Effects of stop-signal probability in the stop-signal paradigm: The N2/P3 complex further validated

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Abstract

The aim of this study was to examine the effects of frequency of occurrence of stop signals in the stop-signal paradigm. Presenting stop signals less frequently resulted in faster reaction times to the go stimulus and a lower probability of inhibition. Also, go stimuli elicited larger and somewhat earlier P3 responses when stop signals occurred less frequently. Since the amplitude effect was more pronounced on trials when go signals were followed by fast than slow reactions, it probably reflected a stronger set to produce fast responses. N2 and P3 components to stop signals were observed to be larger and of longer latency when stop signals occurred less frequently. The amplitude enhancement of these N2 and P3 components were more pronounced for unsuccessful than for successful stop-signal trials. Moreover, the successfully inhibited stop trials elicited a frontocentral P3 whereas unsuccessfully inhibited stop trials elicited a more posterior P3 that resembled the classical P3b. P3 amplitude in the unsuccessfully inhibited condition also differed between waveforms synchronized with the stop signal and waveforms synchronized with response onset whereas N2 amplitude did not. Taken together these findings suggest that N2 reflected a greater significance of failed inhibitions after low probability stop signals while P3 reflected continued processing of the erroneous response after response execution.

1. Introduction

Cognitive control refers to the higher-order organizational processes that monitor and command lower-level processes to intervene and adjust behavior. One frequently investigated aspect of cognitive control is inhibition. This construct has been interpreted and defined in various ways (Kok, 1999; Logan, 1994) and likely entails the involvement of various cognitive processes and different brain circuits (e.g., Kramer, Humphrey, Larish, Logan, & Strayer, 1994). An important aspect of inhibition concerns response inhibition or the ability to suppress or abort ongoing actions. The capacity to interrupt inappropriate motor responses is also referred to as stopping behavior and can be investigated by using the stop-signal paradigm. In this paradigm, subjects are engaged in a primary task, usually a choice reaction time task (CRT). Occasionally and after variable delays a second stimulus, the stop signal, is presented that tells the subject to withhold the response to the primary task—a go stimulus.

Performance in the stop task has usually been interpreted within the framework of the horse-race model. This model represents stop performance in terms of a race between two sets of processes that operate stochastically independent and run for completion in a winner-takes-all fashion (Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984). The first set of processes is invoked by the onset of the primary-task stimulus and controls the stages of stimulus identification, response choice, the preparation and execution of the response, whereas the second set pertains to the processing of the stop signal and controls the detection of the stop signal.
signal and response inhibition. The outcome of this race determines whether a response is stopped or executed; if the stop process finishes before the go process, the response is successfully inhibited, otherwise, a response is given (Lappin & Eriksen, 1966; Logan & Cowan, 1984).

The major dependent variable in the horse-race model of stop performance is the estimated finishing time of stop-signal processing. In contrast to the direct observation of processing speed in the primary task, the duration of the non-observable, internal reaction time to the stop signal (Stop Signal Reaction Time or SSRT) can only be estimated indirectly (for details see Logan, 1994; Logan & Cowan, 1984). From a considerable number of studies with the stop-signal paradigm, it appears that the SSRT is rather stable in speed, with a duration ranging from 200 to 250 ms (for extensive simulations with the stop-signal paradigm, see Band et al., 2003) although it has been shown that that SSRT is increased by distractor stimuli in a flanker task (Ridderinkhof, Band, & Logan, 1999).

The horse-race model is not concerned with the specific nature of processes underlying stopping behavior. Event-related potentials (ERPs) have proven to be useful in providing additional insights into the processes underlying stop performance. In one of the first experiments in which the stop task was combined with ERPs, De Jong and colleagues observed an enhancement of the P3 component in the successfully inhibited compared to the unsuccessfully inhibited condition, which was interpreted as a reflection of inhibition of the primary-task processing elicited by the stop signal (De Jong, Coles, Logan, & Gratton, 1990). Kok, Ramautar, De Ruiter, Band, and Ridderinkhof (2004) recently reported that ERP components following the stop signal and go stimulus in the stop task could be separated from another. Effects were manifested as an enhancement of P3 amplitude elicited on stop-signal trials relative to ERPs on no stop-signal trials (i.e., trials that did not contain a stop signal). It also appeared that P3 components elicited on successful stop trials (SST) and unsuccessful stop trials (UST) differed markedly with respect to their scalp topographies and dipole sources. SST P3 showed a fronto-central focus and UST P3 a medial posterior focus on the scalp, suggesting the involvement of precentral and more widespread posterior areas, respectively. Moreover, SST P3 was elicited slightly earlier than UST P3 that could reflect differences in the timing of stop processes. Based on these findings Kok et al. (2004) suggested that in the stop task the P3 component elicited on SST probably reflected aspects of active inhibition of the motor response, while N2 and P3 components that were elicited on UST could also have reflected aspects of monitoring of erroneous responses.

Other stop studies in which similar ERP results were observed, involved child studies in which the stop task was used to investigate inhibitory deficits in dyslexia (Van der Schoot, Licht, Horsley, & Sergeant, 2000) and in ADHD (Dimoska, Johnstone, Barry, & Clarke, 2003; Overtoom et al., 2002; Pliszka, Liotti, & Woldorff, 2000).

In Kok et al. (2004), stop-signal trials and no stop-signal trials were presented equally frequent. Logan et al. (1984) however has argued that low frequency stop signals are needed to ascertain that subjects do not develop a response strategy in which speed is traded for inhibition success. The major performance effects of presenting stop signals less frequently are faster reaction times to the go stimulus and an increase of the percentage of false alarms (% UST). This is usually interpreted by assuming that stop performance is controlled by a trade-off between processing of the go and stop signal: when stop-signal probability decreases subjects sacrifice success of inhibition for faster responses to the go signal (Logan, 1984). Another aspect of presenting stop signals less frequently, which is of relevance for the present study, is that more inhibitory pressure is needed to overcome the strong tendency to produce rapid responses to the go stimuli. Brain areas that are engaged in response suppression involve in particular the prefrontal areas as indicated by several lesion studies (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Fuster, 1997) and imaging studies (Casey et al., 1997; Garavan, Ross, & Stein, 1999; Konishi et al., 1999).

The principal aim of the present study was to further examine the functional and topographical characteristics of N2/P3 components that are elicited in the stop task by using stop-signal probability as the major manipulation of an internally generated act of control. Our first objective was to gain a more precise understanding of the functional significance of ERP components that are elicited on successful and unsuccessful inhibit trials of the stop task. Contrasting these ERPs in tasks when stop signals were presented at low and high frequencies was assumed to provide a more detailed insight into involvement of inhibitory brain processes. According to the race model presenting stop signals less frequently would create a stronger bias to the go stimuli. This was however expected to affect components elicited on SST and UST in a different way. When stop signals occur less often successful inhibition can only be accomplished because a greater inhibitory pressure is applied to overcome the stronger bias to the go stimuli. The
study of Kok et al. (2004) suggested that the fronto-central P3 elicited on successful stop trials was a suitable candidate for the expression of response-inhibitory processes. The scalp topography and location of the dipole generators of SST P3 component was however more consistent with a locus in the precentral (motor) areas than the prefrontal areas. Presenting stop signals less frequently could however be more effective in activating neural sources in prefrontal cortex. Stop-signal frequency was also assumed to affect ERP components elicited on unsuccessful stop trials. According to the trade-off model subjects could place less emphasis on stopping when stop signals are presented less frequently (Logan et al., 1984). Following this logic, subjects should perceive failed inhibitions as less meaningful or important in the low than in the high stop-signal probability task. This was assumed to lead to smaller amplitudes of UST N2/P3 components in the condition when stop signals occurred less often than when they occurred more frequently.

A second objective of our study was to verify if N2/P3 components elicited on unsuccessful stop trials reflected processing of the incorrect response rather than processing of stop signals. This research objective was inspired by studies showing that an N2-like component (i.e., error-related negativity: Ne or ERN) and a P3 like component (i.e., error positivity: Pe) are elicited after incorrect actions. These components have been localized in anterior (Ne/ERN) and posterior (Pe) areas of the brain and likely reflect aspects of error processing (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). An additional result in the latter study showed that error probability did not influence the Ne/ERN component.

It was anticipated that if N2/P3 components would primarily reflect aspects of response-monitoring, this would become apparent in the following pattern of results. First, N2/P3 elicited on stop-signal trials with failed inhibition of button-press responses should show larger amplitudes in the response-locked than stimulus-locked waveforms, irrespective of the quality of the response (i.e., correct or incorrect). Second, N2/P3 should show larger amplitudes and in case of the N2, possibly a different (more anterior) scalp distribution following incorrect responses to stop signals than correct responses emitted on no stop-signal trials.

2. Method

2.1. Participants

Fourteen healthy undergraduate students (seven women) from the University of Amsterdam served as subjects. The subjects ranged in age from 18 to 23 years (\(M = 20.14, SD = 1.99\)) and all reported to have normal or corrected-to-normal vision. Two subjects were left-handed and all received course credits for their participation.

2.2. Stimuli and apparatus

Subjects were tested in a dimly lit, sound attenuating room and were comfortably seated in a chair. They were faced with stimuli at a distance of 90 cm in front of the screen and were instructed to look at the fixation plus sign during the execution of the task. Subjects responded using button boxes that were attached to the arms of the chair. Response timing was accurate to 1 ms. The primary-task stimuli (further referred as go stimuli) consisted of blue circles and squares, subtending a 0.4° visual angle and were presented against a black background on a 14-in. monitor with refresh rate of 100 Hz. A blue fixation plus sign was presented at the center of the screen during trials, subtending a 0.15° visual angle. Each trial started with the fixation plus sign for 250 ms that was followed by one of the go stimuli which was displayed for 100 ms. During the stop task, a blue cross (stop signal) was presented after onset of the go stimulus and randomly at one of the 5 fixed delays (100–150–200–250–300 ms). The duration of the stop signal was also 100 ms, with a visual angle of 0.4°. Go and stop stimuli were also presented centrally on the screen. Trial duration of the choice reaction time task (or CRT see below for more details) and stop task varied between 3.5 and 4.5 s.

2.3. Design and procedure

The experiment involved one training session and three experimental sessions that were held on four separate days. In the training session, that always preceded the experimental sessions, subjects practiced the CRT task (without stop signals) and the stop task to achieve stable response levels. A session always started with a CRT task to calculate the individual speed level. Mean RT was then used as a baseline for evaluating the RTs to the go signals of the stop task (i.e., these RTs were not expected to deviate substantially from the RTs in the CRT). Second, stop tasks were administered under two conditions: a 20% (low probability stop signals) and a 50% (high probability stop signals) condition. Each condition consisted of stop signals that were presented in random order and equally often at each of the 5 delays; the remaining 80 or 50% of the trials did not contain stop signals (i.e., no stop-signal trials).

In the experimental sessions, three blocks of CRT tasks and a total of 37 stop-signal blocks were presented. The CRT tasks contained 120 trials per block whereas
the stop-signal tasks consisted of 25 blocks of low probability stop-signal trials (each containing 100 trials) and 12 blocks of high probability stop-signal trials (each containing 120 trials). In total, an amount of 3940 trials was presented. This procedure ensured that an approximately equal number of stop-signal trials was administered under low and high probability stop-signal conditions for averaging of the ERPs.

Task order of the stop blocks was counterbalanced across subjects. The assignment of response finger (left index finger and right index finger) to the reaction stimuli (circle and square) was also counterbalanced across subjects. Subjects were instructed to respond as quickly as possible to the go stimuli while maintaining a stable level of accuracy. The importance of responding to the go stimuli was emphasized and subjects were told not to sacrifice speed to anticipate the stop signal. It was also explained that it would not always be possible to withhold their response after detecting the stop signal. Furthermore, subjects were given feedback about speed of responding at the end of each block. Halfway through the sessions a 15 min break and 1 min breaks between blocks were introduced.

2.4. Psychophysiological recording and data analysis

EEG recordings (Neuroscan) were taken from 64 sintered Ag–AgCl electrodes in an extended system (EasyCap) referenced to the left mastoid. The electrooculogram (EOG) was recorded from sites above and below the left eye and from electrodes lateral to each eye. The AFz electrode served as ground electrode. Electrode impedance was kept below 5 kΩ. The EEG signals were digitized online at a rate of 250 Hz with low-pass filter at 40 Hz and a time constant of 5 s. For each trial, an epoch of 2048 ms was obtained starting 248 ms before the onset of the go stimulus and lasting until 1800 ms after go-stimulus onset. Extraction of single trial epochs occurred offline, then EOG artifacts were corrected using the algorithm described by Woestenburg, Verbaten, and Slangen (1983).

For each subject and in each condition, artifact free, go stimulus-synchronized average waveforms were computed for both the no stop-signal trials and stop-signal trials subtracting the 100 ms pre-stimulus period as baseline per delay. No stop-signal ERPs were used for computation of ERP components to the go stimuli, while isolation of ERP components to stop signals was based on difference waves. Following the procedure of De Jong et al. (1990) and Kok et al. (2004) corresponding no stop-signal waves were computed for each delay by averaging that part of the no stop-signals in the no stop-signal distribution that corresponded with the percentage of UST which will be referred as no stop-signal fast (or CNS fast) and the percentage of SST which will be referred as no stop-signal slow (or CNS slow). The difference waves were computed by subtracting for each stop-signal delay from the stop-signal ERPs the corresponding no stop-signal ERPs (i.e., UST–CNS fast, SST–CNS slow). Prior studies have shown that this procedure is effective in removing overlap from the stop-signal ERPs caused by the go-stimulus ERPs (De Jong et al., 1990; Kok et al., 2004). Notice that the stop-signal ERP waveform comprises a summation of two different types of ERP components, namely: (a) fixed latency ERP components elicited by the go stimulus and (b) stop-signal ERP components that move systematically in time with longer duration of the stop-signal delay. Since our subtraction was carried out for each separate stop-signal delay, it is unlikely that it would generate artificial amplitude variations of ERP components caused by subtraction of ERP components that differ in latency.

Finally, ERPs on no stop-signal trials and UST trials were also averaged in synchrony with the onset of the button-press response. This was done to verify if ERP components would differ in amplitude and topography between the response-locked and stimulus-locked averages. The ERP components in these data were also extracted from difference waves computed by subtracting fast no stop-signal trials (CNS fast) from UST trials with a 100 ms pre-response period as baseline.

N2 and P3 amplitudes and latencies were subsequently calculated from the corresponding no stop-signal ERPs, as well as from the difference waves. These measures were taken from the electrode sites Fz, Cz, and Pz after visual inspection of the individual averaged waveforms, and always corresponded with segments of ERP waveforms where maximal amplitudes and latencies were observed for each component.

2 The use of difference waves entails the danger of misinterpreting results. This is true especially when the two signals are similar in morphology but differ in component latency. At the same time, however, not using differences waves in the stop task invokes the even more serious problem of allowing differential go-signal related ERP components to contaminate the stop ERPs. For instance, as the stop signal was presented later, the progressively developing P300 of the go stimulus became more discernible and contaminated the processing of the stop signal (best seen in the latest delays). Thus, examining raw waves involved spurious effects, while the common objections associated with the use of difference waves are hardly applicable to the present task. Taken together, the use of difference wave is the most preferable solution (compared to the absolute waveforms) in interpreting ERP reflections of stop-signal processing.

3 To examine the extent to which changing frequencies of stop signals would induce different strategies in processing the go signal, we might inspect go-locked ERPs in no-stop trials averaged across the entire RT distribution rather than the ‘corresponding no-stop’ ERPs that we used here. Comparison of these two waveforms yielded essentially no differences. Therefore, since stop-signal related waveforms are best examined relative to corresponding no-stop waveforms, we chose to show only the corresponding no-stop ERPs.
(150–800 ms following the stop signal). In the response locked analysis, N2 and P3 amplitudes and latencies were selected from 80 to 400 ms following the button press onset.

Statistical analyses of the behavioral data were based on repeated-measures Analysis of Variance (ANOVA) using Probability (20 and 50%) and Delay (100, 150, 200, 250, and 300 ms) of the stop signal as independent factors. ERP data were analyzed using four different repeated measures ANOVAs. First, an ANOVA was carried out on N2/P3 area measures of the (absolute) corresponding no stop-signal ERPs, using Probability (20 and 50%), Response Speed (CNS fast, CNS slow), and Leads (Fz, Cz, Pz) as factors. Second, N2/P3 area measures elicited on stop-signal difference waves were analyzed by using Probability (20 and 50%), Stop-type (UST, SST), Leads (Fz, Cz, Pz), and Delay (100, 150, 200, 250, and 300 ms) as factors and finally, an ANOVA was executed to examine if there were differences between the stimulus-locked and response-locked N2/P3 components. This comparison was restricted to the difference waves of the UST condition and consisted of the factors Synchronicity (Stimulus-Locked and Response-Locked), Probability (20 and 50%), Leads (Fz, Cz, Pz), and Delay (100, 150, 200, 250, and 300 ms) as independent factors. Third, an ANOVA was performed on N2/P3 measures extracted from the response-locked waveforms (UST–CNS fast), using Probability (20 and 50%), Leads (Fz, Cz, Pz), and Delay (100, 150, 200, 250, and 300 ms) as factors and finally, an ANOVA was executed to examine if there were differences between the stimulus-locked and response-locked N2/P3 components. This comparison was restricted to the difference waves of the UST condition and consisted of the factors Synchronicity (Stimulus-Locked and Response-Locked), Probability (20 and 50%), Leads (Fz, Cz, Pz), and Delay (100, 150, 200, 250, and 300 ms) as independent factors. Appropriate adjusted was accepted as statistically significant, and was adjusted with the Greenhouse-Geisser correction (GG) where appropriate.

It appeared from the statistics that effects of stop-signal probability on the amplitude of major ERP components were highly similar across the five stop-signal delays. This held for the stimulus-locked as well as the response-locked waveforms. In view of the large amount of ERP data in the present study, it was therefore decided to limit our report of the statistical effects of stopping on the amplitudes and latencies of stop-signal locked and response-locked ERP components to the effects of the three major factors, namely Probability (50 and 20%), Stop-type (UST and SST), and Leads (Fz, Cz, and Pz), and to show ERP waveforms that were averaged across delays. This was realized by pooling the waveforms that were calculated for each separate delay together. For computation of stop-signal ERPs the difference waveforms were first aligned with onset of the stop signal and subsequently pooled across the five stop-signal delays. In these pooled waveforms, the average onset of the stop signal relative to the go stimulus was 200 ms. An additional advantage of this procedure was that much more trials were available for computation of the ERP averages, which would increase the robustness of the statistical tests of effects of stopping, and the precision of dipole source analyses (see further below). For reasons of transparency, however, the stop-signal locked ERP difference waves that were obtained in the separate stop-signal delays are also presented in a separate figure to allow visual inspection of these data. Furthermore, the main effects of Delay are presented.

2.5. Source localization

Source localization analyses (BESA version 4.2) were carried out to explore the differences in spatial dynamics of ERP components that might result from different probabilities of the stop signal. In order to increase the precision of dipole solutions, the pooled ERP difference waveforms synchronized to the onset of the stop signal were used. Source analyses of stop-signal ERPs were applied to individual difference waveforms and were performed on the components elicited on SST and UST trials.

Modeling was performed on signals that were re-referenced to the average signal across all channels, using a four-shell spherical head model. In addition to an energy constraint that was used as a criterion to be minimized in fitting, the residual variance (RV) was included as a second criterion (<10%). Source configurations with optimal results always corresponded with one single pair of symmetrical dipoles. For each individual difference wave, instantaneous dipole models were derived. The solution parameters found in the grand average waveform were used as a marker for the single-subject solutions (cf. Kenemans, Lijffijt, Camfferman, & Verbaten, 2002). Dipole parameters that are associated with dipole location and orientation were estimated separately for each stop-signal probability. Each parameter was then subjected to repeated measurement ANOVA with Probability (20 and 50%) and Stop-type (UST and SST) as factors.

4 One of the reviewers identified a potential mathematical confound in pooling ERPs across delays. This objection, involving over-representation of fast cq slow responses, would be an issue if raw waves were used, but for the present difference waves things are more complicated. From a functional perspective there is no way (that the authors can think of) in which over-representation of fast cq slow responses, if at all an issue, would affect the interpretation of results. Since the horse race model assumes that trial-by-trial variability in the speed of Go processes is independent of trial-by-trial variability in the speed of inhibitory processes, it is assumed that those differences in the speed of inhibitory processes that are expressed in the UST vs SST difference ERPs are independent of trial-by-trial variability in the speed of Go processes. Thus, these differences (in the speed of inhibitory processes between SST and UST trials) should appear regardless of Go-RT and therefore regardless of SOA. Consequently, these differences should be expressed equally in the difference ERPs of each SOA, as well as in the differences ERP pooled across SOAs (irrespective of whether or not the latter comprised an over-representation of fast cq slow responses).
3. Results

3.1. Behavioral performance

**Reaction times.** RTs differed significantly between the CRT task, and the no stop-signal trials in the conditions in which stop signals occurred with 20 and 50% probabilities, \( F(1,13) = 31.53, p < .001 \). The average RTs in these conditions were 406 ms (SD = 45 ms), 423 ms (SD = 29 ms) and 471 ms (SD = 25 ms), respectively. With subsequent paired t tests, only RTs in the 50% no stop-signal condition were found to be significantly longer than RTs in the CRT task, \( t(13) = 7.44, p < .001 \). RTs of the no stop-signals trials were also shorter in the 20% than the 50% stop task, \( t(13) = 9.92, p < .001 \). Consistent with the assumptions of the horse-race model (Logan et al., 1984), reaction times for UST were shorter than the RTs on no stop-signal trials in either probability condition (20% stop-signal task; \( F(1,13) = 23.51, p < .001 \), and 50% stop-signal task; \( F(1,13) = 28.27, p < .001 \)). Percentages of misses to the no stop-signals in both probabilities were below 0.5% whereas percentages of wrong responses were 5.1% in the 50% stop task and 11.6% in the 20% stop task \( t(13) = 14.15, p < .001 \).

With respect to the RTs of the UST, ANOVAs further yielded a significant main effect of Probability: commission errors were faster in the 20% stop-signal task than in the 50% stop-signal task \( F(1,13) = 11.13, p = .005 \); see Fig. 1A). A main effect of Delay was also observed \( F(4,52) = 23.52, p < .001 \), indicating that RTs became slower with longer delays.

**Commission errors.** The proportion commission errors (i.e., % UST) increased with longer stop-signal delays \( (F(4,52) = 398.72, p < .001, GG = .47, \text{see Fig. 1B}) \) and was higher in the 20% than in the 50% probability condition \( (Probability (F(1,13) = 164.65, p < .001)) \). The significant Probability × Delay interaction \( F(4,52) = 9.12, p < .001, GG = .64 \) further indicated that the increase of commission errors with longer delays was somewhat stronger in the 50% than 20% condition.

**SSRT.** Stop-signal reaction times results are presented in Fig. 1C. Two aspects of these data are worth emphasizing. First, the SSRTs in both probabilities are in good agreement with the horse-race model; reaction times in the order of 200–250 ms are commonly found in stop-signal studies. The main effect of Probability was as expected not significant \( (F(1,13) = .012, p = .914) \). This notion was further confirmed as the interaction effect of Probability × Delay also did not reach significance \( F(4,52) = 1.10, p = .404 \). Second, SSRTs decreased as the stop signal was presented at later delays \( F(4,52) = 4.00, p < .001 \).

3.2. Event-related potentials

For reasons of transparency, it was decided to first report the analyses of the ERP components that were time-locked with the go stimulus, and then report the analyses of the ERP components that were synchronized to the onset of the stop signal and button-press response, respectively.

3.2.1. No stop-signal trials: Effects of fast and slow responding to the go stimulus

Fig. 2 presents the waveforms synchronized with the go stimuli, pooled across the five stop-signal delays and plotted for two RT conditions (CNS slow and CNS fast) separately for the 50 and 20% stop tasks. Main effects were found for Leads and Probability: the amplitude of the N2 component elicited by the go stimuli on no stop-signal trials was most pronounced at the anterior leads \( (F(2,26) = 55.65, p < .001) \) and was somewhat larger in the 50% task relative to the 20% task \( (F(1,13) = 12.08, p = .004) \). Furthermore, N2s with longer latencies were found on CNS (slow) trials than on CNS (fast) trials as indicated by a main effect of Response Speed \( (F(1,13) = 12.37, p = .004) \) and this latency was the largest at the frontal lead as indicated by a main
effect of Leads \( (F(2,26) = 6.04, p = .015) \). N2 latency was also somewhat longer in the 50% stop task compared to the 20% stop task at Fz, as indicated by an interaction between Probability \( \times \) Leads \( (F(2,26) = 7.80, p = .007) \).

On no stop-signal trials go stimuli that produced fast RTs (CNS fast) always elicited larger P3s than go stimuli that produced slow RTs as indicated by a main effect of Response Speed \( (F(1,13) = 37.46, p < .001) \). This effect was also more pronounced on posterior leads (Response Speed \( \times \) Leads interaction \( (F(2,26) = 4.44, p = .036) \)). Furthermore, larger go P3s were found in the low (20%) than high (50%) probability stop task \( (F(1,13) = 27.30, p < .001) \) and this effect was most pronounced at the posterior leads as indicated by a Probability \( \times \) Leads interaction \( (F(2,26) = 22.94, p < .001) \). On no stop-signal trials, P3 latency was longer in the CNS (slow) relative to the CNS (fast) trials \( (F(1,13) = 3.41, p = .032) \).

### 3.2.2. Stop-signal trials: Effects of the go stimulus

ERP components elicited by the go stimulus on stop-signal trials are depicted in Fig. 3. Of relevance here is the highlighted area showing the N2 downturn and rising flank of P3 around 400 ms after go-stimulus onset (i.e., approximately 200 ms after stop-signal onset). Notice that in this area the P3 amplitude difference between UST and SST appears to overlap almost completely with the P3 amplitude difference between fast and slow responding on no stop-signal trials. These early P3 effects are visible in the absolute waveforms, since in the difference waveforms experimental manipulations that affect no stop signal and stop-signal ERPs in a similar fashion are eliminated. Effects occurring later than 400 ms after the go stimulus are obscured by overlapping ERPs elicited by the stop signal. Note also that these longer-latency ERPs to the stop signals are

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**Fig. 2.** Grand average ERPs at the three locations of the corresponding no stop-signal trials, synchronized with the onset of the go-stimulus and displayed for two categories of no stop-signal trials (CNS slow and CNS fast) and two stop-signal probabilities (50 and 20%). Waveforms are computed by pooling over the five stop-signal delays.
3.2.3. Stop-signal trials: Effects of the stop signal

Fig. 4 depicts the difference waves that were calculated by subtracting no stop-signal trials (CNS fast and CNS slow) from the respective stop-signal ERPs (UST and SST) for each stop-signal delay. These difference waves were aligned with the onset of the stop signal and subsequently pooled across the five stop-signal delays. Difference waveforms were characterized by a prominent negativity (labeled stop-N2 at about 200 ms after stop-signal onset) followed by a large late positivity (labeled stop-P3 at about 400 ms after stop-signal onset). Furthermore, stop P3 is followed by a large central-posterior negativity on SST which is much more prominent in the high than low probability task. Also visible is a shift in latency of the N2/P3 complex on UST relative to SST.

Stop-N2 amplitude. N2 amplitudes were more enhanced in the UST than in the SST condition, $(F(1,13) = 24.27, p < .001$, see also Fig. 5 and Table 1A). Although no main effect was found for Probability, smaller amplitudes were observed in the UST condition in the 50% stop task relative to the 20% stop task, while the opposite effect (N2 amplitude 20 > 50%) was found for SST, as evidenced by a Probability $\times$ Stop-type interaction $(F(1,13) = 7.74, p = .016)$. A main effect was also found for Leads $(F(2,26) = 7.16, p = .009)$. The largest amplitudes were observed at the centroparietal leads and in the UST condition, as indicated by an interaction effect between Stop-type $\times$ Leads $(F(2,26) = 7.16, p = .009)$.

Stop-N2 latency. With respect to stop-N2 latency, main effects were found for Stop-type, $(F(1,13) = 20.83, p = .001)$ and Leads $(F(2,26) = 9.59, p = .003, G^2 = .58)$. This confirmed the visual impression that N2 peaked later on UST than SST, and somewhat later at posterior electrode sites than at the central and the anterior sites, see also Table 1B.

Stop-P3 amplitude. Stop-P3 amplitudes were larger in the 20% than in the 50% stop-signal probability task,
and were larger on UST than SST ($F(1,13) = 6.86, p = .021$; see also Fig. 5 and Table 1A). An interaction between Probability $\times$ Stop-type ($F(1,13) = 8.72, p = .011$) confirmed the visual impression that the enlarged P3 amplitude in the 20% probability task was more prominent for UST than SST. The interaction effect of Probability $\times$ Leads ($F(2,26) = 25.14, p < .001, GG = .66$) indicated that the probability effect was more prominent at the parietal electrode. Finally, the interaction effect between Stop-type $\times$ Leads ($F(2,26) = 37.45, p < .001$) reflected that stop-P3 amplitude was largest at the frontocentral leads on SST, but largest at the parietal lead on UST.

**Stop-P3 latency.** P3 latency appeared to peak later in the 20% stop task compared to the 50% stop task, which was reflected in a main effect of Probability ($F(1,13) = 34.29, p < .001$). ANOVA also yielded significant main effects of Stop-type ($F(1,13) = 33.02, p < .001$) and Leads ($F(2,26) = 19.44, p < .001, GG = .72$), that is, P3 peaked later in the UST compared to the SST condition, and later at frontal and central leads compared to the parietal lead. The probability
difference was affected by Leads which implies that the increase of P3 latency to lower frequency stop signals was more pronounced at the posterior electrodes, \(F(2, 26) = 6.16, p = .014, \text{GG} = .88\). The latter effect also appeared to be strongest for SST, as reflected in the three-way interaction effect between Probabil-
Fig. 5 depicts difference waves for both probabilities separately for the five consecutive delays. As can be noticed in the figure, successfully and unsuccessfully stop trials varied as a function of delay and as expected, main effects of Delay were found for P3 latency ($F(4,52) = 349.68, p < .001, \text{GG} = .67$), P3 amplitude ($F(4,52) = 6.81, p = .006, \text{GG} = .47$), N2 amplitude ($F(4,52) = 17.50, p < .001$) and N2 latency ($F(4,52) = 640.02, p < .001, \text{GG} = .54$). That is, larger and longer values were observed for these components as a function of delay. Most importantly, however, the patterns of effects seen in the difference waves pooled across delays remain basically unaltered when these difference waves are examined per separate delay, see also Fig. 4.

3.2.4. Stop-signal ERPs: Response-locked averages

Fig. 6 (left panel) depicts the ERP activity synchronized with onset of the button-press response on no stop-signal trials and stop-signal trials, for the high and low stop-signal frequency tasks. At the anterior electrode sites UST waveforms show two negative peaks around 80 and 100 ms post-response, followed by a large positivity peaking around 300 ms post-response in both probability tasks. These components are of much smaller amplitude in the no stop-signal trials. The difference waves (right panel Fig. 6) show a large negative peak (N2) around 100 ms followed by a larger positive wave peaking around 300–350 ms post-response (P3).

Response-related negativity (N2 amplitudes). A main effect of Leads was observed for the N2 amplitude ($F(2,26) = 8.65, p = .005$) indicating that the largest amplitude was found at the central leads rather than the frontal and parietal leads, see Table 2A.

Response-related negativity (N2 latencies). No results reached significance.

Response-related positivity (P3 amplitudes). A main effect of Probability was found ($F(1,13) = 44.31, p < .001$) indicating that larger amplitudes were found for the low than high probability stop task, see also Fig. 6 and Table 2A. Amplitudes also varied per lead, with the central and parietal leads showing the largest difference, as indicated by an interaction between Probability × Leads ($F(2,26) = 6.06, p = .015$).

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Fig. 6. Grand average of corresponding no stop-signal ERPs (CNS fast) and UST ERPs displayed for the two stop-signal probability tasks. Waveforms are computed by pooling over the five stop-signal delays in synchrony with the onset of the response. Left panel: absolute waveforms and right panel: difference waveforms.
Values are taken from the leads (Fz, Cz, Pz), see also Fig. 6.

Table 2A
Means and standard deviations of N2 and P3 amplitudes derived from the pooled response-locked difference waves in both probability tasks (in µV)

<table>
<thead>
<tr>
<th></th>
<th>N2</th>
<th>P3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fz</td>
<td>Cz</td>
</tr>
<tr>
<td>UST–CNS (fast) 50%</td>
<td>-4.6</td>
<td>-5.8</td>
</tr>
<tr>
<td>SD</td>
<td>3.1</td>
<td>3.7</td>
</tr>
<tr>
<td>UST–CNS (fast) 20%</td>
<td>-4.3</td>
<td>-5.3</td>
</tr>
<tr>
<td>SD</td>
<td>2.3</td>
<td>3.1</td>
</tr>
</tbody>
</table>

Values are taken from the leads (Fz, Cz, Pz), (see also Fig. 6).

Table 2B
Means and standard deviations of N2 and P3 latencies derived from the pooled response-locked difference waves in both probability tasks (in µV)

<table>
<thead>
<tr>
<th></th>
<th>N2</th>
<th>P3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fz</td>
<td>Cz</td>
</tr>
<tr>
<td>UST–CNS (fast) 50%</td>
<td>120</td>
<td>114</td>
</tr>
<tr>
<td>SD</td>
<td>23.3</td>
<td>29.7</td>
</tr>
<tr>
<td>UST–CNS (fast) 20%</td>
<td>110</td>
<td>112</td>
</tr>
<tr>
<td>SD</td>
<td>26.1</td>
<td>23.5</td>
</tr>
</tbody>
</table>

Values are taken from the leads (Fz, Cz, Pz) (see also Fig. 6).

Response-related positivity (P3 latencies). With respect to latency, main effects for Probability \((F(1,13) = 5.98, p = .029)\) and Leads \((F(2,26) = 50.45, p < .001)\) were observed. That is, P3 latency peaked slightly later in the 20% as compared to the 50% stop task, and was longer at the parietal lead as compared to the frontocentral lead, see also Table 2B.

Stimulus-locked vs response-locked N2 amplitudes. The N2 amplitude was the smallest on the parietal lead, somewhat larger on the frontal lead and the largest on the central lead \((F(2,26) = 15.03, p = .001)\). No effects were found of the factor Synchronicity.

Stimulus-locked vs response-locked P3 amplitudes. With respect to P3 amplitude, main effects of Probability \((F(1,13) = 44.47, p < .001)\) and Leads \((F(2,26) = 7.30, p < .020, GG = .69)\) were observed, that is, P3 amplitudes were larger in the 20% stop task, and larger at the parietal lead. Furthermore, in the 20% stop task slightly larger P3 amplitudes were observed in the stimulus-locked analyses as compared to the response-locked analyses (Probability × Synchronicity interaction \((F(1,13) = 8.04, p = .014)\)). Finally, larger amplitudes were found in the 20% stop task for the parietal lead compared to the frontocentral leads, as evidenced by a Probability × Leads interaction \((F(2,26) = 11.64, p = .002)\).

3.2.5. Stop-signal ERPs: Topographical and source analyses
As the previous statistical analyses revealed that largest effects of stop-signal frequency occurred for the P3s elicited on UST and SST, source modeling was limited to this component. Voltage maps and dipoles were based on P3 peak amplitudes. These amplitudes were derived from the same difference waves that are displayed in Fig. 4. Figs. 7A and B show the effects of stop-signal probability on the scalp topographies and dipoles of P3 elicited in the SST and UST conditions. Fig. 7A shows that for SST P3 approximately similar scalp distribution were found for both the high and low frequency stop-signal conditions. Visual inspection indicated that the symmetric dipoles were located more anteriorly and slightly deeper in the cortex in the 20% than in the 50% stop-signal condition (50% probability: residual variance (RV) of the grand average pair of dipoles = 6.8%: location of the grand average dipoles: \(x = -19.9; y = 13.4, z = 67.7, 20\% \) probability: RV = 5.3%: location: \(x = -16.9; y = 27.6, z = 29.1\). For UST P3 in the 50% stop task, the majority of the individual dipole pairs were localized in the anterior part of the frontal lobe, but deeper in the brain than for SST P3. (RV = 4.0%, \(x = -16.9; y = 27.6, z = -04\). In contrast, UST P3 dipoles in the 20% stop task were located in the lower part of the parietal lobe (RV = 6.8%; \(x = 17.7; y = -30.0; z = 9.5\).

ANOVA's carried out on location parameters globally confirmed the prior observations. Dipoles were located more eccentric in the SST than in the UST condition (\(x\)-parameter Stop-type: \(F(1,13) = 12.57, p = .004\)). This effect was enhanced in the 20% stop task (Probability × Stop-type: \(F(1,13) = 6.79, p = .022\)). SST dipoles were also located more anteriorly than UST dipoles (\(y\)- parameter Stop-type: \(F(1,13) = 11.22, p = .005\), and this effect was more pronounced in the 20% than in the 50% stop task (Probability × Stop-type: \(F(1,13) = 14.03, p = .002\)). Finally the UST dipoles were located deeper (i.e., more ventrally) in the brain than the SST dipoles (\(z\)-parameter Stop-type: \(F(1,13) = 26.72, p < .001\).
Orientation parameters of the dipoles were also subjected to ANOVAs. Dipoles were oriented more laterally in SST than in the UST condition, \( (x\text{-parameter Stop-type} \quad F(1,13) = 14.56, \quad p = .002) \) and more anteriorly in the 50% than 20% stop task \( (y\text{-parameter Probability} \quad F(1,13) = 9.48, \quad p = .009) \).

### 4. Discussion

The primary purpose of this study was to explore the effects of stop-signal frequency on response inhibition using performance indices and components of the ERP as dependent measures. It was argued that presenting stop signals at lower probabilities would result in a stronger bias to the go stimuli. We further assumed that this manipulation would also affect processing of the stop signals, and generate different types of processes on successful and unsuccessful stop-signal trials. On successful stop-signal trials more inhibitory pressure is needed to overcome the stronger set to the primary (go) task. This was presumed to lead to an anterior shift of the frontocentral P3 elicited on successful inhibit trials, reflecting a stronger engagement of prefrontal cortex. A different pattern was predicted to occur for ERP components that were elicited on failed inhibit trials, in particular the posterior N2/P3 complex. A previous study (Kok et al., 2004) suggested that these components were more associated with error-related processing activities. Presenting stop signal less frequently implies that processing of stop signals and possibly also errors of action receive less emphasis.
Therefore, these components were assumed to be less pronounced in the condition when stop signals were presented less frequently.

The second purpose of this study was to verify if N2/P3 components that were elicited on failed inhibit trials would be associated with processing of the error response or with processing of the stop signals. For this purpose response synchronized ERPs were compared with ERPs that were synchronized with onset of the stop signal. Enhanced amplitudes of N2/P3 components in response-locked averages would constitute evidence for the notion that these components were uniquely related to error-related processing activities.

### 4.1. Behavioral results

When stop signals occurred less frequently, go stimuli elicited faster RTs and stop signals elicited more commission errors than when stop signals occurred more frequently. This result clearly appears to confirm the notion that when stop signals are presented less frequently, subjects tend to sacrifice success of inhibition to speed of responding to go stimuli. RTs in this study were similar in magnitude compared to previous stop studies with fixed delays (e.g., Logan & Burkell, 1986; Logan et al., 1984). The number of commission errors increased as a function of delay, and was substantially higher in the task when stop signals were presented less frequently, which also replicates the results of Logan and co-workers. In agreement with previous stop studies, the speed of processing of the stop signal (SSRT) was in the order of 200–250 ms, and decreased as a function of delay. SSRT remained constant between probabilities, indicating that the latency of response inhibition was itself unaffected by the frequency of presentation of the stop signals. This finding further corroborates the notion derived from prior stop-signal studies that an increased set-to-go is the dominant mechanism behind faster responses to go stimuli in the lower frequency stop-signal task (Logan et al., 1984). Note that a closer look at the behavioral results show that the increase in % UST in the first delay in the low probability stop task corresponds to the % UST in the second delay in the high-probability stop task and that this pattern is consistent for all delays. This is probably due to a shift in the no stop-signal distribution in the low-probability stop task that also causes a shift in the distribution of inhibition. This indicates that despite the increase of % UST trials, inhibition is not necessarily worsened.

Notice also that the present behavioral findings are not conclusive with respect to the question which information processing stages were specifically affected by stop-signal probability. After almost three decades of research the locus of the effect of stimulus probability, that is, the elements of information processing that are affected by preparatory set, still remains a matter of controversy (Gehring, Gratton, Coles, & Donchin, 1992; Miller & Pachella, 1973; Sanders, 1980; Sternberg, 1969). The present ERP findings however did permit us to segregate the effects of stop-signal probability on processes associated with the primary (go) stimulus and stop signal, as well as the processes that preceded and followed commission errors to the stop signals. These findings will be further elucidated below.

### 4.2. ERPs to the go-stimuli

The suggestion, derived from the behavioral results, that a lower stop-signal frequency created a stronger set or bias to the go-stimuli was further supported by the ERP amplitude findings derived from no stop-signal trials. N2 and P3 components that were elicited by go stimuli occurred slightly earlier in the task when stop signals occurred less frequently. It further appeared that the amplitude of P3 to the go stimuli was enhanced in the low relative to the high stop-signal frequency task, and on fast relative to slow no-stop-signal trials (Fig. 2; keep in mind that on no-stop-signal trials the frequency of presentation of go-stimuli in the low- and high-probability stop-signal tasks was 80 and 50%, respectively). This effect was most conspicuous for the ascending flank of go P3. It could also have represented a modulation of an earlier component (i.e., P2), since close inspection of the no-stop-signal ERPs suggests that a late positivity around 500 ms is preceded by a smaller positivity around 400 ms.

At first sight, this amplitude effect is in contradiction to the typically observed inversed relationship between event probability and P3 amplitude (e.g., Duncan-Johnson & Donchin, 1977; Low & Miller, 1999). It is proposed however that in the present study this P3 enhancement to high frequency go stimuli reflects the operation of the same mechanism that also caused the amplitude enhancement of P3 on fast RT trials, namely a stronger bias toward to the stimuli of the primary task. Interestingly, effects of successful versus unsuccessful stopping were also manifested in the amplitude of the P3 to the go stimulus (see Fig. 3, highlighted area). Since this early amplitude difference between UST/SST P3 mirrored the effect of fast/slow responding on no stop-signal trials on the same component, it likely reflected processing of the go stimulus and not of the stop signal. Thus, these findings suggest that processing of the go stimulus was affected in a similar fashion by factors like low signal probability, fast responding, and unsuccessful stopping. The common process underlying these three factors could have been a stronger set to produce fast responses to go stimuli. A stronger preparatory set is known to be reflected in a sustained anticipatory negativity or CNV-type of waves (e.g., Rösler, 1991). This type of activity was not visible in our baseline corrected...
ERPs, but the enlarged P3 (or P2) to the go stimulus could very well have reflected some form of resolution of an anticipatory negativity that preceded the go signal.

4.3. ERPs to the stop signal

Stop signals used in this study elicited a N2/P3 complex that seems specifically related to the visual modality of the stop signals (Ramautar, Kok, & Ridderinkhof, in preparation).

Effects of stopping on N2/P3 globally replicated the effects reported in our earlier stop study (Kok et al., 2004). A frontocentral P3 emerged on trials where a response was successfully withheld, while a more posterior P3 was elicited on trials where a commission error was made. The major effect of presenting stop signals less frequently was a shift in latency and an amplitude enhancement of N2/P3 components to the stop signal, which was manifested more strongly on SST than UST.

Latency effects. The N2/P3 complex to the stop signal shifted systematically in time with longer duration of the stop-signal delay. The latency of the N2/P3 was also slightly longer on low- than high-probability stop tasks and was longer on UST relative to SST. These effects are of theoretical importance because they indicate that in contrast to performance measures (SSRT remained stable), ERP latency measures did suggest that subjects needed more time to process low-probability stop signals, as well as stop signals on unsuccessful inhibit trials, in comparison to high-probability stop signals and stop signals on successful inhibit trials. It is possible that both latency effects were generated by the same mechanism, namely a stronger set to respond to the go stimuli. Differences in timing of the internal response to the stop signal could have been caused both by a trade-off between go and stop processes, as well as spontaneous fluctuations in the speed of stop processes. A subtle delay in processing the stop signal could have meant that the stop signal was no longer effective in disrupting the concurrent response inhibition.

Amplitude and topographical effects. SST P3 seems to have much in common with the frontocentral NoGo-P3 that has been found in Go-Nogo tasks (Kiefer, Marzinik, Weisbrod, Scherg, & Spitzer, 1998; Kok, 1983, 1986; Pfefferbaum & Ford, 1988; Pfefferbaum, Ford, Weller, & Koppell, 1985; Roberts, Rau, Lutzenberger, & Birbaumer, 1994) and also in the stop-signal paradigm (De Jong et al., 1990, Fig. 6; Kok et al., 2004). Presenting stop signals less frequently enhanced the amplitude of SST P3 and resulted in a subtle but significant shift of its dipole sources from precentral to more frontal locations. This finding confirms our prior hypothesis that when stop signals are presented less frequently, more inhibition is needed to overcome the strong response bias. One possibility, suggested previouly (Kok et al., 2004) is that generators in precentral (or motor) cortex are more active when control of movement is guided by external events (Goldberg, 1985; Kalaska & Crannie, 1995). On the basis of the present results it can further be speculated that presenting stop signals less frequently could also have induced a shift from external to internal control mechanisms that rely more strongly on frontal areas in the brain. We do not intend to assert that SST P3 represents the actual suppression of the go response. The point in time when this component reached its maximal amplitude (i.e., around 400 ms after onset of the stop stimulus) was too late to reflect neural activity that could have caused the actual inhibition of the motor response in the stop task. However we verified in a separate statistical analysis that the SST P3 waveform already started to deviate from the no-stop-signal waveforms around 280–300 ms after stop-signal onset (see Ramautar, Kok, & Ridderinkhof, in press). This effect is possibly early enough to provide a marker of processes that were involved in actual response inhibition.

Partly in contrast to our prediction, only posterior P3 components that were elicited on failed inhibition trials appeared to be much larger when stop signals were presented less frequently. Thus, despite the finding that priority is shifted from stop to go processes when stop signals occur less frequently, the present ERP finding suggests that low-frequency stop signals that are followed by failed inhibitions are perceived as more rather than less meaningful. One possibility is that this reflected a combined effect of subjective probability and a higher significance of errors of action. Subjective probability - or the oddball effect - is known to affect in particular the amplitude of the classical P3b component (Donchin, Karis, Bashore, Coles, & Gratton, 1986; Duncan-Johnson et al., 1977). P3b is further known to have a widespread distribution in the more posterior areas of the brain, which also includes structures in the brainstem (McCarthy, Luby, Gore, & Goldman-Rakic, 1997; Me-non, Adleman, White, Glover, & Reiss, 2000). Dipole source analyses of UST P3 yielded a pattern that was consistent with generators reported to underlie P3b. The dipole pattern however was highly variable across individual subjects. This could be a sign that multiple local sources were involved in the generation of UST P3, each of which varied in location across individual subjects. UST P3 could also have represented a ‘mix’ of generators, namely generators associated with the attempt to inhibit the response as well as generators associated with processing of the error responses (after failed inhibitions). The symmetric dipoles of the grand average UST P3 were localized in lower medial temporal lobe in the 50% probability stop task, whereas the 20% probability stop task activated generators in lower parietal sub-lobar areas, in particular in the vicinity of the thalamus and pulvinar. Perhaps the most plausible interpre-
tation of these deep dipoles is that they represented a center of gravity of a more widely distributed network extending from areas in posterior cortex to areas in anterior cortex.

4.4. Response-locked versus stimulus-locked averaging

A second aim of this study was to verify whether N2/P3 components that were elicited on unsuccessful stop trials reflected processing of the error response or processing of the stop signal. The N2 component did not differ between go-synchronized and response-synchronized data, see also Tables 1A,1B, and 2A, 2B. This finding is most readily explained by assuming that N2 was not uniquely generated by response-related processes such as monitoring of errors (see further below under alternative explanations) but is also possibly related to processes related to the stop signal. On the other hand, P3 amplitude and topography did differ between go-synchronized and response-synchronized waveforms. The larger P3 in stop-signal locked averages indicates that in contrast to the N2, the P3 amplitude could have reflected processes that were generated by the stop signal. However, considering that UST P3 reached its maximum amplitude at approximately 300 ms after response onset, and its more posterior source compared to the SST, this component could also have incorporated processing of the erroneous response.

**UST N2/P3: Attempt at integration of principal findings.** A tentative explanation why latencies of N2/P3 were longer and P3 amplitudes much larger on unsuccessful stop trials in the low probability stop-signal task is the following. The stronger preparatory set to the go stimuli when stop signal occurred less frequently could have caused a concomitant delay of processing of stop signals, as reflected in a prolonged latency of N2/P3. Furthermore, a stronger set to go stimuli will not only cause fast RTs but will also increase the likelihood of committing errors of action on a substantial number of trials of the stop task. On these trials subjects may have been aware (either consciously or unconsciously) that the presented stop signals were no longer effective to prevent execution of the motor response, even when they had not yet completed the motor response. This probably led to an increase of significance or meaning of the stop signals as reflected in an amplitude increase of N2 before the response was emitted. Thus despite the higher priority of the primary (go) task relative to stopping subjects could still have perceived stop signals that forewarned a failed inhibition as more meaningful especially when these signals occurred less often. The same reasoning holds for UST P3, but this component could also have reflected continued processing of errors of action after emission of the button press response.

4.5. Alternative accounts of the present ERP findings

The present findings can also be considered from alternative theoretical perspectives that have recently received much emphasis, and are briefly discussed below.

**Error related negativity (ERN/Ne) and error positivity (Pe).** The error-related negativity is elicited both by errors of choice in a choice reaction task (Falkenstein, Hoormann, Hohnsbein, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993) and by errors of action in go/nogo task (Falkenstein, Hohnsbein, & Hoormann, 1995; Scheffers, Coles, Bernstein, Gehring, & Donchin, 1996). ERN/Ne has also been shown to occur after feedback stimuli signaling that an error has been made (Müllner, Braun, & Coles, 1997). The enhanced N2 to unsuccessful inhibit trials that was found in the present study shows some functional similarity to the ERN/Ne, in particular the ERN/Ne that is elicited by errors of action (e.g., Scheffers et al., 1996). However, two findings render it less likely that this component reflected the same neural systems or mechanisms that are indexed by the error negativity. First, numerous studies have presented evidence that the generators of Ne/ERN are located in medial frontal areas (particularly in the anterior cingulate cortex), but there was little evidence that this area was involved in the N2 elicited on failed inhibition trials. Second, the error negativity is typically larger on response-locked than stimulus-locked waveforms. As noted earlier, inspection of response-locked ERPs that were elicited on failed inhibition trials (Fig. 6) did not confirm the suspicion that the N2 component was triggered specifically by the error response. In the absolute waveforms however, two frontal negativities were more pronounced in the unsuccessfully inhibit condition compared to the no stop-signals. Interestingly, the first negativity did have the same scalp distribution and timing as ERN/Ne. Since this early N2 component could not be clearly observed in the stop-locked waveforms it could indeed have represented monitoring of the error response similar to the ERN/Ne.

A second ERP component that seems to be related to detection or monitoring of errors is the error positivity. This component is also elicited when subjects detect an error in performance and shows larger amplitudes for errors of action in go/nogo tasks than errors of choice (see Falkenstein et al., 2000 for a review). Thus, it is possible that the present UST P3 was similar to the Pe. The functional significance of Pe is however still a matter of debate (e.g., Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001), and it is not yet clear to what extent this component can be separated from the traditional P3b or involves the same or different generators as P3. The present finding showed that UST P3 was much larger to errors after infrequent than frequent stop signals. This can therefore be interpreted from two different perspectives. It can be seen as a demonstration that, (a) Pe
is typically larger after infrequent errors or that (b) P3b amplitude is sensitive to the a priori probability of task-relevant events, in particular when this event is associated with an error in performance.

**NoGo N2.** Previous studies have repeatedly shown that in Go-NoGo tasks, NoGo-stimuli are followed by an anteriorly distributed N2. ERP studies have further suggested that the NoGo N2 reflects the operation or outcome of an inhibitory process in frontal cortex (Jodo & Kayama, 1992; Kok, 1983, 1986; Kopp, Mattler, Goertz, & Rist, 1996; Pfeflerbaum et al., 1985). Neuroimaging (fMRI) studies also indicated involvement of prefrontal cortices in NoGo trials (Garavan et al., 1999; Konishi et al., 1999; Rubia et al., 2001; Rubia, Smith, Brammer, & Taylor, 2003). The NoGo N2 is also more pronounced when NoGo stimuli are presented at lower than higher probabilities (Eimer, 1993; Bruin & Wijers, 2002). In the present study the N2 was larger in the low- than in the high-probability stop task, but more pronounced for unsuccessful stop-signal trials than the successful stop-signal trials. The present stop N2 however showed a less pronounced anterior topography that is typical for NoGo-N2. These findings indicate that the present stop-signal N2 might not be simply equated with the No-Go N2, and its interpretation in terms of response inhibition.

**Conflict-monitoring.** According to conflict theory (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001), any situation that causes a simultaneous activation of two competing response tendencies (such as two alternative responses or generation or suppression of a single response) should increase activity of a conflict monitoring mechanism. This mechanism is presumed to be located in anterior cingulate cortex (e.g., Carter et al., 1998). Furthermore, it has been argued that low-frequency responses involve a stronger conflict than high-frequency responses (Braver, Barch, Gray, Molfese, & Snyder, 2001; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). Several findings of the present study speak against an interpretation in terms of conflict monitoring. If indeed performance in the stop task can be modeled by a single mechanism (i.e., conflict), then presenting stop signals less frequently should result in a stronger involvement of frontal-mesial generators, irrespective of the outcome (successful or unsuccessful) of the stopping process. However, the different configurations of the dipole sources and scalp topography of the ERP components (in particular P3) in this study clearly speak against a single mechanism underlying stopping performance in the stop task. With respect to N2 which has been associated with response conflict in Go/NoGo tasks (Nieuwenhuis et al., 2003), it appeared that although generators were located in the typical frontal areas involved in conflict monitoring, the probability effect was much larger on UST than SST trials. Finally, conflict theory predicts that the timing of conflict on error trials should follow the onset of the response, while in the present study UST N2 was visible in the waveforms that were time-locked with the stop signal as well as the waveforms that were time-locked with onset of the erroneous response, although the moment at which these responses were maximally activated certainly depends on the relative timing of the two (go and stop) processes.

### 4.6. Conclusion

To summarize, presenting stop signals less frequently was accompanied by a stronger set to produce fast go responses. This was manifested not only in the profile of performance measures, but also in ERP components elicited by go and stop signals. The amplitude of go P3 was enhanced in conditions or trials that favored fast responses to the go stimuli and probably reflected a return to baseline or resolution of a negative anticipatory shift that preceded the go stimuli. With respect to ERPs elicited by the stop signal it is concluded that processes underlying successful and unsuccessful stopping were functionally and anatomically separable. Successful stop P3 probably reflected processes that were related to withholding a prepotent motor response. These processes became more dependent on frontal regions in the brain when stop signals were presented less frequently. The P3 component elicited on unsuccessful stop trials probably reflected a greater significance of stop signals and responses on error trials. These components depend on a more diffuse network in the brain and are typically emitted when subjects become aware that they are unable to withhold an immediate response to low probability stop signals.

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### References


