

Bernhard Hommel · Klaus Kessler · Frank Schmitz  
Joachim Gross · Elkan Akyürek · Kimron Shapiro  
Alfons Schnitzler

## How the brain blinks: towards a neurocognitive model of the attentional blink

Received: 20 May 2004 / Accepted: 24 March 2005 / Published online: 20 October 2005  
© Springer-Verlag 2005

**Abstract** When people monitor a visual stream of rapidly presented stimuli for two targets (T1 and T2), they often miss T2 if it falls into a time window of about half a second after T1 onset—the attentional blink (AB). We provide an overview of recent neuroscientific studies devoted to analyze the neural processes underlying the AB and their temporal dynamics. The available evidence points to an attentional network involving temporal, right-parietal and frontal cortex, and suggests that the components of this neural network interact by means of synchronization and stimulus-induced desynchronization in the beta frequency range. We set up a neurocognitive scenario describing how the AB might emerge and why it depends on the presence of masks and the other event(s) the targets are embedded in. The scenario supports the idea that the AB arises from “biased competition”, with the top-down bias being generated by parietal-frontal interactions and the competition taking place between stimulus codes in temporal cortex.

time window of about half a second after onset of T1 (e.g., Broadbent and Broadbent 1987; Raymond et al. 1992). In analogy to an overt blink of the eyes, Raymond et al. (1992) have coined this insensitivity to the second of two sequential targets—attentional blink (AB).

Available accounts of the AB (e.g., Chun and Potter 1995; Duncan et al. 1994; Jolicœur et al. 2000; Shapiro et al. 1994) have linked the effect to capacity limitations of short-term memory or working memory (WM). The general idea underlying these models is that reporting a stimulus presupposes that its sensory representation is transferred to, and consolidated in WM, a process that is assumed to draw on attentional resources. If these resources are allocated to consolidating T1—to a degree that depends on how severely T1 is masked by following items—fewer resources are left to consolidate T2. This makes T2 codes vulnerable to interference from other items competing for representation in WM, so that it is less likely to be maintained and reported later on.

Even though most researchers subscribe to this general characterization of the functional problem underlying the AB, very little is known about the details of this scenario. For example, it is far from clear how the hypothesized consolidation process works or exactly how it prevents temporally overlapping events from being identified, which again makes it difficult to test the proposed models and to relate them to other models and phenomena. The present article makes an attempt to move one step further in conceptualizing AB-related processing limitations by considering recent findings from neuroscientific analyses using brain imaging techniques and patient studies. Given that most available research has been restricted to the visual modality, we will only consider findings on visual processing, also because the behavioral findings are the least controversial for this domain. We will try to substantiate, concretize and, if possible, synthesize (often but not always uncontroversial) functional assumptions by relating them to brain processes, and vice versa—as far as available findings allow. However, given that

### The attentional blink

The number of events we can attend to at the same time is sharply limited, which is nicely demonstrated by a phenomena that occurs in tasks with rapid serial visual presentation (RSVP) of stimulus sequences: when people monitor a visual stream for two targets (T1 and T2), they often miss the second target (T2) if it falls into a

B. Hommel (✉) · E. Akyürek  
Department of Psychology, Cognitive Psychology Unit,  
Leiden University, Postbus 9555, 2300 Leiden, The Netherlands  
E-mail: hommel@fsw.leidenuniv.nl

K. Kessler · F. Schmitz · J. Gross · A. Schnitzler  
Heinrich Heine University, Düsseldorf, Germany

K. Shapiro  
University of Wales, Bangor, Wales

neuroscientific investigations of the AB are still sparse, most of the available evidence must be considered tentative and in need of replication and extension. Accordingly, we are unable to present a full-fledged neurocognitive theory resting on a broad empirical basis. Rather, we suggest a preliminary theoretical framework, a working model that is still speculative in some aspects and in need of further specification in others. Its main function is not to close the case but, on the contrary, to stimulate further research that allows us to develop a more complete theory of the AB in particular, and of the integration of visual information in general.

In the next section we will identify the components that seem to constitute the attentional network producing the AB and consider their functions within the network as well as the way they communicate. Then we go on to set up a neurocognitive scenario describing how the AB emerges and why, for instance, it depends on the presence of masks (Brehaut et al. 1999; Raymond et al. 1992) and the other event(s) the targets are embedded in (Sheppard et al. 2002), before we conclude with a brief summary.

### The attentional network underlying the attentional blink

#### The players

Neuro-imaging and patient studies of the AB and related phenomena have revealed a relatively converging picture of which are the main cortical players involved in attentional selection and, under particular circumstances, in producing attentional limitations. First, information from visual stimuli registered in occipital areas is selectively propagated to the infero-temporal cortex (see Fig. 1). That is, whereas both targets and distractors in a RSVP stream produce systematic activation patterns in the occipital cortex, activation in the temporal cortex mainly reflects task-specific target stimuli only (Kessler et al. 2005a; Shapiro et al., submitted). The infero-temporal cortex is widely assumed to subservise the identification of familiar stimuli (e.g., Milner 1968), which fits with the observation that, in AB studies, the precise location within the temporal lobe varies with the type of stimuli used (e.g., Marois et al. 2004).

A second component has been localized in the posterior-parietal cortex (PPC), commonly in the right hemisphere (cf., Giesbrecht and Kingstone 2004). Using functional magnetic resonance imaging (fMRI), Marois et al. (2000) compared RSVP conditions with high and low interference and observed that the former increased activation in the right intraparietal sulcus—apart from more frontal areas discussed below. Comparable parietal activations related to the difficulty to identify letter targets from RSVP streams have been obtained using fMRI (Marcantoni et al. 2003; Marois et al. 2004) and magnetoencephalography (MEG; Shapiro et al., submitted). These observations fit well with the finding of

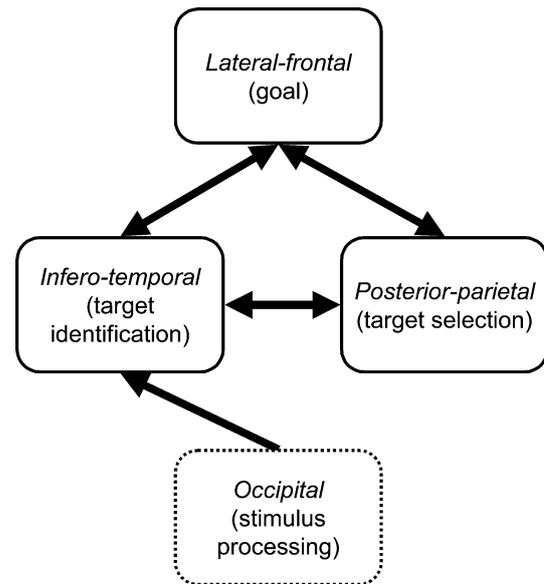


Fig. 1 Components, functions, and main interactions within the attentional network responsible for the attentional blink

Shapiro et al. (2002) that patients with focal lesions at the junction of the superior temporal gyrus and the inferior parietal lobe (IPL) show more of an AB than patients with lesions in the superior parietal lobule.

The right posterior parietal cortex (rPPC) has been associated with visuospatial attention in general and with assigning task relevance to stimuli in particular (Goldberg et al. 2002). More specifically, at least the more ventral areas of rPPC have been suggested to be part of an attentional network subserving top-down control of stimulus processing and identifying targets (e.g., Behrmann et al. 2004; Corbetta et al. 2000; Friedman-Hill et al. 2003; Menon et al. 1997; Wojciulik and Kanwisher 1999). Accordingly, rPPC structures may be conceived of biasing the choice between competing stimulus representations in the infero-temporal areas, presumably in interplay with frontal areas (see below).

Finally, several studies have identified a third component localized in the lateral-frontal cortex. In particular, activation of lateral-frontal areas is associated with selection problems induced by temporally close distractors (Marois et al. 2000) or competing targets (Feinstein et al. 2005; Marcantoni et al. 2003; Marois et al. 2004). Some studies have also provided evidence for an involvement of the anterior cingulate in the AB task, that is, success in reporting a temporally close T2 was found to be associated with increased cingulate activation (Gross et al. 2004; Marois et al. 2000, 2004). Indeed, the frontal cortex is known to be involved in the control of multiple-task performance (Adcock et al. 2000; D'Esposito et al. 1995; Szameitat et al. 2002) and to be particularly sensitive to the temporal overlap of tasks (Richer et al. 1998; Richer and Lepage 1996). Along the lines of Desimone and Duncan's biased-competition model (Desimone and Duncan 1995; Duncan 1996), the role of the frontal

component may thus be conceived of as maintaining the task goal including a functional description of the target(s), which then provides top-down support for stimuli matching this description (see Fig. 1).

### The interplay

We now have identified three anatomically and functionally specified components that we, as other neurocognitive AB researchers, consider to be involved in both solving attentional selection problems and—under unfavorable circumstances—creating the attentional bottleneck expressed as the AB. However, to speak of an attentional network needs more than identifying components and making sure that they are co-activated with the same task. For instance, conventional fMRI studies (not enhanced by EEG) integrate activation changes across time intervals of tens or hundreds of trials and even event-related fMRI has integration windows of more than 1 s. This means that even though two structures may show reliable activation changes within the same condition and interval, the timepoints of their activities may lie hundreds or even thousands of milliseconds apart. Accordingly, coactivation of cortical structures in studies using imaging techniques with a temporal resolution as low as fMRI is a necessary but by no means sufficient condition for considering them as components of a common neural network—so that previous claims that such a network has been demonstrated (e.g., Marcantoni et al. 2003; Marois et al. 2000) are actually not substantiated by the data they are based on.

Apart from coactivation in the same condition one would require the components of a neural network to fulfill at least two further requirements: their activation should overlap in time and be contingent on each other. Evidence suggesting that the first condition is met comes from a recent AB study of Kessler et al. (2005a). In this MEG study, subjects were presented with four-letter stimuli in a row: T1, an irrelevant letter to mask T1, T2, and another irrelevant letter. Figure 2 provides an overview of the averaged waveforms for each of the four cortical clusters analyzed. First, it is easy to see that the occipital source is activated earlier and less selectively (i.e., it responds to target and distractors equally strongly) than the other three sources, suggesting that targets were filtered out after occipital processing. Second, and more importantly, we see that all three remaining sources—which correspond to the components we have identified above—show similar activation patterns and patterns that clearly overlap in time. Obviously, then, the structures producing these activations were processing target information concurrently.

Evidence suggesting that the second condition for demonstrating a neural network is also met comes from an analysis of neural synchronization in an AB task reported by Gross et al. (2004). Gross et al. (2004) compared time-frequency representations (TFRs) of target- and distractor-related neural activity (as mea-

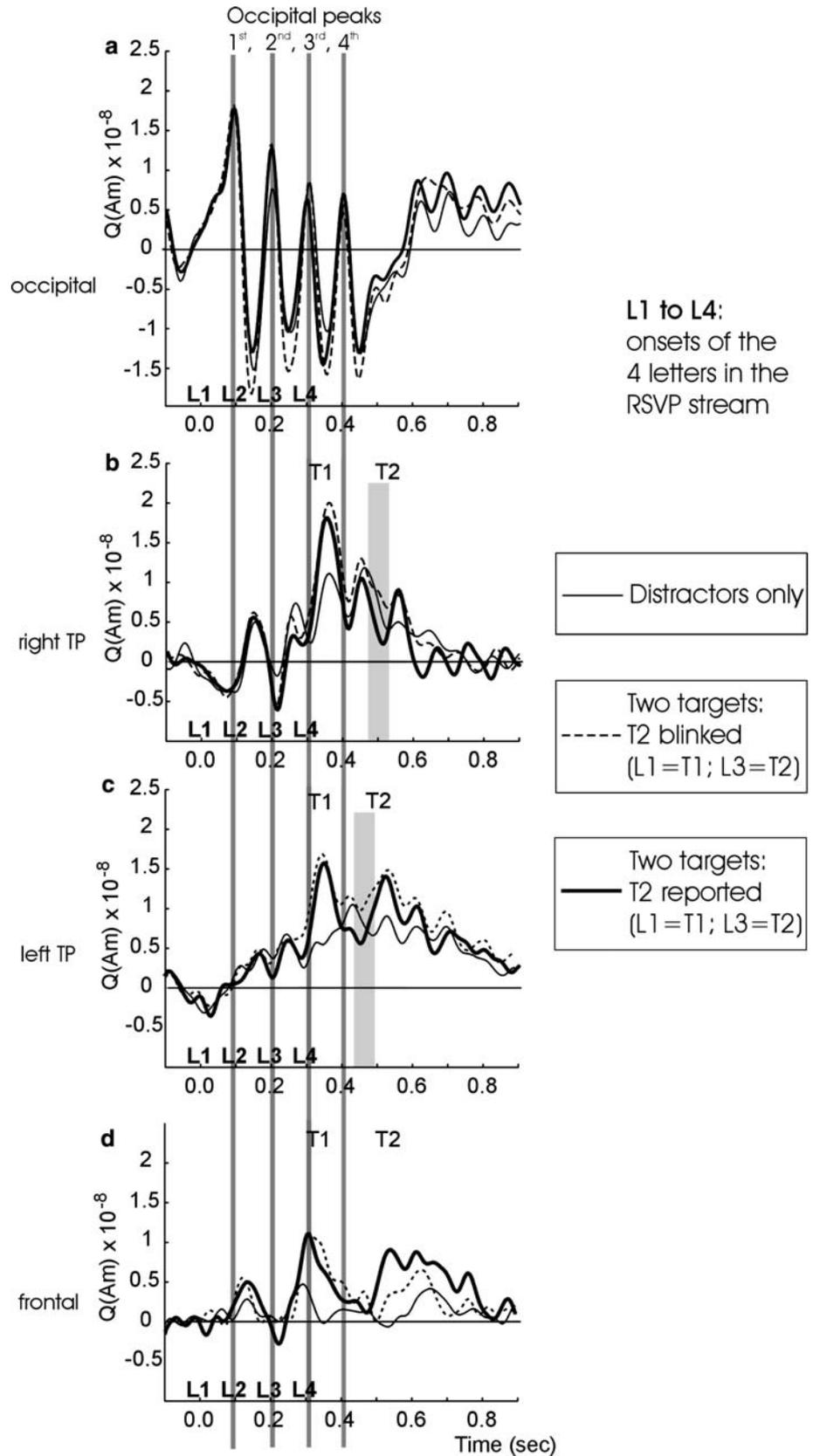
sured by MEG) related to successful report. Subtracting the latter from the former, so to eliminate components common to target and distractor processing, revealed strong neural activity in the beta-band (13–18 Hz) at a time of about 400 ms after target onset.<sup>1</sup> Eight cortical sources contributed significantly to neural oscillation: occipital, temporal left and right, posterior parietal left and right, frontal left and right, and cingular areas.

In the next step, Gross et al. (2004) assessed the neural coupling between these sources by calculating the phase synchronization index (SI).<sup>2</sup> Inspection of the time courses of SI in the trials with successful report of both targets revealed that the coupling between the eight sources fall into two categories: one type of connection shows a modulation at the rate of stimulation (7 Hz or every 143 ms), while the other exhibits two maxima separated by about 292 ms, which was the delay between T1 and T2. That is, some connections were apparently concerned with any stimulus that appeared, whereas others were involved in processing task-relevant information only. Figure 3 (left panels) shows an example of a typical stimulus-related connection, with SI peaks reflecting the rate of stimulus presentation (Fig. 3a) and auto-correlations sensitive to both target and distractors (Fig. 3b). In contrast, the SI pattern for target-related connections (Fig. 3, right panels) is dominated by two peaks separated by about 292 ms—the lag between T1 and T2—and is revealed by the autocorrelation. Most interestingly, distractor-related connections primarily link the occipital cortex to left-hemispheric areas (Fig. 3c, left panel), whereas target-related connections link the right PPC with the cingulum, the left temporal and with the frontal regions (Fig. 3c, right panel). These findings provide the first insights into the actual dynamics of an attentional temporo-parietal-frontal network in demonstrating that the suspected cortical players: (a) play at the same time and (b) apparently play with each other to achieve a common goal, i.e., the selection of the task-relevant stimulus event.

<sup>1</sup>To be more precise, phase synchronization was observed in the beta-band at a frequency of about 15 Hz. Beta synchronization is known to play an important role in attentional processes in general (Liang et al. 2002; Wrobel 2000) and in coupling temporal and parietal areas during object processing in particular (Von Stein et al. 1999). Also, simulation studies show that the beta frequency has characteristics that are favorable for long-range interactions, in contrast to the gamma frequency band that is optimal for local processing (Bibbig et al. 2002; Kopell et al. 2000). However, note that the frequency of 15 Hz is close to the first harmonic of the stimulus presentation frequency in the study of Gross et al. (2004) (6.85 Hz), which may suggest that synchronization frequencies are not specific to the operation mode of the communicating network but to the temporal characteristics of the events the communication refers to. In any case, the findings of Gross et al. (2004) do not support the claims of Dehaene et al. (2003) and Fell et al. (2002) that gamma-band oscillations play a crucial role in the AB.

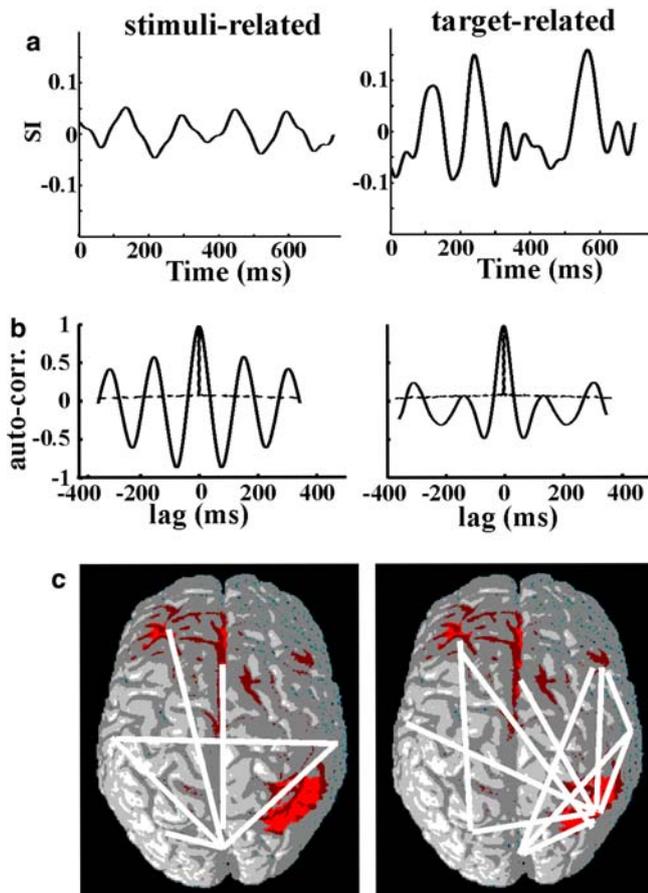
<sup>2</sup>The SI quantifies the phase coupling between different regions. It is computed as the absolute value of the sum of the complex phase differences of both regions divided by the number of epochs and is bounded between 0 (indicating no phase locking) and 1 (indicating perfect phase locking). For further details, see Gross et al. (2004).

**Fig. 2** Source waveforms from occipital (a), right temporo-parietal (b), left temporo-parietal (c) and frontal (d) areas for Lag-2 trials (T1–T2 SOA = 200 ms, ISI = 150 ms), adapted from Kessler et al. (2005a). Early biphasic responses to each of the four letters in the RSVP stream (*letter onsets* are indicated by “L1” to “L4” on the x-axis) are observed in occipital areas that do not differ significantly between distractors and targets. The peaks of the first components of each occipital response are faster than responses evoked in the other three areas as indicated by the *four grey lines* that cut through all panels. In right temporo-parietal areas target-related M300 components (*grey bars*) are observed on-top of regular biphasic responses (best seen in the distractor condition) that mirror the occipital pattern (b). The T2-related M300 peak does not differ in absolute amplitude among blinked and reported targets. However, a stronger decay (*darker grey bar* embedded in the T2-related *grey bar*) is observed preceding the T2-related M300 peak of reported targets (*thick black line*). This T2-related pattern is similar for left temporo-parietal areas (c). Finally, the T2-related M300 component is significantly attenuated for blinked targets in frontal areas (d). In addition, the T1-related M300 peak occurs significantly earlier if T2 can be reported than on trials where T2 is blinked. Furthermore, in noAB trials (*thick black line*) frontal M300 activation related to T1 reaches its peak first (d), i.e., earlier than left (c) and right (b) temporo-parietal activation, possibly suggesting top-down modulation by frontal areas



In order to understand how this goal is achieved, Gross et al. (2004) looked into the temporal dynamics of the SI in trials when dual-target interference prevented

the second target from being reported (AB) as compared to trials when it could be reported (noAB). This analysis revealed two significant effects: first, as Fig. 4a shows,



**Fig. 3** Classification of stimulus-related and target-related connections. **a** Synchronization Index (SI) averaged across all subjects for a typical stimulus-related (*left* occipital to cingulum) and a typical target-related (*right* frontal-left to posterior-parietal-right) connection. The SI index quantifies, at each time point, the regularity of phase relationship between two channels in the selected band (here roughly 13–18 Hz), irrespective of their amplitude and of the actual dominant phase difference between the two channels. **b** Auto-correlations of the SI index were computed for the timecourse of synchronization for each pair of connections. The autocorrelation quantifies the correlation of the timecourse with itself after a shift of “lag” milliseconds (lag being the number displayed on the *x*-axis). Peaks in the autocorrelation indicate increased similarity at the given lag. If the SI is reactive to each stimulus we would expect an increased autocorrelation at a lag of 146 ms and multiples. If it is reactive mainly to targets (separated by 292 ms) we would expect an increased autocorrelation at a lag of 292 ms. Thus, the autocorrelation served as a tool to classify SI timecourses between different areas as stimulus or target related. Hence, connections showing a significant peak at 146 ms were classified as stimulus related (the *left figure* shows an example, occipital to cingulum) and connections showing a significant peak at 292 ms were classified as target-related (see *right panel* for an example, frontal-left to posterior-parietal-right). The *dashed line* represents the 99% confidence limit of the SI time course. The confidence levels for the autocorrelation were computed using 1,000 random permutations of the SI timecourses. The order of points for each timecourse was randomly modified during each permutation. The 99th percentile establishes the confidence level of the null hypothesis that peaks in the autocorrelation are not due to the temporal structure of the SI timecourse. **c** The stimulus-related (*left*) and the target-related (*right*) network is shown

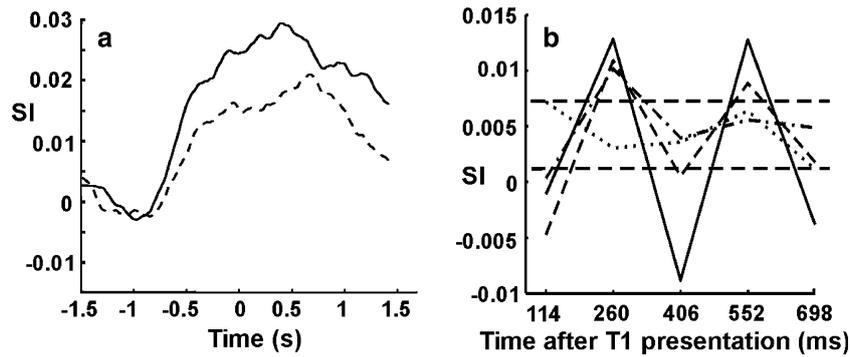
phase synchronization in noAB trials (solid line) was significantly enhanced as compared to AB trials (dashed line). This suggests that the preparedness or “vigilance” of the whole attentional network fluctuates throughout the task, with lower degrees of preparedness leading to a more pronounced AB.

Second, the temporal modulation of synchronization by the targets and their masks is more pronounced in noAB than in AB trials. As shown in Fig. 4b, the noAB function deviates from the AB function in two ways. One relates to the targets: whereas T1 induces strong increases in synchronization in both noAB and AB trials, the T2-induced increase is much less pronounced in AB trials. In fact, noAB trials exhibit comparable peaks for both targets, while the T2 peak just reaches significance in AB trials. The other deviation relates to the distractors: in noAB trials they are in all positions (pre-T1, pre-T2, and post-T2) associated with substantial sub-baseline decreases of synchronization, whereas only weak decreases are obtained in AB trials. This pattern suggests that the network is able to temporarily disassemble itself, that is, to inhibit communication among its members in response to the appearance of task-irrelevant information. As Gross et al. (2004) suggest, this self-desynchronization before a target may represent a processing mechanism that frees or reserves attentional resources for target processing and protects the target against interference at the same time.

## A neurocognitive scenario

Having reviewed evidence for the existence of an attentional temporo-parieto-frontal network, and a central role of it in the AB, let us now consider exactly how the AB may emerge—or be prevented under favorable conditions. It seems obvious, especially when inspecting Fig. 2, that stimulus processing begins with a rather nonselective stage associated with the occipital cortex. Here, distractors are coded just as strongly as targets, while lag effects are absent. This provides further support for the claim that the AB does not reflect purely visual, sensory processes—or process limitations—but the characteristics of later, attentional selection mechanisms (Chun and Potter 1995; Raymond et al. 1992; see the overviews of Jolicœur et al. 2002; Shapiro 2001).

The first processing stage where the task relevance of stimuli affects the strength of their representation is associated with the temporal cortex. Here, stimuli are not only identified (as obvious from the fact that different types of stimuli activate different, content-specific areas within the temporal cortex, Marois et al. 2004) but they are also targeted by top-down modulations from lateral-frontal areas and rPPC. This suggests that temporal areas represent a kind of workspace or desktop, where bottom-up information and top-down prefer-



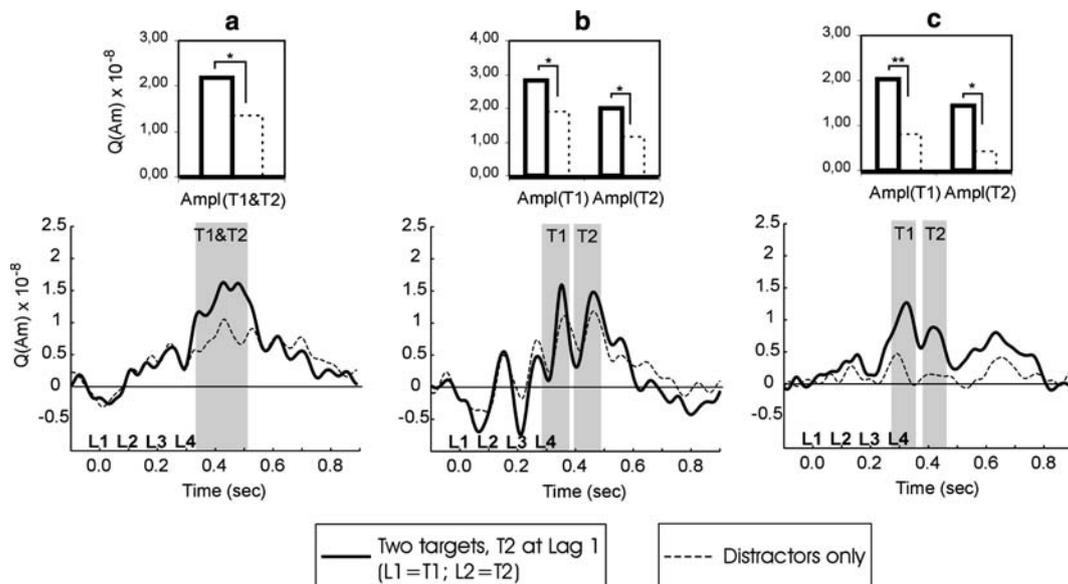
**Fig. 4** Synchronization (SI) in the target-related network. **a** The “noAB” condition (*solid line*) shows a stronger SI during stimulus presentation compared to the “AB” condition (*dashed line*). 0 ms corresponds to the onset of the first target. The beginning of the letter stream ranges from  $-880$  to  $-580$  ms. The SI time courses were smoothed with a Savitzky-Golay filter (polynomial order: 3, frame length: 600). **b** SI for the components of five successive stimuli. Zero on the  $x$ -axis corresponds to a 60 ms long window

centered at 260 ms after presentation of the first target. The other windows were shifted by multiples of the SOA (146 ms). Position 2 corresponds to the target component of the second target (for AB and noAB condition). For each window the mean SI is shown. Conditions are pattern-coded (noAB: *solid*; AB: *dashed*; target: *dash-dot*; distractor: *dotted*). The dashed horizontal lines mark the extent of SI in trials containing only distractors

ences converge, just as sketched in Fig. 1. The central question is, then, why and under which circumstances would this convergence sometimes not lead to a successful report of a target, hence, why does the AB occur?

Luck et al. (1996) provided first evidence that the AB is unlikely to reflect problems in identifying T2 at short lags: even unreported T2s induce the N400, an electrophysiological marker of semantic mismatch, which means that targets undergo semantic analysis whether they can be reported or not. Another piece of evidence speaking against an identification-related processing bottleneck stems from Kessler et al. (2005b), who ob-

served performance in lag-1 conditions (i.e., T2 immediately following T1) in the MEG. As shown in Fig. 5, T2 induced markedly different activation patterns in the three analyzed loci: both targets produced distinct, temporally clearly separated peaks in the frontal cortex and the right temporo-parietal cluster, whereas T1 and T2 activation curves are smeared in the left temporo-parietal cluster. As Kessler et al. (2005a, b) discuss, this is consistent with the assumption that identification processes for the two targets overlap in time, suggesting that the left temporal cortex can identify more than one target at a time (see below). Along these lines one would



**Fig. 5** Source waveforms from left temporo-parietal (**a**), right temporo-parietal (**b**) and frontal (**c**) areas for Lag1 trials (T2 immediately following T1; T1–T2 SOA = 100 ms, ISI = 50 ms), adapted from Kessler et al. (2005b). Letter onsets are indicated by “L1” to “L4” on the  $x$ -axis. At the top of each panel a graph compares amplitude means in the Lag 1 and in the distractor condition. Asterisks denote the 5% (*single*) and the 1% (*double*)

significance levels. In left temporo-parietal areas (**a**) only one M300 component for T1 and T2 is observed that might reflect a single or two overlapping target-related processes. In right temporo-parietal areas (**b**) two target-related M300 components (*grey bars*) are observed on top of regular biphasic responses that mirror the occipital pattern (cf. Fig. 2). In PFC (**c**) two distinct target-related M300 components are the dominant waveform patterns

also expect that the transmission of information from occipital to temporal areas is relatively unaffected by the identity of the incoming stimuli and the success of target report, while the actual outcome of matching should reflect target specificity. Indeed, occipital–temporal synchronization is observed for distractors as well as for targets (Gross et al. 2004), while the amplitude of evoked responses within the temporo-parietal areas reflects target-related processing irrespective of the behavioral outcome (Kessler et al. 2005a).

If identification is not the bottleneck, what else might it be? Several authors have suggested that reporting a target might presuppose that it has previously been re-coded into a more durable format—that is, consolidated into WM—and this consolidation process might be strictly capacity-limited (Chun and Potter 1995; Jolicoeur and Dell’Acqua 1998). Although this account is consistent with a number of findings (for an overview, see Jolicoeur et al. 2002), the concept of consolidation is barely understood and why consolidation should be capacity-limited remains a mystery. So, what do we know about short-term target consolidation or, more generally, about the process that is responsible for the AB?

One systematic feature of the sought-for bottleneck process is that its successful completion seems to produce a P300 potential in the EEG. In trials with successful report of both targets, each target produces a positively peaking evoked response potential (ERP) occurring approximately 300 ms after target onset (Luck et al. 1996; McArthur et al. 1999)—a so-called P300 (Donchin 1981). Interestingly, however, this P300 component has been found to be largely or completely suppressed in trials where T2 is “blinked”, hence, cannot be reported (Dell’Acqua et al. 2003; Kranczioch et al. 2003; Luck et al. 1996; McArthur et al. 1999; Rolke 2001; Vogel and Luck 2002). Comparable observations could be made in MEG analyses, where M300, the magnetic equivalent of the P300, is also attenuated or even suppressed in blink trials (Kessler et al. 2005a; Shapiro et al., submitted).

In general, T2s that can be reported also produce a P300, irrespective of whether the T1–T2 lag is long or short. However, if T2 is not masked (which yields almost perfect T2 report), reducing the lag leads to a delay of the P300 (Vogel and Luck 2002). This implies that the processes that underlie the P300 for T1 and T2 cannot overlap in time and, hence, represent an attentional bottleneck. One of the generator sites of the P300 component seems to be the temporo-parietal junction (TPJ; Downar et al. 2001), which Shapiro et al. (2002) have found to be implied in the AB and which Behrmann et al. (2004) have claimed to be a mediator of selecting visual events. In other words, support from TPJ (and/or other, close-by rPPC systems) seems to be crucial for consolidating a target event but this support can be provided for only one event at a time.

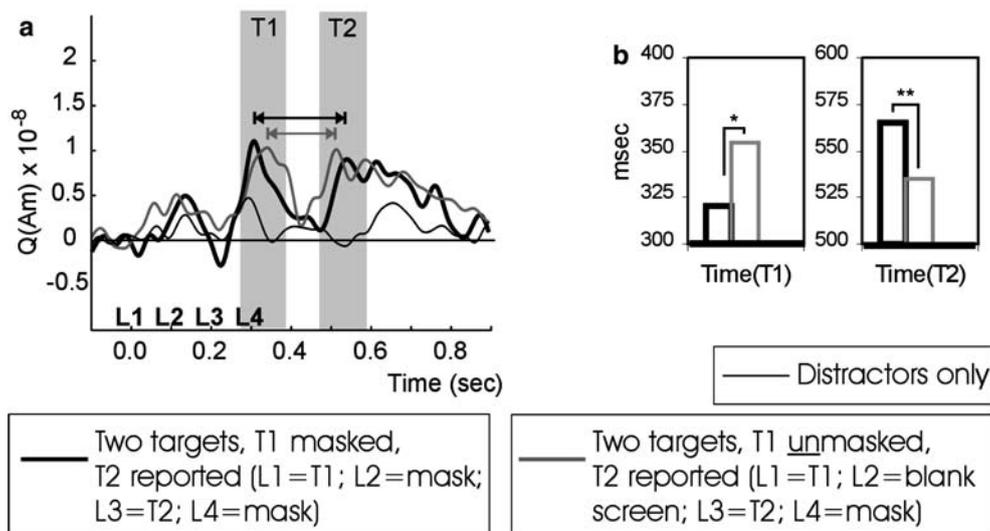
However, there are several indications that TPJ is not the only source associated with the bottleneck. First, a

conscious report of stimuli is not only correlated with rPPC activation but with lateral–frontal activation as well (Dehaene et al. 2001; Marois et al. 2004). In fact, the report success seems to be even more strongly related to frontal than to parietal activation (Kessler et al. 2005a; Marois et al. 2004). Moreover, the timing of the frontal M300 (the magnetic P300) is tightly associated with the incidence of an AB (Kessler et al. 2005a): the T1-induced frontal peak occurs earlier in no-blink than in blink trials (see Fig. 2), which might suggest that completing the processing of T1 earlier helps to overcome the blink. Indeed, behavioral studies provide evidence that the size of the blink is positively correlated with the duration of T1(-related) processing (for an overview, see Jolicoeur et al. 2000). Moreover, M300 activation in the no-blink trials reaches its peak in frontal areas first, i.e., earlier than in left and right temporo-parietal areas (Fig. 2), which points to a crucial role of frontal mechanisms in successful target consolidation. Second, observations in a split-brain patient show that the AB is much stronger if T2 is presented to the right than the left hemisphere—which reinforces the idea that rPPC mechanisms participate in the constitution of a bottleneck—but the AB is still reliable in the latter case (Giesbrecht and Kingstone 2004). And, third, the synchronization analyses of Gross et al. (2004) reveal a very tight linkage of lateral–frontal cortex and rPPC, suggesting that these two components act as a functional unit.

Taken altogether, it seems unlikely that it is one single cortical structure or system that constitutes the processing bottleneck leading to the AB. Rather, the bottleneck seems to be created by the very fact that several components interact as a network that, as Fig. 4 suggests, has the tendency to “silence itself”, presumably in an attempt to preserve the outcome of target selection. By focusing the action of the whole attentional network onto one given target event, the cognitive system creates discontinuous integration episodes (or object files: Shapiro 2001; cf., Hommel 2004) that include all the information within the integration window and exclude—even actively suppress—information that falls outside the window. In other words, attentional selection for action is intrinsically exclusive—not because one component or another would be serial by nature but because the communication within the network is a part of and can refer to only one topic at a time.<sup>3</sup>

Considering that the incoming information is parsed into discrete episodes raises the question of how long

<sup>3</sup>The functional reason for why the system is restricted to, or at least better off focusing communication on one topic at a time may be that this solves one of the many binding problems (Treisman 1996) that distributed systems face. Technically speaking, it may well be possible that different subgroups of codes lead concurrent “private discussions” (to stay with the communication metaphor) but that would make it very hard for a global operation to tell relevant discussions (the outcome of which needs to be considered) from useless babble. This is why members of parliaments commonly agree on sequentially organized contributions.



**Fig. 6** Source waveforms from frontal area (**a**) for Lag-2 trials (T1–T2 SOA = 200 ms, ISI = 150 ms), adapted from Kessler et al. (2005a). Event onsets are indicated by “L1” to “L4” on the x-axis. Two major conditions are compared: two targets with an intervening mask or without an intervening mask—in both conditions only trials with correct report on both targets are

considered here (which is why the T2-related M300 is not suppressed). As specified in (**b**), T1 with a subsequent mask is processed significantly faster than T1 without, whereas T2 with a preceding mask is processed significantly slower than T2 without. Asterisks denote the 5% (single) and the 1% (double) significance levels

these episodes are. One possibility is that all episodes are of the same, uniform length—a kind of information processing quanta (Kristofferson 1967; Pöppel 1997). If so, the number of successive stimuli that can be reported would depend on whether or not they fall into the same quantum. This would explain why the AB often spares the first lag, that is, why performance on T2 is often more or less preserved if it immediately follows T1 (instead of a T1 mask; lag-1 sparing: Potter et al. 1998; Visser et al. 1999). It would also explain why lag-1 sparing is commonly accompanied by the loss of information about the order of the targets (Hommel and Akyürek 2005). If such quanta would really be invariant, we would need to assume that they cover the time span of about one lag, which judging from the most common AB designs should lie in the order of about 100 msec. However, such an estimate is difficult to combine with the finding of Di Lollo et al. (2005) (see also Di Lollo, this issue), that subjects can process at least three targets in a row, as long as no nontarget appears in between. In other words, a target at lag 2 can be successfully reported if lag 1 also contains a target, but is missed if lag 1 contains a nontarget—even though the memory load should be higher in the former than the latter case.

These and other observations suggest that the size of an integration window is variable (Jolicœur et al. 2002; Lupiáñez and Milliken 1999; Raymond 2003; Sheppard et al. 2002), which however raises the question by which factors the effective size is controlled. One such factor may be the presence or absence of goal-irrelevant information: the presence of the target may open an attentional gate (Raymond et al. 1992), which is then closed upon occurrence of the first nontarget (Di Lollo et al.

2005). Nontargets may trigger the closure of the gate in at least two (nonexclusive) ways, by mismatch and/or by episodic retrieval. The mismatch principle may work by matching the incoming information against the target template(s) stored in the working memory, a match signaling to open the gate (or keep it open) and a mismatch to close it. Episodic retrieval may work by binding stimulus representations to task sets in such a way that the reappearance of a stimulus reactivates the previously associated task set (Waszak et al. 2003, 2005, in press). In a RSVP task with fixed target and distractors sets, this would induce bindings between targets and a task set that enables subjects to select, consolidate, and report these targets, and bindings between distractors and whatever task set or cognitive state enables subjects to exclude the available input from processing. Each target would thus activate or, in the case of more than one successive target, further strengthen the already activated “select, consolidate, and report” task set, whereas the appearance of a distractor would trigger the “ignore” task set (cf., Di Lollo et al. 2005, for a related account that however leaves open why and how distractors undermine the current task set). In any case, there are reasons to speculate that the distractor-induced closure of the gate is detected first by the frontal cortex: the frontal area shows the earliest peaks of target-related activation (Kessler et al. 2005a; see present Fig. 5) and a phase lead over other areas when changes in the synchronicity within the attentional network take place (Gross et al., submitted). The occurrence of a mask may thus induce the following sequence of events: detection of a target-template mismatch or code competition in temporal areas may signal a need for support to frontal, goal-related systems

(explaining early frontal peaks), which then provide more support than they would have otherwise. As a consequence, T1 processing is successful but the suppression of competing stimulus codes is more severe than in the absence of competition.

The idea that two or more targets can be processed in parallel, if they only fall into the same, to some degree variable integration window, accounts for a number of recent findings from behavioral and neurophysiological studies. For instance, it fits with Kessler et al.'s (2005b) observation of overlapping M300 peaks for the two targets in an area involved in stimulus identification (Fig. 5). It also fits with the outcome of a related study that looked into the impact of masks on T1 and T2 processing (Kessler et al. 2005a). As we mentioned earlier, T2 is often missed and the related M/P300 is heavily suppressed if only a single distractor intervenes between T1 and T2 (Fig. 2, Panel D). But let us focus on the successful trials, that is, the trials in which the mask did not succeed in preventing T2 report (Fig. 6a, black line, T1 masked), and compare them with successful trials in which no distractor intervened between T1 and T2 (Fig. 6a, grey line, T1 unmasked). Unsurprisingly, we see that the amplitude of the M300 is unimpaired for both trial types, but we also see that the T1- and T2-related peaks differ in time: they are less separated, i.e., T1 activation peaks later and T2 activation earlier, if no mask occurs. This seems to indicate that the processing of T2 overlaps to some degree with that of T1, which delays completion of the latter and speeds up the former. In other words, the absence of an intervening nontarget allows for parallel processing even if the two targets are temporally separated by a lag (i.e., T2 occurs at lag 2). The observation that this parallel processing delays the T1 peak suggests that falling into the same integration window has not only benefits (for T2) but also costs (for T1). Hence, parallel processing creates competition between the two targets. Indeed, behavioral studies have shown that lag 1 does not only spare T2 but also tends to impair performance on T1 (Broadbent and Broadbent, 1987; Hommel and Akyürek 2005; Potter et al. 2002), and this trade-off is stronger the easier the discriminability of T2 and the more difficult the discriminability of T1 is Hommel and Akyürek (2005).

However, according to our considerations regarding the closure of the T1 integration window due to the intervening distractor (cf. Raymond et al. 1992; see also Di Lollo et al. in this special issue) it is worth considering a potentially accelerating influence of the mask on T1 processing (cf. Kessler et al. 2005a), as suggested by the faster peak for masked T1s in Fig. 6. It has been reported (e.g., Turvey 1973) that a mask may terminate target processing instead of delaying it (cf. Chun and Potter 1995). If this was the case then delayed T2 processing would emerge from the necessity to bring the system back into a state where further targets could be processed, i.e., where a new integration window could be opened (Raymond et al. 1992; Kessler et al. 2005a; see also Di Lollo et al. in this special issue). In support,

Gross et al. (submitted) observed a top-down flow of information from left frontal to right PPC prior to target-related enhanced synchronization.

The available evidence clearly points to a bottom-up component in determining the temporal size of the effective integration window. However, this does not exclude that top-down factors are operational as well. Lupiañez et al. (1999; Lupiañez et al. 2001) have suggested and provided evidence that subjects can adjust the duration during which information about a given visual object is collected and when the “object file” holding this collection is closed. For instance, they demonstrated that the transition from priming by location repetition to inhibition of return (i.e., impaired performance if location is repeated) occurs earlier in time when interfering distractors are present than when there are not (Lupiañez et al. 2001). It thus seems possible that the mere expectation of the presence or absence of a distractor affects the time taken to integrate information about a target, suggesting that people are able to control the size of the temporal integration window used to construct object files. Consistent with this assumption, Toffanin et al. (submitted) observed more target-order reversals (indicative of parallel processing of T1 and T2) for lag 1 when the subjects expected a slow RSVP stream than when they expected a fast stream. Given that changes in the synchronicity of the attentional network are sensitive to stimulus-related expectation (Gross et al., submitted), this may indicate that people are able to set their network to create short- or long-attentional episodes.

In sum, then, the consolidation bottleneck is not a direct function of the relative timing of T1 and T2. Rather, consolidation processes are sensitive to intervening events that might disturb ongoing target processing, and they prevent such disturbances by suppressing the corresponding informational sources or, more precisely, by inhibiting communication about these sources among the components of the attentional network (Gross et al. 2004). If the lag between T1 and T2 is short, T2 is more likely to suffer from this suppression and, hence, cannot be reported. Even if it can be reported, such as when the mask following it is omitted, the processes responsible for its consolidation are delayed—as is the associated P300 (Arnell et al. 2004; Vogel and Luck 2002).

---

## Summary

According to our preliminary neurocognitive model the visual AB emerges from a functional bottleneck that renders an actually parallel processing system effectively serial. After unlimited, nonselective processing in specialized perceptual modules in the occipital cortex, raw feature conjunctions are fed into object-specific temporal areas, where the perceptual events are matched against long-term knowledge and identified. Identified objects are then matched against target template(s) maintained in WM (driven by lateral-frontal structures and mediated by TPJ) and they receive top-down support to a

degree determined by that match (Bundesen 1990; Duncan and Humphreys 1989). Support is provided by entraining the firing patterns coding the target and synchronizing them with the relevant structures in frontal and parietal cortex or, reversely, by using temporal characteristics of the target event to entrain the frontal and parietal structures (in case that the relationship between stimulus rate and synchronization frequency observed by Gross et al. 2004, was no coincidence). This synchronization stabilizes the representation of the supported target and increases its competitive strength, which again increases the likelihood that it gets access to action control, such as verbal report. If another stimulus happens to appear while the attentional network is busy with synchronizing, it does not only fail to receive any support, it also suffers from top-down contrast enhancement to preserve ongoing target consolidation. Hence, targets immediately appearing after a distractor interfered with T1 are likely to get “blinked”.

**Acknowledgements** Support for this research by Volkswagenstiftung in the form of a project grant to BH, KS, and AS is gratefully acknowledged. We are also grateful to Andre Achim, Pierre Jolicœur, and an anonymous reviewer for helpful comments on a previous version of this paper. Correspondence and requests for materials should be addressed to Bernhard Hommel, Leiden University, Department of Psychology, Cognitive Psychology Unit, Postbus 9555, 2300 RB Leiden, The Netherlands; hommel@fsw.leidenuniv.nl.

## References

- Adcock RA, Constable RT, Gore JC, Goldman-Rakic PS (2000). Functional neuroanatomy of executive processes involved in dual-task performance. *Proc Natl Acad Sci USA* 97:3567–3572
- Arnell KM, Helion AM, Hurdelbrink JA, Pasiaka B (2004). Dissociating sources of dual-task interference using human electrophysiology. *Psychon Bull Rev* 11:77–83
- Behrmann M, Geng JJ, Shomstein S (2004). Parietal cortex and attention. *Curr Opin Neurobiol* 2:212–217
- Bibbig A, Traub RD, Whittington MA (2002). Long-range synchronization of gamma and beta oscillations and the plasticity of excitatory and inhibitory synapses: a network model. *J Neurophysiol* 88:1634–1654
- Brehaut J, Enns JT, Di Lollo V (1999). Visual masking plays two roles in the attentional blink. *Percept Psychophys*, 61:1436–1448
- Broadbent DE, Broadbent MH (1987). From detection to identification: response to multiple targets in rapid serial visual presentation. *Percept Psychophys* 42:105–113
- Bundesen C (1990). A theory of visual attention. *Psychol Rev* 97:523–547
- Chun MM, Potter MC (1995). A two-stage model for multiple target detection in rapid serial visual presentation. *J Exp Psychol Hum Percept Perform* 21:109–127
- Corbetta M, Kincade MJ, Ollinger J, McAvoy MP, Shulman GL (2000). Voluntary orienting is dissociated from target detection in human posterior parietal cortex. *Nat Neurosci* 3:292–297
- Dehaene S, Naccache L, Cohen L, LeBihan D, Mangin JF, Poline J-B, Rivière D (2001). Cerebral mechanisms of word masking and unconscious repetition priming. *Nat Neurosci* 4:752–758
- Dehaene S, Sergent C, Changeux J-P (2003) A neuronal network model linking subjective reports and objective physiological data during conscious perception. *Proc Natl Acad Sci USA* 100:8520–8525
- Dell’Acqua R, Jolicœur P, Pesciarelli F, Job R, Palomba D (2003) crossmodal attentional blink paradigm. *Psychophysiology* 40:629–639
- Desimone R, Duncan J (1995) Neural mechanisms of selective visual attention. *Annu Rev Neurosci* 18:193–222
- D’Esposito M, Detre JA, Alsop DC, Shin RK, Atlas S, Grossman M (1995) The neural basis of the central executive system of working memory. *Nature* 378:279–281
- Di Lollo V, Kawahara J-I, Ghorashi S M, Enns JT (2005). The attentional blink: resource depletion or temporary loss of control? *Psychological Research*, in press
- Donchin E. (1981). Surprise! ... Surprise? *Psychophysiology* 18:493–513
- Downar J, Crawley AP, Mikulis DJ, Davis KD (2001) The effect of task-relevance on the cortical response to changes in visual and auditory stimuli: an event-related fMRI study. *Neuroimage* 14:1256–1267
- Duncan J (1996) Cooperating brain systems in selective perception and action. In: Inui T, McClelland JL (eds), *Attention and performance XVI*. MIT, Cambridge, pp 549–578
- Duncan J, Humphreys GW (1989) Visual search and stimulus similarity. *Psychol Rev* 96:433–458
- Duncan J, Ward R, Shapiro KL (1994) Direct measurement of attentional dwell time in human vision. *Nature* 369:313–315
- Feinstein JS, Stein MB, Castillo GN, Paulus MP (2005) From sensory to conscious perception. *Conscious and Cogn*, in press
- Fell J, Klaver P, Elger CE, Fernandez G (2002) Suppression of EEG Gamma activity may cause the attentional blink. *Conscious Cogn* 11:114–122
- Friedman-Hill SR, Robertson LC, Ungerleider LG, Desimone R (2003) Posterior parietal cortex and the filtering of distractors. *Proc Natl Acad Sci* 100:4263–4268
- Giesbrecht B, Kingstone A (2004) Right hemisphere involvement in the attentional blink: evidence from a split-brain patient. *Brain Cogn* 55:303–306
- Goldberg ME, Bisley J, Powell KD, Gottlieb J, Kusunoki M (2002) The role of the lateral intraparietal area of the monkey in the generation of saccades and visuospatial attention. *Proc Natl Acad Sci USA* 99:205–215
- 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 USA* 101:13050–13055
- Hommel B (2004) Event files: feature binding in and across perception and action. *Trends Cogn Sci* 8:494–500
- Hommel B, Akyürek EG (2005) Lag-1 sparing in the attentional blink: benefits and costs of integrating two events into a single episode. *Q J Exp Psychol (A)*, (in press)
- Jolicœur P, & Dell’Acqua R (1998) The demonstration of short-term consolidation. *Cogn Psychol* 36:138–202
- Jolicœur P, Dell’Acqua R, Crebolder J (2000) Multitasking performance deficits: forging links between the attentional blink and the psychological refractory period. In: S Monsell, J Driver (eds) *Control of cognitive processes: attention and performance* MIT, Cambridge, pp 309–330
- Jolicœur P, Tombu M, Oriet C, Stevanovski B (2002) From perception to action: making the connection. In: W Prinz, B Hommel (eds) *Common mechanisms in perception and action: attention and performance XIX*. Oxford University Press, Oxford, pp 558–586
- Kessler K, Schmitz F, Gross J, Hommel B, Shapiro K, Schnitzler A (2005a) Target consolidation under high temporal processing demands as revealed by MEG. *Neuroimage*, in press
- Kessler K, Schmitz F, Gross J, Hommel B, Shapiro K, Schnitzler A (2005b) Cortical mechanisms of attention in time: neural correlates of the Lag-1 sparing phenomenon. *Eur J Neurosci*, in press
- Kopell N, Ermentrout GB, Whittington MA, Traub RD (2000). Gamma rhythms and beta rhythms have different synchronization properties. *Proc Natl Acad Sci USA* 97:1867–1872
- Kranczioch C, Debener S, Engel AE (2003) Event-related potential correlates of the attentional blink phenomenon. *Cogn Brain Res* 17:177–187

- Kristofferson AB (1967) Successiveness discrimination as a two-state, quantal process. *Science* 158:1337–1339
- Liang H, Bressler SL, Ding M, Truccolo WA, Nakamura R (2002) Synchronized activity in prefrontal cortex during anticipation of visuomotor processing. *Neuroreport* 13:2011–2015
- Luck S, Vogel E, Shapiro K (1996) Word meanings can be accessed but not reported during the attentional blink. *Nature* 383:616–618
- Lupiañez J, Milliken B (1999) Exogenous cuing effects and the attentional set for integrating vs. differentiating information. *J Gen Psychol* 126:392–418
- Lupiañez J, Milliken B, Solano C, Weaver B, Tipper SP (2001) On the strategic modulation of the time course of facilitation and inhibition of return. *Q J Exp Psychol* 54A:753–773
- Marcantoni WS, Lepage M, Beaudoin G, Bourgouin P, Richer F (2003) Neural correlates of dual task interference in rapid visual streams: an fMRI study. *Brain Cogn* 53:318–321
- Marois R, Chun MM, Gore JC (2000). Neural correlates of the attentional blink. *Neuron* 28:299–308
- Marois R, Yi D-J, Chun MM (2004). The neural fate of consciously perceived and missed events in the attentional blink. *Neuron* 41:465–472
- McArthur G, Budd T, Michie P (1999) The attentional blink and P300. *Neuroreport* 10:3691–3695
- Menon V, Ford JM, Lim KO, Glover GH, Pfefferbaum A (1997) Combined event-related fMRI and EEG evidence for temporal-parietal cortex activation during target detection. *Neuroreport* 8:3029–3037
- Milner B (1968) Visual recognition and recall after right temporal-lobe excision in man. *Neuropsychologia* 6:191–209
- Pöppel E (1997) A hierarchical model of temporal perception. *Trends Cogn Sci* 1:56–61
- Potter MC, Chun MM, Banks BS, Muckenhoupt M (1998) Two attentional deficits in serial target search: the visual attentional blink and an amodal task-switch deficit. *J Exp Psychol Learn Mem Cogn* 25:979–992
- Potter MC, Staub A, O'Connor DH (2002) The time course of competition for attention: attention is initially labile. *J Exp Psychol Hum Percept Perform* 28:1149–1162
- Raymond JE (2003) New objects, not new features, trigger the attentional blink. *Psychol Sci* 14:54–59
- Raymond JE, Shapiro KL, Arnell KM (1992) Temporary suppression of visual processing in an RSVP task: an attentional blink? *J Exp Psychol Hum Percept Perform* 18:849–860
- Richer F, Lepage M (1996) Frontal lesions increase post-target interference in rapid stimulus streams. *Neuropsychologia* 34:509–514
- Richer F, Bédard S, Lepage M, Chouinard MJ (1998) Frontal lesions produce a dual task deficit in simple rapid choices. *Brain Cogn* 37:173–175
- Rolke B, Heil M, Streb J, Henninghausen E (2001) Missed prime words within the attentional blink evoke an N400 semantic priming effect. *Psychophysiology* 38:165–174
- Shapiro KL (2001) Temporal methods for studying attention: how did we get there and where are we going? In: K Shapiro (ed.) *The limits of attention: temporal constraints in human information processing*. Oxford University Press, Oxford, pp 1–19
- Shapiro KL, Raymond JE, Arnell KM (1994) Attention to visual pattern information produces the attentional blink in rapid serial visual presentation. *J Exp Psychol Hum Percept Perform* 20:357–371
- Shapiro K, Hillstrom AP, Husain M (2002) Control of visuotemporal attention by inferior parietal and superior temporal cortex. *Curr Biol* 12:1320–1325
- Sheppard D, Duncan J, Shapiro K, Hillstrom AP (2002) Objects and events in the attentional blink. *Psychol Sci* 13:410–415
- Szameitat AJ, Schubert T, Müller K, Von Cramon DY (2002) Localization of executive functions in dual-task performance with fMRI. *J Cogn Neurosci* 14:1184–1199
- Treisman A (1996) The binding problem. *Curr Opin Neurobiol* 6:171–178
- Turvey MT (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 TAW, Bischof WF, Di Lollo (1999). Attentional switching in spatial and non-spatial domains: evidence from the attentional blink. *Psychol Bull* 125:458–469
- Vogel EK, Luck SJ (2002) Delayed working memory consolidation during the attentional blink. *Psychon Bull Rev* 9:739–743
- Von Stein A, Rappelsberger P, Sarnthein J, Petsche H (1999) Synchronization between temporal and parietal cortex during multimodal object processing in man. *Cereb Cortex* 9:137–150
- Waszak F, Hommel B, Allport A (2005) Interaction of task readiness and automatic retrieval in task-switching: negative priming and competitor priming. *Mem Cognit*, in press
- Waszak F, Hommel B, Allport A (2003) Task-switching and long-term priming: role of episodic stimulus-task bindings in task-shift costs. *Cogn Psychol* 46:361–413
- Wojciulik E, Kanwisher N (1999) The generality of parietal involvement in visual attention. *Neuron* 23:747–764
- Wrobel A (2000) Beta activity: a carrier for visual attention. *Acta Neurobiol Exp* 60:247–260