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A psychophysiological analysis of inhibitory motor control in the stop-signal paradigm

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Abstract

We examined two potential inhibitory mechanisms for stopping a motor response. Participants performed a standard visual two-choice task in which visual stop signals and no-go signals were presented on a small proportion of the trials. Psychophysiological measures were taken during task performance to examine the time course of response activation and inhibition. The results were consistent with a horse race model previously proposed to account for data obtained using a stop-signal paradigm. The pattern of psychophysiological responses was similar on stop-signal and no-go trials suggesting that the same mechanism may initiate inhibitory control in both situations. We found a distinct frontal brain wave suggesting that inhibitory motor control is instigated from the frontal cortex. The results are best explained in terms of a single, centrally located inhibition mechanism. Results are discussed in terms of current neurophysiological knowledge.

Keywords: Inhibition; Stop-signal task; Event-related potentials; LRP; N200; Cardiac deceleration

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1. Introduction

Cognitive control mechanisms are needed to coordinate various cognitive processes involved in human task performance. Several models of human information processing accordingly posit cognitive ('executive') control. For instance, Shallice (Norman and Shallice, 1986; Shallice, 1994) hypothesized the existence of a Supervisory Attentional System (SAS), which governs the selection or suppression of schemata, which in turn control the elementary processing that takes place in the execution of a task. Likewise, Meyer and colleagues described an Executive-Process Interactive Control (EPIC) architecture of the human information processing system, in which a central cognitive processor controls perceptual and motor processors (Meyer et al., 1995; Meyer and Kieras, 1997).

Research on stopping motor responses provides a reasonably direct examination of cognitive control. Logan and colleagues initiated a program of research on inhibitory control that made extensive use of a stop-signal paradigm (see Logan, 1994, for an overview). The stop-signal paradigm employs a primary task, typically a visual choice reaction time task. While the participants are engaged in this task they are occasionally presented with a signal, usually a tone, shortly after the respond stimulus. This signal instructs them to withhold their response to the primary, choice task. The stop signal can be presented at various delays after the primary respond stimulus. The chance of stopping the response declines as the delay from the primary respond signal increases. Cognitive control is implicated because stopping is an internally generated act of control changing the current course of action to meet a new goal (Logan, 1994).

Logan and colleagues demonstrated that a horse race model fits the data from the stop-signal paradigm (e.g. Logan and Cowan, 1984). In the horse race model two sets of processes race for completion. The first set controls primary choice reaction time (RT) performance, and is thought to include the processing of the respond stimulus, response choice, and the preparation and execution of the appropriate response. It starts at the onset of the respond stimulus. The second set of processes, which starts at the presentation of the stop signal, controls inhibition, and is thought to consist of stop stimulus processing and response inhibition. These two sets of processes will be referred to as the respond process and the stopping process, respectively. The process that is completed first wins the race and determines whether a response occurs. If the respond process wins, a response is produced despite the stop signal, whereas the response is successfully inhibited when the stopping process wins the race. The horse race model also allows an estimate of inhibition time. Across a variety of tasks, inhibition time has been about 200 ms for young adult participants (Logan and Cowan, 1984).

The horse race model provides an excellent description of behavior in a variety of tasks involving response inhibition, but does not provide an extensive description of the respond and stop mechanisms. A global, perhaps unitary, inhibitory mechanism is suggested by the robustness of the estimates of stopping times across tasks which varied in instructional, stimulus, and response variables. Indeed, most results reported in the stopping literature can be described by a single global inhibitory

mechanism (for reviews see Logan, 1994; Logan and Cowan, 1984). Empirical studies using both performance and psychophysiological strategies have, however, suggested the possibility of two separate stopping processes. A primary aim of the current experiment is to examine these putative processes and the evidence upon which they are based.

1.1. Two mechanisms of inhibitory motor control

Two early studies suggested the existence of two stopping modes—a quick mode for inhibiting all responses, and a slow mode for inhibiting selectively. Riegler (1986; cited in Logan, 1994) presented two stop signals. In one condition, participants had to stop their response when either signal was presented. In another condition, they had to stop their response to only one signal, but not to the other. Inhibition times were longer in selective as compared to nonselective stopping. Another early study (Logan et al., 1986; cited in Logan, 1994) required participants to perform a two-choice and four-choice reaction in which different stimuli were mapped onto different key presses. Again, there were two conditions. In the stop-all condition, participants had to stop their response when the stop signal was presented, irrespective of which key had to be pressed. In the stop-selective task, however, they had to stop the response only when a key press with the right index finger was required; they responded normally whenever another key had to be pressed. Inhibition times were fast in the stop-all condition and hardly altered by whether two or four choices were required in the primary task. Inhibition times were substantially longer in the stop-selective condition than in the stop-all condition, and the difference between stop-all and stop-selective inhibition times was larger in the four-choice task than in the two-choice task. Logan et al. (1986) reasoned that selective stopping was slower than non-selective stopping because it required discrimination, and the duration of the discrimination process was longer the greater the number of alternatives. However, two inhibitory modes do not necessarily imply two inhibitory mechanisms. These results may also be interpreted by assuming a single mechanism that is differentially engaged in the two inhibitory modes. The required additional perceptual discrimination in the selective as compared to the non-selective inhibition mode might have prolonged processing prior to the engagement of the inhibitory mechanism.

Results obtained from stop-change tasks have also been used to distinguish between two inhibition modes. For instance, Logan and Burkell (1986) required participants to perform on three tasks; dual-task, stop-all, and stop-change. In the stop-change task, participants had to inhibit their response and then execute a different response when the second stimulus occurred after the respond signal. Inhibition times in the stop-change task were longer than in the stop-all task, suggesting two inhibitory modes. However, as for the results of selective inhibition discussed above, these findings may also be interpreted by assuming a single mechanism with different degrees of engagement.

De Jong and colleagues (De Jong et al., 1990, 1995) related psychophysiological findings that suggested two inhibitory modes to the motor control literature.

Bullock and Grossberg (1988) derived from this literature that there are two separate processes involved in the generation of movements. Central processes are concerned with the programming of structural movement parameters, such as their direction or amplitude. More peripherally operating processes were thought to generate a 'GO'-signal, which scales (multiplies) the output of the central processes in order to produce the outflow of motor commands to the muscles. In this way, onset and speed of movements are controlled. Likewise, De Jong et al. (1990, 1995) proposed that movements could be inhibited either by preventing the production of motor commands by the central mechanism, or by preventing the outflow of the motor commands by the peripheral mechanism. Because the 'GO'-signal was thought to operate in a largely nonspecific way, scaling any motor commands from the central mechanism, De Jong et al. (1995) related the peripheral mechanism to the fast, nonselective mode of inhibition. By implication, the central mechanism was associated with the slow and selective mode.

De Jong et al. (1990) introduced a physiological criterion for the identification of the peripheral stopping process. They used measures of brain, muscle, and force activity to assess the time course of response activation and inhibition in a stop-all task. The lateralized readiness potential (LRP; for reviews see Coles, 1989; Eimer, 1998) reflects the response-specific involvement of the left and right motor cortices of the brain, and was employed by De Jong et al. (1990) to indicate the degree of central motor preparation induced by response processing prior to inhibition. They reasoned that the operation of the central inhibitory mechanism should affect central motor preparation, and therefore attenuate the LRP. Peripheral inhibition, on the other hand, was assumed to occur after central motor preparation, and therefore should leave the LRP intact. De Jong et al. (1990) defined successful inhibits as trials with a stop signal, but no muscle and force activity. The maximum LRP for these trials was below the maximum LRP for normal responding on trials without a stop signal, implicating the contribution of a central mechanism. Partial inhibits were trials on which muscle and force activity was present, but the force did not reach a preset criterion. The maximum LRP for these trials was also lower than the maximum for normal response trials, again indicating that the central inhibitory mechanism was involved. However, De Jong et al. (1990) doubted that the central mechanism alone could account for stopping on successful and partial inhibits. They reasoned that the inhibitory effect on the LRP must precede the inhibitory effects on muscle and force activity by at least the time required for the transmission of central commands to the peripheral motor system. Because the observed effects on muscle and force activity seemed to precede the effects predicted by transmission delays, they argued that the central mechanism alone could not account for stopping on partial inhibits. De Jong et al. (1990) also observed that the LRP exceeded a 'criterion level' associated with normal responding both on failed and on partial inhibit trials, even though the response was not completed in the latter case. The notion of a criterion level or threshold was based on the observation that LRP amplitude at the instant of responding is constant across conditions and reaction time bins, suggesting that responses are triggered when the criterion is exceeded (Gratton et al., 1988). Importantly, De Jong et al. (1990) found that the

LRP frequently reached the criterion level even on trials resulting in completely successful muscle and force inhibition. If the criterion level of the LRP is indeed associated with triggering a response (Gratton et al., 1988), then the central signs of lateralization and triggering were present on numerous trials on which responses did not occur. This seemed to be strong evidence for a peripheral inhibitory mechanism. By implication, a below criterion LRP in combination with response inhibition defined a more central inhibition.

Some aspects of the data reported by De Jong et al. (1990) suggested that the peripheral mechanism might operate as an 'emergency brake', capable of intercepting responses that have escaped central inhibition. First, the maximum LRP on successful inhibit trials increased with stop-signal delay, presumably because of an increase in the number of trials with an above-criterion LRP that contributed to the average. The number of trials on which the peripheral mechanism was required to prevent the response seemed to increase with stop-signal delay. Second, the percentage of partial responses also increased with stop-signal delay. De Jong et al. (1990) reported 13.8, 21.0, and 36.3% of partial responses on early, middle, and late stop-signal delays, respectively. It seems that, although the majority of responses were inhibited by the central mechanism, the proposed peripheral mechanism might be increasingly involved as the respond process has proceeded.

Further evidence in favor of two inhibitory mechanisms came from a study by De Jong et al. (1995). They directly compared stop-all, stop-change and stop-selective conditions. The peripheral mechanism was expected to be involved only in the stop-all condition, hence De Jong et al. (1995) expected the LRP to exceed the threshold associated with normal responding. This was indeed found to be the case, replicating their earlier findings (De Jong et al., 1990). Successful inhibition in the stop-change and stop-selective conditions, by contrast, was thought to be mediated by the central mechanism, hence the LRP was expected to remain sub-threshold. The LRP in the stop-change condition indeed remained below the criterion level, and this finding provided support for the two-mechanism hypothesis. Contrary to their expectations, however, the LRP in the stop-selective condition exceeded the threshold associated with normal responding. This finding, and the results of an additional experiment, led them to conclude that the peripheral mechanism was indeed involved in selective stopping.

A second physiological index, known to be influenced by non-cortical centers, also added to the description of the peripheral mechanism. Jennings et al. (1992) observed cardiac slowing on partial and full inhibit trials. Because cardiac slowing can be initiated by midbrain centers, they suggested that it might reflect the actions of the peripheral mechanism. It remains uncertain, however, whether cardiac slowing can be viewed exclusively as reflecting the peripheral mechanism, most notably because central structures may contribute to cardiac slowing (e.g. Skinner, 1991). In the data reported by Jennings et al. (1992), cardiac slowing did not discriminate between full and partial inhibits, but full and partial inhibits are thought to differ in the involvement of the peripheral mechanism (De Jong et al., 1990). Thus, although the cardiac evidence was interpreted in terms of the peripheral mechanism, it did not provide a demonstration of this mechanism.

In summary, two stopping mechanisms have been suggested but not wholly established. A central or cortical mechanism is seen as relatively slow, but selective. A peripheral mechanism is seen as fast, but global. Differences in inhibition times across paradigms, and differences in the amount of cardiac deceleration between successful and unsuccessful inhibition, are compatible with the distinction between the two mechanisms, but also with a single mechanism. The distinction between the two mechanisms is mainly based on differences in a hypothetical triggering mechanism keyed to LRP amplitude. Experimental conditions that are characterized by a LRP below a 'respond threshold' are assumed to involve only central inhibition, whereas conditions exhibiting a supra-threshold LRP are thought to involve a more peripheral mechanism in addition to the central mechanism.

2. The present research

On balance, it seems that the notion of two inhibitory mechanisms provides an attractive account of the data but is far from firmly established. After reviewing the stopping literature, Logan (1994) concluded that "... the evidence for central and peripheral inhibitory mechanisms is scant and depends as much on argument than on fact" (p. 206). The timing arguments of De Jong et al. (1990) depend on very small differences and assumptions generalized from different subjects, tasks, and paradigms. Moreover, the distinction between central and peripheral inhibition mechanisms relies heavily on a dichotomization of LRP amplitudes into sub- versus supra-threshold amplitudes. The aim of the present study was therefore to re-examine the presumed central and peripheral mechanisms of stopping, thereby either sharpening the definition of the two mechanisms or showing that this distinction is more illusory than real. We opted for an in-depth analysis of stopping in the stop-all task. The stop-all task is thought to involve both mechanisms (De Jong et al., 1990), and was therefore used in the present experiment to study these mechanisms in the same task. We attempted to sharpen the distinction between the two mechanisms in three ways: (i) analyzing the LRP in great detail, particularly with respect to the putative threshold associated with normal responding; (ii) incorporating no-go trials into the design and comparing frontal brain waves in no-go and stop-signal situations; and (iii) combining cardiac measurements with the LRP and frontal brain waves.

2.1. Analyzing the LRP in detail

De Jong et al. (1990, 1995) interpreted the LRP to reflect central processes that specify movement parameters such as the responding hand. The output of these central processes is then thought to be scaled by a more peripherally operating 'GO-signal' to produce the actual outflow of central motor commands to peripheral motor structures (Bullock and Grossberg, 1988). In their view, the LRP therefore seems to arise between central motor preparation and the peripheral GO-signal, an assumption that they exploited to distinguish between central and peripheral

inhibition based on the LRP in the first place. If the LRP threshold is exceeded (central motor commands have been issued) but the response is nevertheless stopped, the peripheral mechanism is assumed to be active (the GO signal is set to zero). However, recent evidence from the motor control literature suggests that the LRP might arise after the GO signal, not before. If this is true, then it might be impossible to distinguish between central and peripheral inhibitory mechanisms based solely on the LRP criterion threshold. We will now briefly review this evidence.

Two sequentially operating loops through cortical and subcortical areas are important in the generation of responses (for a recent review, see Band and Van Boxtel, 1999). In the anterior loop, activity from widespread cortical areas is focused back via the midbrain (basal ganglia and thalamus) to more restricted cortical areas, especially the supplementary motor area (SMA). This loop functions as a system for the specification of response parameters. The GO-signal, which is thought to be generated in the basal ganglia (Bullock and Grossberg, 1991), is mediated by the anterior loop. At the scalp, the activity of the anterior loop is reflected in a bilaterally symmetrical slow wave (Deecke, 1987; Goldberg, 1985). Because of the bilateral symmetry of this wave, the anterior loop does not contribute to the LRP. The LRP is mainly determined by activity in the posterior loop, in which activity from predominantly posterior brain areas is transmitted back, via the cerebellum and the thalamus, to the primary motor cortex, contralateral to the responding limb. The posterior loop operates after the anterior loop, which can be inferred, for instance, from the difference in onset times between the symmetrical and lateralized components of the readiness potential (Deecke, 1987). This evidence suggests a sequence of central processing, GO signal, and then LRP. Accordingly, LRP onset should be interpreted to index the start of motor outflow from central structures to the periphery, after specification of movement parameters by central processes and scaling by the GO-signal have taken place. Seen from this perspective, the crossing of the LRP threshold on partial response trials might merely indicate that central outflow to the periphery has started, which makes perfect sense because these trials were characterized by partial muscle and force activity. Moreover, according to this perspective it is LRP duration, not LRP threshold that should distinguish between peripheral and central inhibition mechanisms. The duration of central motor outflow indicates how long the muscles are driven (e.g. Burke, 1981; De Luca, 1997), and therefore determines the magnitude of muscle contraction, and, consequently, of the response. We hypothesize that the duration of central motor outflow may be estimated by LRP duration. Should LRP duration on partial and full response trials be the same, despite differences in muscle and force activity, this would strengthen the notion of a separable inhibitory mechanism operating peripherally.

2.2. Incorporating no-go trials

Observations from the disjunctive or go/no-go reaction time task have not played a direct role in the discussion of stopping mechanisms. In this task participants

have to respond to one stimulus and withhold their response to another stimulus. The go/no-go task is functionally equivalent to a stop-signal task in which the respond and stop signals are presented simultaneously (Logan et al., 1984). Use of the central mechanism in the go/no-go task is suggested by one aspect of the De Jong et al. (1990) results noted earlier; below criterion LRPs occurred when stop-all signals were presented early after the respond signal. This finding agrees with results of several other studies that have reported small LRPs in go/no-go tasks (e.g. Ilan and Miller, 1999; Miller and Hackley, 1992; Osman et al., 1989). Brain potential evidence also suggests cortical involvement in no-go processing; evidence that would implicate the central mechanism. A negative brain potential called N200 is detected over the frontal cortex on no-go trials (e.g. Eimer, 1993; Jodo and Kayama, 1992; Kok, 1986; Pfefferbaum et al., 1985; Naito and Matsumura, 1994, 1996). Brain imaging and microelectrode studies provide support for the frontal origin of the negative potential (Kawashima et al., 1996; Sasaki and Gemba, 1986; Sasaki et al., 1993). Most interestingly, electrical stimulation of this frontal area during normal response activation suppressed the activity in the motor cortex and the overt response (Sasaki et al., 1989). Thus it seems that countermanding a motor response in a go/no-go task involves a central inhibition mechanism indexed by a negative potential that can be recorded at the frontal scalp. We are not aware of any literature linking the frontal N200 to processes operating 'downstream' of the motor cortex (i.e. to a peripheral inhibitory mechanism).

A consideration of the literature thus suggests that a central mechanism is involved in inhibition to a no-go signal. Combining no-go and stop signals in the same stopping paradigm can then be used to study the contributions of the central and peripheral mechanisms on stop trials. On the assumption that no-go trials and short delay stop-signal trials are functionally equivalent, successful stop-signal inhibition trials should exhibit the N200, a cortically generated scalp potential. Moreover, the N200 should be temporally related to the stop signal. An N200 elicited by partial signal-inhibit trials would suggest the contribution of the central mechanism, whereas the absence of the N200 of partial inhibit trials would provide support for a peripheral mechanism. Finding an N200 on unsuccessful inhibit trials might be interpreted to suggest central inhibition that failed. The consequence of combining no-go and short delay stop signal trials in the same task is that all stimuli will be presented in the visual modality. In the stop-signal task, visual respond signals and auditory stop signals have usually been employed to emphasize the independence of the respond and stop processes. We will use the methodology of the horse race model to test whether the important assumption, that responding and stopping are independent, has been seriously violated because of stimuli in the same modality. The horse race methodology will also be used to check whether the inclusion of no-go trials led participants to adopt a different inhibition strategy compared to a standard stop-signal design.

2.3. Cardiac deceleration

The third way in which we attempted to delineate the distinction between central and peripheral inhibitory mechanisms, was to measure heart rate in combination with the LRP and the N200. Jennings et al. (1992) found cardiac slowing on full and partial inhibits in a stop-signal task. On the assumption that global stopping invokes a peripheral mechanism, this result was interpreted as a midbrain manifestation of peripheral inhibition. However, the interpretation is not watertight. Cardiac deceleration is also found on no-go trials (Van der Molen et al., 1985; Van der Veen et al., 2000) and on trials with complex stimulus-response mapping (Jennings et al., 1991). Assuming that these trials invoke central inhibition, it would seem that cardiac deceleration is a manifestation of central rather than peripheral inhibition. These findings are consistent with the growing support for the role of the frontal cortex in cardiac control (Fuster, 1997; Neafsey, 1990; Skinner, 1991). On the assumption that the central inhibitory mechanism is instigated from the frontal cortex, the cardiac deceleration on full and partial inhibit trials observed by Jennings et al. (1992) could perhaps be better explained in terms of central not peripheral inhibition. Here we shall investigate the relation between cardiac deceleration, the LRP and the N200. Finding that cardiac slowing does not discriminate between sub- versus supra-threshold LRP amplitude, or different above-threshold times, would support a hypothesis of a single inhibitory mechanism. In addition, finding a relation between cardiac deceleration and the N200 would support a single-mechanism view.

3. Method

3.1. Participants

Ten right-handed participants, five men and five women, with ages ranging from 19 to 28 years (mean 22.2 years) participated in the experiment. They were all healthy, non-smokers, and had normal or corrected-to-normal vision and hearing. They were paid for the completion of the experiment.

3.2. Experimental task and procedure

The experiment was carried out in a dimly lit, sound attenuating, electrically shielded chamber. The participants were each seated in a comfortable reclining chair with supports for hands, arms, and legs. The stimuli were presented on a 14-in. monitor with a refresh rate of 60 Hz, placed 1 m in front of the subject at eye level. The stimuli, presented on a black monitor background, were arrows pointing either to the left or the right ($P = 0.5$), subtending a visual angle of 1.72°. Stimulus duration was 1500 ms, with an intertrial interval ranging from 4 to 8 s (mean 6 s, rectangular distribution), during which a white fixation square subtending a visual angle of 0.4° was presented.

The participants responded by pressing the left or right index finger, depending on the direction of the arrow, on one of two zero-displacement force transducers (Kyowa LM-20KA) mounted into the hand support, which had the shape of an open, slightly bent hand. In this way a voltage proportional to the force applied to the transducer was generated, which was on-line A/D converted and analyzed, allowing immediate determination of the response characteristics. Before the experiment, the participants' maximum voluntary force was recorded for both hands separately. Response onset was defined as the instant at which the force reached 2% of the maximum force, and response completion when 15% of the maximum force was reached.

On 70% of the trials, the arrow presented on the monitor was colored green (go, or no-signal trials). In this case, the task was to press the force transducer as quickly as possible up to the criterion force level of 15% of their own maximum, with the index finger of the hand corresponding to the direction of the arrow. On 10% of the trials, the arrow was colored red, and the participants had to refrain from responding (no-go trials). On the remaining 20% (stop trials), the arrow was colored green, but after a variable interval it briefly turned red for 100 ms, after which it became green again. The color change signaled the participants to withhold their response, and resembled the insertion of a separate stimulus just as is often done in case of an auditory stop stimulus. The timing of this stop signal was determined by a simple staircase-tracking algorithm described by Levitt (1971). Starting from an initial value of 300 ms after the respond stimulus, the algorithm worked by subtracting 50 ms from the previous value of the stop signal latency if the response was correctly withheld, and added 50 ms to it when a full response was produced. In this way, theoretical probabilities of 50% full responses and 50% correct inhibitions (including partial responses) were obtained. In keeping with the behavioral definition of reaction time and response completion, stop trials were classified as signal-respond trials if the force exerted on the transducer was greater than 15% of the maximum force. If the force remained below the 2% criterion, the trial was classified as a signal-inhibit trial, and if the force was between 2 and 15% the trial was classified as a signal-partial trial. A trial was rejected as soon as more than 2% of force was generated with the wrong hand. That is, we did not allow participants to correct such errors by an ensuing response with the correct hand. Pilot work indicated that 2 and 15% of force were good practical values to control the tracking algorithm during the experimental sessions.

Each subject participated in four sessions on separate days, one training session and three experimental sessions. The experimental sessions were always separated by 1 week. In the training session, which preceded the first experimental session by 1 or 2 days, the participants were verbally coached by the experimenter to produce fast responses reaching the force criterion as quickly as possible. In addition, they received knowledge of results about the response after each trial. The force of their response was plotted as a function of time on the computer screen overlapping an 'ideal' force trace in which the criterion force was reached within 40 ms after force onset. If an error was made, this was also indicated on the monitor. Later in the training session, knowledge of results was eliminated mimicking the case in the

experimental sessions. Training continued until the RT on go trials reached a stable level and the standard-deviation of RT measured within a block of 150 trials, containing 105 go trials, was less than 20% of the mean reaction time in that block. In each experimental session, the participants received an additional training block without knowledge of results after the fixation of the electrodes. They were then given eight blocks of 150 trials, each consisting of 105 go (no-signal) trials, 15 no-go trials, and 30 stop trials. The order of trials within a block was completely randomized. In total, 3600 experimental trials were collected for each subject over the three experimental sessions, consisting of 2520 go trials, 360 no-go trials, and 720 stop trials.

During the experiment the primacy of the go task was stressed to the participants. They were told only after the experiment that the study was about response inhibition. Prior to that, they were led to believe that it was about choice reaction time performance, and that the stopping was included for methodological reasons. They were asked to try and withhold their response when a stop signal occurred, but were told that this would not be possible on all of the stop trials.

3.3. Recording and analysis of psychophysiological signals

Event-related potentials were recorded from 28 Beckman Ag/AgCl cup electrodes with a diameter of 8 mm, affixed to the scalp with Grass EC-2 electrode paste. In the present paper we will present only the activity recorded from the 10–20 system positions F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4 referred to algebraically linked mastoids. These electrode positions cover most of the scalp and allow us to present the most important temporal aspects of the data. Eye movements were monitored by three pairs of Beckman Ag/AgCl electrodes of 2 mm size. Two pairs were placed in a straight line above and below each eye to monitor vertical eye movements and blinks, and one pair was placed in a straight line at the outer canthi of the left and right eye to monitor horizontal eye movements and saccades. Electrode impedance for the scalp and eye movement electrodes was kept under 5 kΩ, and the resulting signals were amplified using a time constant of 3 s, low-pass anti-aliasing filtered at 70 Hz (Butterworth, 42 dB/octave roll-off), and on-line A/D converted at 200 Hz. These filter settings enabled us to record the signals of interest without distortion.

The eye movement records were used off-line to correct the event-related potentials for electrical contamination of both vertical and horizontal ocular artifacts using the autoregression method of Van den Berg-Lenssen et al. (1989). This method relies on an eye movement calibration trial that was recorded before each block in order to estimate the correction parameters for that block. In these calibration trials the participants were asked to visually follow a dot jumping to 12 locations on the monitor, thereby making eye movements of pseudo-randomly determined visual angles. This procedure enabled us to correct rather than reject epochs in which the event-related potentials were distorted as a result of eye blinks, saccades, and slow eye movement. All trials were treated by this procedure. Because ocular artifacts are not the only sources of distortion of the raw signals, the data were subsequently checked for other artifacts by a semi-automatic rejection proce-

dure. Signals were passed through a window and excluded from further analysis when the minimum and maximum of the 2 Hz low-pass filtered potentials in the interval between 500 ms before the respond stimulus until 2 s thereafter differed more than 80 μV. The overall percentage of rejected trials by these procedures was 2.3%. The mean voltage measured in the 500 ms interval before the respond stimulus was used for baseline correction. Statistical analysis of event-related potentials was done by means of multivariate analysis of variance (MANOVA) in order to cope with high intercorrelations between measurements at nearby electrode locations (Vasey and Thayer, 1987).

The LRP was computed by subtracting the waveforms measured at C4 from the waveform measured at C3, separately for all trial categories. The C3 and C4 electrodes are located directly above the left and right hand area of the primary motor cortex (Homan et al., 1987). Subsequently, the difference waveforms obtained for left hand responses in each category were subtracted from the corresponding waveform obtained for right hand responses. In formula, $LRP = (C3_R - C4_R) - (C3_L - C4_L)$. This procedure results in negative-going waveforms of the activity contralateral to the side of the response, while lateralized activity unrelated to the side of the response is canceled out (Eimer, 1998). The onset of the LRP was scored after digital low-pass filtering (Ruchkin and Glaser, 1978) at 8.8 Hz. Such a filter preserves the most interesting direction-specific lateralization while attenuating high-frequency changes in lateralization that might hamper the determination of the onset. The onsets were scored by a segmented regression procedure (Schwarzenau et al., 1998). The onset was defined as the intersection point of two linear regression lines, the first one flat through zero and the second one through the LRP peak. Mordkoff and Gianaros (2000) have recently compared a great number of LRP onset scoring methods, and concluded that this approach (SS1DF in their terminology) yielded the most accurate results.

For recording muscle activity, the agonist and antagonist electromyogram (EMG) was recorded bipolarly by pairs of 2 mm Beckman Ag/AgCl electrodes to the dorsal and palmar aspects of the left and right forearms, using the standard placements described by Lippold (1967). By this placement the activity of the superficial finger flexor and extensor muscles is best picked up. It was amplified, high-pass filtered at 20 Hz, full-wave rectified, and low-pass filtered at 50 Hz. The onset of muscle activity is gradual rather than discrete, and therefore computed off-line on single trials by an accurate statistical method described by Van Boxtel et al. (1993). This method relies on a threshold voltage comparison supplemented with temporal analysis of each EMG burst, and results in an average absolute error of less than 5 ms.

Heart rate deceleration was assessed by measuring the electrocardiogram (ECG) bipolarly from 10 mm Ag/AgCl electrodes using a V6 versus sternum lead, which maximizes the R-wave with respect to the other ECG components, and is relatively insensitive to artifacts (Mulder, 1988). It was amplified with a time constant of 0.03 s, and low-pass filtered at 300 Hz (12 dB/octave roll-off). Six interbeat intervals were calculated from the raw ECG data for each trial; three interbeat intervals before, the interbeat interval during, and two interbeat intervals after the presenta-

tion of the respond stimulus. Interbeat intervals less than 400 ms and greater than 1500 ms were discarded from all cardiac analyses. Respiratory activity was measured by placing a pneumograph around the subject's chest, just above the abdomen. The pneumograph consisted of a distensible tube connected to a sound-sensitive device and an amplifier. Changes in the length of the tube were measured by the phase difference of a 575 Hz tone traveling though the tube, which was converted to a voltage. Respiratory phase was determined by integrating the respiratory signal for 300 ms starting with the presentation of the respond stimulus. The trial was classified as an inhalation trial if the integral was positive, else it was marked as an exhalation trial. Only the exhalation trials were selected for the cardiovascular analyses. This was done to avoid the confounding acceleratory influence associated with inhalation. The muscle activity, the cardiac and the respiratory signals, and the continuous record of the force exerted on the transducers were sampled at a rate of 1000 Hz.

4. Results and discussion

4.1. The horse race model

4.1.1. Task performance

Table 1 presents an overview of RTs and the probabilities of inhibiting, responding, or producing a partial response. Task performance was fast and

Table 1
Task performance for go (no-signal), no-go, and stop trials, averaged over ten participants

Go trials	Number of trials:	2520
	Number of correct responses:	2435.5 (96.7%)
	Observed reaction time:	351 ms (S.E.M.: 31 ms)
No-go trials	Number of trials:	360
	Number of correct inhibitions:	347.2 (96.44%)
Stop trials:	Number of trials:	720
	Overall stop signal latency:	176 ms (S.E.M.: 43 ms)
	Estimated stop-signal reaction time:	174 ms (S.E.M.: 27 ms)
Inhibit		
	Number of correct inhibitions:	243.0 (33.75%)
	Stop signal latency:	140 ms (S.E.M.: 46 ms)
Partial		
	Number of partial responses:	84.0 (11.67%)
	Stop signal latency:	158 ms (S.E.M.: 47 ms)
	Predicted reaction time:	358 ms (S.E.M.: 31 ms)
	Observed reaction time:	365 ms (S.E.M.: 35 ms)
Respond		
	Number of respond trials:	393.0 (54.58%)
	Stop signal latency:	200 ms (S.E.M.: 43 ms)
	Predicted reaction time:	311 ms (S.E.M.: 28 ms)
	Observed reaction time:	334 ms (S.E.M.: 27 ms)

Note: S.E.M., standard error of the mean.

accurate. The proportion of correct responses on no-signal (go) trials and correct inhibitions on no-go trials were both high. The mean RT on go trials was reasonably fast (351 ms), hence participants did not seem to delay their responses to increase the chance of withholding the response when a stop signal occurred. The focus on fast responding also appears from the percentage of erroneous responses to stop signals, which was slightly larger than the expected value of 50% (54.6%; $F(1,9) = 23.03$, $P = 0.000$). Responses were virtually all correct choices on the primary RT task (no-signal trials). Most of the slightly more than 3% errors for the no-signal trials were responses with the wrong hand; there were only 0.2% force undershoots, in which force output started but the 15% criterion was not attained.

Table 1 indicates that 11.7% of the trials on which a stop signal was presented resulted in a partial response. Given the low proportion of force undershoots on no-signal trials (0.2%), it seems unlikely that the partial responses on signal trials were force undershoots. De Jong et al. (1990) observed a greater percentage of partial responses, probably because of their use of a higher force criterion value. Two other aspects of the results given in Table 1 are in good agreement with the horse race model (see Logan, 1994). First, the stop-signal RT in the present study approximates the commonly found value of about 200 ms in a variety of tasks and stop signal delays. Secondly, the tracking procedure resulted in the shortest stop-signal delay for successful inhibit trials (140 ms), the longest delay for signal-respond trials (200 ms), and an intermediate value for partial response trials (158 ms; $F(2,8) = 422.60$, $P = 0.000$).

The predicted RTs given in Table 1 were calculated based on the assumptions of the horse race model (Logan and Cowan, 1984; Logan, 1994). Consistent with the race model, the observed RT on signal-respond trials was shorter than the RT on no-signal trials ($F(1,9) = 33.24$, $P = 0.000$) and on partial response trials ($F(1,9) = 52.78$, $P = 0.000$). This seems to be a very robust finding in stop signal tasks (e.g. De Jong et al., 1990; Jennings et al., 1992; Logan, 1994; Logan and Cowan, 1984; Osman et al., 1990). The finding agrees with the intuitive notion that signal-respond trials are those trials that were fast enough to escape inhibition, corresponding to the leftmost part of the no-signal RT distribution. Signal-partial trials correspond to the middle part of the no-signal RT distribution, and are hence slightly slower. The goodness of fit between the race model and the observed data was tested further by comparing observed with predicted RTs on signal-partial and signal-respond trials. The predicted RTs were calculated by rank ordering the no-signal RTs and averaging the number of RTs corresponding to the proportion of full and partial responses on signal trials. This amounts to isolating the leftmost and middle parts from the no-signal RT distribution. Observed RT matched the predicted value for signal-partial trials (365 vs. 358 ms; $F(1,9) = 1.98$, $P = 0.193$), but not for signal-respond trials (334 vs. 311 ms; $F(1,9) = 201.74$, $P = 0.000$). The 23 ms difference between observed and predicted RTs for signal-respond trials is greater than in some earlier reports (e.g. De Jong et al., 1990; Logan and Cowan, 1984),

but is not uncommon. For instance, Jennings et al. (1992) reported a difference of 27 ms for early stop signals. The significant finding does, however, question the important assumption of the horse race model that the stopping and respond processes are independent. In order to accommodate no-go trials, we presented all visual signals, and it is possible that the use of a single sensory modality led to an interaction of the respond and stop processes. We will argue in the next section that this was not the case.

4.1.2. Independence of respond and stop processes

Simulation studies suggest that the underestimation of signal-respond RT does not necessarily imply an association between responding and stopping, but may be due to a large variability in stopping speed (Band, 1997; De Jong et al., 1990). Yet it can not be excluded that the independence assumption was violated. Therefore we used psychophysiological measures to collect further evidence for the independence assumption. We compared the waveforms for signal-respond trials and corresponding no-signal trials as in the calculations of RT presented above. The average waveforms of the corresponding no-signal trials can be conceived of as the predicted waveforms for each measure. We calculated separate averages from the left, middle, and right parts of the no-signal RT distribution, corresponding to the proportion of full, partial, and inhibited responses on signal trials. The waveforms of signal trials constitute the observed data. Clearly, the observed and predicted waveforms should be identical if the independence assumption is true. This would indicate that the response process would operate in the same way whether or not a stop signal was presented. Fig. 1 shows the averaged waveforms of the force exerted on the response device and the LRP. Two averages are presented for each waveform. In the average synchronized to the respond stimulus (left column in Fig. 1) the differences in onset with respect to the respond stimulus are preserved. Onset differences are lost but the shape of the single trial waveforms is better preserved in the average time-locked to the onset of muscle activity (right column in Fig. 1). Numerical estimates of onset times calculated from the respond signal and of peak amplitudes computed from averages synchronized to muscle-activity onset are presented in Table 2, separately for response force, agonist and antagonist muscle activity, and the LRP.

Fig. 1 and Table 2 suggest that the waveforms of the signal-respond and the corresponding no-signal trials are highly similar. Analysis of these results demonstrated that the only significant differences were those that paralleled the underestimation by the model of signal-respond RT. Since the horse race model underestimated RT, it is no surprise that the onset of muscle activity was also underestimated. This is a result of the fixed mechanical coupling of these response systems (cf. De Jong et al., 1990). Observed and predicted LRP onsets could not be distinguished statistically, suggesting that the underestimation of RT is caused by peripheral factors, such as the friction of bones and tendons. This explanation is also supported by the difference between observed and predicted peak force (Table 2). Peripheral response dynamics cause higher forces to be associated with shorter reaction times (Carlton et al., 1987). The underestimation of RT is therefore probably influenced, at least in part, by the effects of peripheral factors.

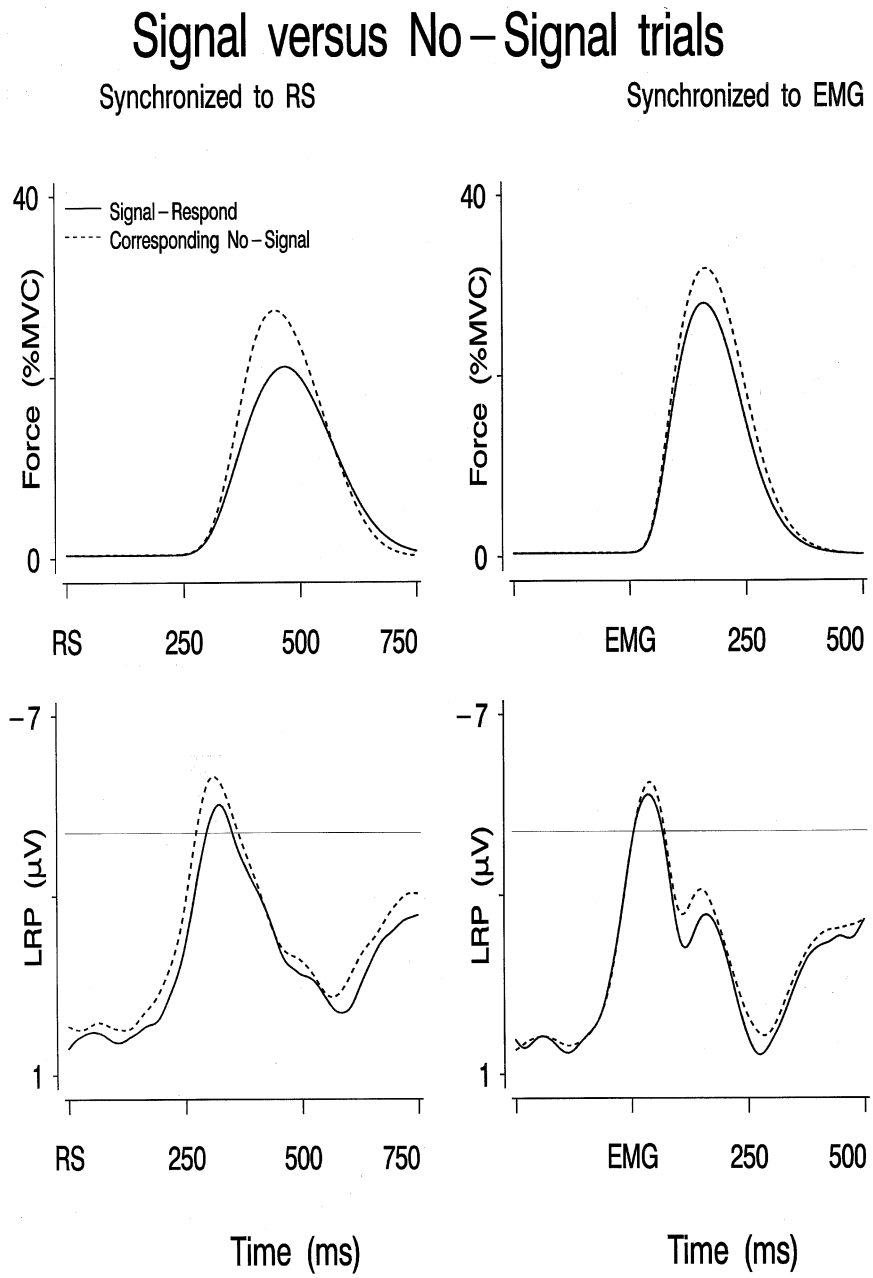

Fig. 1. Waveforms of squeeze activity (force; upper panels), and lateralized readiness potentials (LRP; lower panels), averaged over response hands and participants. Separate waveforms are shown for trials which, despite the stop signal, resulted in a full response (signal-respond trials), and the corresponding (matched for RT) no-signal trials. See text for further detail. In the left panels, the average was computed synchronized to the onset of the primary reaction time signal (RS). In these averages, differences in onset times are best preserved. The averages synchronized to the onset of muscle activity (EMG) are shown on the right. These averages show the characteristics of the single trials better than the stimulus-locked averages, at the cost of eliminating differences in onset times.

Table 2
Mean observed (signal-respond) and predicted (corresponding no-signal) onsets and peaks of response force, agonist and antagonist muscle activity, and lateralized readiness potential (LRP)

Measure	Onset		Peak	
	Observed	Predicted	Observed	Predicted
Response force	334 (27) ms	311 (28) ms*	31.3 (25.4) %MVC	36.1 (29.9) %MVC*
Agonist muscle activity	296 (28) ms	276 (30) ms*	104.8 (32.2) μV	108.5 (30.5) μV
Antagonist muscle activity	324 (27) ms	321 (24) ms*	93.3 (26.3) μV	95.5 (25.6) μV*
LRP	186 (58) ms	176 (29) ms	−6.3 (2.5) μV	−6.4 (2.4) μV

Note: MVC, maximum voluntary contraction. Standard error of the mean (S.E.M.) in parentheses.
* Significant *t*-test observed-predicted.

Taken together, these results indicate that the horse race model provides a reasonably good fit of the data. The independence assumption does not seem to be seriously violated even in the present situation in which the stop signal was presented in the same modality as the primary task respond signal. There is some evidence that the late stages of motor processing were affected by the presentation of the stop signal, but the absolute magnitude of the observed differences was very small and the effects were probably of a mechanical nature. The results replicate the data reported by De Jong et al. (1990), and are also in fairly good agreement with other studies reporting on stop-signal tasks. Therefore, it seems safe to conclude that using visual instead of auditory stop signals, and including no-go trials in the design, did not affect performance.

4.2. The nature of the stopping process

4.2.1. Peripheral response evidence

Partial response trials are of crucial importance for the distinction between central and peripheral inhibitory mechanisms. Partial response trials are trials in which muscle and force output was present but the response did not reach the required criterion force. Inhibitory processing is implied by these partial responses if they are in fact interrupted versions of regular responses. An obvious alternative is that partial responses are just force undershoots. As already argued above, this possibility is unlikely based on the proportion of partial trials on stop-signal as compared to no-signal trials. Another possibility is that partial response trials occurred because the antagonist muscle was contracted as soon as the stop signal was processed, lifting the finger from the response button. The data shown in Fig. 2 clearly rule out this possibility, as both agonist and antagonist muscle activity on signal-partial trials was smaller than on signal-respond trials (peak agonist muscle activity in signal-respond versus signal-partial trials: $F(1,9) = 83.61$, $P = 0.000$). Fig. 2 also shows that there was virtually no agonist, nor antagonist, muscle activity on complete inhibition (signal-inhibit) trials. A third possibility is that participants

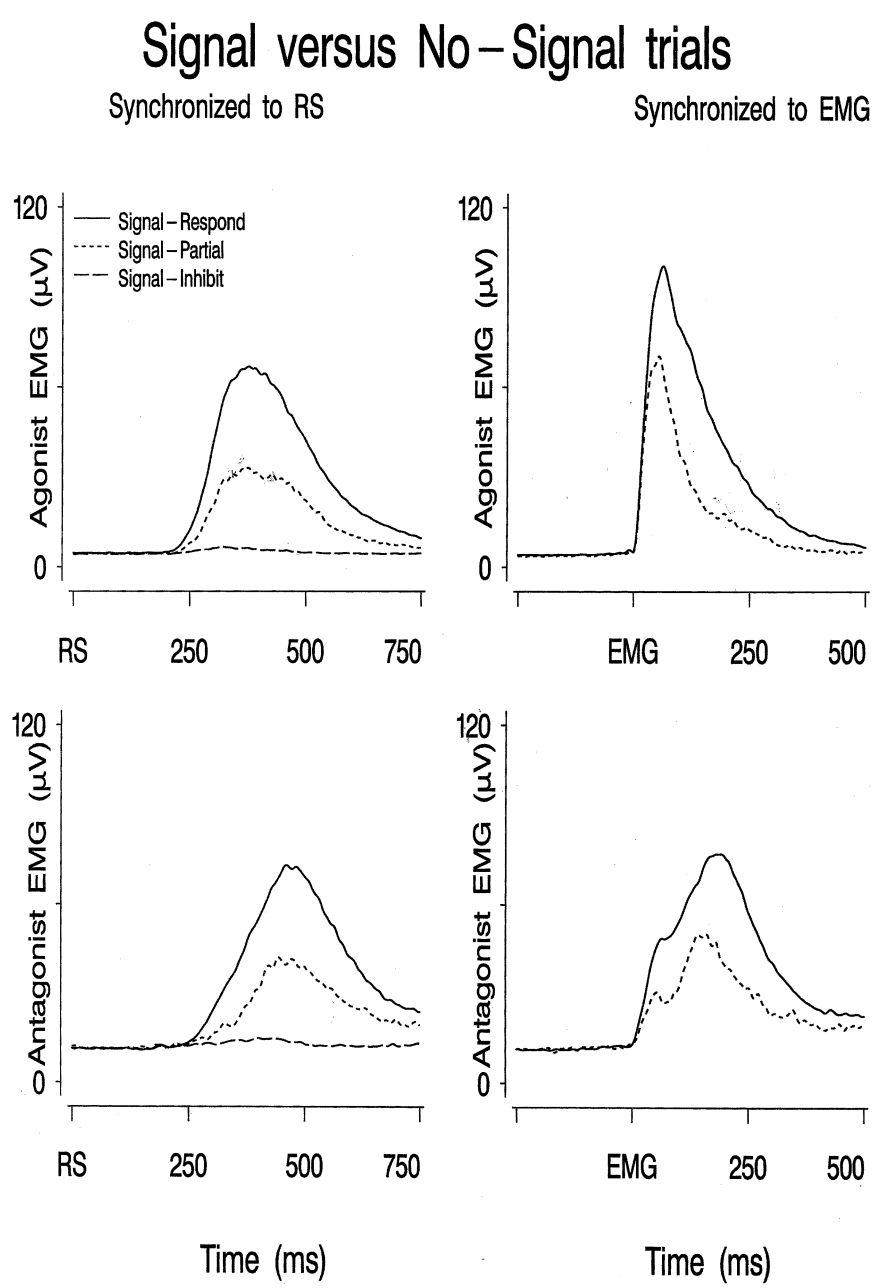

Fig. 2. Agonist (upper panels) and antagonist (lower panels) muscle activity for stop-signal trial categories, synchronized to the onset of the primary respond signal (RS; left panels), or the onset of muscle activity (EMG; right panels). Signal-inhibit trials are not plotted time-locked to EMG onset because the response was absent. Note the absence of agonist AND antagonist muscle activity on signal-inhibit trials.

initiate a partial response and complete it only if they are relatively certain that a stop signal is not going to be presented. In that case a gradual buildup of agonist muscle and squeeze activity would be expected, followed by a sudden increase at the time when the decision to respond became effective. Instead, muscle and squeeze activity consisted of a single quick burst (Figs. 1 and 2), and agonist muscle activity peaked at about the time of force initiation, indicating the ballistic nature of the response (Desmedt and Godaux, 1979). In sum, it seems reasonable to conclude that the partial responses were actually interrupted responses, implicating the operation of an inhibitory mechanism, either central and/or peripheral.

4.2.2. The LRP

The LRP waveforms on stop trials are shown in Fig. 3, separately for failed (signal-respond), partial (signal-partial) and successful (signal-inhibit) inhibit trials. Following Gratton et al. (1988) and De Jong et al. (1990) we determined LRP amplitude of no-signal trials at the onset of muscle activity for each subject separately, and used it as an estimate of the criterion threshold value for central motor outflow. The average criterion value was -4.1 μV (S.E.M. 0.6). De Jong et al. (1990, 1995) argued that the mean LRP at muscle or squeeze activity onset overestimates the actual value of the threshold, because it does not take transmission delays into account. Transmission delay from motor cortex to the muscle can be estimated to be about 20 ms, based on transcranial magnetic stimulation studies (e.g. Romaiguère et al., 1997; see also De Jong et al., 1990). We therefore also estimated the threshold value from LRP amplitude at 20 ms before muscle activity onset, and the resulting value was -2.9 μV (S.E.M. 0.5).

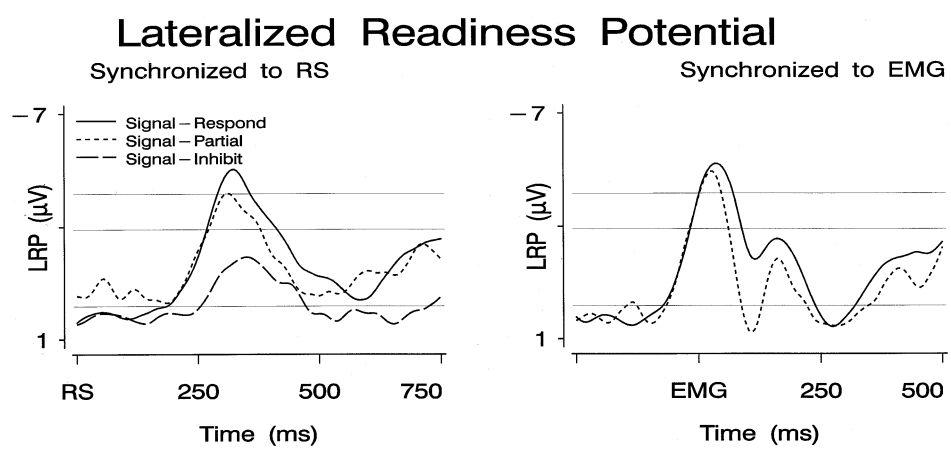

Fig. 3. Lateralized readiness potentials (LRP) for stop-signal trials in which response inhibition was successful (signal-inhibit), partially successful (signal-partial) or in which it failed (signal-respond). The data were synchronized to the onset of the primary respond signal (RS; left panels), or the onset of muscle activity (EMG; right panels). Signal-inhibit trials are not plotted time-locked to EMG onset because the response was absent. The three LRP threshold estimates are plotted as horizontal lines.

In addition, because LRP onset would seem to be a candidate for the start of motor outflow as well, we also estimated the threshold from LRP onset, resulting in a value of -0.2 μV (S.E.M. 0.1). The three threshold estimates differed statistically ($F(2,8) = 23.13$, $P = 0.000$).

We then compared the maximum LRP amplitude for no-go and stop-signal trials to each of these threshold levels. For signal-partial and signal-respond trials we used LRPs averaged with respect to muscle-activity onset, and for no-go and signal-inhibit trials we used LRPs synchronized to the primary task stimulus. Clearly, threshold crossing depended on how the threshold was estimated. When LRP amplitude at the onset of muscle activity was used as an estimate (Gratton et al., 1988), the maximum LRP for no-go and signal-inhibit trials remained below, and therefore statistically differed from the threshold (respectively, $F(1,9) = 13.75$, $P = 0.005$, and $F(1,9) = 5.13$, $P = 0.049$), and the maximum LRP for signal-partial ($F(1,9) = 9.08$, $P = 0.015$) and signal-respond trials ($F(1,9) = 12.08$, $P = 0.007$) exceeded, and hence also statistically differed from the threshold. When the more appropriate estimate at 20 ms before muscle activity onset was used (De Jong et al., 1995), the threshold value was reached but not exceeded, and therefore did not differ statistically on no-go ($F(1,9) = 1.26$, $P = 0.291$) and signal-inhibit trials ($F(1,9) = 0.00$, $P = 0.999$), and it was exceeded for partial ($F(1,9) = 22.89$, $P = 0.001$) and full responses ($F(1,9) = 29.21$, $P = 0.000$). Finally, when LRP onset was used as an estimate, the threshold was exceeded for all trial categories (all Fs > 30, all $P < 0.001$).

Peak LRP amplitudes were slightly greater for signal-inhibit than for no-go trials ($F(1,9) = 8.28$, $P = 0.018$). Although Fig. 3 (right panel) suggests that the peak LRP for partial inhibit trials seems to be lower than for full responses, they could not be distinguished statistically ($F(1,9) = 3.01$, $P = 0.117$). De Jong et al. (1990) found a greater peak for signal-respond than for signal-partial trials, but in the present experiment partial and full responses to stop signals did not seem to differ with respect either to threshold crossing and to maximum amplitude.

We also determined how long the LRP remained above the criterion levels associated with normal responding. This yielded a pattern of results that was similar for all threshold estimates. Most important, it can be seen in the right panel of Fig. 3 that above-threshold time was always slightly shorter in partial relative to full response trials for all three thresholds, and this was confirmed by a main effect of Trial Type ($F(2,8) = 8.11$, $P = 0.012$) in a 2-way MANOVA with factors Trial Type and Threshold Estimate. The main effect of the factor Threshold Estimate in this analysis ($F(2,8) = 18.95$, $P = 0.001$) indicated that supra-threshold time was different for the three threshold estimates, regardless of trial type. Most important for the present purposes is supra-threshold time for the threshold estimated at 20 ms before muscle activity onset, comparing signal-respond and signal-partial trials. The appropriate contrast was significant ($F(1,9) = 7.47$, $P = 0.023$).

Summarizing, we found sub- or near-threshold LRPs on no-go and signal-inhibit trials, depending on the value of the threshold estimate. Supra-threshold LRPs were found on signal-partial and signal-respond trials, irrespective of the value of the estimate. This replicates the findings reported by De Jong et al. (1990), which they interpreted as evidence for a peripheral inhibitory mechanism. On partial inhibits, the central motor threshold was exceeded but the response was nevertheless (partially) inhibited, which must have occurred at some level after or 'downstream' of the LRP. We added findings suggesting that, despite threshold crossing, the duration of the LRP was different on signal-partial and signal-respond trials, suggesting the existence of a single inhibitory mechanism that becomes effective at different instants during response activation.

4.2.3. Event-related potentials

The brain potentials recorded on go (no-signal) and no-go trials are depicted in Fig. 4, averaged over responding hands. Statistical testing was done by means of MANOVA with the following within-subjects factors: Trial Type (Go, No-Go), Response Side (Left, Right), Position (Frontal, Central, Parietal), and Laterality (Left hemisphere, Midline, Right hemisphere). The waveforms were characterized by a broad positivity with a parietal maximum ($F(2,8) = 53.45$, $P = 0.000$), presumably reflecting the processing of the primary stimulus (P300, see Fabiani et al., 1978, for a review). At the parietal electrodes, the P300 was equally large for go and no-go trials, but at central and frontal sites, it was larger for no-go trials (Trial Type × Position: $F(2,8) = 37.81$, $P = 0.000$). A frontocentral positivity related to successful stopping ('no-go P3') has been found before. For instance, De Jong et al. (1990) observed a positive difference wave resulting from a subtraction of signal-inhibit and corresponding no-signal trials in the stop-signal task. In line with, for instance, Kok (1986), however, we would interpret the positive difference wave as a result of response-related negativity on go trials, not as an increase in inhibition-related positivity. Evidence for our interpretation comes from the finding that the positivity for go, but not for no-go trials, at the electrodes over the motor cortex, showed an interaction between the factors Response Side and Laterality ($F(4,6) = 13.82$, $P = 0.004$), which is typical for the contralaterally organized response-related potentials.

The result in Fig. 4 most relevant to the mechanisms of response inhibition is that a small negativity, the N200, interrupts the broad positivity at the frontal electrodes. The peak of the N200 is indicated in Fig. 4 by vertical arrows. The absolute amplitudes of the waveforms at the instant of the N200 peak are hardly different for go and no-go trials, but their morphology is completely different. On go trials there is no distinct N200, and the initial positivity is followed by response-related negativity. On no-go trials, by contrast, a distinct N200 peak is discernible on the positive-going edge of the P300, and the subsequent response-related negativity is absent. We estimated the N200 by calculating the peak negativity in the interval between 300 and 400 ms after the primary stimulus, only for no-go trials at frontal electrode sites, with reference to the immediately preceding positive peak (N200 onset). This analysis revealed that the N200 was bilaterally symmetrical but slightly

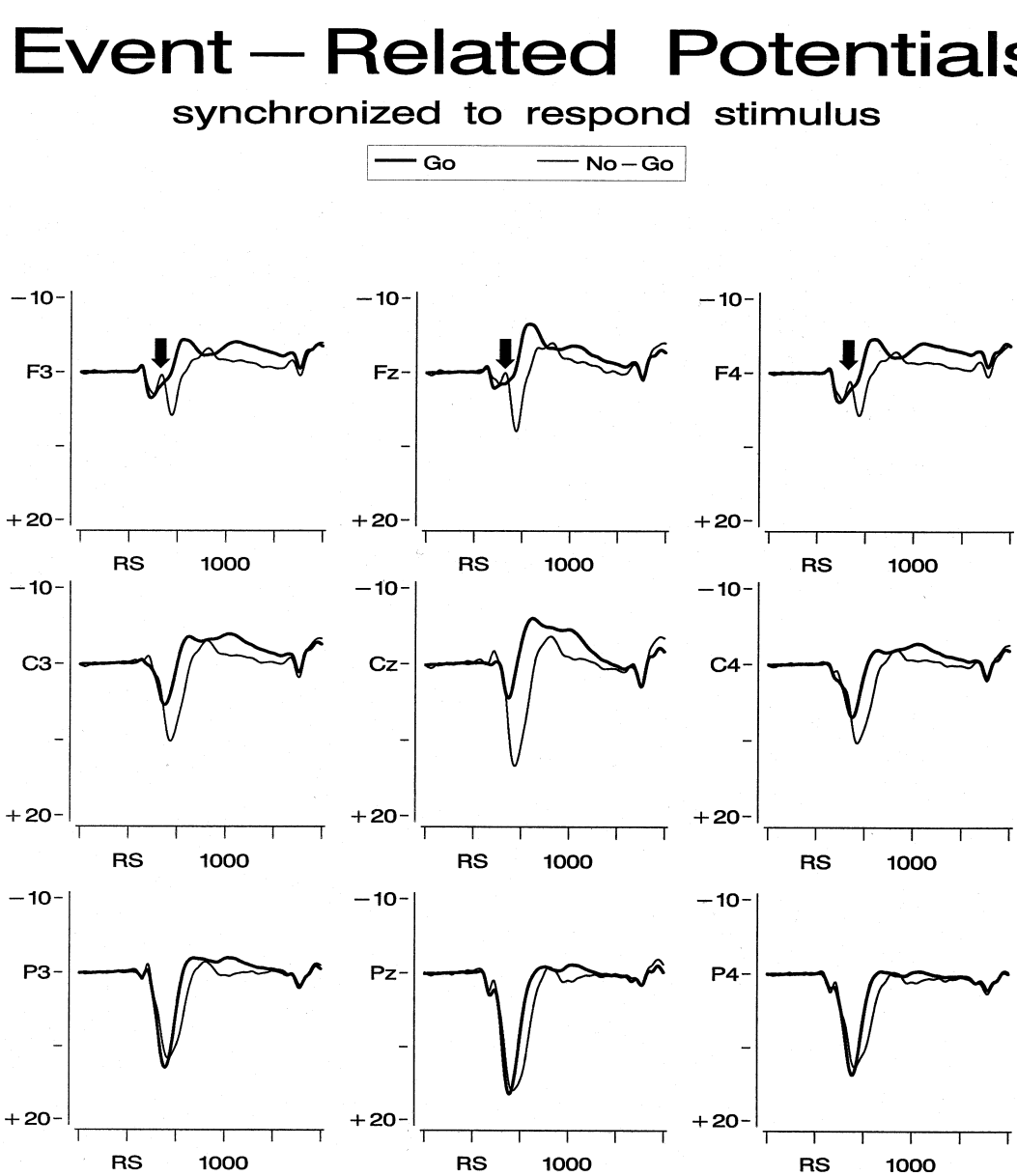

Fig. 4. Overview of event-related potentials for go and no-go trials. The waveforms represent grand averages, synchronized to the respond stimulus, over 10 participants, and both left and right hand responses. The data were smoothed with a spline function. The occurrence of the N200 is indicated by vertical arrows in the upper (frontal) panels.

smaller at the midline than at the lateral electrodes (Laterality: $F(2,8) = 7.48$, $P = 0.015$; contrast for mean of lateral electrodes versus midline electrode: $F(1,9) = 6.45$, $P = 0.032$). The N200 on no-go trials was not affected by variables commonly interpreted as signs of response activation. Neither was it different for left and right hand responses ($F(1,9) = 2.42$, $P = 0.154$), nor was the interaction between Response Side and Laterality found ($F(2,8) = 2.37$, $P = 0.156$). Therefore, together with the observation that there was no distinct N200 on go trials, we interpret the N200 as a cortical sign of stopping—specifically we conceive of it as an index of the central inhibitory mechanism.

The N200 was also present on signal-inhibit and signal-partial trials. Because the N200 is assumed to be elicited by the stop signal, we computed a new set of averages of all stop-signal trials, synchronized to the instant of stop signal presentation. A summary of these averages is shown in Fig. 5, in which the averages of no-go trials are included for comparison. The N200 (upper panel of Fig. 5) was statistically tested as the mean of the two lateral frontal electrodes, and seemed to be virtually identical for signal-inhibit and no-go trials ($F(1,9) = 1.09$, $P = 0.324$). The delay of the N200 on no-go as compared to signal-inhibit trials should be considered in the context of the stimulus conditions. On no-go trials a red arrow pointing left or right was presented as the primary stimulus, indicated by the word 'STOP' in Fig. 5. A no-go stimulus therefore contained two types of information; direction and color. The fact that participants processed both the direction information and the color information follows from the presence of the LRP on these trials (cf. Miller and Hackley, 1992). The signal-inhibit trials, by contrast, commanded 'stop' solely with a color change. The processing of the direction information could already have taken place immediately after the respond signal. Participants had only to process the color information after the stop stimulus. As a consequence, the stop signal could be processed more quickly than the no-go signal, and this might have resulted in the N200 latency difference.

On the assumption addressed above that the N200 on no-go trials is a manifestation of the central inhibition mechanism, it follows that a central mechanism was also invoked on signal-inhibit trials. This interpretation of the N200 results does not exclude the existence of a more peripherally operating mechanism, possibly operating in addition to a more central mechanism. Because partial responses are crucial in distinguishing between the two inhibitory mechanisms, we will analyze the N200 on signal-partial trials in some more detail.

4.2.4. The N200 on signal-partial and signal-respond trials

The two-mechanism view would either predict a similar or a smaller N200 on partial and full response trials, as compared to successful inhibit trials, because on those trials the central inhibitory mechanism is thought to be replaced by the peripheral mechanism. The upper panel of Fig. 5 shows that both partial and full response stop-signal trials were accompanied by a prominent frontal N200. The peak amplitude of the N200 was similar for signal-partial and signal-respond trials ($F(1,9) = 0.19$, $P = 0.673$), and the N200 for full and partial response trials averaged was greater than for signal-inhibit trials ($F(1,9) = 15.14$, $P < 0.004$). The N200

on stop signal trials exhibited a similar laterality effect at frontal electrodes as the N200 on no-go trials (Trial Type × Laterality: $F(4,6) = 31.29$, $P = 0.000$). This finding suggests that the negativity on partial and full response trials was indeed an N200. The greater N200 on signal-partial and signal-respond trials therefore seems to suggest that more central inhibition is needed as central response activation has progressed further.

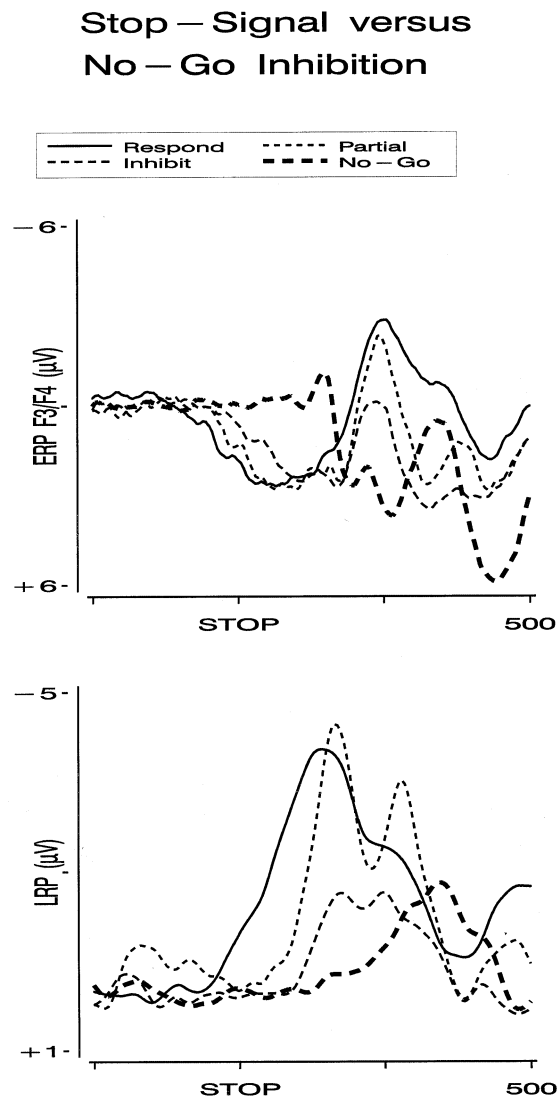

Fig. 5. N200 (mean of lateral frontal electrodes, upper panel) and lateralized readiness potential (LRP, lower panel) in response to no-go and more delayed stop signals. The waveforms are grand averages of ten participants and both left and right responses, synchronized with respect to the stop stimulus. The data were smoothed with a spline function.

The finding of a greater N200 on full and partial response trials might be seen as questioning the N200 association with inhibition. For instance, the additional negativity might be motor-related. In that case, the N200 should become more centrally distributed on partial and full response trials. However, we computed the contrast for frontal versus central electrode positions within trial types, and found no statistical difference (Trial Type × Position: $F(2,8) = 0.59$, $P = 0.578$). Another possibility is that the additional negativity was error-related (Falkenstein et al., 1991). We therefore further tested the association between inhibition and N200. We compared the N200 of efficient and less efficient inhibitors, based on a median split of stop-signal RT. The results of this analysis are depicted in Fig. 6. We found that the amplitude of the N200 in the group of fast inhibitors was slightly greater than in the group of slow inhibitors, but only for signal-partial and signal-respond trials (Group × Trial Type interaction: $F(2,7) = 4.79$, $P = 0.049$). The N200 for signal-inhibit trials was identical for the two performance groups ($F(1,8) = 0.28$, $P = 0.611$). No statistical differences in N200 amplitude were found when performance groups were formed using median splits of RT ($F(2,7) = 2.02$, $P = 0.203$), or the mean latency of the stop signal relative to the primary respond signal ($F(2,7) = 1.52$, $P = 0.283$). These findings are consistent with the interpretation of the N200 as a manifestation of inhibition.

Returning to the complete data set presented in Fig. 5, two further interesting aspects are noteworthy. The averages for signal-inhibit, signal-partial, and signal-respond trials show an almost identical N200 onset. N200 onset was scored as the instant of the most positive peak in the interval between 100 and 200 ms after the stop signal. The average onsets with respect to stop-signal presentation are detailed in Table 3 and did not differ between trial types ($F(2,8) = 0.40$, $P = 0.683$). Assuming that the N200 onset marks the instant at which the stop signal becomes effective, this finding relates to the assumption of the horse race model that the duration of the stopping process is constant, and to empirical observations that stopping time in young adults is constant across many experimental situations. In addition, the observed N200 onset latencies are very similar to the commonly found stopping time of 200 ms.

Another interesting aspect in Fig. 5 concerns the relation between the onset of the N200 and the peak of the LRP. The LRP starts to decrease approximately when the N200 starts to rise, at least for signal-inhibit and signal-partial trials (Table 3). These findings for complete and partial inhibits, seem to suggest that the central inhibitory mechanism, indexed by the frontal N200, attenuates respond processes, indexed by the LRP. Admittedly, this interpretation is speculative because it is based on coincidence not covariation, but as such it is reminiscent of the findings obtained in nonhuman primates that stimulation of the frontal cortex suppresses activity in the primary motor cortex (Sasaki et al., 1989). On signal-respond trials response processing seems fully developed before the inhibitory mechanism could exert its suppressive action, because in this case the LRP peak occurred before N200 onset.

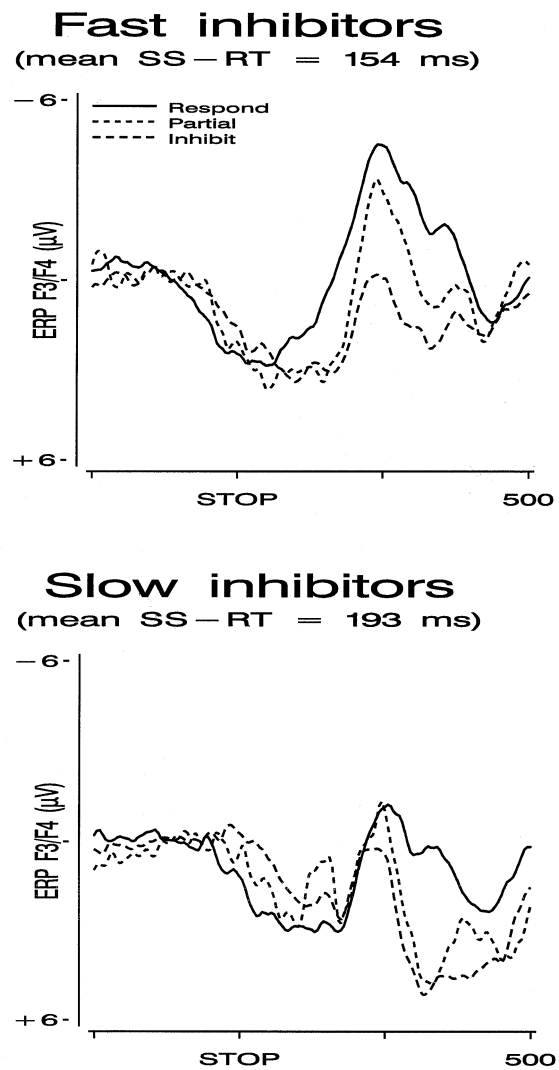

Fig. 6. N200 as a function of inhibition efficiency, as determined by median split of stop-signal reaction time (SSRT). The waveforms are averages of left and right frontal activity, and both response hands, over ten participants, time-locked to the stop signal. The data were smoothed with a spline function.

4.2.5. Cardiac deceleration

Fig. 7 details the pattern of cardiac deceleration observed in the present study. The most relevant heart beat is the beat following the beat of the stimulus (beat 1); the preceding beats serve as a baseline. Comparing no-go and stop-signal inhibition, we found that no-go and signal-inhibit trials showed a similar amount of cardiac deceleration (beat 1: $F(1,9) = 0.01$, $P = 0.923$). Importantly, cardiac deceleration on the first beat after the stimulus for partial inhibits was similar to successful

inhibition on stop-signal trials ($F(1,9) = 0.00$, $P = 0.999$), and to inhibition on no-go trials ($F(1,9) = 0.00$, $P = 0.999$). Signal-respond trials were accompanied by less deceleration than partial inhibits ($F(1,9) = 10.39$, $P = 0.010$). The cardiac deceleration results for stop-signal trials (Fig. 7) agree with the findings reported by Jennings et al. (1992). More generally, deceleration was greater for no-go relative to go (no-signal) trials on beat 1 ($F(1,9) = 29.13$, $P = 0.000$), but not on the preceding beats ($F(1,9) = 1.77$, $P = 0.216$). These findings corroborate earlier results obtained in a go/no-go task (Van der Molen et al., 1985; Van der Veen et al., 2000).

In sum, we observed a very similar pattern of cardiac slowing on all trials with successful response inhibition, complete or partial. The similarity of this pattern is suggestive of a single mechanism operating on all of these trial types. Partial and complete inhibit trials are associated with supra- and sub-threshold LRPs, respectively, and also with different above-threshold times. It can therefore also be concluded that cardiac deceleration is unrelated to LRP threshold crossing and above-threshold time.

Given the apparent association of N200 with stopping and the relationship of cardiac deceleration to stopping, we also assessed the sensitivity of the N200 to phase of the cardiac cycle. Inhibitory effects on the heart are known to be maximal early in the cardiac cycle, i.e. between the P- and T-waves of the electrocardiogram, and less effective late in the cycle (e.g. Lacey and Lacey, 1978; Jennings and Wood, 1977). Lacey and Lacey (1978) further suggested that sensorimotor processing by the cortex was facilitated early in the cardiac cycle. We therefore computed another set of N200 averages, synchronized to the stop stimulus, in which we compared whether the interval between the stop stimulus and the onset of the N200 was contained in either the first 450 ms after the R-wave of the heart beat or thereafter. N200 amplitudes, averaged over left and right frontal electrodes (F3 and F4), were found to be larger early relative to late in the cardiac cycle ($F(1,9) = 11.46$, $P = 0.008$). This finding seems to indicate that the N200 is enhanced when inhibitory influences related to cardiac timing are influencing the frontal cortex. As such, these findings relate cardiac deceleration to the frontal N200 and to the central mechanism of inhibitory control.

Table 3
N200 onset latency and LRP peak latency, in ms after the presentation of the stop signal, as a function of inhibitory success

Trial type	N200 onset	LRP peak
Signal-inhibit	209 (90)	199 (73)
Signal-partial	210 (101)	175 (73)
Signal-respond	198 (87)	129 (59)

Note: LRP, lateralized readiness potential. Standard error of the mean (S.E.M.) in parenthesis.

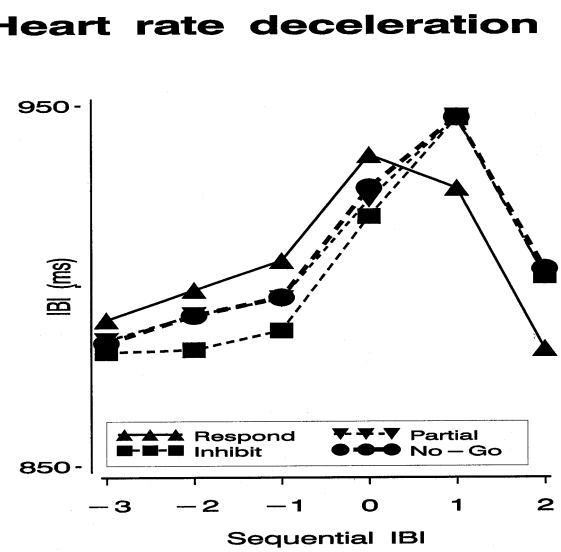

Fig. 7. Cardiac deceleration for completely successful (Inhibit), partially successful (Partial), and unsuccessful (Respond) stop-signal trials, in comparison to no-go trials. The waveforms are inter beat intervals averaged over ten participants, plotted against sequential inter beat interval, where beat 0 is the beat in which the respond stimulus occurred.

5. Further discussion and conclusions

The results of the present experiment replicate and extend the findings reported by De Jong et al. (1990). In both experiments participants were able to withhold responses within 200 ms after stop-signal presentation, while primary task performance remained fast and accurate. In fact, behavioral analyses in both experiments indicated that stop-signal presentation did not affect primary task performance in any way. In both studies the behavioral results fit the horse race model of inhibitory control reasonably well, and in neither experiment did the important assumption of independence between respond and stop processes appear to be seriously violated. The behavioral results of both experiments are thus in good agreement with the literature on stopping motor responses (Logan, 1994). In addition, the present results also agreed with those reported by De Jong et al. (1990) in that the LRP for failed and partial inhibits exceeded the LRP criterion amplitude associated with normal responding.

Apart from replicating the findings of De Jong et al. (1990), we attempted to take their analysis a step further to assess more closely the two mechanisms view of response inhibition that they advanced. We incorporated no-go trials into the design and analyzed psychophysiological measures from brain, heart, and muscles in great detail. We found that stopping to a no-go signal was associated with a negative frontal potential, the N200. We also observed cardiac deceleration associated with no-go stopping. The same pattern of results was found in the case of successful stopping in response to delayed stop signals, suggesting the same mechanism. The N200 can be interpreted as the reflection of a central ‘inhibit

signal' (see also, Kok, 1986). As the inhibit signal is raised, activity in the motor system is attenuated and the relevant motoric output is canceled. The reduced activity of the motor system was reflected in the attenuated LRP that we and De Jong et al. (1990) observed in these situations. Our analysis of the timing coincidence between the N200 and the LRP tentatively suggested that the LRP began to diminish roughly when the N200 started to increase. Four further arguments strengthen the view of the N200 as an inhibit signal. First, the timing of the N200 component corresponds quite well to the estimated inhibition times of about 200 ms observed in this and many other experiments. Second, N200 amplitude was related to inhibition efficiency, because it was greater in fast than in slow inhibitors. Third, N200 amplitude was larger at the time that cardiac inhibitory effects are known to be maximal. Finally, the view parallels neurophysiological findings obtained in nonhuman primates that stimulation of the frontal cortex attenuates activity in the motor cortex and response output (Sasaki et al., 1989). It thus seems that the present findings on successful inhibition, both in no-go and stop-signal situations, can be described by two independent (sets of) processes—a central respond process indexed by the LRP, and a single central inhibitory mechanism indexed by the frontal N200 and by cardiac deceleration.

When partial response output was observed, the negative frontal potential and cardiac deceleration were again found. Despite these indications of a central mechanism, however, LRP amplitude resembled that of full responses, suggesting a more peripheral mechanism that canceled the central motor output. Again, these findings replicate the results previously reported by De Jong et al. (1990). Because the distinction between the two inhibitory mechanisms was based almost exclusively on LRP threshold crossing, we attempted to collect additional evidence for the distinction by examining LRP duration. Accepting the existence of a threshold activity level in the primary motor cortex beyond which cortical outflow to more peripheral structures begins, we investigated whether above-threshold LRP duration was also the same for full and partial responses to stop signals. Our results indicated, however, that LRP duration (above-threshold) was positively related to response force. For long LRP durations, the resulting force exerted on the response device was high, as for full responses. Shorter durations resulted in lower forces produced, as on partial responses to stop signals. We would interpret these results as the effect of the stop signal exerted at the site at which the LRP is generated, that is, the primary motor cortex of the brain. It should be noted that the differences in LRP duration between full and partial response trials were independent of the exact threshold value. The absolute duration was longer when a lower threshold is used, but this increase did not influence the relative differences between full and partial responses.

The current findings are summarized in a simple diagram presented in Fig. 8. The schematic shows that extraction of the relevant features of the respond stimulus leads to lateralization of the motor cortex contralateral to the responding hand. As detailed in the introduction, the lateralization is the result of activity of the posterior loop through cerebellum and thalamus, operating after the anterior loop through the basal ganglia, which give rise to the GO-signal. The lateralization

becomes manifest in the LRP depicted in the upper panel of Fig. 8. At a certain level of LRP amplitude (the LRP threshold), cortical outflow to the periphery begins to arise, which leads, with a certain delay, to muscle activity (EMG, lower panel) and to the response (not shown). The transmission delay between the primary motor cortex and muscle activity is not shown in the diagram, but assumed to exist. The agent of inhibition, or stopping process, is shown in Fig. 8 at the middle left, and is shown to start at three different instants after the primary respond signal. The stopping process emits an inhibit signal to the motor cortex, the site of inhibition, from which outflow to the periphery was initiated. Upon arrival, the inhibit signal attenuates motor activity, which becomes manifest in a decrease of the LRP. The essence of the diagram is that the timing of the stopping process relative to the information flow in the response process determines whether a response occurs. We hypothesize that for complete inhibition, to no-go or to more delayed stop signals, the inhibit signal arrives at the motor cortex around time point A. At time point A the LRP has not yet fully developed and muscle activity has not

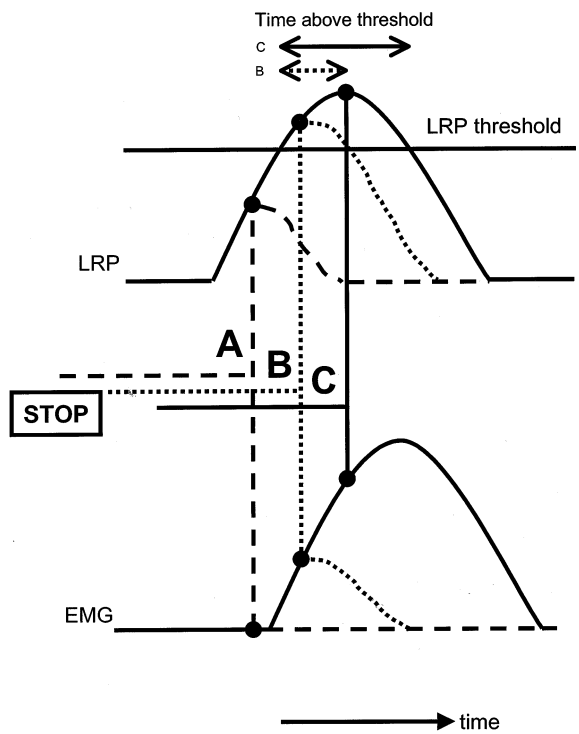

Fig. 8. Diagram representing the effect of different finishing times of the stopping process, relative to the respond process, on the lateralized readiness potential (LRP, top) and muscle activity (EMG, bottom). Time elapses from left to right. The lines A (dashed), B (dotted), and C (solid) show the effect of the relative finishing times for signal-inhibit, signal-partial, and signal-respond trials, respectively. The upper horizontal arrows indicate the difference in the above-threshold time of the LRP. See text for further detail.

started. Hence complete inhibits (signal-inhibit trials) are characterized by a sub-threshold LRP and no muscle activity. If the inhibit signal arrives around time point B, the LRP has increased above the threshold for a short time. Cortical outflow and muscle activity have already started, but the short duration did not allow that the response criterion set for the response device was reached. This situation represents the case for partial inhibits (signal-partial trials), which are thus accompanied by a supra-threshold LRP and partial muscle activity. Finally, on failed inhibits the inhibit signal becomes effective at time point C or later, when central response processes have fully developed. Failed inhibits (signal-respond trials) therefore show a longer supra-threshold LRP as well as fully developed muscle activity. Note that partial and failed inhibits are both accompanied by supra-threshold LRPs, but the duration above the threshold is different, as indicated by the upper horizontal arrows.

The diagram shows how psychophysiological data obtained in the stop-signal paradigm can be accounted for by a single inhibitory mechanism. An important difference between our single-mechanism view and the two-mechanism view entertained by De Jong et al. (1990, 1995) lies in the interpretation of the LRP response threshold. A peripheral inhibitory mechanism is necessary if threshold crossing is viewed as the cortical motor command. Given the issuing of central motor commands, attenuated muscle and force activity is then necessarily the result of a more peripherally-acting mechanism. If the duration of cortical motor output is taken into account, as the diagram details, the results can be more parsimoniously described by a single mechanism.

In sum, in the absence of conclusive evidence for a peripherally-acting mechanism, we propose that simple stopping to no-go and more delayed stop signals is accomplished by a single, central mechanism of inhibitory control. The inhibitory agent is located in the frontal cortex of the brain and is indexed by a negative frontal potential at the scalp and by cardiac deceleration. The site of inhibition is in the motor cortex of the brain. As in the horse race model, the timing relation between the respond and stopping process is sufficient for determining whether a full, a partial or no response occurs.

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