Does P3b guide and sometimes dictate our behavior?

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Abstract

Several studies indicated that the P3b component is related to both stimulus and response processes. Referring to this, we proposed a new functional account of the P3b as manifestation of memory consolidation processes that has short-enduring implications on performance. Using the arrow version of the Eriksen flanker task, we tested this assumption on sequential effects. By modulating the trial sequences, it was predicted that the functional relationship of the P3b and the reaction time could help explain the partial dependence of the compatibility effect on the preceding sequence. Trial repetition sequences were predicted to result in a beneficial relation of the P3b and the RT because of facilitating effects of a build memory trace. Alternation sequence should manifest a negative link between these factors because the existing memory trace impairs subsequent performance. The data did not confirm the expected relationship on the sequential flanker effect. However, the P3 amplitude was affected by the amount of conflict on the present trial where low conflict trials generated greater P3b amplitudes. This result was interpreted in terms of greater consolidation processes and subsequent behavioral benefits due to less conflict.

Introduction

The paper is organized into sections which present different theories that are related to the present empirical question. At the beginning, important concepts of the functional role of P3b are obtained. Subsequently, sequential effects are outlined. Based on the previous theories, the last section introduces a new functional account of the P3 component and presents the associated hypotheses.

P3

The P3 component is an event-related potential which is maximal over the centro-parietal scalp peaking within 300-600ms. The amplitude of the P3 has been shown to be affected by various factors that elicit cognitive processes, such as event probability, attentional resource allocation or motivational significant processes (for a detailed review see: Polich, 2007). More specifically, it has been functionally and structurally shown that P3 consists of two subcomponents, the frontal P3a, and the centro-parietal P3b (Squires, Squires & Hillyard, 1975; Jentzsch & Sommer, 2001). Polich (2007) summarized the P3 as a reflection of "an information processing cascade when attentional (reflected in P3a) and memory mechanisms (indicated by P3b) are engaged" (p. 2128).

One of the most influential accounts of the P3's functional role comes from Donchin (1981) who related the P3 component to the updating of mental representations by external stimuli. His theory stated that initial sensory processing is followed by attentional processes that govern an evaluative comparison of the current stimulus presentation and a previous one in working memory. The detection of a change in the external presentation results in an "update" of the mental representation that is reflected by the P3 component, particularly the P3b. There is a lot of evidence that implied the psychological significance of the P3b component to memory processes (Kok, 2001; Polich, Ladish, & Bloom, 1990). For example, Karis, Fabiani, & Donchin (1984) demonstrated that words recalled from a studied list were accompanied by larger P3 amplitude on initial presentation than words not recalled ("dm" effect: difference due to memory). Increasing memory load has further been found to generate smaller P3b amplitudes (Brookhuis, Mulder, Mulder, Gloerich, van Dellen, van der Meere, & Ellermann, 1981). Interestingly, several investigations discovered that the manipulation of the target-to-target time interval affected the P3 component (Kok, 2001; Gonsalvez & Polich, 2002). Kok (2001), for example,

observed that the amplitude of the P3 was reduced after a rapid target presentation interval compared to longer intervals with enhanced component size. In a different paradigm, the attentional blink, subjects were exposed to a rapid stream of visual stimuli. After the detection of a target within this stream, subjects were subsequently impaired to perceive a second target for a brief period. It was found that the P3 component was totally suppressed after the presentation of the second target (Vogel, Luck, & Shapiro, 1998). It is worth mentioning the two different paradigms because both arrived nevertheless at the same conclusion, namely to P3 as reflection of memory trace development.

But there are also contrasting opinions about the functional role of the P3. Verleger (2008), for example, doubts the relationship to memory related functions because different findings revealed that the peak latency of the P3 was influenced by response processing (Verleger, Jaskowski, & Wascher, 2005). He used this as evidence against the context-updating hypothesis which assumes that the P3 component is independent of response processing. According to Verleger, a possible alternative hypothesis lay in decision-related processes in which the P3 mediates between processes of stimulus identification and response selection. He further questioned the role in memory related processes by referring to the widely distributed scalp generators of the P3 component. Various investigations indicated that especially the temporal-parietal junction (TOJ) affected the component size (P3b), and that memory-related structures as the hippocampus played only an indirect role in the generation of the P3b (Bledowski, Prvulovic, Hoechstetter, Scherg, Wibral, Goebel, et al., 2004). The absence of neuropsychological reports that linked the TOJ to memory formation processes would, according to Verleger (2008), question the validity of the context-updating hypothesis or other memory-related accounts.

As there is still no answer in the ongoing 40 years debate about the functional role of the P3 component, we proposed a new theoretical account which tried to reconcile the two mentioned theories of Donchin, Polich, and Verleger that appeared only initially contrasting. To detail, it was intended to explain why the P3 appeared to mediate both stimulus- and response-related processing.

Sequential effects

Several studies highlighted that the amplitude of the P3b was not only affected by a stimulus but also by the preceding stimulus sequence (Jentzsch & Sommer, 2001; Squires, Wickens, Squire & Donchin, 1976). Repetitive sequences resulted in decreased P3b amplitudes even when the overall event probabilities were equal and randomly ordered (Squires et al., 1976). This discovery resembles the effect on reaction times that were similarly reduced after repetitive sequences (Remington, 1969). Both findings were first explained by expectation effects, where the continuation of an event would result in an expectation-induced information processing- and behavioral benefit. Analogously, alternations would cause expectation-triggered costs in processing and performance. More recently, it has been shown that sequential effects may have properties that are not explainable by expectancies alone (for detail see: Soetens, Boer, Hueting, 1985).

The mentioned sequence effects were similarly found in conflict tasks, like the Simon-, the Eriksen- or Stroop task (Wendt, Kluwe, & Peters, 2006; Egner, 2007). In the arrow version of the Eriksen task, participants are required to respond to the pointing direction of a central target arrow, which is surrounded by task-irrelevant arrows (flankers). These flankers have been found to influence the processing of the target. In an incompatible condition, the flankers point to a different direction than the target stimulus. This incongruence induces conflict so that the reaction time (RT) and accuracy decreases compared to compatible conditions where all arrows (target and flankers) point to the same direction (Eriksen & Eriksen, 1974). Gratton, Coles & Donchin (1992) were the first who reported that the compatibility effect (difference in RT between compatible and incompatible trials) in a conflict task was partially reduced by the sequence of the preceding trial. They observed that the compatibility effect was reduced for trials that were preceded by a high conflict trial (incompatible trial) than when it was preceded by a low conflict trial (compatible trial). RTs on compatible trials preceded by a compatible trial (C-C) received an additional acceleration in comparison to compatible trials that were preceded by an incompatible trial (I-C). In the same manner, RTs on incompatible trials were faster following incompatible trials (I-I) than following compatible trials (C-I). The same applies to error rates. The conflict-monitoring hypotheses which was formulated by Botvinick, Braver, Barch, Carter, & Cohen in 2001, has been most often used to elucidate the sequential dependency effect in conflict tasks. This theory predicts that conflict on the

preceding trial triggers an increase in cognitive control, which subsequently results in reduced conflict impairments on the current trial. The most important, already existing challenge to the conflict-monitoring hypothesis was introduced by Mayr, Awh, & Laurey (2003). They proposed that priming effects caused the faster RT for C-C and I-I trials relative to I-C and C-I trials, thereby emphasizing the role of stimulus specific repetition priming. When associations made in the previous trial were reactivated in the following trial as in the case of C-C and I-I sequences, RT could benefit from the already existing association. Importantly, the authors found that the elimination of repetitions abolished the conflict adaptation pattern (sequence effect). This stands in contrast to the conflict monitoring hypotheses where stimulus repetitions are totally irrelevant.

Interestingly, there are indices that the amplitude and latency of the P3 are correlated to the amount of information extracted from a priming stimulus and subsequent RTs (Gratton, Bosco, Kramer, Coles, Wickens, & Donchin, 1990). Furthermore, the P3b has also been found to have consequences for future behavior by relying on prior events (Munson, Ruchkin, Ritter, Sutton, & Squires, 1984).

New functional account of P3b

The present paper was intended to introduce a new functional significance of the P3 by relating it to one of our most basic cognitive abilities, namely to adapt to the future sequence of event by a preceding event. This capability represents basic learning processes and flexible adjustment of our behavior to everyday demands.

The association of the P3 component to memory formation processes that generates short-enduring performance benefits serves as a link of the P3 to stimulus- and response processes. The theory thereby especially emphasizes the purpose of memory processes. The link of memory processes and a resulting performance benefit is consistent with empirical findings (Radeau, Besson, Fonteneau, & Castro, 1998). By referring to this, we additionally offered an alternative explanation for the sequential effects in the Eriksen task which stands in contrast to the conflict-monitoring hypotheses. We explained the sequential dependency effect by suggesting that the P3 component is an aftereffect of stimulus processing, and that P3b particularly indexes memory consolidation processes. This means applied to a conflict task, that the facilitation effects in the continuation of an event (CC or I-I) are a consequence of a memory trace (reflected by P3b) that is formed in the previous trial and induces

behavioral benefits into the current trial (indicated by RTs). In alternation sequences (IC or CI), the opposite is expected where the consolidation of a stimulus presentation distorts the processing of the following trial and triggers behavioral costs. Thus, at the basis lies the idea that residual activity from the previous trial exerts its influence into the current trial. Therefore, a functional relationship between the P3b amplitude in the preceding trial and the RT in the current trial was hypothesized. Notably, the P3 amplitude was examined on the preceding trial as opposed to previous studies that focused on effects at the current trial. The effect of memory consolidation should be most reflected within a repetition or alternation sequence. Our hypothesis is based on the idea that the amplitude of the P3b informs us on the strength of the build memory trace. As a consequence, within repetition trials fast responses should be preceded by higher amplitudes than slow responses because of greater "consolidation effects". The opposite is predicted within alternation trials, whereby fast responses are preceded by lower amplitudes than slow responses so that less consolidation of a memory trace hampered the processing of the next stimulus trial less (see Figure 1). Our proposal parallels Mayr et al.'s account (2003) because if the P3b component reflects memory consolidation processes which dictates the behavior on the next trial, the priming effect is localized within the information processing chain. To test our hypothesis, we used the arrow version of the flanker Eriksen task as described above.

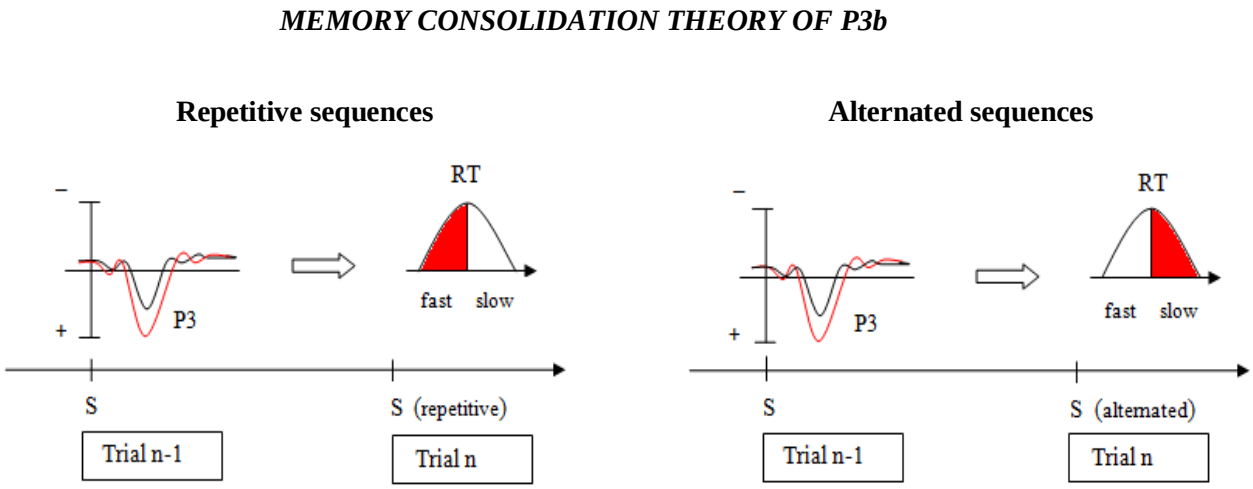


Fig.1. Schematic illustration of the memory consolidation theory of P3b. This model presents the functional relationship of the P3b component and the RT which depends on the type of sequence. The consolidation of a memory trace (indexed by P3b) has facilitating effects on RT on repetitive sequences(C-C or I-I). Higher P3b amplitudes in the preceding trial have therefore above-median RTs on the current trial as consequence (left side of RT distribution). A formed memory trace induces behavioral cost in alternated sequences (IC or CI). The greater the consolidation processes on the preceding trial, the higher the distorting behavioral effects on the current trial indicated by below-median RT (right side of RT distribution).

Methods

Participants

In total, 19 students from the University of Twente participated for about 2 hours in the experiment to meet requirements for their Bachelor's degree. All participants (gender: 15 women, 4 men, age range: 18-25, handedness: 18 right-handed, 1 left handed) had normal or corrected-to-normal vision and were neurologically healthy (self-report). The data of two participants were excluded from the EEG analysis because of great artifacts in the reference electrodes. The study was approved by an ethical board and an agreed, informed consent was necessary for participation.

Stimuli and apparatus

The experiment was conducted on a PC computer with "Presentation" software. The participants were positioned at about 60 cm from a 17 computer screen in a darkened room. The visual angle of the experimental subjects was 4.8 degrees. A white fixation point, centered on a black computer screen, was presented for 500 ms before each trial. A stimulus array of 11cm that consisted of five white arrows was presented within a trial for 500ms on a black background. The total trial duration comprised of 1500ms. The inter-trial interval was 2 seconds.

Task and procedure

An arrow version of the Eriksen task was used. The central arrow which was enclosed by four irrelevant arrows was the target stimulus that determined the response. The surrounding arrows, the flankers (all identical) had to be ignored. The participant's responses were given with the left or right index finger and should be in accordance with the pointing direction of the central arrow. So arrows pointing to the right/ left were correctly responded with the right/ left "ctrl" key, respectively. Feedback was only given when participants made erroneous responses. Each trial was preceded by a central fixation period. To control for eye artifacts, participants were told to fixate their eyes on the central point continuously and to prevent eye blinks during the stimulus presentation. In total, we studied four blocks each consisting of 120 trials, respectively (in total 480 trials). The sequence of trials was manipulated leading to four different conditions 1) compatible trials preceded by compatible trials (C-C),
2) incompatible trials preceded by compatible trials (C-I), 3) compatible trials preceded by incompatible trials (I-C), 4) incompatible trials preceded by incompatible

trials (I-I). The sequences were ordered randomly preventing the formation of expectations about the following trial. The four blocks were separated by a screen that was presented for 5000ms in which the participant could take a short break. Before participants started with the experiment, they underwent a practice session of 20 trials to ensure that they used the right stimulus-responds maps. Speed and accuracy were equally stressed.

Data acquisition

The event-related potentials (ERP) were recorded from 15 Ag/ AgCl ring electrodes (Fz, F3, F4, FCz, Cz, C3, C4, Pz, P3, P4, PO7, PO8, Oz, the left and right mastoid) which were attached to a standard 10/10 cap and ordered according to the international 10-20 system. All electrodes were offline referenced against the left and right mastoid electrode. To exclude trials that were distorted by eye movements bipolar electrodes were utilized to measure horizontal (1cm lateral to the outer canthi) and vertical electrooculograms, EOGs (1 cm above and below the eyes). All channels were amplified by a Quick-Amp amplified and sampled at 1000 Hz. The continuous EEG was filtered with a bandpass from 0.5Hz (down 12dB/oct) to 20 Hz (down to 12dB/oct). Electrode impedance was hold under the 5-10 kΩ. The RT and the accuracy were assessed for the behavioral data.

Data analysis

Trial sequences were rejected from the analysis that consisted of errors in the current or preceding trial, premature responses (earlier than 150ms poststimulus), too slow responses (after 1500ms), and the very first trial because of the non-existing preceding trial. Regarding the EEG signal, trials without ocular artifacts and segments in which amplitudes did not exceed -100uV or +100uV were included in the analysis.

The trials were selected offline by the preceding stimulus type (n-1) as well as by the stimulus type on the current trial and its corresponding RT velocity (n). The RT is here of particular importance because the median values for every participant per stimulus category was calculated to classify fast (above median) and slow responses (below median). In the end, we produced eight stimulus categories [compatibility of the current trial (2x), compatibility of the preceding trial (2x), and response velocity (2x)]. A comparison was made within stimulus sequences (C-C, C-I, I-C, I-I) between the P3b in the preceding trial (n-1) on fast and slow RTs in trial n. This was done to

determine whether there was the expected functional relationship between the amplitude of the P3b and the RT. The Stimulus-locked ERP epoch was baselined to 100 ms prestimulus and ended 1500 ms after the stimulus presentation. Because the RT data showed significant differences (50ms) between compatible and incompatible conditions we used the peak amplitude as dependent variable. This choice prevented any artificially induced amplitude difference due to latency variations (latency jitter) as could be the case with mean amplitudes. Peaks were detected at the specified latency range of 275-475ms focusing on the midline Pz electrode. Amplitude measurements were taken at the latency of 300-400ms on the same Pz electrode for the creation of topographic maps.

Statistical Analysis

Sequential effects were expressed in a significant interaction of the current and preceding trial. Mean RT for correct responses was subjected to repeated measures ANOVA with the within-subject factors current trial type n (compatible, incompatible) × preceding trial type n-1 (compatible, incompatible). The RT was therefore the dependent variable. To test for difference between trial sequences we used the Paired Sample T-Test for each sequence pair C-C & C-I, I-C & I-I.

For the parietal P3b component, analysis of the peak amplitude was restricted to the Pz electrode. The P3b amplitude was evaluated by means of repeated measures ANOVA with the three within-subject factors, previous trial type (compatible, incompatible) × current trial type (compatible, incompatible) × response velocity (fast, slow). A three-way interaction of these factors was assessed to determine whether there was the predicted functional relationship between the P3b amplitude on the preceding trial and RT on the current trial. Further, we used a Paired sample t-test to examine whether there was a difference within sequence conditions on response velocity (fast & slow). The whole analysis consisted of a within-subject design.

Results

Behavioral analysis

The Tables 1 and 2 summarize the main findings on RT and accuracy. As expected, there was a great influence of the current trial type, showing significant difference between faster compatible *(453ms)* and slower incompatible trials *(504ms)* with *F(1, 18) = 185.1* and $p < .001$. Further, the data confirmed a significant interaction between compatibility on the current trial (n) and the preceding trial type (n-1), with *F(1, 18) = 12.8* and $p = .002$ validating the predicted sequential dependency effect. Thus the repeated measures ANOVA supported previous results on RT in an Eriksen task. A pairwise comparison of the trial sequences C-C with I-C showed a significant difference in the RT with *t(18) = -3.8* and $p = .001$. RTs were faster on C-C trial sequences *(448ms)* compared to I-C ones *(457ms)*. There was only a slight difference of *504ms* on C-I trials and *503ms* on I-I trials which was too small to reach the level of significance, *t(18) = -.7* and $p < .5$. From the RT data can be concluded that sequential modulation partially reduced the compatibility effect on reaction times. Slightly different results were observed for accuracy rates. Error rates indicated a significant difference between trial types on the current trial n. Compatible conditions resulted in smaller error rates *(.8%)* than incompatible trials *(3.5%)* with *F(1, 18) = 18.0* and $p < .001$. However, in contrast to the reaction times there was no statistical evidence that the trial type on the current trial (n) was influenced by the preceding trial type (n-1) with *F(1, 18) = 1.0, p = .34.* A Pairwise comparison tested for differences between the trial sequences C-C and I-C, as well as, I-I and C-I. We obtained for none of these pairs a significant difference with *t(18) = .4, p = .72,* and *t(18) = -1.1, p = .28* respectively. This means in other words, that faster but not more accurate responses were given on trials in which the trial types repeated.

Table1

Mean Reaction Times and Standard Error (in Parentheses) as a Function of Flanker Compatibility on Trial n and n-1

Trial n-1	Comp (n-1)	Incomp (n-1)	F(1,18)
Comp (n)	448.36 (17.27)	457.24 (17.84)	12.81
Incomp (n)	504.47 (15.39)	502.69 (16.92)	12.81

Note. Comp= Compatibility, Incomp= Incompatibility

Table 2

Mean Error Percentages and Standard Error (in Parentheses) as Function of Flanker-Compatibility on Trial n and n-1

Trial n-1	Comp (n-1)	Incomp (n-1)	F(1,18)
Comp (n)	0.85 (0.39)	0.77 (0.33)	0.96
Incomp (n)	3.79 (0.76)	3.18 (0.68)	0.96

Note. Comp= Compatibility, Incomp= Incompatibility

Event-related potentials

The compatibility of the trial from which the ERP was extracted affected the peak amplitude significantly (see Figure 2). The repeated measures ANOVA demonstrated the compatibility effect with $F(1, 16) = 15.2$, $p = .001$. Compatible trials invoked therefore higher peak amplitudes, on average $10.8\mu V$ *(SE .92)*, than incompatible trials with an average of $9.0\mu V$ *(SE .82)*. This means applied to the task, that the peak amplitude of the P3 component was reduced by high conflict on that trial.

The data did not result in an interaction effect of the three within-subject factors compatibility of the previous trial (compatible, incompatible), compatibility of the current trial (compatible, incompatible), and response velocity (slow, fast) with $F(1, 16) = .01$ *and* $p = .92$. This generally speaks against our hypothesis that the P3b amplitude could explain reaction time benefits and costs on the basis of sequential effects. Therefore, the partial dependence of the compatibility effect on sequences could not be explained by the P3b amplitude and the associated memory processes.

Surprisingly, we found a significant interaction of the factors compatibility on the current trial and response velocity on the peak amplitude (selected at the preceding trial) with $F(1, 16) = 5.9$ and $p = .027$. The interpretation of this effect was complicated because it is irrelevant that something happening in the future (trial n) had an effect on the immediate past (trial n-1). This result possibly reflects a type 1 error.

We further compared the peak amplitude in trial sequences (C-C, I-C, C-I and I-I) on fast and slow response velocities in a Paired Sample T-Test. The grand mean averages of the P3b for the different conditions are plotted in Figure 3. The only significant difference found was the I-I sequence with $t(16) = -2.5$ and $p = .024$, where fast responses on the current trial were preceded by a higher peak amplitudes $(10.0\mu V)$ on the preceding trial compared to slow responses $(9.0\mu V)$. This difference is in

accordance with our prediction of the trial-repetition effect which is due to the formation of a memory trace that induces behavioral benefits on repetition sequences only. Comparison of the trial sequence C-C on slow and fast responses did not show a main effect of RT velocity with $t(16) = .6$, $p = 0.55$. Similar results were obtained for the I-C and C-I trial sequences on slow and fast responses with a nonsignificant difference of $t(16) = -.1$ with $p = .9$ and $t(16) = 1.7$, $p = .11$, respectively.

Finally, the results reflected in the RTs did not converge with the outcomes of the event- related potentials. While the RT showed significant sequential effects, the P3 amplitude appeared to be influenced only by the compatibility of the trial on which it was recorded.

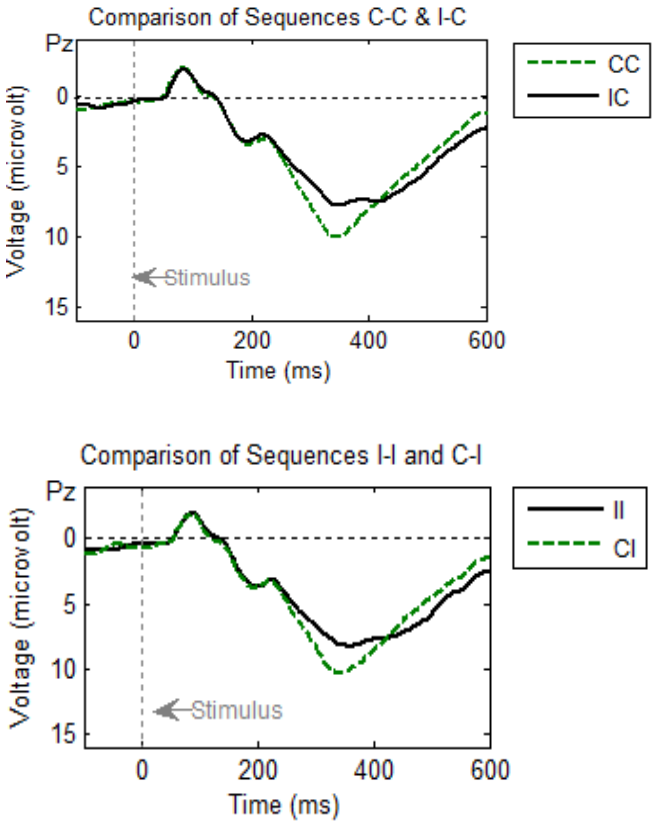


Fig.2 *The effect of conflict is seen in the difference between the mean P3b amplitude on C-C sequence (green) compared to the amplitude on I-C sequence (black). It is similarly reflected in the I-I sequence (black) compared to the amplitude on the C-I sequence (green).*

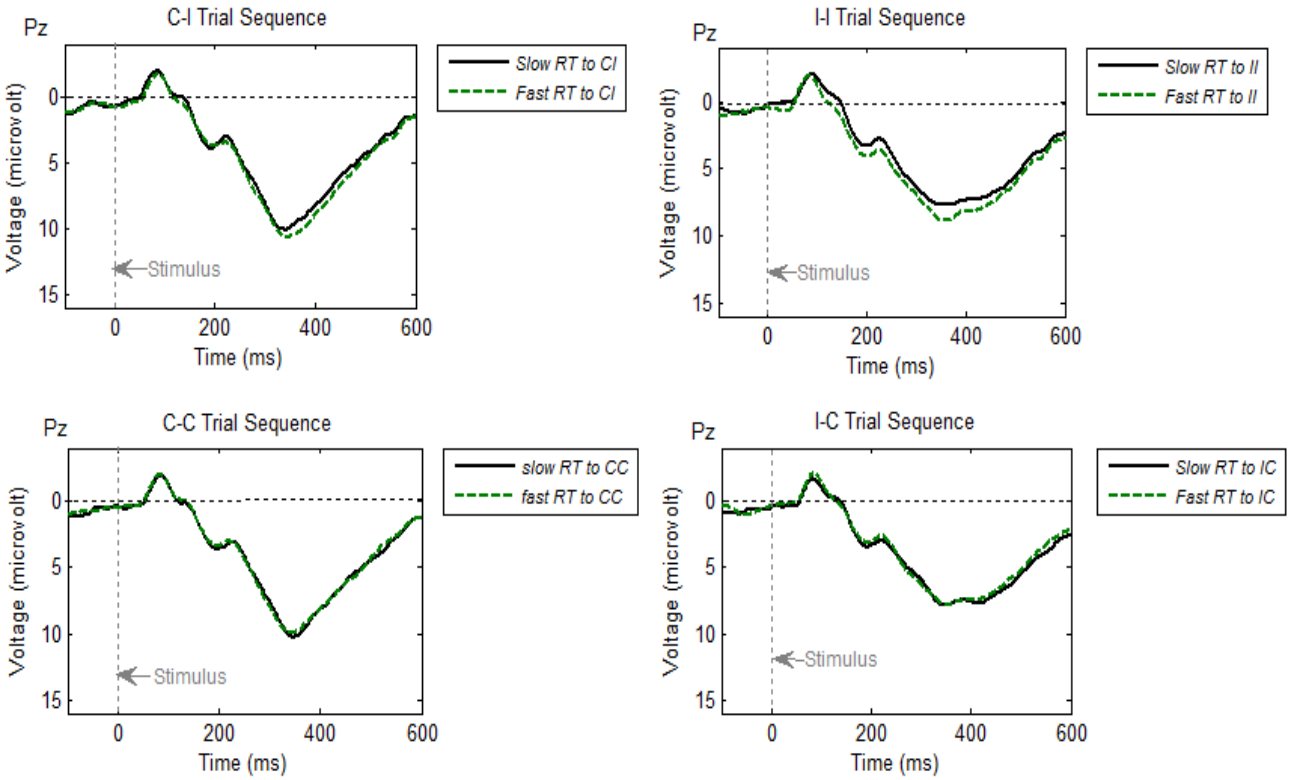
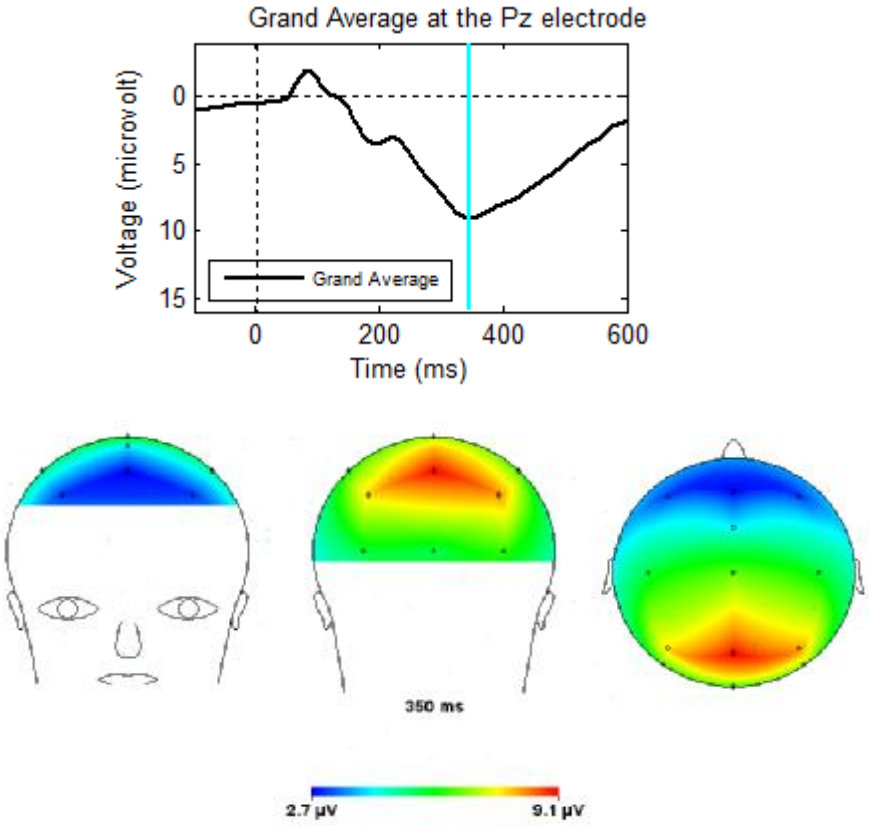


Fig.3 *<u>At the top</u>: Mean P3b amplitudes for the different sequence modulations C-I, I-I, C-C, I-C as function of response velocity (fast= green, slow=black) on trial n. <u>At the button</u>: The Grand Average computed over all conditions at the Pz electrode with the scalp distribution.*



Discussion

This study was intended to introduce a new account of the psychological significance of the P3 component which could provide an explanation for the sequential effects in the flanker task. Previous literature indicated that the P3 component had consequential effects on stimulus and response processing. With the new formulation of P3b as manifestation of memory consolidation processes, we tried to explain the relationship of the P3b to stimulus and response processes on sequential modulations. It was predicted that within repetition trials, P3b had beneficial effects on RTs whereas alternation trials should manifest a distorting influence of the P3b on RT due to their functional relationship. While the behavioral data partially reflected a sequential effect (the RT data) the P3b unfortunately appeared to be unaffected by it.

The RT data confirmed previous results demonstrating that the overt behavior was significantly influenced by the compatibility of the previous and current trial, and also by the compatibility of the current trial only. The sequential effect was greatest in the difference of RT of the C-C and I-C trial sequences. The accuracy was only influenced by the current trial type excluding a sequential effect. This was probably evoked by the ease of the task which stimulated sequential effects in the RT but not in the error rates. The instructions emphasized both speed and accuracy which however did not exclude the possibility of strategic effects taken by the experimental subjects.

The ERP data was in general not consistent with our predictions, reflecting no interaction effect of the three within-subject factors compatibility of the previous trial (compatible, incompatible), compatibility of the current trial (compatible, incompatible), and response velocity (slow, fast). Therefore, the P3b was obviously not responsible for the partial reduction of the compatibility effect by sequence modulations which falsified our theory. However, the trial sequence I-I as a function of the response velocity (fast, slow) reflected significant differences on the P3b peak amplitude. Consistent with our hypotheses, this target repetition sequence resulted in a beneficial relation of peak amplitude on the preceding trial and the RT on the current trial. Higher peaks were therefore followed by faster RT on the next trial. The reason why we could not find such an effect in the C-C repetition sequence could depend on several factors. Various studies supported the idea that working memory is stronger related to performance in high conflict trials compared to performance in low conflict trials (Kane & Eagle, 2003). Additionally, there were indications that priming effects were larger after an incongruent than after a congruent trial which was called conflict

dependent priming effect (Davelaar & Stevens, 2009). However, in our study this was only true for the ERP data in the I-I sequence and was not exhibited in the RTs where C-C sequences reflected the greatest priming effect. We cannot provide an explanation why the RTs and the ERPs were so conflicting on the basis of our theory. Probably the discrepancies between the RT and the P3b on sequential effects may suggest that both rely on different processes.

However, there were few methodological problems in this study which are worth mentioning. One of these is that we determined repetition trials on the basis of the targets only. Our results could be confounded by stimulus-response (S-R) repetitions as Mayrs, Awh and Laurey (2003) criticized earlier regarding the conflict monitoring theory. The underlying problem lies in the fact that 50% of the C-C and I-I consisted of S-R repetitions trials whereas the I-C and C-I trials were totally free of S-R repetitions. Similarly, 50% of the I-C and C-I trials persisted of response repetition trials in the absence of a stimulus repetition. According to the feature integration theory (Hommel, 2004) the co-occurrence of a stimulus and response in time were accompanied by the storage of this association. This means particularly that the activation of one stimulus feature reactivates automatically the associated response. This confounding might have been responsible for the ERP data that appeared independent of sequential effects in our study. Several studies pointed out, though for RT and accuracy, that significant interaction effects between previous and current trial types were only found for (S-R) repetition trials when controlling for the last factor (Nieuwenhuis, Stins, Posthuma, Polderman, Boomsma, & Geus, 2006). As a consequence, we advise future research to investigate the S-R repetitions in their analysis to see whether this factor may have confounded our results.

The aspect of S-R repetitions points further to a possible theoretical problem. We referred to a functional relationship between the P3b and the RT that results from memory consolidations processes and excluded the possibility that the developed trace associates the stimulus with its particular response (Hommel, 2004).

One obvious disadvantage of the flanker tasks is the small stimulus set (**à**, **ß**) where trial-to-trial repetitions of stimulus attributes could consequently induce complex priming effects (Bugg, 2008; Trammell Neill & Valdes, 1992). See for example the situation in which the flanker items on an incongruent trial are selectively inhibited (**à à ß à à**). If this previously inhibited stimulus item changes into the target item on the subsequent trial (**à à à à à**), performance could be distorted by

the residual inhibition associated with this item (negative priming). In contrast, when the previous inhibited flanker item remains the irrelevant item on the subsequent trial, performance gets an additive acceleration (positive priming). This complex priming effects might have masked small effects of interests as for the ERP data and its relationship to RT. Different literature has already indicated that the P3 amplitude was modified by negative priming effects (Kathmann, Bogdahn, Endrass, 2006; Gibbons, 2009). An event-related potential study tested this relation in an Eriksen task and showed significant P3 amplitude reductions with negative primes. Importantly, the negative priming effects differed between RT velocities. Comparable to this study, researchers used the median to divide fast and slow responses. Above-median RTs were preceded by stronger negative priming effects on the P300 amplitude compared to below-median RTs (Gibbons, 2009). An important obstacle for future research is to create a task that has a larger stimulus sets and is able to elicit the same sequential effects as the present Eriksen task. Otherwise, we propose future investigations to control for such effects.

As mentioned before we did not have any explanation for the significant interaction effects of the current trial n and the response velocity on the peak amplitude of the preceding trial. It is possible that the mentioned methodological problems were responsible for complex interactions and confounded our results.

We did find a very strong effect of trial compatibility on the P3b amplitude on which ERPs were recorded. The amplitude was significantly higher on compatible trials than on incompatible trials (see Figure 2). This outcome may probably reflect the disruption of stimulus encoding by the presence of conflict (Eriksen & Eriksen, 1974). Hillyard, Squires, Bauer, & Lindsay (1971) highlighted that those stimuli evoking higher P3 amplitudes were better discriminated than stimuli triggering smaller amplitudes. This interpretation can easily be related to the present tasks. Targets in compatible trials are easier encoded and therefore have more time and processing resources to be consolidated. As a consequence, these targets are associated with improved performance. The memory consolidation account affords a good explanation for the present results by referring to greater consolidation effects on trials with less conflict. The presented data was also consistent with the finding of Verleger et al. (2005) that P3 facilitates behavioral responses where higher amplitudes stimulate enhanced performance benefits (or others Nieuwenhuis, Aston-Jones, & Cohen, 2005). The finding is very new compared to previous research. First of all, most of the

evidence came from studies that focused on the P3 latency to investigate its relationship to compatibility effects (Valle-Inclan, 1995). Studies that examined the amplitude on conflict effects obtained either no influence indicating that the P3 amplitude was unaffected by the presence of conflict (West, Jakubek, Wymbs, & Perry Moore, 2005; Duncan-Johnson & Kopell, 1981) or demonstrated completely the opposite relation to our study where low conflict trials (compatible) reduced the P3 amplitude rather than incompatible trials (Kopp, 1996). The relation found in the present study was very significant. We based our analysis on the Pz electrode only because the P3 component was here best manifested. A study searching for neural correlates of conflict processes revealed identical results on the Pz electrode as presented here (West et al., 2005). Nevertheless, the authors summarized their results as non-significant on the P3 amplitude because they did not find a conflict effect on other electrodes (P3 and P4). To our minds, this conclusion seems to be ungrounded because the authors apparently ignored the significant effects on the Pz electrode. Thus it is possible that the disparate findings were artificially induced by the choice of electrodes. Evidences about the relationship of the P3 amplitude and the compatibility effect are sparse so more research is required to deduce generalizable conclusions. Importantly, research between different conflict tasks were needed to control for different perceptual stimulus dimensions, spatial and temporal features. This was confirmed by a combined fMRI and EEG study that found significant spatial and temporal differences in conflict processing when comparing the Flanker and the Simon task (Frühholz, Godde, Finke, & Herrmann, 2010).

The present results can be further applied to an important discussion related to the Go-NoGo task. Several studies indicated that the P3 amplitude was increased on no-go trials where participants are required to inhibit a response compared to go trials. This Go/NoGo effect was differently explained and triggered a debate about the functional role of the P3 component. Smith, Johnstone and Barry (2003), for example, related the increase in the P3b amplitude on NoGo trials to motor and cognitive inhibition. Verleger, Paehge, Kolev, Yordanova, and Jaskowski (2006) on the other hand, elucidated the smaller P3 component on Go trials with response preparation processes. An overlapping motor-related negative potential should be responsible for the reduced P3 amplitude. The absence of motor-related negativity on NoGo trials would consequently result in greater P3 amplitudes. Inhibitory processes can be similarly engaged in high conflict situation where the processing of conflicting stimuli

(flankers) and their associated responses has to be suppressed. The reduced P3b amplitude on incompatible trials therefore contrasts with both accounts because these would expect the opposite, namely increased P3b amplitude on incompatible trials.

The outcomes of this study are new and not easily applicable to other theories like for example the context updating hypotheses. It was intended to reflect that the P3b causes future behavioral outcomes in a conflict task. Therefore, we examined behavioral effects on the basis of the preceding P3 component. This stands in contrast to previous operationalizations where the P3 amplitude was investigated after specific events. It would be interesting to measure the P3 component with sequential modulations in the Eriksen task but this time to keep the focus on the current trial. The context updating theory would predict greater amplitudes on the current trial after alternations because of greater needs to update existing mental representations.

It is important to note that we tried to offer an alternative account against the conflict monitoring hypotheses. Our results could not provide any arguments that question its validity. In a fMRI study of Kerns, Cohen, MacDonald, Cho, Stenger & Carter (2004) it was tested whether control-related structures were responsible for the sequential effects in a Stroop conflict task when controlling for repetition effects. A direct relation was reported between the activity of the anterior cingulate cortex and the prefrontal cortex on preceding, high conflict trials and subsequent behavioral adjustments. Both structures were correlated to conflict-monitoring functions in the past. Probably, we did not find an effect because of the engagement of control processes that are more important in conflict situations ruling out any other theory.

For the past 40 years there has been a debate over the role the P3 might play in specific cognitive processes that was accompanied by constantly contradicting findings (see for a detailed review Kok, 2001). Possibly it is important to change the focus to more general or multiple processes that are engaged to generate the P3 component. In the last few years, there seemed to accumulate evidence that the P3 component cannot be attributed to one specific cognitive process. For example several others associated the P3 component to the locus coeruleus-norepinephrine system (LC_NE) that has been linked to the adaptive gain hypotheses which consist of two modes of activity (Aston-Jones, & Cohen, 2005; Nieuwenhuis, Geus, & Aston-Jones, 2010). The first one, phasic activation is stimulated by the result of task-related decision processes and therefore plays an important role for the optimization of performance (exploitation). In situations where the significance of the tasks decreases

the second mode, tonic activation, triggers processes for the detachment from the task and promotes exploration behaviors. The interaction of LC and NE activity adjusts the gain of processing and therefore optimizes behavior to external demands. The P3 component has been specifically related to the phasic enhancement of gain in the cortex and has been proposed to manifest neuromodulatory functions. The evidence of the relationship of the P3 and the LC-NE systems comes from temporal and spatial correlations. The LC-NE system is therefore the first account that permits an explanation for the widely distributed spatial generators of the P3 component and reconciles the wide range of seemingly disparate findings.

Conclusion

We could not reflect that the P3 component directs our behavior in situations of sequential modulations in the Eriksen task due to memory consolidation processes. But the P3b amplitude appeared to have consequences on performance within a trial. We related greater memory consolidation processes to the performance benefits in conditions of less conflict within a trial. Therefore, the P3b component guides our behavior in situations where conflicts affect processing within a trial.

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