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Target consolidation under high temporal processing demands as revealed by MEG

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We investigated the nature of resource limitations during visual target processing by imposing high temporal processing demands on the cognitive system. This was achieved by embedding target stimuli into rapid-serial-visual-presentation-streams (RSVP). In RSVP streams, it is difficult to report the second of two targets (T2) if the second follows the first (T1) within 500 ms. This effect is known as the attentional blink (AB). For the AB to occur, it is essential that T1 is followed by a mask, as without such a stimulus, the AB is significantly attenuated. Usually, it is thought that T1 processing is delayed by the mask, which in turn delays T2 processing, increasing the likelihood for T2 failures (AB). Predictions regarding amplitudes and latencies of cortical responses (M300, the magnetic counterpart to the P300) to targets were tested by investigating the neurophysiological effects of the post-T1 item (mask) by means of magnetoencephalography (MEG). Cortical M300 responses to targets drawn from prefrontal sources – areas associated with working memory – revealed accelerated T1 yet delayed T2 processing with an intervening mask. The explanation we are proposing assumes that "protection" of ongoing T1 processing necessitated by the occurrence of the mask suppresses other activation patterns, which boosts T1 yet also hinders further processing. Our data shed light on the mechanisms employed by the human brain for ensuring visual target processing under high temporal processing demands, which is hypothesized to occur at the expense of subsequently presented information.

Keywords: Attentional blink; P300; Prefrontal cortex; RSVP; Visual attention; Working memory

Introduction

An important feature of biological information processing systems in general is that all operations are implemented in a massively parallel and distributed architecture, which works by means of highly complex cooperation among a huge number of simple processing elements. The administration and control of system resources for focused processing of incoming information is commonly termed "attention" and particular emphasis has been put on the investigation of its mechanisms and limitations. For investigating the dynamics of attention in time, the method of Rapid Serial Visual Presentation (RSVP) has proven to be useful, where items are presented very quickly in succession with participants required to identify specified targets within this visual 'stream'. This technique produces the so-called attentional blink (AB; Duncan et al., 1994; Raymond et al., 1992; Shapiro et al., 1997; see Fig. 1), and is the paradigm we employ for the purpose of the present investigation.

The AB is revealed when two targets (T1 and T2), presented in close temporal proximity within an RSVP stream (stimulus onset asynchrony, or SOA < 500 ms), must be detected and/or identified. The effect is defined by a dramatic drop in performance on T2 with the function's minima occurring at approximately 200–300 ms SOA (e.g., Raymond et al., 1992). With very few exceptions, a robust AB has been found regardless of the modality in which the targets are presented and, importantly, whether targets are embedded in a longer RSVP stream or presented within short target-distractor pairs (e.g., Duncan et al., 1994). The post-target distractor stimuli (masks) have been shown to be of particular importance as without adequate masking of both targets attenuated AB effects were observed (Brehaut et al., 1999; Raymond et al., 1992; for a review, see Enns and DiLollo, 2000; Grandison et al., 1997). Preserved performance is also revealed if T2 immediately follows T1 at lag1, an effect that was termed "Lag1-Sparing" (Kessler et al., in press; Raymond et al., 1992; see Visser et al., 1999 for a review). Hence, in order to observe deficits in the

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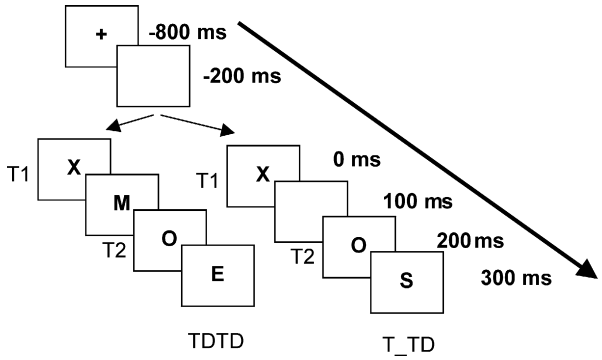

Fig. 1. The experimental paradigm. Each trial was self-initiated. A fixation cross (800 ms) was followed by a blank screen for 200 ms before the presentation of the letter stream was automatically started. In the figure, the onset times of each letter are reported with respect to the onset of the stream. Each letter was on screen for 50 ms followed by a blank screen of 50 ms. On a certain number of trials (see Table 1), a "gap" (blank screen) of 150 ms was inserted between the offset of the first and onset of the next letter in the stream. Each stream could contain either 3 or 4 letters. Targets were defined by identity and comprised the two letters "X" and "O". The figure shows two dual target conditions. On the left (TDTD) T1 was followed by a distractor (mask) while on the right (T_TD) a gap was inserted between T1 and T2. After the offset of the last letter in the stream, a blank screen was presented for 650 ms, followed by the request to report the number and the identity of the targets spotted in the stream. Further explanations in the text.

allocation of temporal attention, it seems necessary that processing of a first target is affected by a non-target stimulus, that is, by the next item in the stream. The present report seeks to understand the precise nature of this effect.

The two-stage model proposed by Chun and Potter (1995) assumes that the post-T1 item (T1-mask) competes for the resources needed by T1 processing at the stage of working memory, which delays T1 consolidation. Consolidation is defined as the generation of a more durable representation in working memory that allows for subsequent report of the target (Chun and Potter, 1995). This consolidation stage is argued to be limited in capacity as only one item can be processed at a time. Hence, during the time T1 consolidation is delayed by the post-target item, T2 cannot be consolidated and T2 report failures are likely to occur (Chun and Potter, 1995). With more emphasis on attentional capacity limitations rather than consolidation per se, a similar conclusion has been advocated by Shapiro et al. (1997). Enns and DiLollo (2000) have further specified that if access of T2 to the consolidation stage is delayed then the perceptual pattern of T2 is likely to be substituted by the next item in the stream ("object substitution"). Indeed, Vogel and Luck (2002) were able to show that cortical responses (the P300 ERP component) to T2 are delayed during the AB, possibly as a result of delayed T1 consolidation. Although the hypothesis of delayed T1 consolidation is plausible and straight-forward, to date, it has not been directly confirmed.

Alternatively, theoretical considerations regarding backward masking suggest that, under certain circumstances, a mask following a target might have an accelerating influence on target processing. Turvey (1973) gives an overview of the state of the art in backward masking and proposes a new integrative model that accounts for masking effects by describing the disruptive effect of a mask on target processing. Under the usual AB conditions (relatively long target display time and ISI) that do not aim at obliterating T1 (T1 should be reportable), Turveys' model predicts that T1 processing is disrupted by the mask, yet enough evidence has already accumulated for T1 to be reported. Disrupting T1 processing at this stage may in turn have an accelerating influence, especially if one takes into account the more recent "re-entrant processing" framework of backward masking proposed by Di Lollo et al. (2000): if enough evidence has already accumulated for T1 to ensure awareness, then stopping the system from persevering through further (redundant) iterations could in fact accelerate T1 consolidation into working memory.[1] Yet, if T1 is accelerated then what may be the cause for attenuated performance on T2, that is, the AB? It is this question that we seek to answer in the present report (Table 1).

This report examines the effect of T1-masking on the neural correlates of T1 and T2 consolidation in the AB, using MEG techniques that focus specifically on the M300 signals, which have been previously validated as an index of visual working memory consolidation. To study this problem, brain activity was recorded by means of a 122-channel whole-head neuromagnetometer Neuromag™ during an AB paradigm that varied the masking of T1. Specifically, we concentrated on the modulation of the evoked cortical response approximately 300 ms after target onset (M300, the magnetic counterpart to the electric P300), as this component has been shown to reflect target-related processing, working memory consolidation, and to play a major role in the AB (Arnell et al., 2004; Kranczioch et al., 2003; McArthur et al., 1999; Vogel and Luck, 2002). Specifically, it has been found that the P300 component to 'blinked' T2s is significantly attenuated (Vogel et al., 1998). To seek a neural locus for this effect, we focused on prefrontal areas, which have been shown to be crucial to working memory functions (see Miller and Cohen, 2001, for a review). To summarize our approach, modulation of the M300 waveform derived from prefrontal cortical sources enabled us to test the role of the first target's mask in yielding the dual-target attentional deficit known as the AB.

Methods

Subjects and experimental procedure

Subjects were right-handed, 4 being members of the University staff and 6 being students at Duesseldorf University. Mean age was 28.8 (SD ± 5.8), and 3 were female and 7 male. Individuals had no neurological deficits and gave their written informed consent prior to the experiment. The study was approved by the local ethics committee and is in accordance with the declaration of Helsinki.

We have pointed out in Introduction that an AB effect is observed whether targets are embedded in a 20-item RSVP stream or just presented on their own followed by masks (Duncan et al., 1994; Ward et al., 1997). In order to save measurement time, we

[1] Bachmann (1984) proposes the "perceptual retouch" account, which also predicts boosted target processing due to the mask at specific target-mask timings (SOAs and ISIs). These effects are postulated to occur because of the interaction of a fast, specific cortical system with a slower, unspecific thalamic system. The phasic "energy" provided by the thalamic system is the major factor for certain stimuli to reach conscious awareness. Due to the different processing speed in the two systems, interactions in form of mutual support vs. interference occur between target and mask at varying SOAs/ISIs.

Table 1
Conditions employed in the experiment

Single target conditions	T_DD	20
	TDDD	20
	DTDD	20
	DDTD	20
Dual target conditions	T_TD	40
	TDTD	40
	TTDD	40
Distractors only conditions	DDDD	40
	D_DD	20

The conditions are labeled according to the qualities of the letter stream that was used. A "T" denotes a target ("X" or "O") at a certain stream position while "D" is a place-holder for any distractor. "Underscore" signifies that there was a gap in the stream. For example, the letter stream "X", "M", "O", "S" is labeled as "TDTD". On the far right is shown the amount of trials for each condition in each block. There were 260 trials in each block.

decided to employ this abbreviated version for our study. Three independent variables were combined in a repeated measures design. Factors were "lag" (1 or 2), "number of targets" (0, 1 or 2 targets present) and "masking" with respect to the first target (masked or unmasked). The factors "lag" and "masking" were not entirely independent. At lag 1, T1 was followed by T2, so masked/unmasked T1 was not applicable for this lag. Therefore, "lag" and "masking" were included into two separate statistical analyses. The dependent variable was percentage of correct target identifications in single target trials and percentage of correct T2 identifications – given T1 correct identification – in dual target trials.

The experiment consisted of 12 to 16 blocks per subject containing 260 trials each. The number of blocks was increased until the respective subject reached at least 90 trials in each condition of interest in order to achieve a good signal-to-noise ratio (note, that the number of "AB" vs. "noAB" trials highly depended on the performance on both targets of the respective subject). An additional block of 24 trials was used at the start of the experiment for instruction of the subject and practice. Target letters were restricted to "X" and "O", while distractors could be any letter of the alphabet. T2 was presented either immediately after T1 (lag 1, SOA 100 ms, ISI 50 ms) or with an SOA of 200 ms (lag 2). In the latter case, there were two possibilities, first, another letter of the alphabet could intervene between T1 and T2 (masked T1 condition) or, second, there was a gap between T1 and T2 of 150 ms inter-stimulus interval (*un*masked T1 condition). A target in single-target trials could appear in any of the first three positions of the stream. To reduce predictability, we also included single-target trials as well as pure distractor trials with an identical gap after the first letter. In summary, all trials consisted of either 3 or 4 letters, while the end of each stream occurred fixed at 350 ms after onset of the first letter as the "missing" letter was always at position 2 (see Fig. 1).

Letters were presented on a back-projection screen with a visual angle of 3.72° at a distance of 1.2 m. Subjects initiated each trial in a self-paced manner by pressing a button on a light-barrier button-box. After presentation of the letter, stream subjects were requested to indicate which targets they had detected (first "how many targets", second "which targets"). After each block of trials, which took approximately 15 min, subjects were asked if they wanted a short (ca. 3 min) or a long (ca. 10 min) break. In the middle of the experiment (after about 6–8 blocks), subjects received a longer break of at least 30 min.

MEG: source localization

Using a 122-channel whole-head neuromagnetometer (Ahonen et al., 1993), brain activity was recorded with a band-pass filter of 0.03–170 Hz and digitized at 514 Hz. Vertical electro-oculogram was recorded simultaneously for off-line rejection of epochs contaminated by eye movements and eye blinks. MEG signals were averaged off-line between −500 and 1000 ms with respect to the letter stream onset.

Source modeling was applied individually and consisted of the search for clear dipolar field patterns, dipole modeling and evaluation of the fitted dipoles (Hämäläinen et al., 1993). The current dipoles were identified one by one, at time points where each specific field pattern was clearest. The channel with the largest magnetic peak and the surrounding eight to ten channel-pairs were selected for dipole modeling. Only sources with a goodness of fit (g) of >85% were accepted. The sources were then brought into a multi-dipole model where the source locations and orientations were kept fixed, whereas their amplitudes were allowed to vary as a function of time to best account for the signals measured by all 122 sensors in all experimental conditions and over the entire length of the trials. The complete models included 7–10 sources per subject. The resulting source waveforms represent the time courses of activation in the cortical source areas.

The location of each source is defined in head coordinates. The position of the head within the magnetometer was found by attaching four small coils on the subject's head, measuring their location in the head coordinate system with a 3-D digitizer (Isotrak 3S1002, Polhemus Navigation Sciences) outside the MEG system, and energizing them briefly, inside the MEG system, to obtain their locations in the magnetometer coordinate system. MEG sources were combined with the individual anatomy by marking the three anatomical points (ear canals and nasion) in the individual MR images. In a further step, individual source locations were mapped onto a standard brain by using SPM99 (SPM99: Wellcome Department of Cognitive Neurology, Institute of Neurology, London).

In eight out of ten subjects, we observed one occipital and two bilateral occipito-temporal sources (in two subjects only one occipito-temporal dipole could be fitted according to our standards) which is in agreement with previous MEG findings regarding visual object processing (e.g., Tarkiainen et al., 2002). Occipital cortex is known to compute early visual feature extraction (e.g., Blasdel and Salama, 1986) and has not been reported to be involved in the generation of the P300 ERP component. Accordingly, the pattern of evoked neural activity in these sources can be taken to reflect early visual signal processing and, indeed, cortical responses (100–140 ms) to targets and to distractors did not differ significantly ($Z = -1.376$; $P < 0.169$, tested for the first stream position). Therefore, in order to focus on our main research questions, occipital waveforms will not be further analyzed here. However, to give an impression of these signals, we generated an occipital cluster based on the most medial occipital source in each individual (dipoles fitted individually within 79–159 ms after stream onset). The averaged waveforms showed regular biphasic responses every 100 ms that accurately reflect the visual letter stream (Fig. 2, Panel A).

The sources in two lateralized temporo-parieto-frontal clusters were anatomically highly distributed across individuals and covered a wide range of temporal, parietal and frontal areas. This is in concordance with the modeling results from one of our MEG pilot studies and with other findings that show a wide range of areas being involved in dual target processing in RSVP tasks (e.g., Feinstein et

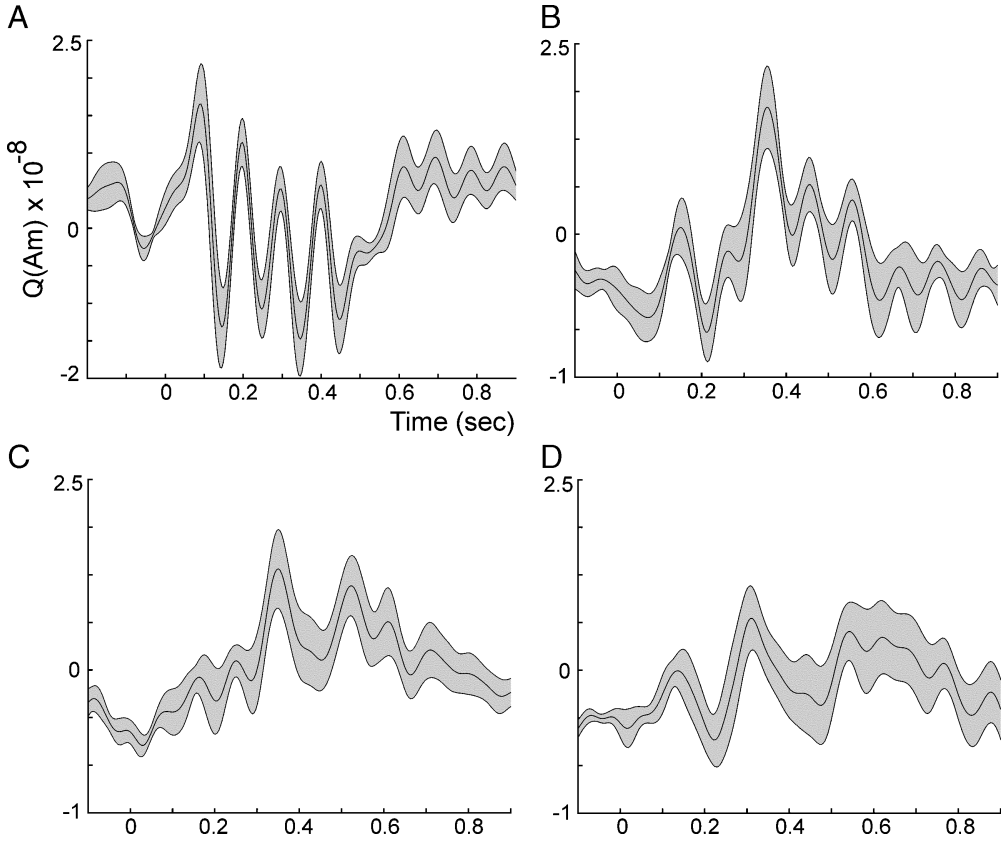

Fig. 2. Average waveforms with standard error of mean (SEM) for each cluster: Panel A—occipital; Panel B—right temporo-parieto-frontal (TPF); Panel C—left temporo-parieto-frontal (TPF); Panel D—prefrontal (PFC) cluster.

al., 2004; Gross et al., 2004; Marois et al., 2001). We interpret this as a symptom of the very complex cognitive processes involved in the AB. Quite large networks might be engaged in some sub-processes and different components might be best to trace in each individual by means of dipole modeling. This method assumes cortical sources with practically no spatial extension yet with a high variability in activation over time in contrast to more wide-spread and slowly activated volumes like the ones obtained with imaging techniques. We therefore decided to trade anatomical correspondence between individual sources for the similarity of their waveform signals.[2] That is, clusters were determined by a rough anatomical classification in a left and a right temporo-parieto-frontal cluster (TPF) but most importantly by the temporal similarity of signal properties (SEM in Fig. 2, Panels B and C) across the subject's multi-dipole models. Hence, those sources were clustered together (one source from each subject) that showed minimal inter-individual variability over time (as an objective criterion the average of the SEM was computed and minimized across the trial interval). Dipoles in left TPF were fitted individually within 303–391 ms after stream onset, and dipoles in the right TPF were fitted individually within 177–287 ms after stream onset. Finally, only one prefrontal source was obtained for each subject (dipoles fitted individually either within 284–395 ms or 547–593 ms after stream onset) and was included into a cluster (PFC) for further analysis. Fig. 2, Panel D shows the average waveform and the relatively low SEM for this cluster.

[2] It is important to note that our focus was not primarily directed on localization but on the temporal dynamics of attentional processes.

MEG: extraction of individual amplitudes and latencies

As pointed out, our main focus of interest was directed towards components around 300 ms that have been shown to reflect target processing in the AB (e.g., P300 component in EEG). Therefore, we identified the peaks for each subject for each source in each cluster (apart from occipital) that lay within a time window of 250–450 ms after target onset by means of automatic peak detection. Consequently, for targets occurring at stream positions 2 and 3, the onset delay was added to the interval resulting in a time window of 350–550 ms after stream onset for targets at position 2 and of 450–650 ms for position 3. As these time windows overlap, we made sure by visual inspection that for the dual target conditions different local peaks in the correct temporal order were picked for each target. Amplitudes and latencies were quantified for each source. In the right temporo-parieto-frontal cluster cortical responses where biphasic in that a negative peak was followed by a positive peak—similar to the occipital pattern in Fig. 2, Panel A. To account for modulations on both components, minimum-to-maximum amplitude differences were employed as quantification for amplitudes in the right TPF cluster. Averages and standard deviations for each source cluster and condition are provided in Appendix A. M300 waveforms and results for the lag1 condition are reported in Kessler et al. (in press).

Note that individual peak extraction and the separate statistical analysis of amplitudes and latencies might lead to some mismatch with the average waveforms shown in the Figs. 5 and 6. This is because individual peaks (highest amplitudes) occur at individual latencies and therefore average waveforms suffer from a smearing across subjects. In addition, subjects differ in the strength of their

cortical responses and the average waveforms will be stronger affected by subjects with high amplitudes. The smearing tends to obliterate effects that turn out significant in analyses that are focused either on amplitudes or on latencies only. As subjects differ substantially in the strength of their cortical responses, non-parametric Wilcoxon tests (two-tailed) are used to determine differences in amplitude between conditions. In contrast, latency differences between conditions are analyzed with paired *t* tests and ANOVAs because inter-individual time scales were comparable.

Results

Behavioral data

The results shown in Fig. 3 reflect the expected pattern of behavioral performance. Preserved performance is observed at lag1 and at lag2 without a T1 mask, while a significant drop in performance at lag2 is observed with a T1-mask, indicating the occurrence of an AB. Statistically, this impression was confirmed by a significant interaction ($F(9) = 50.158$; $P < 0.0001$) of task (single vs. dual targets) with lag (1 vs. 2-masked) and by a significant *t* test ($T(9) = 4.951$; $P < 0.001$) contrasting T1 masked vs. *un*masked at lag 2 (Fig. 3).

Source clusters

Two bilateral clusters were determined in temporo-parieto-frontal areas by minimizing the SEM of the averaged waveforms (see Methods). Due to the rough localization of the sources, we will refer to these clusters as "right TPF" and "left TPF", respectively. The areas between frontal, temporal and parietal cortex have been shown to be essentially involved in the AB as patients with lesions in inferior frontal, superior temporal and inferior parietal areas do show abnormal attentional blink functions (Husain et al., 1997; Shapiro et al., 2002). This is in concordance with our finding that both clusters are involved in the generation of the target-related M300 responses (T1 = 1st and T2 = 2nd gray bar in Fig. 4, Panels A, B) as revealed by comparisons to the distractor only condition (all $Z < -2.090$; $P < 0.037$).

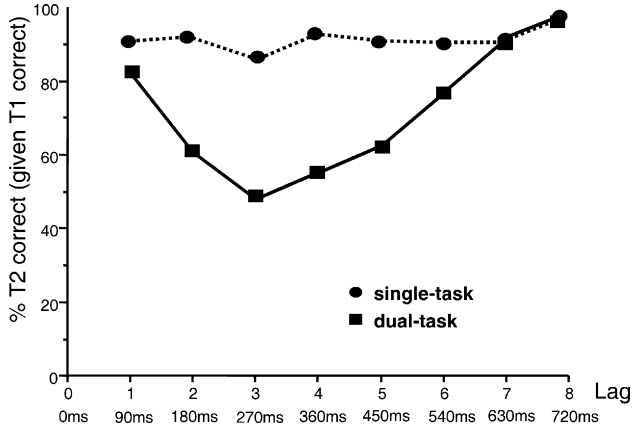

Fig. 3. Behavioral results. This figure shows the observed percentage of correct target reports: percentage of correct single targets for the "single target" condition and correct T2 performance *given* T1 correct for the "dual target" condition, respectively. "Dual no mask" refers to the condition where a target presented at the first position in the stream was followed by a gap of 150 ms (cf. Fig. 1) before the second target was presented (SOA 200 ms). "Lag 1" indicates that T2 immediately followed T1 with an SOA of 100 ms, while at "Lag 2", the SOA between the two targets was always 200 ms (with or without an intervening distractor).

In each subject, at least one prefrontal cortex (PFC) source was fitted (Fig. 4, Panel C). Although there was some variation in the exact location across individuals, there was a quite high similarity in the time course of the signals as is suggested by the relatively low standard error of mean (SEM) across the trial interval (Fig. 2, Panel D). One subject showed an anterior cingulate source, while the rest of the individual sources ranged from dorsolateral, ventrolateral to orbital prefrontal locations. All these areas have been related to high-level attentional processing and working memory (e.g., Feinstein et al., 2004; Glahn et al., 2002; Goldman-Rakic, 1996; Kessler and Kiefer, in press; Marois et al., 2001; Mottaghy et al., 2002; Nobre et al., 1999; Stephan et al., 2003).

The prefrontal region bears the highest impact on the theoretical issues we have raised in Introduction. That is, competition for resources between T1 and the subsequent mask is thought to delay processing at the level of working memory (cf. Chun and Potter, 1995). Accordingly, clear target-related M300 peaks (T1 = 1st and T2 = 2nd gray bar in Fig. 4, Panel C) are observed in the PFC cluster for reported targets in all target conditions as compared to the distractors only condition (all $Z < -2.599$; $P < 0.009$). The modulations of the PFC waveforms will be considered in detail in the next subsection.

Masking effects in PFC

Our main research question was whether T1-related waveform components around 300 ms would be delayed by a subsequent mask at the level of working memory. In this general formulation of the masking effects on T1, the predictions are independent of the T2 processing outcome. As a first step, we therefore averaged trials across blinked T2 trials (AB) and reported T2 trials (noAB), given that T1 had been correctly reported. This procedure was applied separately to *un*masked and masked T1 trials.

The results in Fig. 5 are clear. The T1-related M300 peak for masked trials is significantly earlier than the peak for *un*masked trials ($T(9) = 3.107$; $P < 0.013$). Peak amplitudes did not differ significantly ($Z = -0.255$; $P > 0.798$) among the dual target conditions but differed significantly from the distractors only condition (both $Z > -2.497$; $P < 0.013$). This suggests that the observed peaks around 300 ms after T1 onset are indeed target-related responses (Fig. 5, Panel C). Concluding, a subsequent mask seems to speed-up and not to delay T1 consolidation.

Next, a more detailed analysis was performed that allowed to determine differences between AB and noAB trials and to identify the impact of the mask in noAB trials (Fig. 6). With respect to amplitudes, all three dual target conditions show a significant T1-related M300 response (1st gray bar, Fig. 6, Panels A, B) as compared to the distractor condition (all $Z > -2.599$; $P < 0.009$). This T1-related response does not differ significantly in amplitude among the target conditions (all $Z < -1.172$; $P > 0.241$). In contrast, the corresponding M300 response to T2 (2nd gray bar, Fig. 6, Panel A) is significantly attenuated if T2 was blinked, hence, if AB was observed (masked, noAB vs. masked, AB: $Z = -2.090$; $P < 0.037$; also, *un*masked, noAB vs. masked, AB: $Z = -2.803$; $P < 0.005$), replicating previous findings (McArthur et al., 1999; Vogel and Luck, 2002; Vogel et al., 1998).

The most important outcome with respect to our research question was the pattern of M300 peak latencies in noAB trials

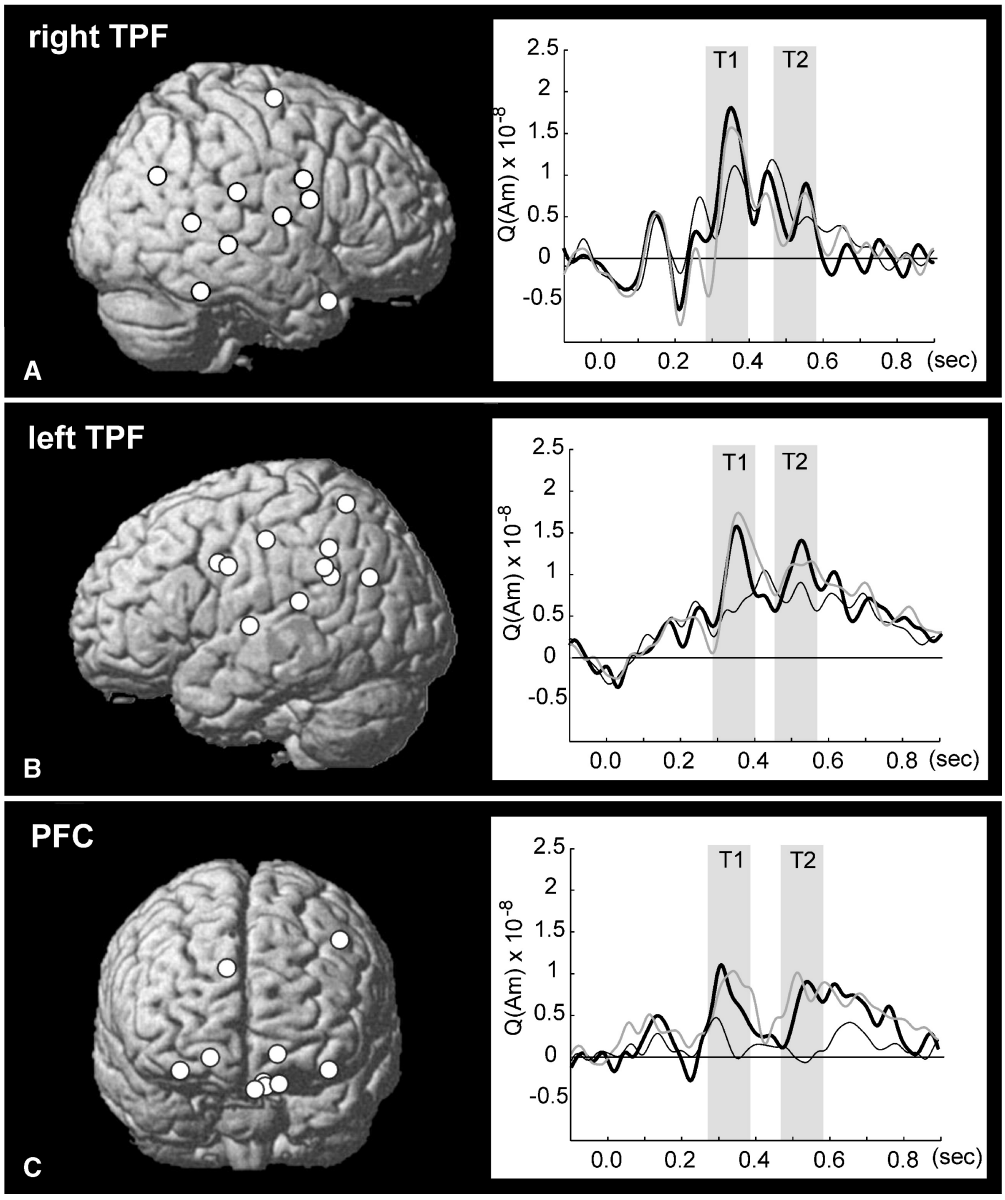

Fig. 4. Locations of the analyzed sources. Individually fitted dipoles where included into the three clusters depicted in Panels A to C (10 dipoles per cluster; one per subject). Panel A shows the sources in the right temporo-parieto-frontal cluster (right TPF), Panel B in the left temporo-parieto-frontal cluster (left TPF) and finally Panel C in the prefrontal cluster (PFC). Individual source locations were mapped onto a standard brain by using SPM99 (SPM99: Wellcome Department of Cognitive Neurology, Institute of Neurology, London). Grand averages, at the far right of each panel, illustrate mean source waveforms for the dual target (noAB) conditions in comparison to the distractor only condition in the respective cluster. Grey bars denote T1- and T2-related M300 components, respectively. Waveforms are time-locked to the first letter in the RSVP stream (0 ms).

(Fig. 6, Panels B, C). Again, T1-M300s in *un*masked trials are significantly *delayed* with respect to responses in masked trials ($T(9) = 2.259$; $P < 0.05$). In contrast, T2 responses show exactly the *reversed* temporal order: T2 processing is significantly delayed *with* a T1-mask (masked vs. *un*masked: $T(9) = 3.051$; $P < 0.014$). Overall, this reveals a longer T1–T2 inter-peak interval in the masked condition than in the *un*masked condition. This is visualized in Fig. 6, Panel B by a black horizontal arrow (masked condition) that is longer than the gray horizontal arrow below (*un*masked condition). The inverse relationship for T1 and T2 timing was statistically substantiated by a significant interaction ($F(1,9) = 14.997$; $P < 0.004$) between "target" (T1 vs. T2) and "masking" (masked vs. *un*masked T1) in an ANOVA that included both factors.

These data suggest that the mask does not seem to delay T1 consolidation and in turn T2 processing as proposed by Chun and Potter (1995). Instead, our findings emphasize that the mask prolongs the interval between (accelerated) T1 and (delayed) T2 processing. We propose that the mask abruptly terminates T1 processing (cf. Turvey, 1973) and interferes with preparation for T2. The mechanisms that might underlie this outcome will be discussed in more detail in General discussion.

Timing differences across source clusters

Mask-related asymmetries in T1 processing across clusters were analyzed by an ANOVA (noAB trials only) that included

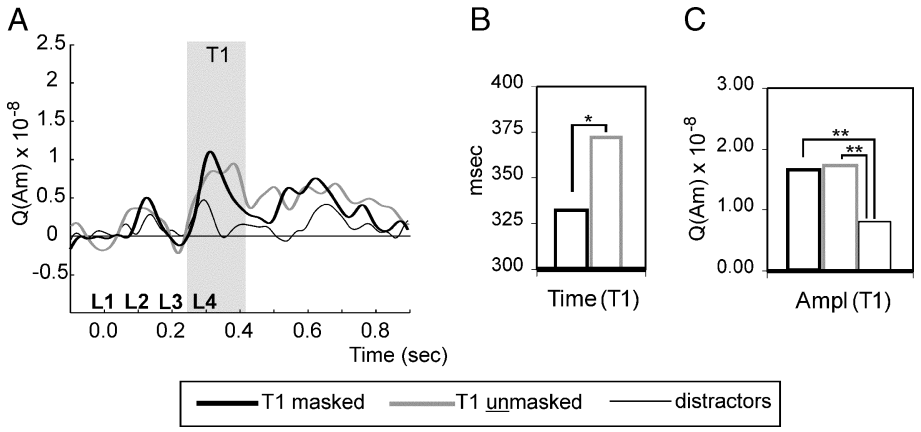

Fig. 5. The general effect of the T1 mask (AB + noAB trials). Panel A: waveforms are shown for the prefrontal (PFC) source cluster, time-locked to the first letter in the RSVP stream (0 ms). The sequence of events on the screen is shown for the dual target conditions (masked: black; *un*masked: gray) below the *x* axes (the position in relation to the *x* axes denotes onset time). M1 and M2 refer to the T1 and the T2 mask, respectively. The distractor condition is merely shown as a baseline for target-related components. The gray bar in Panel A indicates the T1-related M300 components. The significant amplitude differences between these target-related M300 peaks and the distractor-related responses in the corresponding time window are depicted in Panel C. Panel B shows the significant latency delay for *un*masked trials compared to masked trials. Note that amplitudes in the diagrams are generally higher than in the corresponding waveforms. That is because amplitude comparisons were calculated on the basis of individual peaks disregarding individual latency variations. In the average waveforms, individual latency and amplitude variations are intermixed and manifest themselves as a smearing-out of effects. Hence, significant results based on separate analysis of amplitudes and latencies might become less evident in the average waveforms.

the factors "masking" (masked vs. *un*masked T1) and "cluster" (PFC, left TPF, and right TPF). The main effect of "masking" reached significance ($F(1,9) = 6.499$; $P < 0.031$) suggesting that masked T1 was generally processed faster throughout the system than *un*masked T1. A corresponding ANOVA for T2 revealed a significant main effect of "cluster" ($F(1,9) = 4.867$; $P < 0.02$). T2 is processed slower in PFC than in left TPF ($F(1,9) = 9.561$; $P < 0.013$) indicating that T2 processing is especially slowed down at the level of working memory. The main effect of "masking" almost reached significance ($F(1,9) = 4.718$; $P < 0.058$) providing a hint that T2 in masked-T1 trials is processed slower throughout the system than T2 in *un*masked-T1 trials. Hence, the PFC pattern of speeded T1 yet delayed T2 processing with a T1-mask is replicated to a certain degree at all levels of the system involved in the generation of target-related M300 responses.

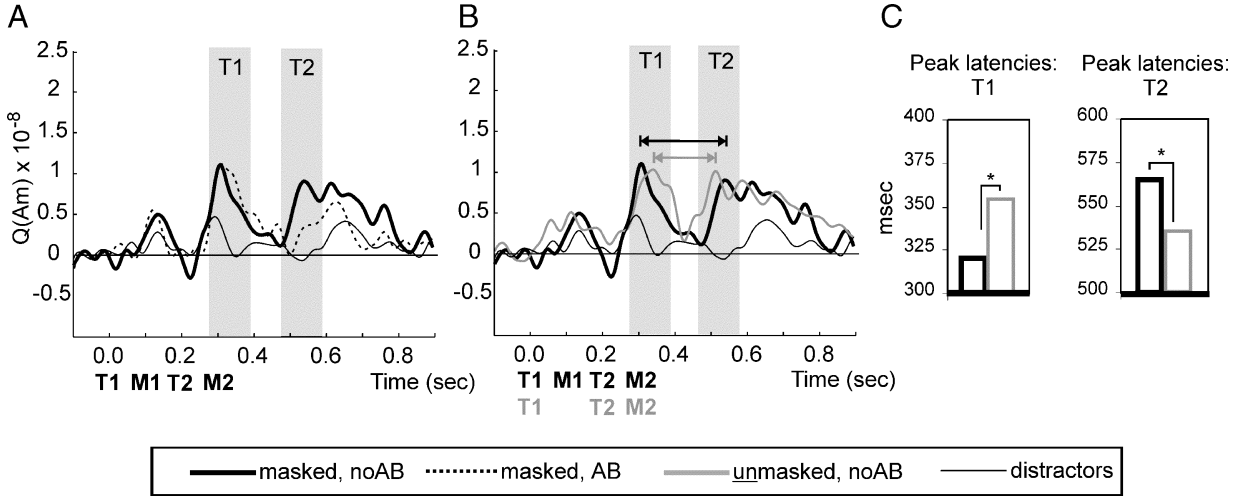

Fig. 6. The specific effects of the T1 mask. Waveforms are shown for the prefrontal (PFC) source cluster, time-locked to the first letter in the RSVP stream (0 ms). The sequence of events on the screen is shown for the dual target conditions (masked: black; *un*masked: gray) below the *x* axes (the position in relation to the *x* axes denotes onset time). The two gray bars in Panels A and B indicate T1- and T2-related M300 components that are referenced in the text. Panel A compares the two masked conditions (masked, AB vs. masked, noAB). Blinked T2s (masked, AB) show a significantly attenuated M300 response (2nd gray bar). Panel B shows a comparison of the masked, noAB condition to the *un*masked, noAB condition. Here, latency differences are observed as indicated by two horizontal arrows (black: masked, noAB; gray: *un*masked, noAB). As the black arrow is longer than the gray arrow, the inter-peak interval in the masked condition is longer than in the *un*masked condition. This in turn is due to accelerated T1, yet delayed T2 with an intervening mask as shown in Panel C, which further specifies the apparent latency differences in Panel B by providing information about mean peak latencies for T1 (left) and T2 (right), respectively. Further explanations in the text.

General discussion

Our primary question of interest was to test whether the data would support the notion of delayed T1 consolidation as the primary cause for the AB. To re-iterate, Chun and Potter (1995) proposed that if target processing is disturbed by a subsequent non-target item then consolidation of this target into working memory would be delayed and subsequent consolidation of targets would be impeded during this delay period (Shapiro et al., 1997). We have tested this hypothesis by comparing T1-related M300 latencies in masked and *un*masked T1 trials and the result was that T1-related M300 peaks were accelerated if T1 consolidation was disturbed by a subsequent non-target item (mask). This strongly contradicts the "delayed T1" hypothesis.

In contrast to the T1-related results and supporting previous findings (e.g., Vogel and Luck, 2002), T2-related M300 peaks were indeed delayed if T1 was masked. Hence, T2 was delayed despite an advantage in processing speed for masked T1 (Fig. 6, Panels B, C). Any theoretical framework that aims at explaining performance deficits in RSVP streams would have to account for this asymmetry. Several accounts predict delayed T2, yet, to our knowledge, speeded T1 processing has not been postulated so far. The framework originally proposed by Raymond et al. (1992), for example, suggests that the potential confusion induced by a mask is noted by the system and then used to initiate a suppressive mechanism to eliminate further confusion. This, in turn, causes an attentional gate, that was opened for T1 processing, to be both shut and locked, making the initiation of the next attentional episode a more time-consuming process than if a locking operation had not been conducted—as in the case of an unmasked T1. The impact of the mask on T1 processing speed is not considered in detail, yet implicitly T1 is assumed to be delayed.

The notion of delayed T2 processing as the main cause for performance deficits is also shared by the account of "temporary loss of control" recently suggested by Di Lollo et al. (2005). Di Lollo et al. propose that the first target task sets up an 'input filter', tuned to the specific detection requirement of the (first) target. Subsequent stimuli that match this filter are passed through for processing, whereas those that do not match require the filter to be reconfigured, which takes time and effort, hence failures on subsequent targets become likely. Similar to Raymond et al. (1992), delayed T2 processing is postulated, yet no explicit predictions are made regarding T1 processing speed. In conclusion, the question remains unresolved of how the accelerating influence of the mask on T1 may be explained, while at the same time preparation for T2 seems to be impeded.

Note that the masking accounts discussed in Introduction may explain the accelerating influence of the mask on T1, yet fail to account for the delay in T2 consolidation. This is, because masking accounts (Bachmann, 1984; Di Lollo et al., 2000; Turvey, 1973) aim at describing the processes that occur when a single mask efficiently extinguishes a single target, while AB accounts in general aim at describing what happens if the mask fails to completely obliterate T1 (T1 should be reportable), that is, what the impact of the residual interference on a subsequent target (T2) is. This also implies a difference between processing stages. Efficient backward masking seems to occur during early perceptual processing, while AB accounts focus on a later stage, that is, consolidation into working memory (e.g., Chun and Potter, 1995; Di Lollo et al., 2005; Raymond et al., 1992; Shapiro et al., 1997; Vogel et al., 1998).

We propose that the 'paradoxical' effect of the mask on T1 and T2 processing at the stage of consolidation may be explained if we switch from a cognitive level of explanation, in terms of competition for limited resources, to a lower level, by taking into account the computational properties of parallel, distributed and recurrent networks that have been introduced as a metaphor for the architecture of the human brain. Rumelhart and McClelland (1986) have pointed out by means of simulated connectionist networks that once parallel distributed and recurrent systems have settled into a stable state, this state is quite difficult to perturb. The stability of an emerging state depends on the signal-to-noise ratio, that is, not only on the strength of "winner"-activation, but also on the strength of suppression allocated to competitors. To return the system to further processing after settling into a stable state, either some sort of self-inhibition (winner) or an automatic system reset needs to be implemented (cf. Schade and Berg, 1992).

In the case of the AB phenomenon, T1 processing might react quite sensitively to a disturbing event (the mask), that is, by suppressing in turn all patterns of activity that do not belong to the T1 pattern, thus increasing the contrast (signal-to-noise) for T1. Although not testing the AB paradigm per se, Loach and Mari-Beffa (2003) provide empirical support for suppression following target consolidation in RSVP, by showing that the post-target item (T1-mask and the next non-target item) reveal negative priming as determined by a probe presented at the end of the trial. This view is also highly compatible with the Desimone and Duncan (1995) neural competition model for visual attention that assumes competition by means of inhibitory interactions. Finally, Houghton and Tipper (1994) describe an implemented connectionist model of selective attention (i.e., negative priming) that makes use of template-based reactive suppression of non-target items below resting level. If such a reactive suppression mechanism is applied to temporal-selective attention in RSVP streams then suppression could be triggered by the post-target item (mask), which interferes with T1 processing. In turn, all patterns of activity that are not compatible with the target are suppressed. If the post-target item (mask) is omitted then no reactive suppression is triggered.

If engaged, such a mechanism would boost T1 processing by enhancing the difference to all other activation patterns, yielding a stable suppressive state (cf. Rumelhart and McClelland, 1986). Accordingly, further input (i.e., T2) is suppressed until such time as this state is perturbed (cf. Schade and Berg, 1992). The longer T2 is delayed, the more likely it becomes that the post-T2 item "substitutes" for T2 (Enns and DiLollo, 2000). We call this explanation the "robust state" hypothesis because suppression of competing patterns leads to boosted T1 processing but also to a robust stable state that is difficult to perturb. The major difference between our account and the "delayed T1" hypothesis is that T1 processing itself is not delayed by the mask (in contrast, it is even boosted), but instead the emerging stable (suppressive) state that impedes processing of T2 is protracted.

It is important to point out, however, that cognitive accounts of the AB, such as the ones advanced by Chun and Potter, 1995; Di Lollo et al., 2005; Raymond et al., 1992; Shapiro et al., 1997, are not generally incompatible with the computational framework proposed by Rumelhart and McClelland (1986). In fact, direct theoretical comparisons are impossible due to differences in the generality, the theoretical constructs and the level of description. The explanation we provide here in terms of stable network states is on a lower level of description. If the

underdetermined notion of "competition for resources" would be substantiated according to the view we have sketched out here than no conflicting predictions would remain. What we have successfully falsified here is 'just' the (implicitly) assumed mechanism for competition, which predicted that competing masks always delay T1 consolidation.[3]

To re-iterate, the data from the present experiment support the "robust state" view in the following way. First, T1 was not delayed by a mask, but instead was processed faster in a brain area associated with working memory (PFC). Second, also in PFC, T1 and T2 were processed rapidly when not in the presence of an intervening mask, whereas the presence of the mask elongated the inter-peak interval between T1 and T2, in concordance with the concept of a robust suppressive state emerging from T1 processing that takes time to be perturbed. Third, speeded T1 yet delayed T2 processing with a mask was found throughout the system with respect to M300 peak latencies. Taken altogether, the "robust state" explanation accounts particularly well for the observed data pattern (Appendix A).

Support for this explanation is also provided by our results from a previous AB study in the MEG, where we specifically analyzed target-related phase synchronization in the beta band (15 Hz) across the attentional network[4] (Gross et al., 2004). Phase synchronization was calculated as phase-coupling between pairs of cortical areas, which provides a measure of the amount of interaction between such areas (see Gross et al., 2004, for details). Moreover, de-synchronization below baseline was taken to reflect the amount of active *de*-coupling between areas. The result of major importance is that Gross et al. report significant de-synchronization which *precedes* synchronization related to reported T2s. De-synchronization is significantly less before non-reportable T2s. This finding suggests that a successful perturbation of the preceding robust state might be indispensable for correct performance on T2. That is, only if the T1-related stable state was effectively de-synchronized (T1 was always masked in this study), T2-related processing (synchronization) could be achieved. Rodriguez et al. (1999) have suggested that de-synchronization (de-coupling) may generally be a necessary stage that allows for the transition from one stable processing state to the next. In the case of the AB task, the transition from T1 to T2 processing state may be impeded by enhanced stability of the first state (T1) that is induced by the intervening non-target item. In addition, effective de-coupling of the target-related network may serve as a mechanism to prevent interfering influences of the mask on target processing.

One of the main implications of the 'robust state' view is that the more efficient the robust state is with regard to T1, the greater the negative consequences for accurate T2 processing will be. A somewhat different formulation of this implication is that if attention is highly focussed on T1, robustness will be enhanced at the expense of T2. It is worth considering whether further existing data would support or contradict this prediction. Subjects who would tend to focus their attention on T1 should show a larger AB than subjects who tend to distribute attention. In a pilot study (Shapiro et al., unpublished results), that employed an SOA of 147 ms, we found that the individual T1 amplitude was correlated with the size of the AB. That is, subjects with a high activation related to T1 showed a higher proportion of blinked trials. This speaks in favor of the prediction that when more emphasis is put on T1 the AB will be larger.

Another piece of evidence comes from investigations on the impact of mood on attention. Euphoric mood seems to bias cognitive processing towards the global shape of visual stimuli while sad or dysphoric mood seems to bias cognitive processing towards local features (Basso et al., 1996; Gasper and Clore, 2002). Moreover, dysphoric mood seems to enforce focussed attention while hampering divided attention (Brand et al., 1997; Riedel et al., 2003; Schmitt et al., 2000). How do these findings relate to the AB and the robust state account? We would expect that under conditions of dysphoric mood (more focussed attention), a stronger and longer lasting AB would be revealed. This is exactly the observation reported by Rokke et al. (2002). Students rated for dysphoric mood showed the following pattern: Nondysphoric and mildly dysphoric participants showed the same size ABs, but the ABs for moderately to severely dysphoric participants were larger and longer.

Correspondingly, we would predict that increased *divided* attention (possibly by inducing euphoric mood) would yield the opposite result: less AB. Preliminary support comes from our informal observation that subjects tended to show less AB if they reported afterwards that they were somewhat unfocussed on the task. This informal observation was also reported by Olivers and Nieuwenhuis (in press). Olivers and Nieuwenhuis (in press) took this as a starting point for their study on the impact of "distraction" on the AB. The major finding was that free association performed concurrently to an AB task (i.e. "think of your holidays" or "think of your shopping plans for a dinner with friends") as well as concurrent listening to music significantly attenuated the AB. The authors also point out that thinking of positive events and listening to music may have induced a positive mood, and hence, a predisposition to divided attention. The major conclusion, however, is that widening the settings of attention seems to attenuate the AB. As a follow-up study Arend et al. (unpublished results) have employed distracting motion in form of a starfield simulation during an AB task. The results replicated the Olivers and Nieuwenhuis findings: visual distraction in form of consistent motion also attenuated the AB. These findings support the robust state hypothesis that predicts attenuated AB with less focussed attention on T1.

There are also other experimental ways to test for this prediction. One could, for example, increase the salience of T1 either by enhancing the perceptual contrast or by putting more socio-emotional emphasis on T1 (i.e. emotional faces). A more salient T1 should increase the AB. Perceptual contrast has been investigated by Hommel and Akyrek (in press) with emphasis on the processes at Lag 1. However, when considering the longer lags, where at least one mask intervenes between T1 and T2, than the results are clear: stronger AB with a perceptually salient T1 (white digit in a stream of black letters).

Similar predictions also hold true for the *T1-mask*. The stronger the interference, by the mask, the stronger the reactive suppression. Target-mask similarity has been investigated in experiment 4 of the Chun and Potter (1995) paper. As expected, a stronger performance deficit (AB) was found at Lags 2 and 3 if the post-target item was more similar to T1, hence, when the maximum S/N ratio was necessary to be achieved.

To summarize, in a parallel architecture such as that found in the human brain, resource limitations emerge from the antagonistic constraints of robustness versus speed of informa-

[3] It is also important to point out that we do not claim that a mask would always accelerate T1. For example, with more complex stimuli, more iterations would be necessary to gather evidence for identity, thus, possibly occluding differences in processing speed between masked and unmasked stimuli.

[4] The attentional network was defined as those cortical areas and interconnections that were primarily related to target and not to distractor processing. Details about the employed procedures are provided in Gross et al., 2004.

tion processing. The use of the AB methodology proved to be fruitful in shedding light on the dynamic equilibrium between these two constraints by putting the system under high processing pressure. We conclude that if the consolidation of relevant information (target) is abruptly disturbed by irrelevant information (mask), the system reacts to this interference by attempting to protect ongoing target processing in order to achieve a robust result, that is, successful target report. However, the more efficient this protection mechanism operates, the more robustness must be traded for speed, with the consequence that subsequent relevant information is unlikely to become consolidated.

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Appendix A

Table A1
Means and standard deviations of peak latencies and amplitudes (measured in ms and nAm, respectively) per cluster as described in the text

Right TPF
M300 peak-to-peak for T1 and T2, gray bars in Fig. 4, Panel A

	Masked				AB			
	T1time	T1ampl	T2time	T2ampl	T1time	T1ampl	T2time	T2ampl
Mean	360	2.51	548	1.35	369	2.69	556	0.85
SD	29.81	1.53	29.66	0.92	26.03	1.27	44.61	0.70
	Unmasked				Distractor			
	T1time	T1ampl	T2time	T2ampl	D1time	D1ampl	D3time	D3ampl
Mean	359	2.99	534	1.42	365	1.92	557	0.89
SD	27.23	2.06	44.66	0.83	31.18	1.57	36.65	0.70

Left TPF
M300 peaks for T1 and T2, gray bars in Fig. 4, Panel B

	Masked				AB			
	T1time	T1ampl	T2time	T2ampl	T1time	T1ampl	T2time	T2ampl
Mean	342	2.03	523	1.67	355	2.29	527	1.85
SD	28.14	1.35	22.07	1.08	47.42	1.45	30.83	1.09
	Unmasked				Distractor			
	T1time	T1ampl	T2time	T2ampl	D1time	D1ampl	D3time	D3ampl
Mean	376	2.20	521	1.83	348	0.95	523	1.18
SD	35.97	1.27	42.32	1.43	31.18	0.57	29.01	0.78

PFC
M300 peaks for T1 (AB + noAB), gray bar in Fig. 5

	Masked		Unmasked		Distractor	
	T1time	T1ampl	T1time	T1ampl	D1time	D1ampl
Mean	331	1.65	373	1.72	323	0.82
SD	43.71	0.91	40.24	1.08	55.98	0.72

M300 peaks for T1 and T2, gray bars in Fig. 6

	Masked				AB			
	T1time	T1ampl	T2time	T2ampl	T1time	T1ampl	T2time	T2ampl
Mean	321	1.92	565	1.76	333	1.90	550	1.10
SD	42.74	1.07	19.31	1.39	39.46	1.10	42.64	1.07
	Unmasked				Distractor			
	T1time	T1ampl	T2time	T2ampl	D1time	D1ampl	D3time	D3ampl
Mean	355	1.70	535	1.84	323	0.82	533	0.56
SD	43.79	1.02	28.02	1.24	55.98	0.72	45.80	0.73

References

Ahonen, A.I., Hämäläinen, M.S., Kajola, M.J., Knuutila, J.E.T., Laine, P.P., Lounasmaa, O.V., 1993. 122-channel SQUID instrument for investigating the magnetic signals from the human brain. Phys. Scr. 49, 198–205.

Arend, I., Johnston, S., Shapiro, K., unpublished results. Does Distraction by Visual Motion Attenuate the Attentional Blink?

Arnell, K.M., Helion, A.M., Hurdelbrink, J.A., Pasieka, B., 2004. Dissociating sources of dual-task interference using human electrophysiology. Psychon. Bull. Rev. 11, 77–83.

Bachmann, T., 1984. The process of perceptual retouch: nonspecific afferent activation dynamics in explaining visual masking. Percept. Psychophys. 35, 69–84.

Basso, M.R., Schefft, B.K., Ris, M.D., Dember, W.N., 1996. Mood and global–local visual processing. J. Int. Neuropsychol. Soc. 2, 249–255.

Blasdel, G.G., Salama, G., 1986. Voltage-sensitive dyes reveal a modular organization in monkey striate cortex. Nature 321, 579–585.

Brand, N., Verspui, L., Oving, A., 1997. Induced mood and selective attention. Percept. Mot. Skills 84, 455–463.

Brehaut, J., Enns, J.T., Di Lollo, V., 1999. Visual masking plays two roles in the attentional blink. Percept. Psychophys. 61, 1436–1448.

Chun, M.M., Potter, M.C., 1995. A two-stage model for multiple target detection in rapid serial visual presentation. J. Exp. Psychol. Hum. Percept. Perform. 21, 109–127.

Desimone, R., Duncan, J., 1995. Neural mechanisms of selective visual attention. Annu. Rev. Neurosci. 18, 193–222.

Di Lollo, V., Enns, J.T., Rensink, R.A., 2000. Competition for consciousness among visual events: the psychophysics of reentrant visual processes. J. Exp. Psychol. Gen. 129, 481–507.

Di Lollo, V., Kawahara, J.-I., Ghorashi, S.M., Enns, J.T., 2005. The attentional blink: resource depletion or temporary loss of control? Psychol. Res. 69, 191–200.

Duncan, J., Ward, R., Shapiro, K.L., 1994. Direct measurement of attentional dwell time in human vision. Nature 369, 313–315.

Enns, J.T., DiLollo, V., 2000. What's new in visual masking. Trends Cogn. Sci. 4, 345–352.

Feinstein, J.S., Stein, M.B., Castillo, G.N., Martin, P., 2004. From sensory processes to conscious perception. Conscious. Cogn. 13, 323–335.

Gasper, K., Clore, G.L., 2002. Attending to the big picture: mood and global versus local processing of visual information. Psychol. Sci. 13, 34–40.

Glahn, D.C., Kim, J., Cohen, M.S., Poutanen, V.-P., Therman, S., Bava, S., Van Erp, T.G.M., Manninen, M., Huttunen, M., Lönnqvist, J., Standertskjöld-Nordenstam, C.G., Cannon, T.D., 2002. Maintenance and manipulation in spatial working memory: dissociations in the prefrontal cortex. NeuroImage 17, 201–213.

Goldman-Rakic, P.S., 1996. Memory: recording experience in cells and circuits: diversity in memory research. Proc. Natl. Acad. Sci. U. S. A. 93, 13435–13437.

Grandison, T.D., Ghirardelli, T.G., Egeth, H., 1997. Beyond similarity: masking of the target is sufficient to cause the attentional blink. Percept. Psychophys. 59, 266–274.

Gross, J., Schmitz, F., Schnitzler, I., Kessler, K., Shapiro, K., Hommel, B., Schnitzler, A., 2004. Long-range neural synchrony predicts temporal limitations of visual attention in humans. Proc. Natl. Acad. Sci. U. S. A. 101, 13050–13055.

Hämäläinen, M., Hari, R., Ilmoniemi, R.J., Knuutila, J., Lounasmaa, O.V., 1993. Magnetocephalography—theory, instrumentation, and applications to noninvasive studies of the working human brain. Rev. Mod. Phys. 65, 413–497.

Hommel, B., Akyrek, E.G., in press. Lag-1 Sparing in the AttentionalBlink: benefits and costs of integrating two events into a single episode. QJ Exp. Psychol. Section a.

Houghton, G., Tipper, S.P., 1994. A model of inhibitory mechanisms in selective attention. In: Dagenback, D., Carr, T. (Eds.), Inhibitory Mechanisms of Attention, Memory and Language. Academic Press, San Diego, pp. 53–112.

Husain, M., Shapiro, K.L., Martin, J., Kennard, C., 1997. Abnormal temporal dynamics of visual attention in spatial neglect patients. Nature 385, 154–156.

Kessler, K., Kiefer, M., in press. Distributing visual working memory: electrophysiological evidence for a role of prefrontal cortex in recovery from interference. Cerebral Cortex.

Kessler, K., Schmitz, F., Gross, J., Hommel, B., Shapiro, K., Schnitzler, A., in press. Cortical mechanisms of attention in time: neural correlates of the Lag1 Sparing phenomenon. Eur. J. Neurosci.

Kranczioch, C., Debener, S., Engel, A.E., 2003. Event-related potential correlates of the attentional blink phenomenon. Cogn. Brain Res. 17, 177–187.

Loach, D., Mari-Beffa, P., 2003. Post-target inhibition: a temporal binding mechanism? Visual Cogn. 10, 513–526.

Marois, R., Chun, M.M., Gore, J.C., 2001. Neural correlates of the attentional blink. Neuron 28, 299–308.

McArthur, G.M., Budd, T.W., Michie, P.T., 1999. The attentional blink and P300. NeuroReport 10, 3691–3695.

Miller, E.K., Cohen, J.D., 2001. An integrative theory of prefrontal cortex function. Annu. Rev. Neurosci. 24, 167–202.

Mottaghy, F.M., Gangitano, M., Sparing, R., Krause, B.J., Pascual-Leone, A., 2002. Segregation of areas related to visual working memory in the prefrontal cortex revealed by rTMS. Cereb. Cortex 12, 369–375.

Nobre, A.C., Coull, J.T., Frith, C.D., Mesulam, M.M., 1999. Orbitofrontal cortex activated during breaches of expectation in tasks of visual attention. Nat. Neurosci. 2, 11–12.

Olivers, C.N.L., Nieuwenhuis, S., in press. The beneficial effect of concurrent task-irrelevant mental activity on temporal attention. Psychol. Sci.

Raymond, J.E., Shapiro, K.L., Arnell, K.M., 1992. Temporary suppression of visual processing in an RSVP task: an attentional blink? J. Exp. Psychol. Hum. Percept. Perform. 18, 849–860.

Riedel, W.J., Sobczak, S., Schmitt, J.A., 2003. Tryptophan modulation and cognition. Adv. Exp. Med. Biol. 527, 207–213.

Rodriguez, E., George, N., Lachaux, J.P., Martinerie, J., Renault, B., Varela, F.J., 1999. Perception's shadow: long-distance synchronization of human brain activity. Nature 397, 430–433.

Rokke, P.D., Arnell, K.M., Koch, M.D., Andrews, J.T., 2002. Dual-task attention deficits in dysphoric mood. J. Abnorm. Psychol. 111, 370–379.

Rumelhart, D.E., McClelland, J.L., 1986. Parallel Distributed Processing: Explorations in the Microstructure of Cognition. MIT Press, Cambridge (USA).

Schade, U., Berg, T., 1992. The role of inhibition in a spreading-activation model of language production, part II: the simulational perspective. J. Psycholinguist. Res. 21, 435–462.

Schmitt, J.A., Jorissen, B.L., Sobczak, S., van Boxtel, M.P., Hogervorst, E., Deutz, N.E., Riedel, W.J., 2000. Tryptophan depletion impairs memory consolidation but improves focussed attention in healthy young volunteers. J. Psychopharmacol. 14, 21–29.

Shapiro, K.L., Arnell, K.M., Raymond, J.E., 1997. The attentional blink. Trends Cogn. Sci. 8, 291–296.

Shapiro, K.L., Hillstrom, A.P., Husain, M., 2002. Neither dorsal nor ventral. Control of visuotemporal attention by inferior parietal and superior temporal cortex. Curr. Biol. 12, 1320–1325.

Stephan, K.E., Marshall, J.C., Friston, K.J., Rowe, J.B., Ritzl, A., Zilles, K., Fink, G.R., 2003. Lateralized cognitive processes and lateralized task control in the human brain. Science 301, 384–386.

Shapiro, K., Schmitz, F., Martens, S., Hommel, B. and Schnitzler, A., unpublished results. The Neural Correlates of Temporal Attention.

Tarkiainen, A., Cornelissen, P.L., Salmelin, R., 2002. Dynamics of visual

feature analysis and object-level processing in face versus letter-string perception. Brain 125, 1125–1136.
Turvey, M.T., 1973. On peripheral and central processes in vision: inferences from an information-processing analysis of masking with patterned stimuli. Psychol. Rev. 80, 1–52.
Visser, T.A., Bischof, W.F., Di Lollo, V., 1999. Attentional switching in spatial and nonspatial domains: evidence from the attentional blink. Psychol. Bull. 125, 458–469.
Vogel, E.K., Luck, S.J., 2002. Delayed working memory consolidation during the attentional blink. Psychon. Bull. Rev. 9, 739–743.
Vogel, E.K., Luck, S.J., Shapiro, K.L., 1998. Electrophysiological evidence for a postperceptual locus of suppression during the attentional blink. J. Exp. Psychol. Hum. Percept. Perform. 24, 1656–1674.
Ward, R., Duncan, J., Shapiro, K., 1997. Effects of similarity, difficulty, and nontarget presentation on the time course of visual attention. Percept. Psychophys. 59, 593–600.