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Effects of neuronic shutter observed in the EEG alpha rhythm

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Abstract

The posterior alpha rhythm, seen in human electroencephalogram (EEG), is posited to originate from cycling inhibitory/excitatory states of visual relay cells in the thalamus. These cycling states are thought to lead to oscillating visual sensitivity levels termed the "neuronic shutter effect." If true, perceptual performance should be predictable by observed alpha phase (of cycling inhibitory/excitatory states) relative to the timeline of afferentiation onto the visual cortex. Here, we tested this hypothesis by presenting contrast changes at near perceptual threshold intensity through closed eyelids to 20 participants (balanced for gender) during times of spontaneous alpha oscillations. To more accurately and rigorously test the shutter hypothesis than ever before, alpha rhythm phase and amplitude were calculated relative to each individual's retina-to-V1 conduction delay, estimated from the individual's C1 visual-evoked potential latency. Our results show that stimulus observation rates are greater at a trough than a peak of the posterior alpha rhythm when phase is measured at the individual's conduction delay relative to stimulus onset. Specifically, the optimal phase for stimulus observation was found to be 272.41°, where observation rates are 20.96% greater than the opposing phase of 92.41°. The perception-phase relationship is modulated by alpha rhythm amplitude and is not observed at lower amplitude oscillations. Collectively, these results provide support to the "neuronic shutter" hypothesis and demonstrate a phase and timing relationship consistent with the theory that cycling excitability in the thalamic relay cells underly posterior alpha oscillations.

Significance Statement

After accounting for neural conduction delays, we found that threshold intensity stimuli are observed at higher rates when the alpha wave is at a trough phase than at a peak phase, but only when alpha amplitude is high. Our results were derived using methods consistent with a specific hypothesis about a mechanism of visual perception, considering the structure, physiology, and transmission delays in the visual system. The results of this rigorous study design add support to the neuronic shutter hypothesis and are consistent with the theory that posterior alpha reflects cycling excitability in thalamic relay cells, thereby gating the flow of visual information.

Introduction

The posterior alpha rhythm, a 7-13Hz oscillation, is a notable characteristic of the human EEG over the occipital cortex, especially when eyes are closed (Berry and Wagner, 2015). This is thought to assist attention regulation, as its characteristics are often found to correlate with attention over specific sensory cortices (Sauseng et al., 2005; Ben-Simon et al., 2013) – possibly a gating mechanism to limit the flow of sensory information (Cooper et al., 2003; Janssens et al., 2018). This rhythm has analogs in the sensorimotor (Pineda, 2005; Bazanova and Vernon, 2014) and auditory cortices (Tiihonen et al., 1991; Weisz et al., 2011), with both appearing to provide similar gating. This regulatory function is further demonstrated by the observation that alpha activity increases during introspective behavior (Ancoli and Green, 1977) and behaviors involving internally directed attention such as imagination (Cooper et al., 2003), mental arithmetic (Ray and Cole, 1985), autobiographical recall (Yue et al., 2013), and meditative practices (Tenke et al., 2017) presumably because external sensory information is distractive during these behaviors.

However, the cellular mechanism and functional significance of alpha oscillations remains unclear. In the 1950's, alpha oscillations were proposed to represent a 'neuronic shutter' (Callaway and Alexander, 1960) to down-sample incoming sensory information to reduce processing load, and similar discrete perceptual processes are proposed today (VanRullen and Koch, 2003; VanRullen et al., 2014). This shutter is thought to occur at the lateral geniculate nucleus (LGN), which relays visual information to V1. At times, LGN relay cells burst-fire at 10Hz, with a hyperpolarized refractory period between bursts (Lopes da Silva, 1991; Sherman, 2001; Timofeev and Bazhenov, 2005; Alexander et al., 2006; Timofeev and Chauvette, 2011). If many of these cells fire synchronously, visual afferentation during a widespread refractory period is less likely to be relayed to V1, resulting in a 10Hz visual shutter. Each burst in LGN relay cells results in a large excitatory postsynaptic potential (EPSP) at V1, measured as a negativity in the occipital EEG (Timofeev and Bazhenov, 2005), with higher amplitudes indicating more synchronous excitation (Pfurtscheller et al., 1996) resulting, as hypothesized by this model, from LGN excitatory

volleys. Thus, the alpha rhythm is thought to reflect cycling excitability in the LGN visual-relay cells, which would result in cycling visual sensitivity in phase with the alpha oscillation. Evidence of this mechanism producing alpha oscillations has been found in animal models with the associated phasic gating of sensory transfer (Lorincz et al., 2009; Chen et al., 2016). The hypothesis of the present study was based on this model; however, we did not directly test the underlying cellular mechanism.

To more accurately measure the alpha phase relationship to perceptual performance, we accounted for neural conduction delays: If a stimulus strikes the retina at *t0*, that information arrives at the LGN at a later time, *t1*. Therefore, the *t1* LGN excitatory state determines whether the information is relayed to V1. Further, the LGN excitatory state is measurable even later in the EEG, as V1 EPSPs at *t2*. Thus, *t0* to *t2* represents the retina-to-V1 conduction delay: At *t2*, we can *assess* the *t1* LGN excitability, the time of afferentation of the *t0* stimulus. This is the first study, to our knowledge, to directly examine the relationship between visual observation and spontaneous alpha phase relative to each individual's conduction delay, which advances the accuracy and rigor for testing the shutter hypothesis.

This study's objective is to more robustly test the hypothesis that posterior alpha reflects cyclic excitability of the visual system with a phase and timing relationship predicted by underlying physiological model and conduction delays. Therefore, we presented visual stimuli (*t0*) to participants during spontaneous occipital alpha activity, then measured alpha amplitude and phase at *t2* (estimated by the individual's C1 visual-evoked potential component peak latency) (Di Russo et al., 2001). We predicted that visual stimuli at *t0* would be more likely observed with alpha phase at a trough at *t2* (assumed to indicate *t1* LGN excitation) than at a peak (*t1* LGN inhibition). Additionally, this effect was predicted to be greater at high alpha amplitudes, indicating greater cellular excitatory state synchrony. We found that participants responded to 20.96% more stimuli with alpha phase at a trough vs. a peak at *t2* when alpha amplitude was high. At low amplitude, no significant phase effect on observation rate was observed. These results support our hypothesis that the alpha rhythm reflects cyclic excitatory states in the visual system resulting in a visual shutter effect.

Materials & Methods

Participants: Alpha activity varies largely both within (Gonçalves et al., 2006) and between (Wieneke et al., 1980) individuals. For this study, we needed to ensure stimuli were able to be presented during times of observable (e.g., stationary) alpha oscillations and therefore selected participants who more readily and reliably generated observable occipital alpha activity. This selection was made by observing EEG activity as the participants practiced the visual sensitivity task with eyes closed during an earlier portion of the experimental session. Participants who did not readily and reliably produce occipital alpha oscillations during this practice period were excluded from further participation.

In total, 41 participants were recruited for this study. Based on the observable alpha criteria described above, 21 participants were excluded from further participation. The remaining 20 healthy participants (10 males/10 females, mean age: 22.3 years, range: 18-28 years) completed two tasks: one to estimate their individual conduction delay; the other to investigate their visual sensitivity at different phases of alpha oscillations. The experimental protocol was approved by the Institutional Review Boards of Wright State University and the Air Force Research Laboratory. All participants were compensated for their time.

Recording: All recordings were made using the BioSemi ActiveTwo system (BioSemi B.V., Amsterdam, The Netherlands). Recordings were made with a 2048 Hz sampling rate at 64 channel locations based on the modified combinatorial nomenclature extension of the 10-10 system (American Electroencephalographic Society, 1994) excluding the inferior chain with the exception of P9/P10 and Iz (Seeck et al., 2017), with bilateral electrodes on the mastoid process, infraorbital, and outer canthus locations. Participant responses were recorded using a low-latency mechanical keyboard (Cherry MX 6.0 [G80-3930], Cherry GmbH, Auerbach in der Oberpfalz, Germany). This was combined with other task-

state and visual-stimulus timing information via light sensors placed on the monitor to record events on the ActiveTwo's 16-bit trigger line (StimTracker 1G, Cedrus Corporation, San Pedro, California, USA).

Stimuli:All tasks and stimuli were constructed and presented using MATLAB (R2011b; The MathWorks, Inc., Natick, MA, USA) and the Psychophysics Toolbox (v3.0.13) (Brainard, 1997; Pelli, 1997; Kleiner et al., 2007). This software was run on a Dell Precision T3610 computer (Dell Inc., Round Tock, TX, USA) with the Windows 7 Professional operating system (Microsoft Corporation, Redmond, WA, USA). Stimuli were presented to participants on a 24.5", 240 Hz monitor (BenQ ZOWIE XL2540, BenQ Corporation, Taipei, Taiwan) providing 4.2 ms temporal resolution for stimulus presentation. Where relevant, stimulus luminance was measured using a light meter (Light Meter LUX/FC 840020, SPER SCIENTIFIC, Scottsdale, AZ, USA). The participant's head position was fixed using a chinrest placed 58 cm from this monitor. The experiment was conducted in a dark room with a natural sound machine (Dohm Classic, Marpac LLC, Wilmington, NC, USA) to mask noise disturbances.

Data Analysis:All data analyses were performed in MATLAB (R2019b; The MathWorks, Inc., Natick, MA, USA) utilizing the EEGLAB Toolbox (v2019.0) (Delorme and Makeig, 2004) and the ERPLAB plugin (v7.0.0) (Lopez-Calderon and Luck, 2014). Statistical analyses were conducted using MATLAB's Statistics and Machine Learning Toolbox (R2019b; The MathWorks, Inc., Natick, MA, USA). This software was run on a Lenovo ThinkPad P50 computer (Lenovo, Quarry Bay, Hong Kong) with the Windows 10 Enterprise operating system (Microsoft Corporation, Redmond, WA, USA).

Code Accessibility: Task design and data analysis code are made publicly available at https://osf.io/qnryf/ DOI: 10.17605/OSF.IO/QNRYF in addition to the recorded EEG data.

Estimation of t2

The peak latency of each participant's C1 visual-evoked potential (VEP) component was used as an estimate of their individual retina-to-V1 conduction delay, *t2*. This component reflects the arrival of a volley of visual information to V1 from the LGN along the optic radiations. This is analogous to the alpha wave, in the sense that it also reflects excitatory burst volleys from the LGN to V1, according to the hypothesized model. The C1 peak, rather than onset, latency was used to estimate the middle point of these volleys rather than their onset. Due to the specific folding of the V1 cortical area around the calcarine fissure (Figure 1A), the C1 wave shape will vary with the location of the stimulus in the visual field. Stimuli presented horizontally centered in the visual field will stimulate spatially opposing V1 neurons whose dipoles will cancel each other out, yielding no measurable C1 component in the EEG (Clark et al., 1995; Di Russo et al., 2001). This is similarly the case for stimuli presented approximately 3° below the vertical center (Clark et al., 1995; Di Russo et al., 2001). However, with some horizontal spacing, bilateral stimuli presented above -3° in the visual field will produce a negative C1 wave, while those presented below -3° in the visual field will produce a positive C1 wave as shown in Figure 1B (Clark et al., 1995; Di Russo et al., 2001).

C1 waves are found to be measured maximally at electrode POz using an averaged mastoid reference, but they are still quite small in amplitude. To obtain a well-defined C1 VEP component, both negative and positive components were obtained using upper and lower visual field stimuli. Next, the difference between these two signals was taken to create a large-amplitude wave with a clear peak (Figure 1C shows these waveforms from a single participant). This peak latency was taken as an estimate of *t*2.

Experimental Design:The stimulus used for this task was a 17° wide, 6.38° tall black and white checkerboard made of 16x6 1.06° squares placed on an otherwise black screen with a fixation point placed in the center (Figure 1B). The checkerboard was flashed with its center either 5° above or 7.5°

below the fixation point and centered horizontally. The size and placement of these stimuli were selected to generate maximum amplitude waves (Clark et al., 1995). A total of 600 upper and 600 lower stimuli were presented in a mixed random order each with a duration of 33 ms with random 250-450 ms interstimulus-intervals, resulting in a trial time of approximately 7.5 minutes.

Similarly to Di Russo et al. (2001), participant attention and gaze were maintained on the fixation point by a sham task performed during the trial. The fixation point would occasionally flash to a brighter color for 12.6 ms, and the participant was instructed to watch for this flash and to respond by pressing the space bar.

Analysis:For the C1 VEP analysis, the originally recorded 2048 Hz sampling rate was maintained but re-referenced to averaged mastoids. All EEG signals were band-pass filtered from 0.01 to 50 Hz at -6 dB, using a 2nd order IIR Butterworth filter as implemented in the ERPLAB toolbox (Lopez-Calderon and Luck, 2014), to achieve a zero-phase filter response. A bipolar vertical electrooculogram (vEOG) signal was created by subtracting the averaged left and right infraorbital electrodes from the averaged Fp1 and Fp2 electrodes.

The data were epoched from 50 ms prior to 200 ms after stimulus presentation for both the upper and lower visual stimuli. Blinks within these epochs were detected by sliding a 150 ms window at 75 ms steps over the vEOG signal, and any window containing a peak-to-peak amplitude of 100 μV or greater was rejected from analysis. Separate average VEPs were calculated for the upper and lower stimulus trials and were baseline-corrected to the 50 ms pre-stimulus period. The lower visual field VEP was then subtracted from the upper field VEP to create a difference wave containing a C1 wave with a clear peak.

The C1 peak latency was measured at electrode POz by finding the most negative peak within the 0-110 ms time window relative to stimulus onset. This peak latency was calculated for each participant individually and was used as an estimate of their individual *t2*.

Measuring Observation Rates

This task was designed to be conducted with closed eyes in order to evoke more frequent (Legewie et al., 1969) and higher amplitude alpha oscillations (Barry et al., 2007). For this purpose, stimuli were designed such that they could be observed as light flashes through closed eyelids. The brightness of these flashes was designed to be near threshold intensity (where participants reported seeing approximately 50% of the stimuli) to prevent any ceiling or floor effects on performance.

Experimental Design:The stimulus used for this task was an 8.5° square centered on an otherwise black (0.7 lux) screen. The stimulus brightness was defined by coloring the square as a greyscale value from 0 (completely black; 0.7 lux) to 255 (completely white; 89.2 lux). The stimulus was designed to be observable through the closed eyes of the participant. The brightness of the stimulus was adjusted to near the threshold intensity for each individual participant, the value at which the participant reported observing the stimulus half of the number of times it was presented. This threshold was estimated using the staircase method as described in Cornsweet (1962) during a calibration task prior to the main task. In this task, the participant kept their eyes closed as a series of flashes were presented, each time the participant reported observing a flash (using the spacebar) the stimulus intensity was decreased in the next trial. However, if the participant did not report observing the stimulus, the intensity was increased on the next trial. Following the methods described in Cornsweet (1962), threshold intensity is defined as the average of all trials following the third reversal in trial-intensity slope. An example of this task from one

224 participant is shown in Figure 2. In this figure, the third reversal occurred at trial 5, and threshold
225 intensity was calculated as the average of trials 6 to 40.

226 The stimuli used were so dim that the participants' ability to perceive them was sensitive to how well
227 their eyes had adapted to the dark. Pilot testing of the task revealed that, without an adjustment period, the
228 threshold brightness found during the calibration task would become easier to see over the course of the
229 main task. Although visual adaptation to the dark will continue over many hours, the most rapid changes
230 occur in the first 15 minutes, and visual acuity begins to plateau in 20 minutes (Bierings et al., 2018).
231 Therefore, participants were given 25 minutes in the dark as an adaptation period before conducting the
232 calibration task to estimate the threshold brightness.

233 In the main task, the stimuli were flashed for a period of 8.4 ms and the participant was instructed to
234 respond using the keyboard any time they observed a flash. The participant had to respond with the space
235 bar within two seconds of the stimulus to be a valid response. The experimenter triggered the stimuli
236 during the times of occipital alpha oscillation as observed in the real-time data throughout the task. After
237 the experimenter triggered the stimulus, it was presented after a random interval of less than 750 ms (to
238 mitigate potential confounding effects experimenter bias in stimulus presentation timing).

239

240 ***Analysis:***All data were down-sampled to 512 Hz and re-referenced to averaged mastoids. The signal was
241 bandpass filtered from 0.01 to 50 Hz at -6dB using a 2nd order IIR Butterworth filter, as implemented in
242 the ERPLAB toolbox (Lopez-Calderon and Luck, 2014), to achieve a zero-phase filter response. A
243 bipolar vEOG signal was created by subtracting the averaged left and right infraorbital electrodes from
244 the averaged Fp1 and Fp2 electrodes.

245 To isolate the activities of the left and right visual cortices, a left occipital (LO) signal was calculated by
246 averaging the channels O1, PO3, and PO7, and a right occipital (RO) signal was calculated by averaging

channels O2, PO4, and PO8. LO and RO were then convolved with a 10 Hz complex Morlet wavelet to obtain time-domain power and phase signals (Cohen, 2014). A peak-to-peak amplitude signal was then calculated by doubling the square root of the power signal.

The Morlet wavelet was constructed with a center frequency of 10 Hz containing $2^2/_3$ cycles (the recommended minimum (Cohen, 2019)). This cycle count was chosen to optimize temporal resolution and resulted in a wavelet with a full-width at half maximum (FWHM) of 101.6 ms in the time domain and 8.87 Hz in the frequency domain. A FWHM of 8.87 Hz indicates that the wavelet convolution was effectively a bandpass filter with half-power points of 5.57 and 14.44 Hz, approximating the alpha frequency band.

The following analysis was then performed independently for each participant. Amplitude and phase were measured from LO and RO at the individuals' *t2* relative to stimulus onset for both observed and missed trials. Trials in which LO and RO phases differed by more than 90° were rejected from further analysis to control for alpha asynchrony between the hemispheres. For each of the remaining trials, a single amplitude and phase value was obtained by averaging the amplitude and phase of the LO and RO signals. Phase values were averaged using appropriate statistics for circular quantities (Berens, 2009). These trials were then divided by median amplitude into high and low amplitude bins and then further divided by phase using 90° bins centered on 0°, 90°, 180°, and 270° phase angles. The amplitude median was chosen to provide equal sample sizes in each amplitude bin, and since it is appropriate regardless of distribution normality. When the individual amplitude distributions of our 20 subjects were examined, they appeared to be unimodal and approximately normal with no point of division appearing to be more advantageous than the median (see Figure 3). Observation rates (OR, the percentage of trials in which the participant observed the stimulus) were then calculated for every bin, and the overall OR across all bins (i.e., the average OR over all trials) was subtracted from these values to obtain a ΔOR value for each of the 2 (amplitude) x 4 (phase) bins. Note that ΔOR is not a measure of percent change, but simply a difference of the overall OR and the condition specific OR.

Statistical Analysis:This analysis resulted in a within-subjects 2x4 repeated measures design with twenty participants and two factors: amplitude (high, low) and phase (0°, 90°, 180°, 270°). These data were analyzed using a repeated measures ANOVA, testing both main effect of amplitude and phase as well as their interaction. Independent t-test and Tukey's HSD tests were used to probe any effects found to be significant. All statistical analyses were conducted using MATLAB's Statistics and Machine Learning Toolbox (R2019b; The MathWorks, Inc., Natick, MA, USA).

Results

Individual retina-to-V1 conduction delay (t2) measurement

In the first part of the experiment, the goal was to estimate each participant's retina-to-V1 conduction delay, *t2*. To calculate that, we measured in each participant the averaged VEPs in response to upper and lower visual stimuli and calculated the "difference waveform" (based on the difference between two signals); the peak latency of this difference waveform was taken as the participant's *t2* (Figure 4). The mean *t2* for all participants was 75.56 ms (range = [62.99, 88.87] ms, 95% confidence interval (CI) = [72.44, 78.69] ms). These data indicate that it takes, on average, 75.56 ms for visual information to be transmitted from the retina to area V1. For each participant, the phase and amplitude measurements were made at the individual's *t2* relative to the stimulus onset to assess visual sensitivity.

Observation rate measurement

The second part of the experiment aimed to investigate if perceptual performance, when measured at *t2*, is higher at an alpha wave trough (i.e., phase angle of 270°) than an alpha wave peak (i.e., phase angle of 90°). To examine this, we presented light flashes to each participant through closed eyes. Light flashes were near individual threshold intensity estimated using the staircase method (Cornsweet, 1962) during a calibration task prior to the main task to prevent ceiling and floor performance effects. This resulted in the stimuli of average luminance across participants of 6.48 lux. Use of these near-threshold stimuli resulting in total observation rates averaging 58.28% across participants.

On average, participants were presented 161.65 stimuli during the main task (range = [120, 200]) with an average interstimulus interval of 12.52 seconds. Left occipital (LO) and right occipital (RO) amplitude and phase signals were measured for each participant and analyzed at the participant's *t2* time point. Trials in which LO and RO phases differed by more than 90° were rejected from further analysis resulting in 135.75 trials remaining per participant on average (range = [85, 183]) . The amplitude measures were split by their median value into two bins (high and low), and the phase measures were split into four 90°

bins centered on 0°, 90°, 180°, and 270° phase angles. A within-subjects 2 (amplitude) x 4 (phase) repeated measures ANOVA analysis was conducted on the participant ΔOR values (calculated as the OR per bin minus the overall OR). Mauchly tests indicated no significant violation in the assumption of sphericity for phase ($\chi^2(5) = 10.169$, $p = 0.0706$), or the interaction of phase and amplitude ($\chi^2(5) = 3.250$, $p = 0.6615$). The ANOVA analysis showed that phase and the interaction between phase and amplitude have statistically significant effects on observation rates, as shown in Table 2. Accordingly, these ANOVA results support our hypothesis that a relationship between alpha phase and visual observation exists, and that this relationship is modulated by the alpha wave amplitude.

Table 1. Two (amplitude) x Four (phase) within-subjects repeated measures ANOVA results.

Source	Statistic	p	η_p^2	Power
phase	F(3,57) = 7.971	0.0002	0.2955	0.986
amplitude	F(1,19) = 1.751	0.2014	0.0844	0.242
phase*amplitude	F(3,57) = 3.770	0.0154	0.1656	0.786

Multiple comparison tests were performed to investigate the precise nature of the effects found to be significant in the ANOVA. Figure 5 shows the results of independent t-tests (significance indicated by #) comparing ΔOR to zero in each of the phase and amplitude levels. Only phase bins 90° and 270° in the high amplitude condition were found to be significantly different [$t(19) = -5.632$, $p = 0.0002$; $t(19) = 3.466$, $p = 0.0207$ respectively, Bonferroni corrected for eight comparisons (Bland and Altman, 1995)]. Tukey's HSD tests (indicated by *) then compared ΔOR distributions between phase bins within each amplitude level. These tests found significant differences in the phase bins only in the high amplitude condition. Specifically, the 90° phase bin was statistically different from the 0° ($p = 0.0077$), 180° ($p = 0.0308$), and 270° ($p < 0.0001$) phase bins in the high amplitude condition (Figure 5). Importantly, the greatest difference in the high amplitude condition was found between phase bins 90° and 270°, with a 22.20% increase in observation rates (95% CI = [9.77% , 34.63%], Figure 5). Collectively, these results

show that stimuli are observed with greatest probability when the alpha wave is near a trough at t2, and with lowest probably when near a peak, when alpha amplitude is high. The perception-phase relationship is robust only at high alpha wave amplitude, likely because during low amplitude oscillations the measured phase is less representative of the underlying neural population activity.

Phase-varying analysis of visual observation

For a high-resolution characterization of the phase effect on ΔOR, we repeated the original procedure at *t2* using 90° phase bins rotated in 1° increments for all 360° phase angles. Figure 6 shows ΔOR for each of the twenty participants in both the high and low amplitude conditions as a function of the centered alpha phase angle of the bins. Indicated along the circumference is the direction of the center of mass of these phase diagrams for each condition, calculated as the angle of the vector mean of each of the 360 points forming the ΔOR distribution. This angle is referred to here as the preferred phase and indicates the phase at which stimuli have the highest probability of being observed given the whole distribution. Note that in the high amplitude condition, the preferred phase is between 180° and 360° (the negative-amplitude portion of the alpha cycle) in 18/20 participants. These preferred phase angles have associated magnitudes indicating how far the distribution's center of mass is offset in that direction and the strength of the effect of phase on observation rates. These preferred phase angles and magnitudes for all participants are shown in Figure 7, panels B and D.

Figure 7, panels A and C, show the ΔOR distributions averaged across all participants. Indicated in black along the circumference is the group's preferred phase. Note that calculating the group's preferred phase as the direction to the center of mass of the group's ΔOR distributions (Figure 7, panels A and C) is equivalent to calculating the vector mean of the twenty individual preferred phases and associated magnitudes (Figure 7, panels B and D). By taking the ΔOR value at the preferred phase angle and subtracting from it the corresponding opposite phase angle (by 180°, referred to as the pessimal phase), we calculated the preferred phase effect (PPE), which indicates the increase in observation rate from pessimal to preferred alpha phase angles, relative to *t2*. Our results showed that the preferred phase was

similar in both the high and low amplitude conditions (272.41° and 284.65°, respectively) with a PPE of 20.96% in the high amplitude condition but 9.12% in the low amplitude condition. These data further substantiate an interaction effect on observation probability between alpha wave phase and amplitude, which was observed in the prior ANOVA analysis.

Additionally, we estimated the 95% confidence intervals (CI) for the group's preferred phase, which are shown as the shaded regions along the circumference of the phase diagrams in Figure 7. These CIs could only be estimated since there is not a known method of computing an angular CI on *non-unit* vectors directly. To estimate these CIs, the twenty individual preferred phase vectors (with associated magnitudes) were first converted to cartesian values. Then, two 95% CIs were calculated separately on both the X and Y components, indicated respectively as the width and height of the shaded rectangles in Figure 7, panels B and D. The angular interval that encompassed both CIs was then used to represent the estimated 95% CI on the preferred phase in the polar space, although this interval will not necessarily be centered on the preferred phase (i.e., mean). In the high amplitude condition, this resulted in an estimated 95% CI of [234.75°, 309.84°], and in the low amplitude condition a much wider CI of [206.22°, 344.45°]. Together with Figure 7A and B, these results show that observations rates are highest at the trough of the alpha wave, and lowest at the peak. This effect is prominent in the high amplitude condition, but also to a much lesser extent in the low amplitude condition recalling these effects did not reach significance in the prior ANOVA.

Measuring induced activity

Alpha band desynchronization (ERD) and phase resetting are characteristically observed in response to relevant visual stimuli (Klimesch et al., 2011). These reflect neural processes resulting from the stimulus, not spontaneous activity, and so in our case are only expected in response to observed trials, not missed trials. To ensure that the amplitude and phase measurements made here are the result of spontaneous brain

activity and not caused by the stimulus itself, we verified that a drop in amplitude or sudden change in phase coherence across trials does not occur in the observed trials at *t2*. Thus, we first calculated the percent change in alpha amplitude, relative to a prestimulus baseline period of -300 to -100 ms, for both the observed and missed trials and these are shown in Figure 8A. There was the expected characteristic drop in alpha amplitude (i.e. ERD) in response to the observed stimuli, however the this was not observed until after 100 ms post stimulus which did not drop below that of the missed stimuli until after 200 ms. This result indicates that measurements made at *t2* (whose mean value is 75.56 ms and range = [62.99, 88.87] ms) were safe from stimulus induced amplitude effects.

To ensure that there was no event related phase resetting, we next calculated phase coherence across all trials, as well as just the observed and missed trials (Figure 8B). As shown in Figure 8B, no phase resetting was apparent in the observed trials or in all trials together, especially prior to 150 ms. No event related phase resetting would be expected in the missed trials, and this was not seen. However, in the phase coherence across missed trials, there is a prominent peak measured at the 70.31 ms time point, marked by the arrow in Figure 8B, but this not an event related phase reset as will be described further in the Discussion. In sum, Figure 8 confirmed that no event related amplitude change or phase reset occurred prior to 100 ms post stimulus, demonstrating that the phase and amplitude measurements taken in the present study are not the result of stimulus induced activity, but of ongoing spontaneous oscillations.

t2 measurements versus prestimulus measurements

To evaluate the benefit of using the individual *t2* time point for measuring the alpha phase effect on perceptual performance, we repeated the above analysis at a time point 100 ms prior to stimulus onset (t= -100ms) and compared the results to those obtained above at *t2*. These measurements have been taken in previous studies in prestimulus periods or at stimulus onset in order to avoid measuring induced oscillatory effects as mentioned above (Mathewson et al., 2009; Mathewson et al., 2012; Milton and

Pleydell-Pearce, 2016; Kizuk and Mathewson, 2017). Whereas in the previous analysis, the four phase bins centered at 0°, 90°, 180°, and 270° were chosen *a priori* based on their theoretical significance at the *t2* time point, no prediction could be made about the relevant phase bins at this prestimulus time point. Instead, the analysis was first performed in 90° phase bins in 1° increments, as was performed previously (i.e. in Figure 7), and the four relevant phase bins were chosen empirically based on the observed preferred phase in the high amplitude condition. In other words, the phase bins were chosen *post hoc* in order to reveal the highest effect phase has on amplitude for this prestimulus t= -100ms time point.

The results of this analysis performed over all 90° bins in both the high and low amplitude conditions are shown in Figure 9. In the high amplitude condition, a phase effect on perceptual performance can again be observed, however in this case the preferred phase was measured at 33.53° (95% CI = [-31.69°, 80.29°]) with a 15.48% PPE. In the low amplitude condition, the preferred phase was measured at -17.22° (95% CI = [-90.04°, 90.07°]) with a 5.22% PPE. Based on the preferred phase measured in the high amplitude condition for prestimulus t= -100ms, the four 90° phase bins centered at 33°, 123°, 213°, and 303° were therefore chosen for the 2x4 repeated measures ANOVA analysis. Mauchly tests indicated no significant violation in the assumption of sphericity for phase ($\chi^2(5) = 5.194$, $p = 0.3926$), or the interaction of phase and amplitude ($\chi^2(5) = 8.861$, $p = 0.1147$). As shown in Table 2, only the main effect of phase was found to have a statistically significant effect on observation rates. Despite these phase bins showing the highest phase effects at t= -100ms, phase – whose bins were chosen *a priori* based on theoretical significance – had stronger effects on observation when the measurements were taken at *t2* (with roughly twice the effect size).

Table 2. Two (amplitude) x Four (phase) within-subjects repeated measures ANOVA results repeated at 100 ms prior to stimulus onset.

Source	Statistic	*p*	η_p^2	Power

phase	F(3,57) = 3.378	0.0243	0.1510	0.735
amplitude	F(1,19) = 0.008	0.9289	0.0004	0.051
phase*amplitude	F(3,57) = 1.695	0.1784	0.0819	0.420

418

419 Figure 10 shows the multiple comparisons result for this data collected at 100 ms prior to stimulus onset
420 for better comparison to those from *t2* shown in Figure 5. Bonferroni corrected independent t-tests
421 compared each ΔOR value to zero, but none were found to be significant, indicating that none of the
422 observation rates in any of these four phase bins differed from the overall mean observation rate either
423 amplitude condition. Tukey's HSD tests (indicated by *) were used to compare ΔOR distributions
424 between phase bins within each amplitude level, but these tests only found a significant difference
425 between 33° and 213° (p = 0.0143) where there was a 15.47% difference in observations rates (95% CI =
426 [2.39%, 28.55%]).

427 In our original hypothesis, we predicted that the high amplitude preferred phase would be 270° at *t2*,
428 which was approximately t=75ms on average. Assuming 1) a perfectly stationary and 2) an exactly 10Hz
429 signal, we would have predicted that the preferred phase at t=-100ms, as well as at *t0*, would have been
430 0°. This was approximately what was found here, however the inexactness is expected since neither
431 assumption is exactly met in the natural alpha rhythm. The non-stationarity would additionally explain the
432 decrease in effect size from *t2* to t=-100ms. Collectively, these results show that although an alpha phase
433 effect on observation rates in a prestimulus period can be observed, the strength of the effect is much
434 weaker than when measurements are taken at *t2*. This finding shows that measurement of alpha
435 oscillations amplitude and phase have the strongest correlation with visual perception at *t2* than at t= -100
436 ms.

Discussion

This study presents a novel, controlled method for examining the neuronic shutter effect, hypothesized to be reflected in the posterior alpha oscillations observed in human EEG. Our hypothesis was that perceptual performance is modulated by the alpha rhythm in a precise phase and timing relationship. To test this relationship, we (1) presented near-threshold intensity visual stimuli through closed eyes to participants; (2) accounted for individual retina-to-V1 conduction delays (*t2*); (3) controlled for asynchrony between hemispheres; and (4) measured the alpha phase and amplitude relative to *t2*. Our results confirm the hypothesized relationship between the alpha rhythm phase and perception, with the greatest rates of observation occurring at the alpha wave trough (excitatory state) and the lowest rates at the alpha wave peak (inhibitory state). The perception-phase relationship appears to be modulated by alpha amplitude as it is observed at high, but not low, alpha wave amplitude. This work is novel in considering the underlying neural structure and function of the visual system when predicting the exact phase relationship to perception, and in controlling for individual neural conduction delay and asynchrony between hemispheres.

While the alpha wave shutter effect has been previously examined (Dustman and Beck, 1965; Mathewson et al., 2009; Mathewson et al., 2012; Milton and Pleydell-Pearce, 2016; Kizuk and Mathewson, 2017), this is the first study, to our knowledge, to directly examine the relationship between perceptual performance and spontaneous alpha phase as measured relative to each individual's conduction delay. Previous studies have either measured alpha phase at stimulus onset, *t0*, or during prestimulus periods (Mathewson et al., 2009; Mathewson et al., 2012; Milton and Pleydell-Pearce, 2016; Kizuk and Mathewson, 2017). Accordingly, the phase measurements were not always well aligned with the LGN excitability state according to the proposed cellular mechanism. Here, when we accounted for each individual's conduction delay, as opposed to the group average conduction delay or measuring at stimulus onset *t0*, we demonstrated the existence of a hypothesized relationship between the alpha phase and perception. Notably, these results provide the most robust evidence consistent with cyclic LGN

462 excitability as the cellular mechanism mediating this effect – though we do not directly examine observe
463 this mechanism. Dustman and Beck (1965) measured reactions times, as opposed to observation rates, at
464 alpha phase adjusted to group average, as opposed to individual conduction delay. This study, similar to
465 ours, found slowest reaction times at the peak (90°) of the alpha wave, and fastest reaction times close to
466 the alpha trough (240°).

467 A 2x4 repeated measures ANOVA found that alpha phase at *t2* had a significant effect on perceptual
468 performance as hypothesized. The interaction effect of phase and amplitude was also found to be
469 significant; the multiple comparisons tests in Figure 5 show that low amplitude attenuates the phase
470 effect. This supports the hypothesis that low amplitude alpha waves reflect asynchronous inhibition
471 within the visual system. Although not part of our original hypothesis, it is somewhat surprising that no
472 main effect of amplitude was found since increased alpha power has sometimes been found to predict
473 poorer perceptual performance (Ergenoglu et al., 2004; Mathewson et al., 2009; Bruers and VanRullen,
474 2018). This perception-power relationship is usually attributed to more generalized early visual inhibition
475 as evidenced by the attenuation of early (C1 and N150) VEP components with increased alpha power
476 (Iemi et al., 2019). Figure 7 gives a higher phase angle resolution view of the effects seen in Figure 5. In
477 both the high and low amplitude conditions, the preferred phase was near the wave trough (272.41° and
478 284.65° at high and low amplitude, respectively). However, in the low amplitude condition, the 95% CI
479 of the preferred phase was much wider, and the PPE was lower (20.96% and 9.12% at high and low
480 amplitude, respectively). These results show how alpha amplitude modulates the effect of alpha phase on
481 perception.

482 To demonstrate the significance of taking alpha amplitude and phase measurements at *t2*, we repeated our
483 analysis at t= -100 ms and compared them to those obtained at *t2*. This prestimilus time point was chosen
484 for two reasons. First, given the risk in poststimulus measurements getting altered by stimulus processing,
485 a prestimilus time point is, therefore, "safer" than a post-stimulus or stimulus-onset time points. Second,
486 given that alpha oscillations have a primary frequency of 10Hz (i.e., has a period of 100ms), the

amplitude-phase relationship would repeat roughly every 100ms. Thus, our results at t= -100ms could help to predict this relationship at stimulus onset, which is 100ms (or one period) away, but under safer signal analysis conditions. At prestimulus time point of -100 ms (t= -100 ms), we did find a significant phase effect, but by using different phase bins more relevant to that time point (i.e., 33°, 123°, 213°, 360°). These phase bins were chosen *post hoc* to reveal the highest effect phase has on observation for this prestimulus time point. Despite showing the highest phase effects, phase – whose bins were chosen *a priori* based on theoretical significance – had stronger effects on observation when the measurements were taken at *t2*. Specifically, the new analysis shows that while phase was found to have a statistically significant effect on observation rates between the preferred and pessimal phase bins in the high amplitude condition, the effect size was weaker and roughly half of that at *t2*. These results show the advantage of making phase and amplitude measurements at *t2* rather than in prestimulus periods.

Poststimulus measures of ongoing spontaneous oscillations are generally avoided since there is a risk that they will be altered by stimulus processing. We verified that our *t2* amplitude and phase measurements were not influenced by that by measuring alpha band ERD/S and ITPC over the -300 to 500ms time range relative to stimulus onset (Figure 8). No stimulus related ERD/S or ITPC were observed prior to 100ms indicating that our measures at *t2* were independent of stimulus processing responses and instead reflected spontaneous activity. Interestingly, there was an overall increase in phase coherence values of the observed and missed trials compared to that of all trials combined (Figure 7B). This increase is expected since, according to the phase effects on observation rates found above, trials that are observed and missed will predominantly occupy specific, opposing phase ranges; thereby resulting in relatively high phase coherence across trials when grouped by observational outcome, and low phase coherence when grouped together. It is also important to note that the missed trial phase coherence trace features a prominent peak at 70.41 ms (indicated by the arrow in Figure 8B). We do not interpret this peak as an event related phase reset since 1) it is the peak of an upward trend beginning at least 300 ms prior to stimulus onset, 2) a decreasing trend is observed immediately afterwards, and 3) no phase reset would be expected in response

512 to an unobserved stimulus. Instead, we interpret this peak as probably marking the time where phase most
513 accurately predicts that a stimulus will not be observed, which – in support of our hypothesis – is within 5
514 ms of the average estimated *t2* (75.56 ms), the point hypothesized to best predict observational outcome.

515 ***Neural mechanism***

516 Understanding the biological mechanism underlying alpha oscillation is important for explaining its
517 function. Although simultaneous recordings with fMRI and PET correlated alpha activity with
518 fluctuations in the thalamus, thus suggesting this might be the origin of alpha rhythm (Hughes et al.,
519 2004; Omata et al., 2013), so far the precise mechanism in humans is not yet agreed on. However, sleep
520 spindles, a ~10 Hz alpha-like oscillation that occurs in 1-3 second bursts during transition to sleep, do
521 have a well-established mechanism. The sleep spindle mechanism includes a negative feedback loop
522 between the LGN and the reticular nucleus (RN, shown in Figure 11). Both nuclei have T-type Ca^{+}
523 channels that, when activated, exhibit burst firing. Between each burst of the LGN, the Ca^{+} channels of
524 the LGN are known to enter a refractory period, and the RN sends a similar, but inhibitory, burst to the
525 LGN. During this time, sensory information from the optic nerve would be less likely to be relayed to V1.
526 Importantly, the refractory periods of these T-type Ca^{+} channels are known to result in each nucleus firing
527 at a ~10 Hz rate (Lopes da Silva, 1991; Sherman, 2001; Timofeev and Bazhenov, 2005; Alexander et al.,
528 2006; Timofeev and Chauvette, 2011). Each LGN burst firing generates massive EPSPs in V1, measured
529 as a negativity in the occipital EEG (Timofeev and Bazhenov, 2005) and producing the ~10 Hz oscillation
530 of sleep spindles seen on EEG. Chen et al. (2016) showed that in mice, very similar mechanisms give rise
531 to both sleep spindles *and* waking alpha oscillations, and that the mechanism is capable of phasically
532 modulating sensory transfer through the thalamus, and on this basis, they predicted the perceptual results
533 found here. In cats, this mechanism was also found to produce waking alpha oscillations with the same
534 phasic sensory gating effect (Lorincz et al., 2009). Although additional and alternate sources, including
535 extrastriate cortical sources, have been proposed to give rise to this oscillatory activity (Steriade et al.,
536 1990; Connors and Amitai, 1997; Bollimunta et al., 2008; van Dijk et al., 2008; Bollimunta et al., 2011),

there is much evidence supporting this thalamic mechanism as the primary driver (Hughes and Crunelli, 2005). Although the present study does not directly test this thalamic mechanism, it is offered as a purely speculative explanation for our results on the basis of previous and more direct research findings (Lorincz et al., 2009; Chen et al., 2016).

Cellular shutter effect generates a behavioral graded gating effect

Although our speculation and not directly tested here, if the neural mechanism posited above were correct, the neuronic shutter effect would then best be described as occurring at the level of individual LGN relay cells, in which the effect may occur at a nearly binary level. However, the behavioral effects we observe are graded, with increasing and decreasing probabilities of stimulus observation. This may be explained given that oscillating excitatory states are not always synchronized across all LGN cells. It is not necessarily the case that all cells are simultaneously in burst-mode firing state; some may concurrently operate in the normal tonic firing state. Thus, one cell may be in a state of inhibition and not relay to V1, while others may relay visual information as usual. However, in asynchronous states, the alpha rhythm should be decreased in amplitude. As shown in this study, at higher alpha amplitudes, the differences in observation rates between peak and trough phases at *t2* increased, presumably indicating that the measured phase becomes more representative of the excitatory state of larger populations of LGN cells with increasing alpha amplitude. Asynchrony is also important to consider, not just among cells within an LGN, but between the LGNs of the left and right hemispheres. As on a cellular level, one LGN may restrict the flow of visual information while the other relays it. Presumably, if all cells within and between LGNs were in perfect synchrony, behavior relative to peak/trough phase at *t2* would reflect a perfect on/off visual shutter. This simple relationship wasn't observed in this study, and it is therefore more appropriate to describe the proposed neuronic shutter effect at the cellular level generating a graded, gating effect of perception at the behavioral level.

In sum, to our knowledge, this is the first study to examine the neuronic shutter effect with an exact phase and timing relationship to perceptual performance predicted by the underlying physiology of the visual system. The study design is novel and rigorous in controlling for individual visual conduction delays and hemispheric asynchrony. During times of high amplitude alpha oscillations, we found participants on average are most likely to perceive stimuli at waveform trough (272.41°) with observation rates 20.96% greater than at the opposing peak phase (92.41°). Given the rigor in our phase measurements, these results provide strong support for a mechanism that modulates perception described by the neuronic shutter effect and is reflected in the posterior alpha rhythm. Further studies with conduction delay-adjusted phase measurement are needed to investigate this shutter effect in the mu and tau rhythms over the sensorimotor and auditory cortices, to explore the possibility of this mechanism in other sensory systems.

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706 **Legends**

707

Figure 1. (A) According to the cruciform model of the calcarine fissure in the V1 cortical area, the lower portion of the fissure will respond to upper visual field stimuli and vice versa for the upper portion of the fissure. For this reason, and as the upper and lower portions of the fissure contain opposing dipoles, upper and lower visual field stimuli will create waveforms of opposing polarity in the EEG. (B) Upper and lower visual field stimuli used for C1 VEP task are centered 3° below the fixation point to account for the overrepresentation of the lower visual field in the calcerine fissure as shown in (A). (C) C1 VEP from a single participant in response to the upper and lower stimuli, the difference was measured as the lower minus the upper VEP. The peak latency of the difference wave (indicated as '+') was used as the estimate of *t2*.

708

Figure 2. Example of the staircase method used to determine the threshold intensity in a single participant. Each time the participant observed the stimulus, the intensity in the next trial was decreased. If the pariticipant did not observe the stimulus in one trial, the intensity was increased in the next trial. Threshold intensity was deterimed as the average intensity of all trials (trials 6-40 in this example) following the third reversal, or corner, in the trial-intensity trace (trial 5 in this example). The dashed threshold intensity line in this figure spans the trials over which the intensities were averaged.

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Figure 3. Each of the twenty participants' alpha peak-to-peak amplitude distribution for all trials used in the analysis at *t2*.

710

Figure 4. C1 VEP component averaged across all participants in response to upper and lower visual field stimuli. The 'Difference' waveform is the 'Lower' waveform subtracted from the 'Upper' waveform. The mean and range of each participant's estimated retina-to-V1 conduction delay (*t2*) is indicated and was calculated as the peak latency of the C1 component in each individual's Difference waveform.

711

Figure 5. Mean change in observation rate (ΔOR) for each amplitude and phase condition relative to mean OR across all conditions when analysis was conducted at *t2* (error bars indicate standard error). Tukey's HSD tests were performed between phase levels within amplitude conditions with significance indicated by '*'. Bonferroni corrected t-tests compared each mean to zero with significance indicated by '#'. 90° = peak. ([*,#]: $p \leq 0.05$, [**,##]: $p \leq 0.01$, [***,###]: $p \leq 0.001$, [****,####]: $p \leq 0.0001$)

712

Figure 6. ΔOR as a function of phase for each participant in the high and low amplitude conditions with radial axis extending to negative values for analysis conducted at *t2*. The preferred phase is indicated on the circumference, calculated as the direction towards the circle's center of mass. 90° = peak.

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Figure 7. Analysis conducted at *t2.* Panels A and C show ΔOR as a function of phase, with radial axis extending to negative values. Panels B and D show individual participants' preferred phase angles; their magnitude is proportional their individual preferred phase effect. The width and height of the shaded rectangles indicate, respectively, the X and Y 95% CI in cartesian space. In all panels, the group mean preferred phase is indicated in black on the circumference of the diagrams, with the shaded region indicating estimated 95% CIs for each amplitude condition. 90° = peak.

714

Figure 8. (A) Change in alpha amplitude, relative to a -300 to -100 ms baseline interval, and (B) alpha phase coherence for observed, missed, and all stimuli together. The arrow indicates a peak in the missed trial phase coherence reflecting the time point were alpha phase best predicts that a trial will be unobserved by the participant. There was no indication of stimulus-induced effects at the *t2* time point where phase and amplitude measures were hypothesized to be of mechanistic relevance.

Figure 9. Analysis conducted at t = -100 ms. Panels A and C show ΔOR as a function of phase, with radial axis extending to negative values. Panels B and D show individual participants' preferred phase angles; their magnitude is proportional their individual preferred phase effect. The width and height of the shaded rectangles indicate, respectively, the X and Y 95% CI in cartesian space. In all panels, the group mean preferred phase is indicated in black on the circumference of the diagrams, with the shaded region indicating estimated 95% CIs for each amplitude condition. 90° = peak.

Figure 10. Mean change in observation rate (ΔOR) for each amplitude and phase condition relative to mean OR across all conditions when analysis was conducted at t = -100 ms (error bars indicate standard error). Tukey's HSD tests were performed between phase levels within amplitude conditions with significance indicated by '*'. Bonferroni corrected did not find any of the means to significantly differ from zero. 90° = peak. (*: $p \leq 0.05$).

Figure 11. Visual pathway from retina to V1. The LGN/RN negative feedback loop provides a mechanism of cycling LGN excitability state. This LGN/RN network is known to give rise to sleep spindles. The same, or a similar, mechanism is expected to give rise the posterior alpha rhythm.

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Table 1. Two (amplitude) x Four (phase) within-subjects repeated measures ANOVA results at t= *t2*.

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Table 2. Two (amplitude) x Four (phase) within-subjects repeated measures ANOVA results repeated at 100 ms prior to stimulus onset (t= -100 ms).

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