stimuli can facilitate reports of similar stimuli (“priming”), even when subjects are unable to report T2 (Luck et al., 1996; Rolke et al., 2001). This indicates that T2 stimuli in case of a “blink” are processed to a stage just short of awareness. Different mechanisms were proposed to account for the AB phenomenon. Theories explaining the AB have in common that they assume that as long as attention is allocated to T1 less attention is available for T2 and that processing of T2 is therefore more vulnerable to decay or substitution (Shapiro et al., 1997). The two leading theories of AB are the two-stage model (Chun and Potter, 1995) and the interference model (Shapiro et al., 1994). The two-stage model suggests

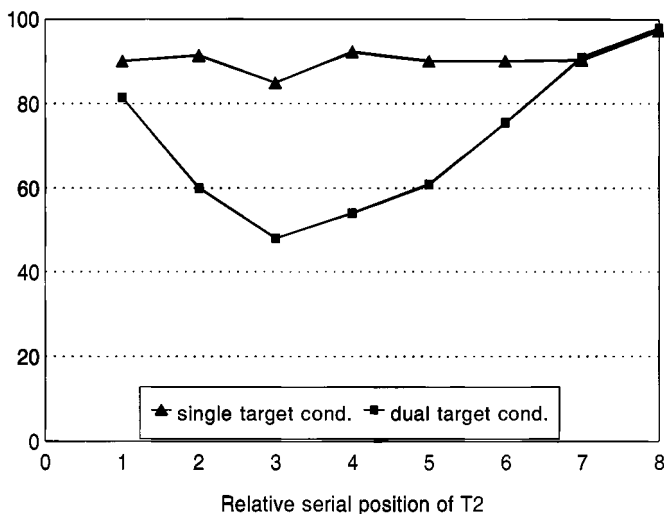


FIG. 1. Typical results of an AB experiment (adapted from Shapiro et al., 1997). Detection accuracy for T2 is plotted as a percentage on the Y axis and the delay of T2 (expressed in 90-ms intervals) with respect to T1 presentation is plotted on the X axis. The single target (ignore T1) condition is marked by triangles and the dual target condition by squares.

that all items presented in the rapid visual stream are processed to the point of conceptual representations without awareness (stage 1). The transfer of T2 representations into working memory is assumed to be impaired, as long as working memory (stage 2) is still engaged with T1. The interference model suggests that not the impaired transfer of T2 to working memory, but the interference of the representations of T1 and T2 within working memory causes the AB. Also hybrid versions of both models have been proposed (Vogel et al., 1998).

The temporal progression of stimulus processing is correlated to specific fluctuations of scalp voltage called event-related potentials (ERPs) (e.g., Luck et al., 2000). Hence, analyses of ERPs are well suited to addressing the question of which subprocesses related to T2 are influenced by T1 processing. Vogel and co-workers (1998) reported that no suppression of T2-related ERP components corresponding to sensory processing (P1 and N1) or semantic analysis (N400) occurs during the AB interval. However, a complete suppression of the T2-related P3 component was observed. The P3 component is understood to be a electrophysiological correlate of updating or closure of a context within working memory (Donchin, 1981; Verleger, 1988). Luck and co-workers thus concluded that the AB reflects an impairment occurring in a postperceptual stage of T2 processing.

CHRONOMETRICAL CONSIDERATIONS

Shapiro and co-workers (1997) have emphasized that existing theories of the AB phenomenon do not account for the fact that T2 stimuli being presented within approximately 100 ms after T1 are identified fairly well. A common explanation is that adjacent T1 and T2 items are processed in a single temporal episode. However, this argument is post hoc in nature and there is no plausible mechanism which would support it. Any reliable theory of the AB has to explain why detection accuracy for T2 reaches its minimum when T2 is presented about 300 ms after T1 and why T2 detection is better when T2 is presented immediately following T1.

A model of the AB thus has to elucidate which process Pr1 being triggered by T1 disturbs another process Pr2 related to T2. In particular, the peak timing of both processes must reveal the critical time lag of 300 ms between T2 and T1. Since the physical features of T1 and T2 are similar, one can assume that peak timing of subprocesses engaged in the processing of T1 and T2 is more or less identical. This leads to the requirement that t_1 (Pr1) - t_2 (Pr2) \cong 300 ms, where t_1 is time relative to T1 onset, t_2 is time relative to T2 onset, Pr1 is a process related to T1, and Pr2 a process related to T2.

In ERP experiments of the AB the P3 component is the latest significant ERP component occurring in response to T1 (Vogel et al., 1998; McArthur et al., 1999). Since in an AB design subjects are instructed to give responses after each trial has finished, response-related potentials are not expected to occur during the trials. For an AB experiment implementing letters and numbers as stimuli the peak of the P3 component related to T1 occurs about 400 ms after stimulus presentation. Because of the critical time lag of 300 ms, the process indexed by the P3 and related to T1 is thus the most probable candidate for Pr1.

Is There an Association between the P3 Component of the First Target and the Attentional Blink?

McArthur and co-workers (1999) have studied the relationship between the AB and the P3 component elicited by T1. Their investigation was motivated by the notion that AB and the P3 component have similar time courses. Moreover, slow surface positive potentials like the P3 have been assumed to represent a decreased excitability of cortical networks (Elbert & Rockstroh, 1987). Thus, both phenomena, the AB and P3, seem to be linked to inhibitory processes. In two RSVP experiments with different task difficulties they compared time course and peak amplitude of the P3 component related to T1 with the magnitude of the AB effect. They found that AB and the P3 component follow a very similar time course at the group as well as at the individual level. The reduction in RSVP task difficulty had a similar effect on the magnitudes of AB and the P3 component on the group level. However, this relationship could not be confirmed on the individual level.

McArthur and colleagues concluded that there is a moderate association between the AB and T1-related P3. The impairment in visual processing of T2 appears to coincide with the amplitude of the P3 occurring in response to T1. It has been reported that reaction times in response to probe stimuli (T2) are prolonged (Woodward et al., 1991) and that startle reflexes are smaller (Schupp et al., 1994) when stimuli are presented after target stimuli (T1) eliciting a large P3. These findings support the model of a reduced cortical excitability correlated to the P3. McArthur et al. thus suggested that the cortical disfacilitation associated with the T1-related P3 is responsible for the impairment in T2 processing. In the following we assume that the process Pr1 interfering with T2 processing is working memory updating corresponding to the P3 component elicited by T1.

Which Process Pr2 Might Be Disturbed by the P3 Component of the First Target?

As Vogel and co-workers (1998) reported, the AB leaves the early components of sensory processing (P1 and N1) of T2 intact, whereas the P3 of T2 is completely suppressed. Thus, the process Pr2 being disturbed in the case of AB on first sight appears to be the updating (or closure) of working memory corresponding to the P3. In order to directly interfere with the P3 related to T2, the peak of Pr1, however, would have to occur about 700 ms after T1. On the precondition to be measurable as an ERP component, such a late process peaking after the P3 of T1 has not been observed in AB paradigms (Vogel et al., 1998; McArthur et al., 1999). The chronometrical considerations described above suggest that working memory updating related to T2 is not directly affected by T1 processing. It is more plausible that another process preceding and being necessary for initialization of working memory updating related to T2 is disturbed by Pr1.

Because N1 and P1 related to T2 are unaffected by the AB, McArthur and co-workers (1999) expected that the interference of T1-related P3 with T2 processing occurs at the time when T2 already has been fully identified and is available for probe discrimination. This time point is supposed to coincide with N2 latency, which occurred about 235 ms after T2 presentation. The underlying idea is that process Pr2

corresponds either to the N2 component or to the transition from the N2 to the P3. McArthur and co-workers thus shifted the AB waveform forward by 235 ms to compare the time courses of T1-related P3 and the AB. Coming back to chronometrical terms this means

$$t_1 (\text{Pr1}) = t_1 (\text{P3}_{\text{max}}) \cong 400 \text{ ms} \wedge t_2 (\text{Pr2}) = t_2 (\text{N2}_{\text{max}} \text{ or } \text{N2/P3}) \\ \geq 235 \text{ ms} \Rightarrow t_1 (\text{Pr1}) - t_2 (\text{Pr2}) \leq 165 \text{ ms}.$$

The time relation between the assumed processes Pr1 and Pr2 obviously does not fulfill the requirement of a 300-ms time lag. For the above model, additional post-hoc explanations would be needed to explain the 300-ms time lag, such as that the peak of the ERP components does not correspond to the peak intensity of the related processes or others. In search of a process, which may be disturbed by Pr1 and which is necessary for successful initialization of the T2-related P3, it would be more straightforward to focus on a process occurring around 100 ms after T2 presentation. If N1 and P1 related to T2 are unaffected by the AB, what kind of process could that be?

AN ALTERNATIVE EXPLANATION

In recent years evidence has accumulated that EEG activity in the gamma frequency range (>20 Hz) plays an essential role in perceptual and cognitive processes (e.g., Engel & Singer, 2000). Two types of gamma band activity occurring in response to sensory stimuli can be differentiated. Firstly, the evoked response, which is observed around 100 ms after presentation of a visual stimulus and is time-locked to the stimulus. The evoked response is followed by induced gamma activity, which occurs in a non time-locked fashion. In simple visual detection tasks, induced gamma activity can be observed in a time range of up to 400 ms after stimulus onset.

Both evoked and induced gamma activity seem to play an important role in attention (e.g., Gruber et al., 1999; Müller et al., 2000; Herrmann & Knight, 2001). In the following, we focus on the evoked gamma response, since it fits with the time requirements for process Pr2. Tiitinen and co-workers (1994, 1997) have shown that the auditory evoked gamma response is enhanced by selective attention, attenuated in the course of long-term stimulation, but is not affected by changes in stimuli. They concluded that the evoked gamma response is closely related to selective and sustained attention. Enhancement of the visually evoked gamma response by task-driven attention has been reported by Sokolov and colleagues (1999).

Recently, Herrmann and Mecklinger (2001, 2000) investigated evoked and induced gamma activity in a visual classification task. They found that the early evoked gamma response occurring about 100 ms after the stimulus is affected by the targetness of a stimulus and the need to discriminate between the features of a stimulus. Herrmann and Mecklinger supposed that the more occipital part of the early gamma response represents the automatic bottom-up processing of the stimuli (Herrmann & Mecklinger, 2000). The more frontally located part, however, seemed to be present only when discrimination of the perceived features is relevant for task performance. These findings suggest that the early gamma response is necessary for the allocation of attention to a selected object and thus for successful stimulus discrimination. An

intact early evoked gamma response thus might be important also for subsequent initialization of the P3 component (working memory updating). However, this interpretation has to be proved by further investigation.

As already mentioned, the timing of the early evoked gamma response fits with the requirements for process Pr2, which is disturbed in the case of the AB. In addition, the functional specificity of the early gamma response as reported by Hermann and Mecklinger (2001, 2000) is consistent with the finding that no P3 component is elicited by the second target (T2) during AB. But is there any evidence that the early gamma response might be impaired by the P3 component elicited by T1? Indeed, several groups reported that gamma oscillations are diminished during the occurrence of P3 responses in auditory oddball experiments (Fell et al., 1997; Marshall et al., 1996; Bertrand et al., 1998). Deviant (target) stimuli produce a significantly larger P3 component than standard (nontarget) stimuli. For target stimuli smaller amplitudes of induced gamma activity were observed in the P3 time range than for nontarget stimuli (Fell et al., 1997; Marshall et al., 1996; Bertrand et al., 1998).

Slow cortical ERP components are understood to provide a threshold controlling the excitability of cortical networks (Elbert & Rockstroh 1987; Schupp et al., 1994). The P3-related suppression of gamma activity thus can be interpreted as resulting from a widespread cortical inhibition associated with the surface-positive P3 component (Fell et al., 1997; Marshall et al., 1996). Although these findings were reported for induced gamma activity, the underlying reduction in gamma power should in principle also diminish time-locked evoked responses, if those were present in the affected time range. However, the described gamma-P3 interactions can only provide indirect evidence for our hypothesis, because they are related to the auditory and not the visual domain and gamma activity and P3 are elicited by the same stimulus and not successive stimuli in those studies. Further investigations are thus needed to explore the interaction between visual P3 and gamma oscillations, particularly in the AB paradigm.

As initially described, focusing attention on T1 stimuli is necessary to produce the AB. The fact that generation of the T1-related P3 is based on the same condition supports our hypothesis. Due to the narrow time window of around 100 ms during which the early gamma band response occurs (Herrmann & Mecklinger, 2000, 2001), our model predicts that the time course of the AB is mainly determined by the relatively broad time course of the T1-related P3. This is indeed the case, as has been noticed by McArthur and colleagues (1999). The second necessary factor for the AB is backward masking of T2 (Kawahara et al., 2001; Brehaut et al., 1999). It has been suggested that backward masks may overwrite the iconic images of the targets and thus force identification of the targets at the time that they are presented (Chun & Potter, 1995). In the frame of our hypothesis, backward masking of T2 may ensure that processes associated with the T2 gamma response cannot be delayed. Thus, interference of the evoked T2 gamma response with the T1-related P3 is inevitable.

In contrast to T2 masking, the AB effect is increased in case of backward as well as superimposed masking of T1 (Brehaut et al., 1999). Compared to T2 masking, the effect of T1 masking is rather nonspecific and has been attributed to the increased amount of attention which T1 requires to be recognized in case of masking (Brehaut et al., 1999; Seiffert & Di Lollo, 1997; Chun & Potter, 1995). The finding that P3

amplitude is sensitive to the allocation of attentional resources (Donchin & Coles, 1988; Wickens et al., 1983; Sirevaag et al., 1984) may link the effect of T1 masking to our hypothesis. For example, an amplitude enlargement of a late part of the visual P3 due to masking was observed by Okita et al. (1985). To our knowledge, however, no systematic studies directly exploring the relation between visual P3 amplitude and masking have been published. In a recent functional magnetic resonance study Marois et al. (2000) investigated the hemodynamic correlates of different manipulations of T1 masking during rapid serial stimulus presentation. This study is only partially related to our hypothesis, because the enhancing effect of T1 masking on the AB, but not the AB effect per se, was investigated. However, the most consistent activation associated with increased masking interference was localized within the intraparietal sulcus in this study. Since this region was also identified by depth recordings as one of the major generator regions of the P3 component (Halgren et al., 1995, 1998), the findings of Marois et al. (2000) are in line with our hypothesis.

In conclusion, we hypothesize that the early evoked gamma band response is the process Pr2, which is impaired by T1 processing. In accordance with the chronometrical requirements for process Pr2, the early gamma response peaks around 100 ms after visual stimulus presentation. Suppression of gamma oscillations accompanying P3 waves has consistently been reported by several groups. Thus, disturbance of the early evoked T2 gamma response by the T1-related P3 component seems likely. Impairment of the early gamma response may then cause the absence of the T2-related P3 and prevent T2 from reaching awareness. We therefore propose that suppression of EEG gamma activity may be responsible for the attentional blink.

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