It’s not too late: The onset of the frontocentral P3 indexes successful response inhibition in the stop-signal paradigm

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Abstract
The frontocentral P3 event-related potential has been proposed as a neural marker of response inhibition. However, this association is disputed: some argue that P3 latency is too late relative to the timing of action stopping (stop-signal reaction time; SSRT) to index response inhibition. We tested whether P3 onset latency is a marker of response inhibition, and whether it coincides with the timing predicted by neurocomputational models. We measured EEG in 62 participants during the stop-signal task, and used independent component analysis and permutation statistics to measure the P3 onset in each participant. We show that P3 onset latency is shorter when stopping is successful, that it is highly correlated with SSRT, and that it coincides with the purported timing of the inhibition process (towards the end of SSRT). These results demonstrate the utility of P3 onset latency as a noninvasive, temporally precise neural marker of the response inhibition process.

Descriptors: Inhibitory control, EEG/ERP, Individual differences, Stop-signal task

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ERP is rarely (if ever) observed in adults. In adults, the frontocentral P3 is the most common ERP index of successful response inhibition. Many studies have shown increased P3 amplitudes for successful versus failed stop trials in healthy individuals (Bekker, Kenemans, Hoeksma, Talsma, & Verbaten, 2005; Dimoska, Johnstone, Barry, & Clarke, 2003; Etchell, Sowman, & Johnson, 2012; Greenhouse & Wessel, 2013; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004; Lansbergen, Boker, Bekker, & Kenemans, 2007; Ramautar, Kok, & Ridderinkhof, 2004; Senderecka, Grabowska, Szewczyk, Gerc, & Chmylak, 2012; Wessel & Aron, 2013). Furthermore, as for the right frontolateral N2, participants with ADHD also show reduced frontocentral P3 amplitudes, with the degree of P3 amplitude reduction being predictive of the degree of stopping impairment that is typical for ADHD (Johnstone, Barry, & Clarke, 2007; Overtoom et al., 2002). Finally, the P3 is sensitive to whether participants prioritize successful stopping over quick going: when stopping is incentivized by monetary reward, the P3 amplitude increases (Greenhouse & Wessel, 2013). However, despite this body of evidence for the relation between successful response inhibition and the P3 ERP, it is disputed whether the P3 is a direct reflection of the response inhibition process. Several authors have argued that the P3 peaks too late relative to SSRT (Dimoska, Johnstone, Barry, & Clarke, 2003; Huster, Enriquez-Geppert, Lalavelle, Falkenstein, & Herrmann, 2013; Naito & Matsumura, 1996). Based on this observation, they suggest that, rather than reflecting response inhibition, the P3 instead reflects a different psychological process, such as the post hoc evaluation of performance (e.g., positive affect at a good outcome; for a review, see Huster et al., 2013).

One key to the debate about whether the P3 reflects successful response inhibition is whether the peak amplitude of an ERP is a good indicator for the timing of a neural process. A better indicator of the neural process reflected in an ERP could be the onset of the
Onset latency indexes response inhibition

All versions of the SST used here, SSD was initially set to 200 ms, which prompts participants to attempt to cancel the go response. In delay (stop-signal delay, SSD) following the onset of the go signal, which prompts participants to make a quick motor response. On a task (Logan et al., 1984). In the SST, a go signal is displayed, All tasks were slightly different versions of the standard stop-signal task (see below). This research was approved by the local ethics committee at UCSD provided written informed consent prior to participation in the studies. Overall, this resulted in a sample of 62 participants (mean age: slightly different versions of the stop-signal task (see below). Participants were pooled from two different sources: two previously published studies (Wessel & Aron, 2013; Wessel & Aron, in press) and two hereto unpublished studies. All of these studies used slightly different versions of the stop-signal task (see below). Overall, this resulted in a sample of 62 participants (mean age: 21.7; SEM: .72; 38 female; 2 left-handed). All participants provided written informed consent prior to participation in the studies. This research was approved by the local ethics committee at UCSD and performed in accordance with the Declaration of Helsinki.

Method

Participants
Participants were pooled from several SST studies into a large sample of 62 participants. In each participant, we applied independent component analysis (ICA; Jutten & Herault, 1991) to identify an independent component corresponding to the frontocentral P3 ERP. This approach increases the single-trial signal-to-noise ratio of an ERP (Wessel & Ullsperger, 2011), and furthermore disentangles it from other, potentially superimposed ERPs (Onton, Westerfield, Townsend, & Makeig, 2006). This increase in signal-to-noise ratio is key in quantifying the onset of an ERP in each individual participant. We then quantified the onset of the P3 in each participant using single-trial permutation statistics, and tested whether this P3 onset measurement related to the speed of stopping (measured by SSRT) across the sample. We tested three main hypotheses: (1) Does the latency of the P3 onset in each participant correlate with SSRT across participants? (2) Is the correlation between P3 onset and SSRT higher than the correlation between P3 peak and SSRT? (3) Is the timing of the P3 onset in each participant different depending on whether stopping is successful or not? We were also interested in finding out, with more temporal precision, when the P3 onsets occur relative to SSRT. Neural data and computational simulations suggest that the actual response inhibition process happens within a few milliseconds (~10 ms) prior to the end of SSRT (Boucher et al., 2007), whereas the remaining time before this period is related to other subprocesses of stopping (e.g., the sensory processing of the stop signal). We thus expected that, if the P3 onset relates to successful response inhibition and occurs before SSRT, the time of occurrence should be more precisely in the time window immediately preceding SSRT.

Behavioral Task
All tasks were slightly different versions of the standard stop-signal task (Logan et al., 1984). In the SST, a go signal is displayed, which prompts participants to make a quick motor response. On a subset of trials (stop trials), a stop signal is displayed with a certain delay (stop-signal delay, SSD) following the onset of the go signal, which prompts participants to attempt to cancel the go response. In all versions of the SST used here, SSD was initially set to 200 ms, and then dynamically adjusted according to participants’ ongoing performance. Specifically, SSD was increased by 50 ms in case of a successful stop trial, and decreased by 50 ms in case of a failed stop trial. This was done in order to achieve a probability of successful stopping [p(stop)] of around .5, which results in a race-like situation between the going and stopping on all stop trials (Verbruggen & Logan, 2009). Participants were instructed that going quickly and stopping successfully were equally important. All studies followed this general template. Differences between the samples concerned the following parameters: response domain (verbal or manual responses), stop-signal domain (visual or auditory stop signals), stop-signal probability (25% or 33%), trial numbers, block numbers, and trial timing. The exact details for each group of participants were as follows:

Sample 1 (verbal responses, visual stop signals): N = 12. Go signals consisted of the letter T or the letter K in white print in the middle of the screen. Participants were instructed to respond as quickly as possible by the letter into a Logitech USB microphone, unless a stop signal occurred. The stop signal consisted of the respective letter turning from white to red ink. 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, overall trial duration: 2,500 ms. The onset of verbal responses was detected from the audio recording by a custom online algorithm and checked for accuracy by offline inspection. (This dataset has been published as part of Wessel & Aron, 2013; see therein for details).

Sample 2 (manual responses, visual stop signals): N = 10. Go signals consisted of a rightwards or leftwards pointing white arrow in the middle of the screen. Participants were instructed to respond as quickly as possible by pressing a response button (left or right) with their right hand using a custom keypad, unless a stop signal occurred. The stop signal consisted of a red exclamation point appearing above 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, overall trial duration: 2,500 ms. (This study is currently submitted as part of Wessel & Aron, in press).

Sample 3 (manual responses, auditory stop signals): N = 14. Go signals consisted of a rightwards or leftwards pointing white arrow in the middle of the screen. Participants were instructed to respond as quickly as possible by pressing a response button (left or right) with their right hand using a custom keypad, unless a stop signal occurred. The stop signal consisted of a 100-ms sine-wave tone with a frequency of 900 Hz. 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, overall trial duration: 2,500 ms.

Sample 4 (manual responses, visual stop signals): N = 26. Go signals consisted of a rightwards or leftwards pointing white arrow in the middle of the screen. Participants were instructed to respond as quickly as possible by pressing a response button (left or right) with their right hand using a custom keypad, unless a stop signal occurred. The stop signal consisted of the arrow turning from white to red ink. 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, overall trial duration: 2,500 ms.
Behavioral Analysis

In order to ensure the validity of the race model, all participants' behavioral data were screened for whether mean go-trial reaction time (GoRT) was slower than the mean reaction time on failed stop trials (FsRT), and whether \( p(\text{stop}) \) was between .4 and .6 (which tests the efficacy of the SSD staircase algorithm). SSRT was computed according to the mean method (Verbruggen & Logan, 2009).

EEG Recording

EEG data were recorded using a BioSemi system (BioSemi Instrumentation, The Netherlands), using 64 scalp electrode sides in samples 1, 3, and 4 (512 Hz sampling rate), and 128 scalp electrode sites in sample 2 (1024 Hz sampling rate). Additional electrodes were placed on the bilateral mastoids (except for sample 2) and canthi, as well as below and above each eye. The data were online referenced to the BioSemi CMS-DRL (common mode sense-driven right leg) reference. In all samples, the offset from the reference was kept below 25 microvolts.

EEG Preprocessing

Data were preprocessed using custom routines in MATLAB 2012a (TheMathWorks, Natick, MA). ICA and dipole fitting (DIPFIT 2.2) were performed using functions from the EEGLAB toolbox (Version 9, Delorme & Makeig, 2004). On import into MATLAB, the data were rereferenced to Cz1 (according to the 10–20 system) in the 128-channel montage (sample 2), or a linked mastoid reference in the 64-channel montage (samples 1, 3, and 4; all datasets were later rereferenced to common average before analysis to ensure comparability across samples). The continuous time series was resampled to 512 Hz (in case of the 128-channel montage), and filtered using symmetric two-way least squares finite impulse response (FIR) filters (.5 Hz high-pass, 50 Hz low-pass). Then, the data were epoched with respect to the go stimulus, beginning at 500 ms before stimulus onset and ranging to 1,500 ms poststimulus onset. The time series were then visually inspected for bad channels and epochs with nonstereotyped artifact activity (e.g., from gross movement or spurious muscle activity). Such data were removed. The remaining data were rereferenced to a common average.

ICA and Component Selection

Each individual participant’s data were subjected to a temporal ICA decomposition using the infomax algorithm (Bell & Sejnowski, 1995; with extension towards subgaussian sources, Lee, Girolami, & Sejnowski, 1999). In case of the 128-channel montage, the data were reduced to 64 principal components first (in order to achieve a comparable ICA model order across all samples). The resulting component matrices were screened for independent components (ICs) representing stereotypic artifacts (blinks, saccades, and electrode artifacts) using outlier statistics (procedure as described in Wessel, Danielmeier, Morton, & Ullsperger, 2012). Such components were removed. On average, 19.6 components remained in the data \( (SEM: 1.6) \). The remaining components were fitted with individual inverse dipole solutions using the DIPFIT 2.2 algorithm. Components with nondipolar equivalent dipole solutions usually represent nonbrain signals (as defined by a residual variance of their equivalent dipole solution of greater than 15%; Delorme, Palmer, Onton, Oostenveld, & Makeig, 2012), and were also removed. The automatic classifications based on these criteria were visually screened for inaccurate classifications and manually rectified if necessary (two participants had ICs that had to be manually rejected, and seven had ICs that were manually retained). The remaining nonartifact components were subjected to further analyses.

From the remaining nonartifact components, we then selected one IC in each participant that represented the P3 ERP. This was done in order to disentangle the P3 ERP from other ongoing EEG processes that are unrelated to the P3 process, but might be superimposed on the ERP in channel space. By restricting the focus of investigation on the ERP of interest using this ICA approach, the single-trial signal-to-noise ratio of an ERP significantly increases (Wessel & Ullsperger, 2011). In order to select components that represented the P3, trials were grouped into successful stop trials, failed stop trials, and go trials. Errors (go trials with wrong response) and misses (go trials with no response) were removed from all analyses. Additionally, stop trials with an SSD < 100 ms were excluded, to ensure that successful stopping was sufficiently difficult and the result of a “true” race process. Furthermore, in order to appropriately match stop and go trial numbers, we selected only one go trial per stop trial, which was matched by SSD (e.g., for each successful or failed stop trial with an SSD of 300 ms, one go trial in which a stop signal would have appeared at 300 ms was selected). ERPs for stop trials were generated from −100 to 500 ms relative to stop-signal onset. ERPs for go trials were generated across the same time period, time-locked to the according time point in the ERP following the go signal (i.e., in a trial in which a stop signal would have occurred at 300 ms following the go signal according to the current SSD staircase, the ERP data were extracted around the 300-ms time point following the go signal). Mean ERP time courses and topographies can be found in Figure 1. We used a modified version of the COMPASS algorithm (Wessel & Ullsperger, 2011) to select an independent component per participant that represented the P3. Specifically, in order to qualify as the P3 component, an IC had to fulfill three requirements:

1. Topography: The topographical representation of the IC weight matrix had to have a local maximum (or minimum, as IC weight polarities are arbitrary) at frontocentral scalp electrodes (FCz or Cz in the 64-channel montage; A1, C1, D1, and C23 in the BioSemi 128-channel montage).
2. Time course: Out of all ICs that fulfilled the topographical criterion, we selected the one IC whose back-projected time course averaged across the frontocentral electrode sites correlated with the original ERP to the highest degree (the original ERP is the ERP based on the back projection of all nonartifact ICs). This was measured by a Pearson correlation coefficient.
3. Functional significance: The ERP back projection of the selected component to the frontocentral channels had to show significant differences between successful or failed stop trials and their respective matched go trials in a positive direction (i.e., stop trials had to show more positive voltage compared to the matched go trials) on at least one sample in the first 500 ms following the stop-signal onset. Significance was determined on a sample-by-sample basis, based on Monte Carlo truth-label switching permutation testing with a \( p \) value of .05, corrected for multiple comparisons using the false discovery rate procedure (FDR: Benjamini & Hochberg, 1995).

Seven out of the 62 participants had no component that fulfilled criterion 3 (i.e., the selected component did not show a significant stop-trial P3 in at least one sample). In most cases, this was explained by an absence of a visible P3 wave in the original ERP.
There was no discernible pattern regarding any particular behavioral abnormalities in these seven subjects. We suspect that the absence of a frontocentral P3 could be explained by slightly abnormal anatomy in these subjects. In the following, we focused our investigations on the remaining 55 participants.

**EEG Analysis**

The onset of the P3 was quantified for successful and failed stop trials separately, in order to compare P3 onset latencies depending on the success or failure of stopping (i.e., to test whether the P3...
onset latency shorter when stopping is successful). The P3 onset latency was defined as the earliest time point at which a statistically significant deviation between stop- and matched go-trial ERPs could be detected in a given participant. For this, single-trial amplitudes were subjected to samplewise, truth-label switching Monte Carlo testing, each of these variables was tested for outliers using Grubbs’ test at $p < .05$. Outliers were removed from further analyses. We then tested the hypotheses as follows: **Hypothesis 1** (P3 onset latency correlates with SSRT) was tested by means of a Pearson correlation between P3 onset latency on successful stop trials and SSRT. **Hypothesis 2** (P3 onset latency correlates more strongly with SSRT than P3 peak latency) was tested in two stages. First, the Pearson correlation between P3 peak latency on successful stop trials and SSRT was measured. Then, the magnitude of this correlation was tested against the magnitude of the correlation between P3 onset and SSRT using a method to compare correlation coefficients based on overlapping samples (Zou, 2007). **Hypothesis 3** (P3 onset latency is earlier for successful vs. failed stop trials) was tested by comparing the mean P3 onset latency on successful stop trials to the mean P3 onset latency on failed stop trials, using a paired samples $t$ test.

### Results

#### Behavior

Average GoRT was 519 ms ($SEM$: 7.5); average FsRT was 444 ms ($SEM$: 7.0). All participants exhibited a $p($stop$)$ between .4 and .6, indicating the efficacy of the SSD staircase algorithm. Furthermore, GoRT was longer than FsRT in all participants, indicating the validity of the race model. Average SSRT was 228.8 ms ($SEM$: 4.5; range: 142–312 ms). The distribution of SSRT values contained no outliers. Average SSD was 290 ms ($SEM$: 8.6). Miss rates and false alarm rates were low (miss rate: 1.1%; $SEM$: 2; error rate: .46%; $SEM$: .08). Data for the three different versions of the task (1—manual responses, visual stop signal; 2—manual responses, auditory stop signal; 3—verbal responses, visual stop signal) can be found in Table 1 (sample 2 and sample 4 were grouped because both samples consisted of manual responses and visual stop signals).

#### P3 Onsets

In order to qualify as a P3 component, either successful stop trials or failed stop trials (or both) had to show a significant P3 (see Method). Out of the 55 participants for whom such a component was found, all 55 showed a significant P3 for successful stop trials, and 50 also showed a significant P3 for failed stop trials. None of the participants showed a significant P3 for failed stop trials but no significant P3 for successful stop trials. One participant’s successful stop P3 onset was classified as an outlier and was excluded from

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### Table 1. Behavioral Data Split by Stop-Signal Task Type

<table>
<thead>
<tr>
<th></th>
<th>GoRT</th>
<th>FsRT</th>
<th>SSRT</th>
<th>SSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manual responses/visual stop signals</td>
<td>mean</td>
<td>499.35</td>
<td>418.85</td>
<td>234.66</td>
</tr>
<tr>
<td></td>
<td>$SEM$</td>
<td>9.02</td>
<td>7.29</td>
<td>4.48</td>
</tr>
<tr>
<td></td>
<td>min</td>
<td>405.99</td>
<td>353.92</td>
<td>161.31</td>
</tr>
<tr>
<td></td>
<td>max</td>
<td>637.80</td>
<td>571.98</td>
<td>290.84</td>
</tr>
<tr>
<td>Manual responses auditory stop signals</td>
<td>mean</td>
<td>536.42</td>
<td>465.52</td>
<td>191.71</td>
</tr>
<tr>
<td></td>
<td>$SEM$</td>
<td>15.52</td>
<td>13.53</td>
<td>8.88</td>
</tr>
<tr>
<td></td>
<td>min</td>
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<td>400.04</td>
<td>142.08</td>
</tr>
<tr>
<td></td>
<td>max</td>
<td>646.64</td>
<td>579.33</td>
<td>277.18</td>
</tr>
<tr>
<td>Verbal responses/visual stop signals</td>
<td>mean</td>
<td>556.96</td>
<td>493.31</td>
<td>254.28</td>
</tr>
<tr>
<td></td>
<td>$SEM$</td>
<td>15.20</td>
<td>14.81</td>
<td>7.33</td>
</tr>
<tr>
<td></td>
<td>min</td>
<td>472.27</td>
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<td>213.91</td>
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<tr>
<td></td>
<td>max</td>
<td>635.93</td>
<td>554.86</td>
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</tr>
</tbody>
</table>

*Note: GoRT = go-trial reaction time; FsRT = failed stop-trial reaction time; SSRT = stop-signal reaction time; SSD = stop-signal delay.*
Onset latency indexes response inhibition

Hypothesis 1: P3 Onset Latency Correlates with SSRT

In the sample of 54 participants, the average P3 onset for successful stop trials was at 225.3 ms (SEM: 4.5). SSRT was 227.5 (SEM: 4.8). Across participants, P3 onset latency on successful stop trials correlated strongly with SSRT (r = .60, p < 10^{-3}, Pearson correlation, Figure 3A). This means that the earlier the P3 reached significance on successful stop trials in a given participant, the faster they stopped.

Hypothesis 2: P3 Onset Correlates with SSRT Better Than the P3 Peak Does

The P3 peak for successful stop trials occurred at 297.6 ms (SEM: 8.6) on average. Like the P3 onset, the P3 peak on successful stop trials correlated significantly with SSRT (r = .37, p < .01, Pearson correlation, Figure 3B). However, this correlation was significantly lower compared to the correlation between the P3 onset and SSRT (p < .05; one-sided), showing that P3 onset latency is indeed a better predictor for the speed of stopping compared to the P3 peak. Further support for this finding comes from a partial correlation analysis between P3 peak latency and SSRT. This reveals that if the variance explained by the P3 onset latency is accounted for, there is no longer a significant correlation between the P3 peak latency and SSRT (r = .069; p = .62).

Hypothesis 3: P3 Onset Is Earlier for Successful Compared to Failed Stop Trials

Out of the 50 participants who had both a significant successful stop-trial and failed stop-trial P3, the onset of the failed stop P3 was at 259.9 ms (SEM: 6.0) on average. The successful stop P3 onset latency (225.2 ms in this sample of N = 50 (SEM: 4.5)) was earlier than the failed stop P3 onset in 43 out of those 50 cases (p < 10^{-3}, binomial test; see Figure 4). The mean difference in latency between the two onsets was 34 ms in this sample. t(49) = 6.9, p < 10^{-8}, paired t test; Cohen’s d: .90. This shows that the timing differences in P3 onset clearly reflect differences in stopping success: when the action was successfully stopped, the P3 onset occurred significantly earlier compared to when stopping failed.

Discussion

We used single-trial permutation statistics and an individual differences approach in a large sample of stop-signal task data to investigate the temporal relationship between the frontocentral P3 ERP and the timing of successful response inhibition. We found that the onset latency of the P3 on successful stop trials was highly correlated with the speed of the stopping process (as measured by SSRT), and also that this correlation between P3 onset and SSRT was significantly stronger than the correlation between P3 peak and SSRT. We further found that the onset of the P3 occurred significantly earlier on trials in which participants successfully stopped compared to when they failed, underlining the key importance of the timing of the onset of the P3 in determining the success of stopping. From this, we conclude that the timing of the P3 is directly related to the success of response inhibition within participants, as well as to the timing of successful response inhibition between participants.

On average, the P3 onset in this study preceded SSRT by only 2–3 ms. This is in line with the latency range predicted by modeling work and animal electrophysiology, which showed that response inhibition takes place immediately before the end of SSRT (Boucher et al., 2007). Notably, the criterion we used here to determine the onset of the P3 is relatively conservative; that is, it probably overestimates the P3 onset latency in favor of statistical reliability. One could argue that the true onset of the P3 actually occurs when the successful stop P3 starts to deviate from the go ERP (i.e., even before this deviation becomes significant). Indeed, if we defined the P3 onset latency as the time point at which the difference wave between successful stop trials and matched go trials became positive (and did not return to zero before significance was reached), it becomes even more evident that the P3 onset latency is clearly before SSRT; the average P3 onset latency according to this more progressive quantification was ~18 ms before SSRT (which was a statistically significant difference between SSRT and P3 onset latency; data not shown). Thus, a major implication of this study is that the P3 does occur in time to reflect response inhibition.
The approach used here provides several key advantages over prior studies. First, the unprecedented sample size (for an EEG study of the stop-signal task) provided a larger than usual degree of statistical power. This also enabled us to use an individual participants approach instead of the group-level comparisons that most regular ERP studies are based on. Second, by using several different versions of stop-signal task, which included different stop-signal modalities (auditory and visual) as well as motor response effectors (verbal and manual), we observed a sizable variance in SSRT, which further aided our attempt at an individual participants approach. Third, ICA served to disentangle the ERP signature of stopping from other ongoing EEG processes that could potentially be superimposed on the stopping ERP, such as motor ERPs (Bekker, Kenemans et al., 2005). The increase in the single-trial signal-to-noise ratio brought about by using this technique (Wessel & Ullsperger, 2011) enabled us to use permutation statistics to quantify the exact time point at which the P3 reached significance in each individual participant. This allowed us, for the first time, to directly relate the exact millisecond timing of the onset of an ERP in individual participants to their SSRT.

Apart from the P3, other ERPs are often reported in the context of stop-signal tasks. This is because stopping action is a complex behavioral sequence, which requires several psychological processes: at a minimum, successful stopping requires the perceptual processing of the stop signal (primary auditory or visual processing), the attentional detection of that signal, and, finally, response inhibition. In accordance with this, the frontalcent P3 (which we here argue to be a measurement of the motor inhibition process) is preceded by several other ERPs, some of which are similarly increased for successful versus failed stopping, but probably do not reflect the response inhibition process. First, the P3 is usually preceded by a frontalcent N2, especially in go/no-go type paradigms (Eimer, 1993). However, we believe that this N2 is unlikely to reflect the success of a motor inhibition process, as contrasts between successful and failed stop trials in the stop-signal task usually show that, if anything, the N2 tends to be increased for failed stop trials (Dimoska et al., 2006; Greenhouse & Wessel, 2013; Kok et al., 2004; Ramautar, Kok, & Riddervinkhof, 2006; Senderecka et al., 2012). Hence, we believe that the N2 is most likely to reflect attentional processes related to the stop signal (Schroger, 1993). Second, an increased right frontalateral N2 is often reported for successful compared to failed stop trials (Liotti et al., 2010; Pliszka et al., 2000; Schmajuk et al., 2006), and is hence a potential candidate to reflect the response inhibition process (like the P3). This is in line with a right-lateralized network for response inhibition in the stop-signal task, for which there is ample evidence from other imaging domains, as well as lesion studies (for a review, see Aron, Robbins, & Poldrack, 2014). In our current study, we did indeed observe a frontalateral N2 wave in the ERP based on all nonartifact ICs (see Figure 1). However, as can be seen from the figures, this ERP was not right lateralized. Importantly, though, it has to be mentioned that the right-frontolateralized N2 has so far been observed only in children and adolescents, for studies of response inhibition in ADHD, and not yet reported in healthy adults. It is possible that, in adults, frontalateral stopping-related activity cannot be easily measured using EEG. One reason for this could be increases in cortical folding from childhood to adulthood (Gogtay et al., 2004; Zilles, Palomero-Gallagher, & Amunts, 2013). As EEG is only susceptible to dipolar activity that is perpendicular to the scalp, increases in cortical folding in frontal brain regions across development could change the electrical dipole structure in right inferior frontal cortex, making it difficult to measure EEG activity from this area in adults. Lastly, early sensory ERPs can be selectively increased on successful compared to failed stop trials (Bekker, Kenemans et al., 2005; Greenhouse & Wessel, 2013; Lansbergen et al., 2007). In conclusion, we argue that, while multiple candidate ERPs exist that potentially map onto psychological processes associated with action stopping in the stop-signal task, our data support the theory that the P3 specifically reflects the response inhibition subprocess associated with action stopping, since earlier ERPs are either clearly mapped onto other processes (sensory N1), not selectively increased for successful versus failed stopping (frontocentral N2), or are not usually observed in adults (right frontolateral N2). However, the processes reflected in these ERPs likely contribute to the sequence of psychological processes that constitute successful action stopping.

Apart from the ERP approach, several recent studies have looked for alternative signatures of successful response inhibition in human scalp EEG. These studies mainly reported frontocentral time-frequency signatures in the theta (5–8 Hz) and delta (1–4 Hz) frequency bands, which show an increase on successful compared to failed stop trials, similar to the P3 (Lavallée, Herrmann, Weerda, & Huster, 2014; Nigbur, Ivanova, & Sturmer, 2011; Schmedt-Fehr & Basar-Eroglu, 2011; Wessel & Aron, 2013; Yamanaka & Yamamoto, 2010). However, while such time-frequency signatures have their utility, the ERP method has several advantages. First, ERPs can be derived without the advanced knowledge of digital signal processing necessary to appropriately conduct time-frequency analyses. Second, the low computational effort necessary to generate ERPs makes them potentially useful for brain-computer interfaces, in which quickly derived neural signals are needed for online feedback. Third, the high time resolution of ERPs allows for the testing of hypotheses that pertain to the relative timing of neural and psychological processes with millisecond precision. The current study is just one example that exploits this high temporal precision to test specific hypotheses about the relative timing of brain activity and a psychological process. Unlike ERPs, time-frequency signatures, especially in lower frequency bands such as the delta/theta range, cannot be measured with such temporal resolution, and do not allow inferences such as the ones made in the current study.

Of course, the P3 is not exclusive to the stop-signal task. Notably, frontocentral positive voltage deflections in the ERP around the 300-ms time range are commonly observed following unexpected or rare events (novels/oddballs; Courchesne, Hillyard, & Galambos, 1975; K. C. Squires, Squires, & Hillyard, 1975; N. K. Squires, Squires, & Hillyard, 1975). P3-like potentials have also been associated with decision making (Squires, Donchin, Squires, & Grossberg, 1977) and information processing (Duncan-Johnson & Donchin, 1982), and are elicited in many different experimental paradigms (for a review, see Polich, 2007). While it has been argued that the P3 reflects “inhibition of ongoing activity” following psychologically significant events of many types (Polich, 2007; also see Wessel & Aron, 2013), this does not necessarily mean that the occurrence of a P3 ERP signifies the presence of an inhibitory mechanism in any given context or task. Still, the data presented here strongly suggest that, within the context of the stop-signal task, the P3 is a clear-cut neural marker of the speed of a (response) inhibition process.

Linking the P3 with successful response inhibition provides a fruitful test bed for studies of response inhibition that require easily derived clinical markers (for a review, see Robbins, 2007;
Onset latency indexes response inhibition


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