J Neurophysiol 122: 251–258, 2019.
First published April 3, 2019; doi:10.1152/jn.00712.2018.

RESEARCH ARTICLE | *Sensory Processing*

Saccadic suppression measured by steady-state visual evoked potentials

Jing Chen,[1] Matteo Valsecchi,[2] and Karl R. Gegenfurtner[2]
[1]*School of Psychology, Shanghai University of Sport, Shanghai, China; and* [2]*Abteilung Allgemeine Psychologie, Justus-Liebig-Universität Gießen, Gießen, Germany*

Submitted 19 October 2018; accepted in final form 28 March 2019

Chen J, Valsecchi M, Gegenfurtner KR. Saccadic suppression measured by steady-state visual evoked potentials. *J Neurophysiol* 122: 251–258, 2019. First published April 3, 2019; doi:10.1152/jn.00712.2018.—Visual sensitivity is severely impaired during the execution of saccadic eye movements. This phenomenon has been extensively characterized in human psychophysics and nonhuman primate single-neuron studies, but a physiological characterization in humans is less established. Here, we used a method based on steady-state visually evoked potential (SSVEP), an oscillatory brain response to periodic visual stimulation, to examine how saccades affect visual sensitivity. Observers made horizontal saccades back and forth, while horizontal black-and-white gratings flickered at 5–30 Hz in the background. We analyzed EEG epochs with a length of 0.3 s either centered at saccade onset (saccade epochs) or centered at fixations half a second before the saccade (fixation epochs). Compared with fixation epochs, saccade epochs showed a broadband power increase, which most likely resulted from saccade-related EEG activity. The execution of saccades, however, led to an average reduction of 57% in the SSVEP amplitude at the stimulation frequency. This result provides additional evidence for an active saccadic suppression in the early visual cortex in humans. Compared with previous functional MRI and EEG studies, an advantage of this approach lies in its capability to trace the temporal dynamics of neural activity throughout the time course of a saccade. In contrast to previous electrophysiological studies in nonhuman primates, we did not find any evidence for postsaccadic enhancement, even though simulation results show that our method would have been able to detect it. We conclude that SSVEP is a useful technique to investigate the neural correlates of visual perception during saccadic eye movements in humans.

NEW & NOTEWORTHY We make fast ballistic saccadic eye movements a few times every second. At the time of saccades, visual sensitivity is severely impaired. The present study uses steady-state visually evoked potentials to reveal a neural correlate of the fine temporal dynamics of these modulations at the time of saccades in humans. We observed a strong reduction (57%) of visually driven neural activity associated with saccades but did not find any evidence for postsaccadic enhancement.

perception and action; saccadic eye movements; saccadic suppression; spectrum analysis; SSVEP

INTRODUCTION

We move our eyes several times a second when we view the world around us. How does the visual system maintain a stable visual representation, while our eyes move constantly? One of the well-documented mechanisms that is thought to support visual stability is saccadic suppression (Binda and Morrone 2018; Ibbotson and Krekelberg 2011; Krock and Moore 2014; Wurtz 2008), which is a temporal reduction of visual sensitivity at the time of saccadic eye movements.

Previous research on saccadic suppression has primarily used behavioral methods to probe visual perception at the time around saccades in human observers (Latour 1962) and electrophysiological techniques to record perisaccadic single neuron responses in nonhuman primates (for reviews, see Ibbotson and Krekelberg 2011; Krock and Moore 2014; Wurtz et al. 2011). Some other studies utilized a computational modeling approach, trying to link behavioral findings in humans and physiological results in the primates (Hamker et al. 2011; Teichert et al. 2010). The assumption of these studies was that visual processing during saccades is essentially similar for humans and nonhuman primates. A recent study (Klingenhoefer and Krekelberg 2017) tested this assumption by assessing perisaccadic visual perception (including detection and localization) in both nonhuman primates and humans. They observed similarities but also substantial differences between species. For example, at a higher level of stimulus contrast, human observers did not show any saccadic suppression, whereas nonhuman primates did. The pattern of perisaccadic mislocalization in the primates was also qualitatively different from that observed in humans under identical conditions. These results demonstrate the need to investigate neural processing of perisaccadic vision in human observers.

A few previous studies did investigate physiological responses during saccades in humans with functional MRI (fMRI) (Kleiser et al. 2004; Sylvester et al. 2005; Sylvester and Rees 2006; Vallines and Greenlee 2006). By comparing blood oxygenation level-dependent signals across different trials (e.g., saccade trials where observers were required to make saccades constantly, versus fixation trials where observers were required to maintain fixations), these studies in general found reductions in brain signals for saccade trials in areas including the lateral geniculate nucleus (LGN), V1, V4, hMT, and V7. Due to the poor temporal resolution of fMRI, however, it was not possible to trace the full time course of saccadic suppression.

The EEG technique offers another noninvasive, high temporal resolution method to investigate human neural processing. The critical problem is that eye movements create large amounts of electrooculography (EOG) signals that contaminate

Address for reprint requests and other correspondence: J. Chen, School of Psychology, Shanghai Univ. of Sport, 650 Qing Yuan Huan Rd., Shanghai 200438, China (e-mail: chenjingps@gmail.com).

the EEG signals originating from the brain. Despite this challenge to use EEG to examine visual processing during eye movements, a few attempts were made (Kovalenko and Busch 2016; Parks and Corballis 2008, 2010; Wauschkuhn et al. 1998). One straightforward approach to deal with the problem of eye-movement-related artifacts is to focus on the EEGs before eye movement initiation. In this way, Wauschkuhn et al. (1998) examined shifts of attention before saccadic eye movements and Parks and Corballis (2008, 2010) investigated the remapping before saccades. Kovalenko and Busch (2016) took another approach by implementing algorithms to remove artifact signals from EEG data in the analysis and revealed a reduction in event-related potentials (ERPs) triggered by stimuli displayed during saccades, which likely reflects the saccadic suppression observed in psychophysics studies (Burr et al. 1994).

In the present study, we chose instead to use the steady-state visually evoked potential (SSVEP) technique to investigate perisaccadic visual perception. The SSVEP is an oscillatory brain response to periodic visual stimulations (Norcia et al. 2015). Compared with ERPs, SSVEP responses have a higher signal-to-noise ratio. SSVEP responses are narrowband, located specifically at the stimulation frequency, while artifacts in EEGs are distributed across a broad range of frequencies. SSVEP signals can, therefore, be reliably separated from noise induced by eye movements, making it an effective approach to examine neural responses during eye movements in human observers (Chen et al. 2017a, 2017b). Here, we use SSVEPs to show that visual responses are transiently reduced at the time of saccades.

METHODS

Participants. Eight observers (6 women and 2 men, average age of 25, ranging from 20 to 28) participated in the experiment. All had normal or corrected-to-normal visual acuity. They signed written informed consent forms in agreement with the Declaration of Helsinki. The study was approved by the local ethics committee (LEK FB6 2017-08).

Stimuli and procedure. Stimuli were displayed using the Psychophysics Toolbox (Brainard 1997; Pelli 1997) in MATLAB (MathWorks, Natick, MA) on a 120-Hz Samsung SyncMaster 2230R7 22-in. monitor (Samsung Group, Seoul, South Korea). With a spatial resolution of 1,680 × 1,050 pixels, the screen extended 61° horizontally and 38° vertically at a viewing distance of 40 cm.

Two blue spots (radius = 0.25°), separated by 12° horizontally, were displayed at the center of the screen (Fig. 1*A*). Observers were required to make horizontal saccades back and forth between the two spots (Fig. 1*B*), at a rate of once every 1 to 2 s, for the 90-s duration of each trial. In the background, horizontal gratings were presented on the whole screen. The gratings had a spatial frequency of 0.83 cycles/°. The luminance of the gratings was modulated sinusoidally between 124.4 and 172.3 cd/m^2, corresponding to a contrast of 16%. The gratings were pattern-reverse flickering (square wave) at 5, 10, 15, 20, or 30 Hz depending on the conditions. Note that pattern-reversal stimuli evoke SSVEP responses at even harmonics, because the two counterphase components activate the same visual mechanism (Norcia et al. 2015).

All eight observers underwent six trials in total. Four observers underwent three trials with 20-Hz flicker and three with 30-Hz flicker. The other four observers underwent two trials at 5 Hz, two at 10 Hz, and two at 15 Hz.

Eye movement recordings and analyses. We used an Eyelink 1,000 table-mounted eye tracker (SR Research, Mississauga, ON, Canada) to record the eye movements from the right eye at 1,000 Hz. Observers' head movements were restricted by using a chin rest. Saccades were detected by the default algorithm from Eyelink, which uses a velocity threshold of 30°/s and an acceleration threshold of 8,000°/s^2. We only included saccades with amplitude between 9 and 15° to ensure that the saccades we analyzed were indeed the large saccades made across the two fixation spots (which were 12° apart). In our paradigm, saccades are frequently followed by small corrective microsaccades. These are less of an issue here because they create much less visual suppression (Stevenson et al. 1986). Still, we excluded saccades that were followed by more than one micro-/corrective saccade or by any eye blink in the time window of [0, 500] ms (14.7% of all saccades). Furthermore, we excluded saccades if the follow-up micro-/corrective saccade had an amplitude >1° (22.5% of all saccades). In the end, 185 saccades on average entered the final analysis (between 83 and 513 for different observers).

EEG recordings and analyses. EEGs were recorded from 29 scalp sites according to the international 10–20 system (FP1, FP2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T7, T8, P7, P8, Fz, Pz, Oz, FC1, FC2, CP1, CP2, FC5, FC6, CP5, CP6, TP9, and TP10). A BrainAmp amplifier (Brain Products, Munich, Germany) sampled signals at 1,000 Hz. The ground electrode was placed at the AFz site and the online reference at the Cz site. We kept electrode impedances <5 kΩ.

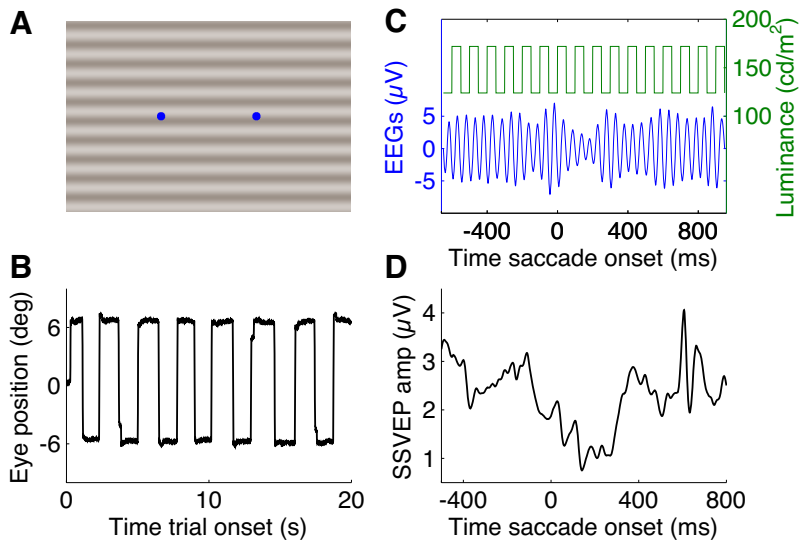

Fig. 1. *A*: stimulus used in the experiment. Participants were required to make saccades back and forth across the 2 blue spots (distance = 12°), once every 1–2 s. The gratings in the background were pattern-reverse flickering at a frequency between 5 and 30 Hz, eliciting steady-state visually evoked potential (SSVEP) responses at the corresponding frequency. *B*: example eye trace for the first 20 s of a trial (90 s). *C*, *left axis*: example EEG trace shown from a case where the stimulus flickered at 10 Hz, thus producing an SSVEP response at 20 Hz. The EEG signals were band-pass filtered between 15 and 25 Hz. *C*, *right axis*: stimulus luminance at a given pixel flickering at 10 Hz (square wave). *D*: evolution of the SSVEP amplitude computed by means of a moving window (300 ms).

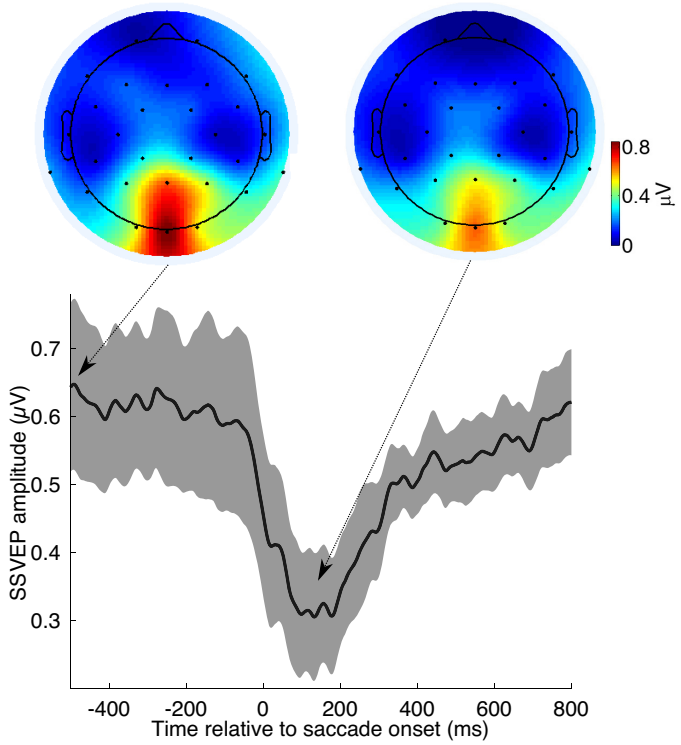

Fig. 2. The grand average of steady-state visually evoked potential (SSVEP) amplitudes at various time point relative to saccade onset. *Top*: images show the topographic distributions of SSVEPs at fixation (*left*: 500 ms before saccade onset) and at saccade (*right*: 100 ms after saccade onset). The SSVEP responses were confined at occipital electrodes (O1, Oz, and O2), which were used for data analysis. Shaded areas indicate SE across all observers.

Customized scripts in MATLAB and functions from EEGlab toolbox (Delorme and Makeig 2004) were used to analyze EEG data (Fig. 1, *C* and *D*). The signals were first re-referenced to a common average reference. We cut out EEG epochs in short time windows (300 ms) centered at variable latencies relative to the onset of saccades. Each epoch was detrended by removing the linear fit (Bach and Meigen 1999) and was multiplied by a Tukey window (i.e., tapered cosine window, $\alpha = 0.2$). The amplitude spectrum of the epoch was then obtained by fast-Fourier transformation (*fft.m* in Matlab). SSVEP amplitude was calculated by subtracting the average amplitude of nearby two bins from the peak amplitude at the stimulation frequency, which effectively discounted the background noise from the SSVEP amplitude (Liu-Shuang et al. 2016). We used the average SSVEP amplitude at O1, Oz, and O2 electrodes for statistics, as SSVEPs in the present study were confined to these electrodes (see Fig. 3). The circular statistics toolbox in MATLAB was used when dealing with phase data (Berens 2009).

RESULTS

Saccadic suppression indexed by SSVEPs. We computed SSVEP power in short time windows (300 ms) centered at variable latencies relative to the onset of saccades. Figure 2 displays the averaged response pattern of all observers, and it shows that saccades did strongly modulate SSVEP responses. The SSVEP responses, as well as the modulation effect, were confined to occipital regions, as shown by the topographic plots in Fig. 2. We also plotted the SSVEP curves for each individual observers in Fig. 3. The reduction of SSVEPs at saccades is clear for all eight observers. The reduction starts from ~100 ms before saccade onset. For the majority of observers, the SSVEP amplitude increased to the presaccade level at ~300 ms after saccades, except for *observers 5* and *8*.

To examine how exactly saccades affect neural processing, we compared SSVEP responses at two specific time points, i.e., the fixation SSVEP at −500 ms relative to saccade onset and the saccade SSVEP at 100 ms after saccade onset (marked by arrows in Fig. 2). As SSVEPs are calculated from a 300-ms EEG epoch, the fixation SSVEP was computed from the epoch at [−650, −350] ms and the saccade SSVEP from the epoch at [−50, 250] ms. Note that we did not choose to calculate the saccade SSVEP exactly at saccade onset (i.e., the [−150, 150] ms epoch), because there is a neural processing delay from visual stimuli to EEG responses. As saccadic suppression is maximum for stimuli presented exactly at saccade onset (Latour 1962; for review, see Ibbotson and Krekelberg 2011), the suppression effect in EEGs will be delayed due to the neural processing delay. In Fig. 2, we can see that maximum suppression for SSVEP is indeed not at saccade onset. We, therefore, decided to analyze the EEG signals within window of [−50, 250] ms to investigate the effect of saccadic suppression.

The amplitude spectrum of saccade epochs versus fixation epochs is shown in Fig. 4, separately for each flicker frequency. The peaks at the corresponding stimulation frequencies are visible in all conditions. Note that Fig. 4, *insets*, shows the normalized SSVEP response, which is the peak amplitude at the response frequency, minus the average amplitude of the two neighboring frequency bins. Compared with fixation epochs, saccade epochs had increased power especially in the lower frequency band (<30 Hz), which most likely resulted from saccade-related EEG signals. Because we calculated the SSVEP amplitude by subtracting the nearby average from the peak, the broadband noise would have minimum influence on

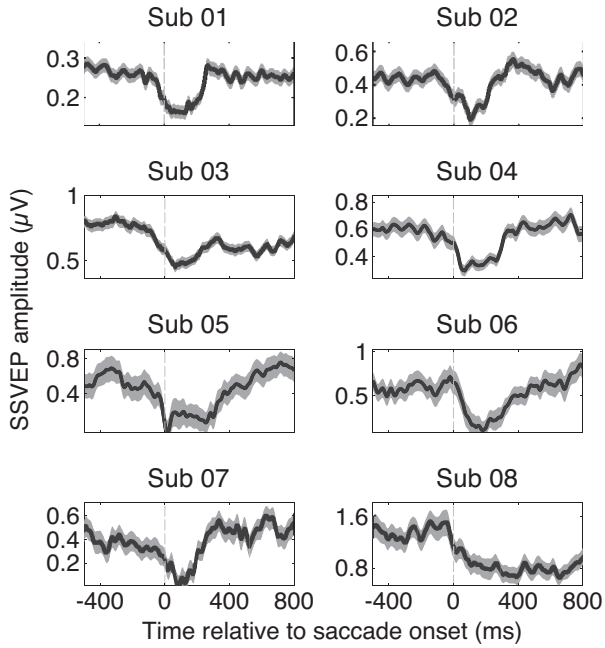

Fig. 3. Steady-state visually evoked potential (SSVEP) amplitude at various time points relative to saccade onset plotted separately for 8 observers. The gray dashed line marks the onset of the saccade. The reduction of SSVEPs at saccade onset is present for all observers. Shaded areas indicate SE across all analyzed saccades, the number of which ranges from 83 to 513 across observers (mean = 250).

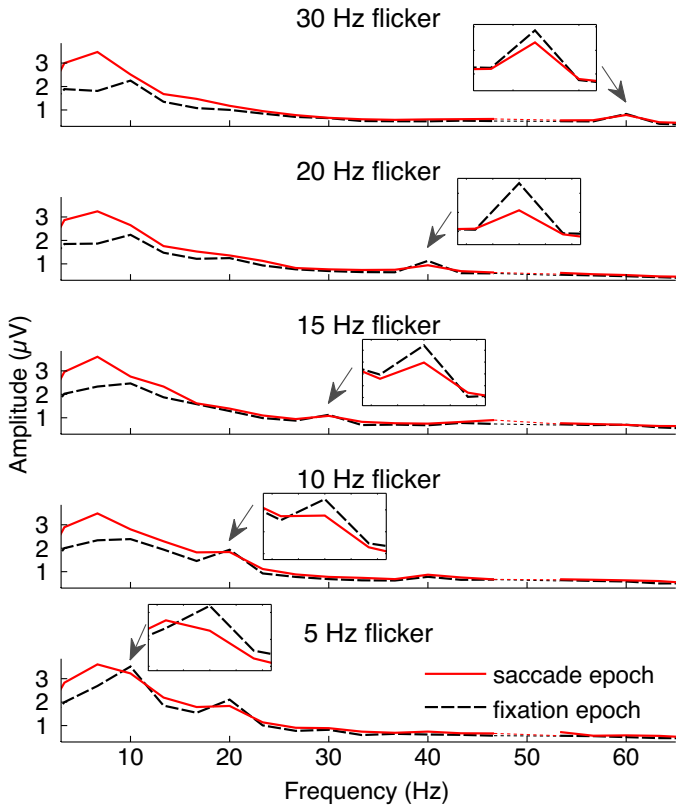

Fig. 4. Amplitude spectrum for different flicker frequencies over all observers. As the flickering stimuli were counterphase pattern reversal stimuli, steady-state visually evoked potentials (SSVEPs) are only observed at even harmonics (see METHODS). Compared with fixation epochs (dashed black), saccade epochs (solid red) show a broadband power increase, which most likely resulted from saccade-related EEG activity. *Inset*: SSVEP responses at the stimulation frequency after normalizing the peak amplitude to the average amplitude of 2 nearby frequency bins. The SSVEP amplitude is reduced in saccade epochs compared with fixation epochs. The amplitude at 50 Hz is dashed out because it corresponds to the power line artifact.

the SSVEP signals, even for the conditions where 5/10/15 Hz stimuli were used.

Saccade epochs, however, had reduced SSVEP amplitude specifically at the response frequency. Figure 5*A* shows the effect in eight individual observers, all of which had a significant reduction of SSVEP amplitude in saccade epochs compared with fixation epochs, $t(7) = 6.05$, $P < 0.001$. The average reduction of eight observers was 56.6%, with a 95% confidence interval of [37.6, 75.5%]. Figure 5*B* shows the SSVEP amplitudes at saccade and at fixation as a function of temporal frequency. There is a trend of larger reduction effect at lower frequencies. As EEG power at lower frequencies is generally stronger, there is probably more room to show the reduction effect at lower frequency range.

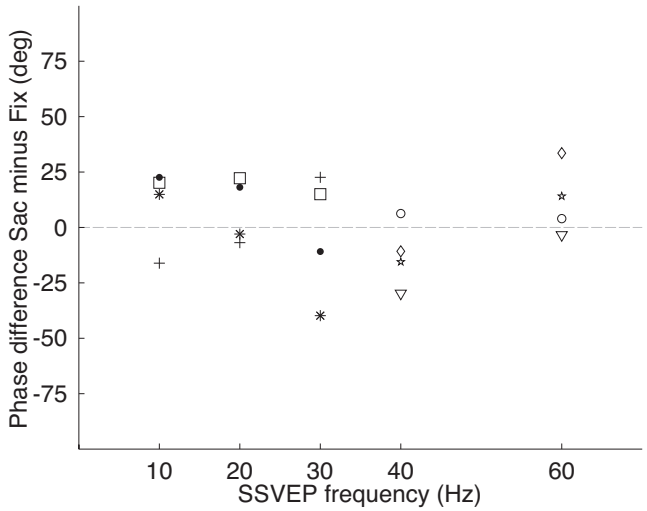

Fig. 6. The difference in phase angles of saccade (Sac) epochs minus fixation (Fix) epochs for each observer, separately for each steady-state visually evoked potential (SSVEP) frequency. Different symbols indicate different observers.

To clarify whether the execution of saccades added a temporal shift to the SSVEP response, rather than a genuine suppression, we further analyzed the phase of the SSVEP oscillation in the saccade and fixation epochs. The two epochs are separated by 600 ms, which, for all frequencies, contains an integer number of flicker cycles. As a result, SSVEPs should be in phase if the SSVEP latency is unaffected by saccades. Note that only the phase difference between saccade and fixation epochs was informative here and the absolute phases were meaningless, as the epochs were decided by the timing of voluntary saccades, which are not phase locked to the flickering stimuli. We focused our analysis on the main response at the second harmonic. Figure 6 shows the phase difference between saccade epochs and fixation epochs for each observer at each response frequency. The phase differences were similar across frequencies, the average being 11, 8, −3, −12, and 12°

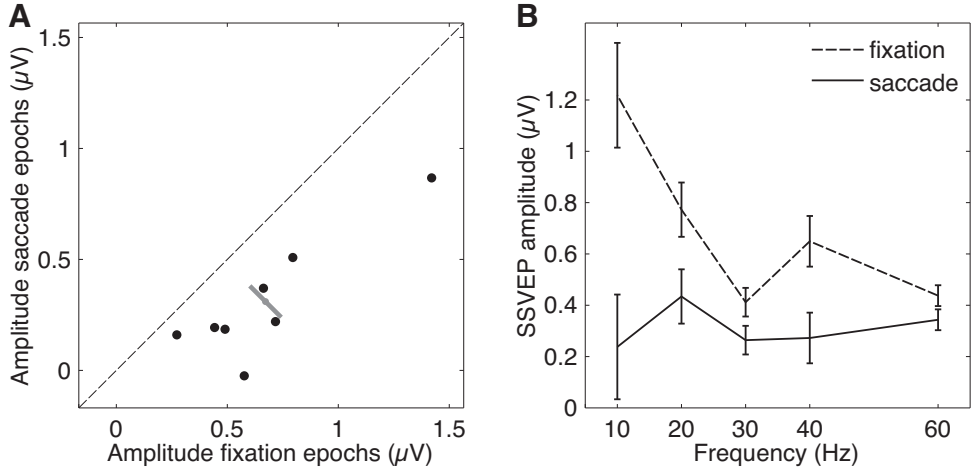

Fig. 5. *A*: steady-state visually evoked potential (SSVEP) amplitudes at saccade plotted as a function of the amplitude at fixation. The amplitude was calculated as the peak amplitude at the stimulation frequency relative to the average amplitude of nearby frequencies. Filled circles denote 8 individual observers. All observers fell below the diagonal line, indicating reduced SSVEP amplitudes in saccade epochs compared with fixation epochs. The gray line shows the average value and the 95% confidence interval. *B*: SSVEP amplitude at fixation and saccade plotted as a function of temporal frequencies. The response of each frequency here was the 2nd harmonic of flicker frequency (e.g., 20-Hz response was from the 10-Hz flicker). Error bars show the within-subject 95% confidence intervals.

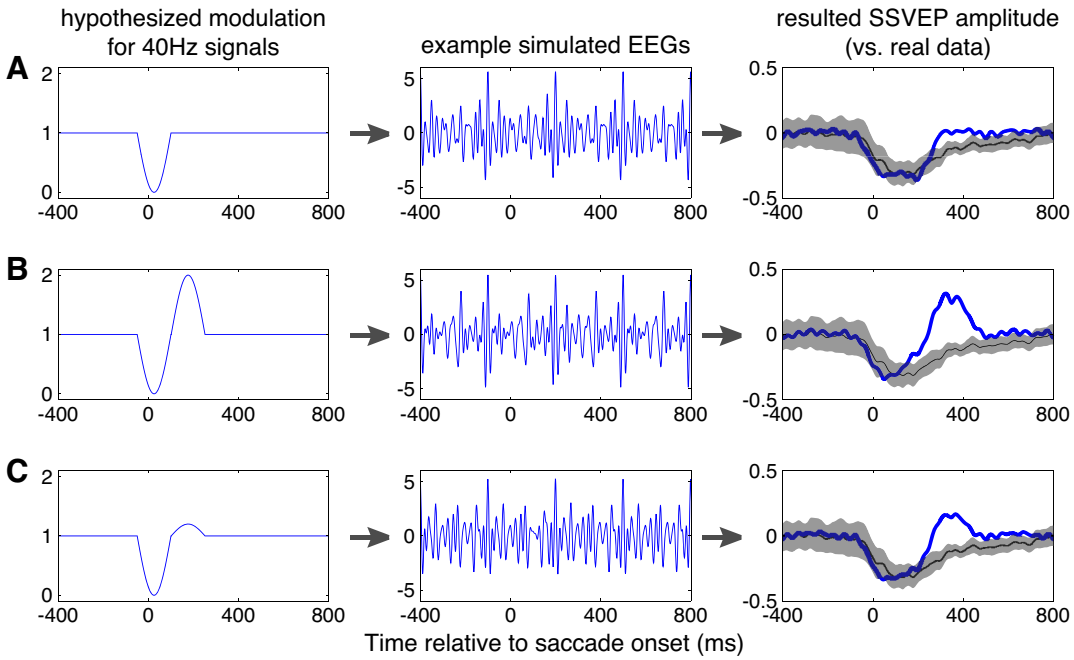

Fig. 7. Simulation results. EEG signals were simulated with steady-state visually evoked potentials (SSVEPs) at 40 Hz (signal-to-noise ratio = 1.7 relative to power at nearby frequencies). *A–C*: the 40-Hz content was convolved with different modulation functions [a suppression window (*A*), a window of suppression followed by enhancement with the same amplitude (*B*), a window of suppression followed by enhancement with 20% of the amplitude (*C*)]. The resulted SSVEP amplitude was the average of 100 iterations. Both the simulation result (blue curve) and the real data (shaded gray, taken from Fig. 2) were baseline corrected to the average value of window of [−500, −300] to make them comparable. The simulation results show that our method in principle recovers both the suppression and the enhancement fairly well (*right*) and that the real SSVEP profile (shaded gray) matches closely to the simulated SSVEPs with a suppression window only (*A*).

(corresponding to 3, 1, −0.3, −0.9, and 0.6 ms in time) for 10-, 20-, 30-, 40-, and 60-Hz responses, respectively. After grouping different frequencies together, we did a one-sample test for the mean angle (*circ_mtest.m* in the circular statistics toolbox in MATLAB) and found that the average angle (2.4°) was not significantly different from 0, with a 95% confidence interval of [−6.2, 11.0°]. SSVEP phases are thus relatively constant during saccades.

No evidence for postsaccadic enhancement. From Figs. 2 and 3, we see no signs of postsaccadic enhancement. The postsaccadic SSVEPs at 300 ms (0.44 μV, SD = 0.20) or 400 ms (0.50 μV, SD = 0.14) were comparable to, if not smaller than, the fixation SSVEPs at −500 ms (0.64 μV, SD = 0.35), both $P > 0.05$. This is in contrary to what was found in multiple single-neuron recording studies [LGN: Reppas et al. 2002; V1: Hass and Horwitz 2011; Kagan et al. 2008; middle temporal (MT)/medial superior temporal (MST): Ibbotson et al. 2007]. One possibility is that our method, which effectively integrates the EEGs over a 300-ms time window, is not able to capture the enhancement effect following suppression. To understand if this limitation was decisive, we simulated EEGs containing 40-Hz oscillations convolved with either a suppression window (Fig. 7*A*), a suppression window followed by an enhancement window with identical amplitude (Fig. 7*B*), or suppression followed by enhancement with only 20% of the amplitude (Fig. 7*C*). The suppression window was a 150-ms window from −50 to 100 ms relative to saccade onset, taken from previous studies (Knöll et al 2011; Latour 1962). Noise was added between 20 and 60 Hz with a resolution of 3.33 Hz, which is the resolution our analysis is able to capture. The signal-to-noise ratio of 40-Hz signals over the signals at the other frequencies was set at 1.7, which is the signal-to-noise ratio of the 40-Hz SSVEP we observed in our data set (Fig. 4, 20-Hz flicker). A processing delay of 100 ms was added to the simulated SSVEPs (see DISCUSSION for details on SSVEP delays). We analyzed the simulated EEG with the identical procedure used above and found that it is able to capture the enhancement effect following suppression (Fig. 7*B*), even when the enhancement effect is at 20% of the amplitude (Fig. 7*C*). Simulations of SSVEPs at other frequencies such as 10/20/30/60 Hz led to similar results. Additionally, our simulation results show that the real SSVEP profile (Fig. 7, shaded gray) taken from Fig. 2 matches closely to the simulated SSVEPs with a suppression window only (Fig. 7*A*).

DISCUSSION

In the current study, we asked participants to make saccadic eye movements, while fast flickering stimuli were presented in the background, which evoked SSVEP responses in EEG signals. The use of horizontal grating stimuli ensured that retinal motion induced by horizontal saccades is minimized. By computing SSVEP amplitudes at variable time points relative to the onset of saccades, we found a significant reduction of neural activity at the time of saccades. This reduction cannot be explained by eye movement-related activities (e.g., EOG artifacts), because these artifacts were associated with a broadband increase in the power spectrum, whereas what we observed in the SSVEPs was a relative reduction specifically at the stimulation frequency. This approach, therefore, is able to capture the neural signature of saccadic suppression. Our method offers a new approach to investigate perisaccadic neural activities in humans, extending previous fMRI and ERP

approaches. While fMRI suffers from poor temporal resolutions and ERP suffers from contaminations of EOG artifacts, the current SSVEP-based method is relatively immune to artifacts and has a good enough temporal resolution to capture the temporal dynamics (Figs. 2 and 3).

Saccadic suppression is a well-established phenomenon in behavioral measurements in humans and in single-cell recordings in nonhuman primates (see reviews in Ibbotson and Krekelberg 2011; Krock and Moore 2014). There are also a few studies on humans using fMRI (Kleiser et al. 2004; Sylvester et al. 2005; Sylvester and Rees 2006; Vallines and Greenlee 2006) and ERP techniques (Kovalenko and Busch 2016). Kleiser et al. (2004) used a block design by comparing the fMRI response in blocks of saccadic trials against that in blocks of fixation trials without any eye movements. They found saccadic suppression in brain areas of V4, hMT+, and V7 but not in early visual areas V1 and V2. Subsequently, two studies using a similar design did reveal suppression effects in V1 and LGN (Sylvester et al. 2005; Sylvester and Rees 2006). As the recorded responses in these studies integrated signals over full trials in the block design, the results were difficult to interpret if one considers the fact that postsaccadic enhancement is usually observed following saccadic suppression in nonhuman primate studies (e.g., Reppas et al. 2002). Vallines and Greenlee (2006) took another approach by flashing stimuli at different times before saccades and measuring blood oxygenation level-dependent signals in V1. They showed that the neural response was reduced as the stimulus was displayed closer to the saccade onset. Kovalenko and Busch (2016) used a similar design and recorded ERPs to a flashed stimulus presented before saccades. After computationally removing EOG artifacts, they showed that the evoked ERPs at occipital regions were reduced in saccadic trials compared with trials without saccades. Our study provides evidence, in additional to these previous studies, for saccadic suppressions in early visual cortex in humans.

One possible issue with our approach is that if SSVEP is considered as a superimposition of repeated visual-evoked potentials and the latency of responses changes in the perisaccadic period, then the EEG power might be transiently smeared across different frequencies, thus producing an apparent reduction in SSVEP power. If neural latency was reduced during saccades, we would expect a positive phase difference between saccade epochs and fixation epochs and the phase difference should get larger with higher flicker frequency. However, we found that the phase did not differ between saccade epochs and fixation epochs, which speaks against this possibility. This result seems to be in conflict with a previous report (Ibbotson et al. 2008) showing that neural response latency was reduced by 8 ms at the time of saccade. The results of Ibbotson et al. (2008) were, however, from neurons in MST, whereas SSVEPs are thought to originate mainly from V1 (Norcia et al. 2015; Wittevrongel et al. 2018). It has not been investigated yet, to our knowledge, whether V1 neurons show similar response latency changes during saccades.

Our approach offers the unique advantage of tracing the temporal dynamics of neural activities throughout the time course of a saccade. In particular, we were able to study the postsaccadic neural activity (Figs. 2 and 3) in ways that were precluded in previous physiological studies in humans, i.e., without having to deal with eye movement EEG artifacts. We did not observe any postsaccadic enhancement following the saccadic suppression, even though our simulation results showed that the analysis would have been capable of capturing it even if the effect of enhancement is at 20% of the effect of suppression. One possibility is that SSVEPs are saturated during the postsaccadic time window. This is unlikely for our low-contrast stimuli (16%). It is generally found that SSVEPs are a linear function of log stimulus contrast for a substantial range of contrasts, up to ~30–40% (see Norcia et al. 2015 for a review). Therefore, SSVEP saturation is unlikely to explain the lack of enhancement.

Previous animal studies showed a postsaccadic enhancement in many brain areas, including LGN, V1, and MST (reviewed in Ibbotson and Krekelberg 2011), with a magnitude varying from much larger (e.g., Reppas et al. 2002) to slightly smaller than the magnitude of suppression (e,g., Bremmer et al. 2009). As SSVEPs are thought to originate largely from V1 (Di Russo et al. 2007; Müller et al. 1997; Wittevrongel et al. 2018), one may expect that we should see postsaccadic enhancement in SSVEPs. In psychophysical studies in humans, however, postsaccadic enhancement is not generally observed (Diamond et al. 2000; Klingenhoefer and Krekelberg 2017; Knöll et al 2011). It might be possible that human V1 does not show enhancement as nonhuman primate V1 does. Alternatively, the absence of a postsaccadic enhancement could be due to the fact that we elicited SSVEPs by means of a full-field, task-irrelevant stimulus in the present study. It is plausible that enhancement might be limited to the saccade target location, which was tested in many previous studies (Bremmer et al. 2009; Ibbotson et al. 2008; MacEvoy et al. 2008), or other predefined task-relevant locations (Yao et al. 2016), so a large part of the stimulation that produced the SSVEP signals in our paradigm might have been immune to a localized postsaccadic enhancement.

Another possibility is that the micro-/corrective saccades, which are associated with suppression as well (e.g., Hafed and Krauzlis 2010), may mask the postsaccadic enhancement. The micro-/corrective saccades occurred in ~70% of main saccades, on average 259 ms after the onset of main saccades (from 217 to 333 ms across observers). To minimize their impact, we excluded saccades from data analysis that were followed by more than one micro-/corrective saccades, or followed by one with an amplitude larger than 1°, in the postsaccadic time window (0–500 ms). As there is less visual suppression for smaller saccades (Stevenson et al. 1986), this procedure made sure that the impact of micro-/corrective saccades in our analysis is minimal. However, follow-up studies are needed to examine how exactly corrective saccades contribute to postsaccadic visual enhancement.

Notice that the maximum suppression point in the curve is not at exactly at the onset of saccades but is delayed by ~100–150 ms (Fig. 2 and 3). This makes sense if one considers the neural processing delay from stimulus onset to the SSVEP responses. The previous studies that investigated the temporal dynamics of saccadic suppression/enhancement were not affected by this delay, because they charted the observed responses as a function of stimulus onset time relative to saccade onset. In our study, however, the neural responses were plotted as a function of the time of SSVEP response relative to saccade onset. How large do we expect the delay for SSVEP responses after stimulus onset? The best estimate may come from studies

that cross correlate EEG responses to visual stimuli of random, nonperiodic luminance sequences (Gonçalves et al. 2014; Lalor et al. 2006; VanRullen and Macdonald 2012). The random luminance approach has a similar rationale as the SSVEP technique, but the use of nonperiodic stimulation makes it possible to find the exact delay of the responses. For example, in the study of Lalor et al. (2006), the delay was around 75–100 ms. In Gonçalves et al. (2014), the delay was around 90–115 ms (see their Fig. 3). Taken such a ~100-ms delay for SSVEPs into consideration, our simulation (Fig. 7, *right*) found that the simulated SSVEP curve fit well with the real SSVEP profiles at the time of saccades (Fig. 2). Our result is therefore consistent with previous studies (Latour 1962; see a recent review in Binda and Morrone 2018) which showed that stimuli presented around saccade onset (and SSVEPs occurring 100 ms later) get maximally suppressed.

In our hands, the time window of suppression expands from −100 ms to 300 ms relative to saccade onset for most observers (Fig. 3). In previous studies, saccadic suppression usually takes place in the window of [−50, 100] ms for both single neural responses (see a review in Ibbotson and Krekelberg 2011) and behavioral measurements (Latour 1962). The fact that the window is longer in our results is likely due to the fact that we needed to compute the FFT over signal segments of 300 ms. This is also evident in our simulation results (Fig. 7*A*), where a 150-ms hypothesized suppression window resulted in an expanded SSVEP window of [−100, 300] ms, which matched the results in Figs. 2 and 3 fairly well.

To conclude, the present study proposed a method based on SSVEPs to examine perisaccadic neural processing in humans. We showed that this approach is able to reveal the fine temporal dynamics of the neural response modulation at the time of saccades. We conclude that the SSVEP method is a useful technique to investigate the neural correlates of visual perception during saccadic eye movements in humans.

GRANTS

The study was funded by the Deutsche Forschungsgemeinschaft (DFG; German Research Foundation) Project No. 222641018 SFB/TRR 135, TP A1 and A8, and DFG International Research Training Group IRTG 1901, "The Brain in Action, and the Shanghai Sailing Program (19YF1445900)."

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

J.C., M.V., and K.G. conceived and designed research; J.C. performed experiments; J.C. analyzed data; J.C., M.V., and K.G. interpreted results of experiments; J.C. prepared figures; J.C. drafted manuscript; J.C., M.V., and K.G. edited and revised manuscript; J.C., M.V., and K.G. approved final version of manuscript.

ENDNOTE

At the request of the authors, readers are herein alerted to the fact that additional materials related to this manuscript may be found at https://doi.org/10.5281/zenodo.2614368. These materials are not a part of this manuscript and have not undergone peer review by the American Physiological Society (APS). APS and the journal editors take no responsibility for these materials, for the website address, or for any links to or from it.

REFERENCES

Bach M, Meigen T. Do's and don'ts in Fourier analysis of steady-state potentials. *Doc Ophthalmol* 99: 69–82, 1999. doi:10.1023/A:1002648202420.

Berens P. CircStat : a MATLAB toolbox for circular statistics. *J Stat Softw* 31: 257–266, 2009. doi:10.18637/jss.v031.i10.

Binda P, Morrone MC. Vision during saccadic eye movements. *Annu Rev Vis Sci* 4: 193–213, 2018. doi:10.1146/annurev-vision-091517-034317.

Brainard DH. The Psychophysics Toolbox. *Spat Vis* 10: 433–436, 1997. doi:10.1163/156856897X00357.

Bremmer F, Kubischik M, Hoffmann KP, Krekelberg B. Neural dynamics of saccadic suppression. *J Neurosci* 29: 12374–12383, 2009. doi:10.1523/JNEUROSCI.2908-09.2009.

Burr DC, Morrone MC, Ross J. Selective suppression of the magnocellular visual pathway during saccadic eye movements. *Nature* 371: 511–513, 1994. doi:10.1038/371511a0.

Chen J, Valsecchi M, Gegenfurtner KR. Enhanced brain responses to color during smooth-pursuit eye movements. *J Neurophysiol* 118: 749–754, 2017a. doi:10.1152/jn.00208.2017.

Chen J, Valsecchi M, Gegenfurtner KR. Attention is allocated closely ahead of the target during smooth pursuit eye movements: evidence from EEG frequency tagging. *Neuropsychologia* 102: 206–216, 2017b. doi:10.1016/j.neuropsychologia.2017.06.024.

Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods* 134: 9–21, 2004. doi:10.1016/j.jneumeth.2003.10.009.

Di Russo F, Pitzalis S, Aprile T, Spitoni G, Patria F, Stella A, Spinelli D, Hillyard SA. Spatiotemporal analysis of the cortical sources of the steady-state visual evoked potential. *Hum Brain Mapp* 28: 323–334, 2007. doi:10.1002/hbm.20276.

Diamond MR, Ross J, Morrone MC. Extraretinal control of saccadic suppression. *J Neurosci* 20: 3449–3455, 2000. doi:10.1523/JNEUROSCI.20-09-03449.2000.

Gonçalves NR, Whelan R, Foxe JJ, Lalor EC. Towards obtaining spatiotemporally precise responses to continuous sensory stimuli in humans: a general linear modeling approach to EEG. *Neuroimage* 97: 196–205, 2014. doi:10.1016/j.neuroimage.2014.04.012.

Hafed ZM, Krauzlis RJ. Microsaccadic suppression of visual bursts in the primate superior colliculus. *J Neurosci* 30: 9542–9547, 2010. doi:10.1523/JNEUROSCI.1137-10.2010.

Hamker FH, Zirnsak M, Ziesche A, Lappe M. Computational models of spatial updating in peri-saccadic perception. *Philos Trans R Soc Lond B Biol Sci* 366: 554–571, 2011. doi:10.1098/rstb.2010.0229.

Hass CA, Horwitz GD. Effects of microsaccades on contrast detection and V1 responses in macaques. *J Vis* 11: 1–17, 2011. doi:10.1167/11.3.3.

Ibbotson M, Krekelberg B. Visual perception and saccadic eye movements. *Curr Opin Neurobiol* 21: 553–558, 2011. doi:10.1016/j.conb.2011.05.012.

Ibbotson MR, Crowder NA, Cloherty SL, Price NS, Mustari MJ. Saccadic modulation of neural responses: possible roles in saccadic suppression, enhancement, and time compression. *J Neurosci* 28: 10952–10960, 2008. doi:10.1523/JNEUROSCI.3950-08.2008.

Ibbotson MR, Price NS, Crowder NA, Ono S, Mustari MJ. Enhanced motion sensitivity follows saccadic suppression in the superior temporal sulcus of the macaque cortex. *Cereb Cortex* 17: 1129–1138, 2007. doi:10.1093/cercor/bhl022.

Kagan I, Gur M, Snodderly DM. Saccades and drifts differentially modulate neuronal activity in V1: effects of retinal image motion, position, and extraretinal influences. *J Vis* 8: 1–25, 2008. doi:10.1167/8.14.19.

Kleiser R, Seitz RJ, Krekelberg B. Neural correlates of saccadic suppression in humans. *Curr Biol* 14: 386–390, 2004. doi:10.1016/j.cub.2004.02.036.

Klingenhoefer S, Krekelberg B. Perisaccadic visual perception. *J Vis* 17: 16, 2017. doi:10.1167/17.9.16.

Knöll J, Binda P, Morrone MC, Bremmer F. Spatiotemporal profile of peri-saccadic contrast sensitivity. *J Vis* 11: 386–15, 2011. doi:10.1167/11.14.15.

Kovalenko LY, Busch NA. Probing the dynamics of perisaccadic vision with EEG. *Neuropsychologia* 85: 337–348, 2016. doi:10.1016/j.neuropsychologia.2015.12.012.

Krock RM, Moore T. The influence of gaze control on visual perception: eye movements and visual stability. *Cold Spring Harb Symp Quant Biol* 79: 123–130, 2014. doi:10.1101/sqb.2014.79.024836.

Lalor EC, Pearlmutter BA, Reilly RB, McDarby G, Foxe JJ. The VESPA: a method for the rapid estimation of a visual evoked potential. *Neuroimage* 32: 1549–1561, 2006. doi:10.1016/j.neuroimage.2006.05.054.

Latour PL. Visual threshold during eye movements. *Vision Res* 2: 261–262, 1962. doi:10.1016/0042-6989(62)90031-7.

Liu-Shuang J, Torfs K, Rossion B. An objective electrophysiological marker of face individualisation impairment in acquired prosopagnosia with fast periodic visual stimulation. *Neuropsychologia* 83: 100–113, 2016. doi:10.1016/j.neuropsychologia.2015.08.023.

MacEvoy SP, Hanks TD, Paradiso MA. Macaque V1 activity during natural vision: effects of natural scenes and saccades. *J Neurophysiol* 99: 460–472, 2008. doi:10.1152/jn.00612.2007.

Müller MM, Teder W, Hillyard SA. Magnetoencephalographic recording of steady-state visual evoked cortical activity. *Brain Topogr* 9: 163–168, 1997. doi:10.1007/BF01190385.

Norcia AM, Appelbaum LG, Ales JM, Cottereau BR, Rossion B. The steady-state visual evoked potential in vision research: a review. *J Vis* 15: 4, 2015. doi:10.1167/15.6.4.

Parks NA, Corballis PM. Electrophysiological correlates of presaccadic remapping in humans. *Psychophysiology* 45: 776–783, 2008. doi:10.1111/j.1469-8986.2008.00669.x.

Parks NA, Corballis PM. Human transsaccadic visual processing: presaccadic remapping and postsaccadic updating. *Neuropsychologia* 48: 3451–3458, 2010. doi:10.1016/j.neuropsychologia.2010.07.028.

Pelli DG. The VideoToolbox software for visual psychophysics: transforming numbers into movies. *Spat Vis* 10: 437–442, 1997. doi:10.1163/156856897X00366.

Reppas JB, Usrey WM, Reid RC. Saccadic eye movements modulate visual responses in the lateral geniculate nucleus. *Neuron* 35: 961–974, 2002. doi:10.1016/S0896-6273(02)00823-1.

Stevenson SB, Volkmann FC, Kelly JP, Riggs LA. Dependence of visual suppression on the amplitudes of saccades and blinks. *Vision Res* 26: 1815–1824, 1986. doi:10.1016/0042-6989(86)90133-1.

Sylvester R, Haynes JD, Rees G. Saccades differentially modulate human LGN and V1 responses in the presence and absence of visual stimulation. *Curr Biol* 15: 37–41, 2005. doi:10.1016/j.cub.2004.12.061.

Sylvester R, Rees G. Extraretinal saccadic signals in human LGN and early retinotopic cortex. *Neuroimage* 30: 214–219, 2006. doi:10.1016/j.neuroimage.2005.09.014.

Teichert T, Klingenhoefer S, Wachtler T, Bremmer F. Perisaccadic mislocalization as optimal percept. *J Vis* 10: 19, 2010. doi:10.1167/10.8.19.

Vallines I, Greenlee MW. Saccadic suppression of retinotopically localized blood oxygen level-dependent responses in human primary visual area V1. *J Neurosci* 26: 5965–5969, 2006. doi:10.1523/JNEUROSCI.0817-06.2006.

VanRullen R, Macdonald JS. Perceptual echoes at 10 Hz in the human brain. *Curr Biol* 22: 995–999, 2012. doi:10.1016/j.cub.2012.03.050.

Wauschkuhn B, Verleger R, Wascher E, Klostermann W, Burk M, Heide W, Kömpf D. Lateralized human cortical activity for shifting visuospatial attention and initiating saccades. *J Neurophysiol* 80: 2900–2910, 1998. doi:10.1152/jn.1998.80.6.2900.

Wittevrongel B, Khachatryan E, Fahimi Hnazaee M, Carrette E, De Taeye L, Meurs A, Boon P, Van Roost D, Van Hulle MM. Representation of steady-state visual evoked potentials elicited by luminance flicker in human occipital cortex: an electrocorticography study. *Neuroimage* 175: 315–326, 2018. doi:10.1016/j.neuroimage.2018.04.006.

Wurtz RH. Neuronal mechanisms of visual stability. *Vision Res* 48: 2070–2089, 2008. doi:10.1016/j.visres.2008.03.021.

Wurtz RH, Joiner WM, Berman RA. Neuronal mechanisms for visual stability: progress and problems. *Philos Trans R Soc Lond B Biol Sci* 366: 492–503, 2011. doi:10.1098/rstb.2010.0186.

Yao T, Ketkar M, Treue S, Krishna BS. Visual attention is available at a task-relevant location rapidly after a saccade. *eLife* 5: e18009, 2016. doi:10.7554/eLife.18009.