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Establishing a right frontal beta signature for stopping action in scalp EEG: implications for testing inhibitory control in other task contexts

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Abstract

Many studies have examined the rapid stopping of action as a proxy of human self-control. Several methods have shown that a critical focus for stopping is the right inferior frontal cortex (rIFC). Moreover, electrocorticography (ECoG) studies have shown beta band power increases in the rIFC and in the basal ganglia for successful vs. failed stop trials, before the time of stopping elapses, perhaps underpinning a prefrontal-basal-ganglia network for inhibitory control. Here we tested whether the same signature might be visible in scalp EEG – which would open important avenues for using this signature in studies of the recruitment and timing of prefrontal inhibitory control. We used Independent Component Analysis and time-frequency approaches to analyze EEG from three different cohorts of healthy young volunteers (48 participants total) performing versions of the standard stop signal task. We identified a spectral power increase in the band 13–20Hz that occurs after the stop signal, but *before* the time of stopping elapses, with a right frontal topography in the EEG. This right frontal beta band increase was significantly larger for successful compared to failed stops in two of the three studies. We also tested the hypothesis that unexpected events recruit the same frontal system for stopping. Indeed, we show that the stopping-related right lateralized frontal beta signature was also active after unexpected events (and we accordingly provide data and scripts for the method). These results validate a right frontal beta signature in the EEG as a temporally precise and functionally significant neural marker of the response inhibition process.

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Data Sharing: Two stop signal data-sets standing alone, and one stop signal plus unexpected events data set were analyzed. We provide access to the EEG and behavioral data for the latter (combined) data-set along with detailed instructions and scripts to generate the results shown in the paper: <https://osf.io/ukvmh/>

Keywords

beta oscillations; neural stopping network; electrophysiology; prefrontal inhibition; Independent Component Analysis

Introduction

One component of executive function is top-down inhibitory control. The domain in which this is best explored is the stopping of action. In the lab, this is often operationalized using the stop signal task (SST, Logan and Cowan, 1984). On each trial, participants initiate a go response, which they sometimes have to try to stop when a stop signal is subsequently presented. The latency of the stop process, referred to as stop signal reaction time (SSRT), can be estimated with models that take into account the stop signal delay, the probability of stopping and the reaction time on go trials (Logan and Cowan, 1984). Understanding the neural architecture of the putative inhibitory process in stopping action is important for several reasons. One of these is that if a clear brain signature of inhibitory control can be established for the stopping of action, it could then be asked if the same signature occurs in other behavioral and cognitive contexts.

There have been many studies of the neural correlates of action stopping, specifically for the stop signal (and related) tasks, across species, and using many methods, see reviews by (Aron, et al., 2016a; Chambers, et al., 2009; Jahanshahi, et al., 2015; Kenemans, 2015; Schall and Godlove, 2012; Schmidt and Berke, 2017). Here we focus on the cortical aspect. Lesion and stimulation approaches in humans have pointed to the critical importance of the right inferior frontal cortex (rIFC) (reviewed by Aron, et al., 2014), and this region is also activated in many fMRI studies (for meta analysis see, e.g. Cai, et al., 2014; Swick, et al., 2011). Moreover, electrocorticography (ECoG) studies, which record electroencephalography (EEG) signals directly from the surface of the brain, have shown that one electrophysiological signature of stopping is a power increase in the rIFC for successful vs. failed stop trials, before the time of stopping elapses i.e. Stop Signal Reaction Time, SSRT (Swann, et al., 2009; Wessel, et al., 2013) [but see Fonken et al. (2016)]. These power increases occurred in the beta band, as do power increases in the basal ganglia during stopping (e.g. Ray, et al., 2012; Wessel, et al., 2016a), perhaps underpinning a prefrontal-basal-ganglia network for inhibitory control (reviewed by Aron, et al., 2016b; Zavala, et al., 2015). The cortical electrophysiology results are especially important because they show both the frequency and the timing of activation of a region known to be critical for the stopping process. Here we test whether a similar signature is detectable in scalp EEG. If so, this would open avenues for testing the generalizability of inhibitory control in other task contexts, moreover in healthy individuals (rather than surgical patients). It would also open up more ecologically-relevant scenarios since scalp EEG is much more portable than ECoG or fMRI.

We used ICA and time-frequency approaches to analyze scalp EEG data from three different cohorts of healthy young volunteers performing versions of the standard stop signal task. First, based on the results from ECoG (above), we expected to detect a spectral power

increase in the band 13–20Hz that occurs after the stop signal, but *before* SSRT with a right frontal topography in the scalp EEG. We also predicted that this right frontal beta band increase would be larger for successful compared to failed stops. Second, we aimed to establish the reliability of this signature by testing for it in the three independent data-sets. Third, we aimed to test if this same signature of inhibitory control occurs in a different behavioral context – the processing of unexpected events. We could test this because, for one of the data-sets, each participant had performed an unexpected events task and then a stop signal task. For the unexpected events task, on each trial the subject prepares to respond – and just before the imperative stimulus a sound occurs; on a minority of trials the sound is a deviant (we refer to these as ‘novels’), c.f. (Barcelo, et al., 2006; Gentsch, et al., 2009; Parmentier, et al., 2008; Vachon, et al., 2012; Wessel, et al., 2012). While an earlier study, Wessel & Aron (2013), showed that unexpected events and stopping recruit a common central midline component in the EEG – negative deflection at 200ms (N2) and a positive deflection at 300ms (P3) – here we aimed to test if there was a common right frontal beta increase for unexpected events and stopping. As above, this was motivated by the putative specificity of the right frontal beta as a signature of rIFC (based on ECoG). We suppose this reflects a different part of the stop process compared to the N2/P3 complex in the EEG.

We tested the idea of a common process as follows. In each participant, we concatenated the EEG data from the unexpected events task and the stop signal task. We then used Independent Components Analysis to decompose the concatenated EEG data into temporally independent source signals (Onton, et al., 2006). The goal was to identify a component that has the characteristic frontal beta signature of stopping, and then look at whether this component is active after unexpected events (Wessel, 2016). If there is a common inhibitory process, then the same Independent Component (IC) with a right frontal topography identified in the stop signal task should show a spectral power increase in the beta band after unexpected events.

Materials and Methods

Participants

We analyzed data from participants in three different studies, all using slightly different versions of the stop signal paradigm. Other results from these data-sets were reported in (Wessel and Aron, 2013; Wessel and Aron, 2014; Wessel and Aron, 2015). Importantly, one of the studies also included an unexpected perceptual events task (Wessel and Aron, 2013). The number of participants was: Study 1: 11; Study 2: 13; Study 3: 24; overall mean age 21.47 SEM 0.77, 34 female, 3 left handed. All participants provided written informed consent according to the local ethics committee at UCSD.

Stop signal versions

The SST requires subjects to make a quick motor response to a go signal. On a minority of trials a stop signal is presented which requires participants to try to stop their response. The delay at which the stop signal occurs after the go signal (stop signal delay, SSD) was initially set to 200ms in all studies and then dynamically adjusted during the task according to the participant’s performance. i.e. SSD was increased by 50ms when participants

successfully stopped and decreased by 50ms when they failed to stop their response. The staircase method typically yields a probability of successful stopping of around .5. See Figure 1 for an overview of the different versions of the stop signal task.

Study 1: (manual responses, visual stop signals) N = 11 (Wessel and Aron, 2014)—Go signals were white arrows pointing left or right at the center of the screen. Participants pressed a left or right key on a standard computer keyboard. The stop signal was a red exclamation mark over the white arrow. The probability of a stop signal was 25%. Participants performed 240 trials split across 6 blocks. Trial timing was as follows: 500 ms fixation, 1,500 ms response deadline, 500ms ITI, overall trial duration: 2,500 ms.

Study 2: (verbal responses, visual stop signals) N = 13 (Wessel and Aron, 2013)—Go signals were white letters (K or T) at the center of the screen. Participants spoke the letter into a microphone. The stop signal was the white letter turning red. The probability of a stop signal was 25%. Participants performed 400 trials split across 10 blocks. Trial timing was as follows: 1,000 ms fixation, 1,000 ms response deadline, 500ms ITI, overall trial duration: 2,500 ms.

Study 3: (manual responses, visual stop signals) N = 24 (Wessel and Aron, 2015)—Go signals were white arrows pointing left or right in the center of the screen. Participants pressed a left or right key on a standard computer keyboard. The stop signal was the white arrow turning red. The probability of a stop signal was 33%. Participants performed 300 trials split across 6 blocks. Trial timing was as follows: 500 ms fixation, 1,000 ms response deadline, 1000ms ITI, overall trial duration: 2,500 ms.

Unexpected Events Task (Wessel and Aron, 2013)

On each trial a sound was played followed by a letter. Participants spoke the letter into a microphone. On 80% of trials (standards), the sound was a 600 Hz sine wave, on 20% of trials (novels), a unique birdsong segment was presented. Trial timing was as follows: 1000 ms fixation plus a variable jitter (100 to 500 ms), 200ms sound, 300 ms delay before letter appeared, 1000 ms response deadline (see for full details Wessel and Aron, 2013). Participants performed 450 trials equally divided into six blocks of 75 trials (15 of which were novel trials and 60 of which were standard trials). Only subjects in Study 2 did this task, and *before* the SST (i.e. the order was always unexpected events task followed by stop signal task).

Behavioral Analysis

For the stop signal task, for each participant, we estimated Go RT, number of erroneous or missed Go trials, Failed Stop RT (which should be faster than Go RT according to the race model, Logan and Cowan, 1984), the probability of stopping (which should be in the range of .4 and .6, confirming the efficacy of the SSD staircase algorithm, and the prospect of accurately estimating SSRT) and SSRT itself. SSRT was computed using the mean method – i.e. subtracting the observed mean SSD on stop trials from the observed mean RT on go trials.

EEG recording

EEG data were recorded using a BioSemi system (BioSemi Instrumentation, The Netherlands), In studies 2 and 3, 64 scalp electrode sites in the extended five per cent international 10/20 electrode system (Oostenveld and Praamstra, 2001) were recorded at 512 Hz sampling rate. In study 1 128 scalp electrode sites were recorded at 1024 Hz sampling rate. The 128 channel montage was in accordance with the BioSemi designed equiradial system. In each dataset we also recorded six electrooculogram electrodes placed above and below each eye and at the outer canthi of the eyes.

EEG preprocessing

EEG data analysis was performed using custom scripts in MATLAB2015b (The MathWorks) incorporating EEGLAB 14.0b functions (sccn.ucsd.edu/eeglab) (Delorme and Makeig, 2004). For study 3 the EEG data were down sampled to 512 Hz. Preprocessing then proceeded as follows. First, the EEG data were high-pass filtered at 2 Hz (to minimize slow drifts) [zerophase FIR filter order 3381], and low pass filtered at 200 Hz [zerophase FIR filter order 565]. Second, EEG channels with prominent artifacts were identified by visual inspection and removed. Subsequently, channels that were de-correlated ($r < 0.4$) from neighboring channels for more than 1% of the time were automatically removed. On average, 122/134 channels for study1, 64/70 channels for study 2, and 68/70 channels for study3, per participant (study1: SD 3.1; study2: SD 2.3; study3: SD 1.7) remained in the analysis. Third, EEG data were then re-referenced to a common average reference. Fourth, the continuous EEG data were then visually inspected for artefacts and affected segments were removed from further analysis. Fifth, to perform automatic rejection of affected segments the data were partitioned into epochs of 0.5 s. Epochs containing values exceeding the average of the probability distribution of values across the data segments by 5 SD were rejected. On average, the number of stop and go trials and the % of EEG data that remained in the analysis were as follows. For study 1: 131 Go, 64 Stop and 81 % of EEG data; for study 2: 224 Go, 94 Stop and 80 % of EEG data; study 3: 175 Go, 87 Stop and 87 % of EEG data.

ICA approach

Stop signal datasets—The preprocessed EEG data were decomposed using adaptive mixture ICA (AMICA) (Palmer, et al., 2006; Palmer, et al., 2008). AMICA is a generalization of the Infomax ICA (Bell and Sejnowski, 1995; Makeig, et al., 1996) and multiple-mixture (Lee, et al., 1999; Lewicki and Sejnowski, 2000) ICA approaches. AMICA performed blind source separation of all preprocessed data for each subject individually, based on the assumed temporal near-independence of the effective EEG sources (Makeig, et al., 2004; Makeig, et al., 2002).

ICA results in as many independent components for each dataset as there are channels recorded, thus studies 2 and 3 resulted in an average of respectively 64 and 68 ICs per participant while study 1 resulted in an average of 122 ICs per participant (i.e. some channels were removed for each subject). We then calculated a best-fitting single equivalent current dipole matched to the scalp projection of each independent component (IC) source using a standardized three-shell boundary element head model implemented in the DIPFIT

toolbox (Delorme and Makeig, 2004; Oostenveld and Oostendorp, 2002). The IC scalp projection or scalp map represents the relative weights with which the ‘source’ projects to each of the scalp channels. The theoretical basis of ICA assumes brain source locations and projection maps to be spatially fixed, while source ‘activations’ represent their activity time courses (Onton, et al., 2006).

The standard electrode locations corresponding to the extended 10–20 System were aligned with a standard brain model (MNI). The biosemi equiradial system montage used in study 1 and the 5% International 10/20 System partly overlap, allowing us to align equiradial system electrode positions to the standard 10/20 system and in the following to a standard brain model (MNI) using electrode landmarks such as Cz.

We excluded ICs from further analysis for which the equivalent dipole model was located outside of the brain and explained less than 85% of variance of the corresponding IC scalp map.

Below we explain the process by which automatic clustering of ICs is done across subjects.

Overview: Clustering used feature vectors derived from 1) IC dipole locations, 2) scalp projection 3) power spectra in the range 3–200 Hz, 4) event related potentials in the time window 0–600 ms following the stop signal), and 5) event related spectral perturbations (ERSPs) in the frequency range from 3–20Hz and time window from 0–600ms following the stop signal. Pre-clustering dimensionality reduction: Before clustering we reduced the dimensionality of the features as follows. First we selected the relevant parts of each feature. For example for the ERP we selected the mean over trials 0–600ms after the stop signal which resulted in ~307 data points (given the sampling rate of 512Hz). We then reduced these 307 points to 10 dimensions using Principal Component Analysis (PCA). PCA finds orthogonal subspaces that explain maximal variance of the data, with the first principal component explaining the largest part of the variance of the data. We applied the same procedure to the spectra, ERSP and scalp maps. Dipoles have inherently only 3 dimensions (the tailarach coordinates x,y,z). We thus ended up with 5 feature vectors, 4 each with 10 dimensions and 1 with 3 dimensions (related to dipoles) for each IC.

Weighting of feature vectors: The dimensionally reduced feature vectors were then weighted for subsequent clustering i.e. dipole locations: weight 12; scalp projection: weight 4; power spectra: weight 3; event related potentials: weight 3; and event related spectral perturbations (ERSPs): weight 10. These weights were chosen based on our hypothesis: we expected a beta band increase (13–20Hz) with a right frontal scalp distribution and a timing from 0–600ms after the stop signal. We also know that an ERP occurs at around 300ms following the stop signal. The relatively large weight for the ERSPs made clustering similarly sensitive to IC ERSP differences as for dipole location.

Concatenation and clustering: The 5 feature vectors were then concatenated for each IC and further reduced to 10 principal components using PCA. We then ran k-means clustering ($k = 11$).

Outlier clusters: ICs were identified as outliers if their locations in the clustering vector space were 4 SDs from the obtained cluster centers. Only clusters including ICs from more than half of the participants were considered for further analysis. ICs were separated into 11 clusters representing either muscle or cortical activity. Cortical clusters were then screened for remaining artifact ICs by visually inspecting single IC spectra and ERSP images for broadband activity from 20–100Hz. It is possible that some ICs contain cortical activity mixed with EMG, or the clustering algorithm did not correctly assign all ICs containing muscle activity to a separate cluster. Those ICs were moved to the outlier cluster.

Unexpected events (Study #2 only)—The approach was slightly different. Now, in each participant, the EEG data from both the unexpected events task and the stop signal task were preprocessed as above and concatenated. Then ICA was run on the *combined* data. The process of clustering ICs was automated, as above (using the same features). We then identified a cluster that had a right lateralized frontal scalp distribution (as above for SST only) and showed a beta band increase (13–20Hz) relative to baseline in the stop trials of SST that elapses following the stop signal but before SSRT. This cluster was defined as the *stop* cluster (the ‘prototype process’, see Wessel, 2016) and each component therein (most subjects contributed just one component) was used to compute time-frequency decompositions for the unexpected events task (the ‘candidate process’).

Hypothesis-testing using the event-related spectral perturbation (ERSP)

Stop signal task—The data were segmented into time epochs relative to onsets of the go cue (i.e., from –1 to 2 s around the go cue). ERSPs (Makeig, 1993) were computed for each IC in the cluster of interest. We used Morlet wavelets (Morlet et al., 1982) for time-frequency analyses (4–30 Hz). Starting with 3 cycles at 4Hz we linearly increased the number of cycles used by 0.5 as frequency increased. To generate ERSPs single trial spectrograms were computed and time warped to the median RT latency for Go trials and to the median stop signal delay latency for stop trials (across subjects within each study) using linear interpolation. This procedure aligned time points of reaction times and stop signals over trials following each go cue. Relative changes in spectral power were obtained by computing the mean difference between each single-trial log spectrogram and the mean baseline spectrum (the average log spectrum between –1 s and 0 s preceding the go cue). If participants had more than one IC in a cluster the respective IC ERSP matrices were averaged for that participant. Significant deviations from the baseline were detected using a nonparametric bootstrap approach (Delorme and Makeig, 2004) and corrected for false discovery rate (Benjamini and Yekutieli, 2001) with a significance level set a priori at 0.05.

Unexpected events task—The data were segmented into epochs relative to novel and standard tones (i.e. from –1 to 2 s around the tone). Again, relative changes in spectral power were obtained by computing the mean difference between each single-trial log spectrogram and the mean baseline spectrum – in this case, the average log spectrum between –1 s and 0 s preceding the tone. Across-subject ERSP maps were then generated for novel trials, standard trials and novel-standard.

A follow-on analysis specifically tested whether unexpected events recruited the beta band. To do this, we selected the beta frequency band for each participant individually using combined successful and failed stop trials from SST (the ‘prototype process’). For each participant we selected the band in the range from 13–20Hz that was maximally modulated over time in the window 300–800 ms relative to the go signal. For, each participant, we then averaged a band of ± 2 Hz around this selected beta band but now in the *unexpected events* data (the ‘candidate process’), and averaged over participants.

We also checked for a single trial correlation on novel trials between beta band power and RT for each participant. We selected the frequency band as described above and averaged beta power in single trials in the interval 150ms–300ms after the unexpected event. We then correlated those values to single trial RT. We did this analysis on the first half of the novel trials, since the effect of unexpected events in this study wore off over time.

Effect size

We computed effect size as a guide to powering future studies. This was for the right frontal beta power increase for successful stop trials vs. baseline. We selected a time window of -150 ms to $+50$ ms around mean SSRT for each study and averaged power values at each frequency over time. We then averaged power over frequencies in the beta band from 13–20Hz resulting in one value for beta power increase and one value for baseline power per participant. We then computed one overall value for all three studies for Cohen’s d .

Results

Behavior

Stop Signal Task—Behavioral performance was fairly typical for healthy young participants (Table 1), with quite fast Go RT, $p(\text{stop})$ about 50%, few omissions or errors on Go trials (error rates at 1%, SEM: 0.3), and Failed Stop RT faster than Go RT, consistent with the race model. Go reaction time was longer than reaction time on failed stop trials in all participants, indicating the validity of the race model. Mean SSDs were 241ms 300ms and 315ms in Study 1, 2 and 3, and mean SSRT were 247ms, 257ms and 204ms for Study 1, 2 and 3. These are typical values. Notably, SSRT was longest for study 2 which involved stopping of speech. For each of the 3 studies $p(\text{stop})$ was approximately .5, indicating the efficacy of the SSD staircase algorithm.

Unexpected Events Task—As reported in Wessel & Aron (2013) over all blocks, RT was numerically slower for novel compared with standard trials. While the main effect of trial type was not significant ($F(1,12) = 1.6$, $p = 0.23$) there was a significant block $\times$ trial type interaction ($F(5,60) = 7.61$, $p < 0.0001$), indicating that the difference between novels and standards wore off over time. There was a significant novelty-induced slowing for Block 1 ($t(12) = 5.37$, $p < 0.001$, $d = 0.75$), but not the other blocks (all $ps > 0.25$).

Cortical IC source Clusters related to stopping

Our main analysis focuses on cluster RF with a right frontal scalp distribution (possibly related to the ECoG rIFC signature Swann, et al., 2009; Wessel, et al., 2013) as explained

above. In addition we report analysis from two other putatively stopping-related components, cluster MC with a midline central scalp distribution (c.f. Wessel and Aron, 2013; Wessel and Aron, 2015), and cluster SML with a left ‘sensorimotor’ topography (contralateral to the hand participants were responding with in studies 1 and 3). Although in studies 1 and 3 the cluster RF appeared to have a more frontocentral than right lateralized scalp distribution, the respective distribution of IC dipoles (not shown here) was skewed to the right hemisphere.

ERSPs

Cluster RF—As predicted we found that in all studies the cluster RF showed a stopping-related power increase in the beta band (13–20Hz) significant from baseline starting as early as 150ms after the stop signal (see Figure 2). This beta band power increase was present for successful stop and failed stop trials but absent for go trials. Furthermore, for studies 1 and 2 the beta power increase was significantly different from baseline only for the successful stop trials, not for failed stop trials and in study 1 and 2 the beta band increase was significantly greater for successful vs. failed stop trials. In study 3 the beta power increase was significantly different from baseline for both successful and failed stop trials but now there was no difference between successful and failed. The Cohen’s effect size for right frontal beta power increase in successful stop trials above baseline was 0.74 for Study 1, 0.96 for Study 2 and 0.77 for Study 3. The mean effect size was 0.82. Note that the features that went into the clustering method were 1) scalp distribution, 2) dipoles, 3) spectra, 4) ERP after stop signal, 5) ERSP after stop signal for 600 ms in the range 3 to 20 Hz. These features, do not guarantee a stopping-related beta increase, or the specific timing (just before SSRT), or a successful vs. failed difference, or a right frontal topography.

We also observed low frequency power increases in the RF cluster after both successful and failed stop trials. This increase is probably related to perceptual processing of the stop signal and not to the actual stopping process since it occurs for both failed and successful stop trials.

Cluster SML—For all three studies, cluster SML (putative M1) showed a power decrease in the mu (7–12Hz) and beta band (13–30Hz) significant from baseline relative to the go signal. This mu and beta band power decrease was present for go trials, successful stop and failed stop trials (Figure 4). The decrease was most pronounced for failed stop trials > go > successful stop (in that order). This is a nice validation of earlier results with ECoG from the hand area of M1 in a stop signal task (Swann, et al., 2009) and also findings from motor physiology (Stinear, et al., 2009) as will be discussed below.

ERPs

Cluster MC—This cluster was similar to that reported in earlier studies of stopping (Wessel and Aron, 2013; Wessel and Aron, 2014; Wessel and Aron, 2015). Analysis of ERPs in this cluster confirmed prior results by showing, for all three studies, P3 onset latencies earlier for successful stop compared to failed stop trials before SSRT (see Figure 3). Note that this cluster is different in spatial location from the RF cluster.

Cluster RF—While this and other studies have suggested that the N2/P3 has a dorsomedial topography relative to the stop signal (Huster, et al., 2013; Wessel, 2017; Wessel and Aron, 2013; Wessel and Aron, 2014; Wessel and Aron, 2015) we did observe a N2/P3 complex in the RF cluster, albeit much smaller in amplitude than in central midline areas (MC cluster). This N2 in the RF cluster could have arisen because not all sources were completely separated leading to a ‘merged source’ in some subjects, i.e. one containing a right frontal beta increase and the N2/P3 complex. Indeed in some participants there was only one ‘frontal source’, and this source was localized in between midline and right frontal topographies and it combined features from both clusters. Another possibility is that the N2 is a distributed phenomenon like the P3a and P3b – with the P3a occurring earlier and over medial frontal areas and the P3b occurring later and over more posterior medial areas. In our study the N2 occurring over dorsomedial areas (MC cluster) had a shorter latency than the N2 occurring over right frontal areas (RF cluster). We note that Schmajuk et al. (2006) observed a N2 relative for stopping with a right frontal voltage distribution at 200ms latency. It would be interesting to look at the voltage distributions Schmajuk et al. (2006) observed, at fine grained time points between 150ms–250ms after the stop signal, to see how scalp distributions change over these latencies.

Unexpected Events Task

We ran ICA over the concatenated EEG for the SST and unexpected events task. We identified a cluster RF with a right frontal scalp distribution similar in spatial location and activity to the cluster that we found for the stop signal task in studies 1–3. When testing the ERSF in this cluster for the unexpected events task we observed a power increase for novel versus standard trials in the range from 10–16Hz in the time from around 100ms to 500ms after the novel (see Figure 5). To more specifically test the beta band, we first selected the frequency band in the range from 13–20Hz in each participant that was maximally modulated over time in the window 0–500 ms after the stop signal in SST (‘the prototype process’). We then averaged a band of ± 2 Hz around this selected beta band but now in the *unexpected events* data (the ‘candidate process’). In this way we confirmed that the RF cluster displays a significant beta band power (13–20Hz) increase for novels vs. baseline starting around 100ms after the tone and lasting for around 200ms. This beta band power increase was absent after standard tones and the novel vs. standard difference was significant. However the correlation between single trial beta power and RT on novel trials was not reliable.

Discussion

We reanalyzed scalp EEG from 3 stop signal data sets. In each case, we identified a brain signature for action-stopping with a beta power increase following the stop signal, that occurred before the time of stopping, and with a right frontal topography. In 2 of the 3 data-sets, beta band power was stronger for successful than failed stop trials. We interpret this signature as the scalp EEG counterpart to the stop-related beta power increase in rIFC shown with intracranial ECoG indexing prefrontally-mediated inhibitory control. Furthermore we show that the same characteristic brain signature of stopping is active after unexpected

events. This provides important validation of a scalp EEG signature of inhibitory control, that could be leveraged by other investigators in other task contexts.

Right frontal beta band increase as a putative index for action stopping

Using ICA we identified a cluster of independent components over right frontal areas in each of the three studies. These clusters show a transient beta power (13–20Hz) increase that occurs after the stop signal and before the time of stopping and is absent on go trials. This beta band power increase was significantly larger for successful compared to failed stop trials in studies 1 and 2 (see Figure 2). Our study was motivated by ECoG studies showing a beta band (13–20Hz) power increase in the rIFC for successful vs. failed stop trials, before SSRT (Swann, et al., 2009; Wessel, et al., 2013). Here we show something strikingly similar with scalp EEG in healthy young participants. While only limited anatomical claims can be made from EEG results alone (without individual MRI-based head model), the similarity of these features (right lateralized, frontal and beta power) in the current scalp EEG results to the abovementioned ECoG results strongly suggests that EEG reflects the same process.

One discrepancy in our results was that studies 1 and 2 showed increased beta power for successful vs. failed stop trials, but study 3 did not (notably, however, there *was* a strong beta increase in that study for successful stop trials). This lack of difference between conditions could be due to the higher percentage of stop trials in study 3 (33%, compared to 25% for studies 1 and 2). Also, in study 3, subjects performed the simple stop task that we analyzed, after they had already performed, in the same session, a different kind of stop task that induced more preparation to stop, and effectively had a 50% stopping rate (unpublished). Thus they might have been biased to use a different strategy (indeed RT was much longer than the other studies).

A recent study by Huster et al, (2017), using a group level ICA approach and classification methods identified 15 ICs and 45 time frequency features in the EEG contributing to the stopping process. Interestingly an IC with a scalp map that had a somewhat right frontal distribution and showed a frequency cluster with an increase in the beta band around 150ms after the stop signal was one of the 10 best predictors for differences between successful stop and go trials. That study did not, however, differentiate between successful and failed stop trials as we did here.

In addition to the independent component with the right lateralized beta effect, ICA also revealed, for each of the 3 data-sets, another stopping-related signature – a dorsomedial frontal topography with a negative deflection around 200ms and a positive deflection in the EEG occurring around 300 ms after the stop signal. The P3 latency occurred earlier for successful compared to failed stop trials. The N2/P3 signature has now been well-established as relating to the stopping process in several EEG studies (Huster, et al., 2013; Wessel, 2017; Wessel and Aron, 2013; Wessel and Aron, 2014; Wessel and Aron, 2015). Yet this has different features from the right lateralized beta effect and could putatively reflect the preSMA rather than rIFC – although this remains to be established. The preSMA, which is dorsomedial, has also been implicated in stopping by a variety of brain stimulation, lesion, imaging, ECoG and connectivity studies, (see Aron, et al., 2014; Herz, et al., 2014; Jahanshahi, et al., 2015).

Although we also observed an N2/P3 complex with a smaller amplitude over right frontal brain areas similar to Schmajuk et al. (2006), it is unclear how these two phenomena are related. Increases in beta power relative to the stop signal have been shown in ECoG in the rIFC (Swann et al, 2009, Wessel et al, 2013) and the rIFC is thought to be part of a prefrontal-basal-ganglia network for inhibitory control (reviewed by Aron, et al., 2016b; Zavala, et al., 2015). Further, the role of beta oscillations in motor control is well defined (Gilbertson et al, 2005, Pogosyan et al, 2009). Thus a beta power increase with right frontal topography is functionally more specific than the N2/P3 complex which can occur in a wide range of tasks. We expect that further studies will look into the functional relationship between the N2 and the right frontal beta power increase.

We note here a limitation of our paper which is that all three stop signal studies were visual-go/visual-stop paradigms, whereas often stop signal studies are done with visual-go/auditory-stop. For this latter kind of paradigm, Kenemans (2015), reviewing ERP findings, observed that there is little sign of right frontal ERP signature of successful vs. failed stop. While our results here mainly concern a right frontal time-frequency signature, it would be important to validate, in future studies or analyses, that this does also apply in the case of a visual-go/auditory-stop paradigm.

Unexpected events

For study 3, where each subject did both an unexpected events task and a stop signal task, we used ICA to decompose the concatenated EEG data. We then used the automated clustering method to identify a cluster localized over right frontal scalp areas that showed a transient beta power (13–20Hz) increase elapsing after the stop signal and before the time of stopping (the ‘prototype process’). When testing this same component for the candidate process (i.e. unexpected events) we identified a transient beta band power increase (13–20Hz) that was significantly greater for unexpected events vs. standards. This increase started about 150 to 200 ms after the unexpected event, consistent with the timing of a putative suppression process indicated by other methods such as TMS and STN LFP (see Wessel and Aron, 2013; Wessel, et al., 2016b). While this striking finding supports the idea that unexpected events activate the same (pre)frontal system as stopping in the stop signal paradigm, there are some caveats. First, we did not observe a reliable relation between single trial beta power and RT on unexpected events trials. However, we note that post-novel slowing was quite small in this particular study. Also, our theory of unexpected events is that they recruit the stopping system within say 150 ms and disrupt action (motor suppression) and task-set. Yet this particular task was so simple, with the subject saying ‘K’ or ‘T’ to an imperative stimulus about 1 sec later, that there might have been little task-set to interrupt. Future studies will need to more carefully test whether this right frontal beta signature of unexpected events relates to behavioral and cognitive interruptions. A second caveat is that the unexpected event in this study was a 200 ms birdsong segment (20%), whereas the standard was a 200 ms sine wave (80%). This means the birdsongs were unexpected at the informational level and at the stimulus characteristic level too (saliency, pitch). This does not speak against the finding that such unexpected events activated the same brain signature as the stop signals, but it leaves open the question about what is the critical element that drives

this (Parmentier, 2014; Parmentier, 2016). Future studies should better control the type of unexpectedness.

Sensorimotor Cortical Areas

A useful validation of our ICA method is that we also identified an independent component cluster for each study located over left sensorimotor areas, and contralateral to the responding hand in studies 1 and 3. In all three studies we found event related desynchronization (ERD see (Pfurtscheller and Da Silva, 1999) in the mu and beta band starting after the go cue for stop and go trials. This desynchronization was more pronounced for go and failed stop trials than for successful stop trials, and it was more pronounced for failed than go trials (consistent with a faster/stronger movement on those trials). This is a nice validation of earlier results with ECoG from the hand area of M1 in a stop signal task (Swann, et al., 2009). The fact that failed stop showed a greater mu/beta power decrease (desynchronization) than go trials is consistent with failed stop trials having faster RT (an earlier/more energetic movement). Moreover relatively less desynchronization on successful stop trials is consistent with both a slower RT and also a stopping-related synchronization in M1 – for example stopping increases GABA in M1 (Stinear, et al., 2009) and increased GABA in M1 has been related to increased power in mu/beta (Jensen, et al., 2005).

In study 2 with verbal responses, in addition to the left SML discussed above, we also identified a right SML cluster with the same properties. This fits with bilateral representations of lip and tongue in the motor cortex – which should be active in both hemispheres during verbalization.

Methodological considerations

Our results help define methodological guidelines for EEG studies that want to employ a frontal beta band signature of inhibitory control. 1) Number of participants: We estimated that with an overall Cohen's effect size of 0.82 for all three studies we would need 18 participants to get 95% power to detect a significant beta band power effect with alpha of 0.05, 1-tailed). 2) Total number of stop trials: in study 1 we only had on average 60 stop trials for the analysis (only about half of these were successful). Nevertheless, even with the small number of trials we detected a significant difference between successful and failed trials in the beta band. We would therefore suggest an absolute minimum with these numbers but preferably many more trials. 3) Percentage of stop trials: Studies 1 and 2 revealed a difference in the beta band between successful and failed stop trials whereas study 3 did not. Notably the first two studies had a 25% stop rate while study 3 had a 33% rate. Several lines of evidence suggest that the way in which stopping is done changes with increased probability of stop signals (Chikazoe, et al., 2009; Verbruggen and Logan, 2009; Wessel, 2017) and we therefore recommend the use of a 25% stop signal rate.

Conclusion

We show with scalp EEG in healthy participants that we can derive a signature of stopping action that manifests as a beta power increase for successful vs. failed stop trials, before the time of stopping, and with a right frontal topography. We interpret this signature as the scalp EEG counterpart to the stop-related beta power increase in rIFC in ECoG. We furthermore

show that the same frontal right lateralized beta signature of stopping is active after unexpected events – which strikingly supports the argument that unexpected events leverage the same brain system as stopping in the stop signal paradigm (Wessel and Aron, 2017). We anticipate that other researchers can deploy this same signature for stopping in scalp EEG to test the recruitment and timing of inhibitory control in other task contexts (c.f. van Gaal, et al., 2008; Wagner, et al., 2016). Accordingly, we provide companion material which shares the data and analysis stream.

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Figure 1.

Three stop signal tasks for the 3 data-sets. Note the higher percentage of stop trials in study 3.

Figure 2.

Event Related Spectral Perturbation Images (ERSP) for right frontal (RF) clusters in (A) study 1, (B) study 2 and (C) study 3. From left to right in each row: Mean cluster scalp projection; cluster-mean ERSP images time-locked to the go signal for go trials, successful stop trials, failed stop trials and the successful-failed difference. Nonsignificant differences are masked in transparent. In all three studies these RF clusters show a significant increase in beta band power after the stop signal that elapses before SSRT for successful stop trials. In study 1 and 2 this beta band increase is significantly larger for successful than failed stop trials.

Figure 3.

Event Related (ERPs) and mean cluster scalp projections for mid-central (MC) clusters in (A) study 1, (B) study 2 and (C) study 3. ERP plots are time locked to the stop signal and show a positive deflection in the EEG occurring around 300 ms after the stop signal (P300) previously shown in EEG studies (Wessel and Aron, 2015). For all three studies, P3 latencies occurred earlier for successful stop compared to failed stop trials relative to the stop signal.

Figure 4.

Event Related Spectral Perturbation Images (ERSP) for sensorimotor left (SML) clusters in (A) study 1, (B) study 2 and (C) study 3. From left to right in each row: Mean cluster scalp projection; cluster-mean ERSP images time-locked to the go signal for go trials, successful stop trials and failed stop trials and the successful-failed difference. Nonsignificant differences are masked in transparent. All three SML clusters show a significant decrease in mu and beta bands after the go signal for go trials and successful and failed stop trials. This decrease was most pronounced for failed stop trials > go > successful stop (in that order).

Figure 5. Event Related Spectral Perturbation Images (ERSP) for the right frontal (RF) cluster in study 2 for the unexpected events task. (A) From left to right: Mean cluster scalp projection; cluster-mean ERSP images time-locked to the sound for novel and standard sounds and the difference between the two conditions. (B) Task schematics for the unexpected events task; (C) Mean beta band power changes (solid line) for novel (red) and standard (blue) sound trials, and one standard error confidence interval (rose and turquoise areas).

Table 1

Reaction times and stop signal delays for each study.

	Go RT	FS RT	SSRT	SSD	p(stop)	error rates
Study 1 (manual responses, visual stop signals)	mean	488	415	247	241	.52
	SEM	45.0	31.3	20.3	13.3	.02
Study 2 (verbal responses, visual stop signals)	mean	557	495	257	300	.52
	SEM	14.8	13.9	6.4	13.3	.005
Study 3 (manual responses, visual stop signals)	mean	520	446	204	315	.52
	SEM	12.4	11.0	6.2	14.6	.005

Note: Go RT = go-trial reaction time; FS RT = failed stop-trial reaction time; SSRT = stop-signal reaction time; SSD stop-signal delay; SEM = standard error of the mean

Table 2

Number of contributing participants and ICs to each cluster

	Cluster RF (right frontal)	Cluster MC (midline central)	Cluster SML (left 'sensorimotor')
Study 1	10 Subj 13 ICs	9 Subj 9 ICs	10 Subj 12 ICs
Study 2	10 Subj 10 ICs	10 Subj 11 ICs	8 Subj 8 ICs
Study 3	19 Subj 22 ICs	20 Subj 23 ICs	20 Subj 21 ICs