Awareness is related to reduced post-stimulus alpha power: A no-report inattentional blindness study

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Abstract

Delineating the neural correlates of sensory awareness is a key requirement for developing a neuroscientific understanding of consciousness. A neural signal that has been proposed as a key neural correlate of awareness is amplitude reduction of 8–14 Hz alpha oscillations. Alpha oscillations are also closely linked to processes of spatial attention, providing potential alternative explanations for past results associating alpha oscillations with awareness. We employed a no-report inattentional blindness (IB) paradigm with electroencephalography to examine the association between awareness and the power of 8–14 Hz alpha oscillations. We asked whether the alpha-power decrease commonly reported when stimuli are perceived is related to awareness, or other factors that commonly confound awareness investigations, specifically task-relevance and visual salience. Two groups of participants performed a target discrimination task at fixation while irrelevant non-salient shape probes were presented briefly in the left or right visual field. One group was explicitly informed of the peripheral probes at the commencement of the experiment (the control group), whereas the other was not told about the probes until halfway through the experiment (IB group). Consequently, the IB group remained unaware of the probes for the first half of the experiment. In all conditions in which participants were aware of the probes, there was an enhanced negativity in the event-related potential (the visual awareness negativity). Furthermore, there was an extended contralateral alpha-power decrease when the probes were perceived, which was not present when they failed to reach awareness. These results suggest alpha oscillations are intrinsically associated with awareness itself.

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Perceptual awareness – the subjective experience of perceiving the environment and the objects in it – is intrinsic to human experience. Critical to understanding perceptual awareness is the identification of neural processes that accompany, and potentially give rise to it; the so-called neural correlates of consciousness (Crick & Koch, 1990). A number of potential neural correlates of consciousness have been identified (for review, see Rees et al., 2002; Koch et al., 2016). Determining which of these are associated with sensory awareness itself, and which are associated with distinct but commonly coincident cognitive processes (e.g., attention, memory encoding, etc.) is an important challenge for understanding the neural processes that give rise to subjective perceptual experience. One neural signal that has been repeatedly associated with perceptual awareness is oscillatory amplitude reduction in the 8–14 Hz 'alpha' band (e.g., Vanni et al., 1997; Babiloni et al., 2006; Bareither et al., 2014). However, alpha oscillations are also commonly associated with the allocation of spatial attention (Foxe & Snyder, 2011; Klimesch, 2012), leading to potential alternative explanations for previous results relating alpha oscillations to sensory awareness. In the present work we address some of these alternative explanations. We measured alpha oscillations during a no-report inattentional blindness paradigm to determine whether awareness itself is accompanied by alpha amplitude reduction, or whether past reports of alpha amplitude reduction associated with awareness were due to attention-related confounds such as goal-relevance or visual salience.

Much of the literature examining the link between alpha oscillations and visual processes has focused on their association with the allocation of spatial attention (Foxe & Snyder, 2011; Klimesch, 2012). The scalp topography of posterior alpha amplitude strongly reflects the locus of spatial attention. When participants are cued to attend to one side of space, the power of alpha oscillations is reduced contralateral to the attended hemifield, and relatively increased ipsilateral to the location of attention (Worden et al., 2000; Sauseng et al., 2005; Kelly et al., 2006; Thut et al., 2006; Gould et al., 2011; Rohenkohl & Nobre, 2011; Ikkai et al., 2016). Alpha oscillations are also modulated in this lateralized manner when attention is involuntarily captured to one visual hemifield (Feng et

al., 2017; Harris et al., 2017), or voluntarily allocated in the absence of a spatial cue (Bengson et al., 2014). Moreover, studies employing multivariate approaches have demonstrated that the spatial information contained in the distribution of alpha oscillations across electrodes is far more detailed than simply ipsilateral versus contralateral, allowing tracking of both the breadth of attentional distribution, and its specific location (Samaha et al., 2016; Foster et al., 2017; Voytek et al., 2017).

Other studies have suggested that post-stimulus alpha amplitude change may be a neural correlate of consciousness. When a visual stimulus is perceived, alpha oscillations measured over parieto-occipital cortex typically show an amplitude decrease that is absent or reduced when the same stimulus fails to reach awareness (Vanni et al., 1997; Babiloni et al., 2006). For example, Babiloni et al. (2006) had participants report whether or not they had seen a masked stimulus, and found that perceived stimuli elicited significantly lower post-stimulus alpha power than stimuli that did not reach awareness. In light of the literature linking alpha oscillations to attention, however, this awareness-related alpha effect is also consistent with allocation of attention accompanying the perception of a stimulus. When participants are required to report their awareness of a stimulus on each trial, perceived stimuli become task-relevant (Aru et al., 2012). This may produce alpha amplitude reduction as a result of attentional allocation to task-relevant stimuli (Harris et al., 2017) and not because alpha amplitude reduction is intrinsically associated with awareness. Perhaps when stimuli are not task-relevant they may be perceived without being attended (Koch & Tsuchiya, 2007), and so produce no alpha amplitude change.

The practice of having participants report their awareness of a stimulus on each trial has been criticized for confounding neural responses related to awareness with those related to other processes such as task-relevance and report (Aru et al., 2012). This has led to the development of “no-report” paradigms (Tsuchiya et al., 2015), which do not require participants to report their awareness of a stimulus on each trial. Studies employing no-report paradigms have revealed that brain responses previously considered as neural correlates of consciousness, such as frontal BOLD activity (Frässle et al., 2014), the P3b event-related potential (ERP) component (Pitts et al., 2014a,b; Shafto & Pitts, 2015), and occipital gamma activity (Pitts et al., 2014b), are in fact correlates of decision- or response-

related processes.

In the phenomenon of *inattentional blindness* (Mack & Rock, 1998; Simons, 2000), participants performing an attention-demanding task often do not perceive an unexpected stimulus presented in the display. Recently, Pitts et al. (2012; see also Pitts et al., 2014b) developed a no-report inattentional blindness paradigm to examine the neural correlates of consciousness with electroencephalography (EEG). Participants fixated a central array of small line segments that changed orientation roughly twice per second and detected unexpected contrast decrements of a stimulus in the periphery (**Figure 1**). On half the trials, unknown to participants, line segments in the central array briefly arranged themselves into a geometric shape (square or diamond; the *probe*). The experiment proceeded in three phases, each of which was followed by a questionnaire assessing participants' awareness of the probes. In Phase 1, participants were not informed about the presence of the probes; and indeed, half of them remained unaware of their occurrence, thus showing inattentional blindness. In Phase 2, all participants now reported being aware of the probes, presumably because they had been cued to their presence at the end of Phase 1. In Phase 3, participants were instructed to respond whenever a diamond shape appeared in the central display, thus effectively making shape information task-relevant.

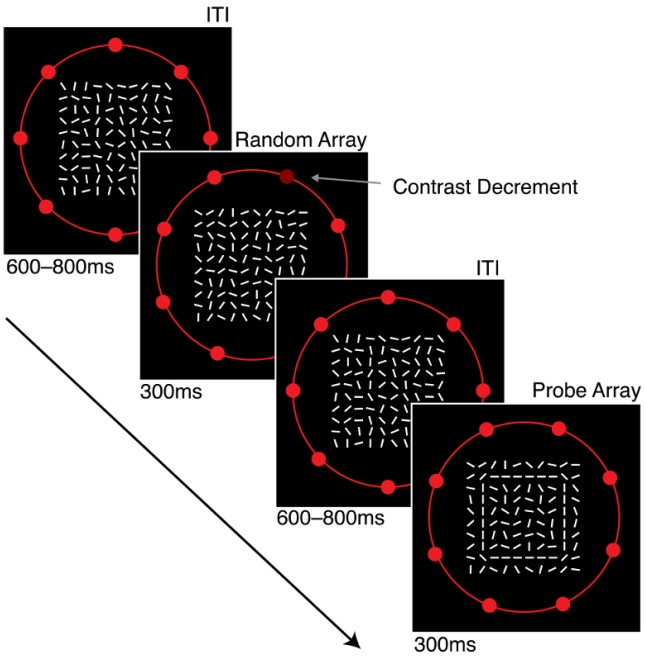

Figure 1. Schematic of stimuli from Pitts et al. (2014). See the main text for a description of the task. Line arrays here are simplified schematics, and in the experiment contained 20 x 20 line segments. ITI = inter-target interval.

Pitts et al. (2014b) used EEG to examine neural responses elicited by the probe events in each of the three phases and found no P3b component or gamma activity in Phases 1 or 2, despite the fact that the probes were perceived by half of the participants in Phase 1, and by all participants in Phase 2. A P3b component and increased gamma response were present only in Phase 3, when shape information was now task-relevant. These results run counter to the widely-held view that the P3b (e.g., Dehaene & Changeux, 2011) and gamma activity (e.g., Fisch et al., 2009) are neural correlates of awareness itself. Instead, Pitts et al. (2012) found that awareness was related to a negativity in the ERP to the shape probe in all aware conditions, and this ERP was absent in participants who were not aware of the probe in Phase 1. The negativity that arises when a stimulus is consciously perceived versus missed has been labelled the *visual awareness negativity* (VAN; for review see Koivisto & Revonsuo, 2010). These results suggest that no-report paradigms can be used to dissociate neural correlates of awareness from those related to task-relevance or report (Aru et al., 2012).

One previous study used a no-report paradigm to examine post-stimulus alpha activity related to awareness, without the confound of task relevance (Bareither et al., 2014). The authors presented brief peripheral luminance stimuli either at 25% of contrast detection threshold (the *subliminal condition*) or at 500% of detection threshold (the *supraliminal condition*), while participants performed a central counting task. Participants were required to ignore the peripheral stimuli and, to maintain the no-report nature of the task, awareness of the peripheral probes was not assessed. Rather, it was assumed that stimuli well above detection threshold would be perceived on a majority of trials, and stimuli well below detection threshold would not reach awareness on a majority of trials. Consistent with past studies showing alpha amplitude reduction associated with awareness (Vanni et al., 1997; Babiloni et al., 2006), the results revealed a contralateral alpha power reduction for supraliminal peripheral stimuli, relative to when no peripheral stimuli appeared. By contrast, there was no alpha power decrease, and instead a small alpha power *increase*, following presentation of subliminal stimuli. These results seem to suggest that alpha amplitude decreases when stimuli are perceived, even when those stimuli are not task-relevant. It has long been known, however, that stimulus onsets, particularly those involving salient luminance changes, tend to capture attention involuntarily under many task conditions (e.g., Yantis & Jonides, 1984; Franconeri et al., 2005). Without a stimulus-matched unaware condition, therefore, it is impossible to know whether the alpha power effects observed by Bareither et al. (2014) were related to awareness per se, or to attentional capture by the highly salient onset stimuli. It may be that without a salient onset, or any other property that involuntarily captures attention (e.g., Abrams & Christ, 2003; Franconeri & Simons, 2003; Guo et al., 2010), task-irrelevant stimuli might be perceived without the involvement of attention or any related reduction in alpha power.

To address the ambiguities in previous studies that suggested a link between alpha power and awareness (Vanni et al., 1997; Babiloni et al., 2006; Bareither et al., 2014), here we employed a no-report inattentional blindness paradigm to examine changes in alpha power associated with awareness of task-irrelevant, non-salient stimuli. We modified the paradigm developed by Pitts et al. (2014b; also, Pitts et al., 2012) to present irrelevant

probes in the left and right periphery while participants performed a central task, allowing us to examine EEG amplitude changes at both ipsilateral and contralateral electrode sites. This allowed us to link alpha amplitude reduction to the specific location of any irrelevant probes, and to rule out more general processes such as non-spatial alerting (Klimesch et al., 1998). We employed two groups of participants: an inattentionally blind group who were unaware of the probe stimuli in the first phase, and a control group who were aware of the probes throughout the experiment. If awareness is associated with alpha power reduction, we would expect to observe a contralateral alpha power decrease in all conditions in which participants were aware of the probes. If awareness is not associated with alpha power reduction, however, and alpha power change in past studies was due attention (e.g., due to task-relevance or attentional capture; Vanni et al., 1997; Babiloni et al., 2006; Bareither et al., 2014), then we would expect awareness of the peripheral probes to produce no lateralized alpha-power decrease when these factors are controlled. Previewing the findings, our results were consistent with the former possibility. Despite the probe stimuli being task irrelevant and non-salient, and producing little-to-no behavioural interference, awareness of the probes was associated with a contralateral power decrease in the alpha range that was not present when participants remained unaware of the probes.

Materials and Methods

Participants

Forty-eight individuals participated in the experiment (aged 18-30 years, mean = 21.69, SD = 2.26, 25 females). Twenty-four individuals were allocated to an inattentional blindness (IB) group, and the other 24 were allocated to a control group. All participants self-reported as right handed, had normal or corrected-to-normal vision, and provided informed consent prior to participating in the experiment. One participant was excluded from the IB group because he removed his EEG cap halfway through data collection, leading to early termination of the experiment. Participants were compensated for their time at a rate of $10 per hour. All participants provided written informed consent, and the

study conformed with the World Medical Association Declaration of Helsinki. The study was approved by The University of Queensland Human Research Ethics Committee.

Behavioral Task

We used an inattentional blindness paradigm adapted from Pitts and colleagues (2014b; also, Pitts et al., 2012). Participants fixated a central red cross (15' x 15'; RGB: 255, 0, 0) on a black background (RGB: 0, 0, 0). At the center of the screen, three 20 x 20 arrays of small white line segments (RGB: 255, 255, 255), were laid out side-by-side (**Figure 2**). As described in detail below, the central array was used to display target stimuli, and the left- and right-sided arrays were used to display peripheral probes. Each line segment within the arrays subtended 15', and each 20 x 20 array subtended 6°. The three arrays were separated by 30'. By default, every line segment was randomly arranged in one of eighteen orientations (every ten degrees from 10° to 180°). On each trial, two displays were presented; an inter-target interval of 600 - 800ms, followed by a target display for 300ms. On both displays, a new random orientation was selected for each line segment (except where noted below), so that the lines in the arrays appeared to be 'jittering' (for a demonstration, see: https://youtu.be/ivXgLgrbn3w). On 50% of target displays, either the left or right peripheral line array contained a square, centered within the line array, formed by the alignment of 12 x 12 line segments on the borders of the square (see **Figure 2**). Fully crossed with these *peripheral probe trials*, 50% of target displays were *central target trials*, which contained either three or four red patches within the central line array. Each red patch was a 2 x 2 set of lines presented in red rather than white. Red patches all overlapped the 12 x 12 line border of an imaginary square (but in the center line array), and were positioned such that no two red patches touched. Half of central target trials contained three red patches, and half contained four. Participants were instructed to maintain fixation on the fixation cross, and to respond whenever they saw three red patches, but not four, or vice versa (counterbalanced across participants).

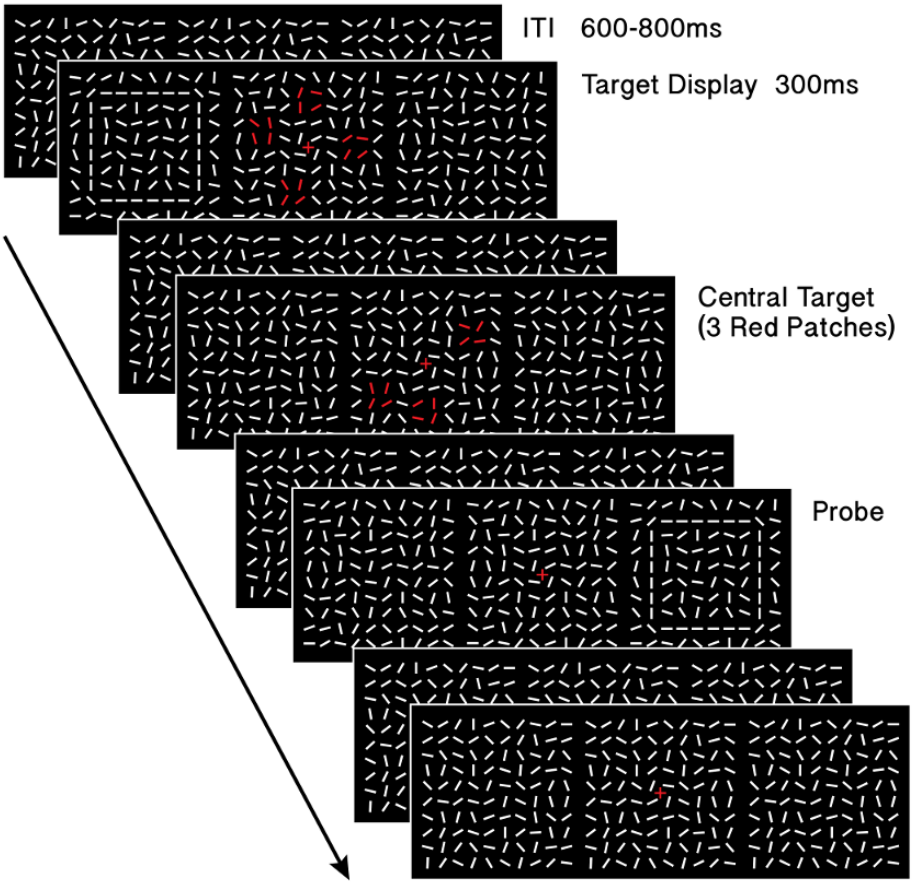

Figure 2. Schematic of experimental paradigm (not to scale). Participants fixated centrally and responded when they saw 3 or 4 red patches (counterbalanced across participants), that only ever appeared in the central array. Participants were instructed to ignore the peripheral arrays, in which probes (squares) appeared on 50% of trials. The control group were told they may see some shapes in the periphery, whereas the inattentional blindness group were not informed about the presence of the peripheral probe shapes. Line arrays here are simplified schematics, and in the experiment contained 20 x 20 white lines each. ITI = inter-target interval.

At the beginning of the experiment, participants in the IB group were told that the peripheral arrays were irrelevant to the task, and they should ignore them and focus on the task in the center line array. No mention was made of the shape probes. By contrast, participants in the control group were told they might sometimes see the lines in the peripheral arrays arrange themselves into a shape (the specific shape – a square – was not mentioned), but that these were irrelevant to their task, and they should ignore the peripheral arrays and focus on the task in the center array. This was the only difference in

the instructions given to the two groups. This manipulation was expected to cue the control group, but not the IB group, to the presence of the probes from the start of the experiment.

Participants first completed 300 trials in which peripheral probes were not presented, to allow them to become used to the task prior to the presentation of the probes. These trials were treated as practice and were not analyzed. Participants then completed 760 trials of the full task (*Phase 1*), including peripheral probe trials, before being given a questionnaire to assess their awareness of the probes (see below). It was expected that this questionnaire would cue any previously unaware participants to the presence of the probes. After completing the first questionnaire, participants undertook a further 760 trials (*Phase 2*), before completing the questionnaire a second time. The experiment was thus divided into two phases, such that the IB group should have been unaware of the peripheral probes in Phase 1, and aware of them in Phase 2. By contrast, we expected the control group to be aware of the probes in both Phases 1 and 2. It should be noted that, due to the nature of no-report paradigms, we are limited to comparing average responses across a whole phase of trials. We cannot determine whether participants perceived all or only some of the probes in any particular 'aware' phase, and we cannot determine whether a participant was aware of the probe on any individual trial. However, what is key is the comparison of Phase 1 performance between the two groups (IB versus control), and also the comparison between Phase 1 and Phase 2 performance for the IB group. Any conscious registration of the probes in Phase 1 for the IB group would contribute toward null differences between the groups and phases. Participants were given a self-paced break at the end of every 60 trials, and a forced break of 30 seconds after every 300 trials.

In the awareness assessment questionnaires, participants were first asked whether they noticed any patterns within any of the three sets of line arrays. If participants responded 'yes', they were then asked to write or draw a description of what they saw in as much detail as possible. Following completion of the first two items, participants were given examples of line arrays containing six different shapes (diamond, horizontal rectangle, X pattern, one large square, four small squares, vertical rectangle), and

completed two rating scales. The first rating scale asked participants to report how confident they were that they had seen each of the six shapes, on a scale from 1 = very confident they did *not* see the shape, to 5 = very confident they *did* see the shape (where 3 = unsure). The second rating scale asked participants to estimate how often they saw each shape, from 1 = never, to 5 = very frequently/more than 100 times. The questionnaires were identical to those of Pitts et al. (2012); see the Appendix of Pitts et al. (2012) for examples of the questionnaire with rating scales. Participants in the IB group were excluded from analysis if in Phase 1 they rated their confidence in having seen a square as 4 or 5, or if they described seeing a square in the first question of the questionnaire. Participants in the control group were excluded from analysis if in Phase 1 they rated their confidence in having seen a square as 3 or below, unless they described seeing a square in the first question of the questionnaire.

Stimuli were presented on an Asus VG248 LCD monitor with a resolution of 1920 x 1080 and a refresh rate of 60 Hz. Stimulus presentation was controlled using the Psychophysics Toolbox 3 extension (Brainard, 1997; Kleiner et al., 2007) for MATLAB, running under Windows 7. Viewing distance was maintained at 57cm with the use of a chinrest. Participants made their responses by pressing the spacebar on a standard USB keyboard with their right hand.

EEG recording

Continuous EEG data were recorded using a BioSemi Active Two system, digitized at a rate of 1024 Hz with 24-bit A/D conversion. The 64 active Ag/AgCl scalp electrodes were arranged according to the international standard 10–10 system for electrode placement (Chatrian et al., 1985), using a nylon head cap. As per BioSemi system design, the Common Mode Sense and Driven Right Leg electrodes served as the ground, and all scalp electrodes were referenced to the Common Mode Sense during recording. Eye movements were monitored online using bipolar horizontal electro-oculographic (EOG) electrodes placed at the outer canthi of each eye, and bipolar vertical EOG electrodes placed above and below the left eye. Left and right mastoid electrodes were employed for use as a reference for the ERP analysis.

EEG analysis

Offline EEG preprocessing was performed with the EEGLAB Toolbox (Delorme & Makeig, 2004) for MATLAB, and analyses were performed with custom-written MATLAB functions (some adapted from Cohen, 2014).

ERPs were analyzed to allow comparison of our results with those of Pitts et al. (2012). For the ERP analyses, the data were down-sampled to 256 Hz and re-referenced to the average of the mastoid electrodes. The appearance of the target arrays roughly every 1 second produced a large ~1 Hz steady-state visual evoked response (Regan, 1989; Norcia et al., 2015) that made the waveforms difficult to compare between conditions. To remove this component, we high-pass filtered the data at 1.25 Hz, using a Kaiser windowed FIR filter with a passband deviation of .0001 and a filter order of 5138 samples, giving a transition bandwidth of 0.25 Hz. The data were then low-pass filtered at 30 Hz with a Kaiser windowed FIR filter with a passband deviation of .0001 and a filter order of 130 samples, giving a transition bandwidth of 10 Hz. Trial epochs were extracted from -300ms to 800ms post target-array onset, and baseline adjusted relative to a period between -40ms and +40ms (see below). The data were contralateralized by flipping the EEG topographies horizontally on trials in which the probe appeared on the left. This served to combine data that were contralateral (or ipsilateral) to the target, regardless of the target's actual location. Trials containing large muscle artefacts, blinks, or eye movements were automatically rejected if their activation levels exceeded ±75μV on any channel. The data were then visually inspected to remove any remaining trials containing artefactual activity. The 75μV threshold might have missed some small eye movements, but the centre of each peripheral array was >6° from fixation. Thus, any problematic eye movements were typically large when they occurred and were therefore readily detected and eliminated. These procedures resulted in an average loss of 15.6% of trials per participant in the IB group, and 16.5% of trials in the control group.

For ERP analyses, we employed an unconventional baseline period from -40 to 40ms, rather than the typical baseline from -100 to 0ms. This was due to a large prestimulus difference in the ERPs between probe and no-probe trials in Phase 1 for the

Control group, which led to a large offset between the ERPs for probe and no-probe trials at all post-target-onset time points when the typical baseline was used. We chose to baseline our ERPs from -40 to 40ms, as this period began after the baseline difference had disappeared and ended before the earliest visually-evoked ERP responses are observed (e.g., the C1 component; Luck et al., 2000). We ran control analyses to confirm that this unusual baselining did not induce spurious ERP differences between probe and no-probe trials at any time point. First, we compared the probe minus no-probe difference waves calculated with a typical baseline from -100 to 0ms to those calculated from a -40 to 40ms baseline, for each group in each phase of the experiment. We found the two baselines to be equivalent for the IB group in both Phase 1 and Phase 2, *p*s > .616, and for the Control group in Phase 2, $p = .720$. As already described, there was a significant difference between the difference waves produced by the two baseline periods in Phase 1 for the control group, $p < .001$. This analysis suggests that the use of a -40 to 40ms baseline period produces equivalent results to a -100 to 0ms baseline when there is no difference in the baseline period. Next, to confirm that the -40 to 40ms baseline adequately aligned the probe and no-probe ERPs for the Control group in Phase 1, we compared probe minus no-probe difference waves between Phase 1 (which showed the baseline difference) and Phase 2 (which showed no baseline difference, and no effect of baseline choice), with both baselines corrected from -40 to 40ms. This comparison was made at all time points throughout the trial. Participants in the control group were aware of the probes in both phases of the experiment (see below), so we would expect the difference waves in each of the phases to be equivalent at all time points. The difference waves significantly differed from one another in the pretrial period (from -102 to -58ms; *p*s > .013), as expected. The only other effect was a small difference at two post-target time-points (160-164ms; *p*s > .036, uncorrected). Note that this is fewer than the 10.2 false positives that would be expected from 204 post-target-onset comparisons, and does not survive correction for multiple comparisons, suggesting it is likely due to chance. In summary, we observed little or no discrepancy between the ERP difference waves for Phase 1 versus Phase 2 in the control group when using a -40 to 40ms baseline period, as would be expected when employing an appropriate baseline correction. These results

suggest the period from -40ms to +40ms is a valid baseline period. As a final note, it is worth pointing out that any ERP differences were not of primary interest in our study and were included only for purposes of comparison with Pitts et al. (2012). Rather, our primary interest was in time-frequency amplitude differences between probe and no-probe trials, which are not influenced by baseline activity (see below).

EEG responses to the probes were only analysed for trials in which no central targets were present, to avoid contamination by factors related to task-relevance. It was not appropriate for us to examine the same electrodes as Pitts et al. (2012), as our probes were presented peripherally rather than centrally, and so would be expected to produce a different topography. Instead, we followed the same procedure for selecting electrodes as that described by Pitts et al. (2012). Two symmetrical clusters of electrodes were selected as regions of interest (ROIs) for analysis by visually examining the location and time of greatest difference between peripheral probe and no-probe trials, collapsed across the two phases of the experiment and across the two groups (Figure 3). It should be noted that this electrode selection method is not circular, as our primary interest is the difference between phases 1 and 2 for the IB group, and between the groups at Phase 1, and these were collapsed together in the selection procedure. The selected ROI electrodes were CP3/4, P1/2, P3/4, P5/6, PO3/4, across the period from 260ms to 320ms, which is similar to that of Pitts et al. (2012). The earlier difference between ~200ms and 260ms (Figure 3), was not included in the analysis because it had a more central topography, consistent with the Nd1 component which Pitts et al. (2012) demonstrated was not associated with awareness. Statistical analyses were performed by comparing the probe versus no-probe difference waves between the two groups and between the two halves of the experiment. As there are no contralateral or ipsilateral electrodes on no-probe trials, probe trials were compared against the average of the left and right electrode clusters on no-probe trials.

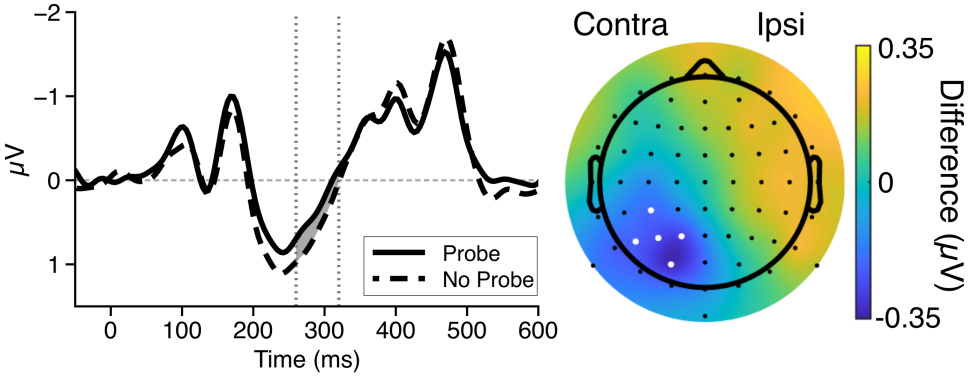

Figure 3. Grand mean ERP difference between probe and no-probe trials, collapsed across groups and phases. Vertical dotted lines indicate the selected time window for analysis from 260-320ms. The scalp topography represents the ERP difference at all electrodes, averaged across this period.

For the time-frequency analyses, the raw data were down-sampled to 256 Hz and referenced to the average of all scalp electrodes, then epoched from 2000ms prior to 2000ms post target-array onset. The same artefact-containing trials as identified in the ERP analysis were excluded from the time-frequency analyses. Power estimates for 30 logarithmically spaced frequencies from 2 Hz to 80 Hz were extracted using Morlet wavelets, with the number of wavelet cycles logarithmically scaled from 3 to 10 cycles. Power estimates for ipsilateral and contralateral electrode clusters on peripheral probe trials were separately compared with those measured at the same electrodes on probe-absent trials, normalized by the average of probe and no-probe trials, as follows:

$$PowerDifference_{tf} = \frac{Probe_{tf} - NoProbe_{tf}}{2^{-1}\left(Probe_{tf} + NoProbe_{tf}\right)}$$

where subscript *t* denotes a particular time point, and subscript *f* denotes a particular frequency. For example, to produce the power difference at contralateral electrodes, trials in which probes appeared on the left had their contralateral electrodes (on the right side of the scalp) compared with the same right side electrodes on no-probe trials, and this was

averaged with the result of comparing left-side electrodes on trials in which probes appeared on the right with left-side electrodes on no-probe trials. The same procedure was employed for ipsilateral trials, comparing ipsilateral electrodes with the same electrodes on no-probe trials, normalized by the average of the two sets of trials. This modulation index approach was employed as it does not use a pretrial baseline, and thus cannot be subject to the issue with baseline differences that was apparent in the ERP analyses. Statistical comparisons, controlling the familywise error rate, were made by down-sampling to 128 Hz and performing cluster-based permutation tests (Groppe et al., 2011) across all frequencies and all times from 0ms to 800ms following onset of the probe displays, using an alpha level of .01 and a null distribution calculated across 5000 random permutations.

Results

Behavioral Results

The awareness assessments showed that all but one of the participants in the control group were aware of the peripheral probes in both phases of the experiment. Two additional participants from the control group rated their confidence in having seen a square as '3 = uncertain' in Phase 1, but were included in the sample as each spontaneously reported seeing a square in the initial open-ended question of the questionnaire (prior to being exposed to information on the available shape categories). Although only one participant in the IB group spontaneously reported perceiving the square, four additional participants rated their confidence for having seen a square as '4 = confident I saw it', or '5 = very confident I saw it', and so were excluded from further analyses. In Phase 2, all participants in the IB group met our criteria for awareness of the peripheral probes. The frequency with which each rating was selected for each shape is shown separately for the two groups and the two phases in Figure 4. The conclusion of inattentional blindness in the IB group in Phase 1, and awareness of the probes in all other conditions, was confirmed by performing separate ANOVAs on the confidence and frequency ratings, each with a between-subjects factor of group (2 levels: Inattentionally

blind, Control), and within-subjects factors of phase (2 levels: Phase 1, Phase 2) and shape (6 levels: Large Square, Diamond, Horizontal Rectangle, X Pattern, Four Small Squares, Vertical Rectangle). One participant from the IB group was excluded from the frequency-rating analysis as they omitted a frequency rating for the Horizontal Rectangle in Phase 2. These analyses both revealed significant 3-way interactions between group, phase, and shape (Confidence: $F(5,195) = 11.95$, $p < .001$, $\eta^2 = .17$; Frequency: $F(3.75,142.67) = 9.29$, $p < .001$, $\eta^2 = .15$), demonstrating that squares received higher confidence and frequency ratings than the other shapes in both phases for the control group, but only in Phase 2 for the IB group (**Figure 4**).

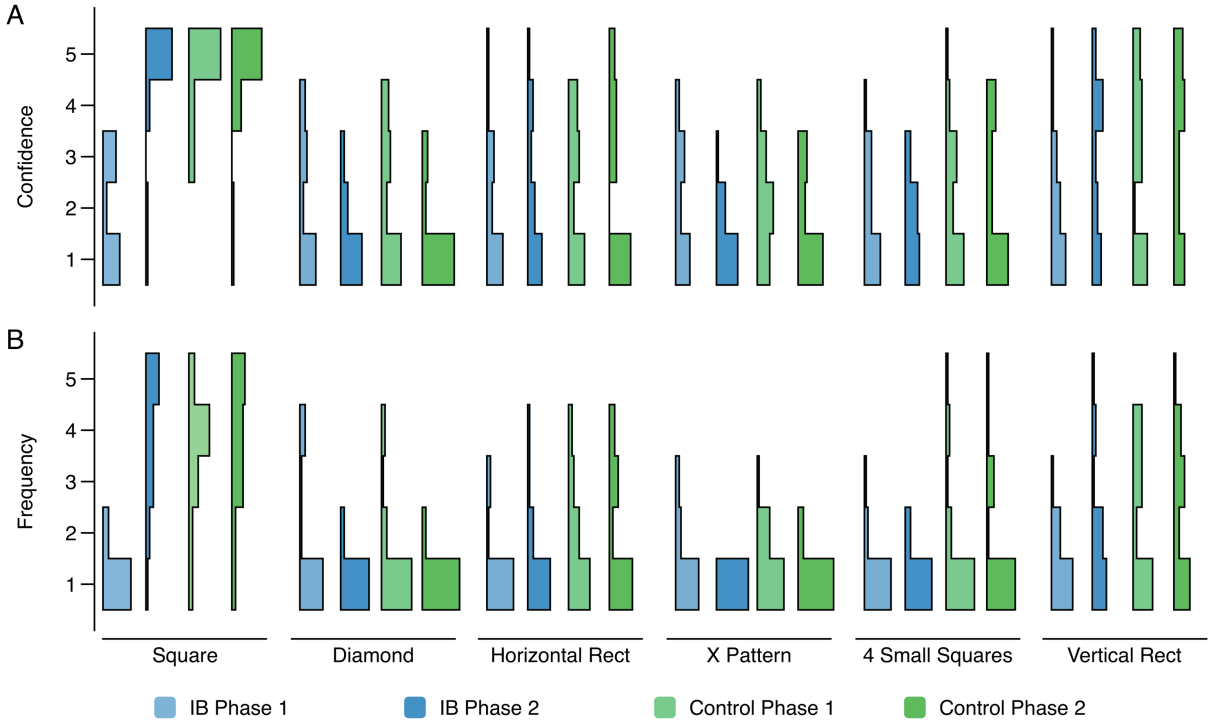

Figure 4. Histograms of awareness ratings for the peripheral probes. **A**) Confidence ratings. Confidence was assessed from 1 = 'Very confident I did not see the shape', to 5 = 'Very confident I did see the shape'. **B**) Frequency ratings. Estimation of presentation frequency for each shape was assessed from 1 = Never, to 5 = Very frequent, more than 100 times. Probe stimuli were only ever large squares, but five other shape options were given in the awareness questionnaire to permit quantification of false alarms (diamond, horizontal rectangle, X pattern, four small squares, vertical rectangle). When completing the rating scales, participants were presented with examples of each shape embedded within line arrays.

Accuracy (hits) in the central-target task was above 95% on average for both groups. A mixed ANOVA with a within-subjects factor of Phase (1,2) and a between-subjects factor of Group (IB, control) revealed no significant main effect of group, $F(1,39) = 0.10$, $p = .760$, $\eta^2 < .01$, and no significant interaction, $F(1,39) = 0.01$, $p = .913$, $\eta^2 < .01$. There was, however, a significant main effect of Phase, $F(1,39) = 21.55$, $p < .001$, $\eta^2 = .36$ indicating that both groups were significantly more accurate in Phase 1 than in Phase 2 (Table 1) likely due to boredom or fatigue. Participants responded when no target was on the screen on fewer than 1% of trials on average and responded to the incorrect target stimulus on fewer than 3% of trials on average.

Table 1. *Accuracy (%) in the Central-Target Task for the Two Experimental Groups*

	Phase 1	Phase 2
Inattentional Blindness	97.51 (1.62)	95.96 (2.97)
Control	97.73 (1.56)	96.11 (2.25)

Note: Values represent means (standard deviations)

The same ANOVA performed on the reaction time (RT) results showed a similar pattern (Table 2). Participants were significantly faster in Phase 1 than in Phase 2, $F(1,39) = 6.43$, $p = .015$, $\eta^2 = .14$, but there was no RT difference between the groups, $F(1,39) = 0.05$, $p = .831$, $\eta^2 < .01$, and no interaction, $F(1,39) = 1.10$, $p = .301$, $\eta^2 = .02$. There were no significant RT differences between responses to the target stimuli on probe trials relative to no probe trials for either group in either phase, however, the IB group did show a trend towards faster responses when the probes were present in Phase 1 (IB group, Phase 1: $t(17) = 2.06$, $p = .055$, Cohen's $d = .51$, all Cohen's d values for repeated measures t-tests have been corrected for the dependence between means using Morris & DeShon's (2002) Equation 8, allowing comparison with Cohen's d from between groups tests; IB group, Phase 2: $t(17) = 0.38$, $p = .712$, Cohen's $d = .09$; control group, Phase 1: $t(22) = 1.13$, $p = .269$, Cohen's $d = .25$; control group, Phase 2: $t(22) = 0.21$, $p = .839$,

Cohen's d = .08). Together, the accuracy and RT results suggest very little impact of awareness of the irrelevant probes on behavioral performance in the central-target task.

Table 2. *Reaction times (ms) in the Central-Target Task for the Two Experimental Groups*

	Phase 1	**Phase 2**
Inattentional Blindness	554 (52)	569 (59)
Control	557 (53)	562 (50)

Note: Values represent means (standard deviations)

Event Related Potentials

To ensure we had enough trials to produce reliable ERPs in all conditions, participants were excluded from EEG analysis if they had greater than one third of all trials rejected due to blinks or other artefacts throughout the experiment. This resulted in the exclusion of two participants from the IB group (mean number of rejected trials for remaining participants = 186 or 12.24% of trials), and three participants from the control group (mean number of rejected trials for remaining participants = 159, or 10.46% of trials).

With a similar paradigm, Pitts et al. (2012) demonstrated a negativity in the EEG when participants perceived the probe stimuli that was not present when the probe stimuli were not perceived. Thus, we would expect a significant VAN in both phases of the experiment for the control group, but only in Phase 2 for the IB group. A mixed ANOVA on VAN magnitude (the difference between probe and no-probe trials; this factor was not included in the ANOVA as this difference was used to select the times and electrodes for our ROI. Analyzing this difference would be circular), with the within-subjects factor Phase (1,2), and the between-subjects factor Group (IB, Control) did not produce the expected significant interaction ($F(1,34) = 1.35$, $p = .254$, $\eta^2 = .04$; Figure 5). However, as we had a-priori hypotheses regarding which conditions should or should not produce a significant VAN, we ran further pairwise contrasts to follow these up.

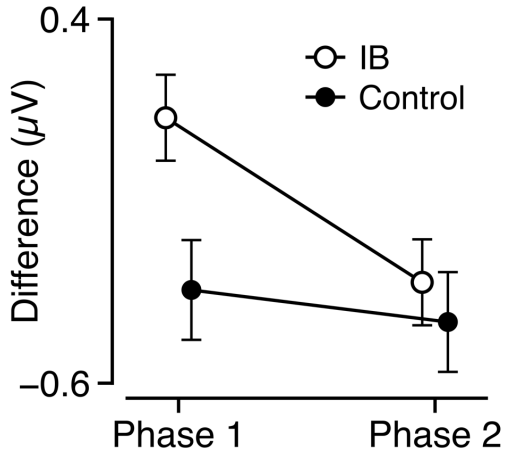

Figure 5. ERP difference between probe and no-probe trials for each condition, plotted separately for the two groups and the two phases. Error bars represent within-participants standard errors.

ERPs at contralateral electrode sites (Figure 6) produced results similar to those of Pitts et al. (2012). VAN magnitude (probe minus no probe) differed significantly between the two phases for the IB group, $t(15) = 2.31$, $p = .036$, Cohen's $d = .58$, but not for the control group, $t(19) = 0.38$, $p = .708$, Cohen's $d = .09$. Despite the times and electrodes for analysis being selected on the basis of the maximum VAN location (collapsed across groups and phases), thus biasing the outcome toward a significant VAN, the IB group (Figure 6A) showed no significant VAN in Phase 1 of the experiment, when participants were *not aware* of the probes, $t(15) = 0.75$, $p = .466$, Cohen's $d = -.19$. They did, however, show a significant VAN in Phase 2 when they were aware of the probes, $t(15) = 2.22$, $p = .043$, Cohen's $d = .56$. For completeness we also show the VAN results for the control group (Figure 6B). Participants in the control group were aware of the probes in both phases of the experiment and, as expected, showed a significant VAN in both Phase 1, $t(19) = 2.19$, $p = .041$, Cohen's $d = .49$, and Phase 2, $t(19) = 2.37$, $p = .029$, Cohen's $d = .53$ (note that as VAN magnitudes were the basis of our electrode and time selection, these results are not surprising. Of more interest to us are the differences between conditions). As expected, we also found some evidence of a difference in VAN magnitude between the IB group and the control group in Phase 1, $t(34) = 2.02$, $p = .051$, Cohen's d

= .68, but no difference between the groups in Phase 2, $t(34) = 0.45$, $p = .655$, Cohen's d = .16. Thus, in all conditions in which participants reported awareness of the probes, we observed a VAN contralateral to the location of the probe that was not present when participants did not perceive the probes.

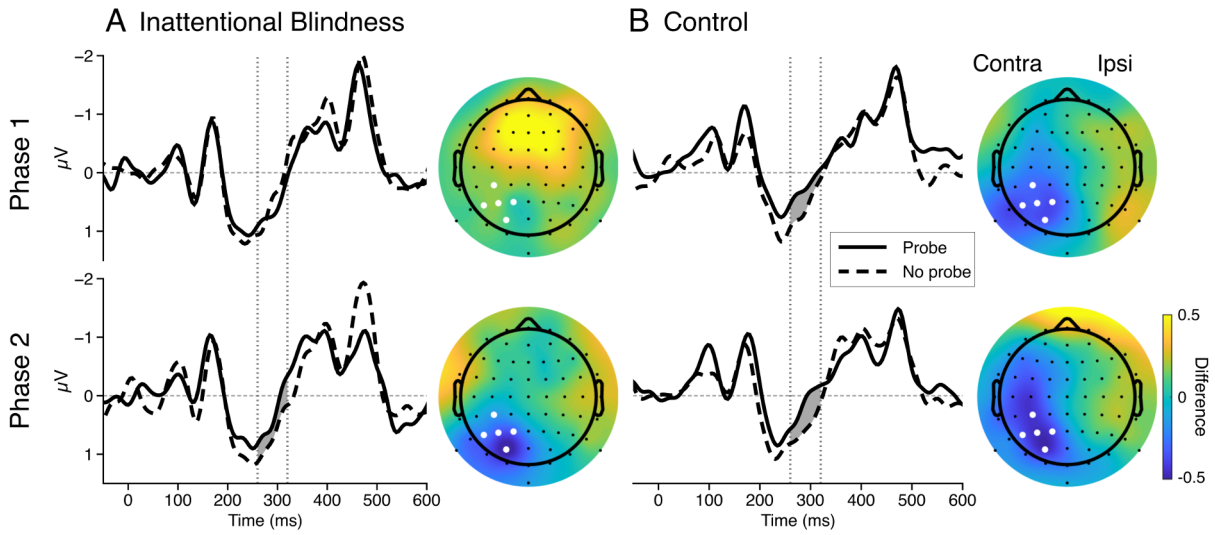

Figure 6. Average ERP waveforms recorded at ROIs contralateral to peripheral probe shapes, and contralateralized scalp topographies of the difference between peripheral probe trials and probe-absent trials, averaged across the time range of interest for the VAN (260–320ms). A) ERPs for the IB group. The top plot shows the results from Phase 1 of the experiment, with no significant VAN when the IB group did not perceive the probes. The bottom plot shows the results from Phase 2, with a significant VAN when the IB group perceived the probes. B) ERPs for the control group, showing significant VANs in both phases, consistent with this group's perception. Scalp topographies have been contralateralized such that electrodes on the left are contralateral, and electrodes on the right are ipsilateral to the peripheral probes. Contralateral ROI electrodes are presented in white. Vertical dotted lines in the ERP plots represent the bounds of the time-range of interest. Filled grey areas between ERPs for probe and no-probe conditions represent significant differences.

Consistent with a contralateral locus of the VAN when probe stimuli are lateralized, ipsilateral electrodes showed no significant difference between probe and no-probe trials in either phase of the experiment for either group (IB group, Phase 1: $t(15) = 1.13$, p = .276, Cohen's d = .28; IB group, Phase 2: $t(15) = 0.16$, $p = .873$, Cohen's d = .04; control

group, Phase 1: $t(19) = 0.57$, $p = .578$, Cohen's $d = .13$; control group, Phase 2: $t(19) = 0.40$, $p = .694$, Cohen's $d = .09$).

Time-Frequency Power

Recall our prediction that, if alpha oscillations are associated with awareness there should be an alpha power decrease (relative to no-probe trials) contralateral to the irrelevant probes when they are perceived, but not when they fail to reach awareness. Alternatively, if alpha oscillations in previous studies were reduced due to attentional allocation on the basis of task-relevance (Vanni et al., 1997; Babiloni et al., 2006) or visual salience (Bareither et al., 2014), then our irrelevant, non-salient probes should not draw attention and we should observe no decrease in alpha power contralateral to the probe in any condition. To address these hypotheses, we selected a time and frequency range of interest, computed the average alpha-power difference between probe and no-probe trials in this range, and compared these scores between the groups and phases. The time-frequency range of interest was selected by collapsing the data from both groups and both phases and performing a cluster-based permutation test (Groppe et al., 2011) on the difference between probe and no-probe trials, with a threshold of $p = .001$ for inclusion in the cluster (Figure 7A). We selected the resulting time-frequency region of significant power difference as our time-frequency range of interest (note: this selection method is not circular, as we are comparing these difference scores between groups and phases that were collapsed together in the range-of-interest selection). This analysis revealed significant amplitude differences from 7–16 Hz between 220ms and 800ms. For our time-frequency range of interest we selected the frequencies from 8–14 Hz, and times between 304ms and 734ms, as this was the largest time-frequency range in which all times and all frequencies were included in the significant cluster (see the red outline in Figure 7A).

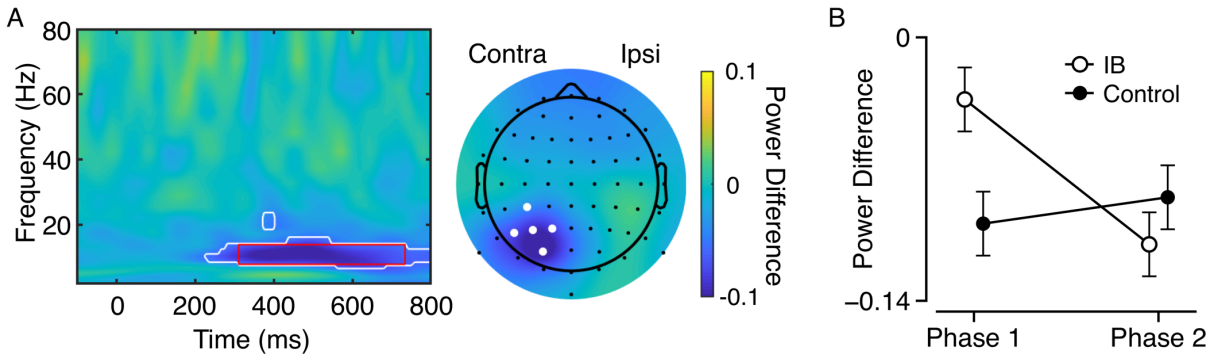

Figure 7. Alpha power difference between probe and no-probe trials. A) Grand average power difference between probe and no-probe trials, collapsed across groups and phases. White outlines represent times and frequencies of significant difference, as assessed by cluster permutation analysis. The red outline indicates the time-frequency window selected for analysis, from 8-14 Hz, between 304-734ms. The scalp topography represents the power difference at all electrodes averaged across this time-frequency range. **B**) Mean power differences between probe and no-probe trials plotted separately for the two groups and the two phases. Error bars represent within-participants standard errors.

We computed the average alpha power difference between probe and no-probe trials across our time-frequency range of interest and compared these scores with a mixed ANOVA that had a within-subjects factor of Phase (Phase 1, Phase 2) and a between-subjects factor of Group (IB, Control). This analysis revealed a significant interaction between Phase and Group, $F(1,34) = 7.47$, $p = .010$, $\eta^2 = .17$ (Figure 7B). Following this up with independent-samples *t*-tests revealed that alpha power on probe relative to no-probe trials was significantly lower in the control group than in the IB group in Phase 1, $t(34) = 2.12$, $p = .041$, Cohen's $d = .71$, whereas there was no significant difference between the groups in Phase 2, $t(34) = -.73$, $p = .473$, Cohen's $d = .24$.

To gain a more fine-grained picture of awareness-related alpha power change we analysed oscillatory power across time and frequency, comparing between probe and no-probe trials at both contralateral and ipsilateral electrode clusters using cluster-based permutation tests (Groppe et al., 2011). In Phase 1, the IB group produced no significant differences between probe and no-probe trials at any time or frequency (Figure 8A). In Phase 2, however, the IB group produced a single significant cluster of reduced power,

cluster $p < .001$, from 164-800ms, in the alpha frequency range between 7-17 Hz. This activity spread up to 37 Hz in the period from 336-523ms post probe onset.

The results of the control group also support the conclusion that reduced alpha power is associated with perception of the irrelevant probes (Figure 8B). In Phase 1, the control group produced a single significant cluster of reduced power, cluster $p < .001$, from 172-800ms, mostly focused across the alpha frequency range, from 7-17 Hz, but with a brief period of power reduction spreading up to 23 Hz from 367-422ms, and spreading down to 4 Hz after 570ms. In Phase 2, the control group produced a single significant cluster of reduced power, cluster $p = .017$, from 250-800ms, spanning 6-20 Hz, but mostly focused in the alpha range between 8-13.5 Hz.

The scalp distribution of alpha oscillations (Figure 8C) reveals a similar contralateral topography to that of the VAN (Figure 6), consistent with alpha power being reduced in response to the perceived stimulus. The combined results of our time-frequency analysis suggest that alpha power is reduced in response to irrelevant, non-salient probes when they are perceived (IB group, Phase 2; control group, Phases 1 and 2), but not when they go unperceived (IB group, Phase 1), consistent with a link between alpha oscillations and awareness.

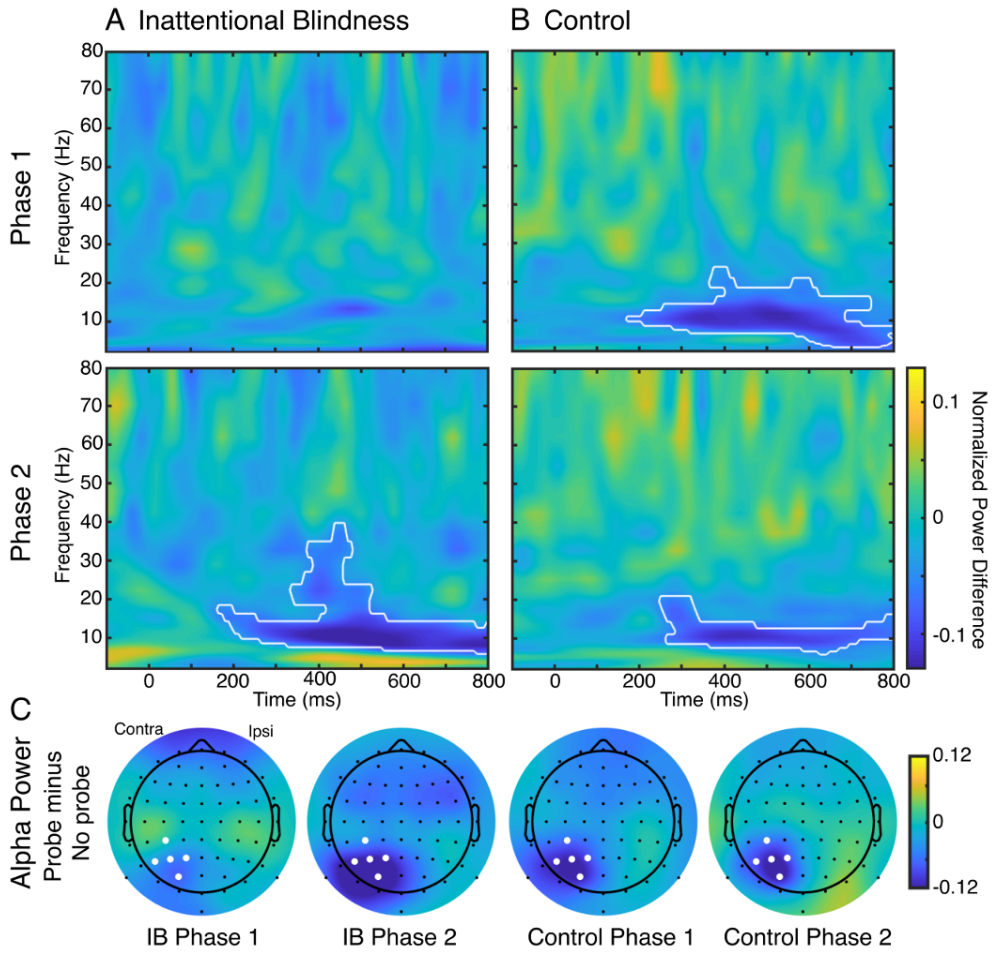

Figure 8. Awareness related power change for peripheral probes in the inattentional blindness task. Time-frequency plots of the normalized power difference between Probe and No-probe trials for the inattentional blindness group (A) and the control group (B). Within A and B, the top panels show data from Phase 1 of the experiment, and the bottom panels show data from Phase 2. White lines indicate regions of significant differences, with the family-wise error rate controlled using cluster-based permutation tests (Groppe et al., 2011). C) Contralateralized scalp topographies of alpha power (8-14 Hz) averaged across the period from 304–734ms. Contralateral electrodes are presented on the left, and ipsilateral electrodes are presented on the right. Contralateral ROI electrodes are shown in white.

The same time-frequency power analysis performed at electrode sites ipsilateral to the probes produced a significant cluster of reduced 2-4 Hz amplitude from 0-531ms, in Phase 1 for the IB group, p = .008. There were no significant ipsilateral amplitude

differences for the IB group in Phase 2 of the experiment, or in either phase for the control group.

Discussion

We set out to test whether alpha oscillations are a neural correlate of visual awareness by examining alpha power change in an inattentional blindness paradigm. We employed non-salient probe stimuli in a no-report paradigm (Tsuchiya et al., 2015), so that any alpha responses corresponding to awareness could not be attributed to task-relevance, or to attentional capture by the probe stimuli. If alpha oscillations are a correlate of sensory awareness itself, we would expect a reduction in alpha power contralateral to any perceived stimulus but not contralateral to stimuli that do not reach awareness. Alternatively, if alpha oscillations are not a correlate of awareness, and past studies have shown awareness-related alpha responses due to confounding awareness with task relevance (Vanni et al., 1997; Babiloni et al., 2006; Palva et al., 2005) or salience (Bareither et al., 2014), then we may expect stimuli to be perceived without any concomitant alpha power reduction when these factors are controlled. We first review our behavioral and ERP findings before turning to a discussion of the pattern of alpha power change related to awareness in the current study.

As intended, the inattentionally blind group reported being unaware of the peripheral probes in Phase 1 of the experiment but were aware of the probes in Phase 2 (after being cued to their presence by the questionnaire at the end of Phase 1). The control group, who were cued to the presence of the probes at the start of the experiment, were aware of the probes in both phases. Consistent with past results (Koivisto & Revonsuo, 2010; Pitts et al., 2012), we observed a contralateral negativity in the ERP response – the VAN – in response to perceived peripheral probes, which was absent when the probes were not perceived. The observed timing of the VAN was somewhat later than that typically observed when the to-be-detected stimulus is goal relevant (Koivisto & Revonsuo, 2010; Phase 3 of Pitts et al, 2012; Railo et al., 2015), but roughly matched the timing previously observed when shape probes were irrelevant in a similar paradigm (Phases 1 and 2 of Pitts, et al., 2012). The contralateral topography of the VAN we observed is

consistent with previous reports suggesting a contralateral temporal-occipital locus of the VAN following lateralized stimuli (Koivisto & Revonsuo, 2010). Although the timing of the VAN in our paradigm was consistent with the results of Pitts et al. (2012), the shape of the waveform on which the VAN appeared was not. Pitts et al. (2012) observed the VAN as an increase of a negative peak in the ERP, whereas we observed it as a negative deflection on the tail-end of a positive peak. These discrepancies are likely due to differences in the stimuli and tasks used by us and by Pitts et al. (2012), which in turn would be expected to yield differences in the overall waveform on which the VAN is superimposed. This is likely why the VAN is sometimes observed as an increased negative peak (e.g., Pitts et al., 2012; Shafto & Pitts, 2015), and at other times is observed as a decreased positive deflection (e.g., Koivisto et al., 2008; Koivisto & Revonsuo, 2010; Pitts et al., 2014a).

Analysis of oscillatory power revealed no differences between probe and no-probe trials when participants were unaware of the probes. When the probes were perceived, however, they elicited an amplitude decrease in alpha/beta oscillations contralateral to the probe. Thus, alpha power reduction is produced in the same conditions in which the VAN is observed (e.g., Pitts et al., 2012). These results are consistent with previous demonstrations of alpha-power reduction associated with the perception of visual stimuli (Vanni et al.,1997; Babiloni et al., 2006; Bareither et al., 2014). By using a no-report paradigm and identical stimuli for the aware and unaware conditions we were able to avoid the confounds present in previous studies and show that alpha power reduction is associated with visual awareness. This is demonstrated particularly strongly in the control group in Phase 1, where we observed a probe-related alpha power reduction despite participants having *no knowledge* at that time that they would ever need to report the probes, making any argument that participants might have attended the probes, and produced an attention- rather than awareness-related alpha reduction, unlikely. Furthermore, by using non-salient peripheral probes that were stimulus matched across the aware and unaware conditions, we can be confident that the post-stimulus alpha power reduction we observed was related to awareness of the probes, and was not due to involuntary salience-based attentional capture that could potentially be independent of

722 awareness. Our results provide a strong link between alpha amplitude reduction and
723 processes intrinsic to awareness.
724 Much of the literature relating alpha oscillations to perception has examined the
725 impact of alpha oscillations *prior* to stimulus presentation on the likelihood of perceiving an
726 upcoming stimulus. These studies have consistently shown that lower alpha amplitude
727 prior to stimulus onset predicts increased likelihood of the stimulus being perceived (e.g.,
728 Ergenoglu et al., 2004; Babiloni et al., 2006; Hanslmayr et al., 2007; van Dijk et al., 2008;
729 Romei et al., 2010; MacLean & Arnell, 2011; Limbach & Corballis, 2016; Iemi et al., 2017).
730 Lower alpha power also increases the likelihood of perceiving a stimulus when no stimulus
731 is presented, both in terms of false positives (Limbach & Corballis, 2016; Iemi et al., 2017),
732 and visual illusions (Lange et al., 2013; Cecere et al., 2015; Gulbinaite et al., 2017),
733 consistent with alpha's role in spatial gain modulation. These studies and others have
734 supported the conclusion that alpha oscillations are a key mechanism underlying the
735 effects of spatial attention (Jensen & Mazaheri, 2010). Here, we attempted to control the
736 effects of attention by ensuring the peripheral probes were not goal-relevant and had low
737 bottom-up salience. Still, one could argue that once participants were cued to the
738 presence of the probes the probes became attended, and this attention is what led to the
739 alpha reduction we observed. This is a possibility. It is difficult to see why participants
740 would voluntarily attend the probes in the absence of a reason or incentive to do so,
741 particularly as they would have to attend the probes many times for the associated alpha
742 response to appear in the condition average. It may be the case that when stimuli are
743 perceived they automatically attract some degree of attention (Flevaris et al., 2013). If the
744 alpha power reduction associated with attention (Thut et al., 2006) and that associated
745 with awareness are determined to be produced by the same source and mechanism, and
746 if no cases of awareness without alpha power change are found, this would be strong
747 evidence supporting claims that awareness cannot be dissociated from attention (Cohen
748 et al., 2012; de Brigard & Prinz, 2010; O'Regan & Noë, 2001; Posner, 1994).
749 Interestingly, we observed no decrement in performance on the central task when
750 participants perceived probes in the periphery. This may suggest that the probes were not
751 attended, and therefore that attention-related alpha (typically pre-stimulus) and awareness-

related alpha (typically observed post-stimulus) are different. This is a logical possibility, but we hesitate to draw strong conclusions on this point for at least two reasons. First, performance on the central task was close to ceiling, so the task may not have been sensitive enough to show attention related behavioural effects. Second, no-report paradigms make it impossible to determine whether any individual peripheral probe event was perceived. Thus, it may be that when attention was captured to the red stimuli in the central task, the peripheral probes present on those trials were not perceived, and so did not interfere with behaviour. It may be that the probes were only perceived on trials with no central target. There is no way to rule out this possibility with the present data.

As noted earlier, Bareither et al. (2014) also examined alpha responses in a no-report paradigm, in which peripheral stimuli were either supraliminal (presented at 500% of detection threshold) or subliminal (presented at 25% of detection threshold). In addition to a contralateral alpha-power decrease following the presentation of supraliminal stimuli, Bareither et al. (2014) observed an *increase* in alpha power following the presentation of subliminal stimuli. This result was not apparent in our data; we observed no change in alpha power following probes that did not reach awareness (IB group, Phase 1). This difference between experiments may be stimulus-related. For their subliminal condition, Bareither et al. (2014) employed small stimuli presented well below detection threshold. We, by comparison, employed high-contrast stimuli (white line segments on a black background) on both probe-present and probe-absent trials. Our probe-present trials were differentiated from probe-absent trials only by their configural properties. Given alpha's inhibitory role in modulating activity levels in visual cortex (Jensen & Mazaheri, 2010; Lange et al., 2013; Iemi et al., 2017), it may be that sub-threshold stimuli like those employed by Bareither et al. (2014) elicit increased alpha oscillations because they are interpreted by the visual system as 'noise' to be suppressed (Bareither et al., 2014). In contrast, when our configural shape probes were not perceived, there was no perceived absence of input to be maintained through suppression of noise. Participants in our paradigm (and those of Pitts et al., 2012; Pitts et al., 2014a,b; Shafto & Pitts; 2015) were unaware of the higher-level configural properties of the probe stimuli, rather than being unaware of the presence of the stimuli themselves.

In summary, perception of a configural stimulus was accompanied by a reduction in alpha amplitude that was not present when the same stimulus went unperceived. This was true despite the stimulus being task-irrelevant and non-salient, and in one condition, despite participants' lack of knowledge that the stimulus would need to be reported. Thus, the current evidence suggests that alpha power reduction constitutes a true neural correlate of consciousness (Crick & Koch, 1990), rather than a consequence of attention-related confounds.

Conflict of Interest: The authors declare no competing financial interests

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Data Accessibility: The ethical clearance held for this project restricts data access to members of the research team only. In the event that an individual outside of the research team might wish to obtain access to the data, we will seek to amend the ethical clearance by adding the name of that individual to the list of research team members.

Author Contributions: AMH, PED, and JBM designed the study. AMH collected and analysed the data. AMH, PED, and JBM drafted the paper.

Abbreviations

ANOVA	Analysis of variance
BOLD	Blood-oxygen-level dependant
EEG	Electroencephalography
EOG	Electro-oculographic
ERP	Event-related potential
FIR	Finite impulse response
IB	Inattentional blindness
ROI	Region of interest
RT	Reaction time
VAN	Visual awareness negativity

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