How to withhold or replace a prepotent response:

An analysis of the underlying control processes and their temporal dynamics

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Abstract

The present study isolated and compared ERP components associated with flexible behavior in two action-control tasks. The 'withhold' groups had to withhold all responses when a signal appeared. The 'change' groups had to replace a prepotent go response with a different response on signal trials. We proposed that the same chain of processes determined the effectiveness of action control in both tasks. Consistent with this idea, lateral (Experiment 1) and central (Experiment 2) signal presentation elicited the same perceptual and response-related components in both tasks with similar latencies. Thus, completely withholding a response and replacing a response required a similar chain of processes. Furthermore, latency analyses revealed intra-individual differences: When the signal occurred in the periphery, differences between fast and slow change trials arose at early perceptual stages; by contrast, differences arose at later processing stages when signal detection was easy but stimulus discrimination and response selection were harder.

Changes in the environment or internal state often force us to update our actions or behavior in order to meet new requirements. In the laboratory, action control in response to an unexpected signal or cue has been studied using the stop-signal paradigm (e.g., Lappin & Eriksen, 1966; Logan, 1994; Verbruggen & Logan, 2008a), and the go/nogo paradigm (e.g., Donders 1868/1969; Wundt, 1880; Catell, 1886; Luce, 1986 for a review). The present study explored action control in different variants of these tasks.

The stop-signal and go/nogo paradigm

In the standard version of the stop-signal paradigm, subjects are instructed to respond to a go stimulus (e.g. press left for a left arrow and right for a right arrow), unless a stop signal appears after a variable delay. In the standard version of the go/nogo paradigm, subjects are instructed to respond when a go signal (e.g. 'O') appears, but to withhold their response when a nogo signal (e.g. 'X') appears. In the cued variant of the go/nogo task (Band, Ridderinkhof, van der Molen, 2003; Bekker, Kenemans & Verbaten, 2004; Bruin, Wijers, & van Staveren, 2001; Jonkman, Lansbergen & Stauder, 2003; Randall & Smith, 2011; Smith, Jonstone & Barry, 2006), a cue provides information about which response is probably required and subjects are asked to prepare this response (a key press with a left finger, a right finger, or no response). Whether or not the cued response is subsequently required is clarified by a second stimulus that follows after a variable delay.

In the literature, both stop-signal and go/nogo tasks have been used to study response inhibition. Neuroimaging studies suggest that both tasks require similar processes. ERP studies have shown that both nogo trials and stop-signal trials are associated with a N2 and a P3 (e.g., Simson, Vaughan & Ritter, 1977; Pfefferbaum,

Ford, Weller & Kopell, 1985; Eimer, 1993; Donkers & van Boxtel, 2004; Lavric, Pizzagalli & Forstmeier, 2004). Furthermore, fMRI studies found large overlap in the neural circuitry involved in stop-signal and go/nogo tasks. For example, the right inferior frontal cortex and the pre-supplementary motor area (pre-SMA) are activated on both stop-signal and nogo trials (for a meta-analysis, see Swick, Ashley, & Turken, 2011).

However, the meta-analysis of Swick et al. (2011) revealed some between-task differences as well, and they argued that the fronto-parietal control network was activated to a greater extent in the go/nogo task than in the stop-signal task. Furthermore, Eagle, Bari and Robbins (2008) reported only subtle neuroanantomical differences but large neurochemical differences. For example, serotonin seems to play an important role in inhibitory control on nogo trials but not on stop-signal trials. Finally, Schachar et al. (2007) found a dissociation between nogo performance and stop performance in children with ADHD but not in healthy control children.

Thus, the go/nogo and stop-signal tasks seem to require similar cognitive resources and neural pathways, but there appear to be some differences as well (especially in clinical populations and at a neurochemical level). The go/nogo task may place greater demands on action selection, whereas the stop-signal task may place greater demands on the motor inhibition system (Rubia et al., 2001). Furthermore, learning may play a greater role in standard go/nogo tasks than in stop-signal tasks (Verbruggen & Logan, 2008b).

Withholding vs. replacing a response

In daily life, people often have to replace the stopped or cancelled actions with a new action. To study this form of action control, variants of the stop-signal and go/nogo

paradigm have been developed. In the stop-change paradigm (Logan & Burkell, 1986; Verbruggen & Logan, 2009), subjects are instructed to stop their initially planned response in the primary task (hereafter referred to as the go1 response) when a stop-change signal is presented, and replace it with a new response (hereafter referred to as the go2 response). Others have used a similar variant of the cued go/nogo task (e.g. Band et al., 2013; Randall & Smith, 2011). In these studies, subjects had to cancel a prepared go response and execute an alternative response instead.

Behavioral and modeling work has tried to determine which processes are involved in replacing planned or prepotent responses. For example, Verbruggen, Schneider and Logan (2008) introduced in a stop-change paradigm a delay between the stop signal and the go2 signal to examine whether the go1 response can be inhibited simply by activating the go2 response (go1 ← go2) or whether it also requires a top-down inhibition process (go1 ← stop + go2). The results of two experiments were consistent with models that included a stop process. This conclusion is further supported by computational modeling studies. For example, Camalier et al. (2007) used an oculumotor variant of the stop-change task (i.e. the double-step paradigm). They fitted three computational models to the data of both humans and macaque monkeys. The models including the stop process fitted the data better than the model without it, suggesting that a stop process was required to explain performance in the oculomotor stop-change task.

Some studies suggest that the same inhibitory processes are involved when stopping all actions (as in the stop-signal task) compared to stopping the primary response (go1) and implementing an alternative one (go2) (as in the stop-change task). Based on their review of behavioral and neurophysiological data, Band and van Boxtel (1999) argued in favor a model consisting of a single inhibitory network,

which involves multiple cortical and subcortical structures. The majority of subsequent fMRI studies support this view because both stop signals and stop-change signals activate the hyperdirect fronto-basal-ganglia stopping network (e.g., Mars, Piekema, Coles, Hulstijn, & Toni, 2007; Kenner et al., 2010; Boecker et al., 2011; for a review of the stop-signal and stop-change comparison see Boecker, Gauggel, & Drueke, 2013).

However, some findings suggest that there might be differences as well. For example, the estimated latency of the stop process (stop-signal reaction time; SSRT) is often longer in the stop-change paradigm than in the standard stop-signal paradigm (e.g. de Jong, Coles, & Logan, 1995; Logan & Burkell, 1986), which could indicate that different inhibitory processes are involved in the two paradigms. Furthermore, the ERP literature has provided conflicting results. In one of the first stop-signal versus stop-change task comparisons, de Jong et al. (1995) compared the lateralized readiness potential (LRP), which is a marker of motor preparation, on signal trials in the two tasks. They found below-threshold motor activation on signal trials in the stop-change task but not in the stop-signal task. This led them to conclude that a fast but non-selective inhibition mechanism is involved in the stop-signal task (consequently, responses could be suppressed at late, peripheral stages), whereas a slower but more selective mechanism is involved in the stop-change task (consequently, responses would be suppressed at central stages). Subsequent studies using different stop-change paradigms were not able to replicate de Jong et al.'s LRP results (Band et al., 2003; Krämer, Knight, & Münte, 2011). Nevertheless, Krämer et al. (2011) still argued that different inhibitory mechanisms were involved in both tasks because they observed a fronto-central N2 component on stop-signal trials but not on stop-change trials. Boecker et al. (2013) argued that this N2 differences might

have been caused by the nature of the paradigm, which combined the Erikson flanker task with a stop/change-signal paradigm. Indeed, a recent ERP study (Rangel-Gomez, Knight, & Krämer, 2015) used a novel method (Laplacian transformation and independent component analysis, ICA) to disentangle activity elicited by the go stimulus from activity elicited by the stop and stop-change signals. This study found a bilateral parieto-occipital negativity around 180 ms and a fronto-central negativity around 220 ms for both stop-signal and stop-change trials, confirming previous fMRI results of a common inhibitory mechanism. Thus, an N2 can be observed when subjects have to withhold a response in the stop-signal paradigm and when they have to replace a response in the stop-change paradigm.

The cued go/nogo ERP literature also produced conflicting results. Some studies compared trials on which subjects had to cancel a planned go response (go/nogo) with trials on which they had to replace it (go/change) (e.g. Band et al., 2003; Randall & Smith, 2011). Band et al. observed an N2 in both conditions (although there were some subtle differences), and proposed that similar inhibitory mechanisms might be involved. Randall and Smith (2011) also observed an N2 in both conditions. However, compared with the go condition, they found a P3 in the nogo condition but not in the change condition. They proposed the N2 reflects conflict detection, whereas the P3 would reflect the cancellation of a planned response. In other words, they argued that inhibition was only involved in the go/nogo condition.

In sum, it is still unclear to what extent withholding a response and replacing a response require similar cognitive and neural mechanisms. We addressed this issue in the present study using ERPs.

The present study

The stop-signal task puts higher demands on motor inhibition than most variants of the go/nogo task. However, a methodological challenge of combining the stop-signal task with ERPs is the short succession of the go stimulus and the signal, which leads to an overlap of neural activity associated with the two stimuli (see Bekker, Kenemans, Hoeksma, Talsma & Verbaten, 2005, for a discussion), complicating the interpretation of ERP modulations. Several procedures (which are discussed in more detail in the General Discussion) have been proposed to disentangle the activation patterns, but they can be complex. Furthermore, the refractoriness of ERPs could still lead to a false assessment of signal trial amplitudes (Woodman, 2010). To address these issues, the present experiments introduce a hybrid version of the stop-signal task and the cued go/nogo task (note that it also shares some features with the response-priming paradigm; see Schmidt, Haberkamp, & Schmidt, 2011 for a review of the original response-priming tasks). Our paradigm (see Figure 1A) greatly reduces the amount of overlap caused by two successive stimuli. This is particularly important for the assessment of early perceptual components following signal onset.

On each trial, subjects saw a digit (the go stimulus) and they were instructed to prepare a response on all trials. After one second, a signal appeared. On most trials, the signal instructed subjects to execute the planned response. But on a minority of the trials, a withhold/change signal appeared. Subjects in the 'change' condition were instructed to replace the planned go response with a different response on signal trials, whereas subjects in the 'withhold' condition[1] were instructed to cancel all responses on signal trials. The majority of the trials were go trials, and we forced subjects to respond as quickly as possible by using a tracking procedure to adjust the response

[1] Whether the task is more similar to a nogo task or a stop-signal task is debatable. Therefore, we use 'withhold' instead of 'stop' or 'nogo'.

deadline. Thus, responding was prepotent in this task (in fact, due to the short time intervals and the highly predictable nature of events, responding could be initiated before the go cue appeared on the screen). We anticipated that this would increase the inhibitory-control demands on withhold/change trials (making this a stop/nogo hybrid).

We combined this paradigm with event-related potentials (ERPs) to offer a detailed description of the neural processes involved in withholding and replacing a response. The first aim of the present study was to explore the overlap between ERP components generated when subjects are required to simply withhold a response compared to when they have to implement an alternative response. Several studies show that a chain of processes determines the effectiveness of response inhibition on stop-signal trials, including perceptual, decisional and motor related processes (e.g., Bekker, Kenemans, et al., 2005; Boucher, Palmeri, Logan, Schall, 2007; Elchlepp, Lavric, Chambers & Verbruggen, 2016; Logan, Van Zandt, Verbruggen, 2014; Logan, Yamaguchi, Schall, & Palmeri, 2015; Overtoom et al., 2009; Salinas & Stanford, 2013; van de Laar, van den Wildenberg, van Boxtel, & van der Molen, 2010; Verbruggen & Logan, 2015; Verbruggen, Stevens, & Chambers, 2014). Therefore, we performed a detailed process analysis (using ERPs) to examine the overlap between our 'withhold' and 'change' tasks.

The second aim of the study was to explore at which processing stages inter-trial differences in the change task arise. As noted in the previous paragraph, in simple stop-signal tasks, successful stop performance requires more than a single neural inhibitory process. Therefore, researchers should consider at which processing stage(s) differences between groups or conditions arise (Verbruggen & Logan, 2015). For example, Bekker, Kenemans et al. (2005) used a stop-signal task with an auditory

stop-signal and found that N1 amplitudes were larger for successful stops than for failed stops. This led them to conclude that inhibitory performance depends to a certain extent on switching attention to the stop signal. Similarly, Boehler et al., (2009) showed that early perceptual processing of the task stimuli strongly influenced the success of stopping. In particular, enhanced processing of the go stimulus improved motor execution (making stopping more difficult) and enhanced processing of the signal improved inhibition performance. It is likely that performance differences in change paradigms can also arise at various processing stages. Therefore, we compared fast and slow change trials, and examined which ERP components differed.

Experiment 1

In our tasks, on each trial a digit (the go1 stimulus) was presented in the center of the screen and was flanked by two letters (M's or W's). Subjects were instructed to classify the digit as lower or higher than 5 and prepare their response in a preparation interval. They were told they could only respond when a go cue was on the screen. Once the go cue disappeared, they could no longer respond. This response window was adjusted with a tracking procedure, pushing subjects to fully prepare their response and react quickly. In other words, responding was prepotent. On the remaining one third of the trials the arrows did not appear; instead, one of the flanking letters changed. In the change version of the task, subjects had to withhold the prepotent response to the digit and respond with their left or right foot to the location of the changing letter instead (Figure 1A). In the withhold version of the task, subjects had to withhold their digit response when one of the flanking letters changed.

By comparing peak latencies and amplitudes of ERP components in the two tasks, we could explore to what extent similar processes were involved in withholding and replacing a response. Note that we used a between-subjects manipulation. A within-block manipulation (with task cues indicating whether subjects had to withhold or replace responses) could encourage subjects to treat both tasks as one, with the only difference what to do with the signal (respond to it or not). Blocking the conditions could lead to differences in practice or learning effects. Therefore, we opted for a between-subjects design. Note that this also helped to reduce the length of each individual session.

To map the chain of processing on withhold and change trials, we examined in Experiment 1 a series of ERP components, namely the N1pc, P2pc, N2pc, the N2 and the P3. These components were selected before data collection, based on a pilot study[2]. The first three components following signal onset (N1pc, P2pc, N2pc) are related to signal detection and visual attention. When a visual signal is presented, it has to be processed, which typically results in a component that peaks around 150 ms after signal onset, the visual N1. When stimuli are presented laterally (as in the present experiment), the N1 is larger contralateral to the visual field of the stimulus (e.g., Johannes, Muente, Heinze, & Mangun, 1995; Wascher, Hoffman, Saenger, & Grosjean, 2009), also termed N1pc. It is often found that N1pc amplitudes are larger when more attention is directed to processing the visual stimulus (e.g., Luck, Fan & Hillyard, 1993; Wascher & Beste, 2009). The N1(pc) is followed by the P2(pc). Most researchers assume that the P2 relates to some aspects of higher-order perceptual processing, such as matching of sensory input to stored memory (e.g., Luck &

[2] In this pilot study (N = 20), we used a task that was very similar to the change task in Experiment 1. The only difference was that subjects executed all go1 responses with the right hand. On change trials, we observed lateralized N1pc, P2pc, N2pc components, a N2 and a P3.

Hillyard, 1994; Freundberger, Kliemesch, Doppelmayr & Holler, 2007). Third, the N2pc is elicited when stimuli are presented laterally and appears contralateral to the side of stimulus presentation. It may reflect a filtering mechanism, allowing one object to receive attention while others are ignored (e.g., Luck & Hillyard, 1994; Woodman & Luck, 2003). Eimer (1996) showed that the N2pc is not only elicited by pop-out items in a multi-item array but also when two stimuli are presented together. This led to the suggestion that the N2pc reflects covert, consciously directed attention. In sum, the existing literature suggests that the N1(pc), P2(pc), and N2pc are suitable markers of (top-down) perceptual mechanisms related to signal detection and analysis.

After the signal is detected and its features are analyzed, the appropriate response needs to be selected. The processes leading up to response selection in stop-signal and go/no-go paradigms are typically reflected in the N2 and the P3. The N2 was previously linked to response inhibition based on the relationship between its amplitude with inhibition efficiency (van Boxtel, van der Molen, Jennings & Brunia, 2001). However, later studies have shown that it is also present when a response is required, for example when go trials are unexpected or when maximal force go trials are compared with normal force go trials (e.g. Donkers & Van Boxtel, 2004; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Smith, Smith, Provost & Heathcote, 2010). This led to the interpretation that the N2 reflects conflict monitoring, more precisely the monitoring of concurrent and competing stimulus- and response representations, with larger amplitudes when conflict is increased (Botvinick, Cohen & Carter, 2004; van Veen & Carter, 2002).

The P3 may reflect resource allocation during the response decision process (Johnson & Donchin, 1982; Johnson, 1984; Kramer, Wickens, & Donchin, 1985)

and/or linking the decision with the correct response (Verleger, Jaskowski, & Wascher, 2005). P3 latency is thought to reflect the speed of the classification process (Kutas, McCarthy, Donchin, 1977; Magliero, Bashore, Coles, & Donchin, 1984). The P3 has also been linked to response inhibition because P3 amplitudes are typically larger for successful versus failed stop trials in healthy adults (Bekker, Kenemans et al., 2005; Dimoska, Johnstone, Barry, & Clarke, 2003; Greenhouse & Wessel, 2013; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004; Lansbergen, Bocker, Bekker, & Kenemans, 2007; Ramautar, Kok, & Ridderinkhof, 2004; Senderecka, Grabowska, Szewczyk, Gerc, & Chmylak, 2012) and larger for nogo than go trials (see Huster, Enriquez-Geppert, Lavallee, Falkenstein & Herrmann, 2013 for review). Furthermore, P3 onset latency is shorter when stopping is successful and correlates with SSRT (Wessel & Aron, 2015). Hence, the P3 may be associated with the decision to withhold a response and/or the selection of the alternative response on change trials.

We analyzed two additional components in Experiment 1. First, we analyzed the lateralized readiness potential during the preparation interval to assess the degree to which subjects had prepared the go response to the digit. Preparing a manual response (i.e. the go1 response in this experiment) results in a negative wave over the motor cortex contralateral to the response hand (the Readiness Potential, e.g., Deecke, Grozinger, & Kornhuber, 1976). Thus, the amplitude difference between electrodes positioned over the ipsi- and contralateral cortex can be used as a marker of response preparation in our task. Second, in tasks where an informative stimulus (in our case, the digit) precedes the imperative stimulus (in our case, the go cue), a Contingent Negative Variation (CNV) is typically elicited in the between-stimulus interval (Walter, Cooper, Aldridge, McCallum & Winter, 1964). Its amplitude varies as a

function of the attention required to perform a task and the extent to which the first stimulus predicts the second (Walter et al., 1964). Particularly the later part of the CNV has also been associated with the expectancy and preparation of a response (e.g., Brunia & Damen, 1988; McCallum, 1988). Therefore we added a post-hoc test of CNV amplitudes as a measure of stimulus anticipation and response preparation[3].

The lateralized signal display in Experiment 1 allowed a careful study of signal detection, signal analysis and decisional processes in the change task and a direct comparison of change and withhold performance (after all, computational work indicates that most of the stopping latency is occupied by perceptual and decisional components; see above). However, the lateralized signal display complicates the interpretation of the (motor-related) LRPs in the post-signal interval because the lateralized perceptual ERPs can spill over into central electrodes (Smulders & Miller, 2012; note that the LRP during the preparation interval was not influenced by lateralized perceptual ERP components because the digit was always presented centrally). To investigate execution- or motor-related processing stages in the change task, in Experiment 2 we changed our tasks so that the withhold/change signals appeared in the center of the screen (see Figure 1B). In sum, we used the excellent temporal resolution of ERPs to examine similarities or differences between withholding and changing responses. Further, we tested at which processing stage inter-trial differences arose by contrasting ERP waveforms associated with fast and slow change performance.

Method

Subjects. Forty right-handed adults (20 in the change condition, 13 females; 20 in the withhold condition, 12 females) with an average age of 20 (ranging from 18

[3] We thank two anonymous reviewers for this suggestion.

to 22) received two course credits or were paid £10 for their participation in this study. No subjects were excluded or replaced. Subjects did not differ significantly between the change and withhold groups in age ($p = 0.3$) or gender ($p = 0.8$). All present experiments were approved by the local research ethics committee at the School of Psychology, University of Exeter. Written informed consent was obtained after the nature and possible consequences of the studies were explained.

Apparatus, stimuli, paradigm, and procedure. The experiments were run on a 21.5-inch iMac using Psychtoolbox (Brainard, 1997). Go1 stimuli were white digits (1,2,3,7,8, or 9), presented against a black background in the center of the screen. They were flanked to the left and right by the letter M or W (white, uppercase font, 0.8cm x 0.8 cm). For the go1 response, subjects pressed the up or down arrow key on the computer keyboard; for the go2 (change) response in the change condition, subjects pressed a left or right foot pedal (Fragpedal Quad – USB Gaming Foot Pedal).

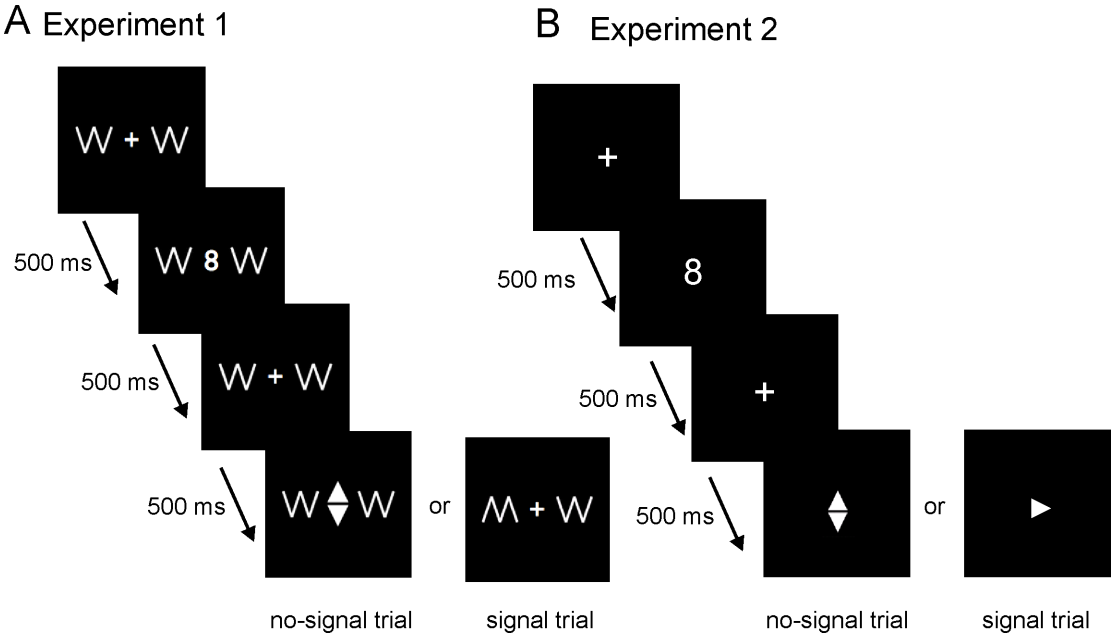

Fig. 1. Trial sequence (A) Experiment 1 (B) Experiment 2. Subjects had to execute the prepared go response when the up/down arrows appeared, but they had to

withhold or change their response when a letter changed (Experiment 1) or when a right (or left) arrow appeared (Experiment 2).

The sequence of events in a trial is shown in Figure 1A. The trial started with a fixation cross in the center of the screen flanked by the letters M or W (*fixation interval*). The cross was always flanked by Ms in half of the blocks, and by Ws in the other blocks. The M- and W-blocks alternated throughout the experiment, and starting order was counterbalanced across subjects. After 500 ms, one of the digits replaced the fixation cross and remained on the screen for 500 ms (*stimulus-presentation interval*). After that, the fixation was shown again for 500 ms (*preparation interval*). Subjects were told to use this interval to prepare the response to the digit. Finally, on no-signal trials, the fixation cross was replaced by up- and downwards pointing arrow cues (*go interval*); on change-signal trials (change condition) and withhold-signal trials (withhold condition), the fixation remained on the screen but one of the flanking letters changed (*signal interval*).

On no-signal trials (2/3 of trials), the arrows instructed subjects to execute the go1 response: they had to press the 'down arrow' key for digits smaller than 5, or the 'up arrow' key for digits larger than 5, using the left or right hand. The response hand alternated from block to block and the starting block was counterbalanced. The duration of the go interval (during which subjects could execute their go1 response on no-signal trials) was initially 500 ms and was subsequently adjusted with a 3-down/1-up tracking procedure: after every three go1 responses executed within the go interval, the response deadline decreased by 50 ms, encouraging subjects to respond faster. When they failed to respond in time, the deadline was increased again by 50 ms. Immediately after the response was executed or after the deadline had passed, the

stimuli were replaced by a feedback message for 1s. We presented ‘too soon’ when subjects had responded before the arrow cues were shown; ‘too slow’ if they pressed a key after the deadline had elapsed; ‘correct’ if they pressed the correct response key during the go interval; and ‘incorrect’ if they had pressed the incorrect key.

On signal trials (1/3 of trials) the left or right letter changed (from M to W, or from W to M; see Figure 1A). In the change condition, subjects had to cancel their planned go1 response to the digit, and instead respond to the location of the changed letter by pressing down the corresponding foot pedal (the go2 response; e.g., a left foot response for a letter changing on the left). Left- and right responses occurred with equal probability, and the order was randomized. The foot response had to be executed within 2,500 ms. Immediately after a response was executed or when the deadline had passed, we presented feedback for 1s: ‘change: incorrect’ if subjects executed the response to the digit (i.e. the go1 response) or pressed the wrong pedal (i.e. an incorrect go2 response), and ‘change: correct’ if they had executed the correct go2 response. In the withhold condition, subjects only had to cancel the go1 response on signal trials. On successful withhold trials the feedback ‘correct’ was shown, on failed withhold trials the feedback ‘try to stop’ was presented.

At the beginning of the experiment, subjects were instructed to respond as quickly and accurately as possible on all trials. Furthermore, they were explicitly discouraged to move their eyes or blink during the digit-response interval. In both conditions, there were eight blocks with 96 trials each (768 trials in total: 512 go trials, 256 signal trials). The experiments lasted around 40 minutes.

EEG/ERPs. The electroencephalogram (EEG) was acquired using 64 Ag/AgCl active electrodes (ActiCap, Brain Products, Munich, Germany) connected to BrainAmp amplifiers (Brain Products, Munich, Germany). The EEG was sampled

continuously at 500 Hz with a bandpass of 0.016-100 Hz with the reference at Cz and the ground at AFz. There were 62 electrodes on the scalp in an extended 10-20 configuration and one on each earlobe (see Figure S1 in the Supplementary Materials for electrode set up). Their impedances were kept below 10kΩ. The EEG was off-line filtered with a 20Hz low-pass filter (48dB/oct). To correct eye blink artifacts, we ran an Independent Component Analysis (Infomax ICA, Bell & Sejnovski, 1995, implemented in Vision Analyzer, BrainProducts, Munich, Germany). ICA components from every participant's EEG were inspected and those with characteristic eye-blink and eye-movement topographies were subtracted from the EEG. We re-referenced the EEG to the linked ears before segmenting. Only the EEG corresponding to correct no-signal and signal trials was used for ERP analyses. The EEG was cut into 1,700 ms long segments starting 100 ms before digit onset and ending 600 ms after onset of the go1 cue or change signal (see Figure 1 for the trial sequence). It was baseline corrected to the 100 ms preceding digit onset. Segments were further inspected visually for any remaining muscle artifacts, large drifts or remaining blink artifacts. Segments showing those artifacts were excluded. 26% of the trials (no-signal: 25%, signal: 27%) were excluded on the basis of behavioral performance (i.e. because the response was incorrect or not executed within the appropriate interval). A further 21% of the trials were excluded due to EEG artifacts. After segmenting into conditions, segments were averaged for each subject containing on average 274 segments for no-signal trials and 138 for signal trials.

We performed four sets of ERP analyses. First, we performed a manipulation check to test whether subjects used the preparation interval to prepare their response to the digit and whether the degree of preparation was similar in both paradigms. Second, we contrasted no-signal and signal trials and tested whether this between-trial

difference was similar in both versions of the paradigm (Study Aim 1). Third, we contrasted latencies and amplitudes of ERP components elicited by the signal in the change and withhold versions (Study Aim 1). Fourth, we compared latencies and amplitudes of ERPs associated with fast change responses versus slow change responses (Study Aim 2).

To confirm that subjects prepared the go1 response after they classified the digit, we analyzed the LRP in the preparation interval (remember that this LRP is not influenced by lateralized perceptual ERP components because the digit was always presented centrally). To calculate the LRPs for hand responses, we subtracted the amplitude in an electrode positioned over the motor cortex ipsilateral to the responding hand (C4 for right hand and C3 for left hand responses) from the amplitude in a contralateral electrode (C3 for right and C4 for left hand responses) and averaged the results of the left and right hand subtractions. We averaged LRP amplitudes in a time window starting 200 ms preceding the go/change stimulus until go/change stimulus onset. In this period, subjects should have prepared the go1 response, but they do not know yet whether a go1 cue or a stop/change signal will appear. We included amplitudes of all trials except those on which the go1 response was given too late (those trials were also excluded from subsequent ERP analyses), and submitted them to a one-tailed t-test assessing whether they are different from zero (see Band et al., 2003 for a similar assessment of motor preparation in the response-priming paradigm).

To test for differences between no-signal and signal trials, we focused on the N2 and P3 components because previous go/nogo and stop-signal ERP studies have linked these components with signal- or conflict detection, conflict resolution, response selection, and/or inhibition on signal trials (for review see Huster et al.,

2013). For statistical analysis of the N2, we averaged amplitudes in electrodes Fz, FCz and Cz; for the analysis of the P3, we averaged amplitudes in electrodes FCz, Cz and Pz[4]. We further averaged amplitudes in time windows around the N2 and P3 peaks (time windows were chosen on the basis of the N2 and P3 peaks on signal trials in the grand average waveform) because most subjects did not show an N2 peak or an unambiguous P3 peak on no-signal trials. In both conditions, the time window was 150-250 ms for the N2 and 300-400 ms for the P3. Thus, for the no-signal vs. signal comparison, the N2 and P3 are defined as the 'no-signal minus signal trial' difference wave in the respective time intervals (e.g., Luck, 2005).

To compare change-signal and withhold-signal trials, we analyzed the N1pc, P2pc, N2pc, N2, and P3. We used the "LRP formula" to calculate the lateralized visual components N1pc, P2pc and N2pc. More specifically, we subtracted the amplitude in an electrode positioned over the occipital cortex ipsilateral to the side where the stimulus was presented on the screen (PO8 for presentation on the right side and PO7 for the left side) from the amplitude in the corresponding contralateral electrode (PO7 for presentation on the right side and PO8 for left side) and averaged the results of the left and right subtractions. This subtraction procedure eliminates all amplitude differences that are non-lateralized, including differences in anticipation of the signal. However, we could not use this subtraction procedure for the N2 and the P3. This is problematic because we observed a CNV during the preparation interval, which potentially has not resolved by N2 and/or P3 onset. We therefore calculated the N2 as the P2-N2 peak-to-peak difference in electrode FCz. We quantified the P3 amplitude as the N2 to P3 peak-to-peak difference. The peak-to-peak method,

[4] Electrodes were chosen on the basis of previous studies (e.g., Eimer, 1993; Bekker, Kenemans et al., 2005; Bekker, Overtoom et al., 2005; Smith et al., 2006; Folstein & van Petten, 2008), knowledge about the neural generators of the N2 and P3 (e.g., Huster et al., 2013) and our pilot study.

although widely used in the ERP literature, does not necessarily solve the problem of the overlapping CNV to 100% because it assumes that the CNV resolves the same way in each condition. Therefore, we also ran a temporal PCA to disentangle the CNV from the subsequent N2 and P3. More details about this rationale and results of the analysis can be found in the Supplementary Materials. Note that for the analysis of the CNV itself, we averaged amplitudes in the 200 ms preceding the signal in electrodes FCz, Cz and Pz.

The P3 typically has a broad scalp topography, which can be subdivided into a fronto-central component, the P3a and a more posterior component, the P3b. According to Polich (2007), the 'P3a originates from stimulus-driven frontal attention mechanisms during task processing, whereas P3b originates from temporal–parietal activity associated with attention and appears related to subsequent memory processing.' (Polich, 2007, p. 2128). To be sensitive to potential differences between the change and withhold conditions with regards to both of these processes, we tested amplitudes and latencies separately in electrodes FCz, Cz and Pz (rather than averaging over these electrodes as in the other comparisons).

For the change condition, we also performed a series of post-hoc analyses to link the ERP components to behavior on change-signal trials. Using a median split, we divided the successful change-signal trials into fast and slow change-signal trials (resulting in two averages with approximately 70 trials per average). We then averaged the ERP segments associated with fast and slow change responses and tested whether component latencies were significantly earlier and amplitudes larger for the fast- compared with the slow trials. The lateralized sensory components (N1pc, P2pc, N2pc) were computed as described above. Without the subtraction procedure, amplitudes for fast and slow trials already differed substantially before and following

signal onset. For the quantification of N2 amplitudes we therefore baseline corrected amplitudes to the interval from 50 ms before to 50 ms after signal onset[5] (see e.g., Karayanidis, Coltheart, Michie & Murphy, 2003; Nicholson, Karayanidis, Bumak, Poboka & Michie, 2006; Nicholson, Karayanidis, Poboka, Heathcote & Michie, 2005, for a similar approach). P3 amplitudes, averaged over electrodes FCz, Cz and Pz, were quantified as the N2 to P3 difference. As discussed above, we could not examine response selection on change trials by means of stimulus-locked LRPs because stimulus presentation was lateralized.

To compare no-signal and signal trials, we used paired t-tests and mixed ANOVAs (with trial type as within-subjects factor and condition as between-subjects factor). To directly compare change and withhold trials, we used independent samples t-tests and Bayes factors to compare latencies and amplitudes of the N1pc, P2pc, N2pc, the N2 and P3 in the change condition with those in withhold condition. Unlike the t-tests, Bayes factors can provide support the null hypothesis.

We performed more than one test in each set of statistical analyses. To correct for family-wise error in multiple t-tests we used the Holm-Bonferroni method (Holm, 1979). This method adjusts the threshold of significance depending on the number of tests performed. In the tables and main text, we report the original p-values and indicate with a star whether results are still significant after the adjustment of the significance threshold. For some analyses, we also ran Bayesian t-tests but one does not have to correct these for multiple comparisons (Dienes, 2011).

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[5] We could also have used a peak-to-peak (P2 to N2) measure to quantify N2 amplitudes, as we did in all cases when there was a clear component preceding the component to be tested. In this case we used baseline correction to be in line with the N2 fast-slow amplitude comparison between the withhold and change conditions of Experiment 2. In those experiments there was no component preceding the N2 in the electrode tested (FCz).

Results

All data are deposited on the Open Research Exeter data repository.

Behavioral data.

Change condition. On no-signal trials, probability of a correct go1 response was .74 (*sd* = .04); probability of an incorrect go1 response was .03 (*sd* = .02); probability of an anticipatory go1 response (i.e. a response executed before the arrows appeared) was .02 (*sd* = .01); and probability of a missed go1 response was .21 (*sd* = .004). The latter indicates that our tracking procedure worked well. Mean correct go1 RT (relative to the presentation of the go1 cue) was 293 ms (*sd* = 58).

On change-signal trials, probability of a correct go2 response was .70 (*sd* = .19). On failed change-signal trials, the probability of executing the prepared go1 response was .90 (*sd* = .09) and the probability of an incorrect go2 response was .07 (*sd* = .06); the probability of pressing an irrelevant key (e.g., an incorrect go1 response) was .02 (*sd* = .06). Mean correct go2 RT on change trials was 518 ms (*sd* = 104). Go1 responses on change-signal trials (i.e. signal-respond trials) had a mean latency of 248 ms (*sd* = 69). This was significantly shorter than go1 latency on no-signal trials (by 45 ms), $t(19) = -3.11$, $p = 0.006$, $d_{av} = 0.71$. The RT difference is consistent with the independent horse-race model (Logan & Cowan, 1984), which assumes that the mean no-signal go1 RT represents the mean of all responses (including the longer right-tail of the RT distribution), whereas the mean go1 RT for signal-respond trials includes only responses that were fast enough to escape inhibition. Note that this RT difference also indicates that subjects had prepared a response and did not just wait for the go1 cue/signal to occur.

Withhold condition. On no-signal trials, probability of a correct go1 response was .77 (*sd* = .02); probability of an incorrect go1 response was .01 (*sd* = .01); probability of an anticipatory go1 response was .005 (*sd* = .01); and the probability of a missed go1 response was .21 (*sd* = .003). Subjects responded on average within 265 ms (*sd* = 26) to the digits.

On withhold-signal trials, probability of an incorrectly executed go1 response was .24 (*sd* = .13). Mean go1 RT on failed withhold trials (201 ms, *sd* = 33) was 64 ms faster than on mean go1 RT no-signal trials, $t(19) = -8.3$, $p < 0.001$, $d_{av} = 2.15$. Again, this is consistent with the independent race model, and confirms that subjects prepared their go response during the preparation interval.

ERPs.

Manipulation check. Figure 2 shows the hand preparation LRPs for the preparation interval. In both conditions, LRPs averaged in the 200 ms preceding the onset of the go1 cue or withhold/change signal differed significantly from zero (-0.4μV), $t(19) = -2.5$, $p = 0.01$ (one-tailed), $d_{av} = 1.1$ (change condition), and (-0.22μV), $t(19) = -1.88$, $p = 0.037$ (one-tailed), $d_{av} = 0.83$ (withhold condition). This indicates that subjects prepared their response to the digit. The LRPs were similar in both conditions, $t(38) = -0.86$, $p = 0.23$, $d_{av} = 0.4$, $BF = 0.41$. Thus, in both conditions, responding was prepotent, just like in other variants of the go/no-go and stop paradigms. The magnitude of the preparation LRP, although significant, appears quite small, which could be due to the potential requirement to withhold or change the prepared response and/or the fact that the response hand only changed across blocks, which avoids between-hand competition on a trial-by-trial basis[6].

[6] We thank an anonymous reviewer for this suggestion.

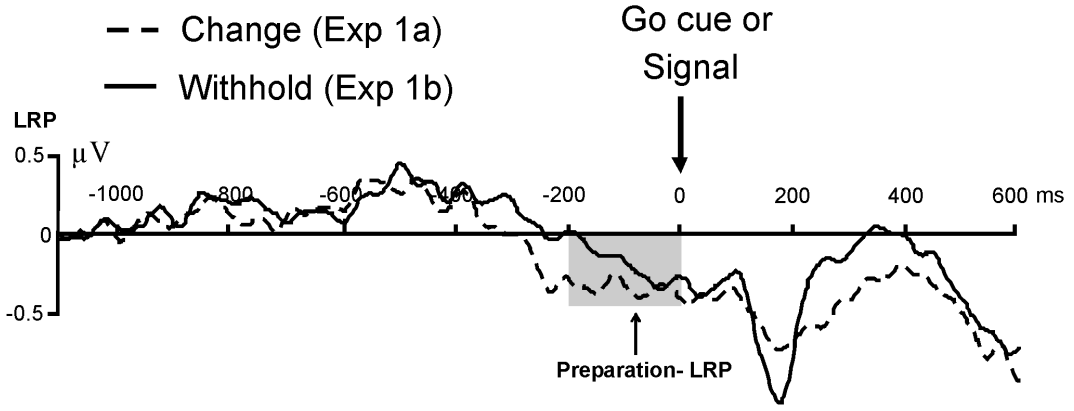

Fig. 2. Experiment 1: LRPs in preparation of the go1 response

No-signal vs. signal trials.

Change condition. For comparison with the existing literature, we examined the differences between change-signal and no-signal trials after presentation of the go1 cue or change signal. Waveforms for change-signal and no-signal trials in electrodes Fz, FCz, Cz and Pz are depicted in Figure 3A. Mean N2 amplitude for change-signal trials (calculated as described in the Methods section) was -3.7 μV; the amplitude for no-signal trials was 0.6 μV. This amplitude difference was significant, $t(19) = 4.0$, $p = 0.001$*, d_{av} =0.608. Mean P3 amplitude was 7 μV for change-signal trials and 4.3 μV for no-signal trials; this difference was also significant, $t(19) = 3.1$, $p = 0.006$*, d_{av} =0.743. Thus, our change task elicited larger (more negative) N2 amplitudes and larger (more positive) P3 amplitudes for change-signal compared to no-signal trials, which is in line with the previous literature (e.g., Huster et al., 2013).

Withhold condition. Waveforms for withhold-signal and no-signal trials are shown in Figure 3B. N2 amplitudes were -4.9 μV for successful withhold trials and -1.6 μV for no-signal trials, $t(19) = 5.49$, $p < 0.001$*, d_{av} =0.9. P3 amplitudes were 11.5 μV for successful withhold trials and 4.3 μV no-signal trials, $t(19) = 7.1$, $p < 0.001$*, d_{av} =1.78.

A between-task comparison of the no-signal vs. signal trial difference. We tested whether the no-signal vs. signal contrast differed between conditions using a mixed ANOVA. We did not find a significant interaction between trial type and condition for the N2, $F(1,38) = 0.05$, $p = 0.8$, *partial* $\eta^2 = 0.001$, but we did find it for the P3, $F(1,38) = 11.58$, $p = 0.002$, *partial* $\eta^2 = 0.233$. Here, the withhold-signal versus no-signal amplitude difference was larger (7.2 μV) than the change-signal versus no-signal amplitude difference (2.6 μV). Possible reasons for this difference are discussed below.

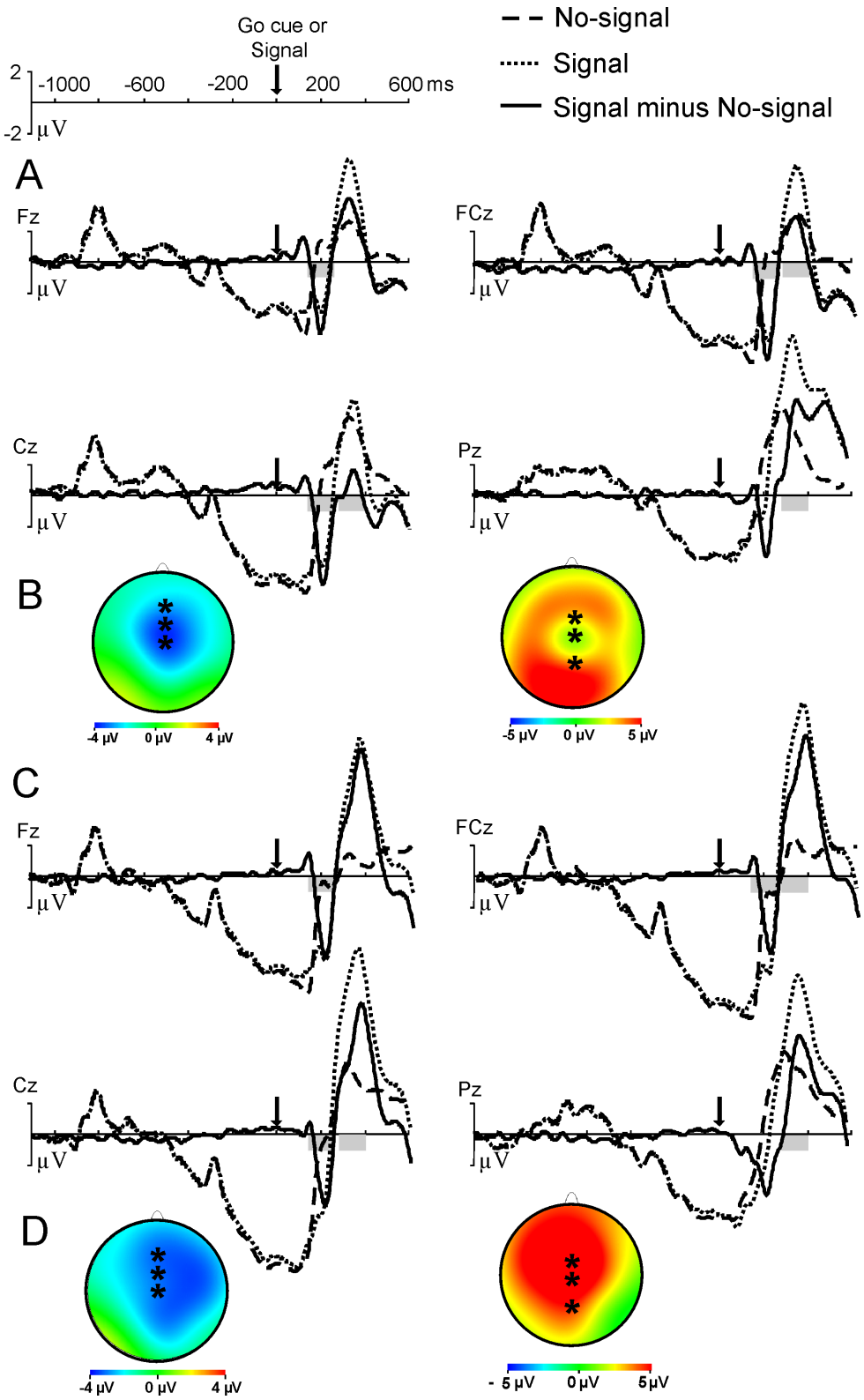

Fig. 3. Panels A-B show the ERP data for the change condition; panels C-D show the corresponding data for the withhold condition. (A) waveforms of signal trials, no-signal trials and difference waves in the change condition; grey bars highlight the intervals analyzed (N2: 150-250 ms, P3: 300-400 ms), (B) left: N2, topography of the signal minus no-signal difference between 150-250 ms; right: P3, topography of the signal minus no-signal difference between 300-400 ms. Stars depict positions of the

analyzed electrodes, (C) waveforms for the withhold condition, (D) N2 (left) and P3 (right) topographies for the withhold condition

Change vs. withhold-signal trials. Figure 4 shows waveforms for the two conditions and the corresponding component topographies. Consistent with our pilot study, the presentation of the signal elicited the lateralized components N1pc, P2pc and N2pc (shown in Figure 4A) as well as an N2 and P3 (amplitudes in FCz, Cz, and Pz shown in Figure 4B). We compared component peak latencies (see Table 1) and amplitudes (see Table 2) for these ERP components following signal onset.

Table 1 shows that the latencies of the various components were numerically similar in both versions of the paradigm. None of the t-tests were statistically significant, and several of the Bayes factors provided substantial support for the null hypothesis ($BF < .33$; i.e. no difference between change-signal and withhold-signal trials). See Table 1 in Wetzel et al., 2011 for the interpretation of Bayes factors.

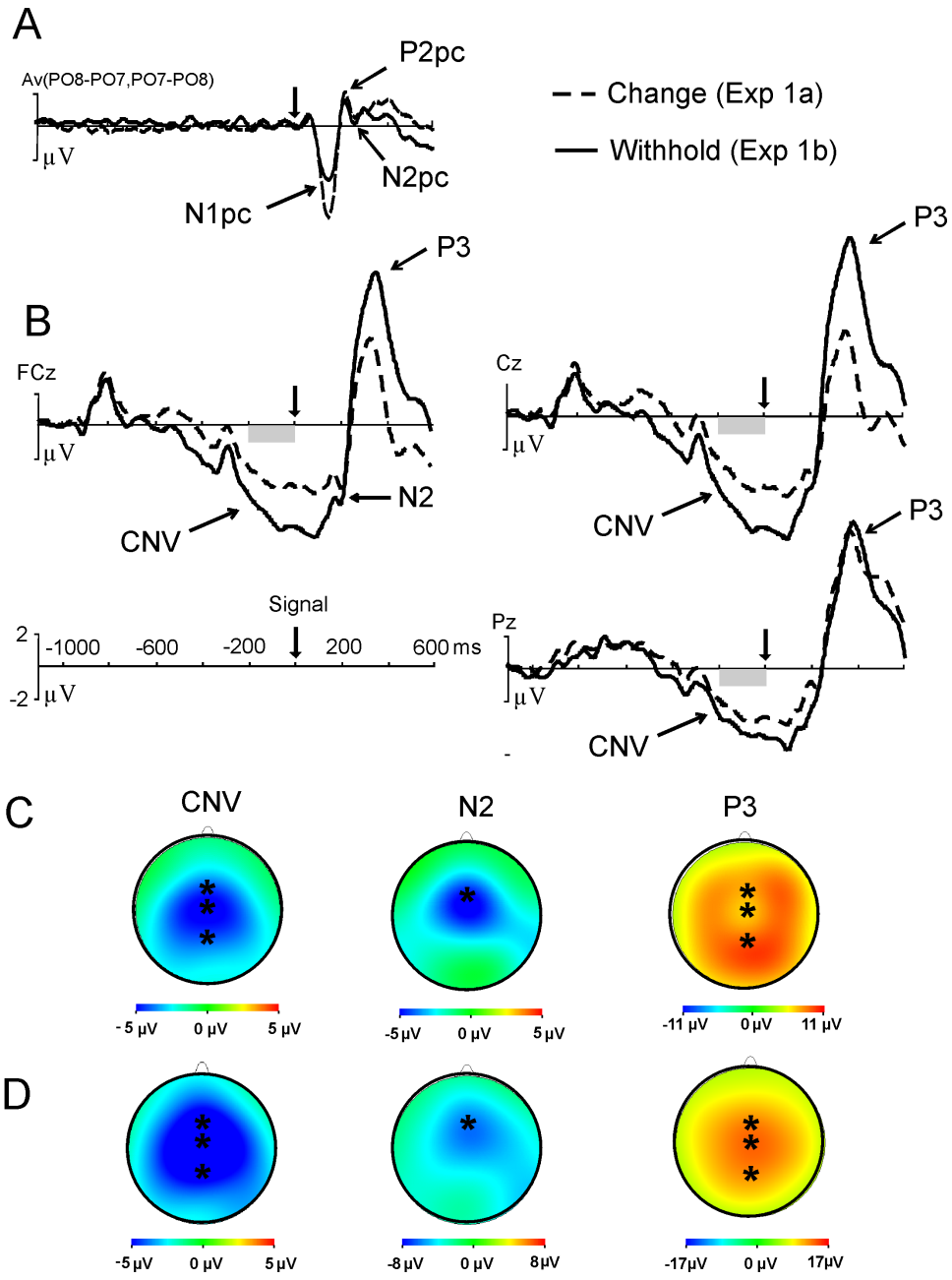

Fig. 4. Signal waveforms of change and withhold trials, (A) lateralised components and (B) non-lateralised components, grey bars show the analysed time window for the CNV[7], (C) component topographies in the change condition, (D) component topographies in the withhold condition; stars depict analysed electrodes

[7] For the other components peak amplitude or peak-to-peak measures were used.

Table 1. Peak latency comparisons between the change and withhold tasks.

	Change, mean in ms (sd)	Withhold, mean in ms (sd)	*t* (38)	*p*	d_{av}	*BF*
N1pc	181 (14)	182 (22)	-0.17	.9	.056	.313
P2pc	249 (22)	243 (22)	0.88	.4	.276	.313
N2pc	295 (29)	284 (24)	1.36	.2	.431	.641
N2	205 (32)	193 (38)	1.02	.3	.323	.466
P3-FCz	350 (62)	350 (27)	0.03	.98	.009	.309
P3-Cz	346 (66)	359 (25)	-0.45	.6	.292	.335
P3-Pz	382(63)	390(48)	-0.84	.4	.144	.409

Amplitudes are shown in Table 2 and Figure 4B. As one can see in the figure, amplitudes of the two tasks already showed numerical CNV differences before signal onset. The Bayes factor analysis suggests that there is substantial evidence for a between-task difference in CNV amplitudes in electrode FCz (and anecdotal evidence for Cz): Pre-signal amplitudes were larger on withhold-signal trials than on change-signal trials. To deal with the potential overlap, we used peak-to-peak measures for the N2 and P3 comparisons and we ran a PCA to disentangle the CNV from the N2 and P3 (see Supplementary Materials). The N1pc, P2pc, and N2pc are lateralized components; consequently, the amplitudes of these components were not influenced by the CNV. Note that the more sensitive PCA analysis could not confirm the CNV differences in any of the electrodes.

The amplitudes of the perceptual/attentional components in the change and withhold conditions were numerically very similar, and the Bayes factors provide more support for the null hypothesis (i.e. no difference between the change and withhold-signal trials; *BF*s < .65) than for the alternative hypothesis (i.e. a trial difference). The N2 was also similar in both versions of the paradigm (*BF* = .466). Neither the peak-to-peak measure nor the PCA found any significant differences.

Peak-to-peak amplitude analysis found no significant difference in the fronto-central (FCz) and posterior (Pz) P3, but the central P3 (Cz) was larger in the withhold task compared with the change task. A possible reason for this difference is that our change response was a foot response. In several experiments, Miller and colleagues (Miller, 2012; Miller & Buchlak, 2012; Miller & Gerstner, 2013) compared motor activations elicited by foot and hand responses and found that activity in the central electrode Cz becomes more negative preceding a foot response (whereas it becomes more positive preceding a hand response). This negativity preceding a foot response would only be present on change-signal trials, leading to the reduced P3 amplitude in Cz for change compared to withhold-signal trials. Consistent with this idea, the temporal PCA did not find any significant differences in the component reflecting the P3 (component 3, see Figure S2 in the Supplementary Materials) in any of the electrodes (after correcting for multiple comparisons) and the Bayes factor only provided anecdotal evidence for a difference between tasks in Cz. The temporal PCA did however extract a component that partially overlapped with the P3 component; this component showed a central negativity for the change condition but a positivity for the withhold condition (see Figure S2). This supports the idea that the reduced P3 amplitude difference in Cz on change-signal trials was caused by the overlap of

response-related activity (the central negativity), rather than differences in inhibitory control mechanisms.

Table 2. Amplitude comparisons between the change and withhold tasks

	change, mean in μV (sd)	withhold, mean in μV (sd)	*t* (38)	*p*	d_{av}	*BF*
CNV-FCz	-4.9	-7.8	2.5	.02	.802	3.340
CNV-Cz	-5.2	-7.7	2.0	.05	.643	1.454
CNV-Pz	-3.8	-4.8	0.96	.34	.303	.444
N1pc	-5 (2)	-3.5 (2)	-2.06	.05	.652	1.593
P2pc	2.4 (3)	1.6 (2)	0.9	.4	.287	.426
N2pc	-0.7 (2)	-0.7 (2)	0.02	.99	.007	.309
N2	-3.4 (4)	-2.1 (2)	-1.40	.17	.465	.668
P3-FCz	16 (6)	22 (8)	-2.47	.02	.791	3.161
P3-Cz	16 (5)	22 (8)	-3.1	.004*	.999	10.959
P3-Pz	17 (5)	19 (6)	-0.9	.4	.291	.575

To sum up, the same early perceptual and later response decision related components were observed in the withhold and change tasks. We found no statistically significant differences in the timing of those components, and the Bayesian analyses provided anecdotal to substantial support for the null hypothesis. The change and withhold amplitudes were also (numerically) similar, with one

exception: Amplitudes of the P3 in Cz were substantially larger in the withhold task than in the change task. Subsequent analysis, however, suggest that this difference was caused by an overlap of the stop-P3 with a response preparation negativity for the foot response in the change paradigm.

Post hoc comparison of fast-slow change response ERPs in the change task. We have isolated a number of ERP components that can be linked to perceptual and response-related processing stages when withholding and changing a response. To examine how the different processing stages (as reflected in their associated ERP components) contribute to successful change performance, we divided ERPs into those associated with fast change responses and those with slow change responses. This way we could examine at which processing stages delays arose. One subject had to be excluded from the fast-slow analysis because they had very high error rates on change trials (84%), resulting in insufficient ERP segments in the averages for fast and slow trials. Another subject had to be excluded because they showed no peaks for the P2 and N2pc in the averages for the fast and the slow trials. Both subjects were excluded from all fast-slow comparisons.

The average go2 RT for fast go2 responses was 412 ms (*sd* = 62) and 605 ms (*sd* = 149) for slow go2 responses. Tables 3 and 4 show the results of the latency and amplitude comparisons, respectively. P-values show one-tailed probabilities because of the strong directional prediction that components would peak earlier and would be of larger amplitude for fast than slow trials. Bayes factors also show one-tailed probabilities (note that Bayes factors do not need to be adjusted for multiple comparisons; this explains some minor discrepancies between the adjusted p-values and the Bayes factors).

First, we analyzed the latencies of the components discussed above. All components were numerically delayed for slow compared with fast trials, but only the differences in the P2 and N2pc survived correction for multiple comparisons. This suggests that a large part of the variance in change performance arises at the stage of stimulus perception and analysis. Although the P3 difference was not significant after adjustment of the *p* threshold, the Bayes factor shows substantial evidence for the alternative hypothesis. This suggests the response decision/selection phase was also delayed on slow trials.

Table 3. Latency comparisons between fast and slow change ERPs

	fast, mean in ms (sd)	slow, mean in ms (sd)	*t* (17)	*p*	d_{av}	*BF*
N1pc	183 (23)	186 (24)	1.76	.05	.138	1.612
P2pc	247 (19)	257 (26)	4.03	.0004*	.455	97.875
N2pc	296 (26)	306 (32)	3.0	.004*	.341	13.167
N2	216 (32)	231 (38)	1.92	.04	.437	2.051
P3	362 (59)	377 (57)	2.24	.02	.253	3.422

Second, we compared the amplitudes of the relevant components. Amplitudes were larger for fast compared with slow trial ERPs for all components, but only the difference in P3 amplitudes survived the *p* value adjustment for multiple tests. Bayes factors provided substantial support for the alternative hypothesis (larger amplitudes for fast than slow trials) also for the P2 and N2. This suggests that on fast trials more

attentional resources are directed to signal analysis (P2), which might lead to enhanced conflict detection (N2), which in turn might lead to more efficiency in making the response decision (stop and change; P3).

Table 4. Amplitude comparisons between fast and slow change ERPs

	fast, mean in ms (sd)	slow, mean in ms (sd)	*t* (17)	*p*	d_{av}	*BF*
N1pc	6.2 (2)	5.7 (2)	1.69	.05	.219	1.455
P2pc	8.5 (4)	7.3 (4)	2.23	.02	.293	3.366
N2pc	4.0 (2)	3.2 (2)	1.9	.04	.376	1.989
N2	1.8 (6)	0.4 (4)	2.22	.02	.277	3.311
P3	18.1 (7)	14.4 (8)	2.64	.009*	.478	6.892

Experiment 2

Experiment 1 identified important perceptual (N1pc, P2pc, N2pc) and response-selection related components (N2, P3). These components were observed on both change-signal and withhold-signal trials, suggesting a substantial overlap in action control mechanisms (Study Aim 1). Furthermore, the post-hoc analyses showed how they related to successful change performance (Study Aim 2): when the signal was presented laterally, a lot of the variance in change performance was found in early components. This finding highlights the importance of fast signal perception for action control (see also e.g., Bekker et al., 2005; Boehler et al., 2009; Overtoom et al., 2009; Verbruggen, McLaren & Chambers, 2014; Verbruggen, Stevens, et al., 2014).

The N2 and P3 analyses suggest that central processing stages did not influence the speed of change performance much (i.e. the differences observed for the later components were numerically similar to the differences observed for the earlier P2/N2pc components). However, for the reasons discussed above, we could not analyze another established marker of response selection, namely the LRPs time-locked to stimulus presentation. We addressed this issue in Experiment 2.

In Experiment 2, the signals were presented centrally. There were no other major changes compared with Experiment 1. In the both conditions of Experiment 2, the signal consisted of an arrow pointing to the left or the right and was presented at the center of the screen (in the same position as the go1 cue on no-signal trials). In the change condition, a left pointing arrow required a left foot response, and a right pointing arrow required a right foot response. In the withhold condition, subjects had to withhold all responses when a left or right pointing arrow appeared.

Consistent with Experiment 1, we compared the ERP components in the two conditions. We then compared ERPs associated with fast versus slow go2 RTs throughout the latent interval starting with the N1 (to pre-empt, no P2 was found in most subjects' waveforms), the N2, the P3 and the s-LRPs.

Method

Subjects. Forty right-handed adults (20 in the change condition, 14 female; 20 in the withhold condition, 18 female) with an average age of 20 (ranging from 18 to 30) received 1.5 course credits or were paid £10 for their participation in this study. Subjects did not differ significantly between the change and withhold groups in age (p = 0.7) or gender (p = 0.12).

Apparatus, stimuli, procedure and analyses. These were the same as in Experiment 1, except for the following: We removed the flanking letters M and W and the trial started with the fixation cross in the center of the screen (Figure 1B). After 500 ms this was replaced by a digit, which was replaced again by the fixation cross after 500 ms. After another 500 ms, either the up- and down pointing arrow cues appeared (no-signal trials), or an arrow pointing to either the left or the right appeared (signal trials).

The same exclusion criteria were applied to the EEG data as in Experiment 1. In Experiment 2, 25% of the trials were excluded on the basis of behavioral performance (24% go trials, 27% signal trials). A further 17% of the trials were excluded due to EEG artifacts. After segmenting into conditions, segments were averaged for each subject containing on average 300 segments for go trials and 145 for signal trials.

For the ERP analyses, we focused on the same components discussed in Experiment 1 although we did not find a P2 and could not calculate an N2pc. In the change condition, we could also test the onset of the s-LRPs for the foot response. More specifically, preparing a foot response (here the change response) results in a negative wave over the motor cortex *ipsilateral* to the response foot (e.g., Brunia, 1980; Miller, 2012). Using the same formula as for the calculation of hand LRPs (see Methods section of Experiment 1), foot s-LRPs present as a positive going waveform. Individual participants' LRPs are typically too noisy for latency difference estimations. Hence, we employed the "jackknifing" method developed to address this problem (Miller, Patterson, & Ulrich, 1998). This method uses leave-one-out averages (including all participants but one) instead of individuals' data to compute the t-statistic.

We estimated the s-LRP latency difference between fast and slow change RT waveforms using a cross-correlation method (Elchlepp, Best, Lavric & Monsell, 2016). This method does not return an onset latency for each waveform but a value representing how much in time one waveform has to be shifted to achieve a maximal correlation with the other waveform. For this we defined a 300-ms-long portion of the LRP associated with slow change RTs (360-660 ms following stimulus onset), and temporally displaced it in steps of 2 ms by up to 100 ms back in time (towards stimulus onset). For each step, we computed a bivariate Pearson correlation between the fast and slow time-series. This resulted in 51 correlations (50 steps plus the zero-shift correlation). "Sliding" the slow waveform back towards stimulus onset resulted in an increase in the correlation from r = .89 to the maximum correlation of r = .995, which corresponded to a shift of 46 ms in the grand-average wave. This 46 ms difference is our estimate of the delay of the slow versus fast waveform. To assess this delay statistically, we computed for each leave-one-out "jackknifing" observation the temporal displacement of the slow time-series corresponding to the maximum of the cross-correlation function, and compared the obtained mean displacement.

S-LRP amplitudes of fast and slow trials already differed substantially before the rise of the positive going foot LRP (see Results section). Before estimating LRP peak amplitudes we therefore baseline corrected the waveforms to the average amplitude of the whole interval (-100 to 600 ms). LRP peak amplitudes were also statistically compared with the jackknifing procedure.

Results

Behavioral data.

Change condition. On no-signal trials, the probability of a correct go1 response was .75 (*sd* = 3.8); the probability of an incorrect go1 response was .03 (*sd* = .4); the probability of a response before the onset of the go1 cue (too soon) was .01 (*sd* = 1.0); and the probability of a missed go1 response was .21 (*sd* = 0.4). Mean correct go1 RT was 338 ms (*sd* = 29) after the appearance of the go1 cue.

On change-signal trials, the probability of a correct go2 response was .75 (*sd* = 10). On failed change-signal trials, the probability of executing the prepared go1 response was .86 (*sd* = .16) and the probability of an incorrect go2 response was .07 (*sd* = .06); the probability of pressing an irrelevant key was .07 (*sd* = .14). Mean correct go2 RT on change-signal trials was 583 ms (*sd* = 77). Go1 responses (i.e. responses to the digit) on failed change-signal trials had a mean latency of 304 ms (*sd* = 56), which was 34 ms faster than on no-signal trials, $t(19) = -3.4$, $p = 0.003$, d_{av} =0.8. This is consistent with the horse-race model, and also indicates that subjects prepared their go1 response during the preparation interval.

Response latencies in the change condition were longer compared to the corresponding condition of Experiment 1 for both no-signal trials (by 45 ms) and change-signal trials (by 65 ms). In Experiment 2, the go1 cue (up and down arrows) and the change signal (left or right arrow) appeared in the same location and were more similar to each other (they were all arrows), making their discrimination harder.

Withhold condition. On no-signal trials, the probability of a correct go1 response was .78 (*sd* = 1.4); the probability of an incorrect go1 response was .01 (*sd* = 1.2); the probability of a response before the onset of the go1 cue (too soon) was .007 (*sd* = 0.8); and the probability of a missed go1 response was .21 (*sd* = 0.3). Mean correct go1 RT was 316 ms (*sd* = 49) after the appearance of the go1 cue.

On withhold-signal trials, probability of an incorrectly executed go1 response was .29 (*sd* = 19). Mean go1 RT on failed withhold trials (268 ms, *sd* = 49) was 48 ms shorter than on mean go1 RT on no-signal trials, $t(19) = -5.08$, $p < 0.001$, $d_{av} = 0.99$ consistent with the independent race model.

ERPs.

Manipulation check. Figure 5 shows the LRPs in preparation to the go1 response. As in the previous experiments, LRPs averaged between -200 ms – 0 ms preceding the go1 cue or withhold/change signal differed significantly from zero; change condition: -0.36μV, $t(19) = -2.17$, $p = 0.02$ (one-tailed), d_{av} =0.97, and withhold condition: -0.32μV, $t(19) = -2.57$, p =0.01 (one-tailed), d_{av} =1.149. The amount of preparation, as measured with LRPs, was similar in both versions of the paradigm, $t(38) = -0.2$, $p = 0.84$, indicating that responding was prepotent in both conditions.

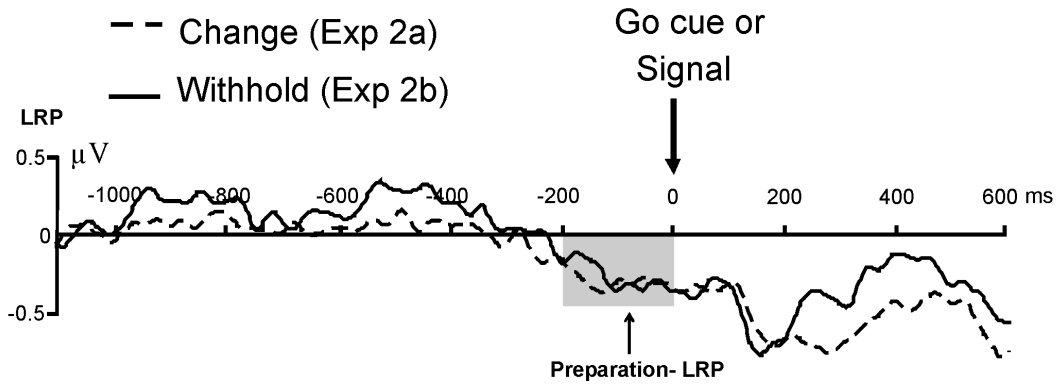

Fig. 5. Experiment 2: LRPs in preparation of the go1 response

No-signal versus signal trials.

Change condition. Differences in waveforms between change-signal and no-signal trials are depicted in Figure 6A and component topographies in Figure 6B.

Mean N2 amplitudes (between 150-250 ms averaged over Fz, FCz and Cz) were 2.7 μV for change trials and 1.6 μV for no-signal trials $t(19) = 3.0$, $p = 0.008$*, d_{av}=0.196.

The P3 peaked slightly later in this experiment compared with Experiment 1. Again, this could be due to the harder signal discrimination (as discussed in the Behavioral Results section). Since our approach was to choose the time window for analysis around the P3 peak, we examined amplitudes between 350-450 ms (as opposed to 300-400 ms in Experiment 1) averaged over FCz, Cz and Pz. P3 amplitudes for change-signal trials were 9 μV and 6 μV for no-signal trials. This difference was significant, $t(19) = 2.74$, $p = 0.01$*, d_{av} =0.615. Thus, we replicate the findings of Experiment 1 (apart from the delay in P3 peak amplitude). The between-trial differences are also consistent with previous go/nogo, stop-signal, and stop-change ERP studies.

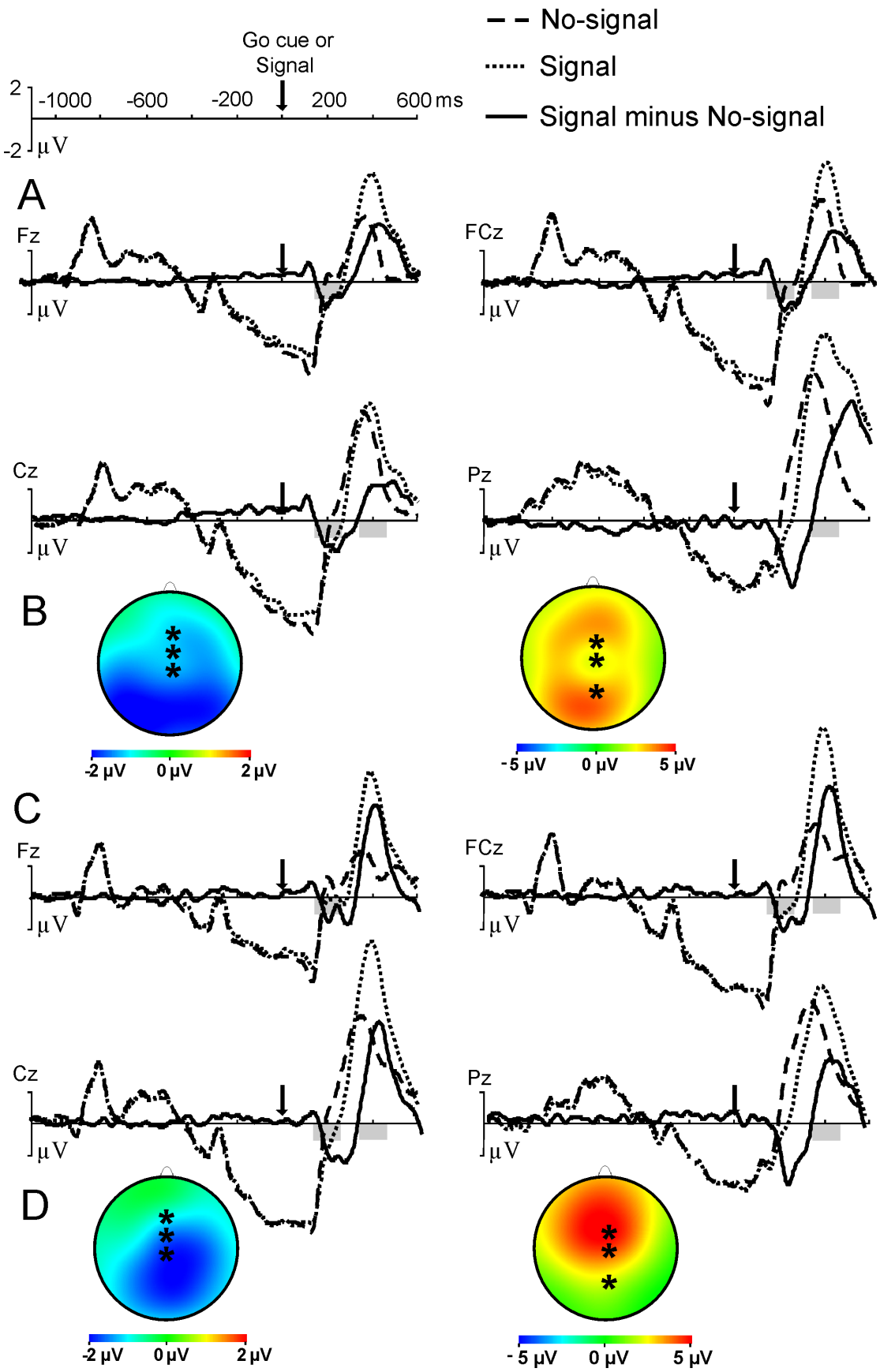

Fig. 6. Panels A-B show the ERP data for the change condition; panels C-D show the corresponding data for the withhold condition. (A) waveforms of signal trials, no-

signal trials and difference waves in the change condition; grey bars highlight the intervals analyzed (N2: 150-250 ms, P3: 350-450 ms), (B) left: N2, topography of the signal minus no-signal difference between 150-250 ms; right: P3, topography of the signal minus no-signal difference between 350-450 ms. Stars depict positions of the analyzed electrodes, (C) waveforms for the withhold condition, (D) N2 (left) and P3 (right) topographies for the withhold condition

Withhold condition. Waveforms for no-signal and withhold-signal trials are shown in Figure 6C and component topographies in Figure 6D. N2 amplitudes were -1.9 μV for successful withhold-signal trials and -0.5 μV for no-signal trials, $t(19) = 3.68$, $p = 0.002$*, $d_{av} = 0.383$. P3 amplitudes were 10 μV for successful withhold-signal trials and 5.4 μV for no-signal trials, $t(19) = 4.08$, $p = 0.006$*, $d_{av} = 0.915$.

A between-task comparison of the no-signal vs. signal trial difference. Similar to Experiment 1, the P3 (signal minus no-signal) difference was numerically larger in the withhold condition (4.7 μV) than in the change condition (2.8 μV). However, the mixed ANOVA with signal (no-signal vs. signal trial) as within-subjects factor and condition as between-subjects factor, showed that this difference was not statistically significant, $F(1,38) = 2.12$, $p = 0.15$, *partial* $\eta^2 = 0.053$. The N2 difference was also similar in both versions of the task, $F(1,38) = 0.24$, $p = 0.6$, *partial* $\eta^2 = 0.006$.

Change-signal vs. withhold-signal trials. Figure 7 shows the signal waveforms and component topographies for both conditions. The presentation of the digit elicited a CNV and the appearance of the signal elicited an N1, an N2 and a P3. Again, we compared component latencies (Table 5) and amplitudes (Table 6). Similar to Experiment 1, peak latencies of the various components were numerically similar (apart from the N2 difference). None of the differences was statistically significant after correction, and the Bayesian analyses of the N1 and P3 differences provided support for the null hypothesis.

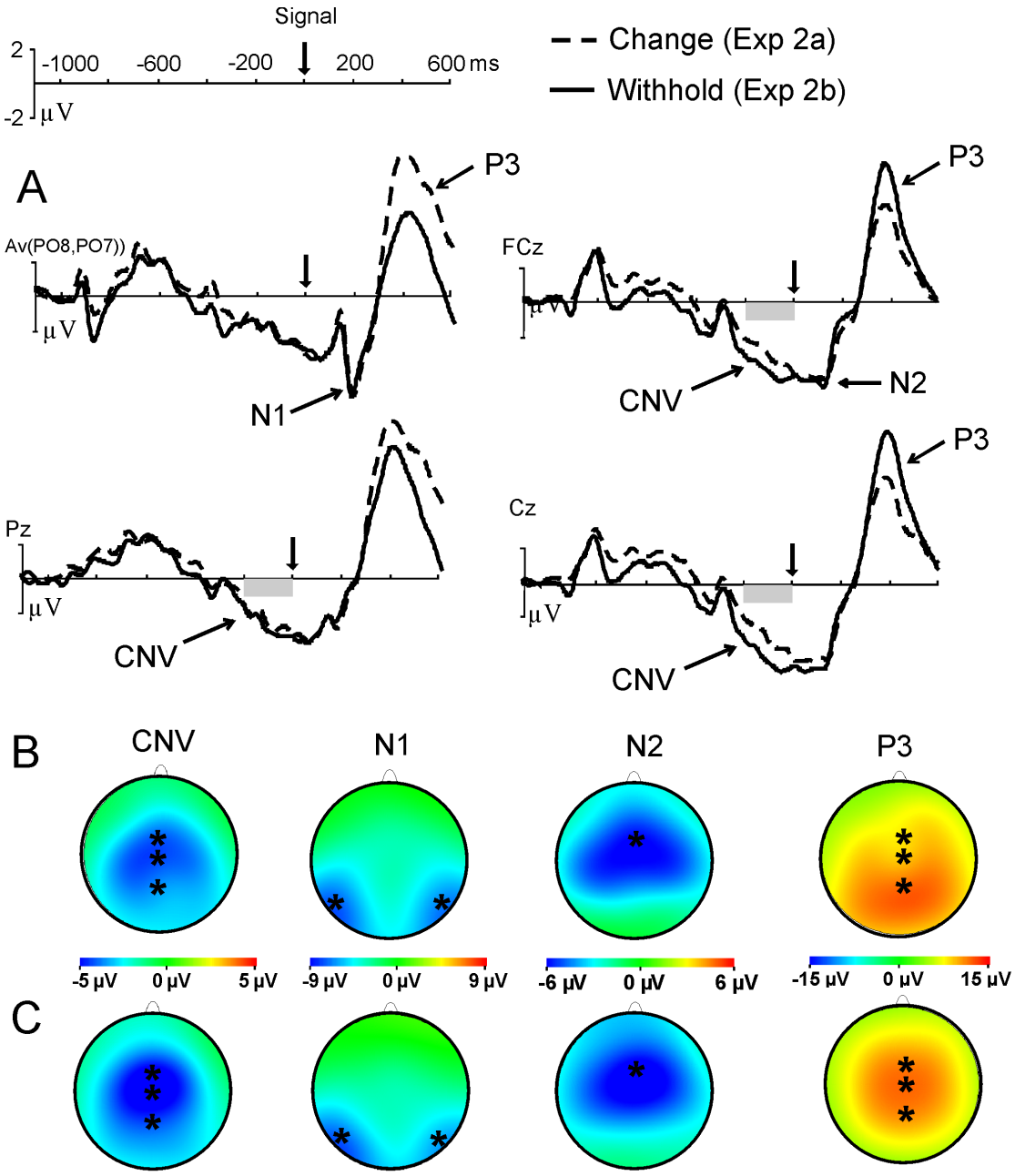

Fig. 7. (A) signal waveforms of change-signal and withhold-signal trials. The grey bars show the analysed time window for the CNV. **(B)** component topographies in the change condition, **(C)** component topographies in the withhold condition. Stars depict analysed electrodes

Table 5. Peak latency comparisons between the change and withhold tasks

	change, mean in ms (sd)	withhold, mean in ms (sd)	*t* (38)	*p*	d_{av}	*BF*
N1	195 (21)	191 (22)	0.6	.55	.190	.357
N2	219 (54)	253 (33)	-2.36	.025	.769	2.604
P3-FCz	398 (22)	391 (23)	0.58	.57	.314	.353
P3-Cz	397 (22)	400 (22)	-0.13	.9	.114	.311
P3-Pz	420 (22)	411 (56)	0.46	.65	.218	.336

Component amplitude comparisons between the change and withhold conditions are largely consistent with the findings of Experiment 1 with a few minor differences. CNV amplitudes were again numerically larger in the withhold condition than in the change condition, but this difference was not significant in the traditional t-tests and Bayes factors were inconclusive or provided anecdotal evidence for the null hypothesis.

In Experiment 1, the amplitudes of the N1, N2, the fronto-central and posterior P3 were similar for change-signal and withhold-signal trials. A similar pattern emerged in Experiment 2, as most BFs provided anecdotal to substantial support for the null hypothesis. In Experiment 1, the central P3 (in Cz) had significantly larger amplitudes for withhold-signal than change-signal trials (although the temporal PCA analysis could not confirm this difference). Numerically, we find the same pattern here but this difference was not significant and the Bayesian analysis provided support for the null hypothesis (*BF* = .389). Altogether, these findings are in line with those in Experiments 1, particularly with the PCA analysis.

Table 6. Amplitude comparisons between the change and withhold conditions

	Change, mean in µV (sd)	Withhold, mean in µV (sd)	*t* (38)	*p*	d_{av}	*BF*
CNV-FCz	-4 (3)	-6 (3)	1.4	.17	.446	.676
CNV-Cz	-4 (3)	-6 (3)	1.71	.09	.200	.971
CNV-Pz	-3 (2)	-4 (3)	0.85	.40	.732	.412
N1	-8 (3)	-8 (3)	0.06	.96	.018	.309
N2	-5 (7)	-3 (5)	-0.72	.48	.232	.379
P3-FCz	14 (6)	15 (7)	-0.7	.48	.228	.375
P3-Cz	14 (6)	16 (8)	-0.76	.45	.242	.389
P3-Pz	18 (5)	14 (7)	1.94	.06	.619	1.330

Fast-slow change response ERPs for the change condition. We performed the same median split analysis as in Experiment 1. Average go2 RT for fast responses was 495 ms (*sd* = 52) and 677 ms (*sd* = 105) for slow responses. In this version of the paradigm no P2 was elicited and an N2pc could not be calculated (because all signals were presented centrally). Therefore, we focused on the N1, N2, P3 and s-LRP.

Table 7. Latency comparisons between fast and slow change ERPs

	fast, mean in ms (sd)	slow, mean in ms (sd)	*t* (19)	*p*	d_{av}	*BF*
N1	193 (19)	194 (22)	0.53	.3	.062	.364
N2	242 (39)	238 (41)	-0.71	.25	.102	.148
P3	393 (59)	424 (57)	2.97	.004*	.599	12.450
s-LRPs	fast-slow diff. = 46 ± 10 ms		2.25	.018	n/a	3.48

First, we analysed component latencies (shown in Table 7). The N1 and N2 latencies were numerically similar for fast and slow change responses. The Bayes factor for the N2 shows substantial evidence for the null hypothesis (driven by the slightly longer N2 latency for fast trials than for slow trials). Whilst P3 latency differences were not significant in Experiment 1, in Experiment 2 we found a large significant delay of 31 ms for slow trials (confirmed by the *BF* providing strong support for the alternative hypothesis). Slow s-LRPs were delayed by 46 ms, an additional delay of 15 ms to the delay already measured in the P3 (see Figure 8A for fast-slow waveforms and Figure 8B for the correlation function of the grand average). This 46 ms difference was not significant after correction for multiple comparisons (p = .018, but the adjusted alpha level for this test was = .017). The Bayes factor, however, provided substantial support for the alternative hypothesis (i.e. a between-trial difference).

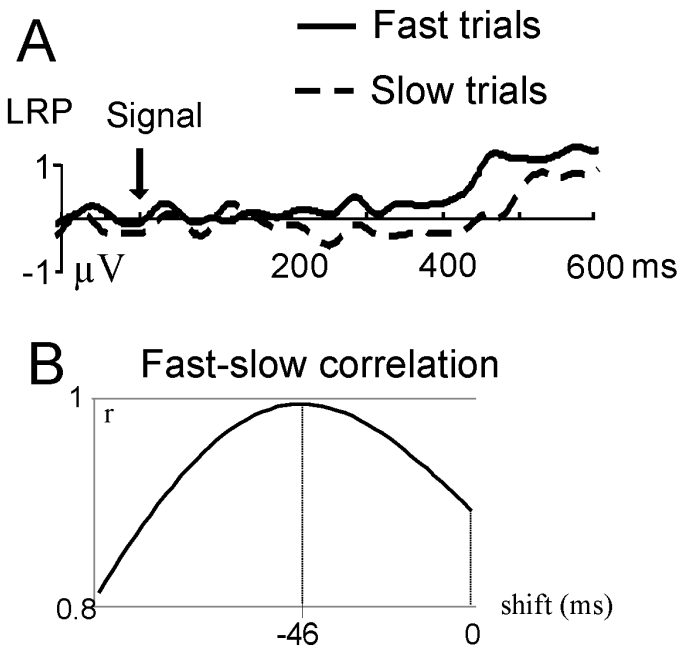

Fig. 8. (A) Waveforms for fast and slow LRPs in the change condition (B) correlation function of the grand average waveform

Table 8. Amplitude comparisons between fast and slow change ERPs

	fast, mean in μV (sd)	slow, mean in μV (sd)	*t* (19)	*p*	d_{av}	*BF*
N1	-7.5 (4)	-7.2 (4)	0.63	.27	.060	.401
N2	-2.7 (6)	-1.8 (5)	1.68	.055	.166	1.434
P3	16 (6)	14 (6)	3.25	.002*	.494	21.143
s-LRPs	.88(.05)	.89(.04)	-0.04	.5	n/a	.225

Next, we compared component amplitudes of fast and slow trials. As in Experiment 1, the P3 amplitudes differed between fast and slow trials. This difference could be due to more efficient processing in the response-decision phase (cancel go1,

implement go2). The differences in N1, N2 and s-LRP amplitudes were not significant. For the N1 and s-LRPs, the Bayes factors provided some support for the null hypothesis.

There are reports that centrally presented arrow cues can also elicit attention shifts to the side that they point to similar to those caused by lateralized stimulus presentations (e.g., Guzzon, Brignani, Miniussi & Marzi). Indeed we found small modulations in the posterior electrodes, but they were too small though to propagate to the central electrodes. Figure S3 in the Supplementary Materials shows a comparison of lateralized components in the change condition of Experiments 1 and 2 calculated using electrodes C3 and C4. One can see clearly that the activity in Experiment 1 resembles that over the occipital cortex (i.e. that it is caused by the processing of the visual stimulus and spreads into the central electrodes), while in Experiment 2 it is close to baseline.

To summarize, in Experiment 1 the signal was presented laterally and most of the variance in change performance was detected in components reflecting attentional resource allocation during signal perception (P2pc and N2pc). In this experiment, detecting a centrally presented signal was easier, and the P3 analyses indicate that the delay arose later in the response-decision and selection phase (e.g., select a left foot response for a left pointing arrow). One could speculate that signal perception was nevertheless affected in this experiment and that possible delays were only picked up in the P3 because of the absence of the signal perception components P2pc and N2pc. However, if this were the case, one would also expect to see some knock-on effect on the N2 (which occurred around the same time as the P2pc in Experiment 1). The N2 component was numerically delayed for slow trials by 15 ms in Experiment 1, but not in Experiment 2. Thus, it seems likely that the delay measured in the P3 does in fact

arise at the stage where the relevant features of the signal are translated into a response. Furthermore, the implementation of the appropriate response, i.e. the activation of the motor cortex associated with the responding foot was also delayed for slow trials compared with fast trials. This finding suggests that (at least in this version of the paradigm) variability this last stage before the execution of the change contributed to successful change performance.

General discussion

The present study explored which processes are involved in withholding and changing a response. We used a hybrid version of the stop-signal and cued go/nogo task that was designed to allow a clean assessment of post-signal ERP components, particularly of early perceptual components. In this paradigm, subjects could only execute their go1 response when a go1 cue appeared, but a tracking procedure adjusted a strict response window to force subjects to prepare their responses in advance. Behavioral (shorter signal-respond RTs than go1 RTs on no-signal trials) and neurophysiological measures (significant LRPs preceding the go/change stimulus, large CNVs) in both conditions of both experiments indicate that subjects engaged in advance preparation and did not just wait for the go1 cue to occur. LRP analyses also confirmed that there were no differences in the amount of motor preparation in anticipation of the go1 cue between the withhold and change paradigms.

Signal versus no-signal trials

In the first set of analyses, we compared signal and no-signal trials. Consistent with the previous literature on stop-signal tasks, go/nogo tasks, and most stop-change tasks (e.g., Huster et al., 2013), we found larger N2 and P3 amplitudes for signal trials

compared with no-signal trials in all conditions. Larger N2 amplitudes in response to signals versus no-signal trials have been interpreted as reflecting the monitoring of the prepotent and competing response- (and stimulus) activations. Larger P3 amplitudes have been argued to reflect resource allocation during the response decision (e.g., Johnson & Donchin, 1982) and/or response inhibition (e.g., Bruin et al., 2001; Wessel & Aron, 2015).

A direct comparison of the change and withhold conditions revealed that the N2 amplitude difference was similar in both versions of the task. Krämer et al. (2011) found an N2 for stop signals but not for stop-change signals, and argued that different inhibitory processes are involved in both tasks. In light of the current results it seems, however, more likely that their findings were specific to their paradigm, which combined an Erikson flanker task with a stop/change signal task. The between-condition comparison in Experiment 1 revealed a statistically significant P3 amplitude difference in Cz. But as discussed below, this difference is presumably due to the extra response-execution demands on change trials.

Processing stages of signal trials in the change and withhold tasks

Various forms of action control, including withholding and changing responses, result from an interplay between three basic and computationally well-defined processes: signal detection, action selection, and action execution (Verbruggen, McLaren, & Chambers, 2014). In the present study, we used tasks that were specifically designed to examine these processing stages.

We performed a detailed examination of change and withhold performance when the signals were presented laterally (Experiment 1) and when they were presented centrally (Experiment 2). Each presentation mode resulted in a unique

pattern of ERP components, which mapped onto the basic processes of signal anticipation (CNV), signal detection (N1pc, P2pc, N2pc in Experiment 1; N1 in Experiment 2), conflict detection (N2), and response decision/selection (P3 in Experiments 1-2 and the s-LRPs in Experiment 2). Overall the pattern of ERP components was very similar in the change and withhold conditions. The components also had similar peak latencies in the two conditions. Finally, amplitudes were also similar for most components. The between-condition comparison of Experiment 1 revealed differences in the CNV (for FCz only) and the P3 (for Cz). However, the temporal PCA could not confirm the CNV difference, and it showed that the P3 amplitude difference measured in the ERP was presumably caused by an overlap with response related activity (a negativity preceding the foot response; Miller, 2012; Miller & Buchlak, 2012; Miller & Gerstner, 2013) rather than a difference in inhibitory control mechanisms.

The strong similarities between the change-signal and withhold-signal trials are consistent with the idea that a stop process or goal is needed to quickly change a response (Verbruggen et al., 2008; Camalier et al., 2007). Furthermore, the results are in line with recent theoretical accounts and fMRI findings (e.g., Band et al., 2003; Mars et al., 2007; Kenner et al., 2010; Boecker et al., 2011), suggesting the same inhibitory mechanisms are at play when subjects have to cancel or replace a response.

Linking ERP components with change performance

To link ERP components with behavior, we compared ERPs associated with fast and slow change responses in change tasks with lateral signal presentation (Experiment 1) and with central signal presentation (Experiment 2). Note that we could not perform this analysis for the withhold task because the covert latency of the stop (withhold)

process has to be estimated, whereas the latency of the change process can be measured directly. We focused on ERP components that reflect the chain of processing on signal trials, starting with signal detection, signal analysis, conflict detection, response selection, and implementation of the alternative response.

When signals were presented out of the focus of spatial attention (Experiment 1) a substantial amount of the variability in change performance arose at early perceptual stages: the perceptual ERP components (P2pc, N2pc) peaked earlier on fast- compared with slow change trials. This suggests that fast attentional re-orienting is important for successful change performance when signal detection is more challenging. Once the signal was detected and analyzed additional numerical differences were found in the subsequent conflict detection (N2) and response decision (P3) phase, but Table 3 indicates that no further delays arose at these stages (i.e. the difference between fast and slow change response did not increase much). It is possible that the spatial congruency between signal presentation side and response side lead to enhanced activation of the go2 response (Kornblum, Hasbroucq & Osman, 1990), thereby reducing the response-selection demands. This could explain the relatively small variability in the timing of the response decision. Analyses of the amplitudes only revealed P3 amplitude differences between fast and slow change-signal trials, which could reflect more efficient processing at the response decision and subsequent selection stages. In Experiment 1, we could not determine whether the speed of activation of the motor cortex further contributed to variability in change performance.

When the signal was presented in the focus of attention (Experiment 2), the timing of signal detection did not differ between fast and slow change trials. There was also no significant difference in conflict detection as measured with the N2. We

did, however, find a large delay for slow trials in the timing of the P3 peak (a delay twice as large as in Experiment 1). In contrast to Experiment 1, in Experiment 2, the signal (i.e. a left or right pointing arrow) shared a number of features with the go1 cue (i.e. upwards and downwards pointing arrows), which might have increased the difficulty to discriminate between the two, which in turn might have delayed the decision of the alternative foot response (left or right side). S-LRPs were also delayed for slow trials suggesting that also the last stage before motor execution is an important contributor to successful change performance in at least some variants of the task. Combined, these findings indicate that it is important to consider at which processing stage(s) differences between groups or conditions arise in response-inhibition and action-control tasks (cf. Verbruggen & Logan, 2015).

From the results above one could conclude that signal perception is only an important contributor to successful change performance when the signal is harder to detect. However, subjects in this study were all young, healthy adults. It is possible that in certain clinical populations (e.g. in patients with attention deficits) or in certain situations (e.g. when subjects are fatigued), differences can arise at early processing stages even when central visual- or auditory signals are used. As noted above, Bekker, Kenemans et al. (2005) found that N1 amplitudes were larger for successful stops than for failed stops. This led them to conclude that inhibitory performance requires switching attention to the stop signal. Interestingly, this N1 difference was absent in adults with ADHD (Bekker, Overtoom et al., 2005), indicating that their stopping deficits could be due to impairments in selective attention, rather than in inhibition. Other studies showed that when the stop-signal paradigm was combined with a reward manipulation, rewarded stop trials showed enhanced N1 amplitudes compared with unrewarded stop trials (Greenhouse & Wessel, 2013; Schevernels et al., 2015),

suggesting more attention is directed to the stop-signal when reward is expected. Surprisingly though, in these studies, N1 amplitudes on rewarded stop trials either did not differ between successful and failed stops (Greenhouse & Wessel, 2013) or were larger for failed than successful stops (Schevernels et al., 2015). While Greenhouse and Wessel suggest that visual attention to the stop-signal might not be related to the stopping process (at least in their task), Schevernels et al. explain their results with a possible interaction between processing the reward information and the attentional processes involved in response inhibition. Based on our present results, and previous behavioral (e.g. Leiva, Parmentier, Elchlepp, & Verbruggen, 2015; Verbruggen, Stevens, et al., 2015), computational (e.g. Boucher et al., 2007; Salinas & Sanford, 2013), ERP (e.g. Bekker, Overtoom, et al., 2005), MEG (Boehler et al., 2009) and pharmacological work (Overtoom et al., 2009) we favor the explanation that early perceptual processes do play an important role in response inhibition/change performance.

Methodological advances

The stop-signal and stop-change tasks belong to the most widely used paradigms to examine inhibitory control in experimental, clinical and developmental settings (Verbruggen, Chambers, & Logan, 2013). In combination with ERPs they can inform researchers about the processes that are involved in stopping and replacing a response, and the timing of these processes. However, combining the stop-signal task with EEG is challenging due to the short delay between the presentation of the go stimulus and the signal (typically around 200 ms). That means in most cases go stimulus related activity is still ongoing when the stop signal is presented. This leads to an overlap and distortion of signal related activity.

Over the last decades ERP researchers have tried to develop appropriate correction techniques to deal with this overlap problem. Even though some suitable techniques are available (e.g. the ADJAR method; Woldorff, 1993), they often pose specific constraints on the paradigm (e.g., it needs particular jittering of SOAs), large trial numbers, and require excellent programming skills or specialized software. As a result, easier or no correction procedures are often used, which makes a comparison of findings difficult or even questionable. This is particularly the case for the analysis of the early perceptual components. Further, early perceptual components are refractory at short inter-stimulus intervals: they show reduced amplitudes when the eliciting stimulus follows soon after another stimulus (see Woodman. 2010 for a discussion). To avoid this problem it is recommended to use an inter-stimulus interval of approximately 1 second.

In standard versions of the go/nogo tasks, there is no overlap between components. However, the inhibitory control demands are much lower than in the stop-signal task and their variants. Therefore, in the present study, we used a hybrid version of a cued go/nogo task and the stop-signal task, in which there was no overlap between the go and stop signal but in which responding was still prepotent. Our paradigms have produced behavioral and neurophysiological results consistent with previous stop-signal and stop-change tasks. Consistent with the independent race model of the stop-signal task, we found that RTs on unsuccessful change and withhold trials were shorter than the corresponding RTs on no-signal trials. This indicates that only the fastest trials could escape inhibition. Furthermore, we have also observed N2 and P3 components, which have been observed in stop-signal and stop-change tasks. Importantly, our task allows a detailed description of the processes involved in stopping and changing a response, and are easy to use. Therefore, it

allows for thorough electrophysiological investigations of response inhibition in cognitive and clinical settings, offering an alternative to the standard stop-signal task when correction techniques like ADJAR are not available/feasible.

Of course, we should point out that we observed between-condition differences in CNV in Experiment 1. If significant differences in anticipatory activity between conditions are found in our paradigm, we suggest to run a temporal PCA and either subtract out the PCA component reflecting the CNV (as demonstrated by Oddy et al., 2005) or if the PCA finds components reflecting the ERP components of interest (e.g., N2, P3, etc.) submit the factor scores of those components to statistical testing.

Conclusions

We isolated ERP components associated with basic cognitive processes following signals instructing subjects to withhold or replace a response. Their comparison revealed great similarities, providing neurophysiological evidence that the same basic processes are involved in cancelling and changing a response. We linked these basic processes to behavior by demonstrating their contribution to fast successful change performance. When signals were harder to detect most of the variability in change performance arose at early perceptual stages, highlighting again the role of early perceptual processes in response inhibition and action control. When signals were presented centrally and their detection was easier, most of the variability in change performance arose at the response decision and selection phase. Thus, breaking action control down into basic processes or mechanisms is important to understand differences between tasks, conditions, or groups.

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SUPPLEMENTARY MATERIALS

1. Electrode montage

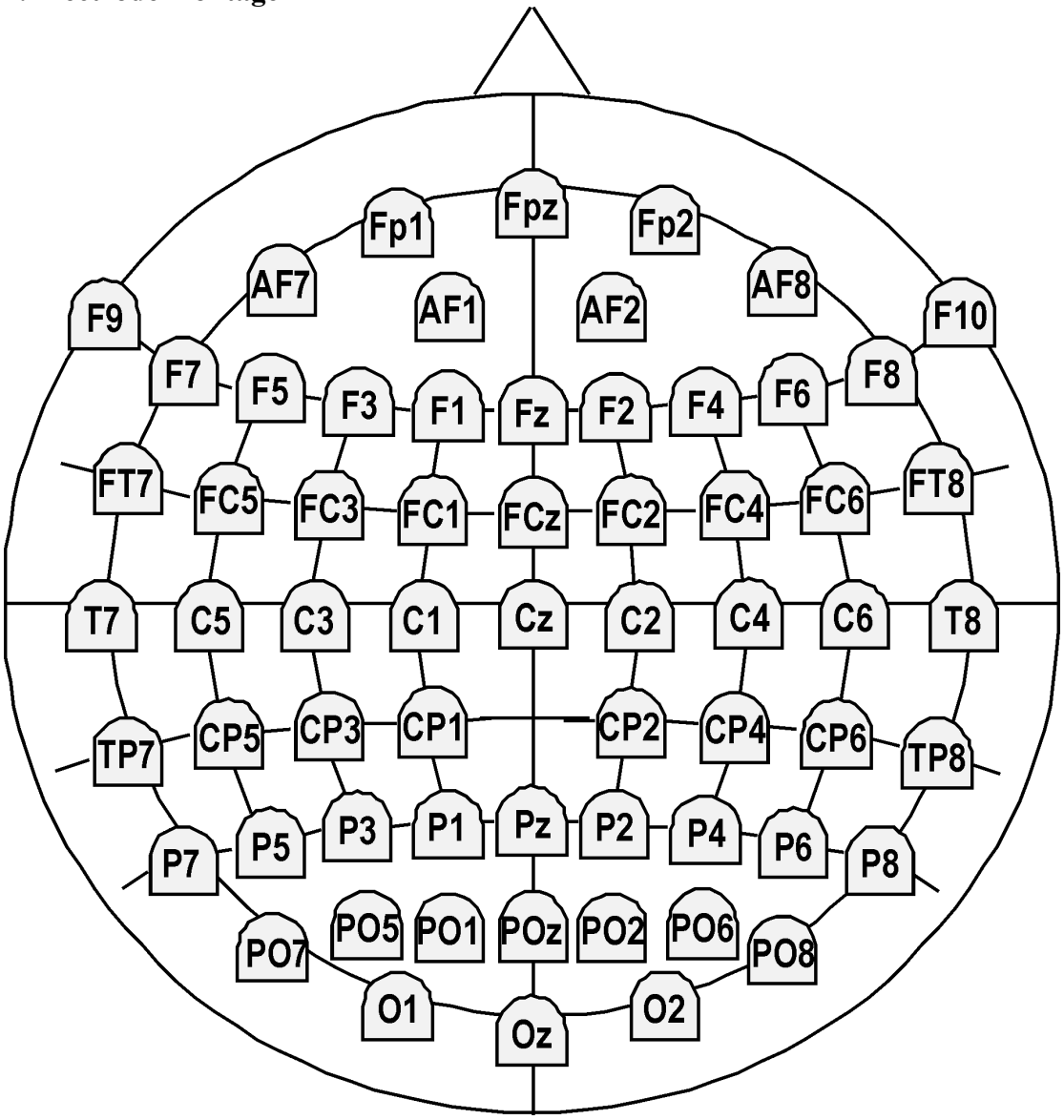

Figure S1. Electrode set-up

2. Temporal principal component analysis

Our task largely eliminates the direct spill over of activity related to go-stimulus processing into the post-signal interval. However, unexpectedly, we found differences in neural activity in anticipation of the signal between the change and withhold conditions in Experiment 1. When an informative stimulus (S1) precedes the imperative stimulus (S2), the S1 often elicits anticipatory activity, which is reflected in a component named Contingent Negative Variation (CNV). Most often this anticipatory activity is not resolved at S2 onset and overlaps with S2 processing. This becomes a problem when the anticipatory activity differs between conditions. To deal with this, common practice is the use of a baseline shortly before or around S2 or taking a peak-to-peak measure for the components following S2. However, these methods have also been criticized because the CNV may resolve or deflect differently in different conditions. For example, Simson, Vaughan and Ritter (1977) argued that it resolved differently for go and nogo trials in a paradigm where the decision to go or not on a combination of two successive stimuli. To test this idea, Oddy, Barry, Johnstone and Clarke (2005) used temporal Principal Component Analysis (PCA) to disentangle the CNV from the subsequent N2 and P3. They isolated a PCA component that captured the CNV and subtracted this component from the EEG. They concluded that the CNV resolution generally did not differ between the go and nogo conditions.

Our paradigm also elicited a CNV, and Bayesian analyses indicated that there was a CNV difference between the change and withhold conditions in electrode FCz. Thus, it is possible that the CNV resolved differently in the change and withhold conditions. This could lead to between-condition differences in the N2 and P3 (note that the lateralized components N1pc, P2pc and N2pc were not influenced by the

CNV overlap, because the CNV was not lateralized and hence subtracted out). To disentangle the temporally partially overlapping ERP components CNV, N2 and P3 we ran a temporal PCA on signal waveforms of the two tasks (change and withhold) and isolated orthogonal PCA components from the ERP time series. If the CNV resolves differently in the two tasks, influencing potential differences in the subsequent N2 and P3, the PCA analysis should provide a clearer picture of between-task differences in the post-signal interval.

We ran a temporal PCA on the interval starting 500 ms preceding signal onset to 600 ms post-signal (=1100 ms). To prepare the data for the PCA we down-sampled the EEG from the original 500 Hz to 250 Hz, which resulted in 275 time points for the chosen interval (one time point every four milliseconds). Temporal PCA (Donchin & Heffley, 1978) uses the covariance between ERP time-points over subjects, conditions (in this case the two task conditions; i.e. change and withhold), and electrodes to identify the underlying temporal components. We ran the PCA on a matrix of 2520 observations (20 subjects x 2 tasks x 63 electrodes) by 275 time-points, with time-points as variables. The PCA was performed on the covariance matrix and its solution was Varimax-rotated to yield uncorrelated temporal components; the criterion for component extraction was that of eigenvalue $\geq$1. PCA extracted 13 components, which together explained 98% of the variance (see Figure S2 for component loadings and selected component topographies). We selected the PCA components reflecting the CNV, the N2 and the P3 for further statistical analysis on the basis of their time course and topographies.

CNV. Component 1 (Figure S2) explains 35% of the variance and rises continuously from the beginning of the interval onwards (500 ms before signal onset) with maximal loadings around signal onset and the following 150 ms. Its time course,

polarity and central topography are reminiscent of that of the CNV. We contrasted factor scores associated with the change and withhold conditions in electrodes FCz, Cz and Pz. Independent t-tests did not reveal any significant differences, FCz: mean difference 0.75, $t(38)= 1.81$; $p=.08$[8], $d_{av} =.59$; Cz: mean difference 0.55, $t(38)= 1.20$; $p=.24$, $d_{av} =.39$; Pz: mean difference 0.24, $t(38)= 0.74$; $p=.46$, $d_{av} =.24$. The corresponding Bayes factors were largely inconclusive (FCz: 1.14, Cz: 0.55, Pz: 0.38). Thus, the PCA analysis could not replicate the FCz difference reported in the manuscript.

N2. Component 10 explains 2.3% of the variance and has highest loadings between 200-250 ms. Its timing, polarity and topography suggests that it represents the N2. The topography of the PCA-N2 is slightly more frontal than that of the ERP-N2 reported in the manuscript. To avoid missing any potential differences, we included electrode Fz in the statistical analysis. No significant differences were found, Fz: mean difference 0.19, $t(38)= 0.51$; $p=.61$, $d_{av} =.16$; FCz: mean difference 0.15, $t(38)= 0.35$; $p=.73$, $d_{av} =.11$, and the Bayes factors provide substantial evidence for the null hypothesis (Fz =0.34: ; FCz = 0.32). This is in line with the ERP amplitude test reported in the manuscript.

P3. Component 3 explains 22% of the variance and has highest loadings between 250-350 ms. Timing, polarity and topography suggest that it represents the P3. Independent t-tests did not reveal any differences that were significant after correction for multiple comparisons, FCz: mean difference 0.59, $t(38)= -1.4$; $p=.17$, $d_{av} =.44$; Cz: mean difference 0.98, $t(38)= -2.26$; $p=.03$ (n.s., Holm-Bonferroni adjusted threshold, $p=0.017$), $d_{av} =.71$; Pz: mean difference -0.14, $t(38)= 0.41$; $p=.68$, $d_{av} =.13$. The Bayes factor for electrode FCz ($BF = 0.67$) provides anecdotal evidence

[8] P values shown are uncorrected. Adjusted thresholds are shown for p values that were significant at $p<0.05$.

for the null, that in electrode Cz ($BF = 2.2$) provides anecdotal evidence for the alternative hypothesis, and the one in Pz ($BF = 0.33$) provides substantial evidence for the null.

To sum up, we used temporal PCA to divide the ERP time series into independent components and extracted components that represent the CNV, N2 and P3. This method is more sensitive to the process that underlies an ERP component because it eliminates temporal overlap of successive components. We did not find any significant differences between the change and withhold tasks in any of the components tested and Bayes factors were either inconclusive or provided substantial evidence for the null hypothesis. Differences that were either significant or where the Bayes factor provided substantial support for the alternative hypothesis in the ERP amplitude analysis (CNV difference in FCz, P3 differences in FCz and Cz) could not be confirmed. Given that the PCA is the more sensitive measure, we can conclude that the change and withhold conditions largely involve the same underlying processes.

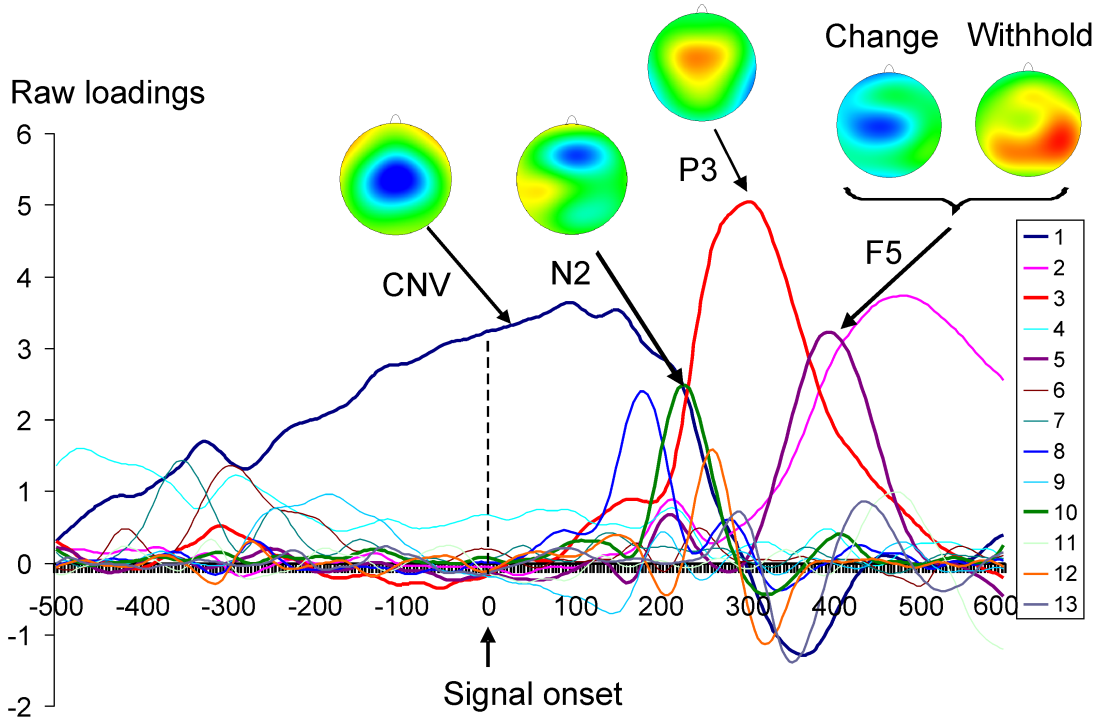

Figure S2. PCA component loadings and their topographies

3. Lateralized activity in central electrodes in Experiments 1 and 2

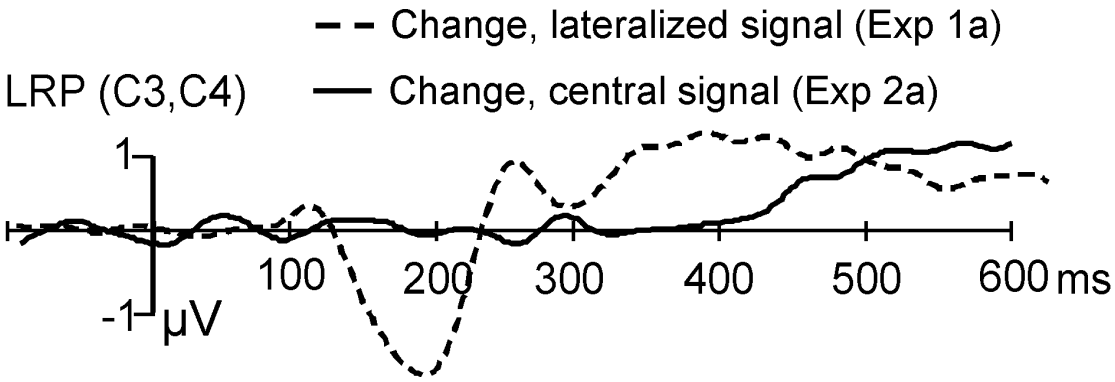

Figure S3. Comparison of lateralised activity (average C3-C4, C4-C3) elicited by signal presentation (change in flanking letter on the left or right) and foot response side in the change condition of Experiment 1, and the direction of the central arrow (left or right) and foot response side in the change condition of Experiment 2. For Experiment 1, there is contamination by visual ERPs, which are still large in central electrodes. In Experiment 2, there is only the rise of the (positive) foot-LRP after about 400 ms without preceding distortions.