Effects of stop-signal modality on the N2/P3 complex elicited in the stop-signal paradigm

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Abstract

The principal aim of the present study was to clarify how stop-signal modality affected the speed and efficacy of stopping, using ERP components as converging measures of stop processes. Both performance and ERP latency findings suggested faster processing of stop signals in the auditory than visual version of the stop task.

The effects of successful versus unsuccessful stopping on the amplitude and topography of N2/P3 components elicited by the stop signals appeared to be largely independent of the modality of the stop signals. Stop signals elicited a fronto-central N2 that was much larger on unsuccessful than successful stop trials in stimulus-locked waveforms. N2 was followed by a P3 component that showed a fronto-central distribution on successful stop trials. P3 elicited on unsuccessful stop trials showed a posterior-parietal focus, but this topography was manifested more clearly in response-locked than stimulus-locked waveforms. A dipole source analyses confirmed these topographical differences of P3, and further showed that the location of the corresponding dipoles remained largely identical across the visual and auditory versions of the stop-signal task. Taken together, the present findings support the suggestion that ERP components in the stop task reflect endogenous aspects of stop-signal processing, such as effective inhibition of responses on successful stop trials and detection of errors on failed inhibition trials.

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1. Introduction

The ability to adjust behavior by means of inhibitory interventions is crucial for the maintenance and control of cognitive and motor events. It is also considered to be a basic act of executive control, a cluster of emergent functions that is typically associated with the prefrontal regions of the brain (Fuster, 1997; Ridderinkhof et al., 2004). Inhibitory interventions can be rather extreme and take the form of complete abortion of overt behavior. Such interruptions of overt movements can occur on the basis of internally generated acts of control but can also be triggered by external events (Logan and Cowan, 1984; Kok, 1999). An effective task to assess abrupt forms of response inhibition is the stop-signal task (Ollman, 1973) in which a choice-reaction task (the primary-task) is combined with the presentation of a stop signal. In the stop task subjects are instructed to perform speeded responses to stimuli in the primary-task (also referred to as go signals) but to inhibit their response whenever a stop signal is presented. The stop signals are usually presented at variable delays after the go signals. This manipulation is necessary for computation of inhibition functions, that is functions that relate the probability of inhibiting the response to stop-signal delay (see further below). In addition, trials on which only go signals are presented (no stop-signal trials) are randomly interspersed among trials on which the go signal is followed by a stop signal (stop-signal trials).

Performance data generated by the stop task have been interpreted in terms of a race between two independent processes, a go process and a stop process that run for completion (Logan et al., 1984; Logan, 1994). The go process is invoked by the onset of the primary-task stimulus...
and comprises the stages of stimulus identification, response choice, preparation and execution of the response, whereas the stop process reflects the processing of the stop signal that includes the detection of the stop signal and the implementation of response inhibition. The winner of this race determines whether a response is given or not (Lappin and Eriksen, 1966; Logan et al., 1984). Consistent with the race model subjects are more effective in stopping as the delay between the go signal and stop signal is shortened and less effective when the delay becomes longer. The stop signal divides the no stop-signal RT distribution (go trials) into two parts. This is represented in Fig. 1 by the vertical line extending upward from the point at which the response to the stop signal occurs. On the left part the response to the primary-task is faster than the response to the stop signal, and on the right side of the line the response to the stop signal is faster than the response to the primary-task.

In contrast to the direct observation of processing speed of the primary-task, the duration of the non-observable, internal reaction time to the stop signal (SSRT) is estimated on the basis of the distribution of reaction times and the proportions of successful stop-signal trials (further referred to as SST) or unsuccessful stop-signal trials (further referred to as UST; see also Fig. 1) and stop-signal delay. This approach has shown that the SSRT is more or less constant and is usually between 200 and 250 ms (Logan, 1994; Band et al., 2003). The horse-race model as such is not primarily concerned with the nature of processes underlying successful and unsuccessful stopping. A number of studies have, therefore, measured event-related potentials (ERPs) to obtain more insight into the dynamics of the motor inhibition process in the stop-signal paradigm (De Jong et al., 1990; Kok et al., 2004; Ramautar et al., 2004a,b; Van Boxtel et al., 2001). A complicating factor is that ERPs elicited on stop-signal trials comprise a summation of a fixed-latency ERP waveform elicited by the go stimulus, and an ERP waveform elicited by the stop signal that moves systematically in time with longer duration of the stop-signal delay. One of the methods that can be followed to isolate the stop-signal ERPs is to compute, separately for each individual and for each stop-signal delay, average ERPs for no stop-signal trials as well as for stop-signal trials, and then subtract from the averaged stop-signal ERPs the no stop-signal ERPs. Prior studies have shown that this procedure is effective in removing from the stop-signal ERP overlap caused by the preceding go stimulus ERP (e.g. De Jong et al., 1990; Ramautar et al., 2004a; cf. Bekker et al., 2005). One of the findings of our earlier studies was that stop signals elicited a N2/P3 complex that shifted systematically in time with longer delays of the stop signal (Kok et al., 2004; Ramautar et al., 2004b). These N2/P3 components were further shown to differ considerably between SST and UST with respect to timing (latency), scalp topography, and dipole sources. Thus, these ERP findings not only demonstrated that N2/P3 represented components were truly linked to processing of the stop signals, but also suggested that these components reflected different aspects of stopping. More specific, we hypothesized that (a) N2 and P3 components elicited on UST reflected a greater significance (possibly as some form of negative feedback) of stop signals and their associated false alarm responses and (b) P3 elicited on SST indexed processes related to withholding a prepotent response.

ERPs have also proven to be useful in clarifying how stimulus variables in the stop task affect inhibition functions, that is, functions that relate the probability of inhibiting to the stop-signal delay. For instance, presenting stop signals less frequently is known to result in faster reaction times to the go stimulus and in a lower probability of inhibition. Ramautar et al. (2004b) reported that when stop signals were presented less frequently, go stimuli on no stop-signal trials elicited larger and somewhat earlier P3-like responses than when stop signals occurred more frequently. Since a similar pattern was found when P3s elicited on fast and slow go responses (on no stop-signal trials) were compared, these ERP results seemed to confirm the suggestion derived from the behavioral literature that in conditions in which stop signals occur less frequently subjects develop a stronger set or bias to produce fast go responses at the expense of success of inhibition (Logan, 1981; Logan and Burkell, 1986).

The modality of the stop signal is another variable that has also been shown to affect the speed and efficiency of stop behavior. Traditionally, stop studies have been performed
with auditory stop signals to stress the salience of the stop signal (Logan and Cowan, 1984; Logan et al., 1984, 1986; Ollman, 1973). Auditory stop signals are thought to enhance the speed of stop processes relative to visual stop signals. Although the precise locus of the modality effect still remains unclear, it is clear that sensory factors could play a crucial role. A classical finding from the RT literature is that simple RTs to auditory stimuli are about 40 ms faster than simple RTs to visual stimuli (Woodworth and Schlosberg, 1954). This RT difference could arise from peripheral factors, such as a faster transmission of sensory information at mechanical receptors of the ear than at the photosensitive receptors of the retina. In the stop-signal task, this effect could give auditory stop signals a small but consistent lead in the race with the go signal in comparison with visual stop signals. Indeed, SSRT has been shown to vary systematically as a function of task variables, such as stop-signal discriminability (Van den Wildenberg and Van der Molen, 2004).

1.1. The present study

The principal aim of the present study was to further clarify how stop-signal modality could affect the speed and efficacy of stopping, using ERP components as converging measures of stop processes and using same and cross-modality stop tasks. In the same modality stop task visual go and stop signals were presented whereas in the cross-modality stop task, visual go and auditory stop signals were presented.

In our earlier studies N2/P3 components elicited by (visual) stop signals were interpreted in terms of internally generated processes involved in inhibition and monitoring of responses. The validity of this interpretation remains to be established more firmly, since it derives primarily from the assumed functional relationship between the frequency of occurrence of the stop signal and ERP components (Ramautar et al., 2004a,b). A deeper insight into the way in which stop-signal modality modulates inhibition mechanisms and N2/P3 components could further validate our prior interpretations of these components. As argued earlier, the effectiveness of stopping might depend to a large extent on sensory or ‘bottom up’ aspects of the modality of the stop signal. At the behavioral level, these factors were expected to contribute to faster stopping to auditory than visual stop signals, as reflected in SSRT and inhibition functions. These bottom up effects of stop-signal processing were further assumed to ‘propagate’ to longer-latency ERP components, such as N2/P3, inducing reduced latencies of these components to auditory compared to visual stop signals.

Regulation of behavior by means of inhibition of thought and action is considered to be driven by central mechanisms of executive control (Ridderinkhof et al., 2004). Our previous findings with visual stop (and go) signals supported the view that P3 elicited on successful inhibit trials reflected endogenous aspects of stop-signal processing, such as effective inhibition (Kok et al., 2004; Ramautar et al., 2004b). These processes were believed to be part of the executive control system, and thus to be independent of the sensory origin (i.e. modality) of the stop signal.

A second aim of our study was to further explore the functional significance of N2/P3 components that were elicited on trials when subjects failed to inhibit the go response. A speculation of our earlier studies was that these components could have partly reflected processing of errors of action (i.e. failed inhibitions) after or shortly before emission of the button press response. Detection and monitoring of errors is also implied by theories on supervisory or top-down control systems (Logan et al., 1984; Gehring and Taylor, 2004). Thus, if these components would indeed be related to error detection, we would expect little or no effects of stop-signal modality on the amplitude or scalp topography of N2/P3 components on unsuccessful inhibit trials, either in the stop-signal locked or response-locked waveforms.

2. Method

2.1. Participants

Fifteen healthy undergraduate students (eight females) from the University of Amsterdam participated in the experiment. They ranged in age between 18 and 24 years ($M = 21.2, \text{ S.D.} = 1.78$) and all reported normal or corrected-to-normal vision and hearing. Two subjects were left-handed and all of them received course credits.

2.2. Stimuli and apparatus

The primary-task stimuli (go stimuli) in the choice reaction task (choice RT) and in the stop task consisted of a blue circle or a blue square, subtending a 0.4° visual angle and were presented against a black background on a 14-in. monitor with a 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 the go stimulus that was displayed for 100 ms.

During the visual stop task, the stop signal consisted of a blue cross with a visual angle of 0.4° whereas in the auditory stop task, a tone of 1000 Hz, 80 dB amplitude was generated by the computer and binaurally administered by headphones. Stimulus duration of the visual as well as the auditory stop signals was also 100 ms. Stop signals were presented after onset of the go stimuli and randomly at one of five fixed delays (100–150–200–250–300 ms). Go and stop signals were also presented centrally on the screen. Trial duration of the choice RT and stop task varied between 3.5 and 4.5 s.
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.

2.3. Design and procedure

The experiment included one training and two experimental sessions. In the training session, subjects practiced the choice RT (which only contained go signals) and both stop tasks to achieve a stable response level. In the experimental sessions, the choice RT task (containing 100 trials) was presented first to calculate the individual speed level. Individual mean RTs from the choice RT task were then used as a reference for mean RTs to go trials in the stop task, and subjects received oral feedback (telling them whether or not their mean RTs were within acceptable ranges). Then, 12 visual and 12 auditory stop blocks of 120 trials each were alternately administered. In each block, 50% of the trials consisted of stop signals which were presented randomly and equally often at each of the five fixed delays. The other 50% of the trials are referred to as no stop-signal trials. In total, 720 trials were presented for each stop-signal modality (144 for each delay).

Assignment of response effectors (left or right index finger) to reaction stimuli (circle and square) was also counterbalanced across subjects. Subjects were instructed to respond as quickly as possible to the go signals by pressing response buttons while maintaining a stable level of accuracy. The primacy of responding to the go signals was emphasized and subjects were instructed 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. The oral feedback about task performance was always given at the end of each block. Halfway through the session a 15 min break and 1 min breaks between blocks were inserted.

2.4. Psychophysiological recording and data analysis

EEG recordings (Neuroscan) were taken from 64 tin electrodes in an extended system (Quikcap) referenced to the left mastoid (as is typical in this type of study). The electro-oculogram (EOG) was recorded from the 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 from 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; subsequently EOG artifacts were corrected using the algorithm described by Woestenburg et al. (1983).

For each subject, modality condition, and stop-signal delay, artifact-free go-stimulus-synchronized average waveforms were computed from corresponding no stop-signal trials and from successful and unsuccessful stop trials conditions, subtracting a 100-ms pre-stimulus period as baseline. Following procedures described elsewhere (De Jong et al., 1990; Kok et al., 2004), two kinds of no stop-signal ERPs were computed for each delay and each subject. This was done in the following way: correct RTs associated with the no stop-signal trials were rank-ordered, and then split into two parts. The fast and slow tails of the no stop-signal RT distribution corresponded with the proportion of UST and the proportion of SST of a particular stop-signal delay, and are further referred to as fast corresponding no stop-signal (corr NSfast) and slow corresponding no stop-signal (corr NSslow), respectively, (see also Fig. 1). Then, for each individual subject separate ERP averages were computed for corr NSfast and corr NSslow, which were subtracted from the stop-signal related ERPs in each stop-signal delay, that is SST – corr NSslow and UST – corr NSfast. This procedure was successfully applied in Ramautar et al. (2004b). In this study, it was demonstrated that speed of primary-task processing affected ERPs to the go signals. It is, therefore, reasonable to assume that ERPs to the stop signals, in particular ERPs elicited on UST and SST trials, would also be affected by effects of fast versus slow responding to the go signals. Thus, by subtracting the fast and slow no stop-signal ERPs from the ERPs elicited on UST and SST, biasing effects of go-signal processing are effectively removed from effects on ERPs that result from processing of the stop signal.

Finally, ERP waveforms synchronized to response onset were also calculated for the no stop-signal trials and UST. The ERP measures in these data were also extracted from difference waves that were calculated by subtracting for each stop-signal delay the corresponding response-locked no stop-signal trials (corr NSfast) from the UST.

The following statistical analyses were subsequently applied to performance and ERP measures. First, behavioral data were submitted to paired t-tests and repeated-measures analysis of variance (ANOVA) using modality (visual, auditory) and delay (100–150–200–250–300 ms) as independent factors.

Second, N2, P2, and P3 components elicited by the go signal on no stop-signal trials were analyzed to examine effects of fast versus slow responding (corr NSfast versus corr NSslow) and the stop-signal modality. Note that although these trials only contained go signals, it was still a theoretical possibility that the blocked presentation of visual and auditory stop-signal conditions would bring about a different preparatory set to the go stimuli on no stop-signal trials. In this analysis, peak amplitudes and latencies were taken from a time window of 250–350 ms (N2, maximum negative peak), 300–450 ms (P2, maximum positive peak), and 450–650 ms (P3, maximum positive peak) following
onset of the go signals. These measures were then submitted to repeated-measures ANOVA with modality (visual, auditory), response speed (corr NSfast, corr NSslow), and leads (Fz, Cz, Pz) as independent factors.

Third, N1, N2, and P3 peak amplitude and latency measures were calculated from the stop-signal related difference waves, separately for each stop-signal delay. These measures were taken from three different time windows following stop-signal onset (N1 and N2: maximum negative peaks within 50–150 and 200–400 ms, respectively, P3: maximum positive peak within: 200–600 ms) and were submitted to repeated-measures ANOVA with modality (visual, auditory), stoptype (UST, SST), leads (Fz, Cz, Pz), and delay (100–150–200–250–300 ms) as independent factors. Since N1 was only present in the auditory ERPs, the visual stop task – and thus the factor modality – was not considered in the analysis of N1. The inclusion of the N1 component was also of importance for the present study, because it is considered to be a typical exogenous component in audition (Nätäinen et al., 1987). Thus, in contrast with N2/P3 elicited by the stop signal, the amplitude of auditory N1 was not supposed to show any effects of processes underlying successful and unsuccessful stopping.

Fourth, an ANOVA was performed to compare N2/P3 measures derived from the response-locked and stop-signal locked difference waveforms (this only concerned UST – corr NSfast waveforms). Since the factor stop-signal delay was not of primary importance in this analysis, ERP s of no stop-signal trials, and UST (that were calculated for each separate delay) were pooled across the five stop-signal delays. Amplitudes of N2/P3 measures in response-locked averages were taken from a time window following 50–400 ms after response onset (N2: maximum negative peak, P3: maximum positive peak). This ANOVA consisted of the following factors: synchronicity (stop-locked averages, response-locked averages), modality (visual, auditory), and leads (Fz, Cz, Pz) as independent factors.

In all analyses, a level of $P < .05$ was accepted as statistically significant, and degrees of freedom were adjusted with the Greenhouse–Geisser correction where appropriate. Only significant main and interaction effects will be reported in Section 3.

2.5. Source localization

Source-localization analyses were carried out on ERPs derived from 62 channels to investigate the differences in spatial dynamics elicited by success of stopping and modality of the stop signal (BESA Version 4.2; Scherg and Berg, 1996). These analyses were applied to N2 and P3 measures derived from the stop-locked and response-locked difference waveforms in both modalities. To increase the precision of dipole source estimations, the analyses were performed on N2 and P3 measures that were derived from the entire set of trials of the stop task. This was again carried out by first aligning the difference waveforms with onset of the stop signal or response button for each separate delay, and then pooling the aligned ERPs across the five stop-signal delays. Modeling was performed on ERPs that were re-referenced to the average ERP across all channels, using a four-shell spherical head model. Next to an energy constraint, the residual variance or RV ($<10\%$) was included as criterion for a satisfactory solution. After testing various dipole configurations to estimate the best solutions for modeling, one symmetrical pair of dipoles seemed to provide the best solutions. Then, instantaneous dipole models were derived from the peak voltages of the ERP component derived from the difference waves of each individual subject. The solution parameters found in the grand average waveform were used as a starting value for the single subjects solutions (cf. Kenemans et al., 2002; Ramautar et al., 2004b). Dipole parameters ($x, y, z$-location) and ($x, y, z$-orientation) were estimated for each subject and experimental condition. For stop-signal locked difference waves, each parameter was then subjected to ANOVA with modality (visual, auditory) and stoptype (SST, UST) as independent factors. Finally, a separate ANOVA with factors synchronicity (stop-signal locked averages, response-locked averages) and modality (visual, auditory) was carried out to evaluate if there were any differences between UST P3 dipole configurations extracted from the stop-signal and response-locked waveforms.

3. Results

3.1. Behavioral data

Faster RTs were found on no stop-signal trials in the auditory than in the visual stop task (auditory: $M = 445$, S.D. = 36; visual: $M = 469$, S.D. = 44), ($t(14) = 6.23$, $P < .001$). Percentages of incorrect responses to the go signals were 3.8 % in the auditory stop task and 1.9 % in the visual stop task ($t(14) = 2.30$, $P < .037$). No omissions were observed on no stop-signal trials.

The following results were found in the ANOVA carried out on data from the stop-signal trials. The percentages of commission errors (UST) increased as a function of delay ($F(4, 56) = 338.32$, $P < .001$, GG = .39) and this increase was larger for visual than auditory stop signals (modality × delay: $F(4, 56) = 2.64$, $P < .034$, GG = .55, see Fig. 2A). RTs on UST increased as the stop signal was presented later, as indicated by a main effect of delay ($F(4, 56) = 63.59$, $P < .001$, GG = .48, see Fig. 2B). This increase was also slightly larger for visual than auditory stop signals.
A separate analysis verified that in agreement with previous stop studies, RTs on UST were faster as compared to RTs on no stop-signal trials, in either modality of the stop task (visual: \( F(1, 14) = 192.02, P < .001 \); auditory: \( F(1, 14) = 33.56, P < .001 \)).

The speed of processing of the stop signal (SSRT) in either modality is displayed in Fig. 2C. SSRTs to auditory stop signals were faster than SSRTs to visual stop signals, as indicated by a main effect of modality (\( F(1, 14) = 55.53, P < .001 \)). Finally, SSRTs also became faster as the stop signal was presented later, as indicated by a main effect of delay (\( F(4, 56) = 32.58, P < .001 \)).

### 3.2. Event-related analyses; go- and stop-signal locked waveforms

#### 3.2.1. No stop-signal trials

ERPs to the go signals are shown in Fig. 3. These ERPs were synchronized with the go stimuli and are depicted for the fast and slow no stop-signal trials: corr NSfast and corr NSslow. The numbers of corr NSfast and corr NSslow trials corresponded with the average proportions of UST and SST of all stop-signal trials obtained in the visual and auditory versions of the stop task (visual: 37.64% UST and 62.36% SST; auditory: 36.11% UST and 63.89% SST).

No stop-signal ERPs show an early negativity at around 280 ms (labeled as go N2 to avoid confusion with stop-signal related components) that is followed by two late positive deflections at around 350 ms and 550 ms (labeled as go P2 and go P3, respectively).

#### 3.2.2. Go N2

Go N2 was largest at the frontal and central leads (leads: \( F(2, 28) = 37.66, P < .001, GG = .71 \)) and also slightly larger in the visual than auditory stop task (modality: \( F(1, 14) = 9.23, P < .001 \)). Smaller go N2 amplitudes were further elicited at corr NSfast trials than at corr NSslow trials (response speed: \( F(1, 14) = 22.74, P < .001 \)), and this effect was somewhat larger at the fronto-central electrode locations (response speed \times \) leads: \( F(2, 28) = 6.64, P < .001, GG = .68 \). The fronto-central effect of response speed was also somewhat larger in the auditory than visual stop task (modality \times \) response speed \times \) leads: \( F(2, 28) = 5.28, P < .021 \).

**Fig. 3.** Grand average ERPs synchronized with go signals on no stop-signal trials. corr NSfast and corr NSslow represent fast and slow tails from the no stop-signal RT distribution in the auditory and visual stop tasks. N2, P2, and P3 are indicated by (*, **, and ***), respectively.
3.2.3. Go P2

P2 to the go signals was more prominent at the parietal lead (leads: \(F(2, 28) = 24.82, P < .001\)) and larger on corr NSfast than on corr NSslow trials (response speed: \(F(1, 14) = 49.61, P < .001\)). The P2 enhancement on corr NSfast trials was manifested most clearly at the central and parietal scalp locations (response speed × leads: \(F(2, 28) = 4.14, P < .041\)). The latter effect was also slightly larger in the auditory than visual version of the stop task as indicated by a modality × response speed × leads interaction (\(F(2, 28) = 4.78, P < .028\)). Finally, go P2 peaked somewhat earlier on corr NSfast than on corr NSslow trials (response speed: \(F(1, 14) = 10.51, P < .001\)) and this latency difference was more pronounced at the frontal lead compared than at the central and parietal leads (\(F(2, 28) = 16.34, P < .001\)).

3.2.4. Go P3

Go P3 also reached its largest amplitude at the parietal lead (leads: \(F(2, 28) = 33.55, P < .001\)). It was also more pronounced on corr NSfast than corr NSslow trials (response speed: \(F(1, 14) = 34.54, P < .001\)), but in contrast to go P2 this amplitude effect was largest at the frontal and central leads (response speed × leads: \(F(2, 28) = 69.69, P < .001\)). Go P3 also peaked somewhat earlier on corr NSfast than on corr NSslow trials (response speed: \(F(1, 14) = 39.68, P < .001\)), and earlier at the frontal/central than the parietal leads (\(F(2, 28) = 18.98, P < .001\)). Finally, the response speed × leads interaction (\(F(2, 28) = 18.14, P < .001\)) indicated that latency effect of response speed was strongest at the central electrode location.

3.2.5. Stop-signal trials (difference waveforms)

Figs. 4 and 5 depict the difference waves associated with successful and unsuccessful stop trials in the auditory stop task (Fig. 4) and visual stop task (Fig. 5) per delay. Furthermore, Fig. 6 provides a compact overview of the SST and UST difference waves of the auditory and visual stop tasks that were pooled over the five stop-signal delays after alignment of ERPs with stop-signal onset. Fig. 7A shows the
average N2 and P3 peak amplitudes that correspond with ERP components displayed in Fig. 6. These measures will be further referred to as stop N2 and stop P3.

3.2.6. Auditory stop N1
Auditory stop signals elicited a large fronto-central N1 component that did not differ between UST and SST conditions, but did decrease in amplitude as a function of delay (delay: \(F(4, 56) = P < .001\)). This effect was more pronounced in the SST than UST condition as indicated by a stop type \(\times\) delay interaction (\(F(4, 56) = 13.71, P < .001\)).

3.2.7. Stop N2
Stop N2 was most prominent at the frontal and central leads (\(F(2, 28) = 11.18, P < .001\); see also Fig. 6 and the upper panel of Fig. 7). Furthermore, visual stop signals elicited larger N2 amplitudes than auditory stop signals (modality: \(F(1, 14) = 32.75, P < .001\)) and this effect was most conspicuous at frontal and central leads (modality \(\times\) leads: \(F(2, 28) = 17.94, P < .001\)). Notice that in the auditory SST condition, N2 is superimposed on the rising flank of a large P2 (see also Figs. 4 and 6) that probably caused the positive absolute polarity of N2 in this condition. A larger (more negative) N2 was also found in the UST compared to the SST condition (stop type: \(F(1, 14) = 77.61, P < .001\)) and this effect was enhanced at fronto-central leads as indicated by a stop type \(\times\) leads interaction (\(F(2, 28) = 12.27, P < .001\)). Finally, the relative enhancement of fronto-central N2 on UST relative to SST (i.e. larger negativity or smaller positivity) was also more pronounced in the auditory than in the visual stop task (modality \(\times\) stop type \(\times\) leads interaction: \(F(2, 28) = 4.21, P < .039\)).

Largest amplitudes of stop N2 were observed at the longer stop-signal delays (\(F(4, 56) = 14.24, P < .001\) and this amplitude increase effect was larger on UST than on SST (stop type \(\times\) delay: \(F(4, 56) = 4.19, P < .026\)). Finally, longer N2 latencies were observed (a) in the UST compared to the SST condition (stop type: \(F(1, 14) = 77.61, P < .001\)) and (b) at later than earlier delays (delay: \(F(4, 56) = 590.90, P < .001\)).

No significant effects were found of stop-signal modality on the latency of N2.

3.2.8. Stop P3
Stop P3 was most prominent at the fronto-central electrode sites (leads: \(F(2, 28) = 59.86, P < .001\)). Larger amplitudes of stop P3 were further found in the SST than in the UST condition (stop type: \(F(1, 14) = 8.63, P < .011\)). This effect of successful stopping was larger at the fronto-central than at the posterior electrode sites (stop type \(\times\) leads: \(F(2, 28) = 57.63, P < .001\)) and larger in the auditory than in the visual stop task (modality \(\times\) stop type: \(F(1, 14) = 6.35, P < .024\), GG = .65; see also the lower panel of Fig. 7).
Statistical analyses further confirmed the observation that there were large differences between P3 amplitude across the various delays ($F(4, 56) = 24.50, P < .001, GG = .63$). Inspection of Figs. 4 and 5 further suggests that stop P3 is more prominent at early stop-signal delays in the SST condition, and more prominent at late stop-signal delays in the UST condition. These observations were corroborated in a delay × stoptype interaction ($F(4, 56) = 19.87, P < .001, GG = .67$).

Stop P3 latency increased systematically with longer stop-signal delays ($F(4, 56) = 464.46, P < .001$). Moreover, stop P3 showed longer latencies (a) in the visual than auditory stop task ($F(1, 14) = 255.19, P < .001$), (b) in the UST than in the SST condition ($F(1, 14) = 136.43, P < .001$), and (c) at the parietal than at the more anterior leads ($F(2, 28) = 33.14, P < .001$). Finally, the modality × stoptype × leads ($F(2, 28) = 8.22, P < .005$) interaction indicated that the latency increase of P3 in the visual relative to the auditory stop task was manifested more strongly at the fronto-central sites in the SST condition (see also Fig. 5).

In sum, for both modalities of the stop-signal task differences in speed of processing of the primary-task became manifest in amplitude modulations of the N2 and the P2/P3 components to the go signal. Effects of stopping were reflected in modulations of amplitude and latency of the N2/P3 to stop signals. Stop N2 had a fronto-central scalp distribution and was more pronounced on (a) unsuccessful than successful stop trials and (b) visual than auditory stop signals. Stop P3 had a predominant fronto-central distribution, and the frontal-central positivity of this component was markedly enhanced on successful relative to unsuccessful stop trials in the auditory stop task. Stop P3 also showed a shorter latency to auditory than visual stop signals and this effect was manifested most clearly on successful stop trials. Finally, the N2/P3 complex shifted systematically in time with longer stop-signal delays, showed larger amplitudes at longer stop signal delays in the UST condition, and larger amplitudes at early delays in the SST condition.

3.3. Event-related analyses; comparison of stop-signal locked and response-locked waveforms

Fig. 8 depicts the difference waves of the UST that were time-locked with onset of the button-press response, pooled across stop-signal delays. Waveforms are characterized by the presence of an N2 and a P3 following at approximately 100 and 350 ms after the response, respectively. N2/P3 amplitude measures that were extracted from these waveforms are presented in Fig. 9 and statistics regarding these components are reported below. Notice that the statistical analysis concerned a comparison of stop-signal locked and response-locked N2/P3 components that were elicited in the UST condition.

3.3.1. UST N2

Larger N2 amplitudes were obtained in stop-signal locked waveforms than in response-locked waveforms (synchronicity: $F(1, 14) = 13.03, P < .003$; compare upper panels of Figs. 7 and 9).

3.3.2. UST P3

UST P3 amplitudes were larger in the response-locked averages than in the stop-signal locked averages (synchronicity: $F(1, 14) = 13.03, P < .003$; compare upper panels of Figs. 7 and 9).
Fig. 10. Upper panel: voltage maps of SST and UST P3 derived from the pooled stop-signal locked waveforms. The lines are separated by 5 μV. Light shaded areas indicate positive voltages; dark shaded dotted areas indicate negative voltages. Lower panel: grand average dipole pairs of the auditory stop task (black) vs. visual stop task (gray) displayed for the left and upper parts of the brain. Left panel depicts the SST P3. Right panel depicts the UST P3.
nicity: $F(1, 14) = 11.24, P < .005$). This amplitude enhancement of response-locked P3 was most conspicuous at the central and parietal leads (synchronicity × leads: $F(2, 28) = 13.54, P < .005$; compare lower panels of Figs. 7 and 9).

3.4. Source analyses

Voltage maps and source modeling were based on N2 and P3 peak amplitudes derived from stop-signal and response-locked difference waves that were pooled across the five delays (grand averages are displayed in Figs. 6 and 8, respectively). Since dipole modeling of the N2 component in both the SST and UST conditions yielded unsatisfactory fits (residual variance was much larger than 10%) it was decided to limit our report of source modeling to the P3 component. Fig. 10 depicts the voltage maps and associated dipole configurations of the auditory and visual P3 amplitudes in the SST and UST conditions, derived from the stop-signal locked averages. The results of the analysis of the UST P3 component derived from the response-locked averages, and a comparison of dipoles sources derived from both types of averages are shown in Fig. 11.

3.4.1. SST P3

The global impression from Fig. 9 is that SST P3 had a similar fronto-central scalp distribution in the auditory and 

Fig. 11. Upper panel: voltage maps of the auditory and visual UST P3 derived from the pooled response-signal locked waveforms. The lines are separated by 5 $\mu$V. Light shaded areas indicate positive voltages; dark shaded dotted areas indicate negative voltages. Lower panel: grand average dipole pairs of the response-locked waveforms (black) vs. stop-signal locked waveforms (gray) displayed for the left and upper parts of the brain. Left panel depicts the auditory stop task. Right panel depicts the visual stop task.
visual stop tasks. It further appeared that visual and auditory stop signals activated exactly the same cortical area when the response is successfully inhibited (dipoles of visual and auditory stop tasks shown in gray and black, respectively). In either modality, the symmetrical dipoles were located in the medial precentral part of the cortex (auditory: residual variance (RV) = 6.6\%: location $x = -18.1; y = -15.4; z = 57.6$, visual: RV = 4.9\%: location $x = -18.9; y = -16.9; z = 58.4$).

3.4.2. UST P3

The RV value of dipole sources of UST P3 was higher than for SST P3. This component seemed to be located more deeply (ventrally) in the medial part of the brain. The symmetrical dipoles were located slightly more anterior and laterally in the visual task than in the auditory task (auditory: RV = 10.5\%: location $x = -11.9; y = -22.5; z = 25.5$, visual: RV = 15.0\%: location $x = -24.7; y = 3.2$ and $z = 10.0$). ANOVAs performed on the location parameters only showed a main effect of stoptype with regard to the z-parameter ($F(1, 14) = 7.64, P < .015$) indicating that UST dipoles were located more ventrally in the brain than the SST dipoles. No further statistics with regard to the other location or orientation parameters approached significance.

Subsequent source modeling was performed on the UST P3 of the response-locked averages. The voltage maps revealed that this P3 had a parietal distribution in both modalities (see Fig. 11; dipoles are shown in black and gray for response-locked and stop-signal locked components, respectively). The symmetrical dipoles were again located at deep medial locations. In the auditory stop task, the locations of UST P3 dipole locations did not differ appreciably between stop-signal and response-locked averages (response-locked auditory: RV = 5.0\%: x-location = $-24.4$; y-location = $-30.7$; z-location = 22.9). In the visual stop-task, they were located slightly closer to the midline and more posterior in the response-locked than stop-signal locked averages (response-locked visual: RV = 4.9\%: $x$-location = $-8.7$; $y$-location = $-28.2$; $z$-location = 11.1). ANOVAs executed on the location parameters revealed that the $x$-parameter differed between response-locked and stop-locked averages (synchronicity: $F(1, 14) = 19.18, P < .001$) that is, UST P3 dipoles were located slightly more medially in the response-locked than in the stop-signal locked averages. For the $y$-parameter, an interaction was observed between synchronicity $\times$ modality ($F(1, 14) = 5.63, P = .032$). This indicated that P3 dipoles in the response-locked averages were located more posteriorly than in stop-signal locked averages in the visual stop task, while no differences between the dipole locations were found in the auditory stop task.

4. Discussion

The central objective of the present study was to examine how the modality of the stop signal affects speed and efficiency of stopping, using ERP components. We hypothesized that in the stop task efficiency of inhibitory interventions would depend both on sensory or ‘bottom-up’ and cognitive or ‘top-down’ aspects of the stop signal. We further assumed as a working hypothesis that stop-signal modality would primarily affect sensory and not cognitive (i.e. executive control) processes. Based on earlier findings from visual stop tasks we further postulated that the higher-order control processes would become primarily manifest in differences between successful and unsuccessful stopping in the amplitude, scalp topography, and dipole configurations of N2/P3 to the stop signal.

4.1. Performance in the stop task

Stop-signal modality affected primary-task performance: on no stop-signal trials subjects responded faster and less accurately to go signals in the auditory than in the visual version of the stop task. Since this effect occurred to visual go signals on trials that did not contain a stop signal, it could have reflected some form of response strategy (taking the shape of a speed-accuracy trade-off) caused by the blocked presentation of auditory and visual stop signals. In addition, shorter SSRTs were found in the auditory than visual version of the stop task. This could have reflected a purely facilitatory sensory effect of auditory relative to visual signals. Alternatively, subjects could have been driven by a stronger bias to produce fast responses to both go and stop signals in the auditory than visual stop task. It is also theoretically possible that in the auditory version of the stop task there was less competition between modality-specific resources associated with processing of go and stop signals than in the visual version of the stop task (cf. Wickens, 1980).

4.2. ERPs and primary-task processes

Go signals elicited larger and somewhat earlier P2 and P3 components on fast than slow trials. Possibly, the amplitude enhancement of the early P2 component reflected a stronger resolution of a CNV-like negativity (e.g. Wastell, 1980) prior to the go stimuli. A larger negativity of the CNV might have been caused by a stronger anticipation to the go signals on fast than slow RT trials. The go P3 resembles the classical P3(b) component. P3 amplitude has been observed to be enhanced on trials associated with fast relative to slow RT quantiles (Roth et al., 1978), or in conditions in which subjects trade speed for accuracy (e.g. Pfefferbaum et al., 1983), suggesting that the P3 effect might reflect a phasic rise in alertness during fast responses to go trials.

4.3. ERPs and stop-signal processes

As predicted, N2 and P3 were elicited at slightly shorter latencies after auditory than visual stop signals, although the effect on N2 latency failed to reach significance. In
findings, these observations indicate that processing of the stop signal (around 400 ms). Consistent with the SSRT auditory stop signals (around 300 ms) than by the visual (Kok, 1983, 1986). It was elicited at an earlier latency by the visual stop tasks (Kok et al., 2004; Ramautar et al., 2004b), and bears resemblance to the NoGo P3 (e.g. Eimer, 1993; Kok et al., 2004; Ramautar et al., 2004b), the auditory (De Jong et al., 1990; Dimoska et al., 2003) and visual (Kok, 1983, 1986) modalities (e.g. Halgren et al., 1998; Stevens et al., 2000). Consistent with this posterior distribution, the UST P3 may be more strongly linked with processing of the erroneous response than with processing of the stop signal. Error detection processes are sensitive to the valence of information conveyed by the current action, but are uncontaminated by elements of non-specific motor-preparation and -execution activity (which were subtracted out from the present difference waves). Thus, the UST P3 could be functionally equivalent to the error-related positivity (Pe). The Pe is assumed to be associated with adjustment of response settings after an error (e.g. Falkenstein et al., 2000) or awareness of the occurrence of the erroneous response (Nieuwenhuis et al., 2001). Note that the UST P3 occurs later and is more time-locked to the response than the UST N2, which is more time-locked to the stop signal. Thus, the UST N2 and UST P3 appear to resemble functionally the ERN/Ne and Pe, respectively, and probably represent two different phases of processing of unsuccessful inhibit responses.

4.3.3. SST P3

The SST P3 was similar to those observed previously in auditory (De Jong et al., 1990; Dimoska et al., 2003) and visual stop tasks (Kok et al., 2004; Ramautar et al., 2004b), and bears resemblance to the NoGo P3 (e.g. Eimer, 1993; Kok, 1983, 1986). It was elicited at an earlier latency by the auditory stop signals (around 300 ms) than by the visual stop signal (around 400 ms). Consistent with the SSRT findings, these observations indicate that processing of the stop signal was faster for auditory than for visual stop signals. Dipole source analysis suggested that SST P3 was generated in modality-unspecific areas of the brain with a precentral/premotor center of gravity. Note that premotor areas tend to be invoked by external control signals (such as stop signals), whereas more voluntarily generated modes of control are presumed to primarily engage the prefrontal areas (e.g. Eimer and Schlaghecken, 2003; Goldberg, 1985).

4.4. Summary and conclusions

Stop-signal modality affected reaction times to the primary-task and the reaction times to the stop signal. As predicted, shorter SSRTs were found for auditory than visual stop signals reflecting faster processing of the stop signals in the auditory modality.

Stop N2 had a fronto-central scalp distribution and was more pronounced in (a) unsuccessful than successful stop trials and (b) visual than auditory stop signals. UST N2 resembled to a certain degree the feedback ERN/Ne and probably reflected a greater negative valence elicited by the stop signals on unsuccessful than successful inhibit trials. Stop P3 had a clear fronto-central distribution on successful inhibit trials in stop-signal locked averages and had a posterior-parietal focus on unsuccessful inhibit trials in response-locked averages. Furthermore, the scalp topography and dipole configurations of stop P3 components did not seem to vary appreciably between visual and auditory stop.

4.3. Stop N2

Auditory stop signals produced smaller N2 components than visual stop signals. One possible explanation of the reduced N2 is that auditory stimuli typically evoke a prominent fronto-central exogenous P2 component around 200 ms (Näätänen and Picton, 1987). This component was also clearly present in the difference waves of auditory stop ERPs where it overlapped and possibly obscured N2. It is worth emphasizing, however, that the effect of unsuccessful relative to successful stopping on N2 amplitude was of almost equal magnitude in both modalities (Figs. 6 and 7, upper panel). Notice also that in contrast with auditory N2, the auditory N1 component did not differentiate between successful and unsuccessful stopping, which supports the notion that N1 was more strongly linked with exogenous/sensory aspects of the stop signal. Consistent with recent findings, no evidence was found of emergence of a typical NoGo N2, that is, an amplitude enhancement to successfully relative to unsuccessfully inhibited trials. This indicates that processing of stop signals in the stop task cannot be simply equated with processing of NoGo stimuli in the typical go/NoGo task. The enlarged stop N2 on unsuccessful relative to successful inhibit trials in stop tasks may reflect a greater significance of stop signals on trials in which subjects are unable to withhold the imminent response to the go signal, and thus commit an error. This interpretation suggests that the UST N2 might be functionally similar to the error-related negativity (ERN/Ne), an ERP component observed immediately following response errors or negative feedback signals that follow feedback stimuli indicating that an unfavorable outcome has occurred (for a review, see Ridderinkhof et al., 2004).

4.3.2. Stop P3

Similar to previous results (Ramautar et al., 2004b), the dipoles and associated generator fields of SST P3 were located more dorsally, and dipoles of UST P3 more ventrally in the brain. These dipole sources were not taken to represent distinct or specific cortical areas, but probably reflected centers of gravity or sheets of generators in a distributed field. These networks differentially mediated response inhibition, the functional characteristics of which will be discussed in more detail below.

4.3.3. SST P3

The SST P3 was similar to those observed previously in auditory (De Jong et al., 1990; Dimoska et al., 2003) and visual stop tasks (Kok et al., 2004; Ramautar et al., 2004b), and bears resemblance to the NoGo P3 (e.g. Eimer, 1993; Kok, 1983, 1986). It was elicited at an earlier latency by the auditory stop signals (around 300 ms) than by the visual stop signal (around 400 ms). Consistent with the SSRT findings, these observations indicate that processing of the stop signal was faster for auditory than for visual stop signals. Dipole source analysis suggested that SST P3 was generated in modality-unspecific areas of the brain with a precentral/premotor center of gravity. Note that premotor areas tend to be invoked by external control signals (such as stop signals), whereas more voluntarily generated modes of control are presumed to primarily engage the prefrontal areas (e.g. Eimer and Schlaghecken, 2003; Goldberg, 1985).

4.3.4. UST P3

The UST P3 was more posteriorly distributed in response-locked than in stop-signal locked averages. The location of the dipoles of the response-locked UST P3 was reminiscent of the neural generators of the classical oddball P3, without appreciable differences between the visual and auditory modalities (e.g. Halgren et al., 1998; Stevens et al., 2000). Consistent with this posterior distribution, the UST P3 may be more strongly linked with processing of the erroneous response than with processing of the stop signal. Error detection processes are sensitive to the valence of information conveyed by the current action, but are uncontaminated by elements of non-specific motor-preparation and -execution activity (which were subtracted out from the present difference waves). Thus, the UST P3 could be functionally equivalent to the error-related positivity (Pe). The Pe is assumed to be associated with adjustment of response settings after an error (e.g. Falkenstein et al., 2000) or awareness of the occurrence of the erroneous response (Nieuwenhuis et al., 2001). Note that the UST P3 occurs later and is more time-locked to the response than the UST N2, which is more time-locked to the stop signal. Thus, the UST N2 and UST P3 appear to resemble functionally the ERN/Ne and Pe, respectively, and probably represent two different phases of processing of unsuccessful inhibit responses.

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signals, suggesting that these components were generated in modality-unspecific areas of the brain.

References


