Attenuation of Visual-Evoked Responses to Hand and Saccade-Initiated Flashes

Nathan G. Mifsud [a], Tom Beesley [a], Tamara L. Watson [b], Thomas J. Whitford [a]

[a] School of Psychology, UNSW Australia, Sydney, New South Wales, Australia

[b] School of Social Sciences and Psychology, Western Sydney University, Bankstown, New South Wales, Australia

Address correspondence to: Nathan G. Mifsud, School of Psychology, Mathews Building, UNSW Australia, Sydney, NSW 2052, Australia. E-mail: nathan.mifsud@unsw.edu.au

Abstract

Sensory attenuation refers to reduced brain responses to self-initiated sensations relative to those produced by the external world. The phenomenon is often explained by universal prediction error mechanisms that are not described in terms of any one sensory modality, yet it is most widely reported for auditory stimuli resulting from self-initiated hand movements. The present study explored the generalizability of sensory attenuation by measuring the electroencephalogram (EEG) of participants exposed to brief flashes initiated by either a button-press or volitional saccade, and comparing these conditions to identical stimuli produced by the computer. Results showed that the largest reduction of anterior visual N1 amplitude occurred for saccade-initiated flashes, while button-press-initiated flashes evoked an intermediary response between the saccade-initiated and externally initiated conditions. This finding indicates that sensory attenuation occurs for visual stimuli, and suggests that the degree of electrophysiological attenuation may relate to the strength of habitual associations between the type of motor action and the modality of the sensory response.

Keywords

- Event-related potentials; Visual N1; Predictive processing; Sensory attenuation; Saccadic movements; Efference copy; Corollary discharge; Visual-evoked potentials

Highlights

- Visual N1 response was smaller to flashes initiated by saccades than button-presses
- Robust demonstration of attenuation of self-initiated sensations in the visual domain
- Associative strength between actions and outcomes may modulate sensory attenuation

Introduction

Sensory attenuation refers to self-initiated stimuli evoking reduced neurophysiological (e.g., Baess, Jacobsen, & Schröger, 2008; Houde, Nagarajan, Sekihara, & Merzenich, 2002; Schafer & Marcus, 1973) and phenomenological (e.g., Blakemore, Frith, & Wolpert, 1999; Cardoso-Leite, Mamassian, Schütz-Bosbach, & Waszak, 2010; Sato, 2008) sensory representations, compared to the sensory representations evoked by physically identical, externally initiated stimuli. The phenomenon is typically explained using a forward model which predicts the sensory consequences of intended actions based on internal motor commands, where these predictions are subtracted from actual sensory input (Bays & Wolpert, 2007; Wolpert, Ghahramani, & Jordan, 1995). Conversely, externally initiated stimuli lack accompanying motor information, and are thus marked by a large disparity between predicted and actual sensory inputs. Notably, Feinberg (1978) first suggested that disruption of this distinction between self and the external world could account for some of the characteristic symptoms of schizophrenia (e.g., delusions of control), and evidence has emerged to support this theory (Ford et al., 2001; Pinheiro, Rezaii, Rauber, & Niznikiewicz, 2016; Whitford et al., 2011).

Studies of sensory attenuation have thus far largely been limited to the auditory domain (e.g., see Table 3 in Hughes, Desantis, & Waszak, 2013), centred on a reliable neurophysiological component that is used by multiple research groups as an index of sensory attenuation—that is, the N1 or N1m component, an evoked potential or magnetic field that is consistently reduced for self-initiated vocalizations and tones (e.g., Baess et al., 2008; Curio, Neuloh, Numminen, Jousmaki, & Hari, 2000; Houde et al., 2002; Mifsud, Beesley, Watson, & Whitford, 2016; Sowman, Kuusik, & Johnson, 2012). Given the well-established positive relationship between the auditory N1 component and stimulus intensity (Näätänen & Picton, 1987), the finding that self-initiated sounds have reduced auditory N1

response suggests that the brain processes them as being "softer"; in other words, that self-initiated sounds have lower perceived loudness. This may reflect an ecological adaptation, in the sense that the strong auditory feedback associated with our own speech might require attenuation to preserve the sensitivity of receptors to incoming sounds (Bendixen, SanMiguel, & Schröger, 2012).

In contrast to the auditory domain, studies of sensory attenuation in the visual domain are relatively scarce, and results are more difficult to reconcile. Differences in visual-evoked potentials (VEPs) between self-initiated and externally initiated visual stimuli have been inconsistent in terms of both the direction and spatial location of the observed differences. This may be due to the diverse range of stimuli which have been used, the event-related components of interest and, by extension, the choices of reference sites. Self-initiation has been shown to result in anterior (but not occipital) reduction of N1 for flashes (Schafer & Marcus, 1973, mastoid-referenced data) and arrow shapes (Gentsch & Schütz-Bosbach, 2011, average-referenced data), and occipital reduction of P2 for faces and houses (Hughes & Waszak, 2014, FCz-referenced data). Conversely, occipital amplification of P1 has been shown for pattern-onsets (Hughes & Waszak, 2011, vertex-referenced data) and occipital amplification of N145 for pattern-reversals (Mifsud, Oestreich, et al., 2016, Fz-referenced data). However, sensory attenuation has also been observed in contrast discrimination tasks using Gabor patches (Cardoso-Leite et al., 2010; Stenner, Bauer, Haggard, Heinze, & Dolan, 2014). Clearly, further experimental work is required in the visual domain which builds on existing self-initiation paradigms. Therefore, in line with the two previous studies which have identified sensory attenuation to the visual N1 (Gentsch & Schütz-Bosbach, 2011; Schafer & Marcus, 1973), we chose to measure the anterior N1 effect (i.e., frontocentral maxima, mastoid-referenced).

A further limiting factor of visual exploration of sensory attenuation is that, to our knowledge, nearly all the studies have participants initiate visual stimuli by pressing a button. Despite this highly specific experimental condition, there is an implicit assumption that we can generalize findings across a much wider range of action–sensation contingencies. In the auditory domain, the limits of this assumption have been tested by van Elk, Salomon, Kannape, and Blanke (2014) and Mifsud, Beesley, et al. (2016), who employed paradigms using foot and saccade initiation respectively to demonstrate that auditory-evoked potentials (AEP) differed depending on the motor output region used to produce the incoming stimulus. In the study by Mifsud, Beesley, et al. (2016), for example, a greater degree of auditory N1 attenuation was observed for button-press initiated tones than for saccade-initiated tones, consistent with the fact that while hand movements are strongly associated with auditory sensations (e.g., the sound of one's fingers on the keyboard), eye movements are not.

The present study sought to test whether a similar pattern is evident for self-initiated visual stimuli, using a new saccade initiation paradigm. We aimed to determine whether attenuation of VEP amplitudes would occur for button-press and saccade-initiated flashes in comparison to externally initiated flashes. We expected that neurophysiological attenuation of button-press initiated flashes would occur, based on the findings of a similar condition reported by Schafer and Marcus (1973). However, in line with eye movements being more tightly coupled with visual sensations than hand movements, we hypothesized that there would be a greater degree of attenuation for flashes initiated by saccades than for flashes initiated by button-presses.

Method

Participants

Forty participants were recruited at UNSW Australia. 7 participants were excluded due to low signal-to-noise ratio in one or more of the stimulus blocks. Of the remaining 33

participants, 15 were female, 29 were right-handed, and mean age was 22 years (*SD* = 6). Participants provided written, informed consent and received either course credit (*n* = 21) or financial reimbursement (*n* = 12, A$30) in exchange for their time. This study was approved by the UNSW Human Research Ethics Advisory Panel (Psychology).

Procedure

Following completion of a demographics questionnaire, participants were fitted with an EEG cap and electrodes. EEG was then continuously recorded while participants completed the experiment. Stimulus presentation was controlled using MATLAB (MathWorks, Natick, MA). Viewing distance was 60 cm from a computer monitor with integrated eye tracking system (Tobii TX300: 300 Hz gaze sampling rate; 23", 60 Hz, 1920 × 1080 resolution TFT screen; accuracy of 0.4° visual angle; system latency under 10 ms), calibrated with a 5-point procedure prior to EEG recording.

The experiment comprised five conditions: two types of self-initiation conditions (i.e., button-press and saccade-initiated flashes) and their corresponding motor control conditions (i.e., button-presses and saccades without consequent flashes), and an externally initiated condition (i.e., flashes initiated without participant input). Each condition was presented in a homogenous 80-trial block, and block order was randomized between participants. Three practice trials preceded each block to ensure participants understood the instructions displayed on screen, and, where appropriate, allowed the experimenter to verbally encourage self-paced rather than speeded responses. Individual trials in all conditions were separated by a uniformly distributed random interval (2–4 s). The EEG recording lasted approximately 50 min.

Press condition. In this condition, a visual stimulus (full-field flash) was self-initiated by a button-press (i.e., hand motor output). Participants were instructed to respond at will any time after the appearance of a red fixation dot (0.7° diameter) presented in the centre of a

black screen, and did so by pressing the space bar on a low-latency keyboard (Ducky Shine 4: 1000 Hz report rate) with their dominant hand. Responses immediately delivered an unstructured full-field white flash (100 cd/m^2 mean luminance) of 33.33 ms duration; i.e., two frames, verified with a photometer.

Saccade condition. In this condition, the visual stimulus was self-initiated by a volitional saccade (i.e., eye motor output). Each trial began with two dots appearing on screen: a solid red circle in the centre of screen (identical to the fixation in the press-initiated condition) and a distal (17° left) hollow white circle. Participants were instructed to initially fixate on the white circle, which would turn solid once the script detected their gaze, based on a 20 ms sample of location recordings. If detection took longer than 5 seconds, trials were skipped with replacement (M = 2.7 skipped trials per participant across both stimulus and motor saccade-initiated blocks). Following fixation on the white circle, participants shifted their gaze at will to the red circle, which immediately delivered a full-field flash identical to the press-initiated stimulus. More precisely, flashes followed detection of the gaze within the 200-px (5°) square area of interest surrounding the central red circle. We confirmed that system latency was identical in both self-initiation conditions using a photometer to detect actual delivery of the flash; that is, mean latency between the end of the action (button-press or the eye attaining fixation in the center) and stimulus delivery was 37 ms (SD = 7 ms).

Motor conditions. The motor control conditions were identical to their respective self-initiated conditions, except that pressing the space bar or shifting gaze between circles did not result in the delivery of a stimulus. The ensuing EEG activity was subsequently subtracted from the appropriate self-initiated conditions to remove EEG activity associated with button-pressing (for the press condition) or a singular, volitional eye movement (for the saccade condition), as is standard practice in button-press studies of this nature (Baess et al.,

2008; Martikainen, Kaneko, & Hari, 2005; Whitford et al., 2011), and as was previously used for the saccade initiation condition in the study of Mifsud, Beesley, et al. (2016).

External condition. In this condition, stimuli were delivered automatically (i.e., without participant input) to assess electrophysiological response to externally initiated, temporally unpredictable stimuli. Trials began with a red fixation dot followed by a uniformly distributed random interval (0.5–2.5 s) before a flash was presented (identical to that in the self-initiated conditions). Participants were instructed to keep their eyes open and maintain their gaze on the screen at all times.

EEG data acquisition. EEG was recorded with a BioSemi ActiveTwo system using 64 Ag-AgCl active electrodes placed according to the extended 10-20 system. Analog signals were anti-aliased with a fixed first-order filter (-3 dB at 3,600 Hz) and continuously digitized at a sampling frequency of 2,048 Hz, with common mode sense (CMS) and driven right leg (DRL) used as reference and ground electrodes. During offline preprocessing, data were re-referenced to the averaged mastoid electrodes as is typical for the visual N1 (Clark, Fan, & Hillyard, 1995; Vogel & Luck, 2000), band-pass filtered from 0.01 to 30 Hz (8^{th} order zero-phase Butterworth IIR), and separated into 600-ms epochs (100 ms pre-onset and 500 ms post-onset). Data were baseline corrected with the average voltage between -100 and 0 ms. To address eye blinks and movement artefacts, we rejected individual epochs at any electrode site which contained EEG activity exceeding ± 75 µV or min-max changes in excess of 75 µV between adjacent 100-ms intervals. At electrode Cz, this resulted in a mean rejection rate of 4.5% ± 5.5% (*SD*) trials (press: 4.7% ± 6.0%, saccade: 3.9% ± 6.4%, external: 5.0% ± 5.5%), with no significant differences between stimulus blocks, $F(2,64) = 0.99$, $p = .378$, $\eta_p^2 = .03$. We then averaged individual trials for each condition to produce event-related potentials (ERPs) for each participant. Lastly, motor waveforms were subtracted from the appropriate self-initiated waveforms to produce difference waveforms. Figure 1 demonstrates

the effect of the motor subtraction procedure by presenting grand-averaged ERPs at electrode FCz for the uncorrected waveforms in the self-initiated conditions and their corresponding motor waveforms. Hereafter, unless explicitly noted otherwise, mentions of the self-initiated waveforms refer to these motor-corrected waveforms. Data preprocessing was done in BrainVision Analyzer 2 (Brain Products GmbH, Munich, Germany), and statistical analyses were performed in SPSS version 23 (IBM Corp, Armonk, US).

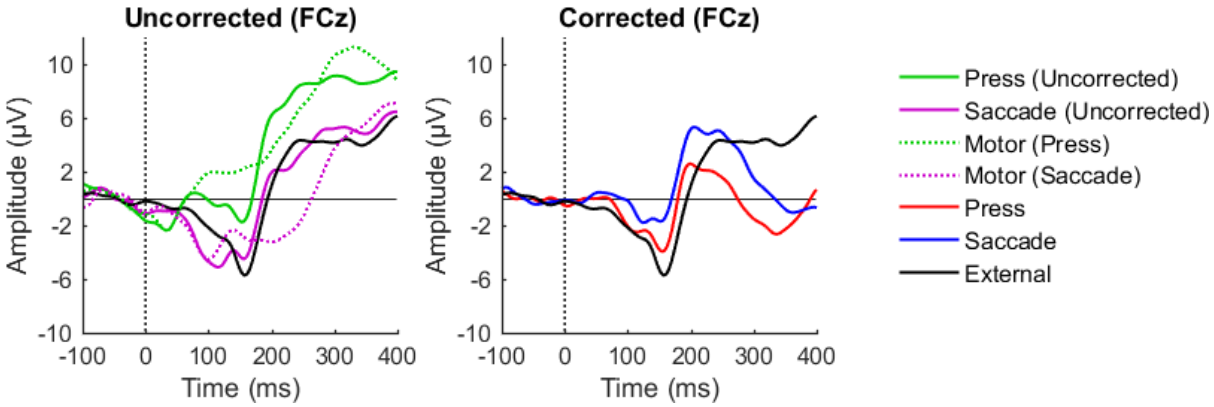

Figure 1. Grand-averaged ERPs at electrode FCz for uncorrected self-initiated conditions (solid traces, left), their corresponding motor conditions (dotted traces, left), and motor-corrected self-initiation conditions (right). The externally initiated condition (black line, both panels) is included for comparison. The x-axes represent time in milliseconds (ms) where 0 is flash onset, and the y-axes represent amplitude in microvolts (μV).

Results

Analysis procedures. As the latency and amplitude of flash ERPs are sensitive to stimulus parameters (Luck & Kappenman, 2012), we used a collapsed localizer approach to guide our analysis procedures; that is, waveforms were averaged across the press, saccade, and external conditions, and these collapsed waveforms were used to identify measurement windows centred on the peaks at which the N1 component was maximal. Accordingly, to investigate the effect of condition on the visual components, we conducted separate one-way analyses of variance (ANOVA) on the condition factor (press, saccade, external) on the mean amplitudes of the N1 (150 to 160 ms) and P2 (230 to 250 ms) at electrode FCz, as this was the electrode for which the N1 component was maximal. Note that while VEPs recorded from FCz remain stable with stimulus position (Clark et al., 1995), early visual responses of competing polarity would occur following a full-field flash due to the physiology of retinotopic cortex, which renders interpretation of occipital sites difficult. Therefore, they were not analysed here. In cases where the assumption of sphericity was violated, a Greenhouse-Geisser correction was applied. To accompany the cluster analysis which was subsequently performed, Figure 2 presents the grand-averaged ERPs at electrode FCz and its neighbouring electrodes (i.e., Fz, FC1, FC2, and Cz). Figure 3 presents the component scalp distributions for each stimulus condition.

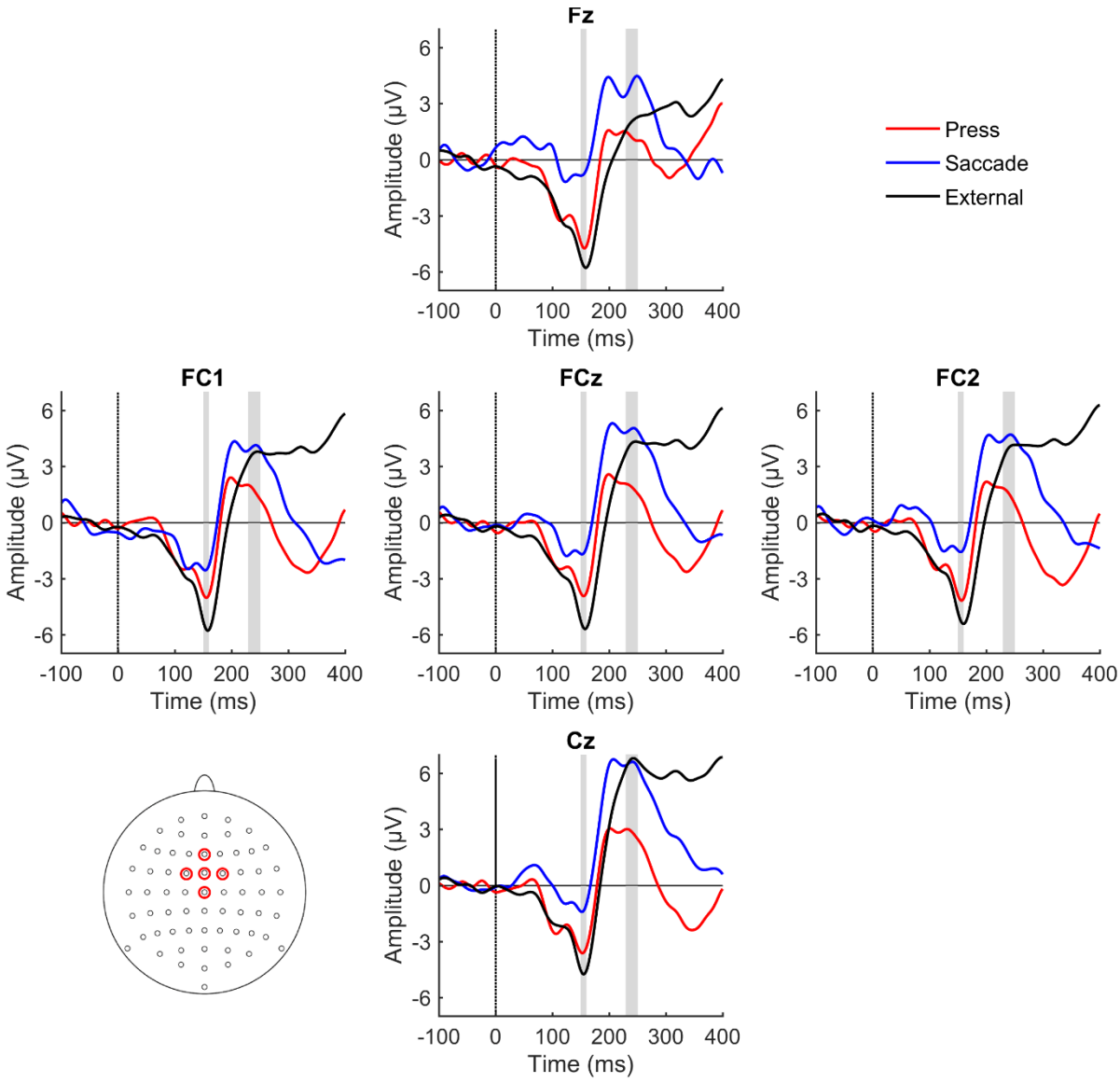

Figure 2. Grand-averaged ERPs for press, saccade, and external conditions at electrodes Fz, FC1, FCz, FC2, and Cz. Self-initiated conditions (i.e., press and saccade) are motor-corrected. The x-axes represent time in milliseconds (ms) where 0 is flash onset, and the y-axes represent amplitude in microvolts (μV). Grey areas indicate measurement windows.

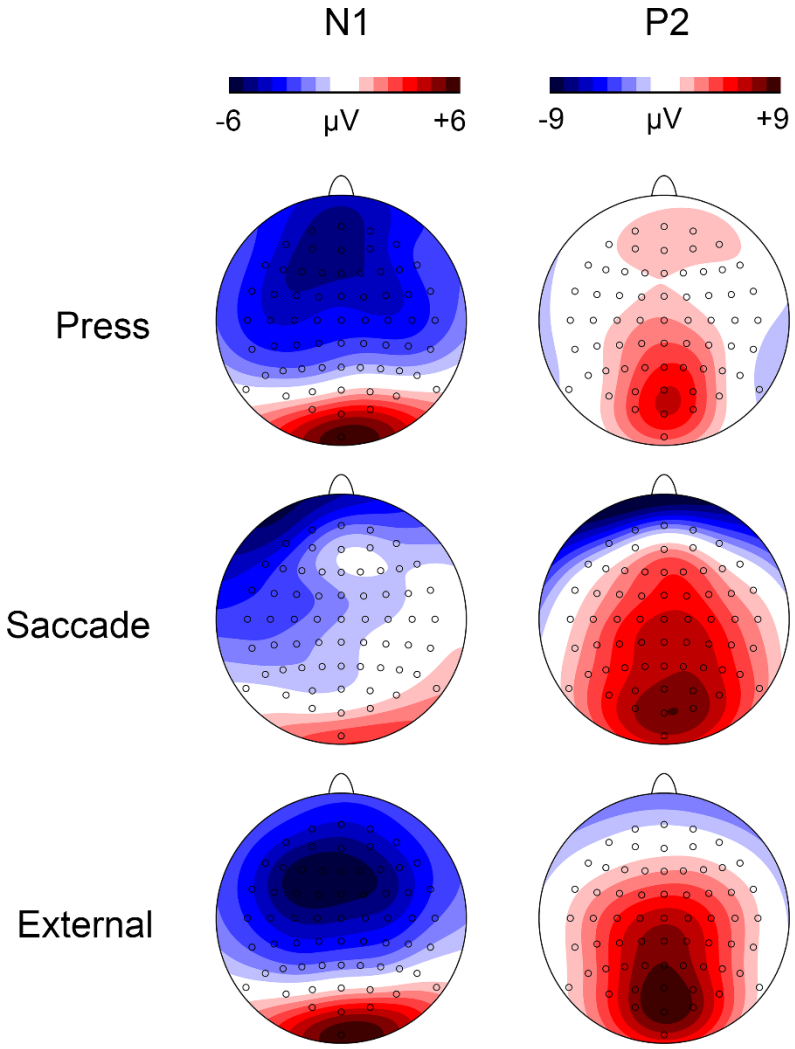

Figure 3. Topographic maps of the visual N1 (150 to 160 ms) and visual P2 (230 to 250 ms) components for each condition. Self-initiated conditions (i.e., press and saccade) are motor-corrected.

FCz analyses. For the N1 component, a main effect of condition, $F(2,64) = 9.19$, $p < .001$, $\eta_p^2 = .22$, indicated that mean N1 amplitude at electrode FCz differed between the press ($M = -3.88$, $SD = 4.34$), saccade ($M = -1.61$, $SD = 5.10$), and external ($M = -5.59$, $SD = 3.45$) conditions. Follow-up pairwise comparisons indicated that external significantly differed from both press, $F(1,32) = 4.89$, $p = .034$, and saccade, $F(1,32) = 16.54$, $p < .001$, and also that press and saccade significantly differed from each other, $F(1,32) = 4.92$, $p = .034$. For the P2 component, there was not a main effect of condition, $F(2,64) = 3.08$, $p = .053$, $\eta_p^2 = .09$, which indicated that mean P2 amplitude at electrode FCz did not differ between press ($M = 1.86$, $SD = 6.52$), saccade ($M = 4.98$, $SD = 6.53$), and external ($M = 4.20$, $SD = 5.66$) conditions.

Cluster analyses. We also conducted 5-site cluster analyses to determine if the observed effects held across adjacent electrodes. As before, for the N1 component, there was a main effect of condition, $F(2,64) = 10.41$, $p < .001$, $\eta_p^2 = .25$, which indicated that mean N1 amplitude across electrodes Fz, FC1, FCz, FC2, and Cz differed between the press ($M = -4.04$, $SD = 0.67$), saccade ($M = -1.54$, $SD = 0.83$), and external ($M = -5.37$, $SD = 0.59$) conditions. Follow-up pairwise comparisons indicated that external significantly differed from saccade, $F(1,32) = 17.31$, $p < .001$, but not press, $F(1,32) = 3.52$, $p = .070$, and that press and saccade significantly differed from each other, $F(1,32) = 7.52$, $p = .010$. There was neither a main effect of site, $F(4,128) = 1.51$, $p = .223$, $\eta_p^2 = .05$, nor an interaction between condition and site, $F(8,256) = 1.68$, $p = .104$, $\eta_p^2 = .05$.

For the P2 component, collapsing across electrodes Fz, FC1, FCz, FC2, and Cz revealed a main effect of condition, $F(2,64) = 3.42$, $p = .039$, $\eta_p^2 = .10$, which indicated that mean P2 amplitude differed between the press ($M = 1.81$, $SD = 1.02$), saccade ($M = 4.86$, $SD = 1.05$), and external ($M = 4.11$, $SD = 0.96$) conditions. Follow-up pairwise comparisons indicated that external did not differ from either press, $F(1,32) = 3.46$, $p = .072$, or saccade,

$F(1,32) = 0.49$, $p = .490$, but that press and saccade significantly differed from each other, $F(1,32) = 5.29$, $p = .028$. There was a significant main effect of site, $F(4,128) = 11.11$, $p < .001$, $\eta_p^2 = .29$, but not an interaction between condition and site, $F(8,256) = 1.96$, $p = .137$, $\eta_p^2 = .06$.

Discussion

The present study investigated the neurophysiological responses to visual flashes which healthy participants initiated by either a button-press or volitional eye movement. We showed that compared to both button-press and externally initiated flashes, saccade-initiated flashes evoked significantly reduced visual N1 amplitude across frontocentral sites centered on FCz, where N1 amplitude was maximal. As predicted, we also showed that button-press initiation led to significantly reduced N1 amplitude compared to external initiation, representing a clear intermediary between the saccade and external conditions. For the visual P2, amplitude was reduced at frontocentral sites following button-press initiation compared to saccade initiation, with no difference between saccade and external conditions. Thus, the present study demonstrates that VEP amplitude attenuation occurred following both button-press and saccade initiation for the visual N1 component, and only for button-press initiation at the visual P2 component. We will compare these outcomes in turn to the existing literature on neurophysiological attenuation of sensory response, with a focus on how the effects of self-initiation differ by type of motor action.

The finding that visual N1 response was attenuated for saccade-initiated flashes compared to externally initiated flashes is highly novel, as our study represents the first saccadic paradigm in the context of investigating the effect of self-initiation on subsequent visual response. That is, while existing saccadic research has focused on the physiological effects around the time of saccadic onset, principally saccadic production and control (e.g., Reingold & Stampe, 2002), the present study focused on the sensory consequences cued by

the eye movement, rather than the movement in itself. With respect to our observation of attenuated visual N1 response at frontocentral sites following button-press initiation, previous studies which measured the anterior N1 component have reported similar findings: Schafer and Marcus (1973) and Gentsch and Schütz-Bosbach (2011) both found reduction of the visual N1 at electrode Cz under similar circumstances. Indeed, Schafer and Marcus (1973) had very similar, though briefer, stimuli (a bright flash of 10-µsec duration). Of key interest, then, is the relative degrees of attenuation observed in each condition.

Given that the N1 attenuation associated with eye movements was greater than the N1 attenuation associated with finger movements (i.e., button-press initiation), even when controlling for between-condition differences in motor-evoked potentials, it is conceivable that the effects observed in the present study relate to the strength of connection between the type of motor action (eye or hand movement) and the resultant perceptual sensations (flashes). That is, the strength of pre-existing associations gained throughout a lifetime of experience is positively correlated with the degree of neurophysiological attenuation. Eye movements are strongly associated with visual sensations, whereas hand movements are only sometimes related to changes in visual sensation. This accords with the pattern of effects observed by Mifsud, Beesley, et al. (2016), where saccade-initiated tones produced *less* auditory N1 attenuation than button-press initiated tones, perhaps because eye movements are less likely than hand movements to be associated with auditory feedback. Relatedly, the forward model account of sensory attenuation—in which predicted sensory consequences in the form of physical "corollary discharge" signals suppress actual sensory feedback—is likely to be most efficacious where there are direct neural connections between the relevant areas of the brain. Such connections conceivably exist between the motor area of the brain involved in eye movements and the visual cortex (e.g., the frontal eye field in prefrontal cortex, see Schall, 2002), whereas it is perhaps less likely that such established connections

exist between the visual cortex and the parts of the motor cortex involved in hand movements.

Regarding later processing of the flash stimuli, button-press initiation was associated with reduced visual P2 amplitude compared to saccade initiation, which did not differ from externally initiated stimuli. Interestingly, the pattern of effects reversed between N1 and P2, with saccade initiation more attenuated at N1, and button-press initiation more attenuated at P2. The significance of this may depend upon the functional dissociation that can be made between visual ERP components. In the auditory domain, the auditory N1 (at least, its supra-temporal subcomponent) is typically associated with sensory processing originating from the auditory cortex (Horváth, 2015), whereas the auditory P2 may reflect cognitive processes such as perceptual learning (Tremblay, Ross, Inoue, McClannahan, & Collet, 2014). No such clear distinction presently exists for visual components. The reduction of visual N1 is likely not due to early visual processing, given the invariance of the anterior N1 effect to retinotopic stimulus properties, as mentioned earlier (Clark et al., 1995). In terms of the visual P2, effect in this latency range are affected by stimulus features (Luck, 2012) and higher-level stimulus features (e.g., faces and houses) have been shown to produce late modulation of potential neurophysiological markers of attenuation (Hughes & Waszak, 2014). However, on the basis of such a small literature, it is difficult to draw any firm conclusions regarding stimulus properties and their effects on the ERP components observed in these procedures. Future directions of merit include a study of occipital effects by using a stimulus which is carefully spatially controlled, and systematic investigations into the effect of stimulus intensity and complexity on the level of sensory attenuation.

In summary, a new saccade initiation paradigm was employed to investigate the sensory attenuation of visual stimuli that were initiated by either a saccade or button-press, introducing a novel procedure to complement the large body of evidence showing sensory

attenuation for auditory stimuli that are initiated by button-press. We observed visual N1 attenuation following saccade initiation, over and above that which occurred following button-press initiation, which we suggest may be related to the strength of the association between eye movements and visual events. This study represents a valuable contribution towards understanding the possible underlying mechanisms that produce the ubiquitous sensory attenuation phenomenon.

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