Visual-Tactile Stop Signal Inhibition

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Contents

Zusammenfassung

Die vorliegende Untersuchung behandelt die absichtliche Hemmung von bereits programmierten Bewegungen infolge der Darbietung eines visuellen oder taktilen Reizes. Die Versuchspersonen waren aufgefordert, sakkadische oder manuelle Antworte auf visuelle oder taktile Zielreize auszuführen. Diese Antwort sollte jedoch annulliert werden in 25% der Fällen, da in diesen ein zusätzlicher "stop" Reiz aus der jeweils anderen Modalität dargeboten wurde. Reaktionzeiten wurden gemessen in allen drei Experimenten, dazu wurden sakkadische Amplituden in zwei Experimenten und EEG-Antworten in einem Experiment erfasst. Die Ergebnisse wurden verglichen mit Hervorsagen beruhend auf dem Wettlaufmodell von Logan & Cowan (1984).

Taktile Stimuli waren wirksam sowohl als imperative "go" Reize als auch als "stop" Reize. Das Gelingen der Versuchspersonen dem "stop" Signal zu folgen und die Bewegung zu unterdrücken war abhängig von dem Zeitpunkt der Darbietung. Etwa die Hälfte der Versuchspersonen griffen unbewusst zu Responsstrategien, bei denen sie ihre Antworten verzögerten, um besser inhibieren zu können. In jedem Experiment waren bei einigen Versuchspersonen kleine Verletzungen der Modellvorhersagen in den Reaktionszeiten vorhanden. Die sakkadischen Amplituden bei nicht gelungene Hemmungen waren eindeutig hypometrisch im Vergleich zu den "go" Antworten, ein Effekt der mit länger dauernden gleichzeitigen "go" und "stop" Verarbeitungen immer stärker wurde. Dieser Einfluss des "stop" Signals auf die Bewegungen wurde als Widerspruch gegenüber der Annahme des Wettlaufmodells von Kontext-Unabhängigkeit gesehen. Keine schlüssigen Belege für einen Effekt der horizontalen räumlichen Position des "stop" Signals wurden gefunden.

Die no-go typische Verstärkungen in den EEG-Daten wurden auch für "stop" Signale bestätigt. Gelungene und gescheiterte Unterdrückungen zeigten stark unterschiedliche Latenzen und Amplituden. Diese blieben jedoch nahezu konstant zwischen den unterschiedlichen zeitlichen Bedingungen des "stop" Signals. Der "stop" Effekt auf die EEG-Ergebnisse wurde als eine Folge einer bereits getroffenen Entscheidung, anstatt als Erscheinungsform dieser, gesehen.

Summary

This study investigated voluntary inhibition of programmed movements upon infrequent presentation of visual or tactile stop signals. Participants were to perform fast saccadic or manual responses to a visual or tactile target, but to cancel the response on the 25% of trials in which a stop signal of the other sensory modality was additionally presented. Reaction times were measured in each of the three experiments reported. Additionally, in two of these amplitudes of saccadic movements were collected, and event-related potentials on inhibition of manual responses were recorded in the third. The data was compared with predictions made by the race model proposed by Logan & Cowan (1984).

Tactile stimuli were successfully utilized both as go and as stop signals. Participants' inhibition success was related to the delay of stop signal presentation. About half of the participants resorted to strategic responding in a subconscious attempt to increase stopping success. In each experiment, the reaction times of some participants showed small violations of the race model assumptions. The saccadic amplitude data from the failed inhibition trials exhibited strongly hypometric eye movements relative to responses on go trials, and the size of this effect increased with the duration of concurrent processing of the go and stop signals. This influence of the stop processing on the go response was interpreted as a contradiction to the context-independent processing supposed by the race model. No conclusive evidence was obtained on the effects of varying the spatial position of the stop signal in the horizontal plane.

Stop signal equivalents of the no-go enhancements were found in the event-related potentials. Their latencies and amplitudes differed greatly between successful and failed inhibition trials, but not across conditions with different stop signal timing. The effects were assumed to be a reflection of a prior decision to inhibit, rather than the manifestation of this decision.

1

Introduction

In its relatively short history as a science, psychology has rather successfully explored the human mind and attempted to explain many of its operations and characteristics. Although many, probably most, of the pieces of the puzzle are still missing, neuropsychology and the different cognitive sciences have been able to mimic and to predict many different behaviours, skills and events, as these are manifested in healthy adults, during development, and in patients. Detailed and experimentally testable cognitive models exist for functions such as sensory processing, selective amnesia after injury, and language development. In the past decades new brain imaging and analysis techniques have added to these developments by providing both new data and a better understanding of cognitive processing.

Bottom-up vs Top-down Influences

Existing theories on specific modules of cognitive processing generally label input to the system as either bottom-up or top-down influences. Bottom-up induced processes are fast, fairly simple and reflexive operations, such as turning your head towards the source of a loud, unexpected sound. Also

called stimulus driven processes, their effects and importance are directly related to the attributes of the stimulus, such as its intensity or suddenness. Stimulus characteristics are relatively easy to manipulate in laboratory settings and hence a great deal is known about, for example, how the visual system responds to colours, to movement, and under different lighting conditions.

In contrast, although almost all cognitive models contain some reference to modifying top-down influences, these are largely undefined and only described in vague terms such as "higher" or "executive" functions. Thus, each cognitive task is largely treated as an isolated process, whereas theories of an overall controlling and coordinating agent are scarce. Executive control risks being reduced to a circular argument of "a 'little man inside the head,' who perceives the world through the senses, thinks, and plans and executes voluntary actions" (Crick & Koch, 2000, p. 107), rather than doing "the main job of psychology [which] is to explain how intentionality can arise out of nonintentional stuff" (Logan, 2003, p. 45).

Voluntary Inhibition of Action

In the absence of agreement on what is meant by the term executive functions — or where the line to non-executive cognitive functions is drawn (see Rabbitt, 1997) — several descriptions have been suggested (e.g., Logan, 1985; Baddeley, 1996; Burgess, 1997). Among the frequently mentioned functions in these descriptions are such abilities as constructing strategies, monitoring behaviour, and changing or switching strategies if needed. The ability to stop an ongoing behaviour or prevent an inappropriate response is noted by many authors, and implicitly assumed by others (as part of the ability to change between tasks). Voluntary inhibition is a small but crucial aspect of executive control, and has increasingly become the subject of interest and research (see, e.g., Dagenbach & Carr, 1994). Problems in inhibition performance have been linked with schizophrenia (e.g., Badcock et al., 2002), attention-deficit/hyperactivity disorder (e.g., Oosterlaan et al., 1998; Nigg,

1999), as well as other clinical conditions (e.g., Amieva et al., 1998), which has further raised awareness of the relevance of inhibition.

The Stop Signal Task

A frequently used experimental paradigm for investigating voluntary inhibition is the stop signal or countermanding task (e.g., Vince, 1948; Lappin & Eriksen, 1966; Logan, 1994). In this reaction time (RT) task, two general classes of stimuli are presented to the participants: imperative go signals requiring a response, and stop signals calling for any response to be cancelled. Each trial contains a go signal, often presented after a variable delay relative to the onset of the trial to avoid guessing. Only in a minority of the trials — the stop trials — a stop signal is additionally presented at one of several delay relative to the go signal. The task of the participant is to respond as fast as possible on the go trials, but to try to inhibit the response on the stop trials. The length of the delays at which the stop signals are presented, relative to the go signal, are set so that each stop signal delay (SSD) produces a certain level of probability (between 0 and 1) of successful inhibition. By comparing the RT means and distributions of the responses obtained in the go condition and in the different delay conditions, along with the probabilites of a response in the latter, insights into the inhibition process can be gained.

In comparison with the related go/no-go paradigm (Donders' (1868/1969) B task), which is frequently used to investigate voluntary inhibition (as well as other processes such as lexical decision [e.g., Perea et al., 2002]), the stop signal task design requires considerably more testing but also provides more detailed information. In each trial of a go/no-go experiment, only one stimulus is presented, either a go or a no-go (stop) signal, often with equal chance of occurance. In contrast, the lower probability of a stop trial in the countermanding task (usually 25–33%), and the presentation of the go signal on all trials, mean that the participant is more likely to prepare a response on each trial. Thus, the stop signal paradigm is more likely to reliably produce voluntary inhibition of ongoing response preparation. Further, as participants

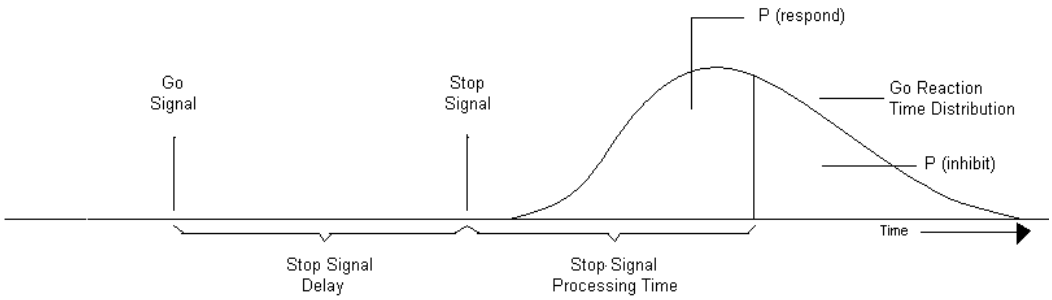
frequently make errors of commission, i.e. respond despite presentation of the stop signal, comparing physiological responses on these trials with those on successful inhibitions or go trials can deliver important information. The final advantage of the stop task is the usage of different SSDs and the further comparisons enabled by the resulting individual sets of data with different levels of inhibition success for each delay condition.

The Logan-Cowan Race Model

Research using the countermanding paradigm in various settings has yielded a model framework describing the processes involved in voluntary inhibition, as well as some general conclusions about these processes (Logan, 1994; Logan & Cowan, 1984). For example, stopping is regarded as a modality-independent operation (Logan, 1994) involving the frontal lobe (e.g., Rubia et al., 2001), and as requiring a surprisingly invariable amount of time to act (Naito & Matsumura, 1994). Logan and Cowan modelled the stop trial events as a parallel race between the go process, starting at presentation of the go signal, and the stop process, which begins at the appearance of the stop signal. The outcome on each stop trial, successful inhibition or a stop failure response, is determined solely by which process is the first to reach a (not necessarily common) threshold and hence win the race. A key assumption is that both go and stop processes have finishing times which are independent random variables, so that for a given stop signal delay the winner of the race can only be predicted with a certain probability. The independence of the processing times is thought to reflect context independent processing, i.e. that neither process is affected by the presence of the other one.

The Logan-Cowan race model makes quite detailed predictions about the reaction times obtained when participants fail to inhibit despite the presentation of a stop signal. These are illustrated by the schematic representation of a hypothetical stop trial shown in Figure 1.1. The RT distribution denotes the response times on the go trials in a stop signal experiment. These are

Figure 1.1: Schematic representation of the assumptions and predictions made by the race model of how the probabilities of response and inhibition depend on the go RT distribution, stop signal processing time, and stop signal delay in the trial. Adapted from Logan & Cowan (1984).



plotted relative to the timing of the presentation of the go signal. As the go processing is assumed to be independent of a possible stop processing, the distribution should not be affected by the stop signal presented after the stop signal delay shown. On a given stop trial, the stop process takes some random time to finish, represented as stop signal processing time (SSPT) in the sketch. The finishing point of the stop process segments the go RT distribution into two parts: the faster responses in the faster tail which would already have been executed at this point, and the slower responses which would be inhibited by this stop signal. Changing the delay between go and stop signals affects the finishing time of the stop process, and thus also the relative proportions of successful and failed inhibitions. As these proportions, along with the go RT distribution, are known from the experimental data and the SSD is determined by the experimenter, the SSPT can be estimated. Note

that the SSPT is drawn as a constant in the sketch, although it is assumed to be a random variable. However, the consensus in the stop signal literature seems to be that treating it as a constant introduces only marginal errors, based on mathematical analyses (Logan & Cowan, 1984, Logan et al., 1984) and simulations (De Jong et al., 1990; Band, 1997), at least for estimations of the mean SSPT across delays (Colonius et al., 2001). The estimated SSPT is a measure of inhibition performance which can be used for comparisons of stimulus arrangements, as well as across groups, individual participants and patients.

Testing the Race Model

The validity of the assumptions made by the Logan-Cowan model can be tested by inspecting the means and distributions of the responses recorded on the stop failure trials. As those are assumed to be the proportion of the go distribution lying to the left of the finishing time of the SSPT, the mean RT should always be smaller than, or equal to, the go mean RT. Similarly, the distribution should be equivalent to the cut off go distribution. Hence, the go RT distribution forms a lower bound for the stop failure RTs. Formally,

$$P(T \leq t| \text{ stop signal at } t_d \text{ ms after the go signal}) \geq P(T \leq t \mid \text{no stop signal})$$

for all $t, t \geq 0$, T being the observed RT and t_d the delay used. Likewise, an upper bound for each delay condition is constituted by this go RT distribution divided by the probability of stop failure at that particular delay (see Colonius, 1990). Plotting the cumulative distribution functions of the stop failure RTs and the upper and lower bounds constitutes a simple test for violations of the race model: The stop failure distribution has to be contained by the upper and lower bounds across its total range to support the model.

The predictions made by the race model have usually been validated by experimental data. However, in many studies participants' performance on

the go trials has not been tested separately as well as in the context of the stop trials. This may allow for unnoted strategic trading of response speed for inhibition success, interfering with the assumed context-independent processing. When such procrastination strategies have been controlled for, the resulting data has contained violations of model predictions for some participants (Colonius et al., 2001; Özyurt et al., 2003), although these violations have also been reported when no testing for strategic responding was done (Logan & Cowan, 1984; Hanes & Carpenter, 1999). Specifically, the RT distributions obtained on the stop failure trials in conditions with very short delays tend to have elongated slow tails, with 5–15% of responses violating the lower bound of the distribution as described above. Unexpectedly slow responses seem to escape from inhibition and be executed when the delay between go and stop signals is short. Alternatively, these results could be interpreted as a slowing down of some of the responses through a possible interaction between the go and stop processes. To allow for investigation of these hypothetical interaction effects, the experiments in this study each included a training procedure to determine the initial go response speed of the participants and to enable detection of possible procrastination strategies.

Stimulus and Response Modalities in Inhibition

Early stop signal experiments (for a review see Logan & Cowan [1984]) mostly involved manual responses to visual go signals, and visual or auditory stop signals. More complicated tasks such as speaking, typing, or mental arithmetic have also been used, producing longer SSPTs (up to 400 ms) than inhibition of simpler tasks such as key presses (around 200 ms or less). In recent years, attention has increasingly turned to inhibition of saccadic eye movements (e.g., Hanes & Carpenter, 1999; Logan & Irwin, 2000; Paré & Hanes, 2003). Voluntary saccades are fast movements of the eyes which bring an area or object of interest from peripheral into foveal vision in order to extract more information from it. As both the neural pathways involved in producing saccades and the likely roles of the structures in these pathways

are quite well established (e.g., Schall, 1995; Findlay & Walker, 1999), they are well suited for research into inhibition of movements.

Several saccadic stop signal studies using visual go and stop signals have produced notably fast inhibition processing, with estimated SSPT as small as 100 ms. Schall and Thompson (1999) suggested that this was due to the stop signal being presented centrally on the fovea. A foveally presented stimulus could contribute to saccadic inhibition in at least two ways: the intended, task-relevant form of inhibition, and a bottom-up interference with saccadic programming which could delay or even inhibit the motor command. Asrress & Carpenter (2001) did not find a difference between the effectiveness of central and peripheral stop signals, but found that combining the two stimuli was more effective than either stop stimulus alone. They suggested, as had Schall and Thompson (1999) and Hanes and Carpenter (1999), that experiments using go and stop signals from different sensory modalities should be conducted to clarify which effects are purely inhibition related and which due to the particular stimulus and response modalities used.

Cabel et al. (2000) employed three different stop signals, a foveally presented visual stimulus, an auditory stimulus, and a signal comprising both stimuli. They found slower saccadic SSPTs for auditory than for visual stop stimuli, with the combined stop signal being the most effective. They concluded that the visual stop signal instigated several inhibitory processes (based on foveal, visual stimulus onset, and nonfoveal instructional information, respectively), whereas the power of the auditory signal relied on the instructional information only, and the combined signal allowed for a statistical facilitation of inhibition processing. They also hypothesized that the location of presentation of the stop signal might have an effect on its efficacy, as the auditory stimuli were delivered via a speaker situated 2 m above the head of the participant, in stark contrast to the foveal visual stimuli.

Colonius and coworkers investigated whether varying the spatial position of the auditory stop signal had an effect on inhibition performance when participants made saccades to visual go signals to the left and the right. Auditory

stop signals which were randomly presented in front, to the right, or to the left of the participant led to very short SSPTs, but to no effects of spatial position of the stop signal (Colonius et al., 2001). When the stop signal location was blocked so that, on a given block of trials, the participant knew on which side it would be presented, ipsilateral stop signals were less effective than contralateral ones, although there was no general shift in attention towards the stop signal side (Özyurt et al., 2003). The authors suggested that the auditory stop signal may, regardless of its task-specific inhibitory effect, also have had a bottom-up facilitatory role on responding, similar to accessory stimuli in focussed attention tasks (e.g., Frens et al., 1995).

In the experiments reported in this study we introduced the usage of tactile stimuli with the stop signal paradigm. In the first experiment, visual go signals and tactile stop signals were used, whereas in the second and third experiments the arrangement was the opposite.

Psychophysiological Correlates of Inhibition

The great majority of electrophysiological and imaging experiments investigating voluntary inhibition have used the go/no-go task discussed above. Different imaging techniques and single cell studies have identified several frontal sites showing selective activation for inhibition. For example, the right prefrontal cortex has been indicated as an important actor in networks of inhibition control (e.g., Gemba & Sasaki, 1990; Kawashima et al., 1996; Durston et al., 2002). Electroencephalograpic (EEG) studies measuring the electrical activity of the brain using electrodes placed on the scalp have identified brain potentials specific to inhibition: The no-go potential starts as a distinct negative deflection at frontal sites around 150 ms after presentation of the no-go stimulus, peaks around 200–250 ms, and then turns into a more central positive deflection with a peak at 300–350 ms (e.g., Falkenstein et al., 1999; Jackson et al., 1999; Filipović et al., 2000). Thus, the response-related N2 and P3 deflections are enhanced when responses have to be inhibited.

Only a handful of studies have used EEG to study stop signal inhibition. Early EEG countermanding experiments were designed to localize the "point of no return" of manual responses, the last processing stage where the movement can be cancelled (De Jong et al., 1990, 1995). These authors did not consider the averaged EEG data directly, but used it to derive the lateralized readiness potentials (LRPs) on successful and failed inhibition trials. The LRP is a measure of central motor activation related to manual responses, obtained by subtracting across left- and right-hand responses to remove hemispheric differences. De Jong and colleagues defined a certain level of the LRP as the threshold for response initiation, and interpreted the patterns of LRP on the different outcomes of the stop trials as evidence for two different inhibitory mechanisms: a central selective one and a peripheral global one. Van Boxtel and colleagues reinterpreted these effects (Band & Van Boxtel, 1999) and showed that they are compatible with a single, centrally located inhibition mechanism (Van Boxtel et al., 2001). Further, they found enhanced N2 components in the event-related potentials (ERPs) both on no-go and on stop trials, regardless of whether the latter involved successful inhibition or stop failure.

Only one study so far has exploited the possibility rendered by the stop signal task to compare data from trials in which the stop signal was presented early with those where it was presented late. Naito & Matsumura (1994) pointed to the frequently found no-go potential described above, and to that this is sometimes found also on go trials (e.g., Jodo & Kayama, 1992; Van Boxtel et al., 2001). They used a pseudo-stop signal task in which a first signal required participants to respond about 500 ms after its presentation, and a second stimulus, presented with different delays (ranging from 100 to 400 ms), indicated whether the intended response should be executed. Following the notion of an effectively constant duration of the stop process inherent in the Logan-Cowan race model (see above), these authors compared the no-go N2 and P3 characteristics on go and no-go trials across the delay conditions. The results showed that the no-go specific deflections were present in the no-go trials on all delay conditions, relative to the go trials. Interestingly, the onset

and peak of the N2 wave were constant, relative to the stop signal, across delay conditions. In contrast, the peak-to-peak time between the N2 and the P3 was reduced with increasing delay between the go and stop stimuli.

The task design in the experiment by Naito and Matsumura differed from that generally used in stop signal experiments in several ways, but most acutely in the non-speeded responses required. Hence, one of the aims of this study was to attempt to replicate their ERP results in a setting in which performance measures were testable against the race model which inspired them.

The aims of this study

The experiments reported here were conducted with respect to the following aims. Firstly, to test the modality-independence of stop signal inhibition by introducing the usage of tactile stimuli, both as go and as stop signals. Secondly, the data was tested against predictions of the Logan-Cowan race model of inhibition, and particular attention was given to detecting possible procrastination strategies by the participants which would in itself contradict context-independent processing. Thirdly, in one experiment the stop signals were presented either ipsi- or contralaterally to the go signal in order to study whether relative spatial position of the signals has an effect on inhibition. Finally, in one experiment EEG data was recorded in addition to the performance measures. We expected to find no-go specific ERP characteristics on those trials on which inhibition was attempted, and that these would differ between successful and failed inhibition but not across delay conditions.

2

Saccadic Inhibition with a Tactile Stop Signal

This study extends previous visual-auditory tests of the modality-independence of the stopping process by introducing tactile stop signals. If the modality of the stop signal does not influence its effectiveness, the reaction time data obtained should be in line with results from experiments using visual or auditory stop signals. In addition to this test of the modality-independence of stop signal processing, participants' performance on the go task was tested separately to disclose and avoid any strategic speed-accuracy tradeoff. Finally, the data obtained was tested against predictions from the Logan-Cowan race model. The experiment described in this chapter has, at the time of writing this study, been submitted for publication in *Experimental Brain Research*.

2.1 Method

2.1.1 Design

In this experiment the participants were presented with visual go signals and, in 25% of trials, additionally with tactile stop signals. In the go trials, the initial central fixation point was followed, after a variable interstimulus interval (gap), only by a go signal either on the left or on the right. The task of the participant was to perform a rapid saccade towards the go stimulus. In the stop trials, the go stimulus was followed by a vibratory stop signal presented to the hands at one of three different stop signal delays (SSDs). In these trials, the participant was required to inhibit any eye movement.

The participants were first trained on blocks containing only go trials until their performance on this task stabilised around some mean RT value. Then stop trials with three randomly chosen SSDs were interspersed with the go trials, and the SSDs were adjusted to produce three distinct probabilities of successful inhibition (approximately 20, 50, and 80%). During this adjustment procedure and the subsequent stop signal experiment participants were urged to focus on performing well on the go trials, in an attempt to match the mean RT value obtained in the preceding go training. Once these criteria had been reached, or the participant ceased improving his or her RT performance, each participant completed a minimum of 3000 trials in the stop signal experiment over a number of sessions.

2.1.2 Participants

Three participants were tested, two males (HB, age 33 years; DS, 20) and one female (PB, 20). All had normal or corrected-to-normal vision and right eye dominance. The experiment was carried out with the informed consent of the participants, who were compensated for their participation partly with compulsory subject hours and partly in money paid out after the last session.

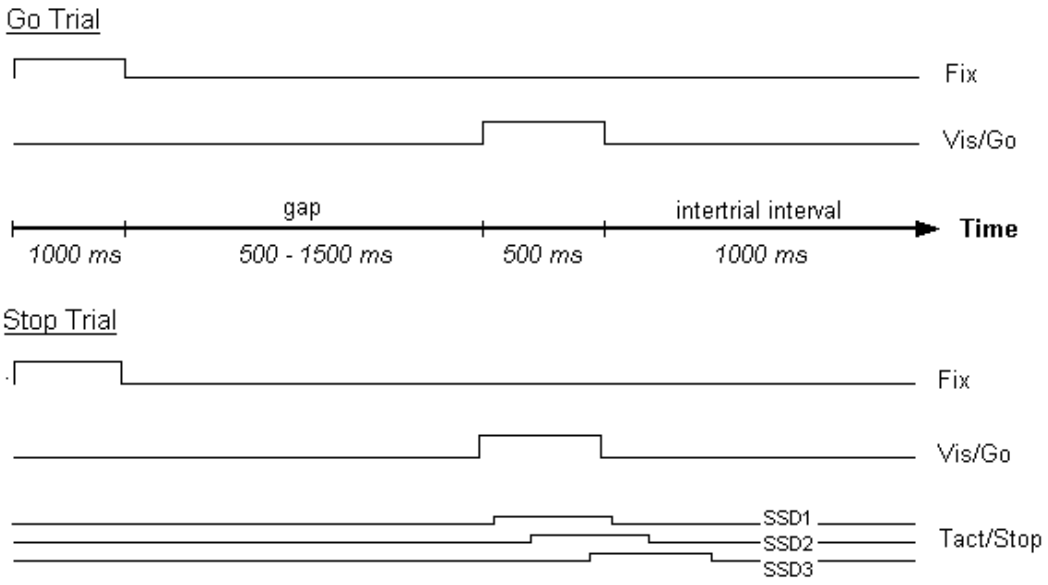
2.1.3 Apparatus and Stimuli

The participant was seated in a darkened, sound attenuated room with the head positioned on a chin rest and the elbows and lower arms resting comfortably on a table. The fixation point and the visual stimuli were red light emitting diodes (LEDs) (5 mm, 8 cd/m^2) situated on the table 60 cm in front of the participant, the fixation point in the centre and the go LEDs 10° to the left and right of it, respectively. The fixation point was presented for 1000 ms and the duration of the visual go stimuli was 500 ms. The tactile stimulation, which had a frequency of 50 Hz and a duration of 500 ms, was generated by two vibration exciters (Brüel & Kjær, Nærum, Denmark) placed on bases situated under the table. Positioned in each shaker was a metal rod extending through a hole in the table approximately 2 cm above the surface. On each rod was a wooden ball of 14 mm diameter, which rested in the palm of the participant and transmitted the vibration to the hand. Eye movements were recorded at a rate of 250 Hz using the infrared light video system EyeLink (Sensomotoric Instruments, Berlin, Germany). One PC controlled the stimulus presentation, and two other interlinked PCs controlled the EyeLink program.

2.1.4 Procedure

The experimental procedure was similar for the go trial training and the stop signal adjustment procedure and experiment. One block consisted of 100 (in go training) or 120 trials (in the stop adjustment and experiment; 90 of which were go, 30 stop). Each trial started with the presentation of the central fixation point. After this, there was a variable interval during which no stimuli were presented. Then the go stimulus, one of the go LEDs (left or right) came on. In the go trials, the go stimulus was followed by an interval of 1000 ms after which the next trial begun. However, in the stop trials the go stimulus was followed by the stop stimulus, presented to both hands after the appropriate SSD relative to the onset of the go signal. Only after the

Figure 2.1: Schematic representation of the stimulus configuration of go training (above only) and of the stop signal experiment (above and below). Note that on each stop trial (below) the stop signal was presented at one of three different delays relative to the go signal.



presentation of the stop signal did the 1000 ms intertrial interval start. The go and stop trials are shown schematically in Figure 2.1.

Participants were instructed to keep their gaze on the fixation point (or, in the intervals, the location where the fixation point had previously been), and to move their eyes to the go target as quickly as possible when it appeared. However, they were to inhibit this movement and continue fixating in the event of a stop signal occurring. They were informed that it would not be possible to do this on all stop trials, but that they should nonetheless try to be as fast as possible in their responses and not slow down in expectance of a possible stop signal.

In go training, trials were sampled in random order from a population of

20 trials, each with a different, randomly chosen length of the gap between the fixation point and the go signal (range: 500–1500 ms). In the stop experiment, all 120 trials were prepared before the onset of the experiment and only the order of presentation of trials was randomized between blocks. For these trials, five different gaps with the same range as in the go training trials were used, each with equal probability. Additionally, in the stop trials the three equiprobable SSDs for each participant were set based on participant performance during training of the stop task. The SSDs used ranged from 30 to 170 ms.

Thus, each of the 30 stop trials had a unique combination of gap length, SSD length (3 levels: individually set), and direction of required response (2 levels: left, right). The 90 go trials that were control trials in the stop experiment were made by reproducing the combinations of gap length and direction of required response so that all levels of these factors were equally probable. Hence, each unique stop trial was matched with three similar go trials, so that for all questions of interest the probability of a stop trial was 25%.

2.2 Results

For each participant, the 300 trials of go training following stabilisation of performance on this task were recorded. A total of 2872 (DS), 3901 (HB), and 4243 (PB) trials from the stop experiment were accepted for further analysis. For both the go and the stop sets the data was collapsed across direction of eye movement response. The dependent measures were saccadic latency and amplitude for both the go condition and the three stop conditions, as well as probability of response for the latter. Trials containing blinks, improper fixations, responses in the wrong direction, saccades with amplitudes larger than 30°, guesses (RT latency shorter than 70 ms), or responses which were too slow (RT latency longer than 500 ms) were excluded from further processing. Movements smaller than 2° were considered as errors in go trials and as inhibitions in stop trials. Whereas go trials were either accepted or

Table 2.1: Numbers of trials in each category in go training and the stop experiment (participant DS).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *30 ms*	SSD2 *110 ms*	SSD3 *170 ms*
responses	291	2155	48	129	213
inhibited	N/A	N/A	175	117	35
rejected	9	95	27	4	2

Table 2.2: Numbers of trials in each category in go training and the stop experiment (participant HB).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *30 ms*	SSD2 *90 ms*	SSD3 *110 ms*
responses	296	2921	70	206	252
inhibited	N/A	N/A	252	122	78
rejected	4	49	8	2	0

rejected based on the above criteria, stop trials resulted in successful inhibition, accepted response (stop failure), or rejection. The numbers of trials in each category for the three participants tested are shown in Tables 2.1–2.3. Also shown are the lengths of delay used for each participant.

2.2.1 Inhibition Functions

The basic requirement for a satisfactory stop signal is that it enables successful inhibition on some proportion of stop trials. The Logan-Cowan race model further predicts that the probability of success depends on the length of delay between go and stop signals: the longer the delay, the smaller the chance of

Table 2.3: Numbers of trials in each category in go training and the stop experiment (participant PB).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *30 ms*	SSD2 *110 ms*	SSD3 *170 ms*
responses	297	3167	59	164	303
inhibited	N/A	N/A	298	195	56
rejected	3	73	3	1	1

inhibition success. By comparing the percentages of successful inhibition (of all accepted trials) across the different stop conditions for the participants in Figure 2.2, it can be seen that these requirements were fulfilled by the tactile stop signal. The likelihood of successful inhibition dropped from around 80% for the condition with the shortest delay to around 20% for the longest delay, with intermediate delays producing probabilities around chance (50%). This monotonic pattern was evident for all three participants.

2.2.2 Saccadic Reaction Times

Table 2.4 compares the mean reaction times of each participant from the go training with those from the stop experiment (i.e. go control trials, stop failure trials in each delay condition of the stop experiment, and stop failures overall). HB was slower than either PB or DS on the pure go task, but was able to keep his average go response latency at the go training level in the stop signal experiment. In contrast, PB and DS were 59 and 65 ms slower on average, respectively, at go responses in the context of the stop trials relative to their average speed of responding before the introduction of the stop trials. All participants had faster average response times for each of the stop conditions than for the go control trials.

Figure 2.2: The inhibition functions of the three participants.

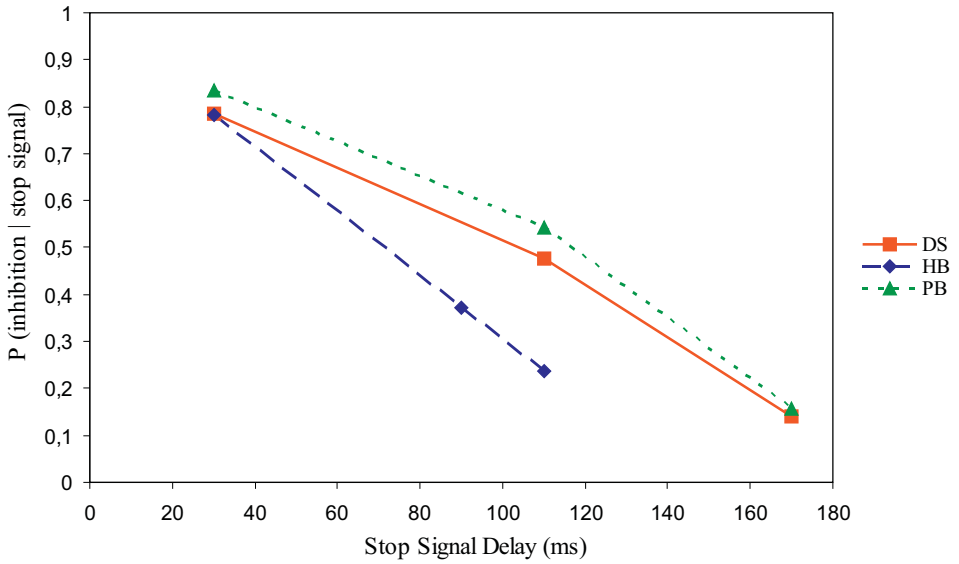
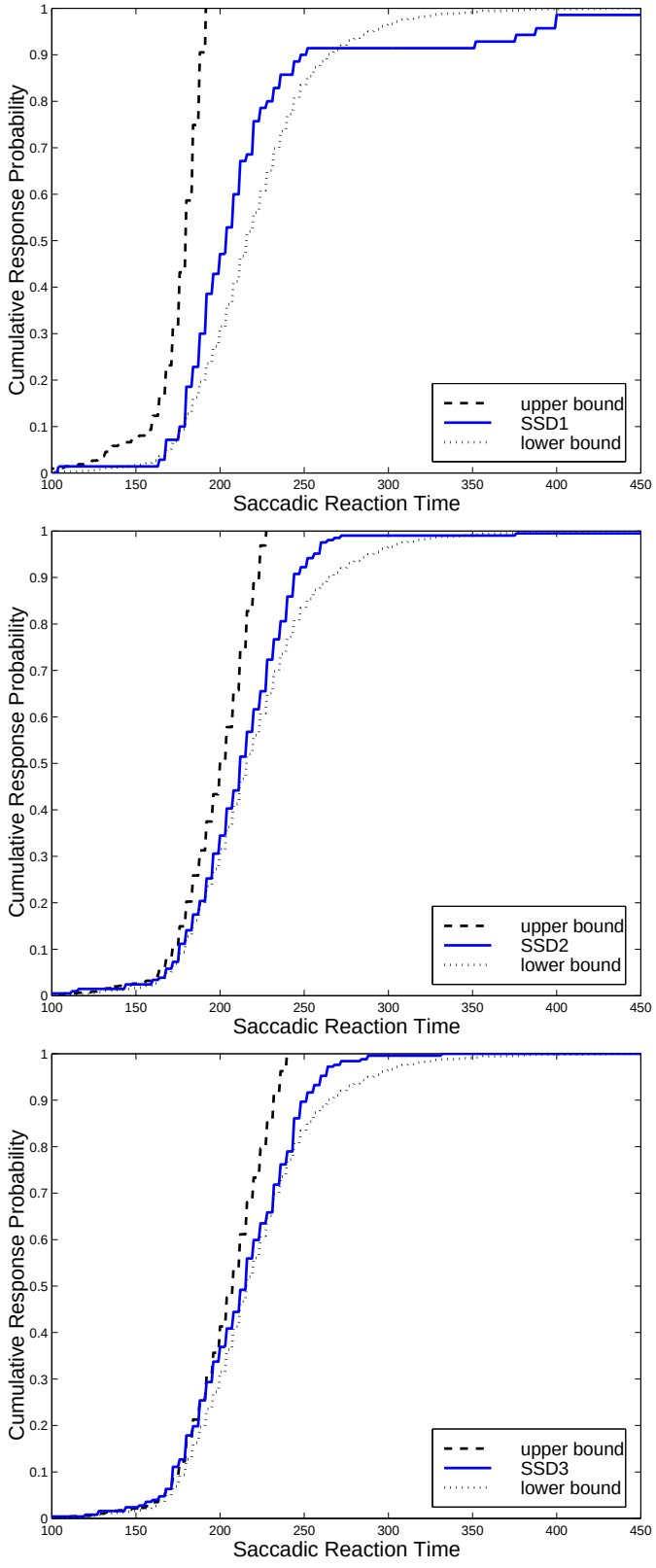


Table 2.4: Mean saccadic reaction times (and standard errors) of the three participants in each condition and across the stop conditions. All values are in ms.

	Go Training	**Stop Experiment**				
		Go Control	SSD1	SSD2	SSD3	All 3 Delays
DS	154 (2.5)	220 (1.3)	194 (12.9)	199 (5.4)	213 (3.9)	206 (3.2)
HB	218 (2.0)	220 (0.7)	218 (7.2)	214 (2.5)	213 (2.0)	219 (0.6)
PB	166 (1.3)	225 (0.8)	221 (7.6)	209 (2.5)	217 (2.3)	215 (1.8)

The slower go responses exhibited by DS and PB in the stop experiment were significantly different from the responses they performed during go training (p <0.001 for both participants, two-tailed t-tests). The difference obtained for HB was nonsignificant. A one-way ANOVA was performed on the RTs

Figure 2.3: Distribution inequality tests for the reaction times from the three delay conditions (participant HB).



in the stop experiment conditions separately for the data from each participant. For DS, it revealed highly significant differences among the conditions (p <0.001). Post-hoc tests showed that the first (p <0.05, Tukey-HSD) and the second (p <0.01) delay condition data had significantly faster means than the go control mean. Highly significant differences were also found between the RT means in the stop experiment data from PB (p <0.001). Post-hoc tests established that two of the delay conditions, SSD2 (p <0.001) and SSD3 (p <0.05), had significantly faster means than the go control condition. The ANOVA on HB's data also found significant differences among the mean RTs (p <0.01), of which only the comparison of go control and the last delay condition was significant in the post-hoc tests (p <0.05).

2.2.3 Distribution Inequality Tests

The individual saccadic response times obtained from the three participants in the different conditions were tested against the predictions of the Logan-Cowan race model, as described in the Introduction. The cumulative distribution of the probability of stop failure responses was plotted against RT, separately for each delay condition. Under the terms of the race model, the control go trial RT distribution forms the lower bound of the stop failure RT distribution, and the higher bound is found by dividing the control RT distribution by the appropriate stop failure probability (see the Introduction).

Figure 2.3 shows the cumulative distributions of the RTs for each delay condition for HB (and Figure 2.4 those for DS, Figure 2.5 for PB). In each graph, the solid line represents the stop failure responses under that delay, the dotted line shows the lower bound, and the dashed line marks the upper bound. As can be seen in the graphs, the accumulated stop failure responses do not violate the upper bound in any condition of any participant. However, the lower bound is violated by the slowest portion of the data in the shortest delay condition for all three participants (upper graphs in Figures 2.3–2.5).

Figure 2.4: Distribution inequality tests for the reaction times from the three delay conditions (participant DS).

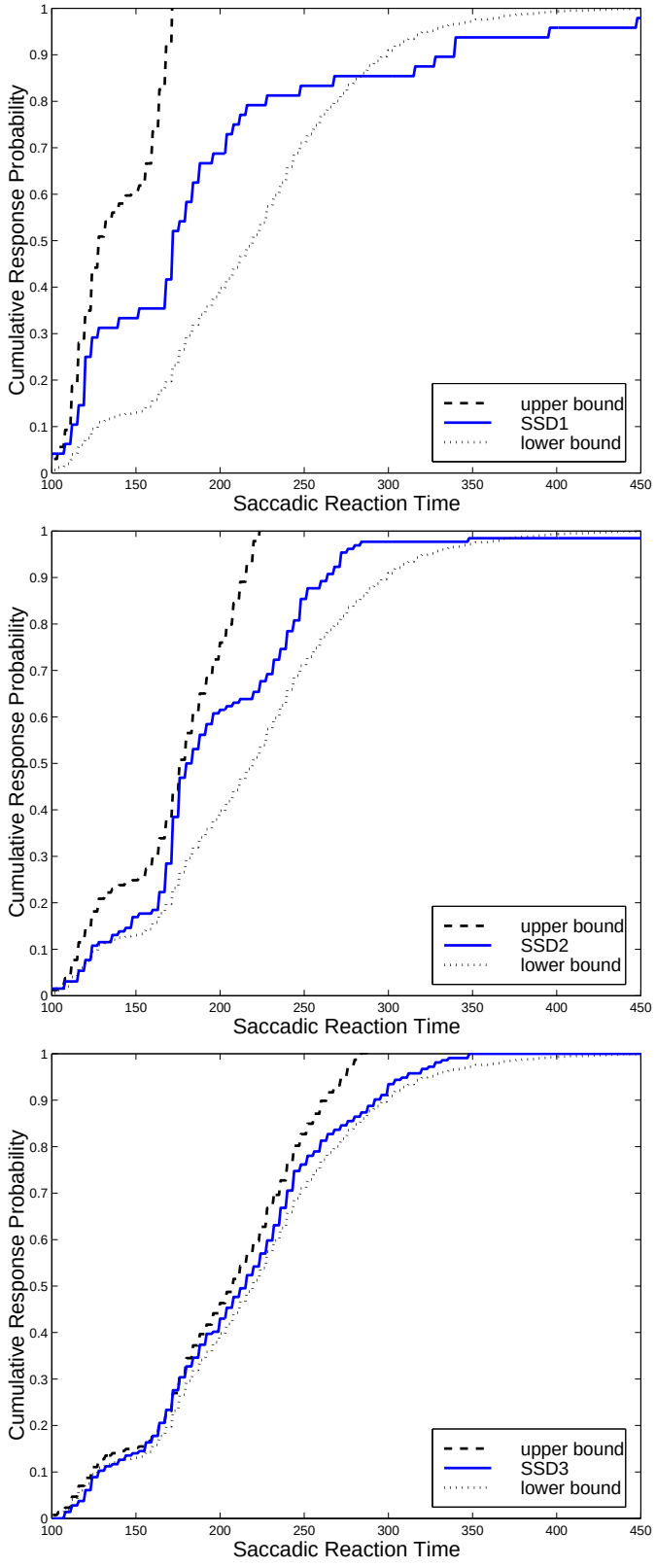
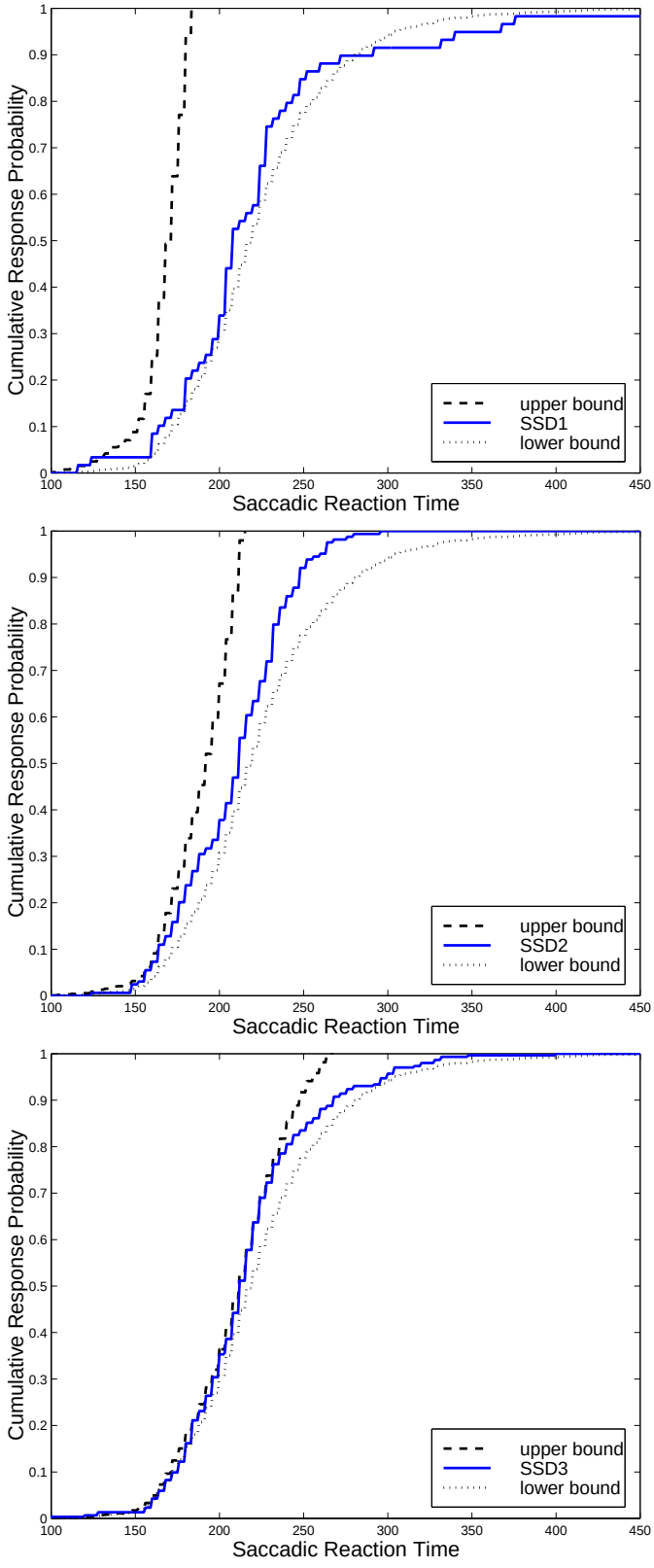


Figure 2.5: Distribution inequality tests for the reaction times from the three delay conditions (participant PB).



2.2.4 Saccadic Amplitudes

The average sizes of the saccades in each condition are shown grouped by participant in Table 2.5. For all participants the mean sizes of the eye movements performed under each stop condition were smaller than the means in the go condition of the stop experiment. Except for the longest delay condition for PB, the pattern was that shorter delays lead to shorter saccades.

Table 2.5: Mean saccadic amplitudes (and standard errors) in degrees of visual angle for the three participants in the go training and the stop experiment.

Participant	**Go Training**	**Stop Experiment**			
		Go Control	SSD1	SSD2	SSD3
DS	9.4 *(0.1)*	12.0 *(0.1)*	9.8 *(0.6)*	11.1 *(0.3)*	11.5 *(0.2)*
HB	11.9 *(0.1)*	11.6 *(0.0)*	10.4 *(0.3)*	10.9 *(0.2)*	11.1 *(0.1)*
PB	12.2 *(0.1)*	12.3 *(0.0)*	12.0 *(0.2)*	12.2 *(0.1)*	12.1 *(0.1)*

Subjecting the amplitude data to statistical testing revealed significant differences for all participants. Between the go training and the subsequent go control trials the saccades made by DS got signficantly larger (p <0.001, t-test), those from HB got smaller (p <0.01), and those from PB did not show a significant change in size.

One-way ANOVAs on the saccadic amplitudes across stop experiment conditions proved significant for all participants, with the data from DS and HB showing very high levels of significance (p <0.001 each), and that from PB only just proving significant (p <0.05). For DS, post-hoc tests showed that the saccadic amplitudes in the SSD1 condition were significantly smaller than both those in the SSD2 (p <0.05, Tukey-HSD) and the SSD3 (p <0.01) conditions, as well as those on the go control trials (p <0.001). The difference between mean saccadic amplitudes in the SSD2 and in the go control condition was also significant (p <0.01). The go saccadic amplitudes produced

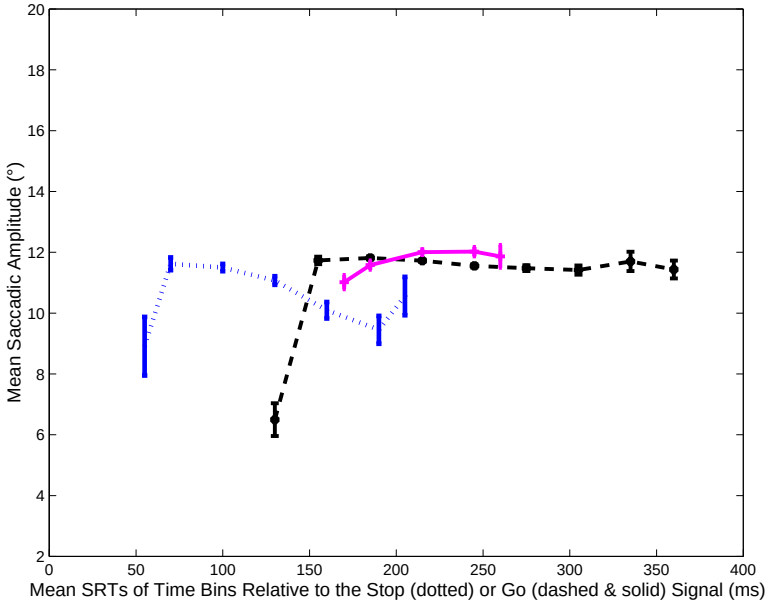
by HB were significantly larger than SSD1 (p <0.001), SSD2 (p <0.001), or SSD3 (p <0.001) saccades. In addition, the difference between the SSD3 and the SSD1 saccadic sizes was significant (p <0.01). None of the post-hoc comparisons for PB showed any significant effects.

Effects of Concurrent Go and Stop Signal Processing on Saccadic Amplitudes

Colonius et al. (2001) and Özyurt et al. (2003) reported findings of hypometric saccades on stop failure trials, and interpreted these as evidence of the stop signal affecting the dynamics of the ongoing saccade. More specifically, in their data the length of time which passed on stop failure trials between the stop signal presentation and the response was correlated with the decrease in saccadic amplitudes, compared to amplitudes on go trials. They suggested that stop signal processing might affect saccadic processing, and that increasing concurrent go and stop processing would accumulate this effect. In both studies, the fast saccades performed by several participants had extremely reduced amplitudes, which was interpreted as strategic responding at a time when the presentation of the stop signal was likely.

A direct comparison between the amount of concurrent go and stop processing and the size of the resulting saccade is made for each participant in Figures 2.6–2.8. Go training data are plotted as a solid line, and control go data as a dashed line. Each data point indicates the mean amplitude of saccades in a 30 ms time bin centred around the RT shown (except the first and the last data points, which include corresponding outliers). The dotted line plots the stop failure data pooled across the delay conditions, but with the appropriate length of delay subtracted from the RTs recorded. Hence, the dotted trace shows the relation between the concurrent go and stop processing and the amplitude, as above grouped into time bins of 30 ms and averaged over the amplitudes of the saccades in each bin. Figures 2.6 and 2.7 exhibit the data from HB and DS, respectively, and show similar patterns of substantially hypometric fast saccades in both go control and stop condi-

Figure 2.6: Mean saccadic amplitudes (and standard errors) for each 30 ms time bin of go training (solid), go control (dashed), and stop data across delay conditions (dotted) for participant HB. Go RT bins were defined relative to the time of go signal presentation, stop bins relative to stop signal presentation.



tions and more similar mean amplitudes among slower movements. For both participants and both tasks, amplitudes also decrease slightly with growing time lapse from the go or stop signal (except for the last bin of stop failures by HB). Standard errors are, however, quite large in the later time bins. PB's saccadic RT versus amplitude data, shown in Figure 2.8, is remarkably similar across both tasks and time bins. She showed no effects of duration of go processing, or duration of concurrent go and stop processing, on the sizes of her eye movements.

Figure 2.7: Mean saccadic amplitudes (and standard errors) for each 30 ms time bin for the data from participant DS (details as in Figure 2.6).

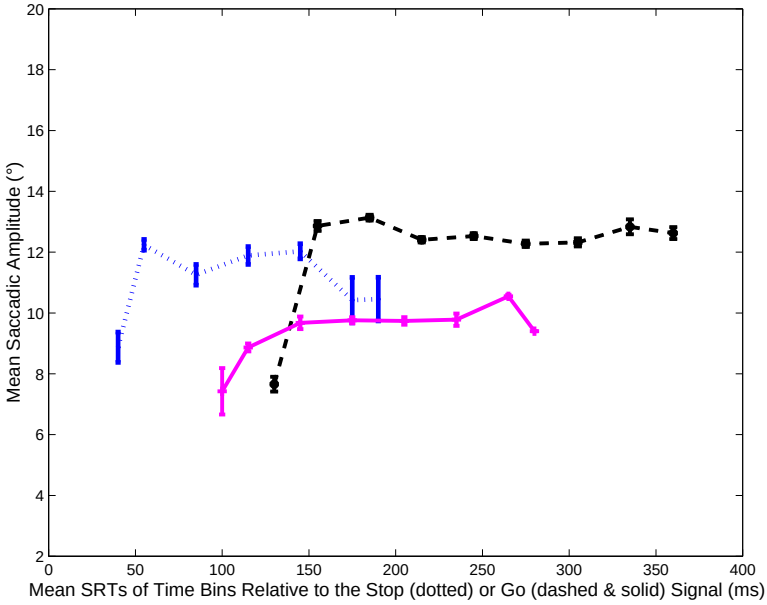
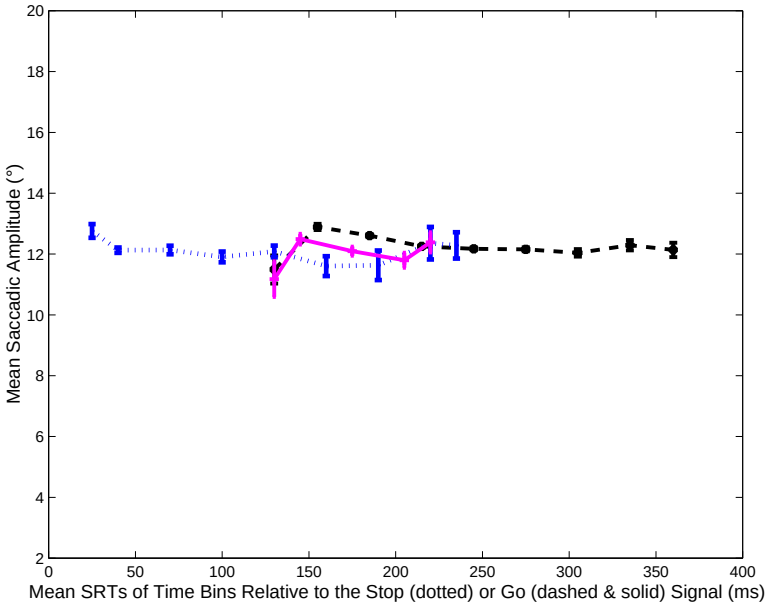


Figure 2.8: Mean saccadic amplitudes (and standard errors) for each 30 ms time bin for the data from participant PB (details as in Figure 2.6).



2.2.5 Stop Signal Processing Times

Following the Logan-Cowan race model, the effectivity of the stop signal was assessed by using the means and distributions functions of the reaction times obtained to estimate the stop signal processing time. Using the method of averaging across the delays (of several methods reviewed by Band et al. [2003]), the estimates for the SSPTs in this experiment were 91 ms (standard error=6.0) for participant DS, 139 ms (4.2) for HB, and 110 ms (14.7) for PB.

2.3 Discussion

The aims of this experiment were, firstly, to introduce the usage of a tactile stimuli in the stop signal task and, secondly, to test the ability of the Logan-Cowan race model to predict the data obtained. In an attempt to assure that the model's assumption of context-independent processing was not violated by procrastination strategies of the participants, go RT data was collected for comparison before the main experiment. The data of the experiment demonstrate that the tactile stimulus was an effective stop signal in that all three participants tested were able to inhibit successfully on a proportion of those trials in which this was required. In agreement with race model predictions, the probability of successful stopping was reduced with longer delays between go and stop signals.

The race model predicts that the mean RT latencies in the stop failure trials should stay below that of the control go trials. In addition, mean RT should increase with the duration of the delay, approaching but not exceeding the go mean RT. Supposing that the go and stop processes are indeed independent of the context introduced by the other process, there should be no difference between RTs on go trials in the training context and those in the stop experiment.

The data from participants DS and PB strongly departed from the above-mentioned hypothesis of context-independence. Both slowed down their — on average relatively fast — responses on the go trials when these were presented intermixed with stop trials. When considering only the mean RTs of the different stop experiment conditions, the data from DS corresponded to the predicted pattern. His slowest mean RT was found in the go condition, and those in the delay conditions grew with increasing delay. PB was unexpectedly slow on the first delay condition. Although her other delay conditions produced significantly faster mean RTs than the go responses, the first delay condition (which should be the fastest) was only marginally, and nonsignificantly, faster than go control. The third participant, HB, also produced faster mean RTs in the delay conditions than on the go control trials, but contrary to model predictions he got slightly faster along the delay conditions.

From the mean RT data it is clear that participants DS and PB were unable to achieve the goal set for the stop experiment of keeping up the mean RT they had exhibited in go training. It was evident that introducing the stop trials had an effect on the go processing of these two participants, probably in the form of unconscious and unintended strategies favouring good stopping performance. This procrastination strategy is a well established consequence of the conflict situation participants are in when trying to inhibit well practiced responses (e.g., Lappin & Eriksen, 1966; Ollman, 1973), and amounts to a contradiction of the race model assumption of context-independent processing. Therefore, the data obtained from HB, who did not seem to adopt a speed-accuracy trading strategy, was of higher relevance and interest for the more detailed fitting of the RT data to the model predictions than the data from the other participants. However, since further analyses of the RT data from DS and PB showed interesting parallels with that from HB, as well as with effects found in the literature, these more detailed tests were reported for all participants.

Plotting the stop failure RT distributions from each delay conditions, along with the appropriate upper bound and the common lower bound for that participant, revealed similar violations of the race model for all three participants. The slowest 10–20% of the stop failure responses performed in the first delay condition were unexpectedly slow and should, according to model predictions, have been inhibited by the stop process. Several earlier studies have shown these violations of race model predictions for some of the participants tested (e.g., Logan & Cowan, 1984; Hanes & Carpenter, 1999; Colonius et al., 2001). The fact that the violations tend to occur on trials with short SSDs, and specifically on the slowest reactions on these trials, suggests that the presentation of the stop signal affects the processing of the go signal on those trials. Özyurt et al. (2003) proposed that stop signal interference with go processing might slow down this operation, and hence also the response made on such trials. Alternatively, go signal interference with stop signal processing might delay the progress of that operation and allow for slow responses to be executed despite proper initiation of the inhibition process.

An interaction between the go and stop processes would be expected to affect not only the RTs and but also the amplitudes of the stop failure saccades. The direction and amplitude of a saccade are thought to be programmed before the start of the actual movement (e.g., Findlay & Walker, 1999), but presenting an additional stimulus may affect the movement as it is carried out, even if the modality of the additional stimulus is different from the target stimulus. For example, Doyle and Walker (2002) found that a visual accessory stimulus affected the trajectory of voluntary saccades to a visual target regardless of the instruction, whereas auditory and tactile stimuli did so only when they were relevant for the task. As the appearance of the tactile stop signal was a highly informative signal in the present experiment, it would be expected that this would affect the saccadic amplitudes as well as the RTs.

The participants in this experiment produced quite accurate saccades to the go signal, which was always presented either at -10 or at 10 degrees of visual angle in the horizontal plane. Across participants, the mean amplitudes of the conditions ranged from 9.4 to 12.3 degrees of visual angle, and the largest standard error was 0.6 degrees of visual angle. It seems that it was very easy to perform an exact movement towards the red light in the otherwise dark experimental room, despite the fact that spatial accuracy was not specifically called for and no feedback was given about it during practice or the experiment. Thus, the small, but in most cases systematic and statistically significant, differences in mean saccadic amplitudes in the stop experiment were unlikely to be due to the natural variability in saccade sizes. For all participants, amplitudes in the three delay conditions were smaller than those on the go control trials, and the smallest mean amplitudes were measured in the first delay condition. Hence, earlier stop signals which failed to produce inhibition had larger effects on the size of the resulting saccade than later ones.

Both the amplitude effects on the go trials and those on the stop failure trials reported in Colonius et al. (2001) and in Özyurt et al. (2003) were replicated in this experiment. In go control trials, DS and HB produced much smaller saccades when the response was fast (<140 ms) than when the movement was around the mean RT or slower. At this point of the trial, a possible stop signal would already have occurred for HB, and would be increasingly unlikely for DS. Thus, a strategy of expecting a stop signal when it is most likely to appear would produce effects around this latency and before it. A state of simultaneous preparation for the possibilities of movement and inhibition might affect motor programming of the saccade so that resulting saccades are hypometric. Accordingly, the go training data of HB was very similar to that in the go control condition, but did not show hypometricity on fast reactions. The go training data from DS featured generally small movements, but without the distinct dip in size on the fast reactions.

However, it is not immediately clear how the possible simultaneous preparation would have produced the similarly hypometric saccades in the first bin of the stop failures, as produced by both DS and HB and also present in the data obtained by Colonius and colleagues. As these responses involved only up to about 50 ms concurrent go and stop processing, and the latest possible stop signal was presented 170 ms (DS) or 110 ms after the go signal, the first bin cannot include slow responses. Instead, the fastest stop failures are likely to be found in this bin, possibly including express saccades. These are extremely fast (around 100 ms in humans), tend to have smaller amplitudes than regular saccades (e.g., Fischer & Weber, 1993), and are most frequent when the stimulus presentation involves a gap between the fixation point and the imperative stimulus, as was the case in this experiment. Thus, the possibility of a substantial proportion of express saccades in the first bin might explain the hypometricity found in the data from DS and HB in both the go control and stop failure conditions, as well as in go training for DS.

Finally, the estimated SSPTs showed that the participants were good at cancelling their eye movements when suddenly required to do so. The differences in inhibitory performance between participants were relatively small. HB, who required shorter SSDs than the other two participants to achieve similar levels of inhibition success, consequently had the longest SSPT. Note that these estimated SSPTs are shorter than those frequently reported in the stop signal literature (e.g., Logan, 1994; Williams et al., 1999). Even shorter SSPTs have, however, been obtained using visual go and auditory stop signals (Colonius et al., 2001; Özyurt et al., 2003). Thus the intermediate peripheral processing time of tactile stimuli, relative to visual and auditory stimuli (e.g., Todd, 1912; Diederich, 1995; Diederich & Colonius, in press), would support rather shorter SSPTs than those obtained in the majority of stop experiments using only visual stimulation. Further, SSPTs have been shown to be shorter for saccadic responses, similarly to the generally shorter latencies for saccades compared to manual responses (Logan & Irwin, 2000). Thus, the estimated SSPTs also mirror the comparatively short RTs obtained in the present experiment.

In summary, the experiment showed that a tactile stimulus can be employed successfully as stop signal in a saccadic reaction time task with visual go signals. Each of the three participants exhibited deteriorating stopping performance with increasing delay of the vibratory stop signal relative to the imperative visual go signal. The estimated stop signal processing times were shorter than those found in the literature, probably due to shorter peripheral processing times of tactile stimuli. The comparisons of mean saccadic reaction times across conditions showed that introduction of stop trials affected the go performance of two of the participants to the extent that they slowed their responding significantly in expectation of the stop signal. All participants were slower on a proportion of responses in the shortest delay condition than what was predicted by the Logan-Cowan race model. These violations of predictions, along with the hypometric saccades on stop failure trials exhibited by all participants, and reaching statistical significance for two of them, support suggestions of earlier studies in questioning the ability of the race model to account for such data.

3

Saccadic Inhibition with Visual Stop Signals

The first experiment of this study showed that a tactile stop signal can be employed in stopping saccadic responses to a visual go signal. In addition, we obtained both RT and saccadic amplitude data challenging the Logan-Cowan race model. To complement the support found for the assumption of modality-independence of the process, we proceeded to investigate whether reversing the roles of the stimuli produces the same pattern of results. Thus, in the second experiment of this study tactile stimuli were used as go signals and visual stimuli as stop signals. The effect of varying the spatial position of the stop signal in the horizontal plane was also investigated.

3.1 Method

3.1.1 Design

This experiment investigated participants' ability to cancel a programmed rapid saccade to a tactile go stimulus at different stages of processing due to

the presentation of a visual stop signal. Participants were presented with a vibratory tactile stimulus at either hand, and their task was to respond to the stimulation as fast as possible by performing a saccadic eye movement towards the stimulated hand. In a pretest, participants practiced this response task so that go data without the context of the occasional stop trials could be obtained. Subsequently, a visual stop signal was presented on some trials after one of three possible delays, either to the right or the left of the midline. On these trials participants were required to inhibit the saccade if possible. This task was also practiced to find suitable stop signal delays and to allow participants to adjust to the new task before the actual stop experiment.

Inhibition success and reaction time data were collected and compared with predictions of the Logan-Cowan race model, while procrastination strategies were discouraged. Saccadic amplitudes were also collected to explore the possible connection between the sizes of the responses and the differences in time pressure of inhibition. Additionally, the possibility of differential effects on stopping performance of ipsi- and contralateral presentation of the stop signals relative to the go signal was considered.

3.1.2 Participants

Three participants were tested after screening for ability to follow the instructions and perform satisfactory saccades. Two of them were female (BH, left-handed, age 25 years); DL, right-handed, 21) and one was male (BW, right-handed, 25). All had normal or corrected-to-normal visual acuity. The experiment was carried out with the informed consent of the participants, who were compensated for their participation partly with compulsory subject hours and partly in money paid out after the last session.

3.1.3 Apparatus and Stimuli

The experiment was carried out in a darkened and sound attenuated room. Stimuli were presented on a table which had an indentation on one side. The participant was seated in the indentation so that the surface of the table provided support for the elbows and lower arms resting on it, while a chin rest supported the head. The tactile stimuli were administered to the hands by two vibration exciters (Brüel & Kjær, Nærum, Denmark) situated under the table, each extending a rod through a hole in the table. The rods were covered by wooden balls resting in the palms of the hands and conveying the vibratory signals (duration: 500 ms, frequency: 50 Hz) to these. The visual stimulation was provided by two light emitting diodes (LEDs) of 5 mm diameters and 8 cd/m^2 luminous intensity each. Another identical LED served as the fixation point. All visual signals were presented for 500 ms. The tactile and visual stimulus pairs were placed 10° to the left and right of the midline, the visual ones 60 cm in front of the participant and the tactile ones adjacent to the chin rest. The fixation point was positioned on the midline at a distance where the apparent position, as seen by the participant during the experiment, was halfway between the hands and the two visual stimuli. Response detection and recording was done using the infrared light video system EyeLink (Sensomotoric Instruments, Berlin, Germany) at a rate of 250 Hz.

3.1.4 Procedure

Two main categories of trials were presented, go and stop trials. Go trials started with the appearance of the fixation point, which after 500 ms was followed by a variable gap (range: 500–1500 ms) during which no stimuli were presented. After this, one of the tactile stimuli was presented as a go signal for 500 ms, after which an intertrial interval of 1000 ms provided a break before the next trial began. The fixation point, gap, and tactile go stimulus sequence was identical for both go and stop trials, but in the latter

either of the visual stop signals was additionally presented. The timing of the stop signal depended on the performance of the participant, and the total range was between -30 and 170 ms relative to the go signal. A go training block contained 100 go trials, whereas a stop training and experimental block was made up of 108 go (control) and 36 stop trials. Hence, the likelihood of a stop signal occurring in a stop experiment trial was 25%. Left and right presentations were equiprobable for both go and stop stimuli.

Participants first practiced responding using the go training blocks. In these, the tactile stimulation of either hand required a quick eye movement from the location where the fixation point had appeared to the stimulated hand. After the participant was comfortable with the task and the measurement procedure, several sessions were run until the mean RT over a block stabilised at some level. Thereafter, the blocks with both go trials and stop trials were presented. As in the preceding go training, participants should perform a fast response towards the stimulated hand on the go trials. On the stop trials, participants were required to cancel the programmed saccade if possible. However, they were informed that inhibition would not always be possible and that they should not slow down responding in expectation of a possible stop signal.

On go trials participants could either perform a valid saccade or commit an error, such as performing a saccade in the wrong direction or responding too late. On stop trials they might perform a saccade (*stop failure*), succeed in inhibiting, or commit an error. Three different delays of the stop signal relative to the go signal were determined for each participant to generate three distinct probabilities of inhibition success. Participants practiced until they either performed as fast on the go control trials as in go training or seized improving their RTs, and until the individual stop signal delays could be determined. Finally, each participant completed several sessions of the stop experiment with the chosen delays over a number of weeks. The dependent measures were saccadic latency and amplitude in both the go and the three stop conditions, as well as probability of response in the stop conditions.

3.2 Results

After exclusion of trials including blinks, improper fixations, or inadequate saccades, a total of 3196 trials were obtained from participant BH in the stop experiment, 3948 trials from BW, and 3666 trials from DL. The corresponding figures for the go training were 261 (BH), 277 (BW), and 222 (DL). In all data sets responses were considered inadequate when they were larger than 40°, guesses (RT latency below 70 ms), slower than 500 ms, or when they were made in the wrong direction. In go trials responses also had to be larger than 2°, whereas in stop trials small responses were classified as successful inhibitions. The number of trials for each participant in each category are shown in Tables 3.1–3.3 together with the individuals stop signal delays used. A negative SSD value indicates that the stop signal was presented before the go signal presentation.

3.2.1 Inhibition Functions

A successfully implemented stop signal should be suitably distinct and be presented at SSDs which allow the participants to succeed in inhibiting their responses on some, but not all, stop trials. When several delays are used, the most information about the processes involved is obtained when these are chosen to produce a distinct probability of success at each delay. Figure 3.1 shows the percentage of successful inhibition plotted against the delay for the three participants. The graph shows three distinct probabilities for each participant, with a remarkable similarity in the functions of participants BH and DL. The data from BW reveals a comparative disinclination to inhibit, as he required the stop signal to be presented up to 100 ms earlier to achieve similar levels of success as the other two.

Table 3.1: Numbers of trials in each category in go training and the stop experiment (participant BH).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *80 ms*	SSD2 *130 ms*	SSD3 *170 ms*
responses	261	2354	71	210	241
inhibited	N/A	N/A	193	78	49
rejected	39	346	36	12	10

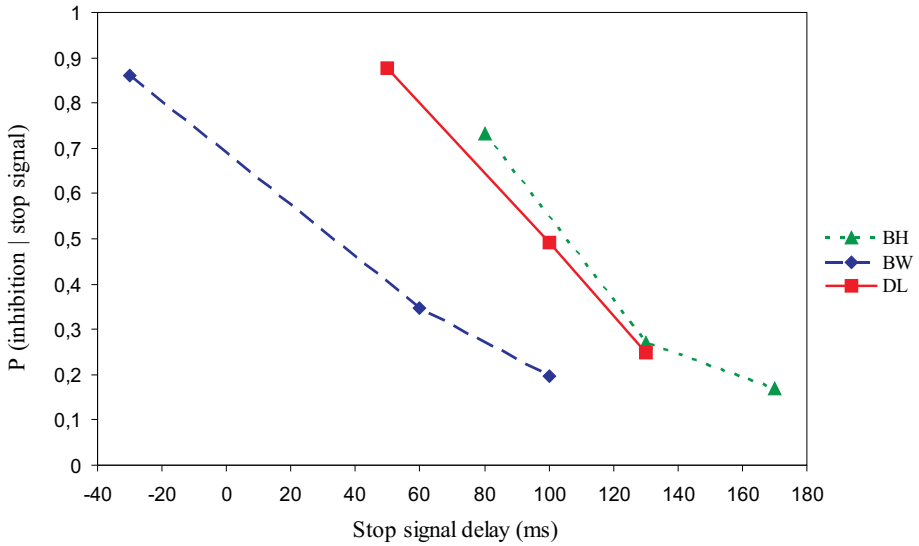
Table 3.2: Numbers of trials in each category in go training and the stop experiment (participant BW).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *-30 ms*	SSD2 *60 ms*	SSD3 *100 ms*
responses	277	2924	46	228	275
inhibited	N/A	N/A	287	121	67
rejected	23	316	27	11	18

Table 3.3: Numbers of trials in each category in go training and the stop experiment (participant DL).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *50 ms*	SSD2 *100 ms*	SSD3 *130 ms*
responses	222	2706	40	162	240
inhibited	N/A	N/A	282	156	80
rejected	78	210	2	6	4

Figure 3.1: The inhibition functions of the three participants.



3.2.2 Saccadic Reaction Times

Table 3.4 shows the mean RTs of the three participants in the go training condition, the go control condition and the three stop conditions, as well as across the three stop conditions. The goal of keeping the mean response speed on go trials in the stop experiment at the level of the go training was best attained by participant BH. Her 2 ms difference in the averages between go training and go control trials was nonsignificant. In contrast, DL produced significantly slower go responses in the stop experiment compared to go training (p <0.05, two-tailed t-test), as did BW (p <0.001).

When comparing RT data from the stop experiment, all participants' RT latencies in the stop conditions were shorter than those in the go control condition, both overall and in each individual stop condition. One-way ANOVAs revealed significant differences in mean RT across the conditions for all participants (p <0.001 for each participant). For BH and BW the mean latencies increased across the delay conditions, with SSD1 having the fastest reactions,

Table 3.4: Mean saccadic reaction times (and standard errors) of the three participants in each condition and across the stop conditions. All values are in ms.

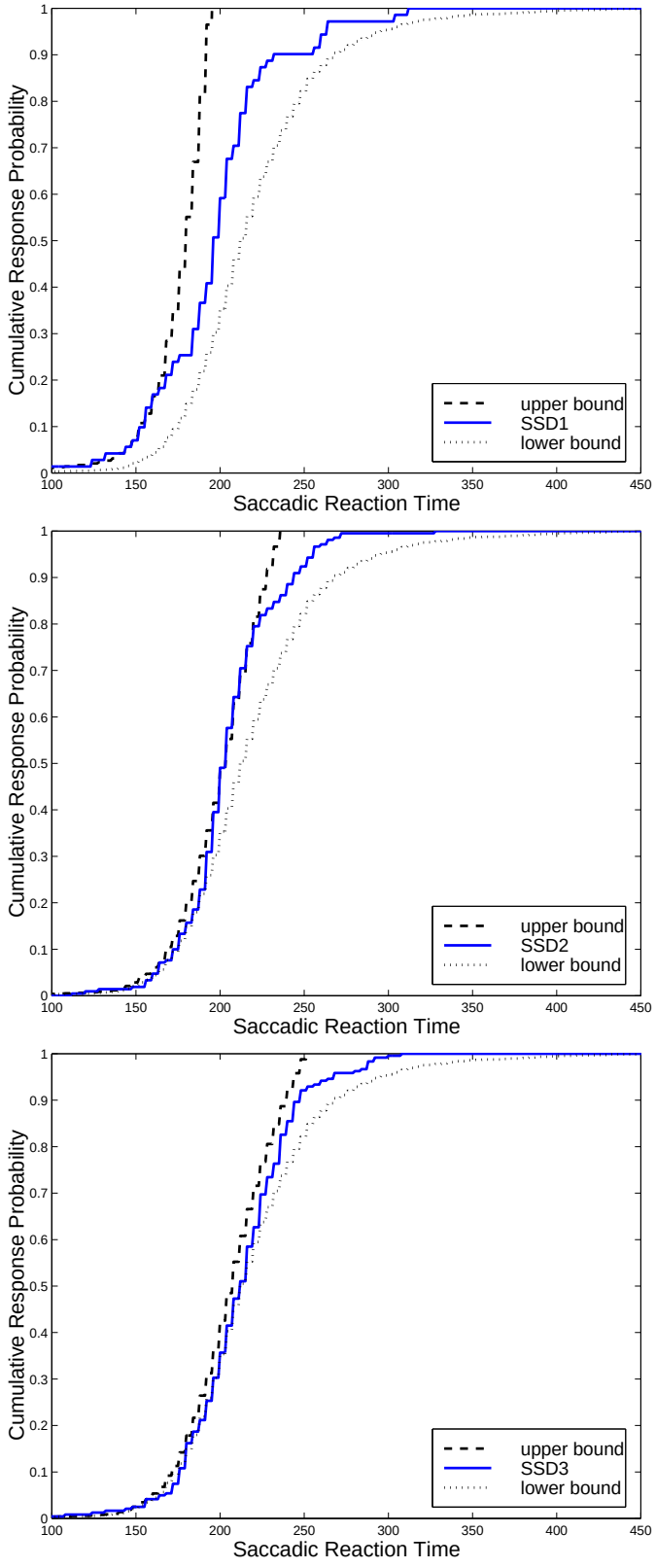
		Stop Experiment				
	Go Training	Go Control	SSD1	SSD2	SSD3	All 3 Delays
BH	218 (2.3)	220 (0.9)	197 (4.4)	205 (1.9)	213 (2.1)	207 (1.4)
BW	210 (2.2)	222 (0.8)	204 (8.7)	213 (2.6)	218 (2.2)	214 (1.7)
DL	212 (2.1)	219 (0.8)	210 (3.9)	215 (2.6)	207 (2.0)	210 (1.5)

followed by SSD2 and then by SSD3. Subjecting the stop experiment data to post-hoc tests showed that the first and second delay condition mean RTs were significantly faster than the mean go RT for BH (Tukey-HSD, $p < 0.001$ for both comparisons). Further, her mean SSD1 RT was also faster than her mean SSD3 RT ($p < 0.05$). For BW, only the faster RTs in the SSD1 and SSD2 conditions relative to the go condition were significant ($p < 0.05$ for both comparisons). For the third participant DL, the ranking of average RTs of the go control, SSD1, and SSD2 conditions was the same, but none of the post-hoc tests for these differences were statistically significant. However, DL was surprisingly fast in the last delay condition, and the difference to the average go control RT was highly significant ($p < 0.001$).

3.2.3 Distribution Inequality Tests

The cumulative RT distributions from the different conditions and participants were plotted along with the limits set by the Logan-Cowan race model, as described in the Introduction. To be in line with the predictions of the model, the distribution of stop failure responses has to lie above (i.e. have faster RTs than) that of the go RT distribution, as well as below the go RT distribution divided by the probability of producing a stop failure in that condition. These distribution inequality tests are shown in Figure 3.2 for

Figure 3.2: Distribution inequality tests for the reaction times from the three delay conditions (participant BH).



BH, Figure 3.3 for BW, and Figure 3.4 for DL. In each graph, the solid line plots the cumulative distribution of stop failure RTs in that particular delay condition, the dotted line denotes the lower bound, and the dashed line the upper bound of the distribution.

The distribution inequality tests for the data from BH showed no evidence of violations of the predictions of the race model in any delay condition. In contrast, BW's data show small violations of the lower bound on all delay conditions. The differences occur on different sections of the distribution in the different conditions: The largest violation was found for the slowest responses in the SSD1 condition, in the SSD2 condition some quite fast responses crossed the lower bound, and in the SSD3 condition a small violation was found around the mean RT. DL also showed clear violations of the lower bound on the first two conditions, and a marginal violation of the upper bound at fast responses in the third delay condition.

3.2.4 Saccadic Amplitudes

Table 3.5 shows the averages sizes of responses of the participants on the different conditions. BH's saccades on the go control trials were of similar size as those on the go training trials. The amplitudes on all three stop conditions were smaller, with SSD1 producing the smallest average responses, followed by SSD2 and then SSD3. A one-way ANOVA on the sizes of the stop experiment data from BH revealed highly significant differences between the conditions (p <0.001). Post-hoc tests showed that the differences between mean amplitude on go compared with SSD1 (p <0.001, Tukey-HSD), with SSD2 (p <0.001), and with SSD3 (p <0.05) were all significant. In addition, the SSD1 mean amplitude was significantly smaller than that from the SSD2 (p <0.01) or the SSD3 condition (p <0.001).

The data from participant BW also showed the similarity between the go training and go control means, as well as the rank order of the means of the stop conditions. The first two stop conditions also produced smaller sac-

Figure 3.3: Distribution inequality tests for the reaction times from the three delay conditions (participant BW).

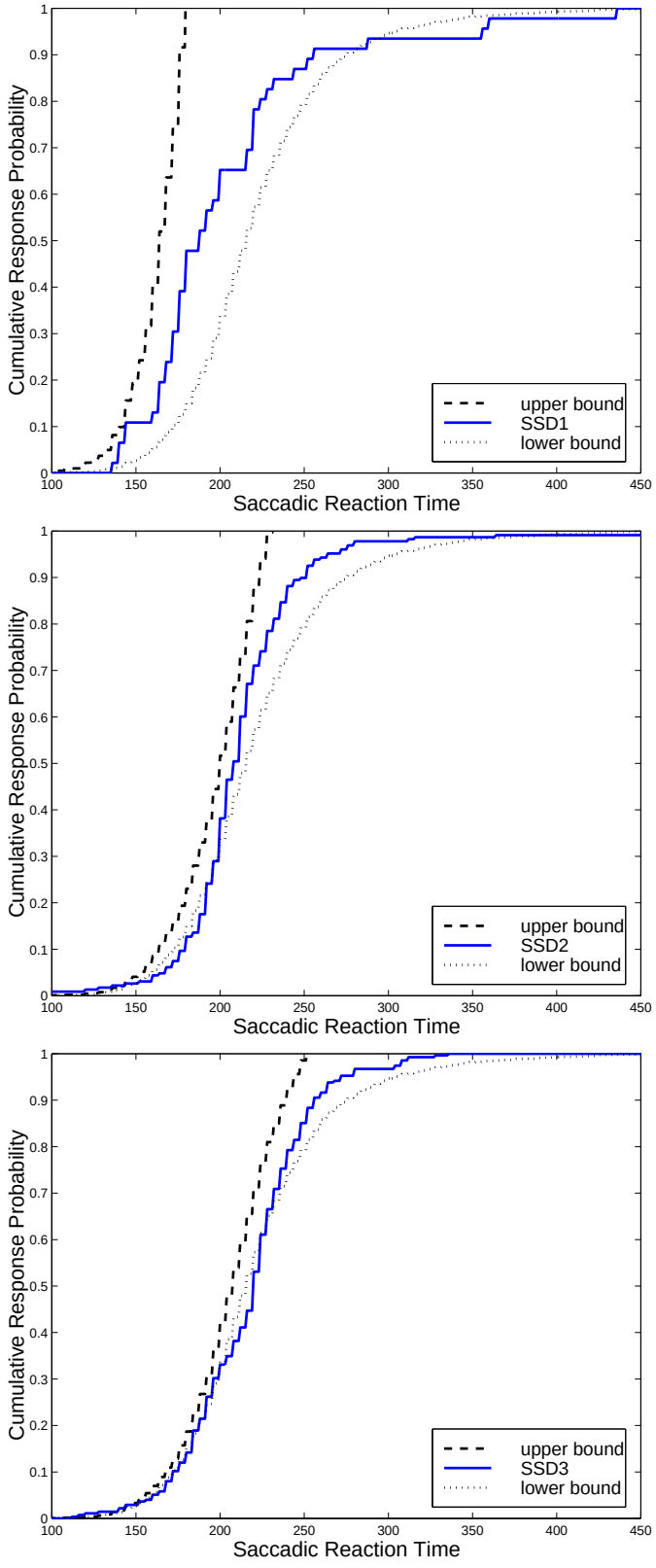


Figure 3.4: Distribution inequality tests for the reaction times from the three delay conditions (participant DL).

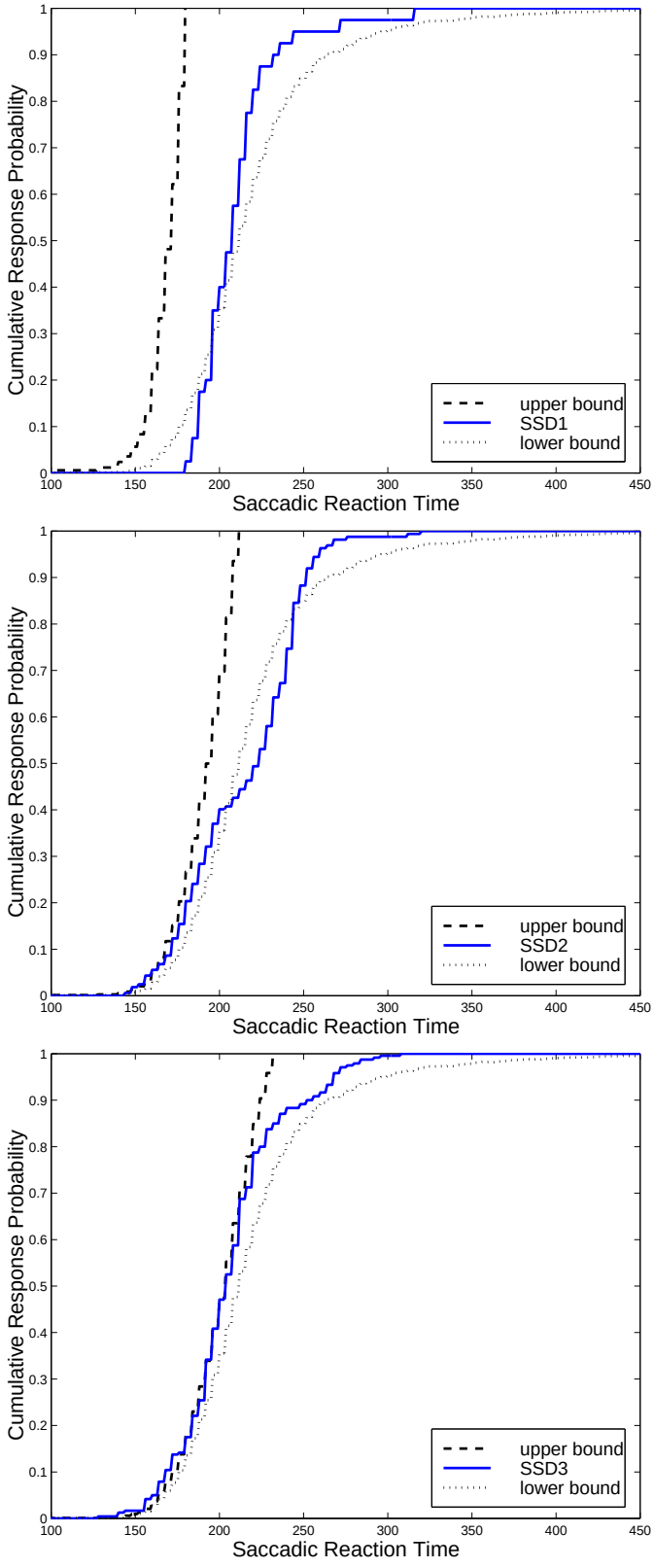


Table 3.5: Mean saccadic amplitudes (and standard errors) in degrees of visual angle for the three participants in the go training and the go and three stop conditions of the stop experiment.

	Go Training	**Stop Experiment**			
		Go Control	SSD1	SSD2	SSD3
BH	16.8 *(0.4)*	16.2 *(0.1)*	11.7 *(0.7)*	14.1 *(0.4)*	15.0 *(0.4)*
BW	20.7 *(0.3)*	20.8 *(0.1)*	18.5 *(1.1)*	19.2 *(0.4)*	20.9 *(0.4)*
DL	20.6 *(0.4)*	16.5 *(0.1)*	12.9 *(1.1)*	14.2 *(0.5)*	15.6 *(0.4)*

cades than both go conditions, but the responses in the last stop condition were marginally larger than those in the go trials. Again, the stop experiment data showed significant differences between the conditions (p <0.001, one-way ANOVA). The post-hoc tests showed that the go saccades were significantly larger than the SSD1 (p <0.05) and the SSD2 (p <0.001) responses. Likewise, the SSD3 condition produced significantly larger saccades than the first (p <0.01) or the second condition (p <0.05).

For DL, the go training responses were considerably, and significantly, larger than the go responses in the stop signal context (p <0.001, two-tailed t-test). Similarly, the differences within the stop experiment conditions proved to be significant (p <0.001, one-way ANOVA). The amplitudes in the stop experiment conditions follow the general pattern seen by the other participants, with go responses being larger than stop responses, and the average size of responses in the stop conditions increasing from SSD1 to SSD3. Post-hoc comparisons showed that amplitudes were significantly smaller relative to go control amplitudes in conditions SSD1 (p <0.01) and SSD2 (p <0.001). There were no significant differences in average sizes between the three stop conditions for this participant.

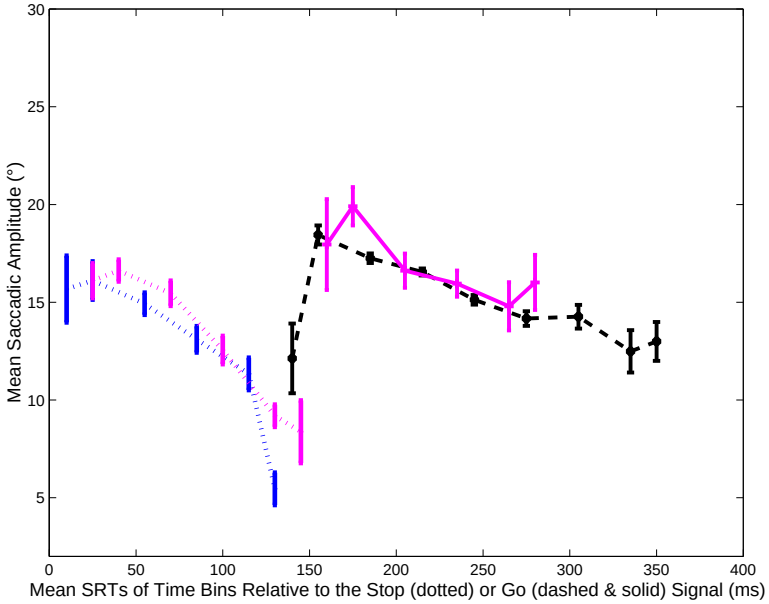
Effects of Concurrent Go and Stop Signal Processing on Saccadic Amplitudes

The substantial reduction in saccadic amplitudes in the delay conditions compared to the go condition, and the observation that this effect was larger on shorter delays, support suggestions by Colonius and colleagues (Colonius et al., 2001; Özyurt et al., 2003) that the effect of the stop signal on the go process is not all-or-none. On the contrary, the presence of the stop signal seems to change the trajectory of the saccade, either at the stage of saccade programming or during the movement itself. In the first experiment of this study, two participants exhibited the pattern found by Colonius and his colleagues: The effect of saccadic amplitude reduction increased with the time period of concurrent processing of the go and stop signals.

The corresponding comparisons in the data from the present experiment are shown in Figures 3.5 (for BH), 3.6 (BW), and 3.7 (DL). The responses were grouped into time bins of 30 ms and plotted against the mean amplitudes of the bins. The go training (solid line) and go control data (dashed line) are plotted relative to the go signal (with outliers included in the first and the last data points). The dotted lines show the stop failure responses for ipsilateral (darker; go and stop signals on same side of the midline) and contralateral trials (lighter; signals on opposite sides of the midline), across delay conditions but with the appropriate length of delay subtracted. Thus, the go data indicates how the saccadic amplitudes of the participants were related to the length of go processing, whereas the stop failure data shows the unfolding of the effects of simultaneous go and stop processing.

All participants showed effects of smaller saccadic amplitudes on longer concurrent go and stop processing. The spatial position of the stop stimulus relative to the go stimulus did not seem to affect this, as the slopes of the ipsi- and contralateral stop failure data are very similar. BH showed greatly reduced amplitudes on her fast saccades in the first bin of the go control condition, and a tendency to produce smaller saccades towards the slowest bins.

Figure 3.5: Mean saccadic amplitudes (and standard errors) for each 30 ms time bin of go training (solid), go control (dashed), and stop data across delay conditions (dotted: ipsilateral is plotted dark grey, contralateral light grey) for participant BH. Go RT bins were defined relative to the time of go signal presentation, stop bins relative to stop signal presentation.



This sloping pattern of longer responses having smaller amplitudes was also found in BH's go training data, as well as in the go control data produced by DL.

3.2.5 Effects of Spatial Position

To explore the possible effects of the relative horizontal position of the stop and the go signals, the stop failure data summarized above was further analyzed by comparing ipsilateral with contralateral trials. Figure 3.8 shows the proportion of ipsi- relative to contralateral cases of stop failures of the total in each delay condition. As can be seen, for all participants fewer ipsilateral than contralateral saccades were performed on stop trials in the first delay condition. Thus, for early stop signals, shown in the inhibition functions

Figure 3.6: Mean saccadic amplitudes (and standard errors) for each 30 ms time bin for the data from BW (details as in Figure 3.5).

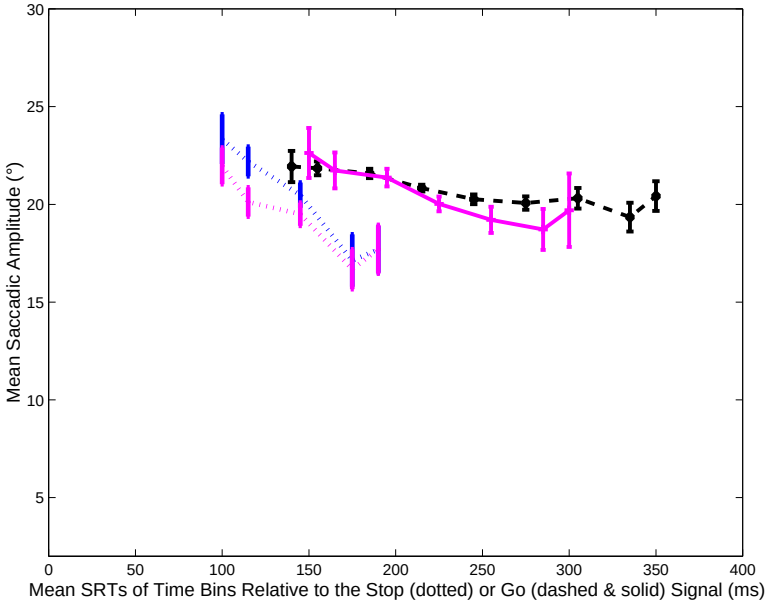
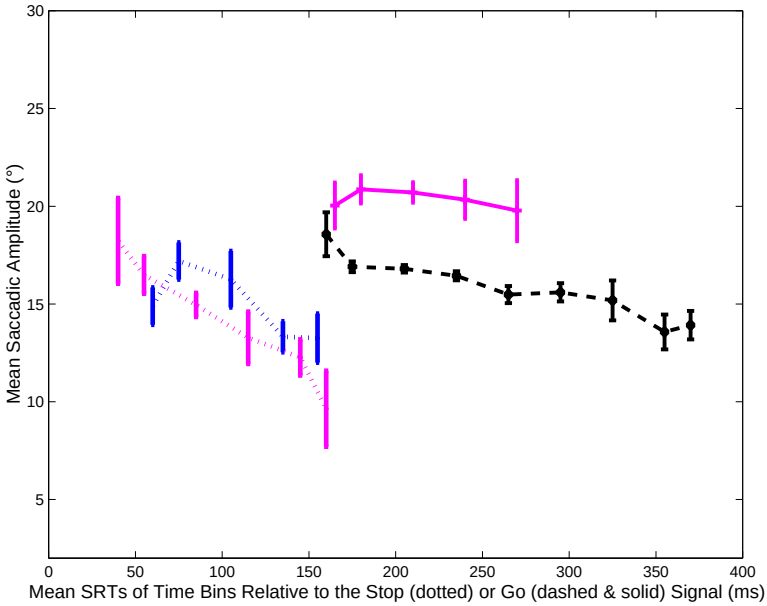
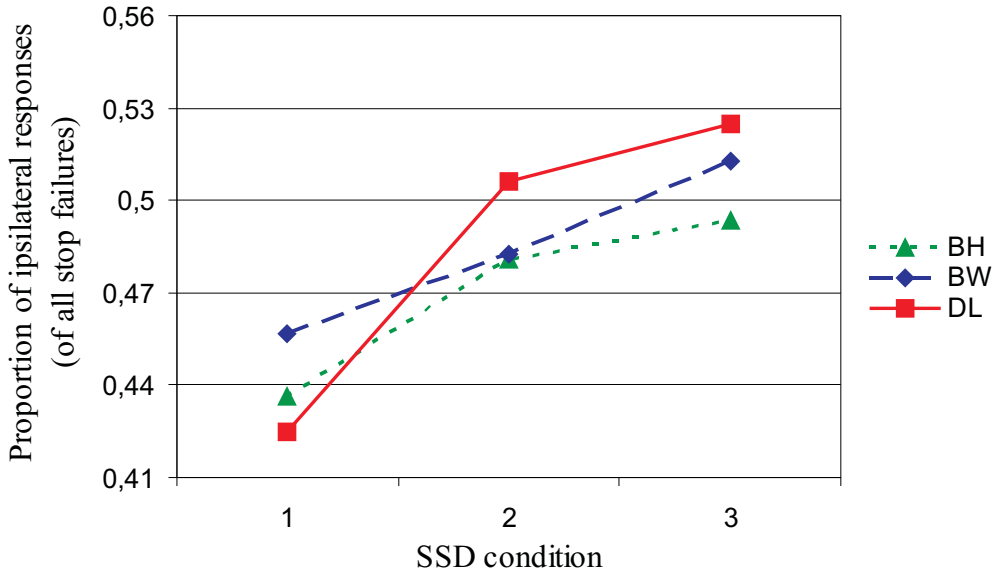


Figure 3.7: Mean saccadic amplitudes (and standard errors) for each 30 ms time bin for the data from DL (details as in Figure 3.5).



to be generally quite effective, ipsilateral presentation increased the chance of successful inhibition of the saccade compared to contralateral presentation. When the stop signal appeared later, producing more stop failures, this difference was cancelled out (BH) or even reversed (BW, DL).

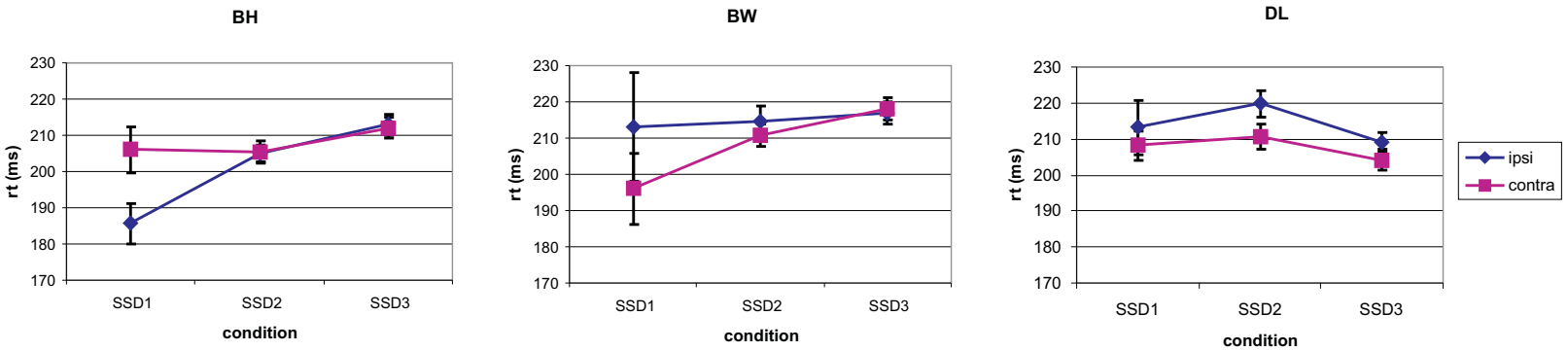
Figure 3.8: The proportion of ipsilateral saccades of the total stop failures.



Spatial Position Effects on Saccadic Reaction Times

Each graph in Figure 3.9 shows the mean RTs of one participant on the stop failure trials, grouped by delay and laterality condition. For participant BH, mean latencies on ipsi- and contralateral trials were practically identical in the two latter delay conditions, whereas for SSD1 ipsilateral responses were faster than contralateral ones. She showed significant overall differences in mean RT between the laterality and delay groups (p <0.001, one-way ANOVA). Post-hoc tests revealed that the origin of this significance was the latency reduction on SSD1 ipsilateral responses relative to ipsi- and

Figure 3.9: Ipsi- and contralateral saccadic reaction times on stop failure trials. Error bars indicate ± 1 standard error.

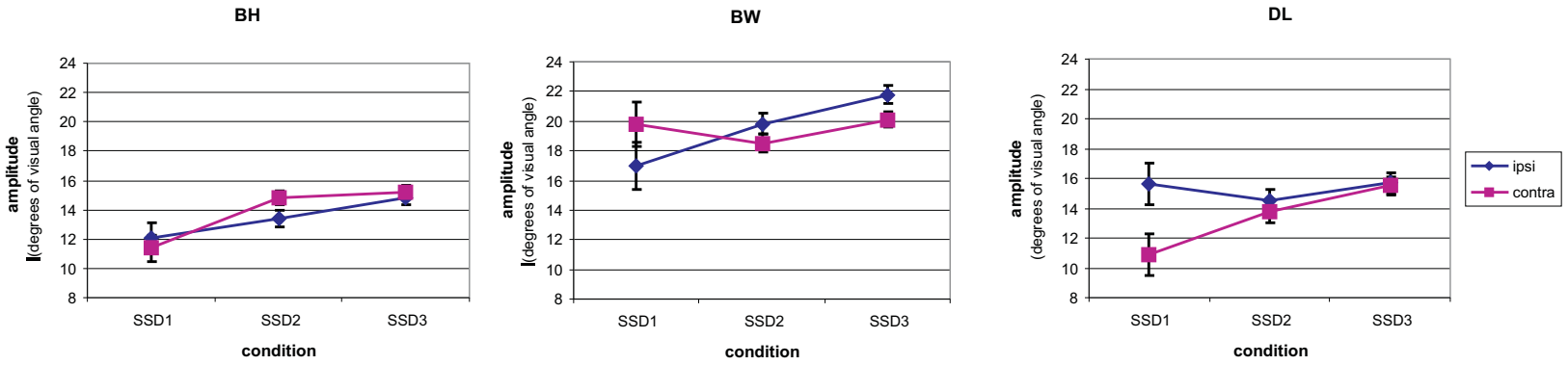


contralateral SSD2 (p <0.05, Tukey-HSD for both comparisons) as well as to ipsi- and contralateral SSD3 responses (p <0.001 for both comparisons). For BW the pattern was virtually the reverse. Again, differences between ipsi- and contralateral means were negligible on SSDs 2 and 3, but for this participant the contralateral responses were faster than the ipsilateral ones in the SSD1 condition. An ANOVA found no significant differences in the data. Finally, the data from participant DL showed slightly faster responses to contra- compared with ipsilateral stimulus configurations for all three delay conditions. An ANOVA revealed significant differences between delay and laterality conditions (p <0.05), and post-hoc testing found the source of the significance to be the comparison between SSD2 ipsilateral and SSD3 contralateral responses (p <0.05).

Spatial Position Effects on Saccadic Amplitudes

Analogously to the ipsi- and contralateral presentation of the RTs above, Figure 3.10 shows the mean saccadic amplitude on each delay and laterality condition grouped by participant. In the first delay condition BH performed slightly shorter saccades on trials with contralateral rather than ipsilateral stimulus configurations, whereas this relation was reversed in the two latter delay conditions. The overall amplitude differences in her data proved to be significant (p <0.001, one-way ANOVA), but post-hoc tests (Tukey-HSD) showed that all within-delay condition ipsi-contralateral comparisons were nonsignificant. Significant differences in the amplitudes were found between responses on SSD1 contralateral trials and SSD2 contralateral (p <0.05), SSD3 ipsilateral (p <0.05), and SSD3 contralateral trials (p <0.01). Also in the data from BW there was a reversal of the ordering of ipsi- and contralateral mean saccadic amplitudes across delay conditions. Here, ipsilateral responses were shorter than contralateral ones in the SSD1 condition, but longer in the other conditions. Again, the overall differences were highly significant (p <0.001, one-way ANOVA), but post-hoc comparisons within each delay condition showed no significant effects. However, ipsilateral re-

Figure 3.10: Ipsi- and contralateral saccadic amplitudes on stop failure trials. Error bars indicate ± 1 standard error.



sponses on the SSD3 condition were significantly larger than either SSD1 ipsilateral responses (p <0.05) or SSD2 contralateral responses (p <0.01). DL's responses on the contralateral SSD1 trials were smaller than those on the corresponding ipsilateral trials, as well as those of the other trial categories. The source of the significant outcome of the ANOVA (p <0.05) was the significantly smaller saccades performed in the SSD1 contralateral condition compared to both SSD3 conditions (p <0.05 for both comparisons).

3.2.6 Stop Signal Processing Times

The quite varied SSDs needed to achieve similar levels of inhibition success across participants suggested that they had very different levels of inhibition proficiency. The mean SSPT for each participant was estimated based on the race model by averaging across delays (see Band et al., 2003). The estimates of the mean duration of the stop process in this study were 82 ms for BH (standard error=3.5), 165 ms for BW (8.7), and 117 ms for DL (8.2).

3.3 Discussion

After having showed in the first experiment that a tactile stop signal can be used to inhibit saccades to visual stimuli, the present experiment extended the support for modality-independence by successfully reversing the roles of the stimuli. Participants were required to respond to tactile stimulation of either hand by performing a saccade towards the stimulated hand, but to inhibit this movement if a visual stimulus was additionally presented at one of three delays. Two visual stimuli, one to the right and one to the left of centre, were used to investigate whether ipsi- and contralateral presentation of the go and stop stimuli had an effect on performance. To achieve adequate inhibition success on stop trials, one of the participants in this experiment needed some of the stop signals to be presented before the go signal, a situation previously unseen in the stop signal literature. All participants showed higher

inhibition success on trials with earlier stop signals, especially if the stop stimulus was presented on the same side as the go stimulus. Comparing go responses before and during the stop signal experiment revealed that two of the participants significantly changed their response speed in the context of the stop experiment. The data obtained were tested against predictions of the Logan-Cowan race model of inhibition.

The predicted pattern of shorter mean RTs on the three stop failure conditions than on go control trials was found. Comparisons across delay conditions also supported, with one exception, the expectation of shorter delays between go and stop signals producing larger reductions in the mean RTs. Participant DL was unexpectedly fast on the last delay condition which should produce similar response latencies to the ones in the go control condition. DL also slowed down slightly, but significantly, on her go responses between the go training and the go trials in the stop experiment. BW's increase in mean RT between the go data sets was highly significant, thus only BH was able to keep her original response speed in the stop experiment rather than responding strategically.

Testing the model predictions of the distributions of RTs revealed very different patterns among the three participants. The data from BH violated neither the upper nor the lower bound of the distribution for any of the three SSD conditions. BW exhibited the frequently reported (e.g., the first experiment of this study; Özyurt et al., 2003) elongated slow tail of the distribution of the stop failures from the first delay condition. Surprisingly, in his data the lower bound was also violated on the other delay conditions, but around the mean of the function or at even shorter latencies. Similarly, the lower bound was strongly violated by the stop failure data from DL in the first and second delay conditions, but at the fast end and the middle of the distribution, respectively. The patterns found across participants in this experiment are likely to be due to a variety of reasons, possibly including the previously suggested interaction between go and stop processes. Owing to the large differences in SSDs for the different participants in this experiment,

as well as to the apparent but unknown procrastination strategies used by the two participants who showed peculiar violations, a satisfactory explanation based on the data obtained in this experiment is unattainable.

Although the go stimuli were presented at 10 degrees of visual angle to the left or to the right of the midline, the mean responses on all go and stop conditions for all three participants were larger than this. The fact that the hands of the participant covered the stimulators may have influenced the judgement of both the stimulus location and the finishing point of the eye movement. Note that, as opposed to the first experiment of the study, the target was not visible to the participants in this experiment. Additionally, spatial accuracy was not stressed in the instructions.

Like the RTs, the amplitudes of the saccades were also affected by the presentation of the stop signal. The mean amplitude in nearly all delay conditions for all participants was smaller than that in the go control condition. Further, for all participants the smallest mean amplitude was found in the first delay condition, whereas the SSD3 mean amplitudes were close to the go control means. Thus, despite the larger variability in saccade sizes in this experiment compared to those in the first experiment reported in this study, an almost identical pattern of reduction in saccadic amplitudes was found. An early stop signal was not only more likely to induce successful inhibition than a late one, but also had a larger effect on the amplitude of the saccade when inhibition was unsuccessful.

Inspecting the amplitude effects relative to the duration of concurrent go and stop processing revealed that all participants produced increasingly smaller movements with longer concurrent processing. The pattern and slope of the effect was remarkably similar for those stop failure trials in which the go and stop signals occurred on the same side and for those in which they were presented on different sides. Interestingly, similar reductions in size with longer processing times were also seen in most of the go data sets collected, although the effects were not as substantial as in the stop failure data. If the hypometric amplitudes of the stop failure responses were indeed due to

increasing effects of go and stop process interaction, as suggested by Özyurt et al. (2003), an additional mechanism is required to explain the effects on go trials. In general, larger saccades tend to have similar or longer latencies compared to smaller (<10–15°) saccades (e.g., Fuller, 1996; Darrien et al., 2001), whereas longer latencies were here associated with smaller movements. One possible explanation concerns the previously addressed matter of the generally large sizes of the responses in this experiment. On those trials in which participants responded more slowly they may have had more time to calculate and program the saccade, in which case the resulting movement would be closer to the target, which was always at 10 degrees of visual angle to the left or right. Thus, more carefully programmed and executed slower go saccades would be smaller than the average, hypermetric, go saccade.

Özyurt and colleagues (2003) found ipsilateral stop signals to be less effective than contralateral ones, and that stop failure responses on ipsilateral trials had faster mean RTs. They suggested that this was due to the stop signal having two different effects on the go processing: a stimulus driven facilitatory interaction with the go signal, and the task-specific inhibitory impact which contradicts the go signal. Despite using several SSDs, the authors did not test whether these effects were present across the delay conditions. In the present experiment, early stop signals proved to be more effective if they were presented on the same side as the go signal, relative to presentation on the opposite side. However, all participants showed a trend towards equal effectiveness or even worse inhibition performance with ipsilateral presentation when the stop signals occurred later. Considering the large differences between participants in the SSDs used, and assuming that the peripheral processing times of the visual stimuli did not vary significantly across participants, it is unlikely that these effects are related to early intersensory facilitation.

Different patterns of laterality effects on RTs and saccadic amplitudes were found for each participant. The faster responses on ipsilateral stop trials reported by Özyurt et al. were only present in the data from BH, and only in the

first delay condition. On her other conditions, and on all three conditions for the other participants, stop failure saccades under ipsilateral conditions were marginally slower than or equally fast as contralateral saccades. Similarly, laterality effects on the amplitudes across delay conditions and participants were small and inconclusive.

The estimated SSPTs confirmed the differences in inhibitory control between the participants. BH was extremely good at inhibiting and had a good chance to stop her response even when the stop signal occurred quite late. In contrast, BW showed a considerable reluctance to inhibit (which was not due to deficits in motivation or in understanding the task), and his SSPT was quite long. However, the range of SSPTs obtained was in line with previous results from stop signal experiments using saccadic responses (e.g., Asrress & Carpenter, 2001; Özyurt et al., 2003).

In summary, visual stimuli were used successfully, but with large variations in effectiveness across the three participants, in inhibiting saccadic responses to tactile stimuli. The efficacy of the stop signal was mainly dependent on the time it was presented, with early stop signals being more effective. Additionally, these early signals were more compelling when they were presented on the same side as the go stimulus. Two of the participants slowed down their responses on the go trials in the context of the stop experiment. In the data from the stop experiment itself, mean RTs on almost all stop condition data sets were faster than the mean go RTs, as predicted by the Logan-Cowan race model. However, the mean amplitudes from the conditions similarly showed generally smaller amplitudes on stop failure than on go trials, indicating a possible interaction of go and stop processing. Different patterns of violations of the race model were found for two of the participants. Thus, several effects present in the RT and amplitude data could not be described adequately by the race model. Further, more research is needed into the effects of spatial position of the stimuli, as well as into the possible interactions of spatial position and stimulus modality.

4

Evoked Responses to Visual-Tactile Stop Signal Inhibition

In this experiment tactile and visual stimuli were used to investigate voluntary inhibition of manual responses. EEG correlates of successful and failed inhibition under different levels of urgency to inhibit were analysed, and the stop signal equivalents of the no-go specific N2 and P3 enhancements studied. The experiment was conducted during a stay as visiting researcher at the Laboratory of Computational Engineering at Helsinki University of Technology in Espoo, Finland.

4.1 Method

4.1.1 Design

The experiment examined the ability of participants to inhibit a well practiced finger lift response to a tactile stimulus when a visual stimulus requiring

inhibition was presented. On 75% of trials (the go trials) a weak electric pulse was presented to the palm of either hand, and the participant was required to respond as quickly as possible by lifting the index finger of the stimulated hand. On the remaining 25% of trials (the stop trials) a visual stimulus was additionally presented, overruling the go command of the tactile stimulus and requiring inhibition of any movement. This stop signal was presented with three different delays for each participant, to produce three distinct probabilities of successful inhibition. Possible procrastination strategies of participants were discouraged and scrutinized. Participants' reaction times on the go trials and the failed inhibition trials were collected and compared with predictions made by the Logan-Cowan race model. Event-related potentials (ERPs) were also recorded on go trials and on failed and successful inhibition trials at 32 scalp sites over a number of sessions.

4.1.2 Participants

Three healthy, right-handed participants were tested, one female (MK, age 21 years) and two males (AN, 24; AW, 23). All had normal or corrected-to-normal vision, and all gave their informed consent to participation in the study. Participants were compensated in money paid out after the last session according to the total number of hours they contributed.

4.1.3 Stimulus Presentation

The experiment took place in a dimly lit, electrically shielded and sound attenuated room. The participant was seated in a comfortable armchair with hands resting on a board supported on the armrests of the chair. The palm of each hand was placed on a plastic case containing bipolar electrodes which provided the tactile go stimuli. The electrical pulses, produced and presented using a Grass Telefactor (West Warwick, RI) S88 stimulator, SIU8T isolation units, and CCU 1A constant current units, had a duration of 0.1 ms.

Their intensities were individually adjusted to be clearly perceptible but not unpleasant, and to allow the participant to keep the hands relaxed. The visual stop stimulus was a red exclamation mark (5° in height) presented at the centre of a monitor 1.5 m in front of the participant. A white dot (0.8° in diameter) was also presented at the centre of the monitor and served as the fixation point. Stimulus presentation and RT recording were done with Presentation software (Neurobehavioral Systems, Albany, CA).

4.1.4 Procedure

In the first session the participant was presented with the different stimuli both in separate blocks and combined into stimulus blocks similar to the ones used later for the actual experiment. Participants were only required to attend to the stimuli and to assist in determining a suitable intensity for the electrical pulse stimuli. ERP responses to the stimulus presentation were collected for later comparison with trials on which stimuli signified different tasks the participants should fulfil. After the first EEG session, participants practiced the go and stop tasks without EEG measurement over a number of trials until their performance stabilised. First, the go task was practiced. Each go practice block contained 72 trials, with the order of presentation of trials randomized within the block. Each trial started with the presentation of the fixation point for 999 ms followed by a variable interval (3 equiprobable durations, range: 500–1487 ms). Then either of the left and right electrical pulses was presented. The participant was required to focus on the fixation point and respond to the pulse as rapidly as possible by lifting the index finger of the stimulated hand slightly. Right and left stimulations were equiprobable. Participants needed one or one and a half sessions of go training to familiarise themselves with the task and increase their response speed until it stabilised at some mean RT level. For the stop task, one quarter of the go trials were changed into stop trials by additionally presenting the visual stop signal (duration: 492 ms) at one of three equiprobable delays. Now the task was to keep responding as quickly as possible to the go signals, but to

try to inhibit the response when a stop signal was perceived. Participants were informed that this would only be possible to achieve on some stop trials, and that they should not slow down responding in expectance of a stop signal. Participants practiced the stop task over a number of sessions, during which the delays of the stop signals relative to the go signal were adjusted in accordance with performance. As a result of this stop task training, three individual stop signal delays were determined for each participant, generating three distinct levels of probability of successful inhibition. The delays used ranged from -36 to 71 ms, with negative values indicating that the stop signal was presented before the signal. Finally, participants completed several sessions of the stop task with the defined stop signal delays and with recording of RT and ERP data.

4.1.5 Data Recording and Analysis

Finger lifts were recorded using two light gates which were taped to the board on each side of the index fingers of the participant. The cables were positioned just above the finger when it was relaxed, so that a small upward movement intercepted the beam and caused recording of the movement. Good operation of the gates was ensured through online monitoring during all stages of the training and experiment blocks. A movement was accepted when it was performed by the appropriate hand and within 70 to 500 ms after the go stimulus. The accuracy of the RT measuring was 1000 Hz.

ERP data was recorded using an electrode cap (BrainCap, Brainproducts, Munich, Germany) with 30 silver/silver chloride electrodes placed at FP1, FP2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T7, T8, P7, P8, Fz, Cz, Pz, FC1, FC2, CP1, CP2, FC5, FC6, CP5, CP6, TP9, TP10. The online reference was at FCz and the data were re-referenced off-line to the tip of the nose. The impedance of the electrodes was kept below 10 kΩ. Vertical and horizontal eye movements were monitored by measuring the electro-oculogram (EOG) from two electrodes placed at the outer canthi of the eyes.

The signal was amplified and filtered with a high-frequency cut-off at 100 Hz, digitised at a sampling rate of 250 Hz, and stored for off-line analysis. The data were then segmented into epochs of 700 ms, starting 200 ms before the go stimulus. The segments were filtered at 1–40 Hz, grouped based on trial type and outcome, and epochs containing EEG or EOG amplitudes exceeding $\pm$ 75 μV were excluded. Finally, the segments were baseline corrected using the first 100 ms of the segment as the baseline interval, and the data in each group was averaged within subjects.

For the statistical analysis of the ERP data, the peaks of the deflections of interest were compared between conditions but within participants. For each condition, the N2 and the P3 peaks were defined as described in the Results section. To quantify the amplitudes and the latencies of the peaks, the trials in the raw data from each condition were arranged into five commensurate groups. For each group, the mean amplitude across trials at each time point in a 32 ms window centred around each peak was calculated. Of these mean amplitudes, the largest absolute value was selected as the group maximum. Thus, for each condition, five sub-averages of the amplitude (the maxima from the five groups) were obtained. Similarly, five sub-averages of the latency were generated for each condition by recording the time point each amplitude maximum occurred at. As these latency sub-averages were determined by the amplitude sub-averages and not independently, comparisons of the peaks across conditions were made by performing multivariate analyses of variance (MANOVAs) with condition as the independent variable and amplitude and latency as dependent variables.

4.2 Results

4.2.1 Behavioural Results

For each participant, the last three blocks of go training after stabilisation of RTs were used as a benchmark for the go performance. For all data in

the RT analysis, a go trial was accepted when the response was performed by the correct hand within the required time window (70–500 ms after the go stimulus), else it was rejected. On stop trials, the lack of a response was classified as a successful inhibition, an adequate response was labelled a stop failure, and incorrect responses were rejected. From the stop experiment blocks a total of 3511 (AN), 3464 (MK), and 3438 (AW) trials were accepted for the RT analysis. The number of trials in each category for each participant are given in Tables 4.1–4.3, as are the respective lengths of stop signal delay used. Negative values indicate that the stop signal was presented before the go signal.

Table 4.1: Numbers of trials in each category in go training and the stop signal experiment in the RT analysis (participant AW).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *-36 ms*	SSD2 *-9 ms*	SSD3 *71 ms*
responses	207	2544	93	129	231
inhibited	N/A	N/A	206	167	68
rejected	9	13	1	4	1

Table 4.2: Numbers of trials in each category in go training and the stop signal experiment in the RT analysis (participant MK).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *-9 ms*	SSD2 *18 ms*	SSD3 *71 ms*
responses	216	2595	136	170	243
inhibited	N/A	N/A	151	122	47
rejected	0	18	7	2	4

Table 4.3: Numbers of trials in each category in go training and the stop signal experiment in the RT analysis (participant AN).

	Go Training	**Stop Experiment**			
		Go Control	SSD1 *18 ms*	SSD2 *45 ms*	SSD3 *71 ms*
responses	212	2593	122	179	239
inhibited	N/A	N/A	184	127	67
rejected	4	0	0	0	0

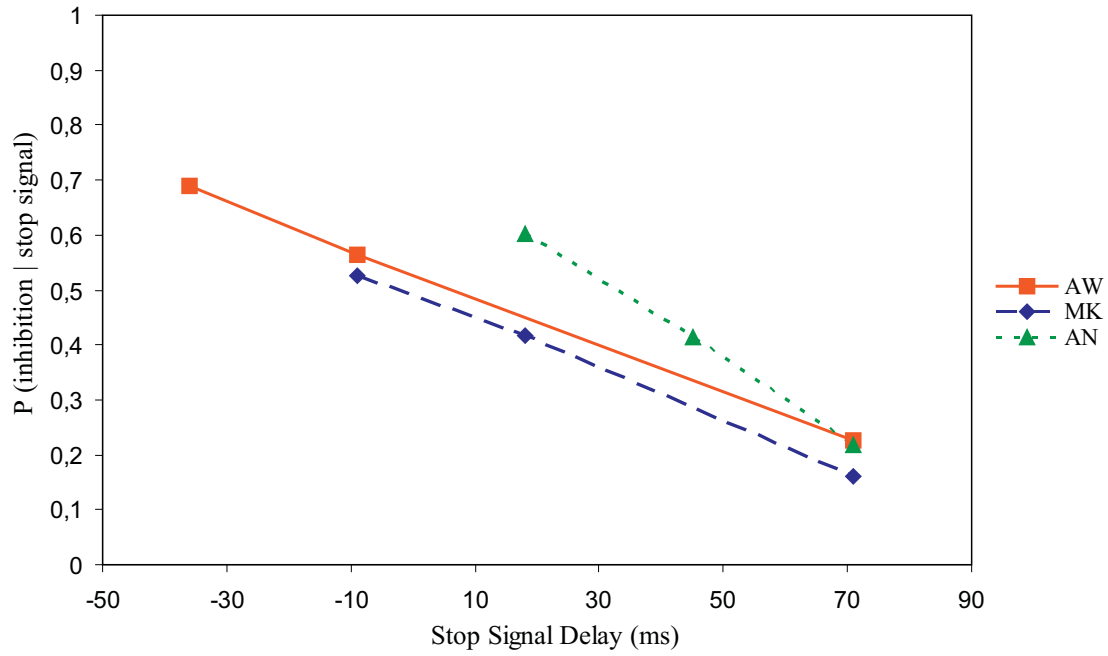
Inhibition Functions

The efficiency of the stop signal was examined by plotting the probability of successful inhibition on a stop trial against the delay between the go and the stop signal. Under the terms of the Logan-Cowan race model, an adequate stop signal should lead to a monotonous rise in the rate of stop failures with increasing delay between presentation of the go and stop signals. As shown in Figure 4.1, the inhibition success rate of the participants in the present experiment varied with the delay used. When the stop signal came early (for AW and MK earlier than the go signal) participants could inhibit on 53–69% of trials, whereas late presentations permitted for only 16–23% success. The slopes of the functions were similar for AW and MK, while AN's steeper slope implies a higher sensitivity to differences in stop signal timing.

Reaction Times

The mean RTs of the participants in the go training condition and the different stop experiment conditions are presented in Table 4.4. All participants showed small changes in their mean RT on go trials between the go training and the context of the stop experiment, and larger changes in mean RT across the go and stop conditions in the stop experiment. The Logan-Cowan race model predicts that stop failure distributions should have faster mean RTs than those of the associate go distribution. Further, the lower the probabil-

Figure 4.1: The inhibition functions of the three participants.



ity of stop failure, the shorter should the accompanying mean RTs be. This pattern was produced by AW, whereas only small differences among mean RTs were found in the stop data from AN. MK's data fits the prediction well except for the slightly slower mean RT in the first compared to the second delay condition.

Comparing the mean go RTs between training and stop experiment showed that MK got somewhat faster, AN slightly slower, and that AW produced virtually the same latencies for these two conditions. Of these, only the RT difference for AN was statistically significant (p <0.05, two-tailed t-test). The stop experiment RT data sets from the participants were subjected to one-way ANOVAs which revealed highly significant differences between the conditions for all three participants (p <0.001 for each participant). Tukey's HSD post-hoc test was applied in further analyses.

Table 4.4: Mean RTs (and standard errors) of the three participants in each condition and across the stop conditions (ms)

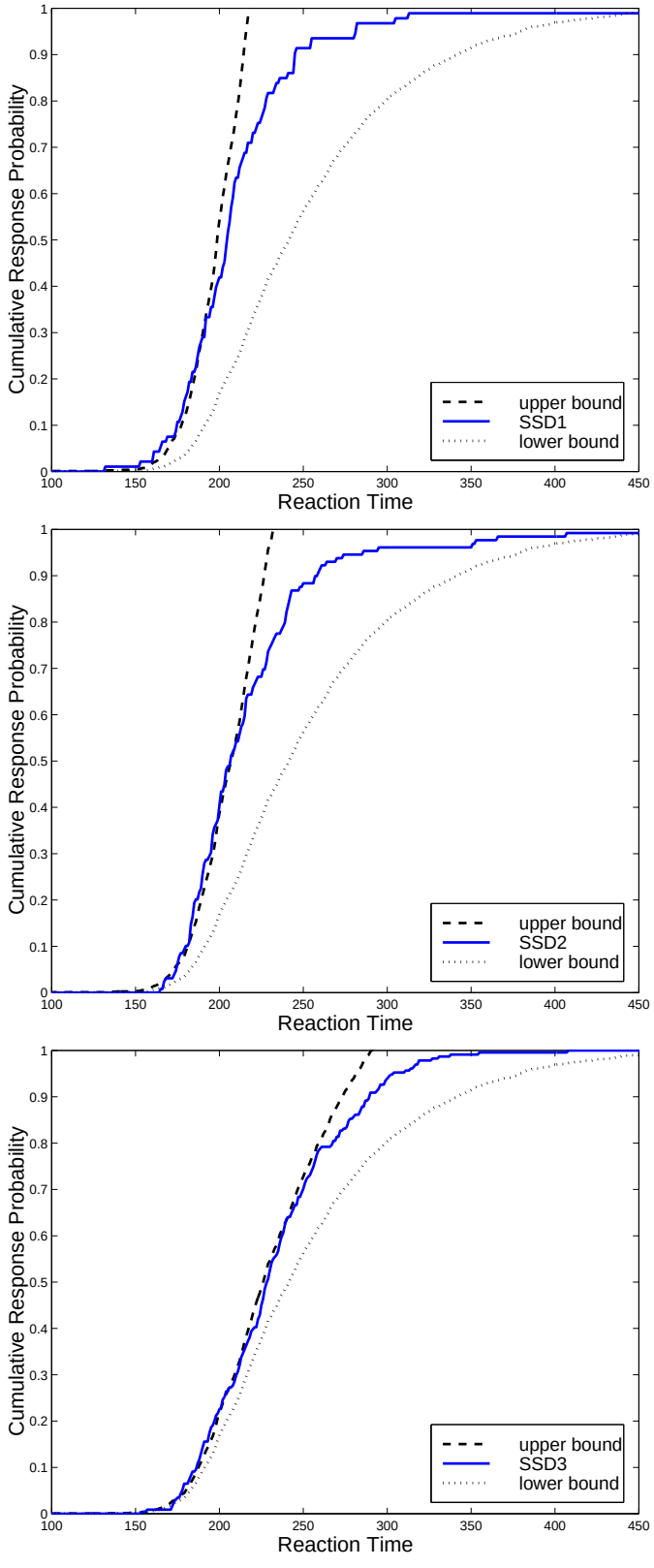
		Stop Experiment				
	Go Training	Go Control	SSD1	SSD2	SSD3	All 3 Delays
AW	257 (3.5)	256 (1.2)	211 (4.3)	217 (3.9)	234 (2.7)	224 (2.0)
MK	268 (3.4)	259 (1.0)	241 (4.5)	238 (3.4)	251 (3.2)	244 (2.1)
AN	247 (3.3)	255 (0.7)	245 (4.0)	243 (3.2)	246 (2.2)	245 (1.7)

The mean RTs from AW in the stop experiment corresponded to the predictions of the race model: The mean RT was significantly lower in each delay condition than in the control condition (p <0.001 for each), and the value increased across the delay conditions. Among the delay conditions the only significant difference for AW was that of slower SSD3 responses compared with SSD1 responses.

For MK, the two later delay conditions fit the expected pattern, whereas the first delay condition produced a slightly slower mean RT than the second. The statistical analysis did, however, support the model predictions, as the only significant differences were those between go control and SSD1 (p <0.01), and SSD2 (p <0.001), respectively.

Also in the case of AN the SSD1 responses were slightly slower than expected, while other mean RTs fit the predictions. The differences between the delay conditions in his data were minimal. Responses in the go condition were significantly slower than SSD2 responses (p <0.01) and SSD3 responses, whereas the comparison with SSD1 responses did not show a significant difference (p =0.056). Finally, as predicted by the race model, the total responses on the stop trials were significantly faster than those in the go control condition (p <0.001 for each participant, two-tailed t-tests).

Figure 4.2: Distribution inequality tests for the reaction times from the three delay conditions (participant AW).



4.2.2 Distribution Inequality Tests

To further explore the responses made under the different conditions in the stop experiment, the individual RTs were considered. The response times from each delay condition were plotted against the cumulative distributions of the probability of a stop failure response, together with the upper and lower bounds specified by the race model (see the Introduction). Figure 4.2 shows the cumulative distribution functions of the RTs from the different delay conditions for AW, Figure 4.3 those for MK, and Figure 4.4 those for AN. In each graph the solid trace corresponds to the responses on one delay condition for the respective participant. The dotted trace indicates the lower bound, and the dashed trace the upper bound.

Previous studies which also employed participant training to avoid procrastination strategies found violations of the lower bound for the slowest 10–20% of responses for some of the participants, mostly in conditions with short SSDs (e.g., Özyurt et al., 2003; see also the first experiment of this study). The graphs depicting the data from the present experiment discloses several minimal violations of the upper and lower bounds of the distribution functions. The data from AW contained no violations of the lower bound, ie. the solid trace had lower RT values than the dotted trace throughout the function. The upper bound was subject to minimal violations in all three conditions by responses with latencies up to about 200 ms. MK produced a small violation of the lower bound in the expected position at the slowest responses in the first delay condition. In addition, the upper bound was violated at a fraction of the short latencies for all three delay conditions. Finally, both findings were also present in the data from AN. The lower bound was violated at slow latencies in both the SSD1 and the SSD2 conditions, and the violation of the upper bound at slow responses was present in all delay conditions

Figure 4.3: Distribution inequality tests for the reaction times from the three delay conditions (participant MK).

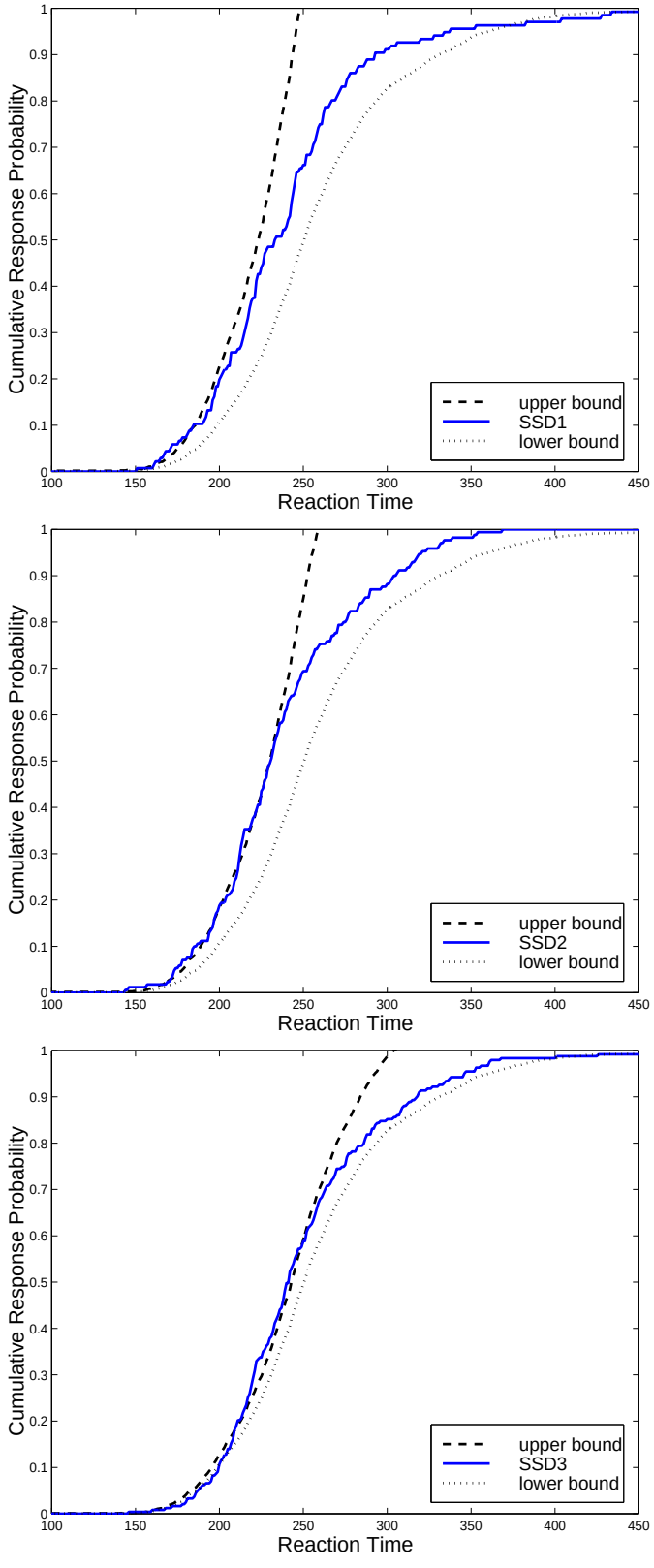
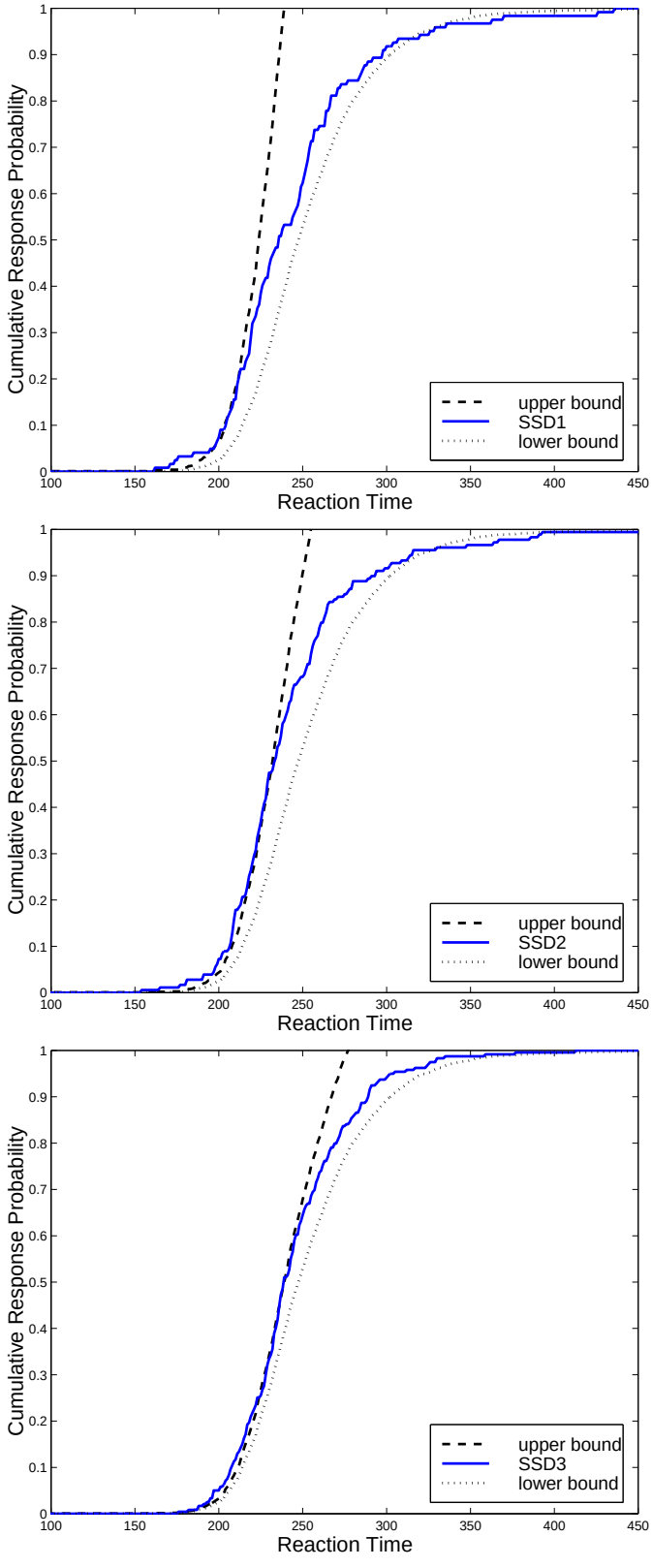


Figure 4.4: Distribution inequality tests for the reaction times from the three delay conditions (participant AN).



4.2.3 Stop Signal Processing Times

As in the previous experiments in this study, the SSPTs were estimated based on the race model assumptions and the RT distributions obtained. Estimates for SSPTs were 209 ms (standard error=11.9) for participant AW, 226 ms (5.8) for MK, and 199 ms (4.5) for AN. These are in line with SSPTs from other experiments with manual responses and using only visual or visual and auditory stimuli (for a review, see Band, 1997), but longer than estimates from saccadic tasks such as the first two experiments reported in this study.

4.3 Event-Related Potentials

For the analysis and classification of the EEG data, the same requirements were used as in the RT analysis described above. Additionally, trials with eye movements or other artefacts were excluded as described in the Method section. The numbers of trials from the stop experiment fulfilling these criteria were 3435 (AW), 2482 (MK), and 1962 (AN). Table 4.5 shows the accepted trials organized by category and participant.

Table 4.5: Numbers of trials from the stop signal experiment included in the ERP analysis for the three participants

		Go Control	**SSD1**	**SSD2**	**SSD3**
AW	responses	2538	91	131	231
	inhibited	N/A	205	169	70
MK	responses	1840	102	124	166
	inhibited	N/A	118	93	39
AN	responses	1415	73	105	138
	inhibited	N/A	109	78	44

4.3.1 Comparisons of Go with Successful and Failed Inhibition Trials

Figure 4.5 shows mean ERPs at electrode positions Cz, Fz, and O1 on the go trials, as well as on the stop failure trials and the successful inhibition trials, organized into columns per participant. In each condition the data were collapsed across left and right go stimulus presentations. Additionally, for both failed and successful inhibitions, data from the three different delay conditions were pooled. The mean RTs measured on the go and stop failure trials are also shown in each graphs. On all trials the go stimulus was presented at 0 ms, the vertex in each graph.

The top leftmost graph shows the ERPs of participant AW at Cz. The go condition average (solid trace) shows a negative potential starting at around 120 ms and peaking at around 190 ms, then gradually turning positive as the response was executed (the solid vertical line shown below the ERP data denotes the mean RT for this condition). This pattern is often recorded at central electrodes in tasks related to preparation and execution of manual responses (for a review, see Altenmüller & Gerloff, 1998). For the stop failure trials (dotted trace, mean RT indicated by dotted vertical line), the pattern is the same until the negative peak of the preparatory potential. Thereafter, the stop failure wave remains increasingly negative until about 325 ms, when it peaks and slowly turns positive. The subsequent positivity is larger than that on the go trials and shows a more pronounced peak at about 450 ms. Finally, successful inhibition on stop trials (dashed trace) also exhibits a distinctive negative deflection followed by a large and distinct positivity. The wave breaks away from the go ERP at an earlier point than the stop inhibition wave, at around 150 ms. Further, the negativity grows larger than in the other conditions and the turn into the subsequent positive deflection is steeper. The positivity peaks earlier and is larger than on the stop failure average.

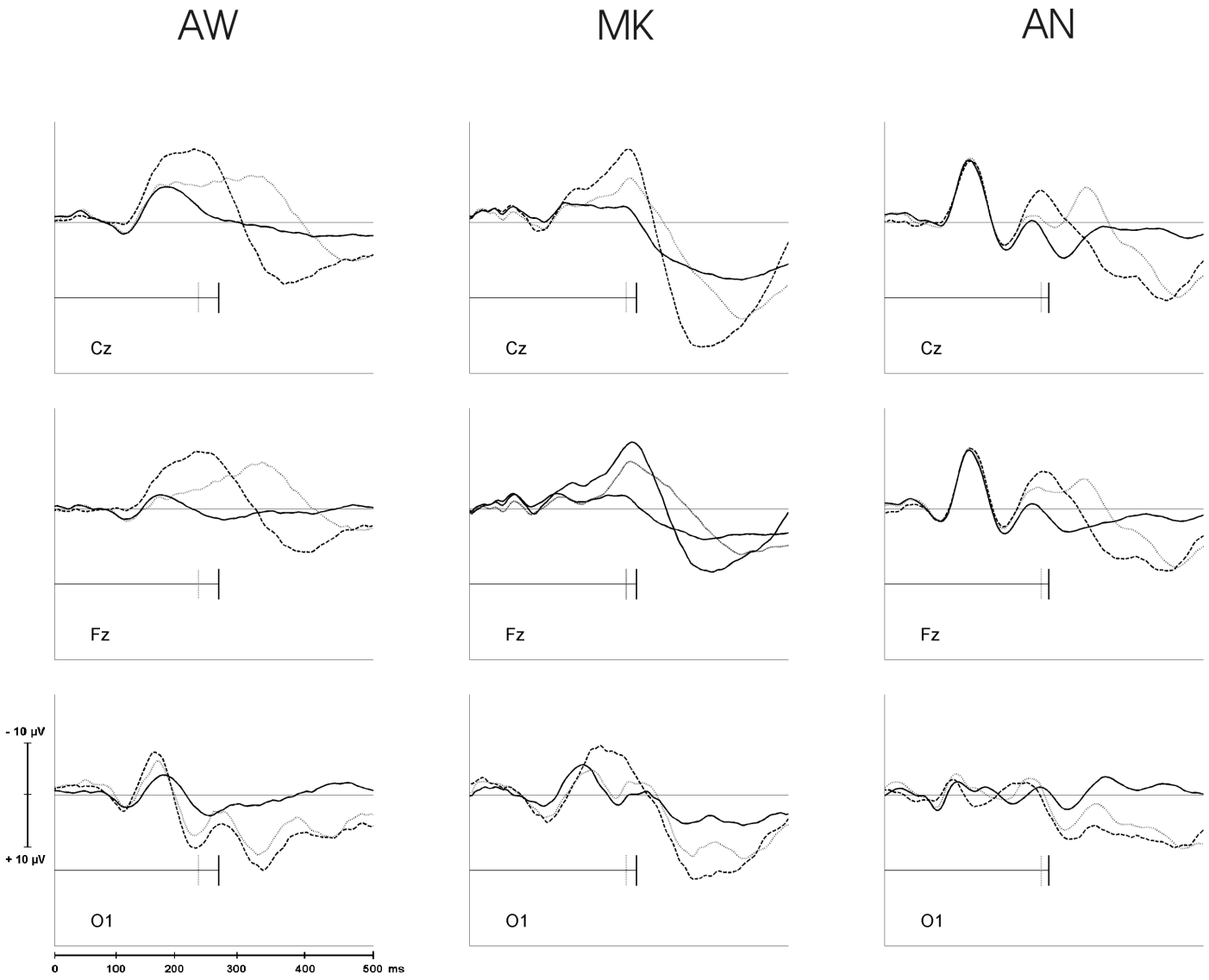


Figure 4.5: Averaged evoked responses for go (solid), all stop failure (dotted), and all successful inhibition trials (dashed) at three electrode sites for the the three participants. Mean RTs for go (solid) and stop failure responses (dotted) are shown below the EEG data.

The patterns found in the ERPs to the different conditions for AW at Cz are also exhibited by MK, as shown in the central upper row graph. The traces from the different conditions separate at about 150–180 ms, after which stop failure and successful inhibition trials show larger negative, followed by larger positive, potentials than those from go trials. Again, both characteristics include distinctive peaks and are more extreme on successful than on failed inhibition trials. For both go and the stop failure ERPs the turn from negative to positive occurs close in time with the mean RTs. In contrast to the data from AW, this turn to positive has almost the same latency for all conditions for MK.

The data from participant AN is presented in the rightmost graph of the upper row. Despite the initial massive evoked response to the go stimulus (present at most electrode sites for AN), similarities with the patterns of the other participants are also seen. After the large stimulus-induced negativity present on all conditions around 100–190 ms, the traces separate and line up as seen in the data from AW and MK. A movement-related negativity in the go trace is surpassed by a larger negative stop failure wave, which in turn is topped by the successful inhibition trace. These negative deflections peak almost at the same point in time, with both RTs also having similar latencies. However, the stop failure trace also shows another later peak before turning positive. Again, the late positive deflections on both failed and successful inhibition trials are larger and more distinctive than that in the go condition.

The ERPs at Fz shown in the central row of Figure 4.5 exhibit strong similarities with those at Cz in the top row. The characteristic negativity is more distinct for AW and MK, starting at about 150 ms, and is also present in AN's data, where it emerges starting around 200 ms, after the initial strong response to the go signal. Likewise, the go data sets show no distinct late positive potential, whereas failed and, in particular, successful stop trials exhibit large positive potentials which peak within the segment.

In contrast to the frontal and central potentials described above, parietal and occipital recordings show much smaller differences among the ERPs from the different conditions. The graphs in the bottom row of Figure 4.5 show the data recorded at O1. On both stop conditions AW shows typical responses to visual stimuli recorded at occipital sites, a small positive dip followed by a sharp negativity and then a more diffuse positivity. His ERPs in the go condition are similar to, but smaller than, those recorded at the frontal and central electrodes. Likewise, MK's data show smaller differences between the conditions at this electrode site than at Cz and Fz. AN's ERPs at this site are generally small, except for a late positive evoked response to the visual stimuli in both stop conditions. ERP effects to the experimental manipulation at other electrode sites not reported here were also negligible compared to those found at frontal and central sites. Thus, the negative deflection starting around 150 ms and the subsequent positive deflection found at these sites on stop trials were the major ERP effects observed. Further, these effects seemed to be larger and occur earlier on successful than on unsuccessful inhibition trials.

To verify the above described amplitude and latency differences between the conditions seen in Figure 4.5, separate MANOVAs were performed for the N2 and the P3 peaks for each participant. For this purpose, the N2 peak was defined as the first negative peak after 150 ms (relative to presentation of the go signal) in the data recorded at Fz. The positive maximum at Cz after the time of the N2 peak was defined as the P3 peak. The MANOVAs, carried out as described in the Methods section, revealed highly significant differences for all participants for both the N2 and P3 data (p <0.001 for each comparison). Post-hoc tests (Tukey-HSD), reported in Table 4.6, showed that most comparisons between conditions were statistically significant. Especially the latency data showed very high levels of significance, particularly for AW and AN. Conversely, the amplitude differences between the successful and failed inhibitions, for both peaks, were either nonsignificant (AW, AN) or significant at a lower level than most other comparisons (MK).

Table 4.6: P-values obtained from post-hoc testing of the amplitude and latency differences in N2 and P3 peaks between go data, successful, and failed inhibition data for each participant.

	Amplitudes		**Latencies**	
	N2	P3	N2	P3
AW				
go vs inhibited	0.001 **	0.009 **	0.000 ***	0.000 ***
go vs failed	0.003 **	0.175 n.s.	0.000 ***	0.000 ***
inhibited vs failed	0.808 n.s.	0.237 n.s.	0.000 ***	0.000 ***
MK				
go vs inhibited	0.000 ***	0.000 ***	0.000 ***	0.000 ***
go vs failed	0.000 ***	0.033 *	0.001 **	0.557 n.s.
inhibited vs failed	0.016 *	0.035 *	0.779 n.s.	0.000 ***
AN				
go vs inhibited	0.000 ***	0.000 ***	0.000 ***	0.000 ***
go vs failed	0.000 ***	0.000 ***	0.000 ***	0.000 ***
inhibited vs failed	0.135 n.s.	0.696 n.s.	0.000 ***	0.002 **

4.3.2 Comparisons Across Delay Conditions

To investigate whether the effects of attempted and executed inhibition discussed above vary across the three delay conditions, the ERPs at Fz and Cz on successful, as well as at Fz on failed, inhibition trials in each condition for each participant are shown in Figure 4.6. The motivation for this was the notion, inherent in the Logan-Cowan race model and investigated by Naito and Matsumura (1994), that the inhibition process (or parts of it) might have an invariant duration, in contrast to the stochastic nature of the go RT. A comparison across delay conditions of the timing of the main inhibition-related features in the ERP data with the timing of the stop signal should give evidence either supporting or rejecting the hypothesis of duration invariance.

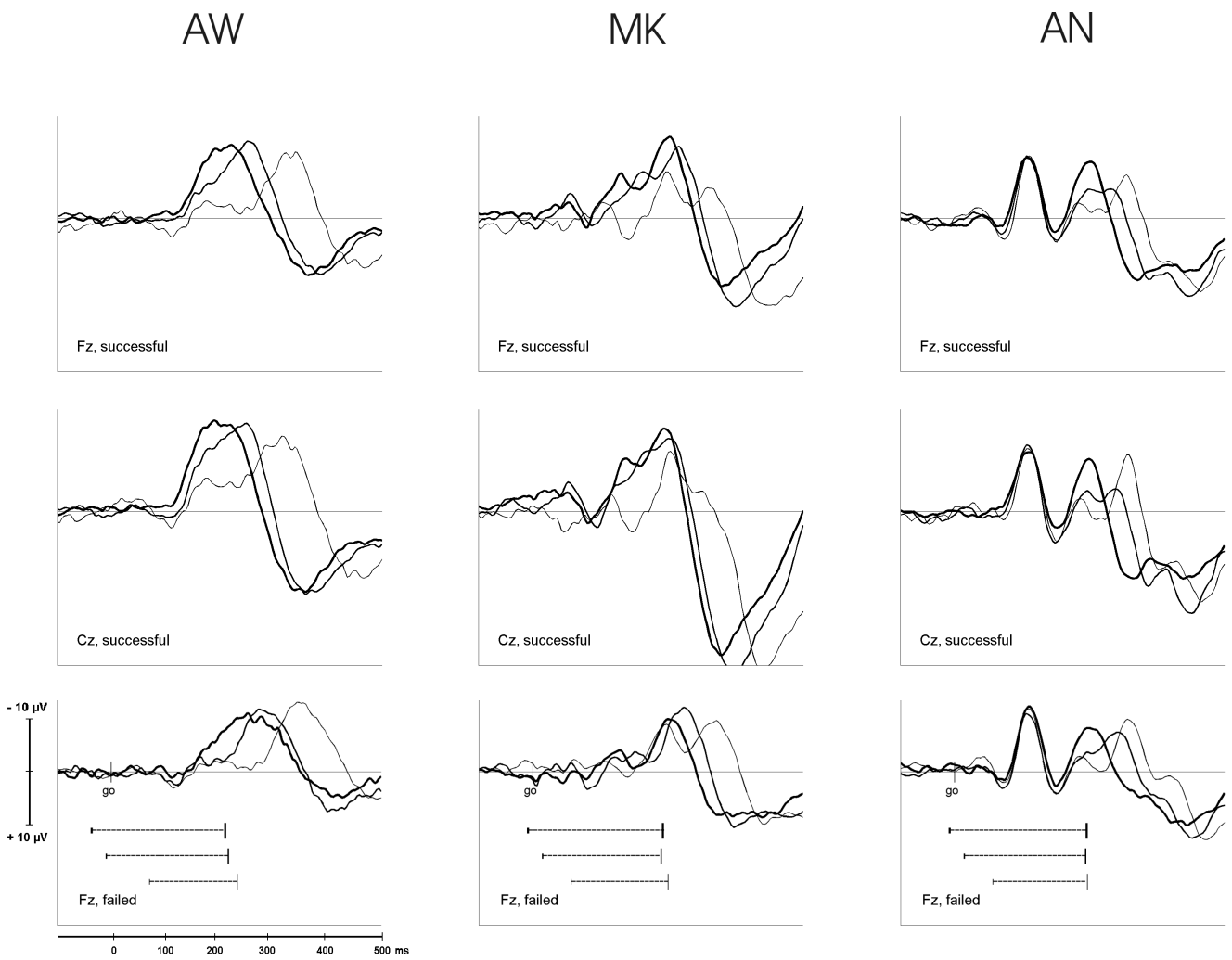


Figure 4.6: The upper rows show the averaged evoked responses on successful inhibition trials at Fz and Cz in the different delay conditions for the three participants. The data from the corresponding stop failure conditions at Fz are shown in the bottom row, including a line indicating the difference between the start of the stop process and the mean RT for each delay condition.

The advantage of first inspecting the ERPs to the successful inhibition trials at Fz and Cz, shown in the upper two rows of Figure 4.6, is that no finger movement was made on these trials. Hence, the data should be free from, or at least to a lesser extent contaminated by, potentials related to motoric programming and execution. The go signal was always presented at 0 ms. The thick line plots the first delay condition, the middle the second, and the thin the third delay condition. For all participants, the peaks of the characteristic ERP features (a pronounced N2 peaking about 240–270 ms after the corresponding stop signal, followed by a pronounced P3 within the segment) were ordered according to SSD. The inhibition-related pattern was produced earlier when the stop signal came earlier, despite the timing of the go signal always being the same.

In order to ensure that the timing patterns seen in the successful inhibition trials in the upper rows of Figure 4.6 were not due to differences in the duration of concurrent go processing, the stop failure ERPs at Fz were similarly plotted. As above, the thick line signifies the SSD1 condition, middle SSD2, and the thin line SSD3. The mean RTs from the stop failure conditions are given by the vertical lines shown below the ERPs, the upper thick line denoting the first, the middle the second, and the lower thin line the third condition. The timing of the go signal is indicated by the vertical mark on the abscissa. As can be seen in the graphs in the bottom row of Figure 4.6, the general patterns were also evident in the data from these conditions, although partly with smaller amplitudes and less defined peaks, despite the simultaneous go processing, which can be assumed to always have started at time 0 (marked with *go* in the graphs). A visual inspection of the distances between the peaks of the ERP data and the mean RTs shows that, while for AW the ordering of the RTs also follows the lengths of delay used, the differences between conditions are much smaller than those seen in the ERPs. For AW and MK, the mean RTs are not ordered according to length of delay. Thus, the influence of stop signal timing on the go processing resulting in the go RT were notably smaller than those on the stop processing as shown in the ERPs.

Table 4.7: P-values obtained from post-hoc testing of the amplitude and latency differences in N2 and P3 peaks between the successful inhibition data from the three delay conditions for each participant.

	Amplitudes		**Latencies**	
	N2	P3	N2	P3
AW				
ssd1 vs ssd2	0.961 n.s.	0.998 n.s.	0.001 **	0.787 n.s.
ssd1 vs ssd3	0.934 n.s.	0.946 n.s.	0.000 ***	0.000 ***
ssd2 vs ssd3	0.811 n.s.	0.926 n.s.	0.000 ***	0.000 ***
MK				
ssd1 vs ssd2	0.798 n.s.	0.249 n.s.	0.047 *	0.001 **
ssd1 vs ssd3	0.001 **	0.311 n.s.	0.000 ***	0.000 ***
ssd2 vs ssd3	0.002 **	0.986 n.s.	0.000 ***	0.000 ***
AN				
ssd1 vs ssd2	0.023 *	0.889 n.s.	0.001 **	0.000 ***
ssd1 vs ssd3	0.390 n.s.	0.959 n.s.	0.000 ***	0.000 ***
ssd2 vs ssd3	0.231 n.s.	0.745 n.s.	0.001 **	0.001 **

The peak amplitude and latency differences between the successful inhibition trials from the three delay conditions of each participant were subjected to statistical analyses analogously to the one described in the previous section. The largest negative deflection in the data recorded at Fz peaking at least 200 ms after the presentation of the stop signal was defined as the N2 peak, and the peak in the following positive deflection at Cz was defined as the P3. For each participant, both N2 and P3 MANOVAs revealed significant differences between the delay conditions (p <0.0001 for each comparison). As is evident from the post-hoc test results shown in Table 4.7, these differences were mainly due to differences in latencies. Amplitude differences in the N2 peaks between the delay conditions rarely reached statistical significance; those in the P3 peaks never did. In contrast, latency differences often proved to be highly significant, and only the difference in P3 timing between the

SSD1 and SSD2 conditions for AW was nonsignificant. Thus, the similarity of the ERP pattern on successful inhibition trials across delay conditions seen in the upper rows of Figure 4.6 was borne out as largely nonsignificant amplitude differences. Correspondingly, the shift in time of the ERP pattern across delay conditions followed the order of the delays and almost always proved highly significant.

If the internal response to the stop signal is indeed time invariant, the differences between ERP peaks across delay conditions confirmed above should be equal to the relevant stop signal timing differences. The latencies of the stop signals and the N2 and P3 peaks, defined as above, are shown in Table 4.8 for the three participants, along with the differences calculated from these. A comparison of the differences reveals that the N2 peaks showed a strong similarity with the delays used for AW, whereas his P3 timing seems less related to the stop signal timing. In contrast, MK's peak latencies followed the stop signal timing fairly closely, especially for the P3. AN's N2 peaks were further apart in time and his P3 peaks closer together than the stop signals, but the general pattern was the same.

To test the invariance hypothesis, the difference in stop signal presentation time between the delay conditions time has to be compensated for. Thus, the length of the relevant delay was subtracted from the N2 and P3 peak latencies of the successful inhibition data. If the differences seen in the latency data were only due to the experimental manipulation, this procedure should eliminate these. The subtracted latencies were subjected to MANOVAs along with the amplitude data, as above. In stark contrast to the original comparisons across delay conditions, only some of the MANOVAs proved statistically significant. For AW, the P3 MANOVA was significant ($p < 0.01$), whereas the test for the N2 was significant for MK ($p < 0.0001$) and for AN ($p < 0.05$). Post-hoc test results are reported, where applicable, in Table 4.9. As the amplitude data were unchanged by the subtraction operation, the post-hoc tests for those comparisons naturally produced identical results to those shown above. For the latency data, only the subtracted P3 peak in

the second delay condition from AW had a significantly different value from the two other conditions, which had virtually identical latencies after delay subtraction. All other latency differences were obliterated by considering the differences in stop signal presentation delay.

Table 4.8: Stop signal delays, ERP peak latencies, and differences in these between delay conditions for the three participants on successful inhibition trials. N2 peaks were determined from data recorded at Fz and P3 peaks from data recorded at Cz. All values are in ms.

AW	SSD1	SSD2	SSD3	SSD2 – SSD1	SSD3 – SSD2
Delay length	-36	-9	71	27	80
N2 peak	224	252	340	28	88
P3 peak	360	360	468	0	108
MK	SSD1	SSD2	SSD3	SSD2 – SSD1	SSD3 – SSD2
Delay length	-9	18	71	27	53
N2 peak	256	272	324	16	52
P3 peak	348	372	424	24	52
AN	SSD1	SSD2	SSD3	SSD2 – SSD1	SSD3 – SSD2
Delay length	18	45	71	27	26
N2 peak	256	288	320	32	32
P3 peak	336	360	380	24	20

Table 4.9: P-values obtained from post-hoc testing of the amplitude and latency differences in N2 and P3 peaks between the successful inhibition data from the three delay conditions, with latencies adjusted for differences in stop signal presentation latency, for each participant.

	Amplitudes		**Latencies**	
	N2	P3	N2	P3
AW				
ssd1 vs ssd2	N/A	0.998 n.s.	N/A	0.006 **
ssd1 vs ssd3	N/A	0.946 n.s.	N/A	0.999 n.s.
ssd2 vs ssd3	N/A	0.926 n.s.	N/A	0.006 **
MK				
ssd1 vs ssd2	0.798 n.s.	N/A	0.051 n.s.	N/A
ssd1 vs ssd3	0.001 **	N/A	0.358 n.s.	N/A
ssd2 vs ssd3	0.002 **	N/A	0.459 n.s.	N/A
AN				
ssd1 vs ssd2	0.023 *	N/A	0.577 n.s.	N/A
ssd1 vs ssd3	0.390 n.s.	N/A	0.111 n.s.	N/A
ssd2 vs ssd3	0.231 n.s.	N/A	0.488 n.s.	N/A

4.4 Discussion

This experiment tested participants' abilities to inhibit a well practiced finger lift response to a tactile stimulus on trials on which an additional visual stimulus was presented. The visual stop stimulus enabled participants to cancel the response to the tactile signal with a certain probability on those trials on which it was required. This probability depended on the relative timing of the imperative and stop stimuli; the earlier the stop signal, the more likely a successful inhibition. The delays required for acquiring probabilities between 0 and 1 in this experiment were extremely short compared to those seen in the stop signal literature. Two out of three participants needed delays

with negative values, ie. where the stop signal was presented before the go signal. The demand for such early stop signals points to the relevance of not only stimulus timing but of the combination of the perspicuity and the timing of both signals. The highly conspicuous electrical pulses used as go stimuli required an exceptionally early presented visual signal to allow for the possibility of inhibition.

4.4.1 Reaction Times

The response time means and distributions were tested against predictions based the Logan-Cowan race model. Participant AN showed effects of context-dependent go processing, in that his mean RT on go trials was significantly longer in the stop experiment compared to go training. The other two participants did not slow down their responses to the go stimulus in expectation of a possible stop signal. The race model predictions of the mean RTs obtained in the the stop experiment were largely supported by the data obtained. All three stop failure conditions for each participant had faster mean RTs than the go control mean RT for that participant. However, the expected ordering of the mean RTs, with faster stop signals producing faster stop failure RTs, was only found for AW. AN and MK produced surprisingly slow responses on the first delay condition. This finding conforms with those reported in the stop signal literature (e.g., Özyurt et al., 2003) as well as with data from the other experiments in this study. Moreover, when present, violations of race model predictions are virtually always found in the condition with the shortest SSD (Logan & Cowan, 1984; Hanes & Carpenter, 1999; Colonius et al, 2001; but see the second experiment of this study). The violations of race model found in the present experiment were extremely small, but included slightly elongated slow tails in the first delay condition for MK and AN, as expected based on the stop signal literature and the mean RTs.

The combined pattern of the delays required for adequate inhibition performance, the fit of the data with race model predictions, and the SSPTs estimated, showed interesting effects. The participant who needed the earliest stop signals to achieve some level of inhibition success, AW, produced no violations of the lower bound set by the race model for the RTs. In comparison, AN required the longest SSDs in this experiment and also showed more evidence of violations of the predictions of the race model, and hence of possible interference between go and stop processing. However, the estimated SSPTs indicated that the differences in inhibition performance between the participants were quite small. Despite needing extremely early stop signals to inhibit, the estimated mean SSPT for AW was only 10 ms longer than that of the best inhibitor AN, and 17 ms shorter than that of MK. Hence, this early presentation of the visual stop stimulus was not an indicator of deficient inhibition, but instead ensured a normal duration of stop processing.

4.4.2 Event-Related Potentials

The reporting of the ERP analysis was focussed on those electrode sites which showed the largest effects of the experimental manipulations. For the comparisons of go, stop failure, and successful inhibition trials these large effects were also contrasted with the ERPs recorded at the occipital O1 electrode. As expected, the stop trial data recorded at this electrode site showed the typical patterns of evoked responses to visual stimuli (with normal individual variations in distinctiveness and magnitude), but not the responses related to the outcome of the trial such as those recorded at Fz and Cz. This was also the case for other parietal and occipital electrodes from which data are not reported here.

In contrast, large amplitude and latency effects of both the experimental manipulation (i.e. go or stop) and participant performance (i.e. stop failure or inhibition) were found in the ERPs at electrode sites Fz and Cz. The N2 and P3 components showed generally highly significant no-go deflections,

with successful inhibitions producing earlier effects, more extreme effects, or both. The sizes and latencies of these effects across delay conditions were compared in the successful inhibition data. These trials should show less interference from motor preparation, and none from motor execution, occurring at different times relative to the stop process in the stop failure trials. The no-go indices of successful inhibition proved significantly different between the delay conditions, but this effect virtually disappeared when the data was synchronised by subtracting the stop signal delays. Thus, the results partially support the conclusions drawn by Naito and Matsumura (1994) from their data: Whereas they suggested an invariant N2 latency and a shorter N2 to P3 peak-to-peak time with increasing stop signal delay, both measures (except for one delay condition for one participant) were largely invariant in the present experiment.

Recent studies have attempted to clarify the connection between the no-go ERPs and performance on stop signal tasks. Van Boxtel et al. (2001) recorded EEG, muscle activity, heart rate deceleration, and respiratory activity in a task which included both stop and no-go stimuli in addition to the majority go stimuli. Among the data which led them to suggest that the N2 is an inhibitory signal was the fact that their calculated LRP, reflecting motor activation, began to diminish when the N2 started to increase. Additionally, they concluded that the amplitude of the N2 deflection was related to inhibitory efficiency, based on the larger amplitudes they recorded for participants categorized as fast inhibitors. Kok et al.(2004) compared the augmented N2 and P3 ERP components produced on successful and unsuccessful stop trials. They found that the P3 peaked earlier on successful inhibitions than on stop failures, and interpreted this as an extension of the race model in terms of showing that the stop process is not invariant. However, they also analysed the topographical distributions of the recordings, and concluded that the successful and unsuccessful P3s were produced by different cortical generators. This was taken to indicate that the P3 reflects the efficiency of inhibitory control as well as inhibition itself.

In stark contrast to these deliberations, other authors have recently attempted to shift the focus in the go/no-go related ERP literature away from the N2 and the P3 and towards earlier components. Filipović et al. (2000) and Yamanaka et al. (2002) have been among those adducing electromyographic evidence which shows that response-related muscle activity often starts before the reported cortical deflections measured by EEG. This would suggest that the decision is already taken at the time of the N2 peak. This notion has been corroborated by measuring neural activity directly from the prefrontal cortices of monkeys during go/no-go performance, setting the essential time window at 100–170 ms poststimulus (e.g., Gemba & Sasaki, 1989). Likewise, techniques directly affecting inhibition performance, such as electrically stimulating the monkey cortex (Sasaki et al., 1989) or applying TMS over the dorsal premotor cortex of humans (e.g., Ro et al., 1997), point to clearly faster decision making processes than those assumed to be coinciding with the N2 peak. Hence, the no-go specific N2 and P3, as investigated in the present experiment, might be reflecting decisional or motor inhibition processes rather than instigating these or being their measurable components.

In summary, in this experiment RTs and ERPs were measured from participants completing a stop signal task with tactile go signals and a visual stop signal. Participants needed extremely early presented stop signals to enable inhibition to a varying extent. They largely managed to avoid response strategies involving speed-accuracy tradeoffs. Comparisons of the RTs with those predicted by the Logan-Cowan race model revealed some small violations at slow responses with early stop signals and at fast responses for all delay conditions. SSPTs were around 200 ms, in line with values found in the stop signal literature for manual tasks. The ERP data showed the expected no-go specific enhancements of the N2 and P3 components at frontocentral electrode sites, with the enhancements being larger and starting earlier on successful than on failed inhibitions. Comparisons of N2 and P3 latencies across the delay conditions supported the proposition of an invariant timing of the N2 relative to the stop signal, but did not corroborate suggestions that

the timing of the P3 varies systematically across conditions. The length of SSPTs, early violations of the race model, as well as early trends towards differences between successful and failed inhibition in the ERP data are all interpreted as that the stop decision is made already before the peak of the N2, probably between 100 and 200 ms after stop stimulus presentation.

5

General Discussion

The main aim of this study was to explore the usage of tactile stimuli in stop signal experiments. In the first experiment the tactile signal acted as the stop signal, in the second and third experiments tactile stimuli applied to both hands of the participants were the imperative go signals. Another aim was to compare the data obtained using these stimulus configurations with results from visual-auditory and visual experiments reported in the stop signal literature, and to test it against the predictions of the Logan-Cowan race model which considers stopping a modality-independent process. As the race model account critically relies on the go and stop processing being context-independent (e.g., Paré & Hanes, 2003; Kok et al., 2004), the RT data were studied particularly closely for presence or absence of possible dependencies between the processes. To monitor for, and if possible avoid, procrastination strategies of the participants, their go RT performance was tested before the stop experiment to obtain a benchmark go response speed. The third aim was to pursue the investigation into the possible effects of the horizontal spatial position of the stop signal, following the mixed results obtained by Özyurt an colleagues (Colonius et al., 2001; Özyurt et al., 2003). Finally, in the third experiment no-go specific ERPs, and in particular the change in these across delay conditions, were investigated.

In each experiment, one or two of the three participants tested did not reach the mean RT level obtained in the go training on the subsequent go control trials in the stop experiment. Participants were aware that the go responding was the primary task, received verbal feedback after blocks with too slow mean go RT, and were tested extensively to give them ample time to improve their go performance. The fact that many still produced significantly slower go responses points to an inherent difficulty of processing two contradictory signals independently, as assumed by the race model. As most previous stop signal studies have not explicitly tested the go task performance without the context of the stop experiment, the data from the participants who slowed their responding was analysed further, despite the obvious effect of the stop experiment on go processing.

The race model predictions of the mean RTs in the stop failure conditions were quite well met in all three experiments. The mean RT across the three delay conditions for each participant were faster than his or her mean go RT. However, the expected ordering of mean RTs across the stop failure conditions, with lower probabilities entailing faster RTs, was not always found. To examine the response times in closer detail each stop failure distribution of the individual RTs was plotted along with the boundaries set by the race model. Violations of these boundaries were found for almost all participants, but these were larger and more systematic for the saccadic data than for the manual responses. Especially the violations found in the first experiment correspond well with those reported in the stop signal literature (e.g., Hanes & Carpenter, 1999; Colonius et al., 2001), while the second experiment produced very varied patterns of violations.

Another difference between the two experiments testing inhibition of saccadic responses was the large variability in both estimated SSPTs and saccadic amplitudes seen in the data from the second experiment. Additionally, error rates (i.e. the proportions of responses in the wrong direction, too fast responses, etc., but not including stop failures) were much higher in the second experiment. The informal comments made by the participants in each were

also indicative of a difference in the two tasks: Whereas the participants in the first experiment found it hard to learn to inhibit the eye movement towards the visual stimulus, those in the second found the go task in itself demanding. Consequently, the saccades performed towards the tactile stimuli in the second experiment were much less accurate than those towards the visual stimuli in the first experiment. Note, however, that the strong go stimulus-response coupling in the first experiment did not lead to faster RTs: The range of mean saccadic RT in the go control condition across participants in both experiments was remarkably small, reaching from 219 to 225 ms.

A possible role of the spatial configuration of the go stimuli in the differences in saccadic accuracy can also not be excluded. In the first experiment, the fixation point was aligned with the visual go stimuli, so that the task of the participant was to perform a saccade to the side of the fixation point. In order not to avoid causing inconvenience to the participants, the position of the tactile stimuli was kept near the body of the participant also in the second experiment. Thus, in that experiment participants were gazing down towards their hands from the fixation point which was now positioned halfway to the visual signals (placed 60 cm to the front as in the first experiment).

The mean SSPTs estimated in the three experiments were in line with those seen in the stop signal literature. The choice of estimation technique was made based on the small number of delay conditions used. With few exceptions, the SSPTs found in the literature suggests that, while inhibition of manual responses regularly takes about 200 ms to implement, the speed of the faster saccadic inhibition is largely dependent on the stimulus modalities used. For example, the SSPTs of auditory stop signals estimated by Colonius et al. (2001) were extremely short, whereas SSPTs to visual stimuli (e.g., by Asrress & Carpenter, 2001) were longer. As those to tactile stop signals in the first experiment reported in this study were intermediate but closer to the visual ones, this mirroring of the peripheral processing times of the stimuli (see, e.g., Todd, 1912) is intriguing. In apparent contrast to the

strong influence of the peripheral processing time suggested here, Cabel et al. (2000) compared visual and auditory stop signals in a saccadic task and estimated SSPTs to be about 90 ms longer for auditory than for visual stimuli. However, their experiment differed from the general stop signal design in that the go stimuli always appeared at the same delay from the time when the participant initiated the trial. In addition, the auditory stop signal was delivered from speaker 2 m above the head of the participants, while visual stimuli were presented on a monitor in front on them. Thus, the difference in the results to other saccadic stop tasks may well have been due to the trial design or spatial features of the stimuli.

Saccadic stopping has been the subject of intensive neurophysiological investigation in the past decade. Single cell recordings from nonhuman primates performing and inhibiting saccades have proved particularly informative. For example, in studying the role of prefrontal executive areas, Hanes and colleagues (e.g., Hanes & Schall, 1995; Hanes et al., 1998) discovered that the frontal eye field (FEF) contains neurons which have activity patterns fitting the outcome of stop trials. Specifically, neurons which increased firing in preparation of a saccade did not respond differently on go and stop failure trials, but their firing rate dropped within the estimated SSPT on successful inhibition trials. Furthermore, activity in fixation neurons in the FEF slowly decreased during saccade preparation, but suddenly increased intensely at the time of the estimated SSPT (Hanes et al., 1998). These results were interpreted as the FEF being the cortical control system for producing and inhibiting saccades. Subsequently, Paré & Hanes (2003) identified neurons with corresponding functions in the superior colliculus, a brainstem structure known to play an important role in saccade production (as well as many other operations such as intersensory interaction, see e.g., Stein & Meredith, 1993).

Based on their primate and human data, Hanes and Carpenter (1999) also extended the Logan-Cowan race model by specifying that the go and stop processes racing independently in a saccadic stop task may be modelled as

two linear rises towards a (not necessarily common) threshold. Some of their data showed similar elongated slow tails of the RT distribution as, for example, the results from the first experiment in this study. They did not consider this to contradict the independence assumption, but suggested that sometimes a stop process winning the race does not cancel the saccade but merely slows it down. However, as pointed out by Özyurt et al. (2003), this account does not explain why conditions with short delays are particularly affected.

Recently, Boucher et al. (2004) compared the performance of different model architectures on prototypical behavioural and neuronal saccadic stopping data. They concluded that neither a pure race model nor a model involving mutual inhibition of the go and stop processes could account for both types of data. Instead, they suggested a two stage model in which the processes first progress independently and then, after some time, interact in the form of mutual inhibition. The specific parameters and neural correlates of this model are yet to be investigated.

The models discussed above all concern the RTs of the responses made and the neural units producing this behaviour. The findings of hypometric saccades are not accounted for, at least not explicitly. Note that Paré and Hanes (2003) reported a small but significant reduction on stop failures compared to the control go saccades performed by their primate subjects. Despite declaring that the reduction in their data was too small to violate the race model, they pointed to hypometric saccades as being one of two indications of interaction between go and stop processes. The other indication they discussed was the systematic SSPT variation with stop signal delay. Considering the data from the present study, this systematic variation was found for all three participants in only one experiment. This was the second experiment, in which all participants showed massive reductions in saccadic amplitude as the duration of simultaneous go and stop processing increased. Thus, these two indicators, as defined by Paré and Hanes were both present in the data from this experiment. In each of the two other experiments reported here,

the systematic variation in SSPTs across delay conditions was shown by only one participant, HB in the first and AW in the third.

The networks controlling inhibition of manual responses are not nearly as well defined as those described above relating to saccadic inhibition. Although imaging and single cell studies have identified several structures involved in inhibition, the causalities and paths of processing are not clear. However, the no-go specific N2 and P3 ERP deflections investigated in the third experiment of this study have been strongly linked to inhibition. Among a large number of studies on this topic, Jodo and Kayama (1992) showed that the amplitude of the no-go N2 was increased in a a group of participants responding under higher time pressure than the other group they tested. Pfefferbaum et al.(1985) observed a no-go N2 although the task which had to be inhibited was not a motor response but a covert operation, such as silent counting. Eimer (1993) found a larger no-go N2 to attended than to unattended stimuli. However, the fact that no-go N2 deflections are only rarely recorded in auditory no-go tasks contradicts the idea that it reflects inhibition.

The infrequent appearance of the auditory no-go effect, together with the effect of attention found by Eimer (1993), clearly distinguishes the no-go N2 from the mismatch negativity, another frontal negativity appearing 100–200 ms post-stimulus on trials with oddball stimuli such as the stop signals. The mismatch negativity is an attention-independent, pre-perceptual change detection mechanism, which signals the change in any physical stimulus parameter of auditory signals (for a review, see Näätänen [1992]). Further, as errors of both omission and commission committed by participants are frequently followed by an error negativity, Falkenstein et al. (1999) hypothesized that this might be a late, unsuccessful no-go N2. However, they found that the no-go N2s and error negativities they recorded had different scalp distributions and showed different changes caused by the experimental manipulations. Falkenstein et al. also concluded that the no-go N2 is probably generated by modality-specific generators, which would explain why the au-

ditory no-go N2 has been so elusive. Supported by single cells studies on visual and auditory no-go potentials by Gemba and Sasaki (1990), this leads to an interesting challenge for the race model of inhibition. Following Logan and Irwin's (2000) comparison of inhibition of saccadic and manual responses to visual stimuli, and their conclusion that these responses are inhibited by different processes operating under similar principles, the disentangling and modelling of the respective effects of response modalities and sensory modalities remain intriguing tasks. As those authors envisaged, explaining these effects may also lead us closer to a general understanding of inhibition, including the locus of it in both neural and processing terms.

In summary, the present experiments showed that tactile stimuli can be used both as go and stop stimuli in a stop signal experiment. RTs were tested against the predictions of the Logan-Cowan race model, which could explain some of the effects found. However, especially the two experiments investigating inhibition of saccadic responses showed several different patterns of violation of the model, including the most frequently reported in the stop signal literature. In addition, the amplitudes of the saccades were affected by the stop signal presence, in contradiction to the assumption of independent processing. The variability in the results on the effects of stop signal spatial position contradicted previous results and warrant further investigation. The no-go N2 and P3 were reliably found on stop trials, and their amplitudes and latencies differed between stop failure and successful inhibitions trials. Though likely to be an effect of inhibition rather than the cause or the manifestation of it, the latencies of the peaks also followed the stop signal presentations closely, and the different delay conditions resulted in no-go peaks with similar amplitudes. Estimated SSPTs were longer for the manual task than for the two saccadic tasks, mirroring the RTs obtained.

6

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Eidesstattliche Erklärung

Ich versichere hiermit, dass ich meine Dissertation *Visual-Tactile Stop Signal Inhibition* ohne unerlaubte Quellen angefertigt und mich keiner anderen als der von mir ausdrücklich bezeichneten Quellen und Hilfen bedient habe. Die Dissertation wurde in der jetzigen oder einer ähnlichen Form noch bei keiner anderen Hochschule eingereicht und hat noch keinen sonstigen Prüfungszwecken gedient.

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Lebenslauf

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