

Top-down control is not lost in the attentional blink: evidence from intact endogenous cuing

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Received: 14 March 2007 / Accepted: 19 September 2007 / Published online: 10 October 2007
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Abstract The attentional blink (AB) refers to the finding that performance on the second of two targets (T1 and T2) is impaired when the targets are presented at a target onset asynchrony (TOA) of less than 500 ms. One account of the AB assumes that the processing load of T1 leads to a loss of top-down control over stimulus selection. The present study tested this account by examining whether an endogenous spatial cue that indicates the location of a following T2 can facilitate T2 report even when the cue and T2 occur within the time window of the AB. Results from three experiments showed that endogenous cuing had a significant effect on T2 report, both during and outside of the AB; this cuing effect was modulated by both the cue-target onset asynchrony and by cue validity, while it was invariant to the AB. These results suggest that top-down control over target selection is not lost during the AB.

Introduction

When observers monitor a rapid serial visual presentation (RSVP) of stimuli, such as letters, digits, words or pictures, and search for two targets in the stream, they usually have

no difficulty in reporting the first target (T1) but they show a deficit in reporting the second target (T2), if the T2 appears within less than 500 ms from T1 (Broadbent and Broadbent 1987). The post-T1 interval during which report of T2 is impaired is referred to as an attentional blink (AB, Raymond et al. 1992).

During the last two decades, a number of models have been proposed to account for the AB (e.g., Broadbent and Broadbent 1987; Raymond et al. 1992; Shapiro et al. 1994; Chun and Potter 1995; Giesbrecht and Di Lollo 1998; Jolicoeur and Dell'Acqua 1998; Bowman and Wyble 2007; Olivers 2007). Most of these models share the assumption that the AB reflects the capacity limit of cognitive processing, the idea being that the consumption of cognitive resources in processing T1 causes the deficit in T2 report. Consistent with this interpretation, results from neuroimaging studies show that there is considerable overlap between the neural areas correlated with the AB bottleneck and those implicated in the capacity limits of visual short-term memory storage and in the phenomenon of psychological refractory period, with all tasks showing increased activation of a fronto-parietal network under conditions of high processing load (for an overview, see, Marois and Ivanoff 2005).

In a recent study, Di Lollo et al. (2005) proposed an account of how the processing load of T1 identification leads to the AB. According to this account, called the temporary loss of control (TLC) model, the sensory system is initially configured to be an input filter which is optimized to process T1 and to exclude distractors. This configuration is assumed to be governed by the top-down signals from the executive control system which is located in prefrontal cortex (PFC). When T1 passes through the filter, the central control system becomes engaged in processing T1 and the top-down control signals are interrupted. At this time the

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distractors following T1 may change the filter settings which are now under the control of the properties of the bottom-up input, resulting in difficulty in selecting T2 because the filter configuration no longer matches the specifications of T2. However, if the item following T1 is not a distractor, but a target that belongs to the same category as T1, the filter settings might not be changed, and this target could be selected and processed without problem. Supporting evidence for TLC comes from findings showing that the processing of T2 is not impaired when the items intervening between T1 and T2 are from the same category as T1 and T2 (Di Lollo et al. 2005; Kawahara et al. 2006; Olivers et al. 2007).

Although the AB has long been thought to be a consequence of the depletion of processing resources by T1, recent work suggests that there are in fact still processing resources available during the AB. For example, it has been shown that targets presented during the AB can in fact be accurately identified when they are precued by a stimulus that matches the attentional set for the targets (Nieuwenstein et al. 2005; Nieuwenstein 2006; see also, Olivers et al. 2007). Importantly, this cuing benefit for T2 does not incur a cost to T1 report, suggesting that not all resources are consumed in processing the first target. Work by Martens and Johnson (2005) further suggests that these remaining resources can be deployed in a purely endogenous fashion during the AB. In particular, this study showed that the AB is attenuated when observers are told on each trial what the target-onset asynchrony (TOA) will be, suggesting that they could somehow prepare for or deploy resources to the moment at which T2 was expected to occur. Given that this effect occurred in the absence of an exogenous cue, it must be concluded that resources were deployed on a voluntary basis even during the AB.

The purpose of the present study was to confirm and further examine the extent to which observers maintain voluntary control over the deployment of processing resources during the AB. To this end, we used an endogenous cuing procedure to directly determine whether the function of prefrontal control system is intact during the AB. In this procedure, an arrow is presented in the center of the display that can indicate where an upcoming target will appear (e.g., Jonides 1981). Studies using arrow cues show that whether or not observers voluntarily orient their attention towards the cued location depends on the validity of the cue, with stronger cuing effects seen for highly predictive cues than for cues with a low validity (e.g., Jonides 1980; Posner 1980; Jonides 1983; Eriksen and Yeh 1985; Madden 1992; Riggio and Kirsner 1997). These results indicate that this type of cuing is contingent on the observers' intention to use the cue, and, thus under top-down control. Moreover, numerous neuroimaging studies have demonstrated that endogenous attentional orienting involves a fronto-parietal

network (e.g., Hopfinger et al. 2000; Miller 2000; Corbetta and Shulman 2002; Miller and D'Esposito 2005; Praamstra et al. 2005; Rounis et al. 2006) in which the PFC is considered the "top" of the top-down control system which can modulate the activity of neurons in visual cortex independent of input information (e.g., Miller 2000; Miller and D'Esposito 2005). For example, a recent neuroimaging study by Vossel et al. (2006) demonstrated more activation in a fronto-parietal network for the endogenous cues with high validity (90%) than that with low validity (60%). These neural studies are congruent with LaBerge's activity-distribution model (e.g., LaBerge and Brown 1989; LaBerge et al. 1997) of spatial attention, in which top-down control processes of spatial attention, located in the dorso-lateral prefrontal cortex (DLPFC), produce a selective activity distribution in posterior parietal cortex (PPC) that is enhanced through the thalamus to visual cortex. If the function of top-down control over attention located in PFC is temporally suspended during the period when T1 consolidation is underway, then the endogenous cuing effect would be impaired during the AB than outside of the AB (as hypothesized by Ghorashi et al. 2007). On the contrary, if the effect of endogenous cuing is not affected by the AB, the presumption of loss-of-control should be re-considered.

An argument might be raised based on the presumption that the different parts of PFC are involved in different aspects of top-down attentional control. For example, according to LaBerge (1997; see also LaBerge 2001), the dorsolateral prefrontal cortex (DLPFC) is selectively involved in voluntary control of spatial attention ("where" circuit), while the ventrolateral prefrontal cortex (VLPFC) is involved in voluntary control of attention for shape and color ("what" or "object" circuit). This entails that our measure of top-down controlled, spatial selection may not be sensitive to the disturbance of the object-based filtering mechanisms implicated in the loss of control account. Crucially, however, results from numerous recent neuroimaging studies have provided evidence against LaBerge's claim in showing that the fronto-parietal network that is recruited when subjects expect to see object or feature clearly overlaps with regions that are recruited by attending to location (for overviews, see Kanwisher and Wojciulik 2000; Corbetta and Shulman 2002; Yantis and Serences 2003; see also, Corbetta et al. 2005). A similar result was obtained in an ERP study that showed that control of spatial and non-spatial visual attention correlates with the same neural activity (e.g., Slagter et al. 2005). Thus, the same fronto-parietal network might be involved in the top-down control over spatial attention in the present study and the top-down control over input filter configuration in TLC model, and this provides fundament for our logic to test whether this network loses its control function during the AB.

To examine how endogenous spatial cuing is affected by the AB, we used the so-called “skeletal” two-target paradigm (Duncan et al. 1994; Ward et al. 1996) in which T1 and T2, presented in one of four locations, are immediately followed by pattern masks. A central arrow cue was inserted between the target presentations. If the function of top-down control is lost during the AB, the endogenous attentional orienting would not work and consequently the cuing effect should be reduced or eliminated compared with the cuing effect when the cue is presented outside of the AB period. If, on the other hand, the functioning of the prefrontal control system is intact during the AB, the cuing effects should be present both inside and outside of the AB.

Given that cue validity plays a significant role in modulating the size of endogenous cuing effects in spatial orienting (e.g., Jonides 1980, 1983; Posner 1980; Eriksen and Yeh 1985; Madden 1992; Riggio and Kirsner 1997; Vossel et al. 2006), we manipulated the cue validity of the central cue, both inside and outside the AB period, across three experiments. In Experiment 1, we used a cue validity of 100% to test whether the central cuing works in the AB context. Then, in Experiment 2, we used a cue validity of 50% to demonstrate that the observers could ignore uninformative cues both inside and outside of the AB. In Experiment 3, the cue validity was 80%, thereby allowing for an assessment of the effects of both valid and invalid cues under condition in which the cue is expected to be valid.

An additional purpose of Experiment 2 (with a cue validity of 50%) was to confirm that any effects of the arrow cue were indeed due to an endogenous shift of attention. As previous work suggests, there are cases in which symbolic cues such as eye gazes or arrows have been found to direct attention in exogenous manner (e.g., Hommel et al. 2001; Ristic et al. 2002). The hallmark of this exogenous component to orienting attention in response to a symbolic cue is that target performance is enhanced even when the cues are non-predictive (i.e., 50% validity). Consequently, if there were to be an exogenous component to orienting in response to the arrow cue we used, the results from Experiment 2 would reveal this, as in this experiment, the cue validity was 50%.

Experiments 1–3

Method

Participants

Sixty right-handed students (30 males and 30 females) from Peking University were recruited to participate in the experiments in return for monetary compensation. Their age ranged from 20 to 28, and averaged on 22 ± 3.5 years.

All the participants reported having normal or corrected-to-normal eyesight. They were randomly assigned to the three experiments, with 10 males and 10 females for each experiment.

Apparatus and stimuli

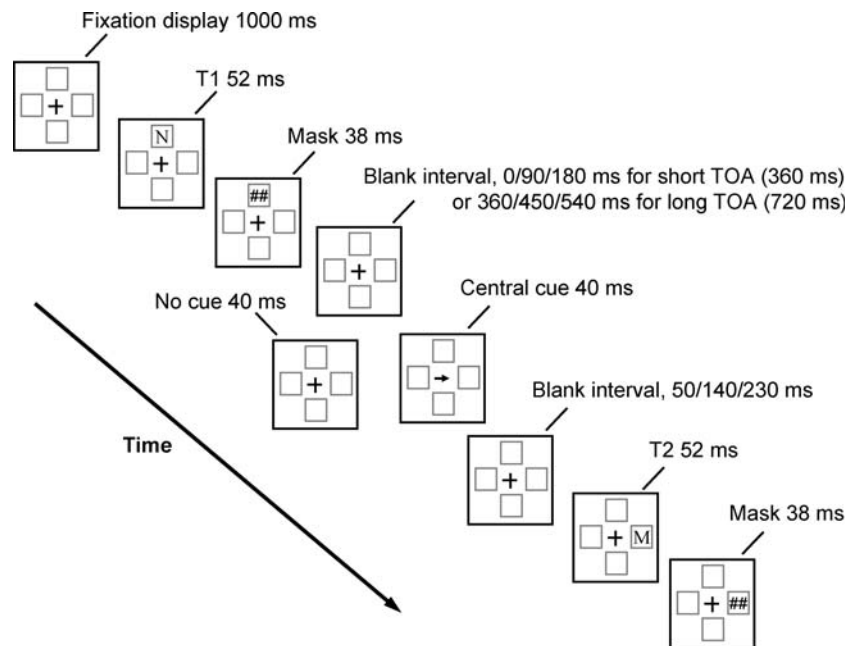
The experiments were run in a dimly illuminated room, with stimuli presented on a 17” CRT monitor running at a resolution of $1,024 \times 768$ pixels. Stimulus presentation and recording of participants’ responses were controlled by Presentation software (<http://nbs.neuro-bs.com/>). The stimulus display consisted of a white central fixation cross [with the RGB values (255, 255, 255)] that was surrounded by four grey outline frames or place-holders [with the RGB values (125, 125, 125)] that designated the potential target locations (see Fig. 1). The size of fixation was 0.6×0.6 of visual angle. The distance between the fixation cross and the center of each of these four place-holders was 2.0° of visual angle. A chinrest was set at 57 cm from the screen. The two targets were randomly drawn from the upper-case letter set [K, M, N, V, W, X, Y, Z] and the mask consisted of two hashmarks next to each other (i.e., “##”; cf. Nieuwenstein et al. 2005). Targets and masks were presented in the Times New Roman font, in white [with the RGB values (255, 255, 255)] against dark background (0, 0, 0), and in a size of $0.3^\circ \times 0.4^\circ$ of visual angle. The central cue, subtending 1° in visual angle, was a white arrow [255, 255, 255] presented at the fixation position and pointed to the left or right place-holder.

Design and procedures

Half of the trials in each of the experiments had the central cue. The onset asynchrony (TOA) between T1 and T2 had two levels, 360 and 720 ms. When a trial had the central cue, the interval separating the onset of the cue from that of T2, the cue-target onset asynchrony (CTOA), was 90, 180, or 270 ms. In Experiment 1, the cue validity was 100%. That is, when present, the cue always indicated correctly the location of the upcoming T2. In Experiment 2, the cue validity was 50%, so T2 only appeared in the cued location on half of the trials. The cue was valid on 80% of the trials in Experiment 3.

Participants began each trial by pressing the space bar while fixating on the central cross in the fixation display. After 1 s, the first target (T1) was presented for 52 ms either above or below the central fixation and followed immediately by the mask which appeared at the T1 location for 38 ms. Following an interval of 270 or 630 ms (for TOAs of 360 and 720 ms, respectively), T2 appeared for 52 ms to the left or right of the fixation cross. Like T1, T2 was followed by a mask which was presented in the same location as T2

Fig. 1 A schematic representation of the trial procedure for Experiments 1–3



for 38 ms. On cued trials, an arrow that pointed to either the left or the right location was presented for 40 ms at the location of the fixation cross. Depending on the CTOA, this arrow could appear at different points in time from T1. For example, when the TOA was 360 ms and the CTOA was 270 ms, the cue appeared immediately following the offset of the T1 mask. The task for the participants was to report the identities of T1 and T2 in their presentation order. If both T1 and T2 were reported as right identities but in reversal order, they were marked as correct report.

In Experiment 1, there were 40 trials for each combination of CTOA and TOA in the cued condition, and there were 120 trials for each TOA in the uncued condition. Thus Experiment 1 consisted of 480 trials. These trials were divided into five testing blocks, with each block containing 96 trials. In Experiment 2, there were 40 cued trials for each combination of CTOA, cue validity and TOA, and 240 uncued trials for each TOA. Therefore, Experiment 2 had 960 trials which were divided into 10 testing blocks. In Experiment 3, there were 36 invalid cue trials and 144 valid cue trials for each combination of CTOA and TOA, and 540 uncued trials for each TOA, yielding a total of 2,160 trials. This experiment was conducted in three separate sessions, each conducted on a different day in the same week. Each session had six testing blocks with 120 trials in each block. Trials from different experimental conditions were equally distributed into different testing blocks and there was an interval of 1–2 min between the blocks. Participants received 60 practice trials before they began the experiment.

Participants were instructed to maintain fixation on the central cross and to try to avoid making eye movements

during a trial. They reported the targets by typing them in the order of appearance at the end of each trial. They were also informed of the validity of the cue before they began the experiment.

Results

The average performance for report of T1 was 92, 90 and 92% for Experiments 1, 2 and 3, respectively (for the accuracy and standard error for each condition, see Table 1). There were no main effects or interactions of the factors cuing and TOA on T1 accuracy ($P > 0.1$). Data analyses were then conducted on T2-identification performance. These analyses only included trials in which T1 was correctly reported (i.e., T2/T1). Figures 2, 3 and 4 show the means and standard errors for accuracy of T2/T1 in different experimental conditions from Experiments 1–3.

Experiment 1: 100% cue validity

Mean accuracy scores for T2/T1 were entered into a 2 (TOA) \times 4 (cue condition: uncued, and cues with CTOA of 90, 180, 270 ms) ANOVA. The main effect of TOA was significant, $F(1, 19) = 133.34$, $P < 0.001$, with better T2 report at the long TOA than that at the short TOA (85 versus 63% correct). The main effect of cue condition was significant, $F(3, 57) = 41.91$, $P < 0.001$, with the lowest accuracy for the CTOA of 90 ms (mean = 67%), the highest accuracy for the CTOA of 270 ms (mean = 82%), and intermediate levels of performance for the uncued condition (mean = 73%), and the cued condition with an CTOA of

Table 1 Means and standard errors of T1 accuracy (in %) from Experiments 1–3

	CTOA (ms)	TOA = 360 ms		TOA = 720 ms	
		M (%)	SE (%)	M (%)	SE (%)
Experiment 1 (100% valid)					
Uncued		92	0.9	92	1.3
Valid	90	90	1.8	90	1.5
	180	90	1.3	91	1.4
	270	91	1.9	92	1.0
Experiment 2 (50% valid)					
Uncued		91	1.7	91	1.9
Valid	90	92	1.8	90	1.9
	180	90	1.8	91	1.9
	270	90	2.0	89	2.1
Invalid	90	90	2.2	90	2.0
	180	92	1.6	90	2.5
	270	91	1.6	92	1.6
Experiment 3 (80% valid)					
Uncued		91	1.5	92	1.5
Valid	90	91	1.7	91	1.5
	180	91	1.6	91	1.6
	270	91	1.8	91	1.6
Invalid	90	91	1.6	91	1.7
	180	90	1.9	91	2.1
	270	90	2.2	91	1.5

M mean, SE standard errors

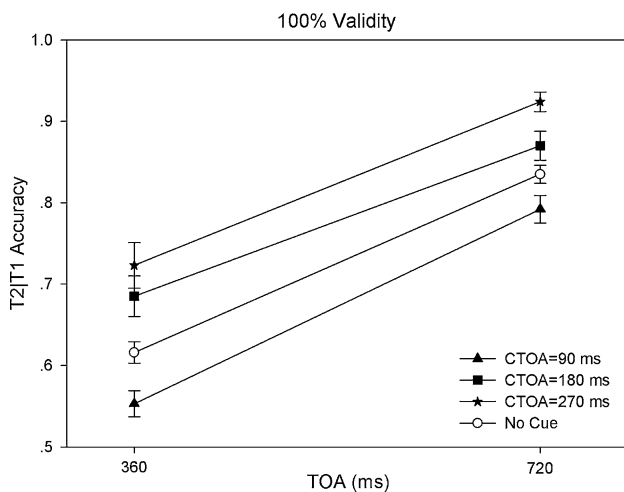


Fig. 2 T2/T1 accuracies from Experiment 1. The error bar indicates the standard error for each condition

180 ms (mean = 78%). Pair-wise comparison of T2 performance in the four cue conditions showed that each of these differences was significant at an alpha corrected for multiple comparisons using Bonferroni adjustment (all P 's < 0.0083). These results indicate that, compared to the uncued condition, T2 performance was enhanced in the

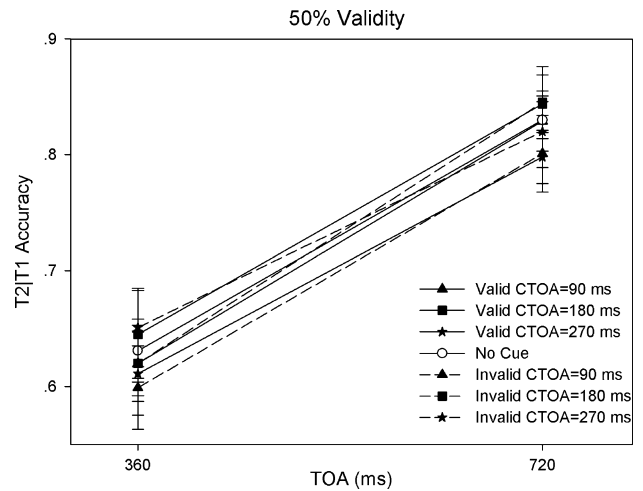


Fig. 3 T2/T1 accuracies from Experiment 2. The error bar indicates the standard error for each condition

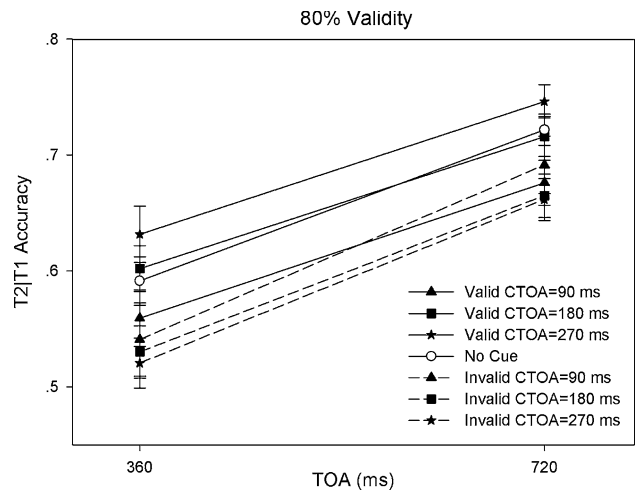


Fig. 4 T2/T1 accuracies from Experiment 3. The error bar indicates the standard error for each condition

cued condition when the CTOA was 180 or 270 ms, while it was relatively impaired for cued trials with a CTOA of 90 ms. Importantly, the interaction between TOA and cue condition was not significant, $F(3, 57) = 1.92, P > 0.1$, indicating that the central cues influenced T2 performance to the same extent however short or long the TOA was. Thus, endogenous cuing did not appear to be affected by the AB.

Experiment 2: 50% cue validity

We performed separate analyses for the effects of valid and invalid cues in Experiment 2.

Effects of valid cues

A 2 (TOA) × 4 (cue condition: uncued, and cues with CTOA of 90, 180, and 270 ms) ANOVA was conducted for T2/T1 in the uncued and valid cuing conditions of

Experiment 2. The main effect of TOA was significant, $F(1, 19) = 171.63$, $P < 0.001$, with lower accuracy for T2 identification at the short TOA (mean = 63%) than at the long TOA (mean = 83%). The main effect of cue condition approached significance, $F(3, 57) = 2.39$, $P = 0.078$. Bonferroni-corrected pair-wise comparisons revealed that none of the pair-wise differences between T2 reporting in different cue conditions reached significance. Moreover, the interaction between TOA and cue condition was not significant, $F(3, 57) < 1$. Thus, in contrast to Experiment 1, there were no significant effects of valid central cues.

Effects of invalid cues

The comparison of T2/T1 for the uncued condition and the condition with invalid cues showed a significant main effect of TOA, $F(1, 19) = 335.87$, $P < 0.001$, with lower accuracy at the short TOA (mean = 63%) than at the long TOA (mean = 82%). The main effect of cue condition approached significance, $F(3, 57) = 2.46$, $P = 0.072$, but pair-wise comparisons revealed no significant differences between the four conditions. The interaction between TOA and cue condition approached significance, $F(3, 57) = 2.69$, $P = 0.055$. Further analyses showed that the main effect of cue condition approached significance at the short TOA, $F(3, 57) = 2.74$, $P = 0.052$. At the long TOA, the main effect of cue condition also approached significance, $F(3, 57) = 2.28$, $P = 0.089$. The Bonferroni-corrected pair-wise comparisons showed that only the difference between invalid cues with the 90 and the 180 ms CTOA was significant, $P < .0083$. These results suggest that, relative to the uncued condition, the invalid cues did not affect T2 performance.

Experiment 3: 80% cue validity

Effects of valid cues

The T2/T1 accuracy for the valid cue and the uncued conditions were entered into a 2 (TOA) \times 4 (cue condition: uncued, and cues with CTOA of 90, 180, and 270 ms) ANOVA. The main effect of TOA was significant, $F(1, 19) = 80.07$, $P < 0.001$, with lower T2 performance at the short TOA (mean = 60%) than at the long TOA (mean = 72%). The main effect of cue condition was significant, $F(3, 57) = 20.70$, $P < 0.001$, with the highest accuracy at the 270 ms CTOA (mean = 69%), the lowest performance at the 90 ms CTOA (mean = 62%), and an intermediate level of performance for the uncued condition (mean = 66%) and the 180 ms CTOA (mean = 66%) condition. Each of the pair-wise differences between the different cue conditions was significant, $P < 0.0083$, except for the difference between the uncued condition and the 180 ms

CTOA condition, $P > 0.1$. The interaction between TOA and cue condition was not significant, $F(3, 57) = 1.16$, $P > 0.1$, indicating that the pattern of cuing effects was the same at the short and the long TOAs.

Effects of Invalid Cues

The analyses of T2/T1 for trials with invalid cues revealed a significant main effect of TOA, $F(1, 19) = 115.61$, $P < 0.001$, with lower T2 performance at the short TOA (mean = 55%) than at the long TOA (mean = 68%). The main effect of cue condition was significant, $F(3, 57) = 8.76$, $P < 0.001$, with the highest T2 accuracy for the uncued condition (mean = 66%), the lowest T2 accuracy for the 270 ms CTOA (mean = 59%), and medium performance for the 180 ms CTOA (mean = 60%) and the 90 ms CTOA (mean = 62%). Bonferroni-corrected pair-wise comparisons showed that all pair-wise differences between the uncued condition and the conditions with invalid cues were significant ($P < 0.0083$) except for the difference between the uncued condition and the 90 ms CTOA condition, which approached significance ($P = 0.06$). There were no significant differences between the three conditions with invalid cues, all P 's > 0.1 . The interaction between TOA and cue condition was not significant, $F(3, 57) < 1$.

General discussion

The aim of the present study was to determine if and how the capacity to endogenously orient spatial attention is affected by the AB. To this end, we examined the effects of arrow cues on the ability to report a second target that appeared during or after the AB induced by a leading first target. The arrow indicated the location where T2 could appear. The validity of the cue was varied across three experiments, with 100% valid cues in Experiment 1, 50% valid cues in Experiment 2 and 80% valid cues in Experiment 3.

Results from the three experiments can be summarized as follows. An AB was observed in each experiment, with T2 report being worse when the time interval between T1 and T2 was short (360 ms) than when the interval was long (720 ms). The effects of valid and invalid cues on T2 performance were modest, yet they appeared to be systematic. Valid cues led to better performance on T2 when the time interval between the cue and T2 was relatively long (i.e., with CTOAs of 180 or 270 ms) but only in Experiments 1 and 3 where cues were 100 and 80% valid, respectively. Invalid cues, in contrast, led to worse performance for reporting T2 in Experiment 3 where the cue was expected to be valid. For trials with a CTOA of 90 ms, both valid and invalid cues produced a decrement in T2 report, perhaps

due to the fact that the arrow captured attention at its own location and observers needed some time to reorient attention from the location of the cue towards the cued location. Critically, the cuing effects observed with the two longer CTOAs in Experiments 1 and 3 were not observed when the cue was uninformative with regard to the T2 location (i.e., in Experiment 2 where 50% of the cues were valid), thereby excluding the possibility that the cuing effects observed in Experiments 1 and 3 were due to some reflexive attentional orienting mechanism evoked by the shape of arrow cue (e.g., Hommel et al. 2001; Ristic et al. 2002). Finally, and, most importantly, the observed pattern of cuing effects did not interact with TOA, suggesting that the AB did not affect the processes involved in endogenous control of spatial selective attention.

A drawback to the interpretation of the present findings is perhaps that our answer to the main question of interest—whether the AB affects endogenous spatial cuing—is based on a null-effect, namely, the lack of interaction between cuing and TOA. To further strengthen the case that endogenous cuing was not affected by the AB we ran an additional analysis on the data collapsed for the 180 and 270 ms CTOA conditions from experiments 1 and 3. In spite of increased statistical power, the interaction of cuing and TOA remained non-significant in this analysis, strengthening our confidence in the conclusion that the AB appears to have little impact on processes involved in the endogenous orienting of spatial attention.

The inhibitory cuing effect for predictive valid cues with 90 ms of CTOA in Experiment 1 and 3 suggests that it takes a certain amount of time to process an endogenous cue and to shift attention accordingly. Meanwhile, the result that the facilitatory cuing effects for predictive valid cues grew with increasing CTOA (i.e., 180 and 270 ms) is consistent with previous studies showing that it takes time to build up endogenous control over spatial orienting (e.g., Müller and Rabbitt 1989). In the present study, there seems a confounding linkage between CTOA and T1-cue SOA. If the processing of T1 and the cue compete for the same resources and if T1 processing consumes most of these resources, then the shorter T1-cue SOA, the stronger the competition between T1 and the cue, the less processing of the cue, and consequently the weaker the endogenous cuing effects. The question is then whether the potentially growing cuing effect with increasing T1-cue SOA is counteracted by the decreasing CTOA. Due to the nature of AB paradigm, it was not possible to manipulate T1-cue SOA and CTOA independently without affecting TOA in this study. However, this linkage is not a problem for the present study. For the trials with a long TOA, the T1-cue SOA was at least 450 ms, putting all the cues near the end of the typical AB period. For the trials with short TOA, the T1-cue SOA ranged from 90 to 270 ms, putting all the cues within typi-

cal AB period. If the processing of T1 and the cue competes for the same resources, either T1 performance or the cuing effect would be different between the short and the long TOA trials. However, the results showed that T1 performance did not change over the short and the long TOA trials; the impact of CTOA upon T2 performance did not vary over the short and the long TOA trials either, indicating that the T1-cue SOA had little impact upon the pattern of cuing effects. This is the key point of the present study: endogenous cuing is not influenced by T1 processing.

In showing that the endogenous orienting of spatial attention is unaffected by an AB, the present findings complement previous studies in which it was shown that selection based on category, color (Nieuwenstein 2006), or the expected moment of arrival of a second target (Martens and Johnson 2005) all continue to operate according to top-down goals during the AB. Together, these results make a compelling case against the loss-of-control account which assumes that the AB occurs because the filter (template) used for target selection is no longer effective due to the load imposed by encoding T1 (Di Lollo et al. 2005; Kawahara et al. 2006). Instead, the available findings converge in suggesting that top-down control over visual selection is sustained during the AB.

While selection thus continues to adhere to top-down goals during an AB, this is not to say that selection operates effectively during this period (e.g., Jiang and Chun 2001; Vul et al. 2007). This is indicated by the fact that errors in report of targets presented during the blink are mostly due to failures of timely selection: Observers will often report the item trailing T2 instead of T2 (Chun 1997; Isaak et al. 1999), and precuing T2 with a stimulus that matches one of the target-defining features substantially reduces the blink effect (Nieuwenstein et al. 2005; Nieuwenstein 2006). For example, in an experiment that used the same method as that used in the present experiments, Nieuwenstein et al. (Experiment 4) found that when T2 is preceded by an exogenous cue (a plus sign that appeared 94 ms prior to T2, in the same location as T2) there is a substantial improvement in T2 report during the blink, but not outside of the blink (but see Ghorashi et al. 2007). This finding was taken to indicate that there is a delay between the detection of a potential target stimulus and the allocation of resources that are needed to sustain the target representation and to consolidate it into short-term memory. Thus, although the AB does not prevent attention from being triggered selectively by stimuli that match our top-down goals, it does affect the rate at which resources are deployed when a potential target stimulus is detected.

The notion that resource allocation is delayed during an AB seems at odds with the present findings that show that the time course of the deployment of attention in response to an endogenous cue is similar during and outside of the

blink period. However, this inconsistency may be explained if one assumes that different mechanisms are involved in endogenous spatial orienting and in the allocation of resources in response to a potential target. As argued by LaBerge and Brown (1989) (see also LaBerge et al. 1997; LaBerge 2001), endogenous orienting of attention may involve a slow build up of attentional resources at the location where a target is expected to appear. This state of preparatory attention may encompass one or multiple locations depending on top-down goals (e.g., Jefferies et al. 2007) and it can be conceived of as a gradient of attention-related activity across the visual field. When a (potential) target appears, a different type of attentional response is elicited. This response is focal, transient, and intense, and therefore capable of facilitating identification of targets that are briefly presented and masked (Nakayama and Mackeben 1989). Crucially, it is this form of transient attention that has been implicated in explanations of the AB (Nieuwenhuis et al. 2005; Bowman and Wyble 2007; Olivers 2007). In particular, it is thought that target detection invokes transient attention, thereby benefiting the encoding of items that appear within a window of about 100 ms following the onset of the target (this explains the so-called “sparing” of T2 identification seen when T2 follows T1 at TOAs of less than 100 ms. see Potter et al. 1998). More important, this transient form of attention is also considered to be the locus of the selection delays seen for targets presented during the AB. For example, one proposal is that consolidating T1 into short-term memory inhibits the mechanism that mediates transient attention so as to ensure that no new inputs can intrude and interfere with the ongoing processing of T1 (B. Wyble et al., submitted). In this view, the delayed engagement of attention during an AB reflects the fact that more time (and target input) is needed to overcome this inhibition so that a second episode of transient attention can be launched.

Thus, transient attention appears to constitute a critical part of the mechanism that gives rise to delayed selection in the AB. On the other hand, the processes that implement top-down goals through setting the weights for different features or locations appear to be unaffected by the processing demands of encoding T1 into short-term memory.

In conclusion, the present findings show that the processes underlying the voluntary deployment of attention to a particular location are not affected by the AB. In this regard, the present study corroborates previous work that also provided evidence consistent with the possibility that top-down control over resource allocation and stimulus selection is not lost during the AB (e.g., Martens and Johnson 2005; Nieuwenstein et al. 2005; Nieuwenstein 2006). Together, these results make a compelling case against the claim that the processing load of T1 identification leads to a

temporary loss of top-down control over selective attention (Di Lollo et al. 2005; Kawahara et al. 2006).

Acknowledgments This study was supported by grants from the Natural Science Foundation of China (30470569, 60435010, and 30770712). We thank two anonymous reviewers for their constructive comments on the earlier versions of the paper. Electronic mail concerning this study should be addressed to Dr. Xiaolin Zhou (xz104@pku.edu.cn).

References

- Bowman H, Wyble B (2007) The simultaneous type, serial token model of temporal attention and working memory. *Psychol Rev* 114:38–70
- Broadbent DE, Broadbent MHP (1987) From detection to identification—response to multiple targets in rapid serial visual presentation. *Percept Psychophys* 42:105–113
- Chun MM (1997) Temporal binding errors are redistributed by the attentional blink. *Percept Psychophys* 59:1191–1199
- 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, Shulman GL (2002) Control of goal-directed and stimulus-driven attention in the brain. *Nat Rev Neurosci* 3:201–215
- Corbetta M, Tansy AP, Stanley CM, Astafiev SV, Snyder AZ, Shulman GL (2005) A functional MRI study of preparatory signals for spatial location and objects. *Neuropsychologia* 43:2041–2056
- Di Lollo V, Kawahara J, Ghorashi SMS, Enns JT (2005) The attentional blink: resource depletion or temporary loss of control? *Psychol Res-Psychol Forsch* 69:191–200
- Duncan J, Ward R, Shapiro K (1994) Direct measurement of attentional dwell time in human vision. *Nature* 369:313–315
- Eriksen CW, Yeh YY (1985) Allocation of attention in the visual-field. *J Exp Psychol Hum Percept Perform* 11:583–597
- Ghorashi SMS, Di Lollo V, Klein RM (2007) Attentional orienting in response to peripheral cues survives the attentional blink. *Vis Cogn* 15:87–90
- Giesbrecht B, Di Lollo V (1998) Beyond the attentional blink: visual masking by object substitution. *J Exp Psychol Hum Percept Perform* 24:1454–1466
- Hommel B, Pratt J, Colzato L, Godijn R (2001) Symbolic control of visual attention. *Psychol Sci* 12:360–365
- Hopfinger JB, Buonocore MH, Mangun GR (2000) The neural mechanisms of top-down attentional control. *Nat Neurosci* 3:284–291
- Isaak MI, Shapiro KL, Martin J (1999) The attentional blink reflects retrieval competition among multiple rapid serial visual presentation items: tests of an interference model. *J Exp Psychol Hum Percept Perform* 25:1774–1792
- Jefferies LN, Ghorashi SM, Kawahara J, Di Lollo V (2007) Ignorance is bliss: the role of observer expectation in dynamic spatial tuning of the attentional focus. *Percept Psychophys* (in press)
- Jiang YH, Chun MM (2001) The influence of temporal selection on spatial selection and distractor interference: an attentional blink study. *J Exp Psychol Hum Percept Perform* 27:664–679
- Jolicoeur P, Dell’Acqua R (1998) The demonstration of short-term consolidation. *Cognit Psychol* 36:138–202
- Jonides J (1980) Towards a model of the mind’s eye movement. *Can J Psychol* 34:103–112 (*Revue Canadienne De Psychologie*)
- Jonides J (1981) Voluntary versus automatic control over the mind’s eye’s movement. In: Long JB, Baddeley AD (eds) *Attention and performance IX*. Lawrence Erlbaum, Hillsdale, pp 187–203

- Jonides J (1983) Further toward a model of the mind's eye movement. *Bull Psychon Soc* 21:247–250
- Kanwisher N, Wojciulik E (2000) Visual attention: insights from brain imaging. *Nat Rev Neurosci* 1:91–100
- Kawahara JI, Kumada T, Di Lollo V (2006) The attentional blink is governed by a temporary loss of control. *Psychon Bull Rev* 13:886–890
- LaBerge D (1997) Attention, awareness, and the triangular circuit. *Conscious Cogn* 6:149–181
- LaBerge D (2001) Attention, consciousness, and electrical wave activity within the cortical column. *Int J Psychophysiol* 43:5–24
- LaBerge D, Brown V (1989) Theory of attentional operations in shape identification. *Psychol Rev* 96:101–124
- LaBerge D, Carlson RL, Williams JK, Bunney BG (1997) Shifting attention in visual space: tests of moving-spotlight models versus an activity-distribution model. *J Exp Psychol Hum Percept Perform* 23:1380–1392
- Madden DJ (1992) Selective attention and visual-search—revision of an allocation model and application to age-differences. *J Exp Psychol Hum Percept Perform* 18:821–836
- Marois R, Ivanoff J (2005) Capacity limits of information processing in the brain. *Trends Cogn Sci* 9:296–305
- Martens S, Johnson A (2005) Timing attention: cuing target onset interval attenuates the attentional blink. *Mem Cogn* 33:234–240
- Miller EK (2000) The prefrontal cortex and cognitive control. *Nat Rev Neurosci* 1:59–65
- Miller BT, D'Esposito M (2005) Searching for “the top” in top-down control. *Neuron* 48:535–538
- Muller HJ, Rabbitt PMA (1989) Reflexive and voluntary orienting of visual-attention—time course of activation and resistance to interruption. *J Exp Psychol Hum Percept Perform* 15:315–330
- Nakayama K, Mackeben M (1989) Sustained and transient components of focal visual-attention. *Vision Res* 29:1631–1647
- Nieuwenhuis S, Gilzenrat MS, Holmes BD, Cohen JD (2005) The role of the locus coeruleus in mediating the attentional blink: a neuro-computational theory. *J Exp Psychol Gen* 134:291–307
- Nieuwenstein MR (2006) Top-down controlled, delayed selection in the attentional blink. *J Exp Psychol Hum Percept Perform* 32:973–985
- Nieuwenstein MR, Chun MM, van der Lubbe RHJ, Hooge ITC (2005) Delayed attentional engagement in the attentional blink. *J Exp Psychol Hum Percept Perform* 31:1463–1475
- Olivers CNL (2007) The time course of attention: it's better than we thought. *Curr Dir Psychol Sci* 16:11–15
- Olivers CNL, van der Stigchel S, Hulleman J (2007) Spreading the sparing: against a limited-capacity account of the attentional blink. *Psychol Res Psychol Forsch* 71:126–139
- Posner MI (1980) Orienting of attention. *Q J Exp Psychol* 32:3–25
- 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* 24:979–992
- Praamstra P, Boutsen L, Humphreys GW (2005) Frontoparietal control of spatial attention and motor intention in human EEG. *J Neurophysiol* 94:764–774
- 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
- Riggio L, Kirsner K (1997) The relationship between central cues and peripheral cues in covert visual orientation. *Percept Psychophys* 59:885–899
- Ristic J, Friesen CK, Kingstone A (2002) Are eyes special? it depends on how you look at it. *Psychon Bull Rev* 9:507–513
- Rounis E, Stephan KE, Lee L, Siebner HR, Pesenti A, Friston KJ, Rothwell JC, Frackowiak RSJ (2006) Acute changes in frontoparietal activity after repetitive transcranial magnetic stimulation over the dorsolateral prefrontal cortex in a cued reaction time task. *J Neurosci* 26:9629–9638
- 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
- Slagter HA, Kok A, Mol N, Talsma D, Kenemans JL (2005) Generating spatial and nonspatial attentional control: an ERP study. *Psychophysiology* 42:428–439
- Vossel S, Thiel CM, Fink GR (2006) Cue validity modulates the neural correlates of covert endogenous orienting of attention in parietal and frontal cortex. *Neuroimage* 32:1257–1264
- Vul E, Nieuwenstein MR, Kanwisher N (2007) Temporal selection is suppressed, delayed, and diffused during the attentional blink. *Psychol Sci* (in press)
- Ward R, Duncan J, Shapiro K (1996) The slow time-course of visual attention. *Cogn Psychol* 30:79–109
- Yantis S, Serences JT (2003) Cortical mechanisms of space-based and object-based attentional control. *Curr Opin Neurobiol* 13:187–193