Dissociable Mechanisms Supporting Awareness: The P300 and Gamma in a Linguistic Attentional Blink Task

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As demonstrated by the attentional blink (AB) phenomenon, awareness for attended stimuli is governed by sharp capacity limits. We used a linguistic AB task to investigate the neural mechanisms that underlie failures of awareness, examining both event-related potentials and oscillatory brain activity to correctly reported and missed second targets (T2s) presented after a correctly reported first target (T1) in a rapid visual stream of distractors. Correctly reported targets occurring at a short lag (250 ms) after T1—within the classic AB period—elicited enhanced late gamma activity relative to incorrectly reported targets but showed no P300 modulation relative to missed targets. In contrast, correctly reported targets presented at a long lag (830 ms)—outside the classic AB period—elicited a greater P300 component but did not significantly modulate oscillatory activity. This double dissociation suggests that there are multiple neural mechanisms supporting awareness that may operate in parallel. Either the P300 or the gamma can index impairment in the cascade of processing leading to a target's entry into awareness. We conclude that the P300 and gamma activity reflect functionally distinct neural mechanisms, each of which plays an independent role in awareness.

Keywords: attentional blink, awareness, gamma, oscillations, P300

Introduction

The human cognitive system is capable of fully processing only a fraction of incoming stimuli. The brain's ability to reduce the potentially overwhelming flood of incoming sensory information to a manageable stream is a crucial component of attention and key in effective processing. A number of striking experimental phenomena, such as change blindness, inattentional blindness, and the attentional blink (AB), are indicative of the limits of human awareness, demonstrating that even salient features in a visual display that elicit robust sensory responses can fail to reach awareness (e.g., Vogel et al. 1998; Simons and Chabris 1999; Fernandez-Duque et al. 2003). The neural mechanisms that contribute to attentional selection, allowing certain stimuli to enter awareness and generate a conscious percept, have come to represent an important topic of scientific investigation in recent years. Although these experimental phenomena represent only a small part of what is termed "consciousness," investigating these failures of awareness allow us to sketch out mechanisms that may underlie our richer conscious qualia.

Two physiological correlates of awareness, both observable in human electroencephalography (EEG), are the P300 component and gamma-band oscillations. The P300 is widely thought to reflect the updating of working memory (e.g., Donchin 1981; Donchin and Coles 1988; Polich and Criado 2006; Polich 2007). According to this view, each incoming stimulus undergoes initial sensory processing and is then evaluated by an attention-driven comparison process to determine whether its representation matches the previous event in working memory. If a new stimulus is detected, such as a target embedded in a stream of standards in an oddball task, additional attentional processes are invoked to update the neural representation of the stimulus environment, generating the P300 (Polich 2007). An alternative account regards the P300 as related to confidence and decision-making processes concerning how to classify and respond to an eliciting stimulus (Squires et al. 1973; Makeig et al. 2004; Verleger et al. 2005). Yet a third hypothesis proposes that the P300 reflects long-lasting global activation of workspace neurons, found throughout a distributed network of cortical association areas (Dehaene et al. 2003; Sergent et al. 2005). Thus, the precise mapping between cognitive processes and P300 modulations is still open for interpretation.

Although these theories differ in a number of important respects, a common assumption shared by all 3 accounts is that some aspect of stimulus awareness and the P300 are correlated. Under each model, the eliciting stimulus triggers a certain set of neural processes, indexed by the P300, that coincide with the stimulus entering awareness and becoming available for explicit report. The P300 is not unitary but consists of 2 subcomponents: the P3a, a central/anterior early (220–280 ms) positivity (Squires et al. 1975) thought to index automatic novelty detection stemming from changes in frontally based working memory representations (Polich 2007) and the later P3b (300–500 ms), a posterior positivity associated with voluntary attention, task-relevant processing, context updating, and subsequent memory storage (Soltani and Knight 2000; Polich 2007).

More recently, gamma-band oscillations have emerged in the neurobiological study of consciousness as another objective neural measure of awareness. The underlying theory is that precise synchronization of neuronal discharges in the gamma frequency range act as a temporal-binding mechanism, integrating distributed neurons into cell assemblies and linking diverse areas involved in different aspects of perception and behavior (e.g., Engel et al. 1999; Tallon-Baudry and Bertrand 1999). A key distinction is often drawn between evoked gamma, which is phase-locked to stimulus onset, and induced gamma, which is non-phase-locked to the stimulus. Evoked gamma tends to occur soon after stimulus onset, is generally focused in the lower gamma-band frequency ranges (30–40 Hz) (Tallon-Baudry and Bertrand 1999; Martinovic et al. 2008), and likely indexes early sensory processes rather than higher level representational processing (Tallon-Baudry and Bertrand 1999). In contrast, induced gamma tends to occur at later and more variable latencies with greater variability in frequency (30–90 Hz; Tallon-Baudry and Bertrand 1999; Martinovic et al. 2008). It is this latter type of gamma that has been linked to feature binding and object representation across a wide range of studies (Muller et al. 1996;

Tallon-Baudry and Bertrand 1999). Induced gamma has been proposed to underlie object representation and object awareness through both "bottom-up" binding as well as "top-down" synchronization processes involving the activation, retrieval, and maintenance of an internal representation (Tallon-Baudry and Bertrand 1999). It is crucial to consider the distribution and time course of gamma-band oscillations when attributing them to brain responses, as some initial reports were likely contaminated with broadband transient electrical artifacts in the gamma band generated from microsaccades (Yuval-Greenberg et al. 2008; Yuval-Greenberg and Deouell 2009). Nonetheless, even with broadband transient gamma excluded, there are still compelling findings relating gamma-synchronization to behavior (Fries et al. 2008; Bosman et al. 2009), although its precise function in awareness is not well understood.

The extent to which the P300 and gamma activity may reflect concomitant neural processes is uncertain and appears to depend upon the type of gamma under discussion. Like the P300, both evoked and induced gamma-band oscillations have been shown to be modulated by attention (Tiitinen et al. 1993; Sokolov et al. 1999; Fries et al. 2001; Muller and Keil 2004). For example, very early gamma-band responses likely to be evoked in primary sensory cortices are modified with attention to visual, auditory, and tactile modalities (Karns and Knight 2009). Studies using a range of target detection tasks have found that targets elicit a greater early gamma response than nontargets (Yordanova et al. 1997; Herrmann et al. 1999; Herrmann and Mecklinger 2000, 2001; Debener et al. 2003). Though at least part of this response is probably due to the change in physical stimulus such as refractoriness between targets and nontargets, it is modulated by task demands (Herrmann and Mecklinger 2001; Debener et al. 2003) and thus also likely reflects aspects of attentive target processing and top-down attention. Similar to the well-established enhancement effects observed in sensory evoked potentials as a function of selective attention (e.g., Woldorff et al. 1987; Hillyard et al. 1995), attention-driven increases in early gamma activity may reflect facilitation in processing at the perceptual stage, in a manner consistent with an early gain control over sensory information flow (Karns and Knight 2009).

In contrast, effects of target processing on later gamma activity have been less consistent, and the functional role of these oscillations is not yet clear. Two studies using auditory oddball tasks found that targets elicited less induced gamma activity than standard stimuli during the time interval of the P300 (Marshall et al. 1996; Fell et al. 1997). Fell et al. (1997) observed that during this time window, induced gamma activity recovered to baseline level for nontargets but remained reduced for target trials. Another study that used a rapid serial visual presentation (RSVP) oddball paradigm found a late induced gamma increase to targets beginning at about 500 ms, after the P300 had begun to descend to baseline levels (Kranczioch et al. 2006). No correlation was observed between the magnitude of the induced gamma response and the P300 amplitude, leading the authors to suggest that induced gamma reflects aspects of target processing beyond those associated with the P300. It has been proposed that the P300 may suppress gamma activity by causing a widespread inhibition of cortical networks responsible for gamma oscillations, leading to a subsequent increase in induced gamma activity coincident with P300 decay (Marshall et al. 1996; Fell et al. 1997, 2002). However, there is some evidence indicating that gamma and the P300 accompany one another temporally and show similar enhancements to targets versus nontargets (Basar-Eroglu and Basar 1991; Gurtubay et al. 2001; Watanabe et al. 2002). Still others find no evidence that target processing modulates late gamma activity in either a positive or a negative direction (Sannita et al. 2001; Debener et al. 2003). In sum, the functional significance of the later induced gamma response and its relationship to the P300 remains to be clarified.

One phenomenon that is especially well suited to investigating the neural mechanisms underlying human awareness is the AB. The AB refers to a deficit in reporting a second target (T2) when it occurs 200–500 ms after the first target (T1) in an RSVP stream (Broadbent DE and Broadbent MH 1987; Raymond et al. 1992). Early evidence that the AB is due to attentional rather than sensory limitations was provided by the finding that the blink does not occur when T1 can be ignored, despite the fact that sensory information remains the same (Raymond et al. 1992). A subsequent series of event-related potential (ERP) experiments provided converging evidence for this conclusion, demonstrating that the P1 and N1 sensory ERP components elicited by T2 show no reduction during the AB period (250 ms following T1), while the P300 component is completely absent (Vogel et al. 1998). Because some stimuli presented during the AB fail to reach awareness as a result of central limitations rather than visual degradation, the paradigm represents a useful experimental manipulation through which to investigate the neural mechanisms that contribute to awareness.

Although it is generally accepted that the AB reflects a postperceptual limitation, the precise stage of processing at which this impairment occurs is not yet agreed upon (Shapiro et al. 1994; Chun and Potter 1995: Giesbrecht and Di Lollo 1998; Vogel et al. 1998; Jolicoeur and Dell'Acqua 1998). Two leading accounts, Chun and Potter's (1995) two-stage model and Shapiro's Interference model (1994, 1997), both propose a first stage of processing in which perceptual features of incoming stimuli that are relevant for target detection are initially processed. However, while the two-stage model assumes that the AB occurs as a result of T2 failing to reach working memory, the interference model proposes that missed T2s enter working memory but are lost later due to interference with T1 and other distracters. To distinguish between these 2 theoretical models, a number of AB studies have focused on the P300 as a useful marker of an incoming stimulus' entry into working memory (Vogel et al. 1998; Vogel and Luck 2002; Dell'Acqua et al. 2003; Kranczioch et al. 2003; Martens et al. 2006; Sessa et al. 2007; Koivisto and Revonsuo 2008; P300: Donchin 1981; Donchin and Coles 1988; Polich and Criado 2006). These studies have typically used a target detection task, in which a target stimulus (such as the letter E) is presented on some proportion of trials. The typical finding of studies using this procedure is that the P300 to T2 is absent or reduced during the AB period (Vogel et al. 1998; Vogel and Luck 2002; Dell'Acqua et al. 2003). Several ERP studies using similar target detection procedures have directly examined the role of awareness during the AB, comparing correctly identified T2 trials from missed trials during the AB period (Rolke et al. 2001; Kranczioch et al. 2003; Martens et al. 2006; Sessa et al. 2007; Koivisto and Revonsuo 2008; Pesciarelli et al. 2007). These studies have generally found that detected T2 items elicit a reliable P300 component, while the P300 to undetected T2s is reduced or absent. Such results have been interpreted as indicating that T2 does not gain access to working memory when blinked, supporting the two-stage AB model.

To date, few studies have focused on oscillatory brain activity underlying target awareness during the AB. However, one recent study found greater gamma activity between 660 and 760 ms poststimulus to targets presented during the AB that were detected relative to those that were undetected (Kranczioch et al. 2007). This finding suggests that gamma enhancement plays a role in awareness during the AB and may be one mechanism by which targets can survive the blink period, or alternatively, that a reduction in gamma prevents a target from entering awareness. In sum, both the P300 and the gamma activity potentially represent important neural processes that contribute to a target's entry into awareness during the AB, although the relationship between them is not well understood.

In the present study, we used a linguistic AB task, analyzing both ERPs and oscillatory brain activity, to address several questions related to the neural mechanisms of human awareness. With this task, we previously demonstrated that incorrectly reported whole-word targets embedded in a linguistic context fail to elicit an N400 effect both during and after the AB period (Batterink et al. 2010). This finding provides evidence that misses occurring at both short and long lags are the result of an impairment in online processing or awareness, and not caused by a later loss of information, such as simply forgetting the target's identity several seconds after stimulus presentation or during the response period. Given these results, this linguistic AB task represents a particularly effective paradigm to examine the neural mechanisms responsible for failures of awareness. In addition, the linguistic nature of this task requires the report of a target's content rather than simply its presence or absence. Thus, this paradigm is well suited to investigating the neural mechanisms that underlie the formation of a coherent conscious percept, such as the gestalt of a familiar word.

Taking advantage of the features of this task, one goal of the present study was to investigate whether and how the P300 and gamma might be related at the physiological level. As reviewed above, past research has yielded inconsistent results regarding the relationship between the P300 and the late induced gamma activity but has generated several intriguing and testable hypotheses. For example, the P300 may actively suppress gamma; the P300 and gamma may reflect connected or concurrent neural processes, correlating across time and experimental manipulations; or the P300 and gamma may reflect largely distinct neural processes without a relationship between them. In the present experiment, we test whether gamma and the P300 show interrelated or dissociable modulations as a function of awareness and lag in order to elucidate whether they reflect overlapping or distinct neural processes.

The present study also aimed to acquire greater insight into the functional significance of the P300 and gamma. As previously described, both of these electrophysiological measures have been proposed to index different cognitive processes (such as working memory updating and binding) and are correlated with awareness. However, how these putative cognitive functions might interact during conscious processing is not known. For example, are the cognitive processes indexed by the P300 and gamma both necessary for awareness or is either one alone sufficient to generate a conscious percept?

Finally, the current study also sought to address the fundamental cognitive limitations driving the AB phenomenon by examining whether failures of awareness occurring within versus outside the classic boundaries of the AB are due to common or distinct neural mechanisms. An underlying assumption made by most models is that the AB reflects a bottleneck specific to the ongoing effects of T1 processing and that this limitation ceases to impact T2 processing at later lags (Shapiro et al. 1994, 1997; Chun and Potter 1995; Giesbrecht and Di Lollo 1998; Vogel et al. 1998; Nieuwenstein et al. 2005). Under this hypothesis, incorrectly reported targets would be expected to elicit dissociable electrophysiological responses as a function of lag. Alternatively, if a similar profile is observed for misses occurring within versus outside the classic AB period, this would point to a more graded boundary between short and long lags than is generally discussed. In support of this possibility, previous research has demonstrated that the AB window is highly variable between individuals (Green and Bavelier 2003; Martens et al. 2006) and is graded as a function of lag rather than sharply defined (e.g., Raymond et al. 1992; Shapiro et al. 1994; Giesbrecht and Di Lollo 1998). These findings provide evidence that the magnitude of the AB effect may vary from trial to trial and may sometime endure well beyond what is traditionally considered the AB window, with at least some proportion of misses at longer lags resulting from the participant experiencing a prolonged AB window. Thus the comparison of P300 and gamma responses at short and long lags may be helpful in clarifying whether distinct processes underlie failures of awareness occurring within versus outside the AB period.

Materials and Methods

Participants
Twenty-one monolingual native English speakers (14 women) were recruited at the University of Oregon to participate in the experiment. Participants were between 18 and 30 years old (M = 23.3, standard deviation = 3.49), were right-handed, had no history of neurological problems, and had normal or corrected-to-normal vision. Participants gave informed consent and received $10/h for their participation. All methods were approved by the Institutional Review Board at the University of Oregon and are consistent with ethical standards of the Declaration of Helsinki.

Stimuli
Each trial began with the presentation of a prime word for 1000 ms, followed by a blank interval for 1000 ms. An RSVP stream was then presented, consisting of 7-character strings of letters that were presented at a rate of 12 items per second (83 ms duration, 0 ms interstimulus interval). The first target (T1), which consisted of a randomly selected number (between 2 and 9) written out in letters and flanked by Xs to create a 7-character string, occurred randomly between positions 5 through 8. The second target (T2) was a word 3 to 7 characters long, flanked by pound signs (#) if the word contained fewer than 7 characters to create a 7-character string. T2 occurred either 3 positions (250 ms) or 10 positions (833 ms) following T1 (i.e., Lag 3 or Lag 10). Distractors were composed of 7-character strings consisting of randomly selected consonants. All distractor items were presented in blue, and both T1 and T2 were presented in red. This paradigm was similar to the one used by Vogel et al. (1998) and is illustrated in Figure 1.

Based on a simple computer algorithm designed to maximize the AB effect, the blue distractor color was adjusted at regular intervals throughout the experiment depending upon participant performance. Within each lag condition, percent accuracy was calculated every 8 trials. If participants correctly reported 6 or more second targets in Lag 3, the blue distractor color was adjusted to become darker, increasing the overall difficulty. If participants incorrectly reported 6 or fewer second targets in Lag 10, the blue distractor color became lighter, making the task easier (beginning RGB value = 0, 100, 255; mean final RGB value in semantic block = 0, 0, 255; mean final RGB value in
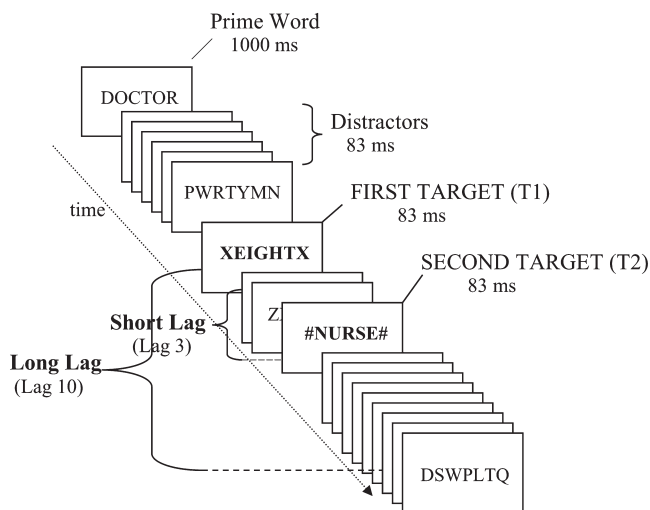
Figure 1. An example trial. A prime word appeared for 1000 ms, followed by a blank interval for 1000 ms. An RSVP stream (83 ms per frame) was then presented. The first target (T1), consisting of a randomly selected number (between 2 and 9) written out in letters and flanked by Xs to create a 7-character string, occurred randomly between positions 5 through 8. The second target (T2) was a word occurring either 3 positions (250 ms) or ten positions (833 ms) following T1 (i.e., Lag 3 or Lag 10). Distractors were composed of 7-character strings consonants. Distractor items were blue, and T1 and T2 were red. A 1000 ms blank interval followed the RSVP stream, which was then followed by the response period.

syntactic block = 0, 75, 255). A 1000 ms blank interval followed the RSVP stream, which was then followed by the response period.

Participants performed 2 blocks in counterbalanced order. In the semantic block, T2 was semantically related to the prime word (e.g., dog-puppy) on half the trials and semantically unrelated to the prime word on the other half of trials (e.g., lemon-puppy). In the syntactic block, the word category of T2 was correctly predicted by the prime word on half the trials (e.g., the-sky) and incorrectly predicted by the prime on the other half of the trials (e.g., we-sky). For each participant, word pairs appeared in random order, assigned randomly to either the Lag 3 or the Lag 10 condition, and were counterbalanced so that each target appeared once in both the related and the unrelated (or syntactically congruent and incongruent) conditions. There were a total of 240 trials in the semantic block and 320 trials in the syntactic block. A more detailed description of the linguistic characteristics of the stimuli can be found in an earlier publication of these data (Batterink et al. 2010).

The stimuli were presented against a gray background on a computer monitor placed approximately 140 cm from the participant. The visual angle of words subtended 3.5° horizontally and 0.5° vertically.

Procedure

Participants were fitted with an elastic EEG cap embedded with electrodes and then seated in a comfortable chair in a dimly lit and acoustically and electrically shielded booth. They were instructed to identify the 2 red targets in the RSVP stream and to make 2 alternative forced-choice responses using a game controller at the end of each trial. In the semantic block, these responses indicated whether the number (T1) was odd or even and whether the word (T2) was semantically related or unrelated to the prime word that appeared at the beginning of the trial. In the syntactic block, participants were again asked to decide whether the number was odd or even and whether or not the word made a syntactically congruent phrase with the preceding prime word. After participants entered their responses, the next trial began automatically after a brief interval. Participants were given as much time as needed to respond but generally responded within 1–2 s after the cue appeared. Before each block, participants were given approximately 10–20 practice trials. Once the experiment was underway, participants were given brief breaks every 60 trials. All participants completed both the semantic and the syntactic blocks, which appeared in counterbalanced order across participants.

EEG Recording and Analysis

EEG activity was recorded from 29 tin electrodes mounted in an elastic cap (Electro-Cap International). The electrooculogram was recorded from electrodes placed at the outer canthi of both eyes and below the right eye. Scalp electrodes were referenced to the right mastoid during recording and for off-line averaging. The EEG was amplified (SA instruments) with a band-pass of 0.01–100 Hz and digitized at a sampling rate of 250 Hz. Recordings were made in an electrically shielded booth.

ERP and spectral analyses were carried out using EEGLAB (Delorme and Makeig 2004) and in-house MATLAB programs. Data were band-pass filtered from 0.1 to 80 Hz, and epochs time locked to the onset of the T2 words were extracted from −750 to 1000 ms. Trials containing large or paroxysmal artifacts, movement or muscle artifacts, or amplifier saturation were identified by visual inspection and removed from further analysis. Data were then submitted to the extended *runica* routine of EEGLAB software. Ocular artifacts were identified from scalp topographies and the component time series and removed. ICA-cleaned data were subjected to a final manual artifact correction step to detect any residual or atypical ocular artifacts not removed completely with ICA. For 5 subjects, ICA did not converge on clean ocular artifact components due to low numbers of vertical or horizontal eye movements or blinks. For these data, ocular artifacts were detected and removed by inspecting eye channels for deflections and polarity inversions with scalp channels. Data from all linguistic conditions (Semantically Related, Semantically Unrelated, Syntactically Congruent, and Syntactically Incongruent) were combined in order to increase the number of trials in the 4 experimental conditions of interest (Lag 10 Correct, Lag 10 Incorrect, Lag 3 Correct, and Lag 3 Incorrect). Following correction for EEG artifacts, the Lag 10 Correct condition contained an average number of 167 trials per subject (max = 226, min = 110), Lag 10 Incorrect contained an average of 33 trials per subject (max = 50, min = 15), Lag 3 Correct contained an average of 159 trials per subject (max = 209, min = 105), and Lag 3 Incorrect contained an average of 56 trials per subject (max = 85, min = 30). Both behavioral and ERP analyses included only those trials on which T1 was correctly reported.

For ERP analyses, epochs were averaged to the onset of the T2 word and baseline corrected to a 100 ms prestimulus interval. Time windows for visual evoked potentials were selected based on grand-average peak amplitudes for each potential. The P1 component was measured between 150 and 170 ms poststimulus, the N1 component between 200 and 220 ms poststimulus, and the P2 between 250 and 270 ms poststimulus.

Channels for all visual evoked potential analyses were selected a priori according to where the peaks were expected to be maximal and included only the 2 most posterior rows of electrodes. For each time window, data were analyzed using a repeated measures analysis of variance (ANOVA) with Lag (Lag 10, Lag 3), Correctness (correct, incorrect), Hemisphere (left, right), and electrode as factors. Time windows for P300 analyses were selected based on visual inspection of the waveforms and their topographical distributions. To capture an earlier, P3a-like effect, a time window between 200 and 500 ms was selected, while a 500–1000 ms time window was selected to capture a later, P3b-like effect. Channels for P300 analyses were selected a priori and included all scalp sites since the P300 is a widely distributed component. For each P300 analysis, data were analyzed using a repeated measures ANOVA with Lag, Correctness, Hemisphere, Anterior/Posterior (frontal, frontotemporal, temporal, central, parietal, occipital), and lateral/medial (lateral, medial) as factors.

Spectral analyses were computed on the entire 1750 ms epochs, then baseline corrected to a 200 ms prestimulus interval. Time-frequency decompositions were computed in 2 Hz steps from 2 to 126 Hz using the *newtimef* function of EEGLAB. Single trials were analyzed using Hanning-tapered sinusoidal wavelets with 1 cycle at the lowest frequency (2 Hz) and increasing by a scaling factor of 0.5, reaching 31.5 cycles at the highest frequency (126 Hz), allowing better frequency resolution at higher frequencies than wavelet approaches using constant cycle length (Delorme and Makeig 2004). The mean event-related spectral perturbation (ERSP), across all scalp channels and subjects, was first calculated separately within each of the 4 conditions (Lag 10 Correct, Lag 10 Incorrect, Lag 3 Correct, and Lag 3 Incorrect). The incorrect ERSP was then subtracted from the correct ERSP at each lag, yielding a time-frequency plot of the spectral awareness effect at each lag.

As an initial characterization of the robustness of spectral perturbations, the ERSP power (in dB) from –200 to 720 ms and from 2 to 80 Hz was averaged across scalp channels for each lag and awareness condition, plotted relative to the prestimulus baseline, and quantified using uncorrected P values. These results are described qualitatively in the results section.

To more rigorously test the significance of the awareness effects at each lag, which represented our main comparison of interest, we performed a cluster analysis as recommended by Maris and Oostenveld (2007). First, the "true awareness effect statistic" was calculated based on the correct and incorrect trials in each lag condition. Within the a priori defined frequency range where we expected gamma (35-80 Hz), we first calculated the T-statistic across the average of all scalp channels for the 21 participants. The T-statistics were thresholded at $P < 0.10$ two-tailed, and the sum of the T-statistics (T-sum) was calculated for each cluster. A P value of 0.10 was used to allow for long-lasting or broad spectrum but potentially weak effects (Maris and Oostenveld 2007). Clusters in the resulting image were determined using the *bwconncomp* function in MATLAB to detect the 8-connected clusters. The sum of the T-statistics (T-sum) was calculated for each cluster. To assess the significance of the T-sum for each cluster, we performed a permutation test. For each subject, a "surrogate awareness effect" was calculated by randomly ordering the trials and selecting surrogate correct and incorrect trials. The number of correct and incorrect trials selected for each surrogate was based on the actual number of correct and incorrect trials for each participant for each lag. This random partitioning of trials was performed 100 times per subject, and the same clustering procedure ($P < 0.10$, two-tailed) was applied to each surrogate. The maximum of the absolute value of the T-sum cluster statistic was used to calculate a surrogate distribution of T-sum values. A critical T-sum was defined as the value in the surrogate distribution corresponding to the 95th percentile ($P \leq 0.05$). We performed a separate cluster analysis for the lower frequencies from 2 to 35 to prevent large clusters in low frequencies from influencing the surrogate of the gamma signal.

In a similar analysis to the ERSP analysis, we calculated the intertrial phase coherence (ITC) for each subject, channel, and condition (Correct and Incorrect Lag 3 and Lag 10) across time and frequency using the same wavelet parameters as in the ERSP analysis. We then performed the statistical analysis across subjects on the average ITC across channels. As with the ERSP, significance was assessed by comparing the "true correctness effect statistic" to the distribution of surrogates, calculated by randomly partitioning the trials for each condition to correct or incorrect, 100 times per subject, based on the true number of correct and incorrect trials for each participant for each lag.

Results

Behavioral Results
Mean T2 discrimination is plotted as a function of T2 lag in Figure 2. A substantial decrease in accuracy for the short-lag condition compared with the long-lag condition was found ($F_{1,20} = 72.2$, $P < 0.0001$), indicative of a significant AB effect. Mean accuracy for T1 discrimination was 87.2% (standard error = 1.6%).

ERP Results
For both short and long lags, P1 (150-170 ms), N1 (200-220 ms), and P2 (250-270 ms) amplitudes were not significantly affected by awareness ($F_{1,20} < 2.0$, $P > 0.1$ in all cases). ERP effects related to the semantic and syntactic processing of target words occurring during and after the AB, including the N400, are described in a previous report of these data (Batterink et al. 2010).

During the P3a time window (200-500 ms), a significant effect of awareness was found on the P300 amplitude at the long lag but not at the short lag (Lag × Awareness: $F_{1,20} = 6.29$, $P = 0.021$; Long Lag Awareness Effect: $F_{1,20} = 6.76$, $P = 0.017$; Short Lag Awareness Effect: $F_{1,20} = 0.44$, $P = 0.52$). At the long lag, the P3a awareness effect had a right medial distribution (Awareness × Hemisphere:
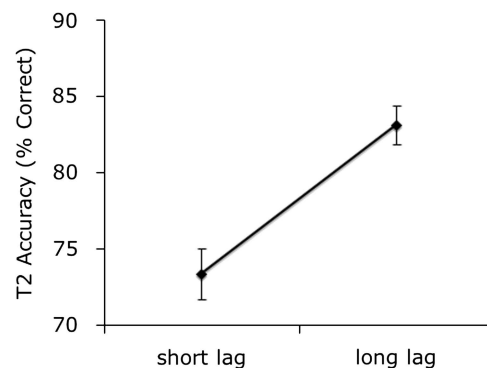

Figure 2. Mean discrimination accuracy for the second target (T2) as a function of lag. For the long lag, T2 appeared 10 frames (833 ms) after T1. For the short lag, T2 appeared 3 frames (250 ms) from T1. Participants reported whether T1 was odd or even, and whether T2 was semantically related or syntactically congruent with the prime (for details, see Batterink et al. 2010). Responses occurred after the end of the trial. Only responses following a correctly reported T1 are included. The decreased accuracy for the short lag demonstrates a significant AB effect. Error bars represent standard error.

$F_{1,20} = 6.95$, $P = 0.016$; Awareness × Lateral/Medial $F_{1,20} = 29.8$, $P < 0.001$). Similarly, during the P3b time window (500-1000 ms), awareness significantly modulated P300 amplitude at the long lag but not at the short lag (Lag × Awareness: $F_{1,20} = 9.02$, $P = 0.007$; Long Lag: $F_{1,20} = 14.9$, $P = 0.001$; Short Lag: $F_{1,20} = 0.11$, $P = 0.74$). At the long lag, the P3b awareness effect was maximal at medial sites (Awareness × Lateral/Medial: $F_{1,20} = 30.7$, $P < 0.001$). Supplementary analyses, in which the number of correct and incorrect trials were equated within each lag, confirmed that these ERP effects were not attributable to the unequal variance between conditions (Supplementary Fig. 1).

In sum, from 200 to 1000 ms, the P300 was significantly modulated by awareness at the long lag but not at the short lag. Although overlapping components associated with T1 processing make it difficult to fully isolate the T2-evoked P300 component, scalp topographies in the 400–600 ms time window (Fig. 3A) appear consistent with the posterior distribution of the P3b for both correct and incorrectly reported T2s at the short lag. At the long lag, the P300 at this latency is reduced for incorrectly reported T2s. ERP topographies for the difference between correct and incorrectly reported T2s are plotted in Figure 3B from 150 to 1000 ms and are consistent with an increased P300 (both P3a and P3b) for correctly reported T2s at the long but not short lag.

Event-Related Spectral Perturbations

Descriptive
Figure 4A shows the spectral profile of the ERSPs from 2 to 80 Hz for correct and incorrect targets at the short and long lag; note that in this paradigm, the rapid presentation of visual stimuli in this paradigm (12 items/s) occurring in the pre- and post-stimulus period will influence the amplitude of the ERSP. As shown in the figure, the amplitudes of spectral changes relative to the prestimulus period for the short lag appear smaller than that of long lag, likely because of the ERSP to T1 still present in the baseline of the short lag. The main comparison of interest is across correct and incorrect trials within the same lag condition, which have the same prestimulus period (Fig. 4B).

Figure 4A illustrates that both correctly reported and missed targets occurring at the short lag elicited a significant increase
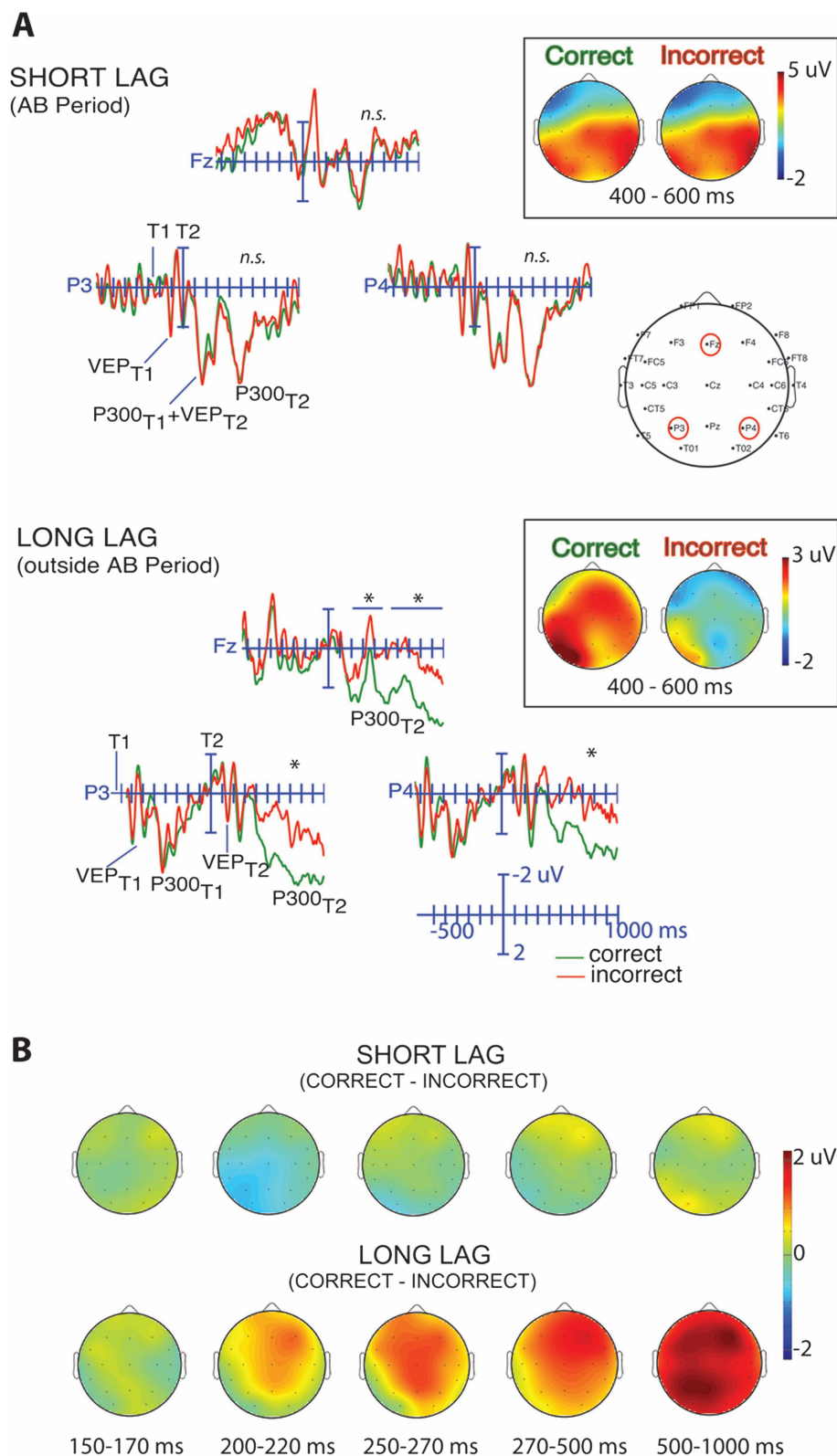
Figure 3. (*A*). Grand-average ERP waveforms at electrodes Fz, P3, and P4 to second targets (T2s) presented at the short lag (upper panel) and at the long lag (bottom panel), divided as a function of whether they were correctly (green lines) or incorrectly (red lines) reported at the end of the trial. Only trials where T1 was correctly reported are included. The onset of T1 and T2 and the ERPs they elicit, namely visual evoked potentials (VEP, comprising the P1, N1, and P2 components) and P300, are labeled for both the short and the long lag. The P300 was not modulated by awareness at the short lag (n.s.—not significant) but showed a robust effect of awareness at the long lag (*—significant modulation), with correctly reported targets eliciting a greater P300 than missed targets. Insets (right panels) show the topographical distribution of the ERP from 400 to 600 ms, to correctly reported and missed targets at both the short (upper) and the long lag (lower). ERPs were low-pass filtered at 40 Hz for presentation purposes. (*B*). Topographical voltage maps of difference between correct and incorrect ERPs across latencies corresponding to VEP and P300 effects, at each lag. This awareness effect was computed by subtracting the incorrect ERP from the correct ERP. At the short lag, no ERP effects of awareness were found at any latency. At the long lag, the P300 was significantly enhanced beginning at 200 ms.
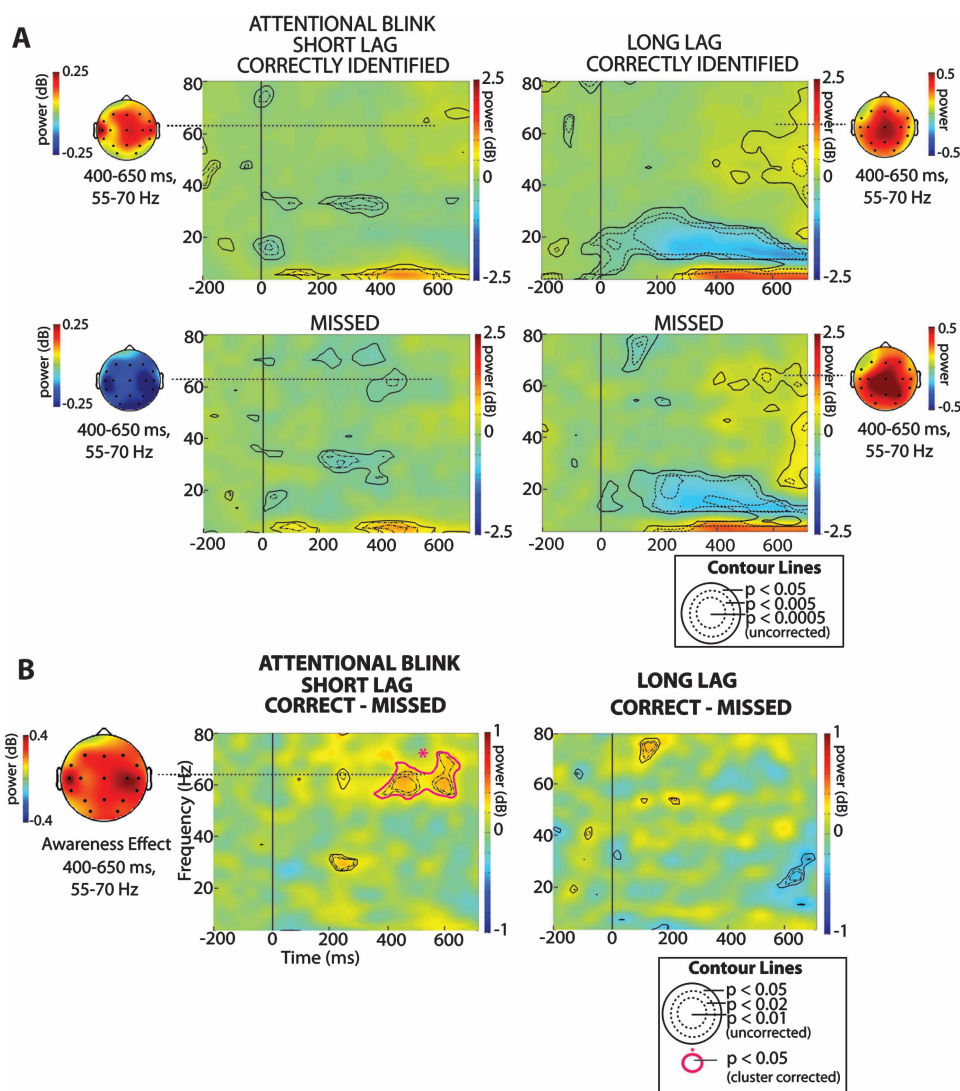
Figure 4. ERSPs measured in dB relative to the prestimulus period [$10\log_{10}(power_1/power_0)$] show oscillatory responses across all scalp electrodes to correctly identified and missed targets at short lags (left panels) and long lags (right panels). Only trials with correctly reported T1s are included. (*A*) The upper panels show ERSPs following correctly reported second targets (T2s) and the lower panels show ERSPs following incorrectly reported T2s. For a quantitative assessment of overall variability, contour lines indicate the *P* values of the uncorrected two-tailed *t*-tests comparing power at each time and frequency bin to prestimulus baseline levels (lines step from $P = 0.05$ to $P = 0.005$ to $P = 0.0005$). The scalp topography of the late gamma response, averaged from 55 to 70 Hz and from 400 to 650 ms, is illustrated for each individual condition. (*B*) The comparisons of interest were the "awareness effects," computed by subtracting the missed target ERSP from the correct target ERSP, and are displayed in the bottom panels. These 2 comparisons were subjected to permutation-based cluster thresholding. The late gamma cluster in the short lag awareness ERSP, ranging from approximately 400–650 ms, was the only effect of awareness to survive cluster thresholding and is outlined in magenta with an asterisk. This cluster is indicative of a significant gamma enhancement to correct trials relative to incorrect trials; the scalp topography of this effect, averaged from 55 to 70 Hz and from 400 to 650 ms, is pictured at left. Contour lines represent uncorrected two-tailed *P* values, stepping from 0.05 to 0.02 to 0.01. Colors indicate power increase (red) or decrease (blue) relative to the 200 ms prestimulus baseline.

in theta amplitude (4–7 Hz) relative to baseline levels extending from approximately 100 ms to the end of the epoch. Correctly reported and missed targets occurring at the long lag showed a significant theta increase relative to baseline beginning at approximately 300 ms as well as significant beta suppression (13–30 Hz) beginning at or near stimulus onset. Correctly and incorrectly reported long-lag targets also showed a significant late gamma increase of 0.5 dB beginning at around 400 ms poststimulus. This value represents a 12% increase in power relative to prestimulus levels (Fig. 4*A*). At the short lag, the gamma increase for correct targets relative to baseline was 0.25 dB, representing 6% increase relative to prestimulus levels, with a similarly sized decrease in gamma power for incorrect targets. Subtracting the ERSP to incorrect trials from correct trials results in an overall difference in gamma power of 10%

(Fig. 4*B*). The magnitude of this modulation (0.4 dB) is comparable to previous reports (Kranczioch et al. 2007).

Statistical Cluster Analysis of Correct–Incorrect ERSPs

The main comparisons of interest were across correct and incorrectly reported targets within each lag, short and long. We performed cluster thresholding on the difference between correct and incorrect targets separately for each lag. Cluster thresholding was based on the sum of the *T*-statistics within a cluster (*T*-sum) (Maris and Oostenveld 2007). One time–frequency cluster survived permutation-based cluster thresholding for Correct–Incorrect ERSPs. This cluster was found in the gamma band (defined as 35–80 Hz) in the short-lag condition with a *T*-sum greater than the critical *T*-sum value determined from the surrogate distribution ($P < 0.05$). This effect was centered at around 60 Hz and ranged

from approximately 400–650 ms, with targets that were correctly reported showing greater gamma activity relative to targets that were incorrectly reported (Fig. 4B). Note that the gamma cluster was centered at 60 Hz but is not likely to be attributed to line noise electrical artifacts; the surrogate cluster analysis we used took account of different numbers of trials in correct and incorrect targets, and line noise would not be differently distributed in true versus surrogate correct and incorrect trial groups. No other clusters in the Correct-Incorrect ERSPs approached significance, in either the gamma-band range (35–80 Hz) or the lower frequency range (2–35 Hz), at either lag.

Gamma Scalp Topography

We also plotted the scalp topography of gamma-band amplitude increases relative to the prestimulus baseline for all 4 trial types (Fig. 4A). It is notable that the scalp topography of the gamma for correct T2s at the short lag (although weaker in amplitude) is consistent with the topography of gamma for both correct and incorrect T2s at the long lag, while gamma for incorrect T2s at the short lag is weakly reduced relative to the baseline period. The topography of the gamma amplitude is centrally distributed and prolonged in time. This pattern of results is not consistent with either a line noise artifact or a transient broadband microsaccade artifact.

Intertrial Phase Coherence

There was no significant modulation of ITC between correct and incorrect trials at either the long or the short lag. All clusters had a T-sum value less than the critical T-sum determined by the distribution of surrogates ($P < 0.05$, two-tailed). The absence of phase coherence in the gamma band across trials is consistent with the profile of "induced" gamma rather than gamma evoked from a sensory stimulus and tightly phase locked across trials.

Discussion

In summary, ERP results revealed that at the long lag—classically considered outside the time period where an AB occurs—the P300 was significantly reduced when the target could not be reported. In contrast, at the short lag, within the AB period, no effect of awareness was found on the amplitude of the P300. Earlier visual evoked potentials were not modulated by awareness at either lag. The main finding revealed by time-frequency decompositions was a late gamma enhancement to correctly reported targets presented during the AB relative to incorrectly reported targets. Oscillatory activity to targets occurring at the long lag was not significantly modulated as a function of awareness. Thus, we found a double dissociation between the gamma and the P300: gamma modulation without P300 modulation during the AB period and P300 modulation without gamma modulation outside the AB period.

Consistent with theoretical models of the AB (Shapiro et al. 1994; Chun and Potter 1995; Giesbrecht and Di Lollo 1998; Jolicoeur and Dell'Acqua 1998) as well as previous empirical findings (e.g., Vogel et al. 1998; Sergent et al. 2005), both ERP and spectral results in the present study support the idea that the AB operates at a relatively late (>200 ms), postperceptual locus of selection. Early visual evoked potentials, namely the P1, N1, and P2, were not affected by awareness, either within or outside the AB period. Similarly, effects of awareness in the spectral domain were not observed before 400 ms. Taken together, these findings indicate that intact early visual processing is not sufficient to

create a conscious percept. Rather, conscious awareness is related to a later cascade of activation that spreads through a distributed network over widespread regions of cortex (Sergent et al. 2005; Gaillard et al. 2009; Dehaene and Changeux 2011).

The P300 and Awareness

At the short lag, an equal P300 component was elicited for both correctly and incorrectly reported targets; ERP awareness effects were not observed during the P300 time window or at any other latency at this lag. Although reduction of the P300 to incorrectly reported targets during the AB period is a common finding (Rolke et al. 2001; Kranczioch et al. 2003; Martens et al. 2006; Pesciarelli et al. 2007; Sessa et al. 2007; Koivisto and Revonsuo 2008), the present results indicate that a reduction in the P300 does not always accompany failures of awareness during the AB.

The lack of a P300 awareness effect at the short lag in our study cannot be readily accounted for by insufficient power, as a highly significant effect was found at the long lag despite the fact that there were fewer trials in the incorrect condition at this lag. Instead, properties of the current task that distinguish it from other AB studies may have contributed to the equal P300 observed to correct and incorrect targets during the AB window. First, our T2, a red word flanked by pound (#) symbols, and distractor items, arrays of blue consonants, may be more perceptually distinct from one another than those used in previous studies, which are typically letters and digits of the same color; the use of a perceptually distinct target may have allowed some perceptual features of both correctly and incorrectly reported targets to initially enter working memory and elicit a P300 (Donchin 1981; Donchin and Coles 1988; Polich and Criado 2006). It is known from visual search studies that targets "pop out" when they differ from distractors by an easily discriminable feature such as color (Treisman and Gelade 1980; Wolfe and Horowitz 2004) and that these color pop-outs elicit a P300 (Luck and Hillyard 1994). Furthermore, our behavioral awareness measure was sensitive to "identification" rather than detection. In contrast to previous AB studies' use of simple letter detection tasks, in which participants make a present/absent judgment on a single, low-level, and unchanging target (such as the letter "E"), in the present study, the identity of the target was not given beforehand. Thus, participants' perception of T2 had to be sufficiently detailed to not only render it recognizable as a target but also to support the formation of a coherent whole-word percept. On some proportion of trials, a crude representation of T2 may have initially entered working memory and been detected as a target, thereby eliciting a P300, but have been of insufficient quality to support explicit awareness.

An additional parameter distinct to the present linguistic AB task is that it may impose higher working memory demands than other AB studies, which could potentially contribute to a late processing bottleneck. The requirement to maintain a unique prime in mind throughout the RSVP stream and to perform a linguistic judgment about a new word on every trial necessarily places an added burden on working memory resources above and beyond typical AB task demands. Previous studies have shown that increasing working memory load increases distractor interference, presumably by compromising the executive system's ability to actively reject irrelevant distractors (e.g., de Fockert et al. 2001; Lavie et al. 2004). Thus, this additional working memory load may have increased T2's

vulnerability to interference after its initial entry into working memory. Under this scenario, short-lag targets that were incorrectly reported would initially enter working memory, generating a P300, but ultimately be lost due to competition for limited working memory resources before a linguistic decision could be made.

From a theoretical standpoint, current results are not compatible with models that propose that the AB is due to a failure of short-lag targets to be consolidated into working memory, such as the two-stage model (Chun and Potter 1995). Under the assumption that the P300 indexes consolidation into working memory (e.g., Donchin 1981; Donchin and Coles 1988; Polich and Criado 2006; Polich 2007), this model would predict that incorrectly reported targets at the short lag should elicit a reduced P300 relative to correctly reported targets, even if some aspects of the stimulus, such as its color, are partially consolidated into working memory. Our findings appear more consistent with the interference model, which posits that T2 enters working memory but is later lost due to competition with T1 and distractor items (Shapiro et al. 1994, 1997). According to this model, both correctly and incorrectly reported targets would initially enter working memory and elicit a P300, a prediction that is supported by current findings. Alternatively, the AB bottleneck could also occur independently of working memory, as a result of impairment in another parallel and limited-capacity processing stream. Subjective states of awareness have been demonstrated to correlate with a number of different objective electrophysiological measures, including the P300 component, gamma-band oscillations, late amplification of sensory activation, joint activation of prefrontal, parietal, and cingulate areas, and phase synchronization across distance regions (Dehaene and Changeux 2011). Though it is not yet known whether these diverse measurements reflect parallel processes occurring along multiple pathways or the same underlying phenomenon, it is possible that the disruption of any one of the mechanisms indexed by these effects may prevent a stimulus from reaching awareness. For example, even if a stimulus generates a P300, suppression of gamma-band oscillations may still impair critical binding processes and ultimately prevent the target from reaching a stable state of awareness.

Overall, the finding that the P300 awareness effect differs as a function of lag suggests that different mechanisms are responsible for misses that occur within versus outside the AB period. Misses that occur at long lags may be caused by a failure of T2 to completely consolidate in working memory, as reflected by the significant P300 reduction to incorrectly reported targets presented after the AB period (Fig. 3). At these long lags, T1-related processes no longer interfere with T2 processing, as a sufficient period of time separates the targets. Rather, T2 may occasionally fail to reach awareness as the result of a more general impairment in processing, likely caused by a high perceptual load induced by the rapid presentation of distracter items. This high perceptual load may decrease the availability of attentional resources, ultimately preventing T2 from being completely consolidated into working memory on some trials. Alternatively, the reduced P300 to incorrect long lag targets may be due to low confidence in the identity of the target as participants prepare a response (Squires et al. 1973; Makeig et al. 2004; Verleger et al. 2005). Under this explanation, the lack of P300 differences between correct and incorrectly reported trials at

the short lag may be attributable to low confidence in the identity of both correctly and incorrectly reported T2s.

Failures of awareness occurring outside the AB period appear to occur with impairments in both earlier mechanisms indexed by a P3a-like effect as well as later processes reflected by a P3b component. As illustrated by the topographical distribution and latency of the P300 awareness effect at the long lag (Fig. 3B), reported targets showed both an enhanced anterior positivity that began relatively early (~200 ms postsimulus) as well as a larger posterior postivity with a later onset (~500 ms), resembling the P3a and P3b, respectively. Thus, both the automatic orienting response indexed by the P3a and the working memory processes indexed by the P3b appear to contribute to a target's entry into awareness (Polich 2007). In contrast, misses occurring within the narrower time window of the AB appear to be caused by a more specific impairment, driven by T1 competition, that appear to act on processes other than those indexed by the P3a and P3b.

Gamma Oscillations and Awareness

The major finding revealed by our spectral analysis was a late (400–650 ms) gamma increase to correctly reported targets relative to incorrectly reported targets during the AB period. This result converges with a previous spectral AB study (Kranczioch et al. 2007), in which a significant gamma increase 660–760 ms postsimulus was found to detected T2s relative to undetected T2 presented within the AB period. In our study, this gamma enhancement relative to baseline appears to have a similar topography—a broad central distribution—for correct and incorrect targets at the long lag and for correctly reported targets at the short lag.

These findings converge with more general findings linking later stimulus-induced gamma to object representation and target awareness. As hypothesized by several groups (e.g., Rodriguez et al. 1999; Tallon-Baudry and Bertrand 1999; Fisch et al. 2009; Gaillard et al. 2009), synchronization in this range may act to integrate spatially distant neural networks that mediate different aspects of the target's representation, allowing the representation to become stable enough to reach conscious awareness. Interestingly, late gamma occurring in the same range as in the present study has also been previously linked with explicit rehearsal or retrieval processes (Tallon-Baudry et al. 1998; Gruber et al. 2001; Kranczioch et al. 2006). One tentative interpretation, then, is that this response may be indexing participants' integration and representation of the just perceived target, allowing it to be correctly reported at the end of the trial.

We did not find a difference in gamma amplitude to reported targets relative to unreported targets outside the AB. Rather, both correctly and incorrectly reported targets showed a late induced gamma enhancement relative to baseline. Although speculative, one possible explanation for this finding is that at long lags, the greater interval of time between T1 and T2 allows the prime word to bias the representation of T2 through top-down processes such as expectancy-induced priming, in which subjects consciously or unconsciously generate a set of likely targets based on the preceding linguistic context (Neely 1991). In the case of missed trials, although T2 has not in reality been correctly perceived, the participant may incorrectly believe that one of the targets in the generated set appeared, producing an incorrect representation of T2's identity. This late gamma may then reflect binding of this inaccurate representation. This explanation is also supported by

an inverted N400 pattern found at the long lag to these stimuli (Batterink et al. 2010). Parallel to our P300 findings, this result suggests that misses occurring to targets at the short and long lag stem from different neural mechanisms.

It is important to note that the broad scalp distribution, constrained frequency profile (55–70 Hz), and late and prolonged time course of our gamma effect are all inconsistent with the spectral profile characteristic of miniature saccades. Induced broadband transient gamma-band responses recorded in scalp EEG reflect miniature saccade electrical artifacts, rather than synchronous neural oscillations associated with object representation and awareness (Yuval-Greenberg et al. 2008).

Relationship of P300 and Gamma in Awareness

Our results shed light on a number of issues concerning the neural and cognitive mechanisms underlying human awareness. At the physiological level, we found that the P300 and gamma doubly dissociate, with P300 enhancement to reported targets occurring only outside the AB, and gamma enhancement to reported targets occurring only within the AB. This apparent dissociation converges with at least one previous finding that found no association between the P300 and the late gamma response in an RSVP task (Kranczioch et al. 2006). However, as we also found no evidence of a negative association between the P300 and the gamma, our results are not consistent with the stronger claim that the P300 actively suppresses gamma activity (Fell et al. 2002). Rather, the present findings provide evidence that late gamma and the P300 reflect largely nonoverlapping neural mechanisms, with no strong connection or interaction between them.

At a cognitive level, our data suggest that the cognitive processes indexed by both the gamma and the P300 appear to be necessary for awareness. During the AB period, failures of awareness were associated with reduced gamma amplitude, while outside the AB period, failures of awareness were predicted by a reduction in the P300. These findings suggest that there are multiple cognitive processes operating in parallel to support awareness, each of which may be vulnerable to disruption by a particular set of external demands. Impairment of either one of the cognitive functions indexed by gamma and the P300 may prevent complete processing of the target, precluding its entry into awareness.

Finally, concerning the fundamental limitation underlying the AB phenomenon, both our P300 and gamma data provide parallel lines of evidence indicating that qualitatively different neural mechanisms underlie misses occurring within versus outside the AB period. While reduced gamma activity appears to increase the likelihood that a target will be missed during the AB, it does not appear to play a role in missed targets outside the AB. Similarly, P300 reduction predicted failures of awareness occurring outside the AB but was not implicated in misses occurring during the AB. These data are consistent with the idea that the AB bottleneck occurs independently of working memory consolidation, instead reflecting impairment in a stage of processing either subsequent or parallel to the target's entry into working memory.

In conclusion, our results provide experimental evidence that the P300 and gamma activity reflect functionally distinct neural mechanisms, each of which plays an independent yet critical role in awareness. Although there have been few studies to directly compare P300 and gamma under different conditions of awareness, this conclusion is consistent with previous literature on the functional significance and neural underpinnings of these electrophysiological measures. As discussed previously, the P300 is most often linked to updating of an incoming representation into working memory and likely recruits a widespread cortical network, including the temporal–parietal junction, the medial temporal complex, and the lateral prefrontal cortex (Soltani and Knight 2000; Polich and Criado 2006). In contrast, gamma activity is thought to reflect transient functional integration between specific groups of neurons, providing the spatial and temporal links necessary to bind together different neural assemblies and create a coherent percept (Tallon-Baudry and Bertrand 1999; Doesburg et al. 2009). In sum, current findings converge with the broader literature's proposal that P300 and gamma are generated by dissociable neural substrates and index conceptually different cognitive processes.

Supplementary Material

Supplementary material can be found at: http://www.cercor.oxfordjournals.org/

Notes

Conflict of Interest : None declared.

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