Biological Psychology 120 (2016) 61–68

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

Attenuation of auditory evoked potentials for hand and eye-initiated sounds

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

ARTICLE INFO

Article history:
Received 16 March 2016
Received in revised form 30 August 2016
Accepted 30 August 2016

Keywords:
Corollary discharge
EEG
N1
Sensory suppression
Saccadic movements

ABSTRACT

Reduction of auditory event-related potentials (ERPs) to self-initiated sounds has been considered evidence for a predictive model in which copies of motor commands suppress sensory representations of incoming stimuli. However, in studies which involve arbitrary auditory stimuli evoked by sensory-unspecific motor actions, learned associations may underlie ERP differences. Here, in a new paradigm, eye motor output generated auditory sensory input, a naïve action–sensation contingency. We measured the electroencephalogram (EEG) of 40 participants exposed to pure tones, which they produced with either a button-press or volitional saccade. We found that button-press-initiated stimuli evoked reduced amplitude compared to externally initiated stimuli for both the N1 and P2 ERP components, whereas saccade-initiated stimuli evoked intermediate attenuation at N1 and no reduction at P2. These results indicate that the motor-to-sensory mapping involved in speech production may be partly generalized to other contingencies, and that learned associations also contribute to the N1 attenuation effect.

1. Introduction

Our brains must regulate a continuous stream of sensory input in order to flexibly generate behaviour and allow interaction with the world. A well-established example of such regulation is sensory attenuation, where the sensory input evoked by self-initiated actions is marked by reduced phenomenological (e.g., Blakemore, Frith, & Wolpert, 1999; Cardoso-Leite, Mamassian, Schütz-Bosbach, & Waszak, 2010; Sato, 2008) and neurophysiological representations (e.g., Baess, Jacobsen, & Schröger, 2008; Houde, Nagarajan, Sekihara, & Merzenich, 2002; Schafer & Marcus, 1973) compared to identical, externally initiated sensory input—a phenomenon typified by the difficulty of tickling ourselves (Weiskrantz, Elliott, & Darlington, 1971). Functionally, sensory attenuation serves to conserve attentional resources and to enable sensory processing in situations where volitional actions would otherwise desensitize sensory receptors, such as during speech production (Bendixen, SanMiguel, & Schröger, 2012). It has also been proposed as fundamental for self-identity, such that dysfunctional attenuation could lead to psychotic symptomology (Feinberg, 1978; Ford et al., 2001).

In the auditory domain, the N1 or N1 m component (an evoked potential or magnetic field which appears approximately 100 ms after the onset of an auditory stimulus) is used as a cortical index of sensory attenuation, because its amplitude, compared to externally initiated stimuli, is consistently reduced for both self-initiated vocalizations (Curio, Neuloh, Numminen, Jousmaki, & Hari, 2000; Heinks-Maldonado, Mathalon, Gray, & Ford, 2005; Houde et al., 2002) and button-press-initiated stimuli (Aliu, Houde, & Nagarajan, 2009; Baess et al., 2008; Martikainen, Kaneko, & Hari, 2005; McCarthy & Donchin, 1976; Schafer & Marcus, 1973; Sowman, Kuusik, & Johnson, 2012). The predominant explanation for these findings invokes a theory of motor control in which a forward model predicts future behavioural states and their sensory consequences (Wolpert, Ghahramani, & Jordan, 1995). According to this theory, the sensory consequences of volitional action can be predicted based on an efference copy (i.e., a copy of the motor command), and sensory attenuation reflects the subtraction of this prediction from actual sensory input (Bays & Wolpert, 2007). Conversely, externally initiated sensory input, for which there is no motor information to form an accurate prediction, will remain unmodulated by the efference copy mechanism (see Timm, SanMiguel, Keil, Schroger, & Schonwiesner, 2014).

Forward prediction is strongly implicated in speech production (Hickok, 2012), which involves a well-defined range of motor output (e.g., shape formed by lips) producing specific, habitual,

* Corresponding author at: School of Psychology, Mathews Building, UNSW Australia, Sydney, NSW 2052, Australia.
E-mail address: nathan.mifsud@unsw.edu.au (N.G. Mifsud).

http://dx.doi.org/10.1016/j.biopsycho.2016.08.011
0301-0511/

sensory consequences (i.e., uttered syllables). As candidate language fibre pathways have been identified (Dick & Tremblay, 2012), upon which the efference copies of speech motor output conceivably travel, there is also a plausible neural basis for forward prediction in speech production. However, the N1 attenuation literature largely comprises experiments based on arbitrary action–sensation contingencies, usually hand movements to press a button and elicit a tone. This is problematic, because it is not clear that motor-to-sensory mapping can be generalized from speech production to auditory input evoked by motor actions unrelated to speech (Horváth, 2015). Unlike speech-induced auditory attenuation, for which there are distinct neural networks proposed to be involved (Behroozmand et al., 2016; Chang, Niziolek, Knight, Nagarajan, & Houde, 2013; Greenlee et al., 2013) which likely encode specific acoustic properties of the upcoming sound, internal predictions arising from non-speech motor actions might be comparatively more crude. This presents the possibility that sensory attenuation of speech and non-speech stimuli are driven by different mechanisms.

An alternate (or perhaps complementary) explanation of sensory attenuation relates to learned associations between actions and sensations. Operationally, learned associations have been proposed to "pre-activate" potential sensory input, increasing baseline neural activity at a greater rate than the signal increase resulting from an incoming stimulus (Roussel, Hughes, & Waszak, 2013). This account differs from forward prediction in that sensory attenuation is attributed to poorer stimulus discrimination (and consequent reduction of the corresponding sensory representations) rather than an efference copy directly reducing the internal response to a predicted stimulus. An associative explanation can be reasonably applied to the classic paradigm of pressing a button to hear an auditory stimulus, given that such a contingency conceivably draws upon a wealth of pre-existing action–sensation associations that have been experienced and learnt over the course of a lifetime. Indeed, there is evidence to suggest that prior experience can affect sound perception (Repp & Knoblich, 2007), and some suggestion that contingency strength alters neurophysiological response in the auditory (Baess et al., 2008; SanMiguel, Widmann, Bendixen, Trujillo-Barreto, & Schröger, 2013) and visual (Roussel, Hughes, & Waszak, 2014) domains (see Horváth, 2015 for a discussion). Hence, efference copy modulation may not be wholly responsible for all cases of sensory attenuation, and an account based on learned associations may explain observed effects in contingencies that lack the highly specific motor-to-sensory mapping that exists during speech production.

One possible test of the contribution of learned associations would be to employ a novel contingency (i.e., one never experienced before) between motor output and sensory input. For example, volitional eye movements cannot directly cause sounds in our natural environment, and therefore it seems implausible that an efference copy of the eye motor command would be sent to the auditory cortex. Hence, for a contingency between an eye movement and an auditory stimulus, it is almost certain that no learned association exists, which provides a strong test of whether learned associations are an essential component of sensory attenuation. Moreover, if neurophysiological auditory attenuation were still observed for this novel contingency, it implies that motor-to-sensory mapping is indeed generalizable to indirect action–sensation contingencies. This result would support the forward prediction model of sensory attenuation. However, if sensory attenuation was not observed, a limit for generalizability will have been identified, which might suggest that learned associations are driving button-press-elicited auditory N1 attenuation. Alternatively, sensory attenuation may be driven by a combination of these processes; for example, it seems possible that forward prediction could be established for novel contingencies, such that levels of N1 attenuation are mediated by associative strength.

Accordingly, the primary aim of the present study was to determine whether auditory event-related potential (ERP) attenuation, particularly with respect to the N1 component, would occur following an action–sensation contingency for which no prior learning exists, and thus provide a valuable contribution to the discussion about the processes underlying sensory attenuation. To do so, our experimental design contained a new condition which associated eye movements (the motor output) with tones (the auditory sensory input), a pairing which cannot occur outside of artificial contexts, and for which it is difficult to imagine an analogous, naturally occurring pairing of events. Specifically, participants were presented a pure tone (as is common in auditory ERP studies of this nature, e.g., Baess et al., 2008) following a singular, volitional saccadic movement. To determine whether N1 attenuation occurred, their resultant electrophysiological response, following subtraction of a motor condition (i.e., the same eye movement without stimulus presentation), was compared to an externally initiated condition in which tones were presented without any participant input. Given the possibility raised above that a combination of efference copy and associative processes drive sensory attenuation, we expected to observe N1 attenuation for saccade-initiated stimuli compared to the externally initiated condition. Even so, it seemed unlikely that a novel contingency could produce the same level of attenuation as previously seen in button-press-initiated experiments, as we have substantial prior experience with auditory sensory input following hand motor output.

In view of this hypothesis, our secondary aim was to quantitatively compare self-initiation effects associated with different regions of motor output (i.e., hand and eye). To achieve this aim, our within-subjects design also included an established button-press-initiated condition, for which convincing N1 and P2 attenuation has been demonstrated (Mifsud et al., 2016; Oestreich et al., 2016; Whitford et al., 2011). We chose to additionally assess the P2 component (a large voltage positivity which peaks approximately 200 ms after stimulus onset), which reflects the processing of specific auditory features (Shahin, Roberts, Pantev, Trainor, & Ross, 2005), for two reasons. First, SanMiguel, Todd, and Schröger (2013) have suggested that it may provide a more direct measure of sensory-specific prediction effects than the N1, because, unlike N1 effects, P2 attenuation was uniform over different stimulus onset asynchronies. Hence, it seems prudent to report P2 effects so as to enable comparison between different paradigms. Second, the P2 component has previously been shown to discriminate between self-initiated conditions which differ by motor output region (i.e., hand and foot; van Elk, Salomon, Kannape, & Blanke, 2014), if not necessarily contingency strength (that is, we have substantive experience with both hand and feet producing auditory stimulation), and is therefore relevant given the disparate eye and hand regions targeted in the present study.

2. Method

2.1. Participants

Forty participants were recruited using an online experiment management system at UNSW Australia. There were 18 females, 36 were right-handed, and mean age was 23 years ($SD = 7$). Participants provided written, informed consent and received either course credit ($n = 24$) or financial imbursement ($n = 16$, A$30) in exchange for their time. This study was approved by the UNSW Human Research Ethics Advisory Panel (Psychology).

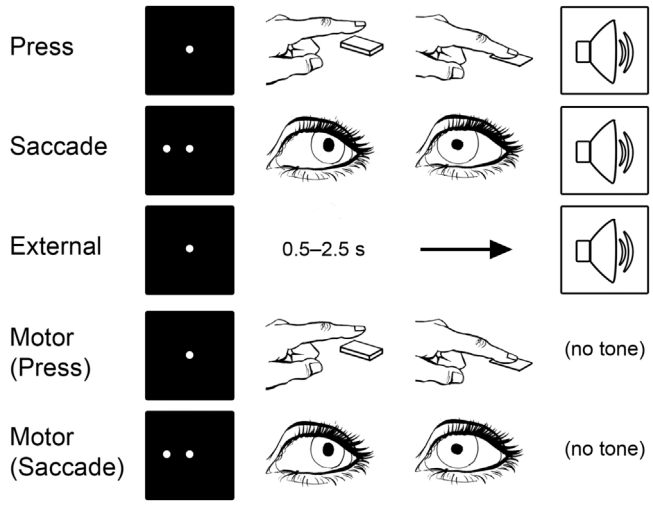

Fig. 1. Experimental protocol. In the press-initiation condition, participants depressed a button at will any time after a fixation dot appeared. Immediately following the button-press, a tone was delivered. In the saccade-initiated condition, participants focused on a distal dot and then shifted at will to the center fixation dot, which immediately delivered a tone. In the externally initiated condition, tone delivery followed a variable delay without motor input. Lastly, control conditions were identical to their respective stimulus conditions, except that motor input did not result in tone delivery.

2.2. Procedure

Participants sat in a quiet, artificially lit room, 60 cm in front of 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). Following a demographics questionnaire, participants were fitted with the EEG cap and electrodes, and underwent a 5-point eye tracking calibration procedure. EEG was then continuously recorded while participants completed an experimental protocol (see Fig. 1) controlled by MATLAB (MathWorks, Natick, MA).

The experiment consisted of five conditions: two types of self-initiation (i.e., button-press and saccade), two corresponding motor control conditions (i.e., button-presses and saccades without consequent tones), and an externally initiated condition (i.e., tones played automatically). These conditions are described in detail below. Each condition was presented in a homogenous 80-trial block, and block order was randomized between participants. Each block was preceded by 3 practice trials to ensure participants understood instructions, and, in the self-initiated conditions, to encourage self-paced responses. Individual trials were always separated by a uniformly distributed random interval (2–4 s). The EEG recording lasted approximately 50 min, including three additional blocks intermixed with those presented here. These additional blocks contained self- and externally initiated visual stimuli, and will be presented in a forthcoming paper, as a discussion here of the visual ERP literature is precluded by space constraints.

2.3. Press condition

This self-initiated condition measured electrophysiological response to an auditory stimulus produced 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 with their dominant hand. Responses immediately delivered a tone (30 ms duration, 500 Hz frequency, 70 dB sound pressure level) to their headphones (AKG K77 Perception).

2.4. Saccade condition

This self-initiated condition measured electrophysiological response to an auditory stimulus produced 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 s, trials were skipped with replacement ($M = 0.8$ 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 tone to the headphones (identical to the press-initiated tone). More precisely, tone delivery followed detection of the gaze within the 200-px (5°) square area of interest surrounding the central red circle.

2.5. 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, as is standard practice in studies of this nature (Baess et al., 2008; Martikainen et al., 2005; Whitford et al., 2011), or with the singular, volitional eye movement associated with the saccade-initiated procedure.

2.6. External condition

In this condition, auditory stimuli were delivered 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), after which a tone was delivered to the headphones (identical to that in the self-initiated conditions). Participants were instructed to keep their eyes open and maintain their gaze on the screen.

2.7. 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 3600 Hz) and continuously digitized at a sampling frequency of 2048 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, band-pass filtered from 0.01 to 30 Hz (8th 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 4.2% ± 5.4% (*SD*) trials, with no significant differences between stimulus blocks. We then averaged individual trials for each condition to produce ERPs for each participant. At this point, a headphone-induced artefact (a solitary spike around

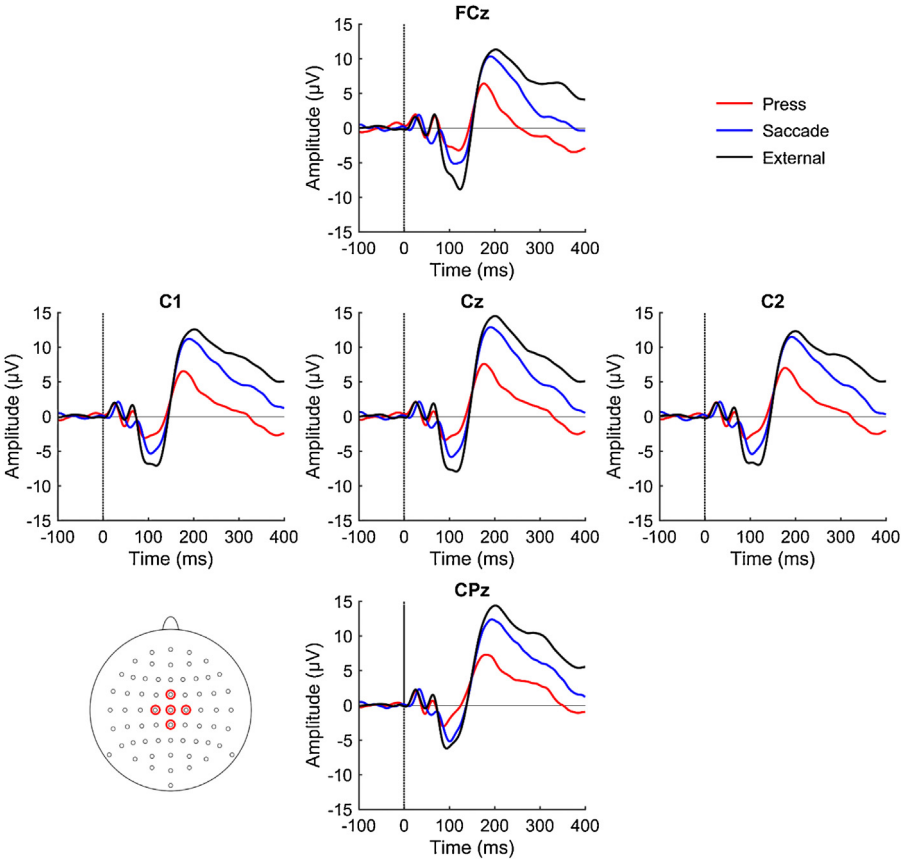

Fig. 2. Grand-averaged ERPs for press, saccade, and external conditions at electrodes FCz, C1, Cz, C2, and CPz. Self-initiated conditions (i.e., press and saccade) are motor-corrected. The x-axes represent time in milliseconds (ms) where tone onset is at 0 ms, and the y-axes represent amplitude in microvolts (μV).

30 ms after tone onset) was removed from 7 participants using independent component analysis (ICA) with the FastICA algorithm (Hyvarinen & Oja, 2000). Lastly, motor waveforms were subtracted from the appropriate self-initiated waveforms to produce difference waveforms, as is typical in other button-press-initiated self-suppression studies (Baess et al., 2008; Martikainen et al., 2005; Whitford et al., 2011) and which has been extended here to our saccadic initiation paradigm. 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).

3. Results

Fig. 2 shows the grand-averaged ERPs at electrode Cz and its neighbouring electrodes (i.e., FCz, C1, C2, and CPz), which represent locations at which the N1 and P2 components are typically maximal, especially subsequent to bilateral auditory stimulation (Ford, Gray, Faustman, Roach, & Mathalon, 2007; Luck, 2012; Näätänen & Picton, 1987). In addition, Fig. 3 shows a complete view of scalp activity during the component windows for each stimulus condition. We conducted separate one-way repeated-measures analyses of variance (ANOVA) to determine the effect of condition (three levels: press-initiated, saccade-initially, and externally initiated) on the mean amplitudes of the N1 (100–110 ms) and P2 (180–200 ms) at electrode Cz.

3.1. N1 (100–110 ms)

There was a main effect of condition for the N1 component, $F(2,78) = 13.85$, $p < 0.001$, $\eta_p^2 = 0.26$, which indicated that mean N1 amplitude at electrode Cz differed between press ($M = -2.81$, $SD = 7.43$), saccade ($M = -5.79$, $SD = 7.30$), and external ($M = -7.73$, $SD = 6.99$) conditions. Follow-up pairwise comparisons indicated that external significantly differed from both press, $F(1,39) = 29.89$, $p < 0.001$, and saccade, $F(1,39) = 4.78$, $p = 0.035$. Additionally, press and saccade significantly differed from each other, $F(1,39) = 8.35$, $p = 0.006$. These outcomes were underscored by a highly significant linear trend for N1 mean amplitudes across the conditions ($F = 29.88$, $p < 0.001$, $\eta_p^2 = 0.43$).

3.2. P2 (180–200 ms)

There was also a main effect of condition for the P2 component, $F(2,78) = 19.85$, $p < 0.001$, $\eta_p^2 = 0.34$, which indicated that mean P2 amplitude at electrode Cz differed between press ($M = 6.78$, $SD = 6.12$), saccade ($M = 12.76$, $SD = 7.79$), and external ($M = 14.09$, $SD = 7.14$) conditions. Follow-up pairwise comparisons indicated that external significantly differed from press, $F(1,39) = 36.86$, $p < 0.001$, but did not differ from saccade, $F(1,39) = 1.40$, $p = 0.244$. However, press and saccade significantly differed from each other, $F(1,39) = 19.20$, $p < 0.001$. As for N1, there was a highly significant linear trend for P2 mean amplitudes across the conditions ($F = 36.88$, $p < 0.001$, $\eta_p^2 = 0.49$).

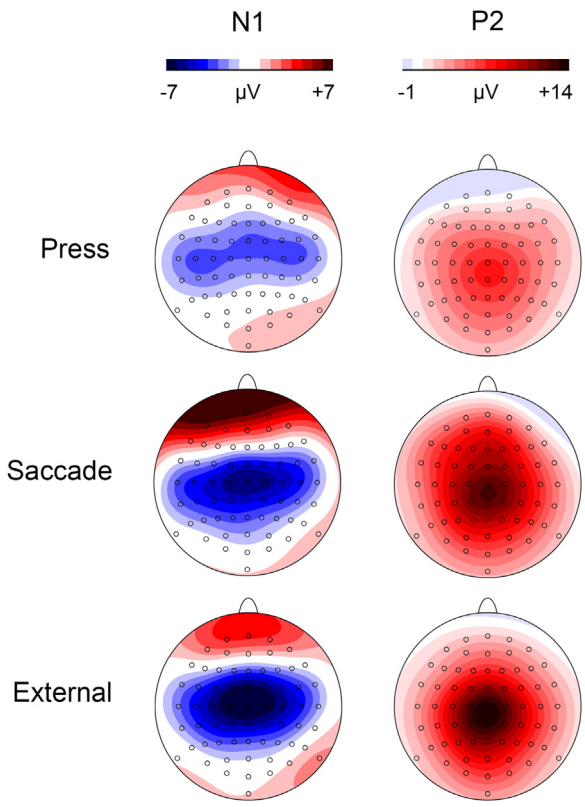

Fig. 3. Topographic maps of the N1 (100–110 ms) and P2 (180–200 ms) components for each condition. Self-initiated conditions (i.e., press and saccade) are motor-corrected.

3.3. Uncorrected waveforms

The effect of the motor subtraction procedure can be seen in Fig. 4, which presents grand-averaged ERPs at electrode Cz and its neighbours (i.e., FCz, C1, C2, and CPz) for the uncorrected self-initiated waveforms and their corresponding motor waveforms. This illustrates that the motor subtraction in fact reduced the N1 difference between button-press and saccade-initiated conditions. Fig. 5 shows scalp activity during the component windows for each uncorrected stimulus condition and its motor control.

For the N1 component, a one-way ANOVA using the uncorrected waveforms revealed a main effect of condition, $F(2,78) = 35.38$, $p < 0.001$, $\eta_p^2 = 0.48$, which indicated that mean N1 amplitude at electrode Cz differed between press ($M = -0.80$, $SD = 7.88$), saccade ($M = -7.22$, $SD = 6.38$), and external ($M = -7.73$, $SD = 6.99$) conditions. Follow-up pairwise comparisons indicated that external significantly differed from press, $F(1,39) = 51.69$, $p < 0.001$, but not saccade, $F(1,39) = 0.39$, $p = 0.535$, and that press and saccade significantly differed from each other, $F(1,39) = 44.84$, $p < 0.001$. There was also a main effect for the P2 component, $F(2,78) = 5.68$, $p = 0.005$, $\eta_p^2 = 0.13$, which indicated that mean P2 amplitude at electrode Cz differed between press ($M = 10.70$, $SD = 6.76$), saccade ($M = 11.62$, $SD = 6.27$), and external ($M = 14.09$, $SD = 7.14$) conditions. Follow-up pairwise comparisons indicated that external significantly differed from both press, $F(1,39) = 8.63$, $p = 0.006$, and saccade, $F(1,39) = 8.54$, $p = 0.006$, but that press and saccade did not differ from each other, $F(1,39) = 0.71$, $p = 0.405$.

4. Discussion

The present study investigated sensory attenuation of self-initiated stimuli in terms of the effect of motor output region (i.e., hand or eye actions) on neurophysiological response to identical contingent sensory input (i.e., auditory tones). We found that button-press-initiated stimuli evoked significantly reduced N1 and P2 component amplitude compared to both saccade- and externally initiated stimuli. In the saccade-initiated condition, we observed an intermediary level of N1 attenuation, that is, a significant reduction compared to the externally initiated condition, but significantly less reduction than in the button-press-initiated condition. In contrast, there was no difference between the saccade- and externally initiated conditions in terms of their P2 component amplitude. Hence, in relation to our primary aim, which was to determine whether ERP attenuation would occur following a novel action–sensation contingency (i.e., saccade–tone), the data indicates that it does at the N1 component, but not the P2 component. We will discuss the possible implications of these outcomes in turn.

Our finding that button-press-initiation was associated with significant N1 and P2 attenuation was expected, as it replicates previous iterations of a similar paradigm (Mifsud et al., 2016; Oestreich et al., 2016; Whitford et al., 2011) and aligns with data from several other research groups (e.g., Aliu et al., 2009; Knolle, Schröger, Baess, & Kotz, 2012; Sowman et al., 2012). More crucially in terms of our experimental rationale was the new finding that N1 attenuation also occurred in the saccade-initiated condition, despite participants' lack of prior experience with the novel saccade–tone contingency. This suggests that sensory attenuation does not rely upon learned associations, and potentially that forward prediction can operate for any combination of motor and sensory events. The finding that saccade-initiated N1 attenuation was not as strong as button-press-initiated N1 attenuation could suggest that learned associations contribute to the size of N1 attenuation in button-press paradigms, or it may be that optimal parameters for saccade initiation are still to be determined. Studies which introduce a wider range of action–sensation contingencies than are presently investigated could provide a clearer indication of the relationship between existing learned associations on sensory attenuation of self-initiated stimuli. Based on the results of the present study, one might hypothesise a positive relationship between N1 attenuation and the existing strength of action–sensation contingencies. As such, an action which has consistently resulted in a given sound over the course of a person's life would be expected to elicit a high degree of N1 attenuation.

For the P2 component, saccade-initiated stimuli did not differ from externally initiated stimuli, which diverges from the press-initiated outcome and indicates that the P2 is sensitive to a property of the motor output region. It has been suggested that P2 attenuation may more directly correlate with 'contingent temporal certainty' (Sowman et al., 2012) or 'sensory-specific predictions' (SanMiguel, Todd et al., 2013) than N1 attenuation. Such factors are more strongly implicated by button-press initiation than saccade initiation, given the greater practice we have with auditory stimulation following button-presses as opposed to saccades, which may explain the present pattern of results. This is similar to the speculation provided by van Elk et al. (2014) in explaining the lack of P2 modulation for feet-initiated compared to hand-initiated stimuli, who argued that associations between feet actions and subsequent sounds are less established than for hands. However, direct evidence for this line of reasoning remains scarce. Shahin et al. (2005) showed that P2 (but not N1) response to tones was enhanced for pianists, and when the tones became more spectrally complex, which indicates that the P2 reflects the specific features of acoustic stimuli. To properly link this study to sensory attenuation, it would be useful to see if there is an inverse pattern for self-initiated tones (i.e., that attenuation increases with spectral complexity). Moreover, in any attempt to functionally dissociate N1 and P2, it is important to consider pertinent research in the speech domain. For instance, Houde and Chang (2015) recently accounted for differences between N1 and P2 response using the state feedback

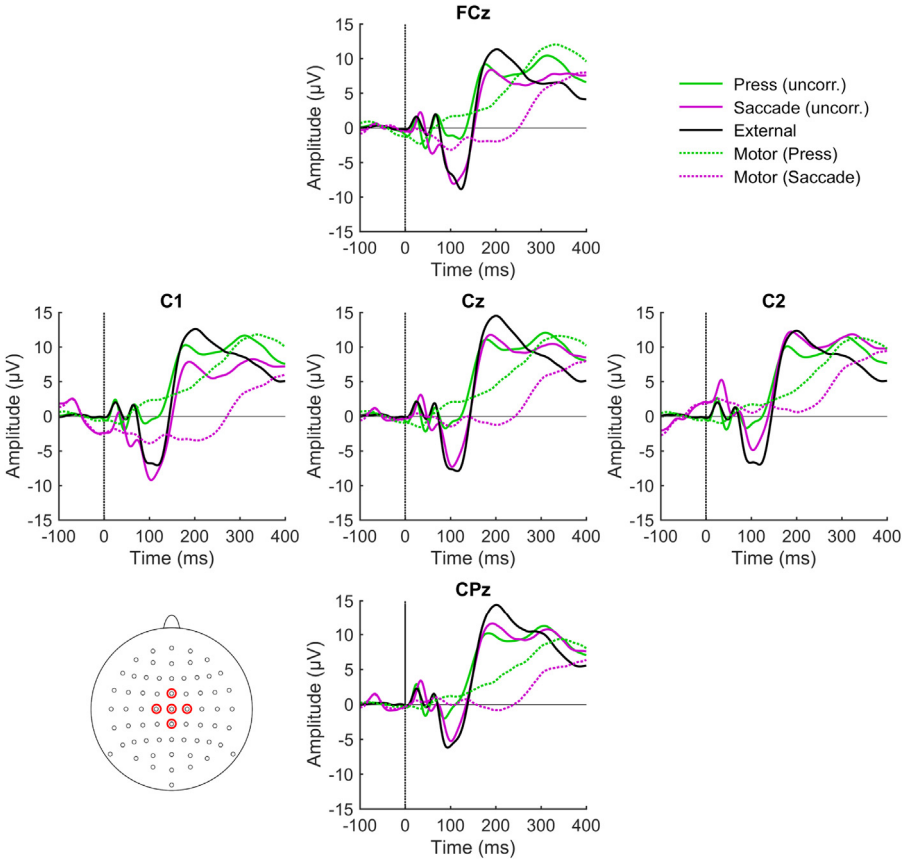

Fig. 4. Grand-averaged ERPs at electrodes FCz, C1, Cz, C2, and CPz for uncorrected self-initiated conditions (solid traces) and their corresponding motor conditions (dotted traces). The x-axes represent time in milliseconds (ms) where tone onset is at 0 ms, and the y-axes represent amplitude in microvolts (μV).

control (SFC) model of speaking. As mentioned earlier, discordance in the pattern of ERP response following speech and non-speech motor actions seems related to the likelihood that predictions of the responsible neural mechanisms differ in their respective degrees of specificity.

As the saccade initiation paradigm presented in this paper is new, we acknowledge that the motor subtraction procedure, standard practice in contingent button-press studies investigating sensory attenuation (e.g., Baess et al., 2008; Martikainen et al., 2005; Whitford et al., 2011), has not been previously applied to eye movements. A direct investigation into possible differences in motor activity between action-only and action-response conditions for both button-press and saccade-initiations would be worthwhile. Nonetheless, it is crucial to highlight that in the present study, button-press and saccade-initiated conditions significantly differed even when uncorrected for motor activity (see Fig. 4). That is, motor subtraction *reduced* the N1 difference between button-press and saccade-initiated conditions, suggesting that there are true N1 attenuation differences related to motor output region.

An explicit assumption so far in this discussion has been that N1 and P2 differences between motor output conditions may be only ascribed to the presence (or lack) of either forward prediction or learned associations, but it should be noted that other

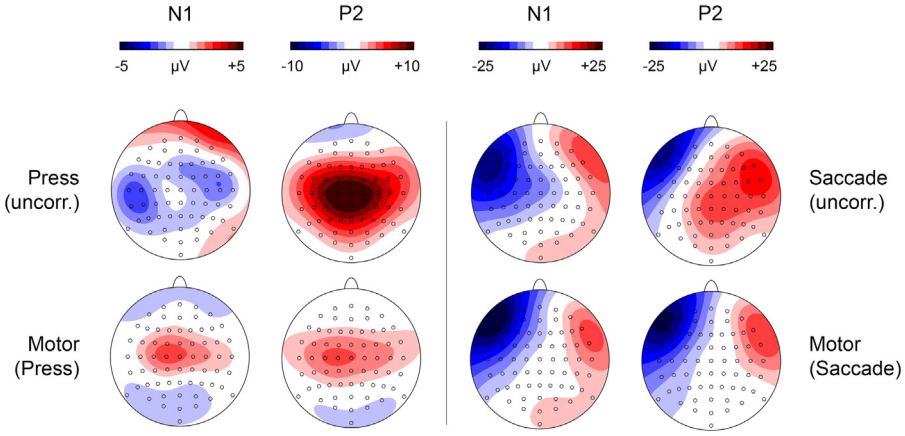

Fig. 5. Topographic maps of the N1 (100–110 ms) and P2 (180–200 ms) component windows for the uncorrected stimulus conditions (Press, Saccade) and their motor controls.

contributory factors are feasible. For instance, exogenous orienting effects may play a role if differences in the strength of learned associations produce differences in the degree to which stimuli are expected (Santangelo, Olivetti Belardinelli, & Spence, 2007). It is also possible that using a saccadic movement to investigate sensory attenuation poses an issue due to the existence of saccadic suppression, a compensatory mechanism classically grounded in forward prediction (Sperry, 1950; Von Holst, 1954), in which the retinal consequences of eye movements are suppressed in order to maintain stability of the visual field. However, exactly why saccadic suppression would have a substantial effect on auditory perception seems unclear. Outside of the laboratory, there are no situations in which auditory consequences directly arise due to saccades. Even beyond saccades, eye movements as a whole produce only visual, and, rarely, social consequences (e.g. staring, eye rolling). If eye movements were to attenuate sensory perception in non-visual modalities, there would be a persistent, resource-intensive source of usually irrelevant suppression in the sensory system. In any case, visual suppression has been shown to disappear within ~50 ms post-saccade (Diamond, Ross, & Morrone, 2000), prior to the time window of the auditory components examined in this paper, further diminishing the likelihood of non-visual modulation. Moreover, in a multimodal localization task, Binda, Bruno, Burr, and Morrone (2007) found that due to blurring during saccades, auditory rather than visual signals were accorded heavier weight. This suggests that if saccades were to affect auditory perception, one would expect the auditory signal to be *enhanced*. That we found the opposite effect indicates that the saccade *per se* is not causing N1 attenuation.

The present study presented a new saccade-initiation paradigm, which demonstrated that auditory N1 attenuation occurs even for an action–sensation contingency for which no prior learning exists. We suggest that sensory-specific forward prediction is an essential component of sensory attenuation, but as saccade-initiated N1 attenuation was less severe than in button-press-initiated N1 attenuation, that learned associations are likely to also play a role. Moreover, we have provided support for the notion that the processes contributing to N1 and P2 effects may be independent, but the specific identity and functionality of these underlying processes, particularly P2, remains an open question.

Acknowledgements

Nathan G. Mifsud is supported by an Australian Postgraduate Award. Thomas J. Whitford is supported by a Career Development Fellowship from the National Health and Medical Research Council of Australia (APP1090507), a Discovery Project from the Australian Research Council (DP140104394), and a Young Investigator Award (Barbara and John Streicker Investigator) from the NARSAD Brain and Behavior Research Foundation (17537).

References

Aliu, S. O., Houde, J. F., & Nagarajan, S. S. (2009). Motor-induced suppression of the auditory cortex. *Journal of Cognitive Neuroscience*, *21*(4), 791–802. http://dx.doi.org/10.1162/jocn.2009.21055

Baess, P., Jacobsen, T., & Schröger, E. (2008). Suppression of the auditory N1 event-related potential component with unpredictable self-initiated tones: Evidence for internal forward models with dynamic stimulation. *International Journal of Psychophysiology*, *70*(2), 137–143. http://dx.doi.org/10.1016/j.ijpsycho.2008.06.005

Bays, P. M., & Wolpert, D. M. (2007). Computational principles of sensorimotor control that minimize uncertainty and variability. *Journal of Physiology*, *578*, 387–396. http://dx.doi.org/10.1113/jphysiol.2006.120121 [Pt 2]

Behroozmand, R., Oya, H., Nourski, K. V., Kawasaki, H., Larson, C. R., Brugge, J. F., & Greenlee, J. D. (2016). Neural correlates of vocal production and motor control in human heschl's gyrus. *Journal of Neuroscience*, *36*(7), 2302–2315. http://dx.doi.org/10.1523/JNEUROSCI. 3305-14.2016

Bendixen, A., SanMiguel, I., & Schröger, E. (2012). Early electrophysiological indicators for predictive processing in audition: A review. *International Journal of Psychophysiology*, *83*(2), 120–131. http://dx.doi.org/10.1016/j.ijpsycho.2011.08.003

Binda, P., Bruno, A., Burr, D. C., & Morrone, M. C. (2007). Fusion of visual and auditory stimuli during saccades: A Bayesian explanation for perisaccadic distortions. *Journal of Neuroscience*, *27*(32), 8525–8532. http://dx.doi.org/10.1523/JNEUROSCI.0737-07.2007

Blakemore, S. J., Frith, C. D., & Wolpert, D. M. (1999). Spatio-temporal prediction modulates the perception of self-produced stimuli. *Journal of Cognitive Neuroscience*, *11*(5), 551–559. http://dx.doi.org/10.1162/089892999563607

Cardoso-Leite, P., Mamassian, P., Schütz-Bosbach, S., & Waszak, F. (2010). A new look at sensory attenuation. Action-effect anticipation affects sensitivity, not response bias. *Psychological Science*, *21*(12), 1740–1745. http://dx.doi.org/10.1177/0956797610389187

Chang, E. F., Niziolek, C. A., Knight, R. T., Nagarajan, S. S., & Houde, J. F. (2013). Human cortical sensorimotor network underlying feedback control of vocal pitch. *Proceedings of the National Academy of Sciences of the United States of America*, *110*(7), 2653–2658. http://dx.doi.org/10.1073/pnas.1216827110

Curio, G., Neuloh, G., Numminen, J., Jousmaki, V., & Hari, R. (2000). Speaking modifies voice-evoked activity in the human auditory cortex. *Human Brain Mapping*, *9*(4), 183–191. http://dx.doi.org/10.1002/(SICI)1097-0193(200004)9:4<183::AID-HBM1>3.0.CO;2-Z

Diamond, M. R., Ross, J., & Morrone, M. C. (2000). Extraretinal control of saccadic suppression. *Journal of Neuroscience*, *20*(9), 3449–3455.

Dick, A. S., & Tremblay, P. (2012). Beyond the arcuate fasciculus: Consensus and controversy in the connectional anatomy of language. *Brain*, *135*(12), 3529–3550. http://dx.doi.org/10.1093/brain/aws222

Feinberg, I. (1978). Efference copy and corollary discharge: Implications for thinking and its disorders. *Schizophrenia Bulletin*, *4*, 636–640. http://dx.doi.org/10.1093/schbul/4.4.636

Ford, J. M., Mathalon, D. H., Heinks, T., Kalba, S., Faustman, W. O., & Roth, W. T. (2001). Neurophysiological evidence of corollary discharge dysfunction in schizophrenia. *American Journal of Psychiatry*, *158*(12), 2069–2071. http://dx.doi.org/10.1176/appi.ajp.158.12.2069

Ford, J. M., Gray, M., Faustman, W. O., Roach, B. J., & Mathalon, D. H. (2007). Dissecting corollary discharge dysfunction in schizophrenia. *Psychophysiology*, *44*(4), 522–529. http://dx.doi.org/10.1111/j. 1469-8986.2007.00533.x

Greenlee, J. D., Behroozmand, R., Larson, C. R., Jackson, A. W., Chen, F., Hansen, D. R., & Howard. (2013). Sensory-motor interactions for vocal pitch monitoring in non-primary human auditory cortex. *Public Library Of Science*, *8*(4), e60783. http://dx.doi.org/10.1371/journal.pone.0060783

Heinks-Maldonado, T. H., Mathalon, D. H., Gray, M., & Ford, J. M. (2005). Fine-tuning of auditory cortex during speech production. *Psychophysiology*, *42*(2), 180–190. http://dx.doi.org/10.1111/j. 1469-8986.2005.00272.x

Hickok, G. (2012). Computational neuroanatomy of speech production. *Nature Reviews Neuroscience*, *13*(2), 135–145. http://dx.doi.org/10.1038/nrn3158

Horváth, J. (2015). Action-related auditory ERP attenuation: Paradigms and hypotheses. *Brain Research*, *1626* http://dx.doi.org/10.1016/j.brainres.2015.03.038

Houde, J. F., & Chang, E. F. (2015). The cortical computations underlying feedback control in vocal production. *Current Opinion in Neurobiology*, *33*, 174–181. http://dx.doi.org/10.1016/j.conb.2015.04.006

Houde, J. F., Nagarajan, S. S., Sekihara, K., & Merzenich, M. M. (2002). Modulation of the auditory cortex during speech: An MEG study. *Journal of Cognitive Neuroscience*, *14*(8), 1125–1138. http://dx.doi.org/10.1162/089892902760807140

Hyvarinen, A., & Oja, E. (2000). Independent component analysis: Algorithms and applications. *Neural Networks*, *13*(4-5), 411–430. http://dx.doi.org/10.1016/S0893-6080(00)00026-5

Knolle, F., Schröger, E., Baess, P., & Kotz, S. A. (2012). The cerebellum generates motor-to-auditory predictions: ERP lesion evidence. *Journal of Cognitive Neuroscience*, *24*(3), 698–706. http://dx.doi.org/10.1162/jocn_a_00167

Luck, S. J. (2012). *An introduction to the event-related potential technique* (2nd ed.). Cambridge, MA: The MIT Press.

Martikainen, M. H., Kaneko, K., & Hari, R. (2005). Suppressed responses to self-triggered sounds in the human auditory cortex. *Cerebral Cortex*, *15*(3), 299–302. http://dx.doi.org/10.1093/cercor/bhh131

McCarthy, G., & Donchin, E. (1976). The effects of temporal and event uncertainty in determining the waveforms of the auditory event related potential (ERP). *Psychophysiology*, *13*(6), 581–590. http://dx.doi.org/10.1111/j. 1469-8986.1976. tb00885.x

Mifsud, N. G., Oestreich, L. K., Jack, B. N., Ford, J. M., Roach, B. J., Mathalon, D. H., & Whitford, T. J. (2016). Self-initiated actions result in suppressed auditory but amplified visual evoked components in healthy participants. *Psychophysiology*, http://dx.doi.org/10.1111/psyp.12605

Näätänen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology*, *24*(4), 375–425. http://dx.doi.org/10.1111/j. 1469-8986.1987. tb00311.x

Oestreich, L. K., Mifsud, N. G., Ford, J. M., Roach, B. J., Mathalon, D. H., & Whitford, T. J. (2016). Cortical suppression to delayed self-Initiated auditory stimuli in schizotypy: Neurophysiological evidence for a continuum of psychosis. *Clinical EEG and Neuroscience*, *47*(1), 3–10. http://dx.doi.org/10.1177/1550059415581708

Repp, B. H., & Knoblich, G. (2007). Action can affect auditory perception. *Psychological Science*, *18*(1), 6–7. http://dx.doi.org/10.1111/j. 1467-9280.2007.01839.x

Roussel, C., Hughes, G., & Waszak, F. (2013). A preactivation account of sensory attenuation. *Neuropsychologia*, *51*(5), 922–929. http://dx.doi.org/10.1016/j.neuropsychologia.2013.02.005

Roussel, C., Hughes, G., & Waszak, F. (2014). Action prediction modulates both neurophysiological and psychophysical indices of sensory attenuation. *Frontiers in Human Neuroscience*, *8* http://dx.doi.org/10.3389/fnhum.2014.00115

SanMiguel, I., Todd, J., & Schröger, E. (2013). Sensory suppression effects to self-initiated sounds reflect the attenuation of the unspecific N1 component of the auditory ERP. *Psychophysiology*, *50*(4), 334–343. http://dx.doi.org/10.1111/psyp.12024

SanMiguel, I., Widmann, A., Bendixen, A., Trujillo-Barreto, N., & Schröger, E. (2013). Hearing silences: Human auditory processing relies on preactivation of sound-specific brain activity patterns. *Journal of Neuroscience*, *33*(20), 8633–8639. http://dx.doi.org/10.1523/JNEUROSCI. 5821-12.2013

Santangelo, V., Olivetti Belardinelli, M., & Spence, C. (2007). The suppression of reflexive visual and auditory orienting when attention is otherwise engaged. *Journal of Experimental Psychology Human Perception and Performance*, *33*(1), 137–148. http://dx.doi.org/10.1037/0096-1523.33.1.137

Sato, A. (2008). Action observation modulates auditory perception of the consequence of others' actions. *Consciousness and Cognition*, *17*(4), 1219–1227. http://dx.doi.org/10.1016/j.concog.2008.01.003

Schafer, E. W., & Marcus, M. M. (1973). Self-stimulation alters human sensory brain responses. *Science*, *181*(4095), 175–177. http://dx.doi.org/10.1126/science.181.4095.175

Shahin, A., Roberts, L. E., Pantev, C., Trainor, L. J., & Ross, B. (2005). Modulation of P2 auditory-evoked responses by the spectral complexity of musical sounds. *Neuroreport*, *16*(16), 1781–1785. http://dx.doi.org/10.1097/01. wnr.0000185017.29316.63

Sowman, P. F., Kuusik, A., & Johnson, B. W. (2012). Self-initiation and temporal cueing of monaural tones reduce the auditory N1 and P2. *Experimental Brain Research*, *222*, 149–157. http://dx.doi.org/10.1007/s00221-012-3204-7

Sperry, R. W. (1950). Neural basis of the spontaneous optokinetic response produced by visual inversion. *Journal of Comparative and Physiological Psychology*, *43*, 482–489. http://dx.doi.org/10.1037/h0055479

Timm, J., SanMiguel, I., Keil, J., Schroger, E., & Schonwiesner, M. (2014). Motor intention determines sensory attenuation of brain responses to self-initiated sounds. *Journal of Cognitive Neuroscience*, *26*(7), 1481–1489. http://dx.doi.org/10.1162/jocn_a_00552

Von Holst, E. (1954). Relations between the central nervous system and the peripheral organs. *The British Journal of Animal Behaviour*, *2*(3), 89–94. http://dx.doi.org/10.1016/s0950-5601(54)80044-x

Weiskrantz, L., Elliott, J., & Darlington, C. (1971). Preliminary observations on tickling oneself. *Nature*, *230*(5296), 598–599. http://dx.doi.org/10.1038/230598a0

Whitford, T. J., Mathalon, D. H., Shenton, M. E., Roach, B. J., Bammer, R., Adcock, R. A., & Ford, J. M. (2011). Electrophysiological and diffusion tensor imaging evidence of delayed corollary discharges in patients with schizophrenia. *Psychological Medicine*, *41*(5), 959–969. http://dx.doi.org/10.1017/S0033291710001376

Wolpert, D. M., Ghahramani, Z., & Jordan, M. I. (1995). An internal model for sensorimotor integration. *Science*, *269*, 1880–1882. http://dx.doi.org/10.1126/science.7569931

van Elk, M., Salomon, R., Kannape, O., & Blanke, O. (2014). Suppression of the N1 auditory evoked potential for sounds generated by the upper and lower limbs. *Biological Psychology*, *102*, 108–117. http://dx.doi.org/10.1016/j.biopsycho.2014.06.007