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Adaptive control of event integration: Evidence from event-related potentials

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Abstract

We investigated whether it is possible to control the temporal window of attention used to rapidly integrate visual information. To study the underlying neural mechanisms, we recorded ERPs in an attentional blink task, known to elicit Lag-1 sparing. Lag-1 sparing fosters joint integration of the two targets, evidenced by increased order errors. Short versus long integration windows were induced by showing participants mostly fast or slow stimuli. Participants expecting slow speed used a longer integration window, increasing joint integration. Difference waves showed an early (200 ms post-T2) negative and a late positive modulation (390 ms) in the fast group, but not in the slow group. The modulations suggest the creation of a separate event for T2, which is not needed in the slow group, where targets were often jointly integrated. This suggests that attention can be guided by global expectations of presentation speed within tens of milliseconds.

Descriptors: Attentional blink, Event-related potentials, Lag-1 sparing, Event integration

Human attention has been shown to be fallible when multiple events need to be processed within an interval of less than 500 ms. Possibly the most striking example of this is the attentional blink (AB) phenomenon, in which the second of two target items in a rapid serial visual presentation (RSVP) stream is often missed when it follows (successful identification of) the first target within about half a second (Broadbent & Broadbent, 1987; Raymond, Shapiro, & Arnell, 1992). The AB illustrates a severe temporal limitation in human perceptual processing that is not easy to overcome. In fact, a particularly remarkable observation about the AB is the finding that increased effort does not seem to improve performance to a level that would overcome the blink deficit. Indeed, there have even been recent reports suggesting the opposite (Olivers & Nieuwenhuis, 2005, 2006). This is somewhat paradoxical, as humans are generally able to adapt to challenging tasks with effort and practice. There are two possible accounts that may explain this finding. The first is that it is simply not possible to optimize performance under RSVP conditions, because the task is too challenging. The second is that optimization does take place, but that it is not sufficient to overcome the attentional bottleneck. Evidence from the Lag-1 sparing phenomenon suggests that the second account is more likely.

Lag-1 sparing occurs when a second target (T2) follows the first target (T1) with no intervening distractor or mask and when no task or location switch is needed between targets (Visser, Bischof, & Di Lollo, 1999). Under these circumstances, identification accuracy on T2 can be as high as it is outside of the AB interval. An initial account of Lag-1 sparing was given by Shapiro, Raymond, and Arnell (1994), who ascribed it to a mechanism involved in setting the attentional filter or gate. They assumed that when Lag-1 sparing occurred, the attentional gate had been slow to close, and hence the second target was able to slip in. It has been suggested that stimuli that do not match the target template act as a closing signal for the attentional gate (Di Lollo, Kawahara, Ghorashi & Enns, 2005). However, because there is no conflicting information coming in between targets when T2 is presented at Lag 1, there is no trigger for the attentional gate to close and to prevent task-irrelevant information from getting in. The high performance on T2 can then be attributed to it being integrated together with T1 in one attentional episode. It is only after the gate closes that any new task-relevant information would require the initiation of a new attentional episode, the costs of which are shown by the magnitude of the AB. Recent reports suggest that the chaining of target items allows extended joint integration up to three items after T1 (Di Lollo et al., 2005; Olivers, van der Stigchel, & Hulleman, 2007). In essence, these studies are consistent with the idea that the attentional gate is flexibly adaptable such that it can accommodate different levels of incoming task-relevant information. This would be an instance of exogenous control over attentional integration.

It has to be noted that the Lag 1 sparing phenomenon is not universally beneficial, but is also a trade-off in a sense: When two targets are integrated into the same attentional episode, temporal

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information tends to be lost (Hommel & Akyürek, 2005; Potter, Staub, & O'Connor, 2002). As a result, when Lag 0 and Lag 1 responses are examined for identity as well as order information, a large proportion are correctly identified, but in the reverse order (henceforth referred to as "order errors"). Because this is most likely to occur if the two events have been integrated within one attentional episode, the prevalence of order errors can be used as a measure of joint integration.[1]

Akyürek, Toffanin, and Hommel (2006) used this response pattern to diagnose whether event integration can be adapted endogenously to task constraints by manipulating task expectations in two groups of participants. One group was randomly presented with a large number of relatively fast RSVP trials and only a small number of slow ones, whereas a second group received the opposite distribution. The idea was that the first group would be led to expect fast trials whereas the second group would expect slow trials. If the integration window is under endogenous control, this expectation should affect the attentional setting and, thus, the length of the attentional window. When a fast trial is expected, a short integration window would help to minimize distractor interference. On the other hand, when a slow trial is expected, a "relaxed" time window would maximize the time available to collect target information. At the same time, however, this could lead to the attentional gate being open for longer than necessary when a fast trial is presented unexpectedly. This would result in an increased chance of T2 being integrated into one attentional episode with T1 at Lag 1.

Akyürek et al. (2006) results indeed showed an increase in order errors at Lag 1 on fast trials in the slow expectation group, relative to performance on the same trials in the fast expectation group. This supported the case for adaptive endogenous control. Furthermore, the expectation effect was also found when slow and fast trials had identical interstimulus intervals (ISIs), and only the ratio of actual stimulus duration to blank duration was changed. In the second and third experiments of Akyürek et al., each stimulus remained on screen for 70 ms with a subsequent 30-ms blank in the "slow" condition, and for 30 ms with a 70-ms blank in the "fast" condition. Under these circumstances, although the illusion of speed with these displays was apparent, there was no real reason to adjust integration time, as the second target would arrive 100 ms after the first in both conditions so that the probability of joint integration should be the same for long and short integration windows. Despite that, participants still acted on the perceived speed difference in the same way, that is, they made more order errors on fast trials with a slow trial speed expectation by extending their integration window.

The shift in the prevalence of order errors is evidence for adaptive control over attentional selection, but it does not shed light on when the critical processes take place and which neural mechanisms are underlying them. Indeed, there might be multiple processes contributing to the eventual behavioral result of order errors. For instance, the behavioral findings offer no real way of discriminating between effects on early selection and memory processing. Recurrent feedback between attention and memory is also likely to occur, given the close link between attentional selection and working memory (de Fockert, Rees, Frith, & Lavie, 2001). For effective control of attentional resources, it is necessary to recall specific filter settings and the results these produced in the recent past, so that frequently successful settings are more likely to be maintained and less successful ones can be modified or replaced. The present study sought to investigate the timing of attentional adaptation by recording event-related potentials (ERPs) by using the equal ISI version of the adaptation task that was used by Akyürek et al. (2006). The constancy of timing in this version allowed for a more direct comparison of the ERPs in both fast and slow expectation conditions.

A number of ERP components have previously been identified in the AB paradigm. Vogel, Luck, and Shapiro (1998) reported the N1 and P1 components, associated with sensory processing, both of which persisted through the AB. Because studies of spatial attention have shown suppression of these components at ignored locations (e.g., Luck et al., 1994), the persistence of the N1 and P1 is evidence that the blink is a relatively late cognitive bottleneck and cannot be attributed to perceptual (sensory) difficulties. Most theories of the AB are in line with that assertion (e.g., Chun & Potter, 1995). Vogel et al. further demonstrated that the P3 and N4 (or N400) are also elicited and that the P3, but not the N4, is correlated with the presence of the blink (i.e., the P3 was suppressed at lags within the blink interval). This result was later confirmed by more direct comparisons of actual missed and detected trials by Rolke, Heil, Streb, and Hennighausen (2001), Kranczioch, Debener, and Engel (2003), and Shapiro, Schmitz, Martens, Hommel, and Schnitzler (2006). The P3 is thought to reflect processing related to working memory (e.g., Kok, 2001). Hence, the absence of the P3 on blink trials can be thought of as a reflection of the failure of the representation of T2 to reach (or to be consolidated in) memory. At the same time, the N4 component, typically associated with semantic processing (Kutas & Hillyard, 1980) was found to be independent of the AB, which is evidence for semantic processing of stimuli even when they cannot be consciously reported. Such processing is likely to be the cause of priming effects of missed targets on subsequent stimuli found previously (Shapiro, Driver, Ward, & Sorensen, 1997). Both Vogel et al. and Kranczioch et al. made mention of a P2 component, but acknowledged it could not be clearly interpreted in their studies. In fact, although Vogel et al. found the P2 to behave similarly to the P3 component at blinked lags, Kranczioch et al. did not observe a similar suppression of the P2 component. Recent work by Sergent, Baillet, and Dehaene (2005) has provided a further index of the ERP components elicited by T2 in AB type tasks by using a target-mask version of the task and obtaining target report on a continuous visibility scale. In particular, although Sergent et al. did not report a P2 component, they did report additional N2 and N3 components that were modulated by the visibility of the target as rated by their participants. In this sense, these negative components seemed to act in a manner similar to that of the P3; a loss of conscious perception was associated with a reduction in their amplitude. Functionally speaking, it has to be noted that the underlying causes of these components remain relatively unclear.

In the present study, the components elicited by T2 were expected to show only when joint integration of targets did not occur, because that would require the initiation of a new event episode for T2, rather than it being able to use the one already created for T1. In particular, the components associated with

[1]One may consider the degree of Lag-1 sparing an equally valid or perhaps even better measure of integration. Unfortunately, however, joint integration of two or more targets increases competition between target identities, which can lead to memory loss (Hommel & Akyürek, 2005; Potter et al., 2002). This means that Lag-1 sparing reflects both gains from joint integration and losses through competition, whereas order errors are a relatively pure measure of integration.

successful report, that is, the N2, N3, and the P3, were expected to show when a new event episode had to be created for T2. The creation of a second event episode (for T2) should be more common in the fast group than in the slow group, as the increase in joint integration in the slow group should lead to T2 being taken aboard with the event representing T1, which means there is no need to create another event episode to represent T2 in that case. The earlier N1 and P1 components are thought to be associated with visual perception mostly, and therefore they should occur regardless of whether targets were jointly integrated or not, depending only on visual clarity (which was not manipulated in the present study). Similarly, the N4 has proven to be insensitive to the blink, which in the context of the present study means that semantic processing is likely to proceed whether there is one event episode or two. In summary then, evidence for N2-, N3-, or P3-related modulation would prove that the order error effect is a true reflection of online attentional processing. The absence of a modulation of the ERP for these components, on the other hand, would point to a pure memory-based source of the increase in order errors observed at Lag 1 when participants have a slow speed expectation.

Method

Participants

Thirty right-handed students at the University of Reading (21 female and 9 male) volunteered to participate in the experiment. Some of these received course credit or monetary compensation for their time. All of them reported normal or corrected visual acuity and reported having no history of neurological problems. Mean age was 22 years.

Apparatus and Procedure

The experiment received ethical approval after review by The University of Reading Ethics and Research Committee. Stimulus presentation and behavioral data collection was handled by a Pentium 4 computer running the E-Prime software (version 1.2) with the Biological Add-ons package. Stimuli were drawn on a 17-in. CRT monitor. Refresh rate was set at 100 Hz at a resolution of 800 × 600 pixels in 16-bit color. Average viewing distance was approximately 50 cm, but was not fixed. Within groups, the design consisted of one variable: T2 Lag, which specified the temporal distance between T1 and T2. Apart from Lag 1, Lags 3 (300 ms post-T1) and 8 (800 ms) were chosen to measure performance within and outside of the AB interval. Between groups, different expectations of stimulus speed were induced in participants by presenting either predominantly fast or predominantly slow trials in random order.

Participants initiated each trial by pressing the Enter key on the keyboard. After a blank delay of 200 ms, a red (RGB 255, 0, 0) fixation cross appeared in the center of the screen for 250 ms, on a light gray background (RGB 128, 128, 128). The background persisted throughout the trial. After another brief blank delay of 50 ms the RSVP ensued, consisting of 19 stimuli in total, all of which were presented in black (RGB 0, 0, 0) 18-pt bold Courier New font. Two target digit numbers selected randomly from 2 to 9 without replacement were presented as the target items. The first target was either the fifth or the seventh item in the stream. The second target followed the first as the first, third, or eighth item (i.e., lags 1, 3, and 8). The remaining stimuli (i.e., the distractors) were capital letters again selected without replacement from the complete alphabet. Between stimuli, a brief ISI was inserted. Each stimulus lasted for 30 ms with an ISI of 70 ms in the "fast" condition, and each stimulus lasted for 70 ms with an ISI of 30 ms in the "slow" condition. After the presentation of the last RSVP item, participants were asked to enter the first digit number. Once they had done so, they were asked to enter the second. After a final blank pause of 100 ms, the next trial was initiated. Fast and slow trials were distributed as follows per group: the "fast expectation" group was shown 80% fast trials and 20% slow trials (randomly intermixed), whereas the opposite distribution was shown to the "slow expectation" group. There were 500 trials in total, 20 of which were practice trials and not considered for analysis. It took on average about 50 min to work through all trials.

Electrophysiological Recording

Electroencephalographic (EEG) activity was recorded using an Electrical Geodesics Inc GSN 200 sensor net, which featured 128 silver/silver-chloride electrodes. EEG was amplified by the EGI NetAmps 200 amplifier with a bandpass of 0.1–100 Hz. Digitization was performed at a sample rate of 250 Hz. Impedances were kept below 50 kΩ. Prior to analysis, a 0.5–20 Hz bandpass filter (2 Hz roll-off) was applied. All sites were recorded with a vertex reference and the data were re-referenced off-line using the polar average reference effect (PARE) corrected procedure (see Junghöfer, Elbert, Tucker, & Braun, 1999). The electrooculogram was recorded using sensor pairs 125 and 128 for the horizontal, and pairs 26 and 127, 8 and 126, 33 and 127, and 1 and 126 for the vertical movements. Trials were scanned for voltage exceeding ± 200 μV, transient voltages exceeding ± 100 μV, oculogram activity higher than ± 70 μV, and flatlining and excluded from the averaged waveforms if any of these events occurred. An average of 10.5% of trials were excluded for each participant, with a minimum of 0.4% and a maximum of 30.0%. These trials were fairly evenly distributed between fast and slow trials: 4.9% of these were fast trials and 5.6% were slow ones. All of the remaining trials were used to compute the ERP averages.

The existing ERP studies of the AB guided the design of the present study. In particular, the time course between 0 and 500 ms after T2 was studied, where the bulk of the components seem to occur. Furthermore, electrode sites were clustered around the midline, because previous work has more or less shown that there is no lateral component of interest in the ERP elicited by central RSVP (Kranczioch et al., 2003; Sergent et al., 2005; Vogel et al., 1998). Averaged ERPs were time-locked to the onset of T2 and baseline corrected to an interval of 200 ms pre-T1. Because the RSVP creates the problem of overlapping ERPs generated by the sequence of incoming stimuli (see Vogel et al., 1998), difference waves were computed by subtracting the infrequent trial type from the frequent one. Although the reverse subtraction could be performed as well (which has the trivial effect of reversing the sign of the waves), the rationale was to treat the most common trial type as the base "signature" of the session. The subtraction of the infrequent trial type takes away all activation common to both trial types, leaving that which is unique in the infrequent type. This approach also keeps the distribution of trials the same in both groups, thereby equating noise. In essence then, the difference waves represent the difference between evoked potentials on slow and on fast trials. Because the difference wave approach takes away all that is common to the experimental conditions, the typical components modulated by the AB were not expected to

show. For example, a P3 elicited by T2 at Lag 8 would be present regardless of whether a slow or fast expectation was held and therefore would be canceled out. Instead, the modulation due to joint integration, which is specific to Lag 1, was the main issue of interest. Mean amplitude was measured in an early time window of 100–250 ms and a late window of 250-500 ms post-T2 onset and statistically tested using analysis of variance (ANOVA). When necessary due to a significant test of sphericity, the probability values were adjusted using the Greenhouse–Geisser epsilon correction. Three electrode groups were added to the ANOVA of the electrophysiological data to determine global scalp distributions, with a particular focus on the midline. Each group comprised 13 clustered electrodes. The first sensor group was the most anterior (consisting of sensors 19, 16, 10, 20, 11, 4, 21, 12, 5, 119, 13, 6, and 113), the second group was centered on the vertex (31, 7, 107, 106, 38, 32, VREF, 81, 88, 54, 55, 80, and 62), and the third group was the most posterior (61, 68, 79, 60, 67, 73, 78, 86, 66, 72, 77, 85, and 76). Figure 1 shows the sensor clusters highlighted on the EGI GSN 200 128 channel net.

Results

Behavior

T1 identification accuracy is shown in the left panel of Figure 2 (closed symbols). T1 accuracy was particularly low at Lag 1 (84.1%), compared to the other lags (90.6% correct on Lag 3 and 91.0% on Lag 8), and this was reflected in a main effect of T2 Lag, $F(2,56) = 38.13$, $MSE = .001$, $p < .001$, $\varepsilon = .83$. This pattern replicated previous findings (cf. Hommel & Akyürek, 2005). The drop at Lag 1 is often observed in situations of joint integration and is thought to reflect a degree of competition between targets (Potter et al., 2002). There were no further significant effects; neither the main effect of expected speed ($F < 1$) nor its interaction with T2 Lag ($F < 1$) was significant.

T2 identification accuracy, given that T1 was identified correctly (T2|T1), is shown in the left panel of Figure 2 (open symbols). Only the T2 Lag variable had an impact on T2 accuracy, $F(2,56) = 105.17$, $MSE = .018$, $p < .001$, $\varepsilon = .67$, which reflected a typical AB with relatively good performance at Lag 1 (92.0%) and Lag 8 (82.7%), but poor performance at Lag 3 (52.3%). Again, there was no significant main effect of expected speed ($F < 1$), or interactions ($F < 1$).

Finally, the number of order errors as an index of joint integration was considered. The right panel of Figure 2 shows the number of order errors as a percentage of the total number of trials. This clearly shows the large number of order errors at Lag 1, $F(2,56) = 374.57$, $MSE = .003$, $p < .001$, $\varepsilon = .67$. Furthermore, more errors of this kind were made in the slow-expectation group (30.5%) than in the fast-expectation group (25.6%), $F(2,56) = 5.04$, $MSE = .002$, $p < .01$. These results essentially replicate Akyürek et al. (2006), supporting the hypothesis that a

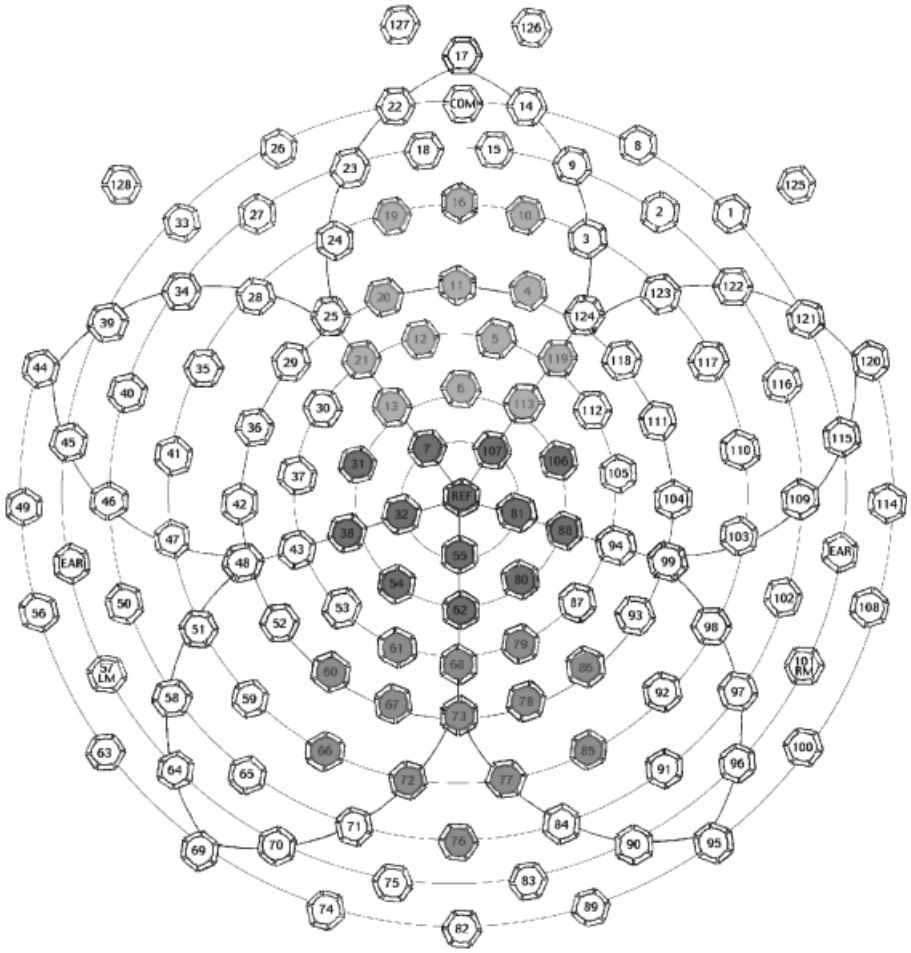

Figure 1. Sensor layout of the EGI GSN 200 net, with the anterior, central, and posterior clusters used in the analyses highlighted. Anterior sensors are light gray, central sensors are dark gray, and posterior sensors are medium gray.

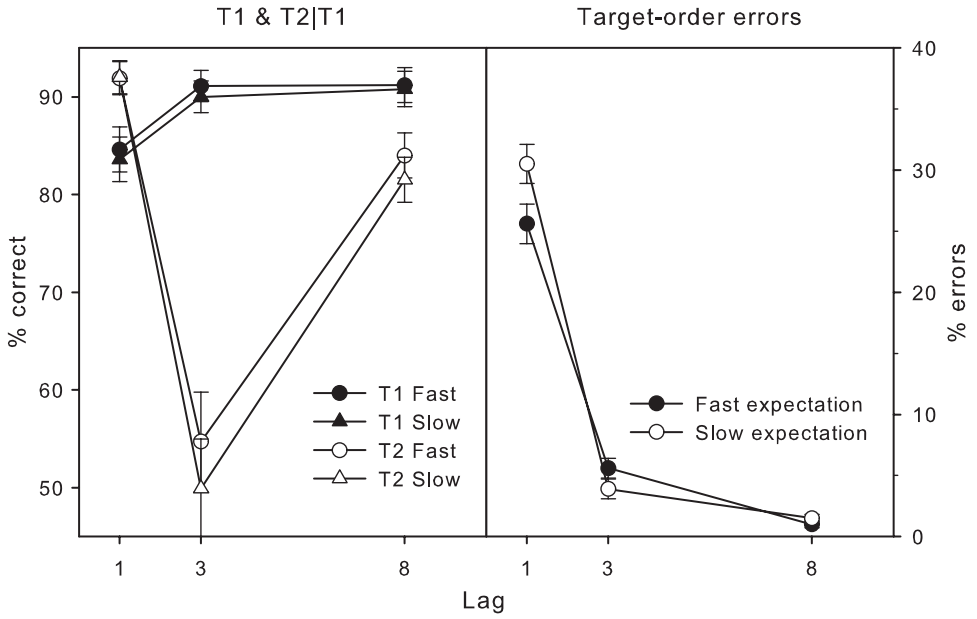

Figure 2. Percentage of correctly identified targets plotted as a function of lag (left panel). Closed symbols represent T1 performance and open symbols represent T2 performance. Separate lines are drawn for fast and slow expectation groups. Percentage of order errors of the total number of trials plotted as a function of lag, for both expectations (right panel).

relatively long window of integration increases the chances of T2 being jointly integrated with T1, thus preserving the identities of both targets but sacrificing their order. Overall, the main effect of expected speed was not reliable, $F(1,28) = 2.02$, $MSE = .002$, $p > .17$.

Electrophysiology

For the fast expectation group, in the early time window (100–250 ms), a negative modulation was apparent at Lag 1 (-2.88 μV), which was not reliable at Lag 3 (-1.08 μV) and Lag 8 (-1.19 μV), $F(2,28) = 4.67$, $MSE = 15.66$, $p < .05$, $\varepsilon = .62$. Figure 3 shows a four-sample average topographical representation based on spherical spline interpolation of the observed differences for both fast and slow groups at 200 ms post-T2 (based on waveform examination). Neither the effect of sensor cluster, nor its interaction with lag was significant, which is evidenced by the rather uniform negativity in the Lag 1 condition shown in Figure 3.

In the late time window (250–500 ms), a positive modulation was present at Lag 1 (3.05 μV), which was largely absent at Lag 3 (0.08 μV) and much weaker at Lag 8 (1.07 μV), $F(2,28) = 19.70$, $MSE = 5.17$, $p < .001$. This positive modulation was stronger in central (1.85 μV) and posterior (1.60 μV) sensor clusters than in the anterior one (0.74 μV), $F(2,28) = 10.24$, $MSE = 1.50$, $p < .001$. This was furthermore only the case in the Lag 1 condition, as evidenced by an interaction between lag and sensor cluster, $F(4,56) = 4.02$, $MSE = 6.98$, $p < .05$, $\varepsilon = .48$. Tukey post hoc tests confirmed that there was a difference between anterior and central sensor clusters at Lag 1 ($q = 4.550$, $t = 4.066$, $p < .05$), but not at Lag 3 ($t = .732$) nor at Lag 8 ($t = .220$). Figure 4 shows a topographical map of the differences at 390 ms post-T2 (a time again chosen for representative quality) for both expectation groups. The time course of the differential activation recorded at electrodes in the center of each sensor cluster is plotted in Figure 5.

The effects observed in the fast expectation group stood in contrast with the analysis of the slow expectation group. Here, T2 lag did not reach significance at all ($F < 1.58$ in the early window, and $F < 1$ in the late window). The overall lack of relevant differences in this group is visible in the bottom rows of Figures 3, 4, and 5.

To quantify the apparent contrast between fast and slow expectation groups, the data were put together and expectation group was added as a between-subjects variable. In the early time window, group was significant, $F(1,28) = 30.49$, $MSE = 2.53$, $p < .001$. Group also interacted with lag, $F(2,56) = 4.93$, $MSE = 6.58$, $p < .05$. These effects indicated that there was a negative modulation of brain activity in the fast expectation group at Lag 1 (-2.86 μV), which was not there in the slow group (-0.45 μV). Tukey tests confirmed that there was a reliable difference between the fast and the slow group at Lag 1 ($q = 4.163$, $t = 3.820$, $p < .05$), but not at Lag 3 ($t = 1.804$) and Lag 8 ($t = .363$).

In the late time window, there were overall differences between groups, $F(1,28) = 4.43$, $MSE = 4.66$, $p < .05$, between lags, $F(2,56) = 10.35$, $MSE = 5.10$, $p < .001$, and between sensor clusters, $F(2,56) = 4.29$, $MSE = 3.16$, $p < .05$, $\varepsilon = .67$. Each of the more specific interactions was also significant: The interaction between lag and group, $F(2,56) = 10.03$, $MSE = 5.10$, $p < .001$, between sensor cluster and group, $F(2,56) = 3.20$, $MSE = 3.16$, $p < .05$, and finally between lag and sensor cluster, $F(4,112) = 5.31$, $MSE = 4.51$, $p < .01$, $\varepsilon = .53$. These effects were mainly caused by the presence of a pronounced difference between Lag 1 and the other lags in the fast group (3.04 μV vs. 0.08 μV and 1.07 μV for Lag 3 and 8), which was lacking in the slow group. Tukey tests confirmed this interpretation and showed that the fast group showed a reliably more positive activation than the slow group at Lag 1 ($q = 4.163$, $t = 4.161$, $p < .05$), but not at Lag 3 ($t = 2.210$, in the opposite direction) and Lag 8 ($t = .897$). The main difference between groups was found in the central sensor cluster ($q = 4.163$, $t = 3.306$, $p < .05$), with a weaker trend in the

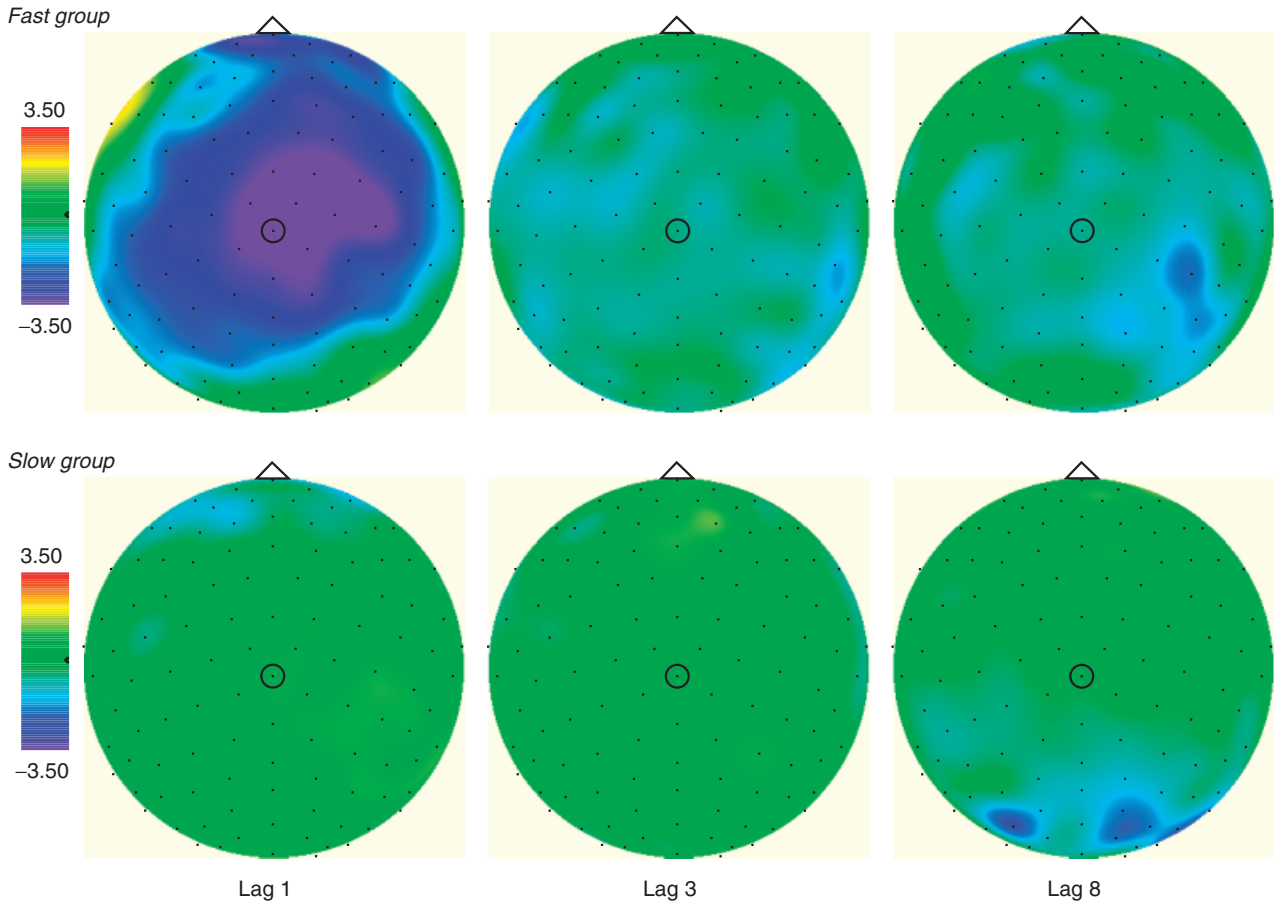

Figure 3. Topographical maps of differential activation in microvolt at 200 ms after T2. The top row shows activation in the fast expectation group for each lag, whereas the bottom row shows the same for the slow expectation group. The vertex has been circled and the nose is represented at the top of each map.

same direction at the posterior cluster ($t = 1.528$) and virtually nothing at the anterior sites ($t = 0.071$). Finally, the positive modulation in central and posterior sensor clusters differed reliably from the activation in the anterior cluster ($q = 4.468$, $t = 3.734$, $p < .05$, and $t = 3.328$, $p < .05$, respectively) at Lag 1, but not at Lags 3 and 8 (all $ts < 1$). The three-way interaction term was not reliable. As was the case in the early time window, the differences between groups that emerged from the individual analyses were confirmed. For reference, the raw waveforms at central electrodes in each cluster underlying the observed differences at Lag 1 are shown in Figure A1 in the Appendix.

In summary, the pattern of results was quite clear: There were differences in the evoked potentials between fast and slow trials at Lag 1 when a fast expectation was held, but no such difference emerged when a slow expectation was maintained instead. The joint integration account explains the occurrence of Lag-1 sparing, and the increase in order errors, by the difference between creating two event episodes and just one. From that perspective, the T2-related modulations observed in the fast expectation group could reflect the creation of the second event episode. The absence of a clear signature of the dual-target, singular event episode in the case of Lag-1 sparing should be regarded with caution, as T1-locked ERPs were not examined. Given the variable interval (and events) between T1 and T2, it would be difficult to establish a decisive analysis of T1-locked ERPs in the present paradigm.

Discussion

If the logic behind the joint integration account holds, then the direct result of the tendency to integrate both targets into one event episode in the slow expectation group is that the second episode normally created for T2 is no longer present. The differences elicited by T2 in the current study support this idea. More specifically, at least some of the components previously reported by others could indeed be involved in the initiation of a second event episode presently observed at Lag 1 in the fast expectation group. The negative modulation peaking near 200 ms observed in the current study could be compared to the N2 component observed by Sergent et al. (2005). Theoretically, because the N1 and P1 components are generally associated with the very early sensory processing of the stimulus and because they have been shown to persist throughout the blink, a modulation of these components seems unlikely. That is, the visual perception of the stimulus should not have been affected by our manipulations. The N2, however, is suppressed when rated visibility decreases (Sergent et al., 2005). In the present paradigm a N2 component would have occurred when a new event episode had to be created for T2 (when there was no sparing) but not when both targets were integrated together. In other words, the components elicited by T1 would serve the same role when the T1 episode also comprises T2 and the targets are integrated together. The observed modulation supports this account.

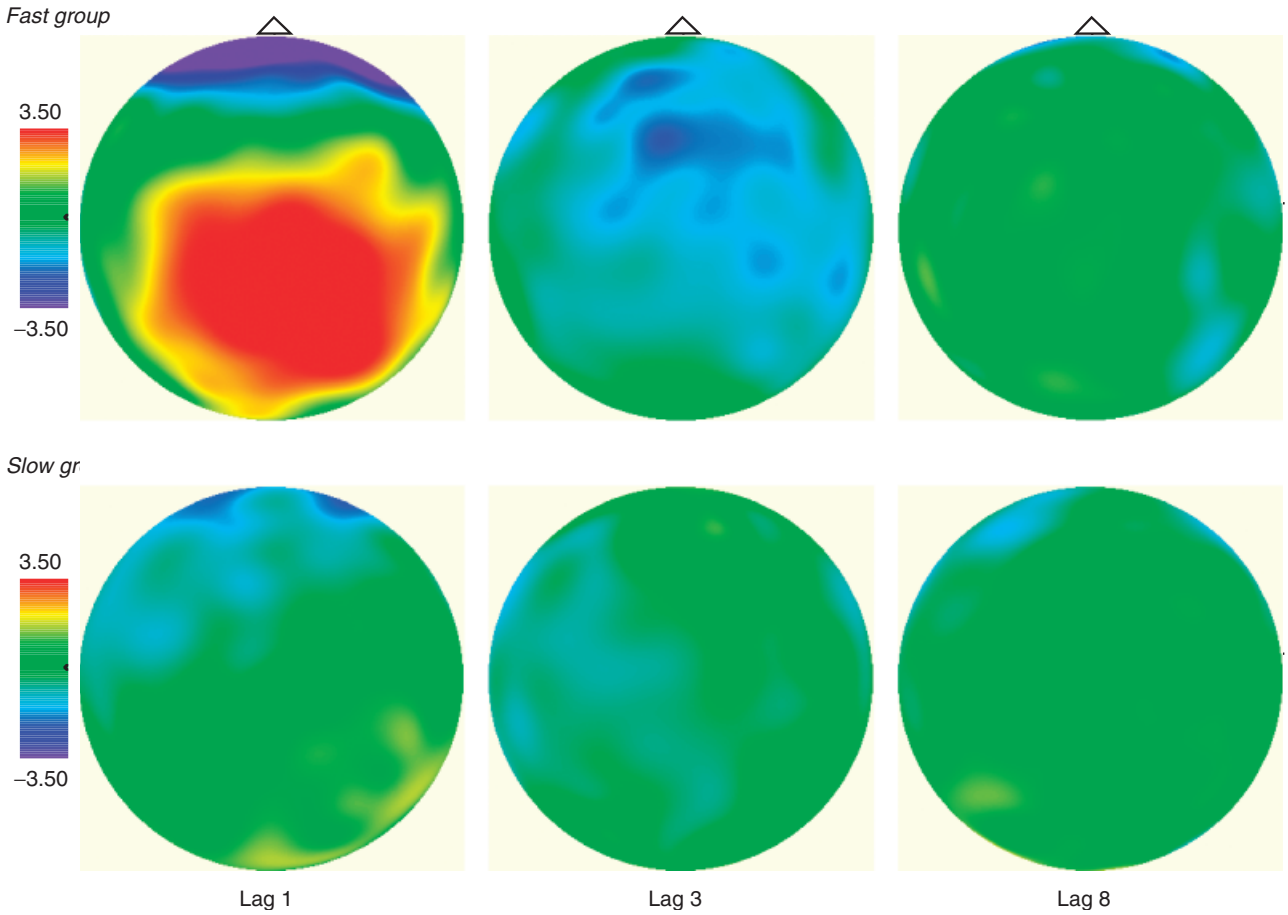

Figure 4. Topographical maps of differential activation in microvolt at 390 ms after T2. The top row shows activation in the fast expectation group for each lag, whereas the bottom row shows the same for the slow expectation group. The vertex and nose have been indicated as in Figure 4.

The positive modulation around 390 ms could be related to the P3 component, broadly fitting with the topographic distribution of activity. As mentioned, this component has also been shown to be sensitive to the AB and hence to the consolidation of the second target (Kranczioch et al. 2003; Sergent et al. 2005; Vogel et al. 1998). As with the N2-related modulation, the presumed absence of the P3 when targets are integrated together reflects their unitary processing mode. It seems therefore that the main effort that has to be undertaken when no sparing occurs is related to working memory processing, which points to the transfer of event episodes into a more durable representation. Interestingly, although P3-related modulations are compatible with the present results, no similar correspondence could be found for the N4 component. This fits well with existing theory, as this component is thought to reflect semantic processing that is relatively unaffected by the blink. This supports the idea that semantic processing of T2 is similar whether it has been integrated with T1 or not. A direct test of this idea would be to check for priming effects of spared and nonblinked targets (at later lags) on subsequent stimuli.

Both the N2 and P3 modulations reported in this study peaked slightly earlier when compared to the peak activations observed by Sergent et al. (2005). This might indicate that the timing of these components was somewhat different, possibly due to procedural differences in stimulus presentation. Determining whether this is true is not feasible in the present study because the overlapping ERPs elicited by the RSVP paradigm preclude meaningful analysis of actual activation. However, exploring this issue seems like a worthwhile course for future investigation, in which a more skeletal version of the task could be used (e.g., Duncan, Ward, & Shapiro, 1994). Although such a study would have to rely on replacement of the mask at Lag 1 by T2 (because there are no distractors to serve as masks when targets do not follow each other directly), the present work now provides a reference point based on traditional RSVP. In summary, the present study has provided evidence that the proposed mechanism of joint target integration at Lag 1 (Akyürek et al., 2006) has a neurological correlate evoked by the presentation of T2. The emerging picture is that the creation of a T2-only event elicits brain activity within a few hundred milliseconds that is not found for a unified event episode containing both targets. The analysis of order errors as an index of integration is thereby supported, as their occurrence cannot parsimoniously be accounted for by off-line memory effects or report bias. In the context of RSVP, it is possible to fine-tune attentional integration by using global task expectations to change the way in which information is processed on a scale of tens to hundreds of milliseconds. To some extent, this conclusion is in line with previous work by Martens and Johnson (2005) and Correa, Lupiáñez, and Tudela (2005), who cued the T1–T2 target onset interval and

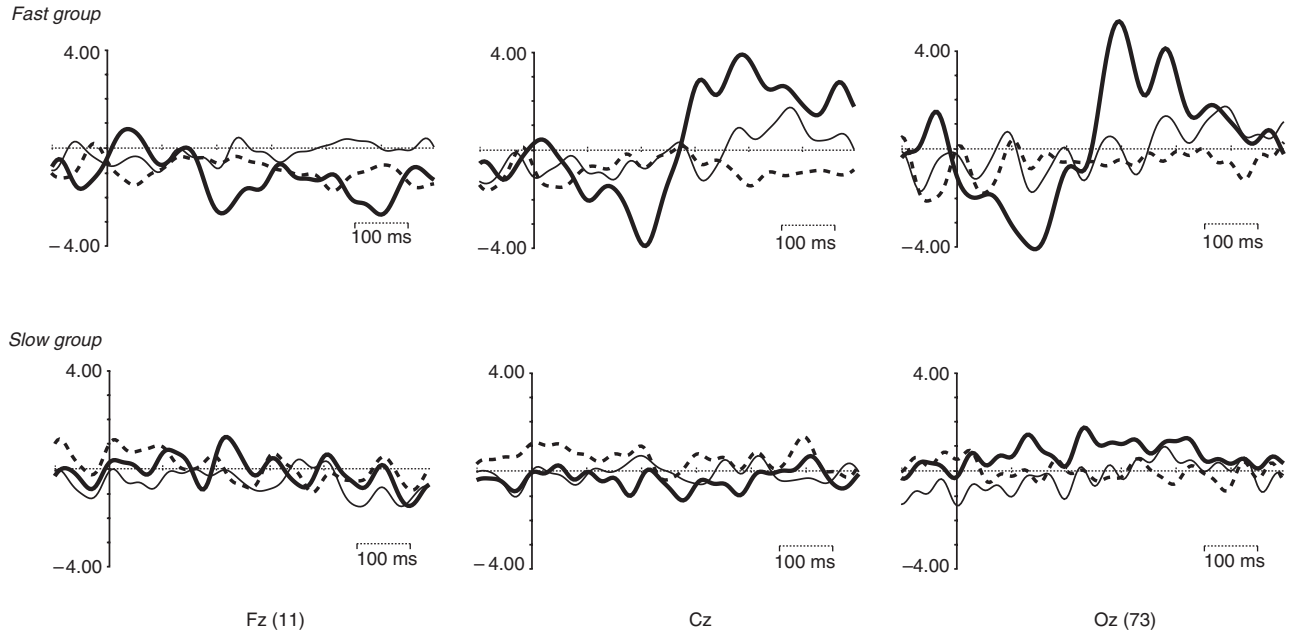

Figure 5. Difference waves (in microvolts) plotted from 100 ms before to 600 ms after T2 for individual electrodes at the center of each sensor cluster. Sensor approximations of the 10-10 system are provided as a guide. Thick lines represent Lag 1, dashed lines represent Lag 3, and thin lines represent Lag 8. The top row shows sensor 11, the vertex, and sensor 73 in the fast expectation group. The bottom row shows the same for the slow expectation group. Note that individual sensor waveforms may deviate from reported cluster averages.

found that the availability of this information reduced the blink. At the same time, the present results go beyond the timing of target onsets, showing that the duration of the integration window of (what is meant to code) a single stimulus can be modulated. Interestingly, Martens and Johnson found no effect of implicit temporal information when they presented lags blockwise, whereas our results were obtained with implicitly induced speed expectations. A possible account for this difference may be that, although implicit information may not be enough to strategically overcome "Stage 2" processing (cf. Chun & Potter, 1995), it can indeed affect earlier attentional selection.

This dissociation between early (Stage 1) and late (Stage 2) attentional processing can explain the joint occurrence of Lag 1 sparing and the attentional blink at the same time. Given the present results, it would seem that the way around the blink is to feed two items (in one chunk) to Stage 2 processing, which in turn suggests that Stage 2 processing is typically not limited by information content but by the number of events that are considered. This account is in line not only with the two-stage theory of the blink (Chun & Potter, 1995), but also with existing accounts of Lag-1 sparing (Shapiro et al., 1994; Visser et al., 1999) and offers a way to bring them together.

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APPENDIX

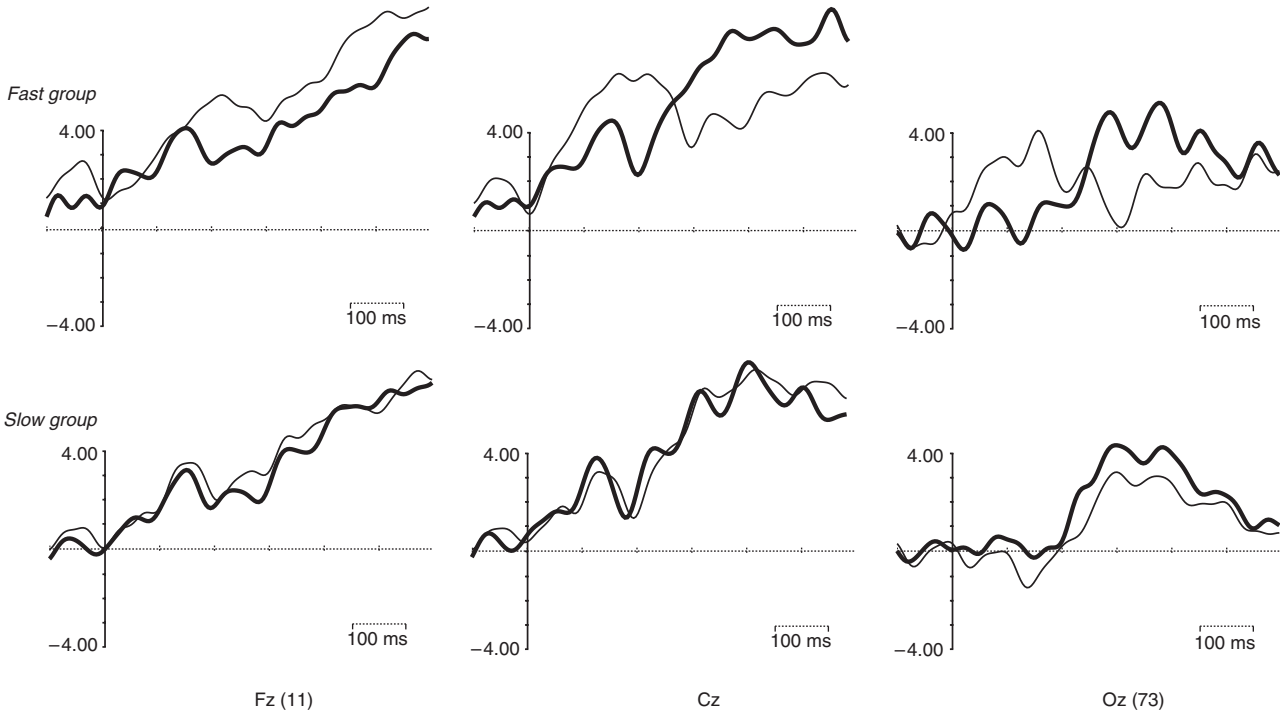

Figure A1. Raw waveforms underlying the difference waves at Lag 1 in both fast and slow expectation groups. Thick lines represent fast trials and thin lines represent slow trials.