Two-timing attention

René Marois

The attentional blink reveals a fundamental limit in the temporal resolution of attention. By describing the entire sequence of electrophysiological events underlying the blink, a new study provides the first glimpse into the neural cause of this bottleneck.

Imagine that, as a participant in a psychological experiment, your task is to detect two letters appearing within a rapid stream of digits presented at the center of gaze, with each character shown only for one–tenth of a second. How will you fare? Dozens of such experiments predict that you will have little difficulty in reporting the first of the two target letters; however, if the second letter appears within about half a second of the first, your performance in detecting that second target may be pitiful[1]. Skeptical? Try it yourself: www.cs.kent.ac.uk/people/rpg/pc52/AB_Webscript/instr.html.

This transient impairment in detecting the second of two targets, termed the attentional blink[2], reveals a severe temporal limitation in our ability to consciously attend to multiple visual events. To put this limitation in perspective, consider that neurons in visual cortex can temporally resolve spikes at rates 200–500 times our capacity to temporally resolve visual targets. How is it that our sophisticated nervous system, equipped with massive parallel processing capacity and comprising 100 billion high-speed processors, yields such poor behavioral performance[3]? In this issue Sergent *et al.* [4] bring us closer to solving this mystery.

It has long been recognized that not all stages of visual information processing are highly efficient. For example, Duncan[5] proposed a two-stage model of visual cognition in which an early stage that permits the rapid, initial evaluation of visual stimuli is followed by a second attentionally demanding, capacity-limited stage that is essential for the conscious report of the stimuli. Such duality in visual cognition is well illustrated by the attentional blink[1,6]: the ease with which the first target (T1) is detected indicates that our visual system can register essentially all the stimuli, even at presentation rates as high as ten per second. In contrast, the difficulty in reporting the second target (T2) reveals a profound temporal limitation in conscious target perception.

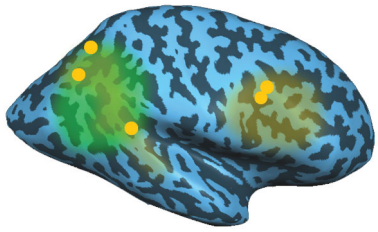

Figure 1 Neuroanatomical substrates of the attentional blink. The gold circles represent activation foci from fMRI studies, whereas the light- and dark-green shaded regions represent areas implicated in lesion and MEG studies, respectively. Together these studies suggest a lateral frontoparietal network as a key neural substrate underlying the attentional blink 'bottleneck'. Adapted from ref. 3.

Although several theories have been proposed to account for the attentional blink[7,8], Chun and Potter's two-stage model[6]—an elaboration of Duncan's model for target detection in rapid serial displays—has received considerable support from the neurobiological literature. Event-related potential (ERP) studies have shown that T2 target words that are not explicitly perceived in an attentional blink situation, nonetheless elicit electrophysiological markers of visual and semantic processing[9,10]; successful detection of T2, however, elicits a P300 response[10], indicating that working memory is being updated. Thus, T2 targets that do not reach working memory are processed by the brain, as predicted by the two-stage model of the attentional blink. Neuropsychological[11] and functional magnetic resonance imaging (fMRI) studies[12,13] complement the ERP results by pinpointing the neural substrates underlying the attentional blink 'bottleneck', the capacity-limited second stage at which T2 is blocked from conscious perception. These studies have not only implicated a lateral frontoparietal network[14] as the neural substrate underlying this bottleneck, but also have revealed activity in the visual, but not frontal, cortex even when subjects failed to detect T2[12,13] (**Fig. 1**). What emerges from this research is a neural account of the two-stage model: activity in visual cortex would correspond to stage 1, whereas target processing in the frontoparietal cortex would correspond to stage 2 (ref. 3).

Despite what these studies have revealed about the attentional blink, they provide little information about the neural processes that prevent two successive targets from reaching consciousness. Also, because the classical attentional blink experiment requires a categorical response (such as: "Did you see the target or not?"), it may not be too surprising that these neurobiological studies are consistent with the two-stage model. A more rigorous way to test this model would be to determine whether a similar dichotomous pattern of brain activity occurs when the task requires a perceptual judgment across a continuum. The new study by Sergent and colleagues[4] addressed both of these issues.

To investigate the 'quantal' nature of awareness in the attentional blink, Sergent *et al.* used a modified T2 task that, rather than calling for target detection or identification, required observers to rate the visibility of each T2 target word along a continuous range (from 0% visible, indicating that the observer missed the target entirely, to 100% visible). Even though this provides subjects with every opportunity to report a graded conscious percept, the T2 scores clustered around two peaks: a sharp one at 0% and a second, broader one between 60% and 90%. This distribution suggests that subjects either did not see T2 at all or saw it quite well. In keeping with this finding, Sergent *et al.* found that some ERPs, generated 300–500 ms after T2 appeared (for example, the P300), occurred in an all-or-none fashion during the blink, showing essentially no response when perceived visibility was below 50%, and strong but invariant response when perceived visibility was above 50%. By contrast, ERP components that occurred earlier (that is, P1 and N1) were unaffected by the perceived visibility of T2. Not only are these results consistent with prior work[10], but they also strongly support Chun and

René Marois is in the Department of Psychology, 530 Wilson Hall, Vanderbilt University, Nashville, Tennessee 37203, USA.
e-mail: rene.marois@vanderbilt.edu

Potter's two-stage[6] and related neural models of the attentional blink by demonstrating a predominantly dichotomous response pattern in several key ERP components associated with the attentional blink.

How can the finding that early ERP components are unaffected by the perceived visibility of T2 (refs. 4,10) be reconciled with fMRI results showing greater activity in the visual cortex when T2 is consciously perceived than when it is missed[13]? Sergent *et al.* provide a potential resolution. Posterior cortical regions showed late reactivations in trials for which T2 was consciously perceived (see Fig. 5 in Sergent *et al.*[4]). Such reactivation, awareness-related presumably of top-down origin, could account for the fMRI results if one assumes that this re-entrant signal integrates with, and thereby amplifies, the visual cortex's BOLD response to target presentation. By the same token, this late reactivation could also be construed as evidence for a contribution of visual cortex activity to stage 2 of the attentional blink[3].

Although these results help pin down which electrophysiological events and neural foci are involved in the attentional blink, they do not tell us how the attentional blink is triggered. This is where the results of Sergent *et al.* become even more interesting. Chun and Potter's two-stage model[6] proposes that T2 cannot access stage 2 until T1 processing is completed. It therefore follows that the sooner T1 processing is completed, the more likely it is that T2 will access stage 2 before it is degraded or replaced (in stage 1) by ensuing distractors. This is precisely what Sergent *et al.* report[4]. The P300 (P3b) wave for T1 peaked earlier in T2 trials rated as highly visible, suggesting that T2 was more likely to access stage 2—and therefore become consciously visible—when T1 processing was completed earlier. Although the times at which the T1-evoked P300 peaked differed only slightly between the visible and invisible T2 targets, it is worth noting that subjects in an attentional blink experiment are at the threshold of conscious perception; thus even tiny stochastic fluctuations in background neural activity may suffice to tip the balance between visible and invisible T2 targets[8]. Earlier T1 processing would facilitate T2's gaining access to stage 2 before its neural representation in stage 1 fades away. Additional support for this idea comes from the relative timings of early and late ERP waves: the P1 and N1 components of T2, likely indexes of stage 1 processing, terminate at about the same time as the P300 wave for T1 starts.

Taken together, these results are consistent with the view that T2 may not access the capacity-limited second stage of information processing that is necessary for awareness, so long as T1 occupies that stage. These findings not only begin to unmask the neural processes underlying the attentional blink bottleneck, they come tantalizingly close to catching that bottleneck in action.

Much remains to be resolved, however. For one, not all of Sergent and colleagues' ERP data fit squarely into two-stage models of the attentional blink, suggesting that these models do not capture the entire cascade of electrophysiological events leading to the blink. In addition, although using a continuous rating scale for the T2 task allowed Sergent *et al.* to provide evidence against a graded nature of conscious perception during the attentional blink, the precise contents of that perception could not be established because subjects were not asked to report the identity of the T2 target. Thus, it will be important to determine exactly what conscious process or processes are reflected by the different ERP components. Indeed, only once we have clearly identified the cognitive processes indexed by each of the neurophysiological events implicated in the blink, and determined how these events are affected by experimental perturbations[12,15], will we have made significant progress into understanding the root of this perceptual deficit. The study by Sergent and colleagues certainly points the way.

1. Broadbent, D.E. & Broadbent, M.H. *Percept. Psychophys.* **42**, 105–113 (1987).
2. Raymond, J.E., Shapiro, K.L. & Arnell, K.M. *J. Exp. Psychol. Hum. Percept. Perform.* **18**, 849–860 (1992).
3. Marois, R. & Ivanoff, J. *Trends Cogn. Sci.* **9**, 296–304 (2005).
4. Sergent, C., Baillet, S. & Dehaene, S. *Nat. Neurosci.* **8**, 1391–1400 (2005).
5. Duncan, J. *Psychol. Rev.* **87**, 272–300 (1980).
6. Chun, M.M. & Potter, M.C. *J. Exp. Psychol. Hum. Percept. Perform.* **21**, 109–127 (1995).
7. Shapiro, K.L., Arnell, K.M. & Raymond, J.E. *Trends Cogn. Sci.* **1**, 291–296 (1997).
8. Dehaene, S., Sergent, C. & Changeux, J.P. *Proc. Natl. Acad. Sci. USA* **100**, 8520–8525 (2003).
9. Luck, S.J., Vogel, E.K. & Shapiro, K.L. *Nature* **383**, 616–618 (1996).
10. Vogel, E.K., Luck, S.J. & Shapiro, K.L. *J. Exp. Psychol. Hum. Percept. Perform.* **24**, 1656–1674 (1998).
11. Shapiro, K., Hillstrom, A.P. & Husain, M. *Curr. Biol.* **12**, 1320–1325 (2002).
12. Marois, R., Chun, M.M. & Gore, J.C. *Neuron* **28**, 299–308 (2000).
13. Marois, R., Yi, D.J. & Chun, M.M. *Neuron* **41**, 465–472 (2004).
14. Gross, J. *et al. Proc. Natl. Acad. Sci. USA* **101**, 13050–13055 (2004).
15. Vogel, E. & Luck, S.J. *Psychon. Bull. Rev.* **9**, 739–743 (2002).

Orexin, drugs and motivated behaviors

Thomas E Scammell & Clifford B Saper

Orexins are known to regulate sleep and feeding, but a study in *Nature* now shows that they are also involved in drug-seeking behavior. This suggests a larger role for orexin-producing neurons as an interface between internal states and motivated behaviors.

Narcolepsy is a sleep disorder caused by the selective loss of the hypothalamic neurons that produce orexin-A and orexin-B (hypocretin-1 and -2). Regardless of how much sleep they get at night, people with narcolepsy often struggle to stay awake during the day. Many require treatment with stimulant medications such as amphetamines that are potentially addictive, but surprisingly few become addicted to these drugs[1]. Harris and colleagues now present a novel perspective on how the orexin peptides may drive drug-seeking behavior[2]. Their findings may, in turn, explain why so many patients with narcolepsy seem able to avoid drug addiction.

Harris and colleagues used an apparatus with two chambers, in one of which they injected rats with morphine or cocaine. They found that the animals later spent more time in that chamber than in the neutral one, even in the absence of drug. This conditioned place preference was associated with activation of orexin neurons in the lateral hypothalamus, as indicated by increased expression of Fos protein. Rats conditioned to morphine demonstrated much less place

Thomas E. Scamell and Clifford B. Saper are at the Department of Neurology, Beth Israel Deaconess Medical Center, 77 Avenue Louis Pasteur, Boston, Massachusetts 02115, USA.
e-mail: tscammel@bidmc.harvard.edu