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RESEARCH ARTICLE

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Central processing overlap modulates P3 latency

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Abstract Two experiments examined the issue of the functional mechanisms exerting a modulatory effect on the latency of the P3. In experiment 1, using a psychological refractory period (PRP) paradigm, two sequential stimuli (T_1 and T_2) were presented in each trial at varying stimulus onset asynchronies (SOAs), each requiring a speeded choice response. Substantial lengthening of the reaction time to T_2 was observed as SOA decreased (i.e., PRP effect). A systematic investigation of the T_2-locked P3 component amplitude and latency was undertaken to discover whether either of these P3 parameters was correlated with the PRP effect. The results showed lengthening of the T_2-locked P3 component latency as SOA was decreased, and, across subjects, a positive correlation between the PRP effect and P3 latency lengthening. No SOA-dependent P3 amplitude variation was observed. In experiment 2, the P3 component was measured under single-task conditions. P3 amplitude was higher under single-task than under dual-task conditions, but no SOA-dependent latency variations were observed in this experiment. Overall, the results of both experiments support the notion that part of the processing reflected in P3 activity occurs at or after the locus of the PRP effect, thus suggesting strongly that central mechanisms are involved in P3 latency variations.

Keywords Central processing · P3 · PRP paradigm

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Introduction

In the psychological refractory period (PRP) paradigm, two target stimuli, T_1 and T_2, are presented sequentially, and separate speeded forced-choice responses, with associated response times RT_1 and RT_2, are to be produced. The stimulus onset asynchrony (SOA) between T_1 and T_2 in most experiments ranges between 0 and 1 s. The usual outcome under these conditions is a progressive lengthening of RT_2 as the SOA is reduced. This SOA-dependent RT_2 lengthening has been termed the PRP effect (Welford 1952; see Pashler 1994 for a comprehensive review of studies using the PRP paradigm). Several researchers have proposed that the PRP effect reflects a forced seriality of central processing for certain mental operations, such as response selection (but see Meyer and Kieras 1997). According to this view (e.g., McCann and Johnston 1992; Pashler and Johnston 1989), under task overlap conditions (i.e., at short SOA), response selection in $task_2$ is postponed until central mechanisms are no longer occupied with response selection in $task_1$. The postponement of response selection in $task_2$ would explain the prolongation of RT_2 at a short SOA compared with a long SOA.

So far, relatively little work has made use of electrophysiological indices of cognitive processing to improve the understanding of the mechanisms that produce dual-task interference in the PRP paradigm. Osman and Moore (1993), using the lateralized readiness potential (LRP), have shown that the PRP effect is correlated with a delay of T_2 processing occurring before the generation of T_2-locked LRP activity. Furthermore, these authors have shown that T_1-locked LRP activity ceases to interfere with LRP activity related to T_2 processing before the emission of a response to T_1. These results help constrain the locus of the PRP effect in two ways. Firstly, the results rule out response execution in $task_1$ as the locus of the PRP effect. Secondly, by showing that the LRP in $task_2$ is postponed, the results suggest that the PRP locus is at or before response

selection. The results obtained by Sommer et al. (2001) using a PRP paradigm and focusing on the LRP component point to the same conclusion.

Two other electrophysiological studies have examined the relationship between ERP activity and PRP effect, though from a slightly different perspective. By relying on assumptions concerning the central locus of the PRP effect, these two studies produced results that were informative about the organization of the mental processes contributing to the generation of an ERP component known as P3. The P3 is one of the most studied ERP components in the field of cognitive electrophysiology (see Donchin 1981; Johnson 1986, for reviews). Studies on the P3 have focused on both its basic parameters, i.e., latency and amplitude (see reviews by Verleger 1997; Kok 2001, respectively). Although there is some disagreement (e.g., Verleger 1988), the P3 is often taken as a measure of "context updating," or of encoding into short-term memory. The two studies reviewed below have both capitalized on the well-established empirical observation that the P3 component is sensitive to the relative frequency of the category assigned to stimuli in categorization and discrimination tasks (Donchin and Coles 1988). Interestingly, P3 activity has been hypothesized to arise prior to response selection, because stimulus–response compatibility, which can be shown to influence RT, has relatively little impact on P3 latency and amplitude (Magliero et al. 1984). The P3 component, therefore, is often taken as a measure of the time required to complete stimulus encoding and classification, which are assumed to take place prior to response selection.

Luck (1998, experiment 1) provided a test of these suppositions in the context of the PRP paradigm. T_1 was a square box varying in color (either red or green with equal probability) and T_2 was a letter. The SOA between T_1 and T_2 was either 50, 150, or 350 ms. For half of the subjects, T_2 was the letter "X" in 75% of trials and the letter "O" in 25% of trials, and these relative frequencies were reversed for the other half of the subjects. Subjects were instructed to make a speeded response to T_1 based on the color of the square, and a speeded response to T_2 based on letter identity. To isolate the T_2-locked P3 component, and segregate it from ERP activity generated by T_1 presentation, Luck (1998) computed T_2-locked ERP difference waves by subtracting, for each SOA, the ERP response to frequent stimuli from the ERP response to infrequent stimuli, with this component referred to as frequency-related P3 difference wave. RT_2 showed the expected PRP effect, namely, an increase in RT_2 as SOA was reduced, with a difference of 220 ms between RT_2 at the shortest SOA and RT_2 at the longest SOA. In the analysis of the electrophysiological results, Luck focused on the latency of the frequency-related P3 difference wave, as a function of SOA. Here, the effect of SOA was much smaller, with a difference of 51 ms between the latency of the P3 difference wave at the shortest SOA relative to the latency at the longest SOA. Interestingly, the amplitude of the frequency-related P3 difference wave also differed as a function of SOA, with significantly smaller P3 amplitudes recorded at the short SOA than at the long SOA. Furthermore, as is often found, there was also a significant frequency-related P2 difference component. In contrast with what was found for the P3, neither the amplitude nor the latency of the P2 component was affected by SOA. Based on this constellation of results, Luck (1998) concluded that stimulus identification and categorization likely take place with negligible dual-task interference, as proposed by bottleneck theories of the PRP effect (Pashler 1994). Luck also argued that the effects observed on P3, particularly the amplitude effects, likely occurred at stages of processing earlier than the PRP locus. Based on the large discrepancy between the large size of the SOA effect on RT and the small size of the SOA effect on the latency of the frequency-related P3 difference waves, Luck argued that the main locus of the PRP effect had to be after stimulus perception and categorization.

One step further in the analysis of the relation between P3 and PRP effects has been taken by Arnell et al. (2004), who presented subjects with the digit 2 or 3 in T_1, followed at SOAs of 100, 200, or 750 ms, by a spoken word varying in pitch in T_2. Stimulus T_2 was presented at a low pitch in 80% of trials and at a high pitch in the remaining 20% of trials. Subjects were instructed to make a speeded response to T_1 based on the digit identity, and a speeded response to T_2 based on pitch. ERP responses to T_2 were generated by subtracting, for each SOA, the ERP response to low-pitch T_2s from the ERP response to high-pitch T_2s. The behavioral results of this study showed a 278 ms PRP effect. From the longest to the shortest SOA, the P3 latency postponement amounted to a significant 69 ms, accompanied by a modest, but significant, decrease in P3 amplitude as SOA was decreased. Arnell et al. (2004) further analyzed their results by looking at a possible correlation, across subjects, between the amount of PRP slowing and the P3 latency shift across SOAs, based on the argument that a positive correlation is an expected pattern on the hypothesis of central processing postponement as the common cause of PRP effect and SOA effects on P3 latency. However, no correlation was found across subjects between the amount of PRP effect and the amount of SOA effect on P3 latency. This finding was taken by Arnell et al. (2004) as evidence for the independence of the sources of these effects.

In the present work, we also studied how SOA affects the frequency-related P3 difference component in a PRP paradigm. Our motivation, in part, stemmed from the observation that both of the studies briefly reviewed in the foregoing paragraphs reported significant delays in the P3 response, as well as significant attenuation of P3 amplitude at short SOA compared with long SOA. There are in addition at least three distinct sets of findings that converge to support the hypothesis that these effects on P3 are evidence for an effect of PRP interference on the identification and/or classification of

T_2: namely, the processing stages held to be responsible for the generation of P3 activity. First, using fMRI and a manipulation of response selection difficulty and perceptual analysis difficulty in distinct tasks, Jiang and Kanwisher (2003a) have recently shown a substantial overlap of brain regions involved in response selection and perceptual discrimination. Second, using ERPs, Dell'Acqua et al. (2003) have shown that a sizable modulation (i.e., amplitude reduction) of a P3 component time-locked to a masked visual T_2 requiring identification can be obtained under conditions in which T_1 is an auditory stimulus and $task_1$ is a speeded forced-choice response, that is, a first task often employed in PRP designs. Third, using a paradigm in which a to-be-identified visual T_1 preceded an auditory T_2 requiring a speeded forced-choice response, Jolicoeur and Dell'Acqua (1998) have found a PRP-like RT_2 lengthening that suggests that one or more stages of processing required for the perceptual identification task conflicted with response selection in the speeded auditory choice task. For these reasons, we believe that a reexamination of the significant SOA effects on the P3 parameters in a PRP context, and of the correlation of such SOA effects with the behavioral PRP effect (i.e., the RT_2 lengthening as SOA is decreased) was warranted. Specifically, our prediction was that, if central mechanisms were implicated in the generation of P3 activity, then a significant postponement of T_2-locked P3 latency should be observed under dual-task conditions in which central processing in $task_2$ was momentarily bottlenecked by ongoing processing occurring in $task_1$ (i.e., at short SOA).

Experiment 1

Method

Subjects, stimuli and apparatus

Thirty observers with a mean age of 26 years, all with normal or corrected-to-normal vision, volunteered to participate. Two stimuli were displayed in sequence on each trial. The first stimulus, T_1, was a square box colored in yellow or blue. The second stimulus, T_2, was a white digit (1, 2, 3, 4, or 8). The stimuli were displayed on a uniformly black background, at the center of 17″ CRT monitor controlled by a 686 Pentium CPU. At a viewing distance of 60 cm, the side of the square measured 3.8°, and all digits could be inscribed in an area of 0.95° x 1.4° (width x height).

Procedure

The experiment was conducted in a soundproof, electrically shielded, and moderately lit room. Each trial began with a 2.5 s presentation of two horizontally arrayed plus signs (+ +) at the center of the screen. Each plus sign subtended 0.28°. After 2.5 s, the plus signs expanded to 0.4° for 700 ms in order to warn the subjects about the imminent presentation of T_1. After a 700-ms blank interval, T_1 was exposed for 50 ms, followed at SOAs of either 100, 350, or 800 ms by the presentation of T_2 for 50 ms. The instructions given to subjects stressed the importance of producing a single response to each stimulus in the same order in which the stimuli were displayed. Subjects were instructed to make a first speeded response to T_1, and a second speeded response to T_2, as quickly as possible while keeping errors to a minimum. Subjects used the index and middle fingers of one hand to press one of two adjacent buttons (e.g., the z and x keys of the keyboard of the computer) to indicate the color of T_1, and the index and middle fingers of the other hand to press one button (e.g., "n") if T_2 was the digit 1, 2, 3, or 4, or a different button (e.g., "m") if T_2 was the digit 8.[1] These stimulus–response mappings were counterbalanced across subjects. After the second response, an interval of 1 s elapsed before the presentation of the fixation points for the next trial. The fixation points served as feedback on response accuracy, with the left plus sign becoming a minus sign ("−") in case of an incorrect response to T_1, and the right plus sign becoming a minus sign in case of an incorrect response to T_2. The experiment was organized in 10 blocks of 60 experimental trials, preceded by one block of 30 practice trials. Within each block, each combination of T_1 color and T_2 digit was equiprobable, and the order of possible combinations randomized. The experiment took about 90 min.

EEG/ERP settings

Using an Electrocap International head cap, the electroencephalographic (EEG) activity was recorded from the sites Fz, Cz, Pz (10/20 System; Jasper 1958), referenced to the left mastoid. Vertical eye movements (EOG) were recorded bipolarly from two electrodes, one below and one above the left eye. Horizontal EOG was monopolarly recorded from one electrode placed on the left lateral canthii. The EEG and the EOG were amplified with a bandpass filter of 0.05–40 Hz, at a sampling rate of 250 Hz. Impedance at each electrode site was maintained below 5 kΩ . The EEG was algebraically re-referenced offline to the average of the left and right mastoids, and segmented into 1,000-ms epochs that began 200 ms prior to T_2 onset. A baseline correction was applied to the recording at each recording site, for each epoch, using the mean activity during the 200 ms pre-T_2

[1]The P3 component is sensitive to the task-defined stimulus probability, and not to the absolute probability of occurrence of one stimulus included in a set of n possible stimuli, when $n > 2$ (e.g., Donchin and Coles 1988). This is shown by the fact that large P3 responses can be generated by stimuli whose absolute frequency of occurrence is higher than any other stimulus included in a stimulus set, provided the response to that particular stimulus is emitted less frequently than the response associated with other stimuli (e.g., Dell'Acqua et al. 2003; Vogel and Luck 2002)

interval. To eliminate ocular artifacts, epochs in which the EOG activity exceeded ±50 μV were eliminated at this stage (9% of the epochs). Only T_2-locked epochs associated with (a) correct responses to both T_1 and T_2 on a given trial, and (b) a response time to T_2 shorter than 2,000 ms contributed to the creation of the ERPs at each recording site. Separate grand-average waveforms for each SOA condition and for each T_2 category were generated. To isolate ERP activity strictly associated with the manipulation of the T_2 stimulus category, and uncontaminated by ERPs associated with the processing of T_1, difference ERP waveforms in each SOA condition were estimated by subtracting the ERP waveform elicited by the frequent T_2 stimulus category (i.e., the digits 1–4) from the ERP waveform elicited by the infrequent T_2 stimulus category (i.e., the digit 8). The amplitude of the P2 and P3 components of the subtracted ERPs was quantified by computing the mean amplitude in time windows of 150–300 ms for the P2 component and 300–700 ms for the P3 component. The latency of the P2 and P3 components was estimated using both a standard algorithm for the detection of the peaks (i.e., local maxima) in the same temporal window used for the component amplitude estimation, and a fractional latency analysis to estimate the point in time at which 25% of each component amplitude was achieved. The data from five subjects were not included in the behavioral and ERP analyses because of an excessive number (greater than 70% in at least two cells of the present design) of ocular artifacts. All retained subjects reported a percentage of epochs not affected by artifacts of 81% or greater.

Results

Behavior

The mean reaction times (RTs) for trials with correct responses to T_1 and T_2 are shown in Fig. 1, as a function of SOA and the relative frequency of T_2. RT_1s longer than 1,500 ms and RT_2s longer than 2,000 ms were excluded from all analyses. These exclusion criteria rejected 1.2% of the correct RT_1s and 2.1% of the correct RT_2s. The means were submitted to an analysis of variance (ANOVA) that considered SOA and T_2 frequency as within-subject factors. RT_1 was 13 ms longer when the category of T_2 was infrequent relative to when it was frequent, $F_{(1,24)} = 4.4$, $MS_e = 1{,}683$, $P < 0.06$, showing an effect of the task$_2$ variable on RT_1. This effect did not vary significantly with SOA, however, $F_{(2,44)} = 2.3$, $MS_e = 553$, $P > 0.13$. No other significant effect was detected in the analysis of RT_1. As can be seen in Fig. 1, RT_2 increased as SOA was reduced, $F_{(2,44)} = 200.4$, $MS_e = 2{,}870$, $P < 0.001$. RT_2 was also longer for the infrequent T_2 category than for the frequent T_2 category, $F_{(1,22)} = 104.0$, $MS_e = 2{,}086$, $P < 0.001$. Furthermore, the effect of relative frequency was larger at longer SOAs (89 ms at 350-ms SOA; 86 ms at 800-ms SOA) than at the shortest SOA (59 ms), which produced a significant interaction between these two variables,

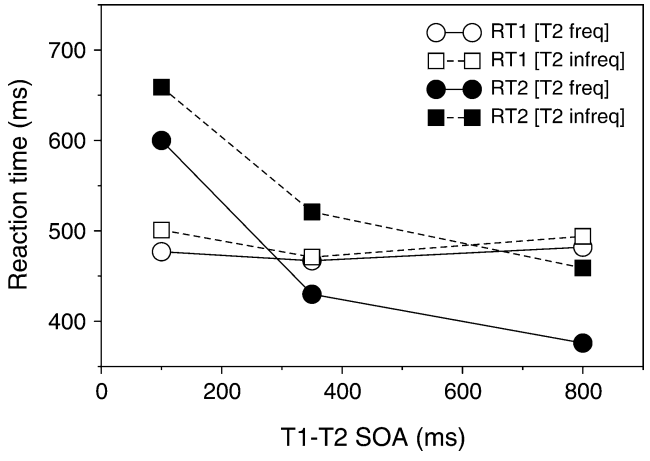

Fig. 1 Experiment 1. Mean RT_1 and mean RT_2 plotted as a function of SOA and as a function of T_2 stimulus category (frequent $T_2 = 1$–4; infrequent $T_2 = 8$)

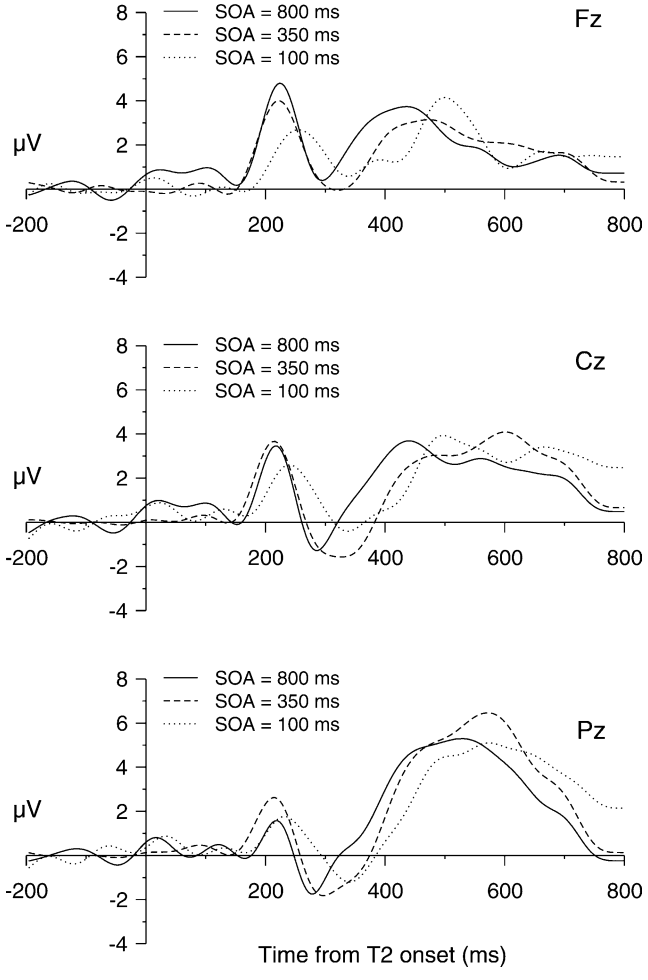

Fig. 2 Experiment 1. T_2-locked grand-average waveforms generated by subtracting ERPs to the frequent T_2 stimulus category ($T_2 = 1$–4) from ERPs to the infrequent T_2 stimulus category ($T_2 = 8$). ERPs are reported for each SOA and for each recording site

$F_{(2,44)} = 3.8$, $MS_e = 874$, $P < 0.03$. The mean proportion of correct responses to T_1 and T_2 was 0.95 and 0.92, respectively. No significant effects of SOA and T_2 category were detected in an ANOVA performed on the mean proportion of correct responses to T_1 and on the mean proportion of correct responses to T_2.

ERPs

Subtracted T_2-locked grand-average waveforms at each recording site are shown in Fig. 2 as a function of SOA. P2 and P3 amplitude and latency values were submitted to ANOVA in which SOA and recording site were considered within-subject factors. A Greenhouse–Geisser correction for nonsphericity was used in each of the tests reported for the present experiment and the subsequent experiment.

P2 The mean amplitude of the P2 difference wave (150–300 ms window) was largest at Fz, intermediate at Cz, and smallest at Pz, $F_{(2,48)} = 12.5$, $MS_e = 2.191$, $P < 0.001$. The amplitude of the P2 was also smaller for the 100-ms condition than for the other two conditions, $F_{(2,48)} = 3.3$, $MS_e = 7.44$, $P < 0.05$. In a separate analysis in which P2 amplitude values at the SOA of 100 ms were temporarily excluded, no significant SOA effects on P2 amplitude were detected ($F < 1$). An ANOVA carried out on the latency to the P2 peak indicated a significant effect of SOA, $F_{(2,48)} = 18.8$, $MS_e = 2{,}232$, $P < 0.001$. The P2 peak latency was lengthened from 211 ms to 240 ms as SOA decreased. An ANOVA carried out on P2 fractional area latency revealed a significant effect of SOA, $F_{(2,48)} = 7.5$, $MS_e = 1{,}140$, $P < 0.002$, with latency increasing from 189 ms to 222 ms as SOA decreased.

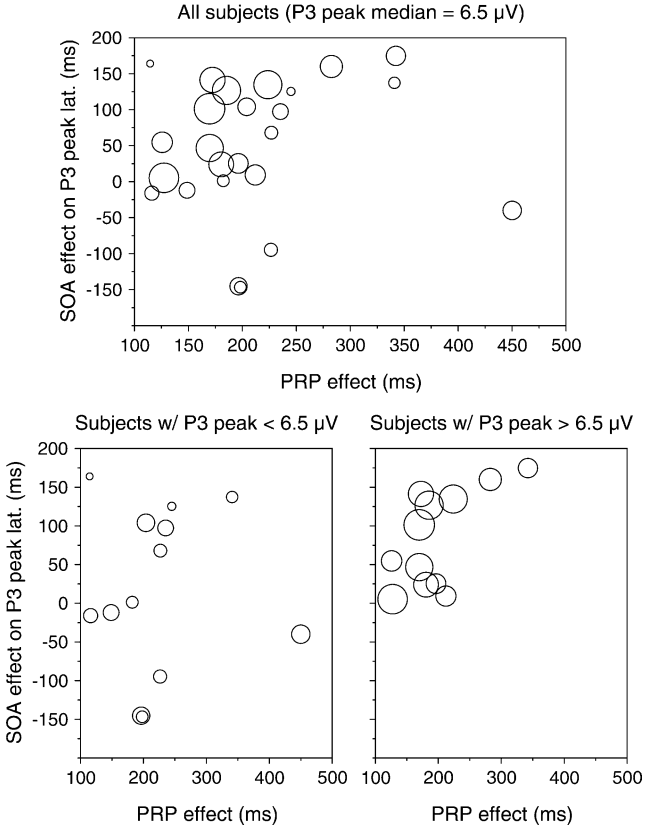

Fig. 3 Experiment 1. Individual estimates of PRP effect plotted against the relative SOA-dependent P3 peak shift. *Upper panel* Scores from all subjects. *Lower panels* Scores partitioned following the application of the median-split applied to the subjects' P3 peak magnitude

P3 The mean P3 amplitude (300–700 ms window) was largest at Pz, intermediate at Cz, and smallest at Fz, $F_{(2,48)} = 4.9$, $MS_e = 5.461$, $P < 0.02$. There was no effect of SOA on P3 amplitude, $F < 1$. An ANOVA carried out on P3 peak latency indicated a significant effect of SOA, $F_{(2,48)} = 3.9$, $MS_e = 13{,}876$, $P < 0.02$. The latency of the P3 peak was lengthened from 470 ms to 526 ms as SOA decreased. Although an SOA-dependent peak shift was maximally apparent at Fz, no significant interaction between SOA and recording site was detected in the P3 peak latency analysis ($F < 1$). An ANOVA carried out on P3 fractional area latency revealed a significant effect of SOA, $F_{(2,48)} = 7.9$, $MS_e = 11{,}642$, $P < 0.001$, with latency increasing from 423 ms to 504 ms as SOA decreased. There was also a significant effect of recording site in this latter analysis, $F_{(2,48)} = 7.8$, $MS_e = 5{,}236$, $P < 0.002$, indicating an earlier P3 at Fz (441 ms) compared with Cz and Pz (480 ms and 484 ms, respectively).

Behavior/ERP correlation

The scattergram including each subject's PRP effect (RT_2 [SOA = 100 ms]−RT_2 [SOA = 800 ms]) plotted against the relative net SOA effects on P3 peak latency (P3 peak latency [SOA = 100 ms]−P3 peak latency [SOA = 800 ms]) is reported in the upper panel of Fig. 3. In the figure, the diameter of the plotting symbol is proportional to the P3 peak amplitude observed in the subtracted ERP waveforms collapsed across recording sites.[2] The correlation between PRP effect and SOA effects on P3 peak latency was $r = 0.11$, and not significant, $t < 1$.

A closer inspection of Fig. 3, however, revealed an unexpected phenomenon. A substantial number of subjects showed very small frequency-related P3 amplitudes, and this is reflected by the large number of small plotting symbols in the top panel of Fig. 3. These subjects tended to show comparable P3 responses to both frequent and infrequent T_2 stimulus categories, resulting in attenuated frequency-related P3 responses in the subtracted ERP waveforms. Other subjects instead showed the expected pattern—namely, P3 responses generally magnified to the infrequent T_2 category compared with P3 responses to the frequent T_2 stimulus category. The principled way adopted in the present context to examine separately these two

[2]Analogous correlation analyses were performed using P3 latency estimates derived from the fractional area latency analysis, with equivalent results.

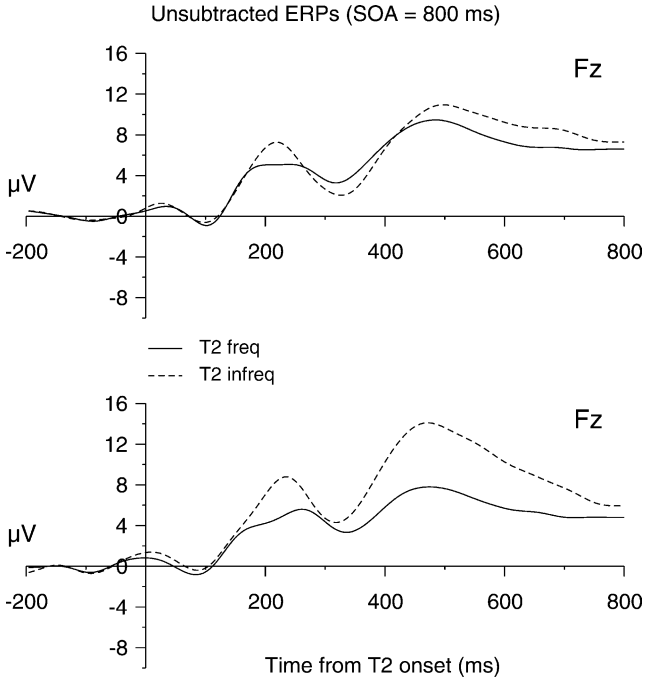

Fig. 4 Experiment 1. Unsubtracted grand-average waveforms under the SOA = 800 ms condition generated after the application of the median-split applied to the subjects' P3 peak magnitude. *Upper panel* Results from subjects with P3 amplitudes that were smaller than the median. *Lower panel* Results from subjects with P3 amplitudes greater than the median

groups of subjects was through a median-split performed on the P3 peak values. The P3 peak values of the ERPs at each recording sites were sorted, and the median value was estimated (6.5 μV). The two groups were generated by including in one group subjects ($n = 13$) producing a frequency-related P3 peak value smaller than the median value, and in the other group subjects ($n = 12$) producing a frequency-related P3 peak value larger than the median value. The distribution of subjects in the large P3 vs small P3 groups was the same at all recording sites. The unsubtracted grand-average waveforms at Fz for the two groups of subjects are separately reported in Fig. 4. The effect shown in Fig. 4 was also evident at the other recording sites.

The PRP effect plotted against SOA effects on P3 peak latency are separately shown for the two groups of subjects in the lower panels of Fig. 3. For the subjects with small P3 amplitudes, the correlation was close-to-nil, and not significant ($r = 0.01$, $t < 1$). For the subjects with larger P3 peak values, the correlation was $r = 0.65$, which was statistically significant, $t(10) = 2.7$, $P < 0.03$. For this latter group, effects of SOA on P3 latency explained a significant proportion of the variance (0.42) in the SOA effect on mean RT_2. The subtracted ERP waveforms, at Fz, for each SOA, are displayed in Fig. 5 for the two groups of subjects. The upper panel, displaying the waveforms for the subjects with small frequency-related P3 amplitudes, makes it clear that estimating P3 latency was difficult for these subjects, and this could explain in part the poor correlation described above when the entire set of values was considered.

Separate analyses focusing on P2 and P3 amplitude and latency were carried out on these two groups of subjects. An ANOVA considering the data from the subjects with P3 peaks smaller than the median revealed a non-significant 40-ms P3 peak shift across SOAs, $F_{(2,24)} = 1.7$, $MS_e = 16{,}501$, $P > 0.2$, and no SOA effect on P3 amplitude, $F < 1$. As in Fig. 5, a P3 response in these subjects was in fact nonexistent in the subtracted ERP time-locked to T_2. There was however a tendency of these subjects to show a significant P2 response, whose latency tended to increase as SOA was decreased, $F_{(2,24)} = 2.9$, $MS_e = 3{,}189$, $P < 0.07$. An ANOVA considering the data from the subjects with P3 peaks larger than the median revealed a significant P3 peak latency increase, $F_{(2,22)} = 10.7$, $MS_e = 6{,}698$, $P < 0.001$, amounting, from the longest SOA to the shortest SOA, to 96 ms. No significant P3 amplitude variations were observed in these subjects. A significant reduction of P2 amplitude was also found, $F_{(2,22)} = 3.7$, $MS_e = 7.8$, $P < 0.05$, reflecting a smaller P2 amplitude at SOA = 100 ms conditions compared with the other two SOA conditions. When the data from the SOA = 100 ms condition were temporarily excluded from consideration, the SOA effect was no longer significant, $F < 1$.

In Fig. 6, the RT_2 results of the two groups of subjects are reported separately as a function of SOA and T_2 relative frequency. An ANOVA considering group as an additional between-subject factor confirmed what is visible to the eye in Fig. 6, namely that the smaller

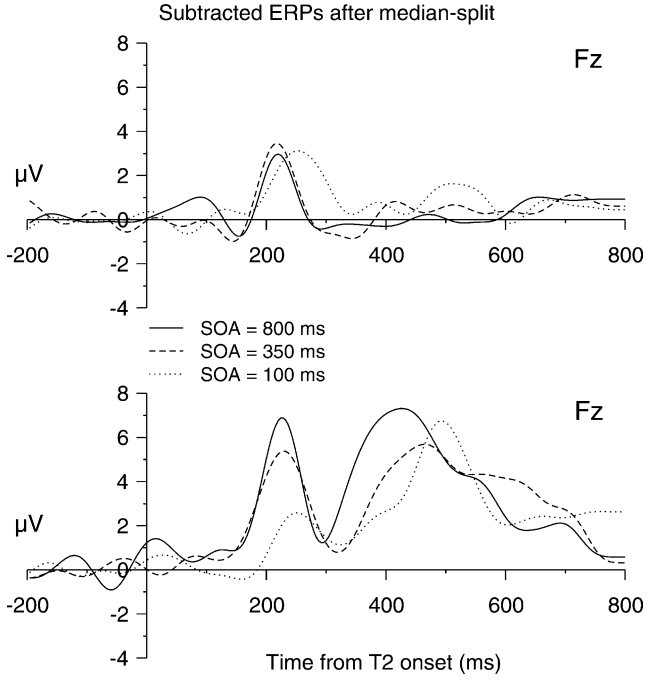

Fig. 5 Experiment 1. Subtracted T_2-locked grand-average waveforms for each SOA for subjects with P3 amplitudes smaller than the median (*upper panel*) and for subjects with P3 amplitudes greater than the median (*lower panel*)

frequency-related P3 group had a larger effect of frequency in their RT_2s than the larger frequency-related P3 group, $F_{(1,22)} = 8.2$, $MS_e = 2{,}086$, $P < 0.01$. No other difference modulated by the group factor was significant ($F < 1$) in this analysis, and in an analysis performed on RT_1.

Discussion

As expected, RT_2 increased as SOA was decreased. Moreover, RT_2 was longer for the infrequent T_2 stimulus category than for the frequent T_2 stimulus category. These results replicated the well-studied PRP phenomenon (Pashler 1994), and the well-known relative frequency effect (e.g., Miller and Pachella 1973). The electrophysiological results showed that SOA affected both of the frequency-related ERP components that emerged from the analyses of the subtracted T_2-locked ERP response, namely, the P2 and the P3 components. As SOA was decreased, P2 amplitude decreased and the P2 latency was lengthened. P3 amplitude remained stable across SOAs, but P3 latency was lengthened at the shortest SOA compared with the longest SOA. The PRP effect and the SOA effect on P3 peak latency were significantly correlated when a group of subjects characterized by clearly well distinguishable P3 components was isolated in the sample of tested subjects. Importantly, the largest P3 amplitude at Pz found in experiment 1 is consistent with the view that the component under study in the present context could be better characterized as a $P3_b$ component. A $P3_a$ component, for instance, would have had its largest amplitude at Fz (e.g., Knight 1991). Further aspects of the results of experiment 1 will be discussed in the "General discussion."

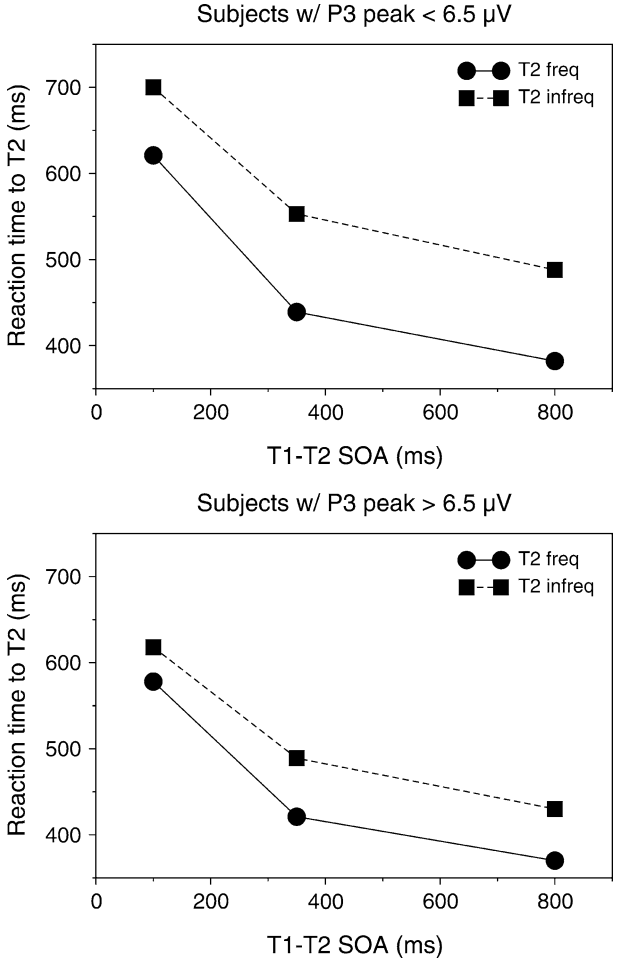

Fig. 6 Experiment 1. Mean RT_2 plotted as a function of SOA and T_2 stimulus category, for subjects with a P3 amplitude smaller than the median (*upper panel*) and for subjects with a P3 amplitude greater than the median (*lower panel*)

Experiment 2

One aspect of the results of experiment 1 was surprising: a large proportion of the subjects did not appear to have a clear frequency-related P3 response. We hypothesized that embedding the classic oddball paradigm embodied in $task_2$ of experiment 1 in a broader dual-task context may have affected the frequency-related P3. There are several ways in which such a context effect could have been produced. For example, the dual-task paradigm used in experiment 1 required holding two different task sets (one for $task_1$, and one for $task_2$) in working memory, which may have increased the working memory load for subjects in experiment 1 (Bourke et al. 1996), or decreased the "automaticity" with which event frequency is normally processed (e.g., Hasher and Zacks 1984). This increased load may have reduced subjects' sensitivity to relative frequency differences between the response categories in $task_2$, leading to an attenuated P3.

Another possibility is that the central processing demands of $task_1$ may have had a direct effect on the P3 in $task_2$ (as suggested by the significant P3 latency differences, as a function of SOA, in experiment 1; and the significant latency and amplitude effects observed by Luck 1998; Arnell et al., 2004). This effect may have been indirect, perhaps by making it more difficult for subjects to keep track of the relative frequency of the response categories in $task_2$. Such ongoing monitoring of relative frequency is presumably crucial in the cause of the frequency-related P3 response (Fabiani et al. 1986). Perhaps the dual-task load effect may have had a more direct impact, by acting to interfere with or delay (or both) the processes that generate the P3. However, the present results would not allow us to tease these two alternatives apart.

Nonetheless, it is straightforward to test the hypothesis that the dual-task context of experiment 1 had an effect on the frequency-related P3 response. This test was carried out in experiment 2 by using exactly the same stimuli as in experiment 1, but asking subjects to ignore the T_1 stimuli and to perform only $task_2$. That is, the task that was used as $task_2$ in experiment 1, was now carried out under single-task conditions. It is important to emphasize two aspects of experiment 2 that make the

results directly comparable to those of experiment 1. Firstly, that the task used in experiment 2 was identical in all ways to that in experiment 1 (number of stimuli, response categories, relative frequencies, and response buttons). Secondly, the physical stimuli in both experiments were identical; consequently, any differences in the frequency-related P3 response across the experiments could not be due to differences in physical stimulation across the two experiments.

Finally, experiment 2 was also important to verify that the effects of SOA on P2 and P3 observed in experiment 1 were not due to physical interactions between the two stimuli. Recall that T_1 and T_2 were both visual stimuli (as in the experiments of Luck 1998). T_2 was a digit and T_1 was a square that surrounded T_2, and they were sometimes presented in close temporal contiguity. It is possible, therefore, that some, or all, of the SOA effects observed in experiment 1 could be due to masking of T_2 by T_1. Alternatively, the effects could reflect processing limitations in visual processing (post masking), rather than central processing limitations usually associated with the PRP effect (Pashler, 1989). It has already been shown that the latency of the P3 can be significantly affected by masking degradation of visual input (e.g., Duncan-Johnson 1981; McCarthy and Donchin 1981). The results of experiment 2 are critical in this respect. If the P3 latency shifts in experiment 1 were the result of masking, or more generally of a processing capacity limitation in the visual system, then equivalent SOA-dependent P3 latency shifts should be evident in experiment 2 because T_1 and T_2 were presented under conditions that were identical to those of experiment 1. If instead the P3 latency shifts were modulated by central processing requirements associated with $task_1$ (in experiment 1), then SOA-dependent P3 latency shifts, if any, should be strongly attenuated in experiment 2.

Method

Subjects, stimuli, and apparatus

Thirteen observers with a mean age of 27 years, all with normal or corrected-to-normal vision, volunteered to participate. Stimuli and apparatus were the same as those described in experiment 1.

Procedure

The sequence of events on each trial of the present experiment was identical to that described in experiment 1, with the exception that subjects in the present experiment were instructed to ignore T_1 and only produce a response to T_2 in the same way subjects did in experiment 1 (i.e., using two fingers of one hand, press one button for $T_2 = 1$–4, or another button for $T_2 = 8$). The experiment was organized in 10 blocks of 30 experimental trials, preceded by one block of 30 practice trials. Within each block, each combination of T_1 color and T_2 digit was equiprobable, and the order of possible combinations randomized. The experiment took about 50 min.

ERP settings

The EEG recording settings were the same as those used in experiment 1. The data from one subject were not taken into consideration in the following analyses because 91% of the epochs were affected by ocular movement artifacts. No more than 18% of trials were rejected in the remaining subjects.

Results

Behavior

The mean RT for trials with correct responses to T_2 is shown in Fig. 7, as a function of SOA and the relative frequency of T_2. RTs longer than 2,000 ms were excluded from all analyses. These exclusion criteria rejected 0.8% of the correct RTs. RTs were submitted to ANOVA that considered SOA and T_2 frequency as within-subject factors. RT was 47 ms longer when the category of T_2 was infrequent relative to when it was frequent, $F_{(1,11)} = 41.2$, $MS_e = 970$, $P < 0.001$. There was also an overall RT increase of 29 ms as SOA decreased, $F_{(2,22)} = 18.5$, $MS_e = 294$, $P < 0.001$. The interaction between T_2 frequency and SOA was not significant ($F < 1$). The analysis on the proportion of correct responses to T_2 indicated that subjects were less accurate when responding to an infrequent T_2 compared with a frequent T_2, $F_{(1,11)} = 10.1$, $MS_e = 0.005$, $P < 0.007$. No other factor or interaction emerged as significant in this analysis.

ERPs

P2 Subtracted T_2-locked grand-average waveforms at each recording site are shown in Fig. 8. The mean

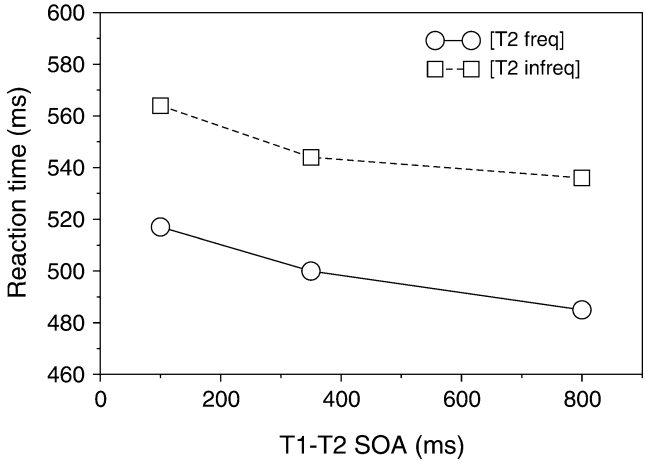

Fig. 7 Experiment 2. Mean RT plotted as a function of SOA and as a function of T_2 stimulus category (frequent $T_2 = 1$–4; infrequent $T_2 = 8$)

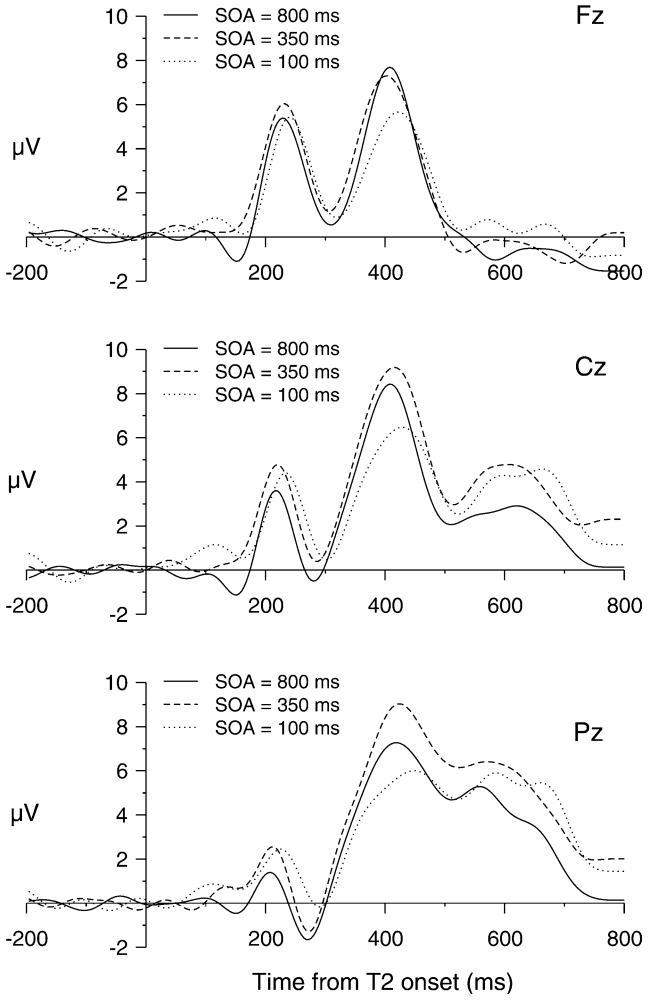

Fig. 8 Experiment 2. T_2-locked grand-average waveforms generated by subtracting ERPs to the frequent T_2 stimulus category ($T_2 = 1$–4) from ERPs to the infrequent T_2 stimulus category ($T_2 = 8$). ERPs are reported for each SOA and for each recording site. Positive voltage is plotted upward. Note the different *y*-scale compared to that of the graphs in Fig. 2.

amplitude of the P2 difference wave (150–300 ms window) was largest at Fz, intermediate at Cz, and smallest at Pz, $F_{(2,22)} = 7.3$, $MS_e = 4.03$, $P < 0.004$. The P2 amplitude did not vary as a function of SOA, $F_{(2,22)} = 1.4$, $MS_e = 7.3$, $P > 0.24$. No interaction between SOA and recording site was detected in this analysis, $F_{(4,44)} = 1.2$, $MS_e = 1.1$, $P > 0.30$. An ANOVA carried out on the latency of the P2 peak indicated no effect of SOA or recording site, all $F < 1$. No significant effects of SOA or recording site emerged from an ANOVA carried out on P2 fractional area latency, all $F < 1$.

P3 The mean P3 amplitude (300–700 ms window) was largest at Pz, intermediate at Cz, and smallest at Fz, $F_{(2,22)} = 14.7$, $MS_e = 7.2$, $P < 0.001$. There was no effect of SOA on P3 amplitude, $F < 1$. No significant effects emerged from an ANOVA carried out on P3 peak latency, all F values were < 1. An effect of SOA was, however, evident in an ANOVA carried out on P3

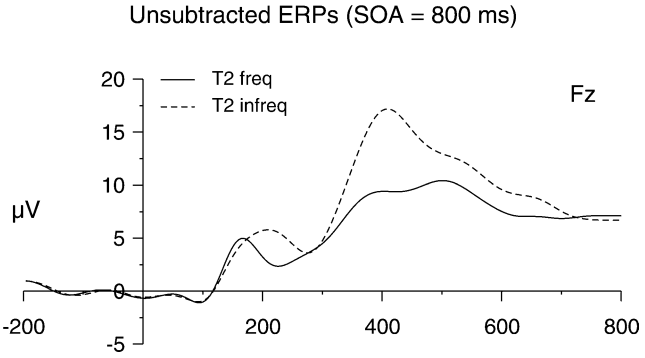

Fig. 9 Experiment 2. Unsubtracted grand-average waveforms under the SOA = 800 ms condition.

amplitude focusing on the 300–500 ms window, $F_{(2,22)} = 14.7$, $MS_e = 7.2$, $P < 0.001$, indicating a moderate reduction of P3 amplitude at SOA = 100 ms. Indeed, when the data from the SOA = 100 ms condition were temporarily excluded from consideration, the SOA effect was no longer significant, $F < 1$ in this latter P3 amplitude analysis. An ANOVA carried out on P3 fractional area latency revealed a significant effect of recording site, $F_{(2,22)} = 10.3$, $MS_e = 1{,}945$, $P < 0.001$, indicating an earlier P3 at Fz (360 ms) compared with Cz and Pz (395 and 405 ms, respectively). No significant SOA effect emerged from an ANOVA carried out on P32 fractional area latency, $F < 1$.

Subject-by-subject analysis

Grand-average T_2-locked unsubtracted ERPs (at Fz) recorded under SOA = 800 ms conditions are reported in Fig. 9, showing the expected pattern of enlarged P2 and P3 components in the ERP time-locked to the infrequent T_2 relative to the P2 and P3 components in the ERP time-locked to the frequent T_2. Across subjects and recording sites, the mean P3 peak magnitude was 8.7 μV (median = 8.8 μV), and the standard deviation was 2 μV. In fact, only one subject produced a small P3 with a peak of 4.3 μV,[3] whereas, all the other subjects produced P3 peaks of 7.0 μV or greater. When compared with the proportion of subjects showing small or nil P3 peaks in experiment 1 (i.e., 13/25), the proportion of subjects with small P3 peaks in experiment 2 (i.e., 1/12) was significantly reduced (Yates-corrected χ^2 (1) = 4.9, Fisher exact $P = 0.027$). The correlation between SOA effect on RT (RT [SOA = 100 ms]−RT [SOA = 800 ms]) and SOA effects on P3 peak latency (P3 peak latency [SOA = 100 ms]−P3 peak latency

[3]During the final debriefing, the subject with the small P3 reported becoming interested in counting how many times a series composed of a repeated digit was followed by 8 (e.g., 2, 2, 2, 8). It is possible that a small P3 could have been generated in this subject because of surprise at the digit following a repeated series, independently of whether this digit was 8 or another (frequent) digit.

[SOA = 800 ms]) was $r = 0.19$, and not significant, $t < 1$ ($R^2 = 0.04$).

Combined analyses of experiments 1 and 2

A series of ANOVAs were carried out on the P2/P3 amplitude and latency data combined from the two experiments. An ANOVA on P2 amplitude results revealed a main effect of experiment, $F_{(1,35)} = 4.0$, $MS_e = 38.7$, $P < 0.05$, with a smaller P2 amplitude in experiment 1 compared with P2 amplitude in experiment 2, and a marginally significant interaction between SOA and experiment, $F_{(2,70)} = 2.8$, $MS_e = 47.5$, $P < 0.07$, supporting the observation of an SOA-locked suppression of P2 amplitude in experiment 1 that was absent in experiment 2. An ANOVA on P2 latency results revealed no significant effects of the variables considered (all F values < 1). An ANOVA on P3 amplitude results revealed a main effect of experiment, $F_{(1,35)} = 3.1$, $MS_e = 50.2$, $P < 0.05$, reflecting more ample P3 responses to T_2 when T_1 could be ignored (experiment 2) relative to when T_1 had to be responded to (experiment 1). There was also a main effect of recording site, $F_{(2,70)} = 15.4$, $MS_e = 6.0$, $P < 0.001$, indicating a P3 amplitude that was smallest at Fz, intermediate at Cz, and greatest at Pz. In addition, there was a significant interaction between experiment and recording site, reflecting the fact that, compared with experiment 1, more ample P3 responses in experiment 2 were more evident at Cz and Pz than at Fz, $F_{(2,70)} = 6.5$, $MS_e = 5.99$, $P < 0.003$. An ANOVA on P3 latency results revealed a main effect of experiment, $F_{(1,35)} = 6.8$, $MS_e = 2{,}344$, $P < 0.02$, indicating P3 latencies generally anticipated in experiment 2 compared with experiment 1, and, more importantly, a significant interaction between experiment and SOA, $F_{(2,70)} = 5.2$, $MS_e = 1{,}945$, $P < 0.03$.

Discussion

The most important results from experiment 2 were that, in contrast with experiment 1, all but one subject produced a substantial P3 frequency-related response, whereas only 12 out of 25 subjects did so in experiment 1. This difference contributed to produce an overall mean amplitude of the frequency-related P3 response that was smaller in experiment 1 (dual task) than in experiment 2 (single task). Given that the stimuli used in both experiments were identical (including the SOA manipulation), the only design difference between the experiments that could lead to the observed differences in results was that subjects had to actively process T_1 in experiment 1, but not in experiment 2. Consistently with previous dual-task investigations on P3 amplitude (e.g., Isreal et al. 1980), these results thus provide good evidence for a sensitivity of the P3 component to dual-task interference. This in turn, with the due caution called for by the fact that the evidence of a significant correlation between P3 latency shift and PRP effect was based on data from only 50% of the subjects participating in experiment 1, is consistent in our view with the present suggestion that the identification and classification of T_2 (in the PRP paradigm) may sometimes be subject to the influence of the PRP bottleneck.

The behavioral results of experiment 2 were also interesting. RT_2 was longer when T_2 was the less-frequent stimulus category than when it was the more-frequent category, replicating what we observed in experiment 1. As expected, the SOA effect was much smaller in experiment 2 than in experiment 1, because no task was associated with T_1 in experiment 2. Consequently, we no longer expected to observe the usual PRP effect. The small SOA effect observed in experiment 2 may represent a form of involuntary distraction caused by the presentation of T_1 (see below).

Furthermore, the ERP results of experiment 2 provide converging evidence that supports our interpretation of the SOA effects on P3 responses in experiment 1 as a reflection of central effects rather than sensory or perceptual interactions between T_1 and T_2. Whereas the SOA effect in experiment 1 caused a variation of P3 latency, the SOA effect in experiment 2 caused only a small P3 amplitude reduction in the subtracted T_2-locked ERP for the shortest SOA. Such P3 amplitude reduction is consistent with a small distracting effect of T_1 onset likely exerted on task$_2$ processing when T_1 and T_2 were displayed in close temporal contiguity (e.g., Hoffman et al. 1985), and rules out sensory corruption (e.g., masking) of T_2 by T_1 presentation in the interpretation of the SOA effects on P3 latency found in experiment 1. If the effects observed in experiment 1 were due to sensory and/or perceptual interactions between T_1 and T_2, identical effects would have been found in experiment 2 because the two experiments used the same stimulation parameters. Similarly, the SOA effects on P2 amplitude that were evident in experiment 1 were absent in the results of experiment 2. Consequently, the effects on P2 observed in experiment 1 must also have been caused by processing capacity limitations at the level of attention, rather than by limitations in early visual processing of the T_2 stimuli.

General discussion

Comparison with prior studies of PRP effects on P3

Several aspects of the present results are particularly informative concerning the relationship between the P3 ERP component and dual-task interference. Consider first the difference between the effects of the T_1–T_2 SOA on the two variables that were of most interest in the present context—namely, the T_2-locked P3 latency and RT_2. Replicating previous findings in similar experimental designs (Arnell et al. 2004; Luck 1998), decreasing the T_1–T_2 SOA in experiment 1 from 800 ms

to 100 ms produced a lengthening of P3 latency of 80 ms, and a lengthening of RT_2 of 220 ms. This difference between the PRP effect on the P3 ERP and RT_2 was also present in the results of a subgroup of subjects who had ample and well-defined P3 components. The analysis of this subgroup indicated an SOA-dependent P3 latency lengthening of approximately 100 ms and a PRP effect of 198 ms. Importantly, a similar ERP versus RT_2 difference was critical for the arguments raised by Luck (1998) and Arnell et al. (2004) against the hypothesis of a common bottleneck for both RT_2 and P3 latency in the PRP paradigm.

On the other hand, two aspects of the latency results are problematic for the view that different and independent bottlenecks explain the latency effects on the P3 response and on RT_2. Firstly, every experiment that has examined this issue has found a significant influence of SOA on the latency of the P3 component (Luck 1998; Arnell et al. 2004; and the present experiment 1). Secondly, in experiment 1 we found a significant positive correlation between RT_2 and P3 slowing, as a function of SOA, across subjects, for subjects producing a frequency-related P3. This correlation falsifies the hypothesis that the R_2 and P3 slowing are caused by independent factors, and dovetails nicely with the view we have exposed in prior work concerning the involvement of central mechanisms in stages of processing other than response selection (i.e., short-term consolidation; Dell'Acqua et al. 2003).

It is natural to wonder whether earlier failures to observe a significant correlation between effects of SOA on P3 latency and RT_2, or more substantial SOA effects on P3 latency, might have been due to the inclusion of data sets for which it was difficult to estimate P3 latency reliably. In about half of the subjects in experiment 1, the frequency-related P3 response was so small that attempts to estimate latency evidently produced essentially noise. Not surprisingly, when these subjects were included in correlational analyses attempting to relate P3 latency to RT_2 slowing, the correlation was not significant. When P3 latency could be estimated reliably (for a subset of the subjects), the correlation between P3 slowing and RT_2 slowing was instead significant (Fig. 3). Arnell et al. (2004) reported a null correlation between P3 slowing and RT_2 slowing and interpreted this outcome as consistent with the view that PRP interference occurs largely after stimulus identification and categorization. Since they did not report the reliability of their measures (in particular the reliability of their measure of P3 latency), we caution against interpreting the low correlation reported by Arnell et al. (2004) as conclusive evidence against an influence of the PRP bottleneck on stimulus identification and classification. In contrast, the positive correlation between the PRP effect and P3 latency, together with the differences across experiments 1 and 2 in overall P3 amplitude, provide evidence supporting the hypothesis of a significant central dual-task interference with the mechanisms responsible for the generation of P3 activity.

SOA effects on P2

Interestingly, we found significant SOA effects on the frequency-related P2 component in the difference ERP waveforms in experiment 1. We found both a reduction in the amplitude of the P2 and an increase in the latency of the P2 component as SOA was reduced. Although the effects were modest in magnitude, they are inconsistent with the view that there are no central interference effects on the identification and/or the categorization of stimuli in the PRP paradigm. Before we can accept this conclusion, however, we need to rule out other possible sources of interference. The most likely such source would be a capacity limitation in visual processing, prior to central mechanisms (Pashler 1989). However, the results of experiment 2 allow us to rule out this possibility. The SOA effects on P2 amplitude and latency that were present in experiment 1 were absent in experiment 2. These results converge with those observed for the frequency-related P3 component and, in overt contrast with Pashler's view (see also Pashler 1993), suggest that one source of dual-task interference in the PRP paradigm is at the stimulus identification and categorization stage. We note that Luck (1998) did not find such SOA effects in his frequency-related P2 responses. Apart from the use of a categorization task as $task_2$ (''O''s vs ''X''s) that was likely to be substantially easier than the categorization task used in the present context (1–4 vs 8), we have no obvious explanation for this difference in results, that underlines however the importance of replications and extensions of work in this domain.

The role of preparation on P2/P3 modulation

One might argue that the differences between experiments 1 and 2, in overall P3 amplitude, suggests that the dual-task effects we observed are not causally related to the PRP bottleneck, but rather to something more diffuse, such as an effect of general load, or perhaps a change in preparatory state. In the latter view, subjects might have been less prepared for the second task in the dual-task context than in the single-task context. And, perhaps such load or preparation effects could explain all of our results. However, this hypothesis is falsified by the fact that the latency and amplitude effects on the P2 and P3 components caused by dual-task interference were modulated by SOA in experiment 1. In experiment 1, every trial involved processing both T_1 and T_2, and thus subjects had to be prepared for both tasks on every trial. Given that SOA conditions were intermixed at random within blocks of trials, it is reasonable to assume that subjects could not differentially prepare for different SOA conditions, for $task_1$ versus $task_2$. Thus, the SOA effects most likely reflect an increased likelihood of processing overlap between the two tasks, as SOA was reduced, with a direct consequence for the efficiency of the identification and/or the classification of stimuli in $task_2$. We note that such interference could take the

form of graded capacity sharing, without jeopardizing locus-of-slack logic for factor effects on RT_2 (see Navon and Miller 2002; Tombu and Jolicoeur 2003).

Although a load and/or preparation account cannot explain the effects of SOA in experiment 1, such effects may have contributed to some of the observed differences across experiments. Experiment 2 showed that, under stimulus conditions that were identical to those of experiment 1 but that differed in terms of processing requirements associated with T_1, almost every subject produced P3 responses that were significantly larger than those produced, on average, by the subjects in experiment 1; and this difference was more evident at Cz and Pz than at Fz. This is not surprising in light of a wealth of data demonstrating P3 amplitude reductions with increasing task complexity (e.g., Hoffman et al. 1985). The crucial point, however, is that the correlation between P3 latency and RT_2 that was found in experiment 1 disappeared in experiment 2, despite the larger P3 amplitudes found in this latter experiment, and this points to the critical role of attending to T_1 (for the production of a response) in generating the dependence between P3 latency and RT_2 in experiment 1.

Locus of modulation of P3 activity in PRP conditions

One way to characterize the functional connection between the PRP effect and SOA effects on P3 latency in experiment 1 (and the absence of such connection in experiment 2) is by making an appeal to the locus-of-slack logic often used in PRP studies to classify variables manipulated in $task_2$ as affecting stages prior to response selection (i.e., the bottleneck stage hypothesized to generate the PRP effect), or as affecting stages at or post response selection (e.g., Pashler and Johnston, 1989). Specifically, variables manipulated in $task_2$ affecting stages prior to response selection (e.g., sensory and perceptual processing) are held to generate effects on RT_2 that are underadditive with SOA effects. This is because, at short SOAs (i.e., when response selection in $task_2$ is waiting for central mechanisms to be freed from response selection in $task_1$), pre-bottleneck variable effects are assumed to be absorbed into the slack caused by $task_2$ response selection suspension. In contrast, variables manipulated in $task_2$ affecting stages at or after response selection (e.g., response execution) are held to generate effects on RT_2 that are additive with SOA effects. In this case, it is because no absorption into slack can occur for the effects of the variable manipulated in $task_2$, which are reflected in RT_2 variations that remain constant across SOAs.

The manipulation of T_2 frequency in the present experiment generated effects that were partly underadditive with SOA effects. According to the above logic, this implies that part of the processing affected by the T_2 frequency manipulation occurred before response selection. The specific frequency manipulation used in our experiment makes it rather unlikely that the stage whose effects were "absorbed" into the slack was related to the identification of stimuli falling in the frequent/infrequent categories, because all T_2 stimuli were physically very similar, and, more importantly, equally likely to occur throughout the experiment. Instead, it seems more reasonable that identified stimuli had to undergo further perceptual processing to be classified into the two categories associated with the different responses that had to be emitted based on the information conveyed by the T_2 stimulus, a stage included in the model used by McCann and Johnston (1992, see their Fig. 6, p. 482) and defined as necessary for "perceptual decision making." Therefore, given the present results, we propose that frequency effects in the present context were affecting the duration of two stages of processing, both affecting RT_2 at a long SOA, a stage of perceptual classification of the stimuli into the frequent/infrequent categories and a later stage of processing engaging the same mechanisms required for response selection. In this view, only the central stage of processing contributing to the generation of a frequency-related P3 component was subject to central postponement at short SOAs, providing an explanation for the smaller P3 latency effects than PRP effects in the present study and in prior PRP studies. It is worth mentioning that underadditive effects of SOA and relative T_2 frequency were not found by Luck (1998) in his PRP paradigm, or by other researchers manipulating the same factors in PRP paradigms (e.g., Van Selst and Jolicoeur 1997). In other dual-task studies, however, underadditive effects of SOA and T_2 relative frequency were consistently found, especially under conditions in which T_2 was difficult to perceive (e.g., Crebolder et al. 2002). There might be a number of methodological differences between the present study and the study by Luck (1998), some of which have already been mentioned, that can account for this specific discrepancy in the results. In particular, we tested RT_2 performance using a wider range of SOAs (100–800 ms) than Luck (1998), who used SOAs ranging from 50 ms to 350 ms. Interestingly, the underadditive effects of SOA and T_2 frequency were found by Crebolder et al. (2002) using SOAs that were more similar to ours than to Luck's (Luck 1998), with T_2 frequency effects much larger at SOAs close to 800 ms than at shorter SOAs. This raises the possibility that underadditive effects can only emerge by testing multitasking conditions in which the separation between the stimuli is longer than that used by Luck (1998), but this certainly would require more systematic investigations to ascertain.

One or more stages for P3 generation?

Positing distinct processing stages as potential sources of P3 activity and observing that P3 latency fluctuations were more evident frontally (Fz) than at more posterior sites (Cz, Pz) call into play a recent proposal on the organization of the functional sources of P3 activity put forth by Falkenstein et al. (1994). Using an oddball

paradigm and different response conditions (i.e., a simple reaction time condition and a choice reaction time condition), these authors have isolated two components hypothesized to be normally confounded in the frequency-related P3 component, a P3 component (P-SR) with a centrofrontal distribution held to reflect stimulus evaluation, and a P3 component (P-CR) with a centroparietal distribution held to reflect response selection. Subsequent work by Dien et al. (2004) has suggested that the P-SR component isolated by Falkenstein et al. might instead reflect a frontal response generated at a processing stage similar to that underlying the generation of a classical, more posterior P3 component. In addition, Dien et al. have hypothesized that the processing stage responsible for the generation of the P-CR component could be stimulus classification, and not response selection as argued by Falkenstein et al. (1994) Distinct aspects of the results of the present experiments 1 and 2 bear some relevance to this controversy. First, we observed an SOA-dependent prolongation of P3 latency, and a significant correlation between P3 latency and RT_2, which we attributed to response selection overlap in dual-task conditions (experiment 1). In this vein, the present results are not consistent with the view that the mental processes reflected in the P3 component are entirely independent of response selection processes, as argued by Dien et al. On the other hand, even the view proposed by Falkenstein et al. of an independent contribution of stimulus evaluation processes to the generation of a frontal subcomponent of the P3 fails to account for the present results. Recall that P3 responses in the present experiments were invariably detected at Pz/Cz, that is, where the classical P3 is normally observed, and not frontally, as the proposal made by Falkenstein et al. would lead us to expect. One way to salvage an explanation of the present SOA effects on P3 based on the work of Falkenstein et al. would perhaps be that stimulus evaluation conditions, which have been hypothesized by these authors to be reflected in the frontal P3, changed across SOA conditions in experiment 1. It this were so, however, an analogous SOA-dependent fluctuation should have characterized P3 responses in experiment 2, in which stimulus evaluation conditions were nominally identical to those implemented in the design of experiment 1. The observed pattern of results disconfirmed this interpretation, and is therefore not consistent with the view held by Falkenstein et al. On the other hand, we note that this pattern of results is convergent with recent hypotheses concerning the ''attentive'' nature of the mental processes reflected in the frontal P3 (or $P3_a$; e.g., Katayama and Polich 1998). Interestingly, some recent ERP and fMRI evidence suggest that the frontal part of the P3 component is modulated by activity in regions located in the dorsolateral prefrontal areas (e.g., Knight 1997; McCarthy et al. 1996). Frontal and prefrontal areas are, as suggested by many (e.g., Duncan and Owen 2000), part of an integrated neural network involved in high-level processing of stimuli requiring overt responses. In this perspective, prefrontal areas, being largely involved in the coordination of tasks in multitasking conditions (Jiang and Kanwisher 2003b; see also Hazeltine et al. 2001; Schubert and Szameitat 2003; Szameitat et al. 2002), would be a natural neural locus of modulation of part of the processing reflected in the frequency-sensitive P3 component.

Furthermore, it is central for an interpretation of the present empirical scenario (and of prior P3 studies implementing analogous paradigms) to consider Verleger's (1997) meta-analytic review of more than 120 studies focusing on the functional localization of the processes concurring in the generation of P3 activity. Converging with the two-component theory of the P3, Verleger's (1997) elegant demonstration that P3 latency sensitivity is amenable to substantial variations depending on the speed at which a cognitive task is executed bears an obvious relation with the electrophysiological results of experiment 1. In detail, the model proposed by Verleger accounts for the finding that P3 latency sensitivity has been found to be negatively correlated with the baseline RT in a variety of speeded tasks. According to this model, this is because fast RTs are more likely to reflect the outcome of a processing route along which both components contributing to P3 activity become postponed after an early stage of stimulus identification. With slow RTs, only one (the latter) of the P3 components—likely sensitive to late-stage response-related factors—becomes postponed, whereas the latency of the earlier component—allegedly sensitive to factors affecting stimulus quality—stays invariant. As modeled by Verleger, the result of the convolution of P3-like functions mimicking the electrophysiological outcome of the concurrent activity of the two components under slow RT conditions is that a second-component shift of 80 ms yields a shift of the entire P3 complex of only about 40 ms (accompanied by a P3 amplitude increase). This finding is crucial in the present context in two main respects. Firstly, as far as the quantitative relationship between the PRP effect and the P3 latency shift observed in experiment 1 is concerned, Verleger's (1997) demonstration of the negative correlation between RT and P3 latency sensitivity appears to provide a natural account for the discrepancy between these estimates. This, in Verleger's terms, would occur because P3 latency sensitivity to experimental manipulations at short SOAs, i.e., when the longest RT_2s are normally detected in a PRP paradigm, would be sensitively diminished compared with P3 latency sensitivity at long SOAs, bringing to the fore the potential consequence that P3 latency shifts observed in PRP conditions should rather be treated as consistent underestimations of latency shifts that are predicted on the basis of the observed PRP effects. Secondly, the results of experiment 1 provide in our view a solid ground to argue that Verleger's (1997) two-route model may be extended to account for the finding that only a portion of the participants showed a P3 latency shift and a significant correlation between such shifts and the

individual estimates of the PRP effect. As is illustrated in Fig. 5, the participants whose performance was associated with attenuated P3 latency sensitivity to the manipulation of T_2 frequency were also those whose behavioral performance in the critical condition (i.e., RT_2 to less frequent T_2) was about 100 ms longer compared with the RT_2 produced by the participants whose P3 was more sensitive to the frequency manipulation (see Fig. 6). If Verleger's model were correct, this would imply that a 100-ms prolongation of RT_2 in experiment 1 was sufficient to generate a condition in which P3 latency shifts were not observable in slower participants because the processing reflected in a P3 increase (following the presentation of a less frequent T_2) occurred prior to processing sensitive to the frequency manipulation.

Future directions of the present line of research

One incidental observation is that the P3 latency variations found in the present experiment (100-ms peak shift across SOAs) were relatively larger than P3 latency variations found in the two previous studies by Luck (1998) and Arnell et al. (2004) (i.e., 51 ms and 69 ms, respectively). In these studies, however, P3 latency variations were accompanied by variations in P3 amplitude, with smaller P3 amplitudes at short SOAs versus long SOAs in the frequency-related T_2-locked ERP wave. P3 amplitude variations were clearly absent in the results of experiment 1. Amplitude variations in these PRP contexts are difficult to understand, for different reasons. One reason is related to the subtraction method used for the generation of frequency-related ERP waves, which hides important information about the amplitude of each of the two contributing ERP waves (i.e., the ERP wave in response to frequent stimuli and the ERP wave in response to the infrequent stimuli). In this perspective, it is virtually impossible to disentangle whether the P3 amplitude variations described by Luck (1998) and Arnell et al. (2004) were determined by an amplitude reduction of the P3 response to the infrequent stimuli, an amplitude increase of the P3 response to frequent stimuli, or a mixture of these effects. From a more conceptual point of view, it is also difficult to understand what these P3 amplitude variations reflect (see, for an analogous conclusion, the detailed review of Kok 2001). An inverse correlation between P3 amplitude and P3 latency is often found in studies based on stimulus frequency manipulation (Johnson 1986). In addition, P3 latency variability also seems to be inversely correlated with P3 amplitude (e.g., Johnson and Donchin 1985). Considering that an increase in RT_2 variability (beside RT_2 mean) is normally observed at short SOAs in PRP paradigms, future investigations may be profitably addressed to understand whether (1) this RT_2 pattern is related to the SOA-dependent P3 amplitude variations, and (2) a behavioral estimate taking RT_2 variability into account might possibly represent a better quantification of the PRP effect against which SOA-dependent P3 latency shifts should be compared, when P3 amplitude variations emerge from the analysis of frequency-related ERP waves.

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