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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., Kranczioch, 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; Kranczioch 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.