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In Search of the Point of No Return: The Control of Response Processes

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Control processes underlying reponse inhibition were examined. Six Ss performed a visual choice reaction task and were occasionally presented with a tone that told them to withhold the response. Reaction time results were in agreement with a model that assumes a race between response activation and response inhibition processes. Event-related brain potentials, electromyogram, and continuous response measures showed that responses could be interrupted at any time. Evidence was obtained for two inhibitory mechanisms: inhibition of central activation processes and inhibition of transmission of motor commands from central to peripheral structures. Results have implications for the distinction between controlled and ballistic processes.

The ability to inhibit planned actions is an important control option that allows efficient reactions to sudden changes in the environment. Such changes may arise as an unexpected consequence of one's own behavior, or they may be due to extraneous factors. In both cases, unexpected changes may render planned actions inappropriate, in which case these actions will need to be inhibited.

The ability to inhibit actions is evident for complex actions that require continuous control and take a substantial amount of time to execute. This ability is less obvious, however, for actions that are relatively simple or highly practiced, so that their execution takes only little time and appears to require little control once it has been initiated. In baseball, for instance, the breaking ball seems to lure the batter into the initiation of a swing that is difficult to interrupt or change if the ball suddenly changes it course. Also, the fact that complex actions can often be decomposed into a series of more elementary components raises the question of whether complex actions can be inhibited at any point during their execution or only at times when one component action has been completed and the subsequent one has to be initiated.

These issues have been the topic of a considerable amount of research (for a review, see Logan & Cowan, 1984). One of the aims of this research is to determine which of the processes underlying overt behavior are controlled and can be inhibited at any time and which, if any, are ballistic so that, once initiated, they necessarily go on to completion (Osman, Kornblum, & Meyer, 1986). Such investigations have commonly been conducted with some version of the stop-signal paradigm in which subjects are required to deliberately withhold or interrupt planned or ongoing actions. For instance, subjects are engaged in a reaction time task (e.g., discriminating visual stimuli), and, occasionally and unpredictably, they are presented with a signal (e.g., a tone) that instructs them to inhibit the response to the stimulus. The stop signal may occur at one of several delays following the presentation of the stimulus. Depending on the stop-signal delay, subjects will be more or less successful in withholding the response, being less successful with longer stop-signal delays. Following Logan and Cowan (1984), we refer to trials on which a stop signal occurred but the subject failed to withhold the response as *signal-respond* trials and to trials on which a stop-signal occurred and the subject did not respond as *signal-inhibit* trials. Trials on which no stop signal occurred are referred to as *no-signal* trials.

The Race Model

To interpret performance in the stop-signal paradigm, most investigators have relied on a race model. This model involves a race between two sets of processes (Logan & Cowan, 1984; Osman et al., 1986). For choice reaction time performance, the first set of processes underlies normal choice performance and includes stimulus recognition, response choice, and the preparation and execution of the response. The second set of processes is invoked when a stop signal occurs and includes the detection of the stop signal and the inhibition of the response. The first set starts with the presentation of the imperative stimulus, and the second set starts with the presentation of the stop signal. Whether a response occurs depends upon which set of processes wins the race. If the response processes win, then there is a response despite the stop signal. If the inhibition processes win, then no response occurs.

Full specification of the race model requires the identification of the point where the race ends. If primary-task processing can be interrupted at any time between stimulus presentation and the response, that point will correspond to the response itself. However, some researchers have maintained that processes that immediately precede overt movement may be ballistic. Once initiated, these processes necessarily go on to completion and result in the overt response. These ballistic processes are preceded by controlled processes that can be interrupted at any time. In this case, the race would effectively end at the point at which the ballistic processes start. Once

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response processing has reached its ballistic phase, inhibition processes can no longer intervene. Accordingly, the point at which processing becomes ballistic has been termed *the point of no return* (Bartlett, 1958; Osman et al., 1986).[1]

At present, there is little direct evidence for the existence of a final ballistic stage in choice reaction performance. Indeed, it is only recently that an experimental procedure has been developed that permits the investigation of the distinction between controlled and ballistic processes in choice reaction performance (Logan, 1981; Logan & Cowan, 1984; Osman et al., 1986). This procedure relies on the use of response functions in the stop-signal paradigm. Response functions give the probability of responding on stop trials as a function of stop-signal delay. It can be shown (Logan & Cowan, 1984; Osman et al., 1986) that any change in the mean of the response function equals the change in the mean duration of controlled processes minus the change in the mean duration of inhibition processes. Also, any change in mean reaction time on no-signal trials equals the sum of the change in the mean durations of controlled and ballistic processes. Under the assumption that inhibition processes remain unaffected by the experimental manipulation under study, the separate effects of this manipulation on controlled and ballistic processes can be obtained simply by comparing the effects on the mean reaction time on no-signal trials with the effects on the mean of the response function. However, the assumption that the inhibition processes remain unchanged is a crucial one; unfortunately, the validity of this assumption is not clear, and, in some circumstances, this validity may be difficult to assess.

In a number of studies the stop-signal paradigm has been used to infer the functional location of a possible point of no return. Typically, these studies involve the manipulation of factors that are believed to affect specific response-related processes, and the response-function procedure is used to separate experimental effects on controlled and ballistic processes in order to determine whether such response-related processes occurred before or after the point of no return. Factors that have been studied include the repetition of the stimulus–response pair from the preceding trial (Osman et al., 1986), stimulus–response compatibility (Logan, 1981), simple versus choice responses (Logan, Cowan, & Davis, 1984), and movement complexity (Logan, 1982; Osman, Kornblum, & Meyer, 1990). Two of these factors, repetition and simple versus choice responses, have been found to affect mean reaction time more than the mean of the response function, a result that might be taken to indicate that these factors affect the duration of ballistic processes. However, considerable caution should be exercised, because it is unclear whether inhibition processes remained unaffected by these manipulations. Indeed, repetition has been shown to have widespread effects throughout the information-processing system (Kornblum, 1973), and, as pointed out by Logan et al. (1984), choice reaction time may require more resources than simple reaction time and so may interfere more with inhibition processes.

Especially problematic for the notion of a final ballistic processing stage are the results obtained by Coles and coworkers in a more conventional choice reaction time paradigm (Coles, Gratton, Bashore, Eriksen, & Donchin, 1985; Eriksen, Coles, Morris, & O'Hara, 1985). They found a substantial number of cases in which the response was initiated at the level of muscle activation but not executed to criterion. Their results suggest that subjects sometimes were able to interrupt their responses in very late stages of response processing and thus cast considerable doubt on the notion that such stages are ballistic.

In the research reported here, we used psychophysiological measures of response-related processes in conjunction with behavioral measures to obtain converging evidence on the issue of controlled and ballistic stages in choice reaction time performance. Psychophysiological measures have been used previously to obtain detailed information about response-related processes in choice reaction time performance (Coles et al., 1985; Coles, Gratton, & Donchin, 1988; De Jong, Wierda, Mulder, & Mulder, 1988; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988). In the present experiment, these measures were used to investigate subjects' ability to interrupt ongoing operations at various stages of response processing.

The second purpose of this experiment was to use psychophysiological measures to obtain information about the mechanisms that underlie response inhibition in the stop-signal paradigm. The race model provides little insight into how inhibition processes actually succeed in interrupting response processes. As discussed below, continuous measures of brain activity, such as those provided by event-related brain potentials (ERPs), may provide direct information about the underlying mechanisms, information that cannot be easily obtained from behavioral measures alone.

Measures of Response-Related Processes

The first measure for assessing response-related processes involved the use of a dynamometer to provide a continuous registration of the force exerted by the subject when executing a response. Subjects were required to squeeze the dynamometer with a certain force in order to register a response. The use of an analogue response device with a fixed criterion level for a response to be counted, rather than a discrete manipulandum (such as a response button), made it possible to identify partial squeeze responses for which the squeeze was initiated but not completed. Thus, the use of dynamometers allowed us to assess subjects' ability to interrupt the actual execution of a brief squeeze response.

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[1] It is possible to define controlled processes more generally as processes that can be initiated without necessarily leading to the initiation of contingent processes or as processes that, once initiated, do not necessarily go on to completion. Similarly, ballistic processes can be defined more generally as processes that, once initiated, necessarily lead to the initiation of contingent processes or as processes that, once initiated, necessarily go on to completion. These definitions would not specify the order of controlled and ballistic processes and would not require that ballistic processes immediately precede the response (Logan, 1983). However, unless mentioned otherwise, we limit ourselves to the more restrictive definitions according to which ballistic processes can occur only in the final stages of processing and are preceded by a series of controlled processes.

The second measure used in this experiment is the electromyogram (EMG) recorded from muscles involved in response execution. Muscle activation must occur for an overt response to be executed. However, muscle activation might occur without an overt response if response processing can be interrupted after muscle activation has started but before the response has been completed. Furthermore, if response processing can be interrupted between the onset of muscle activity and the initiation of the overt movement, we should be able to find cases in which there is muscle activity but no overt movement (Coles et al., 1985). Thus, the use of EMG measures allowed us to assess subjects' ability to interrupt responses after muscle activation has begun but before the overt response has been completed or even initiated.

Finally, we used an analogue measure of central response activation. Premovement scalp-recorded potentials related to voluntary hand movements exhibit a gradually increasing negative shift, beginning 1 s or more prior to movement onset (Deecke, Grozinger, & Kornhuber, 1976). This negative, ramplike potential is known as the *readiness potential.* It has been found to be largest at scalp electrodes placed above motor-related cortical areas contralateral to the hand responsible for the movement (Kutas & Donchin, 1977, 1980). The readiness potential is also observed during the foreperiod of warned reaction time (RT) tasks (Kutas & Donchin, 1977; Rohrbaugh, Syndulko, & Lindsley, 1976). An average contralateral predominance for the readiness potential, however, is found only if the warning stimulus specifies the hand with which the subject should respond to the imperative stimulus (e.g., Rohrbaugh et al., 1976).

There is now considerable consensus that the lateralized part of the readiness potential reflects the differential involvement of left and right motor cortices in preparing to execute unimanual motor acts (Kutas & Donchin, 1980; Rohrbaugh & Gaillard, 1983). Recent neurophysiological and electrophysiological evidence implicate neural activity in the precentral motor cortex (M1) as the primary source of the lateralized potential (Arezzo & Vaughan, 1980; Brunia, 1980; Deecke, 1987; Requin, 1985). This cortical motor area is generally viewed as the major final common pathway for the central control of movements, being involved in the generation of movement-specific commands to peripheral motor structures, though the extent to which other motor areas may participate in this role is currently not clear (Wise, 1985). The amplitude of the lateralized readiness potential has been found to be rather insensitive to movement parameters other than movement side (left–right), such as required force (Kutas & Donchin, 1977) and direction (finger flexion or extension; Deecke, Eisinger, & Kornhuber, 1980). Thus, this potential seems to provide a suitable real-time index of central activation processes involved in the generation of motor commands specific to unimanual movements, being largely invariant over a range of movement parameters other than movement side.

Because motor-related lateralized potentials may be overlapped by a variety of lateralized potentials related to other functional and structural differences between the two hemispheres (Rugg, 1983; Tucker & Williamson, 1984), we used the difference in total lateralization for left-hand responses and right-hand responses. Taking this difference removes lateralized potentials of nonmotor origin that are common to left- and right-hand responses, so that the resulting index will exclusively reflect differential central response activation processes (De Jong et al., 1988); this index is called the *lateralized readiness potential.*[2]

The lateralized readiness potential (LRP) has been used in a number of studies to investigate the mechanisms and timing of motor processes (De Jong et al., 1988; Gratton et al., 1988; for a review, see Coles et al. 1988). In the present experiment it was used to investigate subjects' ability to inhibit the central activation of the response. Such an ability would manifest itself in an LRP that is "cut short" on at least some of the trials in which the response was withheld. Furthermore, as discussed in detail below, this measure was used to assess the relative importance of such a central inhibitory mechanism and other, more peripherally operating mechanisms for the inhibition of overt responses.

Experiment

The measures discussed in the previous section are particularly useful for addressing the issue of controlled and ballistic processes in choice reaction time. The trials of most interest in this respect are the signal-inhibit trials. By considering each of the measures, squeeze activity, EMG activity, and the LRP, for these trials, we can determine the location of a possible point of no return in the processing chain. Finding no LRP, EMG, and squeeze activity on signal-inhibit trials would indicate that the central activation and the execution of the response occurred after the point of no return. Finding LRP but no EMG and squeeze activity on signal-inhibit trials would allow us to locate the point of no return between the central activation and the execution of the response. Similarly, finding LRP and EMG but no squeeze activity on signal-inhibit trials would suggest that the point of no return occurs after muscle activation has started but before the overt movement has been initiated. Finally, finding LRP, EMG, and squeeze activity on signal-inhibit trials would argue against the notion of a final ballistic processing stage in choice reaction performance. Although such a result would not completely rule out ballistic processes as a theoretical possibility, it would serve to locate these processes in such a peripheral part of the processing chain as to be of negligible theoretical and practical significance.

In previous research (Gratton et al., 1988) we have found that partial central activation of incorrect responses sometimes occurred without any overt response activity. This finding suggests that subjects can inhibit or interrupt central response activation processes and, in that way, inhibit overt responses. It is possible, however, that other, more peripherally operating inhibitory mechanisms may be used to inhibit

[2] In formula, lateralized readiness potential = (C3′ − C4′) right hand − (C3′ − C4′) left hand, where C3′ − C4′ is the potential recorded between electrodes located above the hand areas in the left (C3′) and right (C4′) motor cortex. This measure is equivalent to what De Jong et al. (1988) called the *corrected motor asymmetry.*

responses, in addition to, or instead of, central response inhibition.

Bullock and Grossberg (1988) recently proposed a two-process theory for the planning, initiation, and execution of arm movements. One process is concerned with the programming of the movement, computing its direction from the present position and the target position of the limb. The other process, whose output is referred to as the GO signal, is concerned with the energetic aspects of the movement and is involved in the initiation of the movement and the control of its speed. Those investigators implicated the precentral motor cortex as the primary locus of the programming stage, whereas the energizing process was assumed to operate at a more peripheral level in the motor system, multiplying or shunting in a largely nonspecific way the output of the programming stage to control the onset and the speed of the movement. Bullock and Grossberg demonstrated that this two-process model can account for a variety of neurophysiological data and human motor performance results. It is of interest that they noted that in their model "very rapid freezing (of a movement) can be achieved by completely inhibiting the GO signal at any point in the trajectory" (Bullock & Grossberg, 1988, p. 69).

Peripheral motor inhibition has been demonstrated in sleep research. During periods of rapid eye movement (REM) sleep, cortical motor areas may become highly activated, but overt movement is prevented by inhibition of the motor neurons of the spinal cord. Several brainstem and midbrain structures have been shown to be involved in tonic motor inhibition during REM sleep (Morrison, 1979), and it is possible that the same peripheral inhibitory mechanisms may also play a role in the more phasic motor inhibitory phenomena in the stop-signal paradigm.

The possible involvement of peripheral inhibitory mechanisms in response inhibition in the present experiment can be investigated in at least two ways. From the evidence discussed above, it seems reasonable to assume that the LRP reflects the differential activation of the precentral motor cortices; therefore, inhibitory effects on neural activity in the precentral motor cortex should be evident as a decrease in LRP amplitude. The timing of such inhibitory effects on the LRP can then be compared with the timing of inhibitory effects on overt activity. If inhibition of central activation is solely responsible for response inhibition, then the inhibitory effects on the LRP should precede overt inhibitory effects by at least the time required for transmission of central commands to the peripheral motor system. As discussed below, the magnitude of such transmission delays is considerable and can be accurately estimated. An earlier onset of overt inhibitory effects or an approximately simultaneous onset of central and overt inhibitory effects would therefore provide strong evidence against a strictly causal relation between these effects and would suggest the additional involvement of peripheral inhibitory mechanisms in response inhibition.

Another test for the involvement of peripheral inhibitory mechanisms in response inhibition is provided by the LRP amplitude on signal-inhibit trials. In previous research we have found that EMG onset occurs when the LRP amplitude reaches a certain threshold value that is largely invariant over experimental conditions and different reaction time bins (Gratton et al., 1988). This finding implies a rather strict coupling between activity levels in the precentral motor cortex and the onset of motor activity under normal conditions. If response inhibition is effected only by inhibition of central response activation, then the LRP amplitude on signal-inhibit trials without any EMG or overt response activity should remain below the threshold value for EMG onset. On the other hand, finding that the LRP amplitude on such trials exceeds this threshold value would suggest the operation of a peripheral inhibitory mechanism that effectively prevents the occurrence of the normal motor consequences of central motor outflow.

Method

Subjects

Six students (5 males and 1 female) at the University of Illinois were paid $3.50 an hour, plus bonuses, for participation. The subjects (between 19 and 24 years of age) had normal or corrected-to-normal vision and hearing.

Apparatus and Stimuli

The stimuli for the choice reaction time tasks were the uppercase letters M, N, V, and W. On each trial, one of the letters was presented on a DEC VT-11 CRT display. The subject sat facing the screen at a distance of 1 m, such that the angle subtended by each letter was approximately 0.5°.

Each trial began with the presentation of a warning signal. This signal was diamond-shaped and centered around a central fixation dot. The warning signal shrank in five discrete steps of 60-ms duration from 1.0° to 0.1° of visual angle. It was then extinguished and was followed 700 ms later by the letter for that trial, which was exposed for 1,000 ms. After the letter was extinguished, the screen was blank for a 1,500-ms intertrial interval.

The stop signal was a tone (1000 Hz, 50 ms in duration, 65-dB amplitude), generated by a Schlumberger sine-square audio generator (Model SG-18A) and administered binaurally through headphones.

Subjects responded by squeezing one of two zero-displacement dynanometers (Daytronic Linear Velocity Force Transducers, Model 152A, with Conditioner Amplifiers, Model 830A; see Kutas & Donchin, 1977) with the left or the right hand, as a function of the letter. The system generated a voltage proportional to the force applied to the transducer, giving a continuous recording of the force output of both hands for 1000 ms following each stimulus. A Schmitt trigger could be set to any preselected force level such that when the exerted force reached this level, the system recorded the occurrence of an overt "criterion" response. Before the experiment, the value of each subjects' maximum force level was determined for each hand separately. Criterion values for each subject were set at 25% of the maximum force applied by that subject. During the first four blocks in the training session and the first two blocks in the experimental sessions, subjects received a click presented over a loudspeaker when the response force exceeded the criterion.

Procedure

The choice task involved classifying single letters from the stimulus set. The letters V and M were assigned to one response hand, and the letters W and N were assigned to the other.

Subjects participated in three sessions on consecutive days. They performed 18 blocks of 100 trials in each session. The first session was used for training. In the first 4 blocks, subjects practiced the choice task alone. In the remaining 14 blocks, subjects practiced the stopping task. A stop signal occurred on 39% of the trials, at one of three equiprobable delays. The order of trials in a block was completely randomized.

During the training session, stop-signal delays were adjusted individually for each subject by a staircase tracking algorithm (Levitt, 1971), yielding early, middle, and late stop signals, which resulted in the inhibition of 71%, 50%, or 29% of the responses in the choice task.[3]

The first two blocks of trials in the two experimental sessions were excluded from the analysis, and the experimental data were collected in the remaining 16 blocks. The stop-signal delays in the experimental sessions were fixed and set at the mean values obtained during the last 4 blocks in the training session.[4]

The primacy of the choice task was emphasized to the subjects. They were told to respond as quickly as possible while maintaining a high level of accuracy. They were also instructed not to delay their responses in anticipation of a stop signal but to make a concerted effort to stop themselves from responding if they detected a stop signal. It was explained to them that it would not always be possible to withhold the response and that in the training session the computer in fact changed the lags continuously so that they would be successful in withholding responses on approximately 50% of those trials for which a stop-signal occurred.

In order to reduce practice effects across sessions, the assignment of responses to the two subsets of stimulus letters alternated between sessions. The first two blocks of training in the experimental sessions served in part to minimize possible negative transfer between sessions.

Psychophysiological Recording

The electroencephalogram (EEG) was recorded from Fz, Cz, Pz (according to the 10/20 system; Jasper, 1958), C3′ (4 cm to the left of Cz), and C4′ (4 cm to the right of Cz), referenced to linked mastoids, using Beckman Ag/AgCl electrodes. Vertical and horizontal electrooculographic activity (EOG) was recorded using Beckman biopotential Ag/AgCl electrodes placed above and below the right eye and at 2 cm external to the outer canthus of each eye. Ground electrodes were placed on the forehead. The EMG was recorded by attaching pairs of Beckman electrodes to the right and left forearms, using standard forearm flexor placements (Lippold, 1967). For EEG and EOG electrodes, the impedance was less than 5 kΩ; for EMG it was less than 15 kΩ. The EEG and EOG signals were amplified by Grass amplifiers (Model 7P122) and filtered on-line, with a high frequency cutoff point at 35 Hz and a time constant of 8 s for the high-pass filter. The EMG signals were conditioned by using a Grass Model 7P3B preamplifier and integrator combination. The preamplifier had a half-amplitude low-frequency cutoff at 0.3 Hz, and the output of the integrator (full wave rectification) was passed through a filter with time constant of 0.05 s. For each psychophysiological measure (EEG, EOG, and EMG) and each trial, the derived voltages were digitized at 100 Hz for 2,100 ms, starting 100 ms prior to the onset of the warning stimulus and ending with the extinction of the stimulus letter.

Data Reduction and Analysis

The data analysis was organized within the framework of the race model for performance in the stop-signal paradigm. Given a few assumptions, the race model makes detailed predictions about the behavioral results. The first assumption is that primary-task processing and stop-signal processing proceed independently (see Logan & Cowan, 1984, for a discussion of the independence assumption). Second, the stop signal processing time is assumed to be constant. This assumption is unlikely to be valid in a strict sense. However, as shown below, even relatively major violations have only minor effects on the accuracy of the predictions derived from the race model (see also Logan & Cowan, 1984, pp. 326–327).

Under these assumptions, the distribution of primary-task reaction times on no-signal trials, and the probability of responding given a stop signal, can be used to predict mean reaction time on signal-respond trials and to estimate stop-signal reaction times. As depicted in Figure 1, the effect of a stop signal can be thought of as isolating from the reaction time distribution for no-signal reaction times a left part of the distribution that corresponds to the proportion of responses, P(respond), that were fast enough to escape inhibition. Mean signal-respond reaction time, then, corresponds to the mean of the part of the no-signal reaction time distribution lying to the left of the cutoff point. The reaction time at that percentile point represents the point in time at which the internal response to the stop signal must have occurred, relative to the onset of the primary-task stimulus. Subtracting out stop-signal delay yields an estimate of reaction time to the stop signal (see Logan & Cowan, 1984, for more details of these procedures).

More concretely, the distribution of no-signal primary-task reaction times was rank ordered, and the nth fastest value was determined, where n is the number of responses in the primary-task distribution multiplied by the probability of responding on stop-signal trials. Mean signal-respond reaction time was estimated by taking the mean of the n fastest no-signal reaction times. The nth value provided an estimate of the reaction time to the stop signal, relative to the onset of the primary-task stimulus. By subtracting out stop-signal delay, an estimate of stop-signal reaction time was obtained.

Motor Responses

As noted above, subjects were required to squeeze the dynamometers to a criterion of about 25% of maximum force to register a "criterion response." This response criterion was used for on-line feedback during training and classification of trials in terms of reaction time and success of response interruption. An additional overt response classification system was used during the off-line analysis. In particular, two additional measures of the latency of motor response were used: one based on the onset latency of the overt squeeze response and the other based on the onset latency of the EMG response.

The onset latencies of the overt squeeze response and the overt EMG response were determined for each no-signal and signal-respond

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[3] The staircase tracking algorithm adjusted the stop-signal delay according to different rules for the three delays: (a) For early stop signals, the delay was decreased each time the subject responded and was increased every other time the subject inhibited the response; (b) for middle stop signals, the delay was decreased each time the subject responded and was increased each time the subject inhibited the response; (c) for late stop signals, the delay was decreased every other time the subject responded and was increased each time the subject inhibited the response. These rules yield theoretical values of 29%, 50%, and 71% response probability, respectively (Osman et al., 1986). The signal delay was always adjusted in steps of 20 ms.

[4] We assumed that by the end of the training session performance would have reached a stable level so that these mean values would continue to produce the expected percentages of responses during the two experimental sessions. This assumption proved to be warranted.

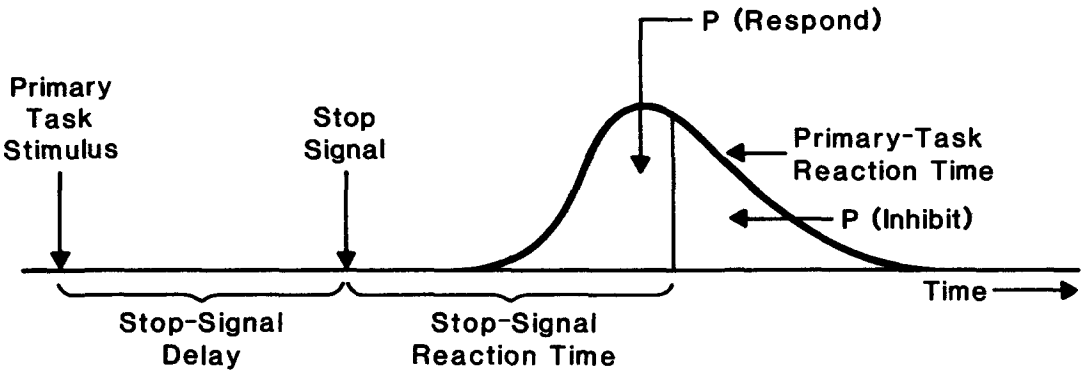

Figure 1. Illustration of the race model for response inhibition. (Depicted is a hypothetical no-signal reaction time distribution. This distribution is divided into two parts in which the left part corresponds to signal-respond trials and the right part to signal-inhibit trials. The location of the cutoff point in the distribution depends on the stop-signal delay and the stop-signal reaction time.)

trial according to the following procedure. For the interval during which the primary-task stimulus was presented (1,000 ms), a computer algorithm determined in the squeeze and EMG records the points that were followed by at least three consecutive points (corresponding to an interval of at least 30 ms) of successively higher amplitude. These points were considered candidate points for squeeze and EMG onset. In the next step of the algorithm, these candidate points were further inspected in a serial fashion, starting with the earliest candidate point for squeeze onset. For the current candidate point for squeeze onset, it was determined whether it was preceded by a candidate point for EMG onset by not more than 100 ms. In preliminary analyses, the interval between EMG onset and squeeze onset had been found to never exceed 100 ms for any of the subjects. If the interval between the two candidate points was less than 100 ms, these points were accepted as onset points for EMG and squeeze; otherwise, the algorithm skipped to the next candidate point for squeeze onset. This algorithm proved to be very robust and produced EMG and squeeze onset latencies for every trial on which the response reached criterion. Unfortunately, the algorithm could not be used for the detection of possible partial responses on signal-inhibit trials, because such responses could be of very short duration and, possibly, involve only EMG activity. For such trials the EMG and squeeze records were displayed, to allow the observer to decide whether and at what latency (to the nearest sample point, i.e., 10 ms) EMG onset and squeeze onset occurred. These decisions were made by eye under the following restrictions. First, EMG and squeeze amplitude had to exceed a fixed criterion that was based on the noise levels in the EMG and squeeze records during the 1,000-ms interval preceding the onset of the stimulus letter. Second, EMG and/or squeeze onset for partial responses should occur no sooner than 100 ms before and no later than 300 ms after the presentation of the stop signal. Finally, when both EMG and squeeze activity exceeded their criterion amplitude, squeeze onset should follow EMG onset by not more than 100 ms.

ERP Measures

For each trial the EEG data were corrected for both vertical and horizontal ocular movement artifacts by using a procedure based on that described by Gratton, Coles, and Donchin (1983). The corrected trials were then stored for further analysis. For the single trial analysis, the data from the five scalp electrodes (Fz, Cz, Pz, C3′, and C4′) were smoothed with a low-pass digital filter (high-frequency cutoff point at 8.0 Hz), and the baseline level was subtracted by averaging the first 10 points of the interval (corresponding to a 100-ms interval preceding the onset of the warning stimulus).

The lateralized readiness potential (LRP) was assessed by using the ERP waveforms recorded at C3′ and C4′. These electrodes were placed on scalp regions close to brain motor areas. Previous research has shown that the amplitude of the readiness potential is maximal at these locations when squeeze responses are required (Kutas & Donchin, 1977). The differences in potential between C3′ and C4′ (C3′ − C4′) was computed separately for left- and right-hand responses; the LRP was then computed by subtracting, point by point, the C3′ − C4′ difference potentials for left-hand responses from those for right-hand responses. In order to assess the statistical reliability of LRP amplitude being different from zero, a Wilcoxon rank sum test was performed on single-trial C3′−C4′ difference potentials, to test for the difference between left-hand responses and right-hand responses. This test was performed for each subject and for each of the 100 sample points (corresponding to 1,000 ms) following onset of the primary-task stimulus; prestimulus differences were corrected by subtracting a baseline voltage averaged over the 100 ms preceding stimulus onset. For each subject a new time series, consisting of Wilcoxon W statistics, was thus obtained. A statistical estimate of LRP onset latency was defined as the latency at which the W statistic first reached significance ($p < .01$, one-tailed). In order to assess the significance of LRP amplitude over subjects, a t test was performed at each of the 100 sample points, in which the hypothesis of zero average W statistic over subjects was tested. This procedure has been described more extensively elsewhere (van Dellen, Brookhuis, Mulder, Okita, & Mulder, 1985).

Results and Discussion

Reaction Time

Stop-signal delays, percentage of signal-respond trials, and mean reaction times for signal-respond trials for each subject and for each stop-signal delay are presented in Table 1. Predictions for mean signal-respond reaction time for each subject and stop-signal delay were derived from the proportion of signal-respond trials and the no-signal reaction time distribution, by using the procedure described in Data Reduction and Analysis. The predicted values are also presented in

Table 1
Stop-Signal Delays, Percentage of Responses, Mean Observed and Predicted Reaction Times For Signal-Respond Trials, and Estimated Stop-Signal Reaction Times

Variable	Subject 1	2	3	4	5	6	*M*
			Early stop signal				
Stop-signal delay	140	210	210	280	240	250	222
% responses	32.5	26.3	25.7	25.2	28.7	21.0	26.6
Observed RT	377	357	396	437	339	418	387
Predicted RT	371	355	388	424	335	405	380
Stop-signal RT	233	187	237	203	151	206	203
			Middle stop signal				
Stop-signal delay	190	270	280	350	300	320	285
% responses	42.8	48.9	45.3	45.7	46.3	38.5	44.6
Observed RT	393	379	421	476	372	435	413
Predicted RT	389	381	415	463	365	436	408
Stop-signal RT	218	178	214	195	147	175	188
			Late stop signal				
Stop-signal delay	260	330	370	420	360	400	357
% responses	65.6	78.6	77.9	67.6	76.5	66.9	72.2
Observed RT	417	418	460	505	410	470	447
Predicted RT	409	417	464	499	411	472	446
Stop-signal RT	196	202	239	198	164	181	197

Note. All times are in milliseconds. RT = reaction time.

Table 1. The correspondence between observed and predicted mean signal-respond reaction times is close, the largest discrepancy being 13 ms. Thus, a race model that assumes constant stop-signal reaction time fits the data very well. Stop-signal reaction times were estimated according to the procedure described in Data Reduction and Analysis. The estimated values for individual subjects and for each delay are presented in Table 1. In accordance with the values reported by Logan and Cowan (1984) from a similar experiment, stop-signal reaction time tends to be around 200 ms. Furthermore, the estimated values do not appear to vary systematically with stop-signal delay, a result that is consistent with the assumption of independence of primary-task and stop-signal processing.

As noted earlier, the assumption of invariant stop-signal reaction time is unrealistic. We therefore ran a number of simulations to test the behavior of the race model when stop-signal reaction time varies between trials, and to estimate the consequences of possible violations of the model's assumption of independence between primary-task and stop-signal processing. These simulations are described in detail in the Appendix. To summarize the results: If the covariance between primary-task and stop-signal reaction time is low, then variability in stop-signal reaction time has little effect on the accuracy of the predictions and estimates derived from the race model. If this covariance is high, however, these predictions and estimates deviate consistently and strongly from the actual values. Thus, the close fit between predicted and observed results in this experiment does not depend on the assumption of constant stop-signal reaction time, but it strongly suggests that the covariance between primary-task and stop-signal reaction time was only low and, therefore, that primary-task and inhibitory processing operated essentially independently without mutual interference or facilitation (see Appendix).

EMG and Squeeze

Partial responses were defined as responses that are incomplete in the sense that, though EMG and/or squeeze activity is present, the response does not reach criterion force. As we indicated above, the presence of partial responses on signal-inhibit trials would suggest that response processing can be interrupted in its late stages and, therefore, would argue against the notion of a ballistic final stage in response processing.

A substantial number of partial responses with both EMG and squeeze activity were found for signal-inhibit trials. Their mean absolute number for each signal delay is presented in Table 2. These numbers are also expressed as proportions of the total number of signal-inhibit trials. Also presented in Table 2 are the observed and predicted mean squeeze onset latencies for partial responses. The predicted onset latencies were derived by using a procedure analogous to the one used to derive predicted mean signal-respond reaction times. According to the race model, primary-task processing on partial response trials should be faster than on signal-inhibit trials with no partial response activity, but not fast enough to escape inhibition. Thus, as illustrated in Figure 2, partial responses should correspond to that part of the no-signal reaction time distribution that is located between a left-most part, which corresponds to signal-respond trials, and a right-most part, which corresponds to signal-inhibit trials with no partial response activity. This middle part of the distribution could be

Table 2
Partial Responses: Mean Absolute and Relative Number, and Mean Observed and Predicted Squeeze Onset Latency (in ms)

Stop-signal delay	Number	Percentage	Squeeze onset latency	
			Observed	Predicted
Early	27.3	13.8	378	377
Middle	29.5	21.0	429	431
Late	23.3	36.3	492	501

Note. Number is absolute number of signal-inhibit trials with partial muscle and squeeze activity; percentage is percentage of signal-inhibit trials with partial muscle and squeeze activity.

estimated from the proportion of signal-respond trials and the proportion of partial response trials. Predicted mean squeeze onset latency for partial responses was computed as the mean squeeze onset latency for no-signal reaction times from that part of the distribution.

A number of aspects of these results should be noted. First, the absolute number of partial responses was not significantly different for the different stop-signal delays, $F(2, 10) = 2.33$, $p > .1$, $MS_e = 17.32$. Because the total number of signal-inhibit trials decreased with stop-signal delay, the proportion of these trials for which partial squeeze activity was found increased strongly with stop-signal delay, $F(2, 10) = 59.5$, $p < .001$, $MS_e = 10.92$. Second, observed and predicted values for mean squeeze onset latencies for partial responses are in close correspondence. This correspondence is remarkable, given the rather limited number of observations on which they are based, and appears to provide strong support for the race model.

Because the finding of partial squeeze activity on signal-inhibit trials may have important implications for the issue of controlled and ballistic processes, it is important to determine whether the partial responses found in this experiment were actually *interrupted* responses. For this to be the case, two alternative interpretations of these partial responses must be ruled out. First, it may be that partial responses are in fact normal responses for which the subject intended to execute the response but failed to squeeze until criterion. This possibility can be addressed by considering the proportion of no-signal trials on which the response did not reach the criterion. This proportion was less than .006 for all subjects. This low proportion, relative to the proportion of partial responses on signal trials, rules out the interpretation of these partial responses as being merely incomplete responses.

The second interpretation of partial responses is based on the idea that subjects may delay their responses in order to enhance the chance of being able to withhold the response when asked to do so (Lappin & Eriksen, 1966; Logan, 1981; Logan & Burkell, 1986; Ollman, 1973). Thus, it is possible that, on occasion, subjects intentionally made a partial squeeze, completing the response only after they were confident that they would not be asked to withhold it. According to this interpretation, partial responses might be partially prepared responses instead of interrupted responses and therefore cannot be taken to indicate that response processing can be interrupted during response execution.

This interpretation accounts for the finding that an approximately equal number of partial responses was found for the different stop-signal delays; when subjects delayed the response in anticipation of a stop signal, they may usually have waited long enough to include the latest possible stop signal. However, in this case one should not only expect to find similar numbers of partial responses for the different stop-signal delays but also similar values for squeeze onset latency for partial responses. In fact, we found a highly significant increase in squeeze onset latency for partial responses with stop-signal delay (see Table 2), $F(2, 10) = 73.7$, $p < .001$, $MS_e = 265.7$. Such an increase was shown to be predicted by the race model. Hence, it is reasonable to conclude that the partial responses were, at least in large part, responses that were interrupted after the squeeze response had already been initiated.

The presence of partial squeeze responses indicates that response execution cannot be completely ballistic, but it does not reveal the degree to which execution processes can be controlled. However, the controllability of execution processes can be estimated by deriving predictions from the race model for the expected number of partial responses if execution processes were to be fully controlled. As shown in Figure

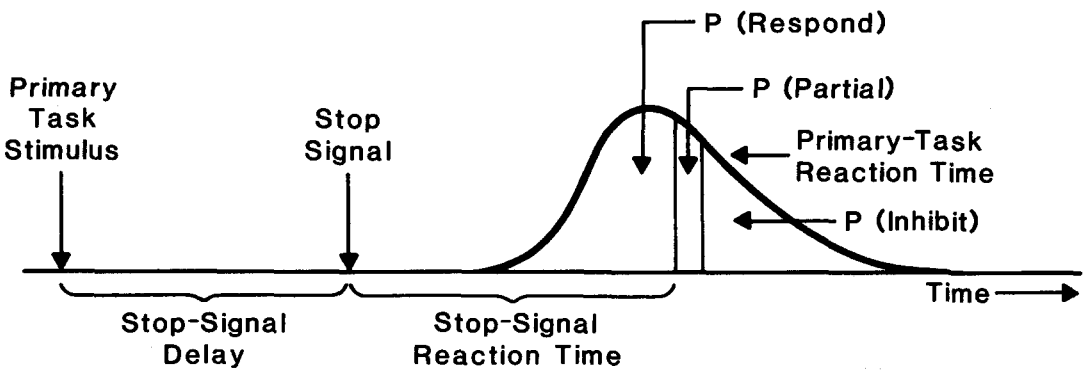

Figure 2. The race model extended to include partial responses. (Depicted is a hypothetical no-signal reaction time distribution. Signal-respond trials correspond to the left part of the distribution, partial responses to the middle part, and signal-inhibit trials to the right part.)

Table 3
Percentages of Partial Responses for Different Force Bins Defined by Maximum Squeeze Amplitude

Force bin	1	2	3	4	5	6	7	8	9	10
% responses	8.7	22.2	16.0	12.8	9.4	9.6	7.8	5.9	3.9	3.8

Note. Force bins are equally sized, with Bin 1 corresponding to 0%–10% of criterion force and Bin 10 corresponding to 90%–100% of criterion force.

2, partial responses correspond to a middle part of the no-signal reaction time distribution. If execution processes are fully controlled so that the squeeze response can be interrupted at any point before it reaches criterion, then the length of that middle part of the distribution should correspond to the interval between the onset of the squeeze and the time it reaches criterion. We estimated this interval for each subject and used it to compute the predicted percentages of partial responses for each signal delay. These percentages, averaged across subjects, were 22.0, 33.8, and 48.2, for early, middle, and late stop signals, respectively. These predicted percentages were significantly higher than those actually observed (see Table 2), $F(1, 5) = 15.8$, $p < .02$, $MS_e = 76.8$, with the interaction with stop-signal delay approaching significance, $F(2, 10) = 3.26$, $p < .08$, $MS_e = 9.03$.

The fact that the race model significantly overestimated the number of partial responses suggests that the control of execution processes is less than perfect. In order to investigate this issue further, partial responses were classified according to the maximum level of force attained, in one of 10 categories. The 10 categories corresponded to consecutive, equally sized force bins, with the first bin corresponding to 0%–10% and the last bin corresponding to 90%–100% of the force level required for a criterion response. Because the force-time characteristic of the initial squeeze response was approximately linear, this analysis allowed us to assess the relative frequency with which partial responses were interrupted at different latencies from squeeze onset. The relative frequencies, averaged across stop-signal delays, are presented in Table 3. With the exception of the first bin, progressively fewer partial responses were found at longer lags from squeeze onset. The low number of responses in the first bin is most likely due to sensitivity limitations that caused us to miss partial responses that reached only a very low level of force. The fact that partial responses were found even in the latest bins demonstrates that responses remained interruptible right up to the criterion force level. However, because fewer partial responses were found in the later bins, the controllability of the squeeze response appeared to decrease as the response gained momentum.

In contrast to the substantial number of partial responses with both EMG and squeeze activity, we found very few (less than 0.4%) partial responses with only EMG and no subsequent squeeze activity on signal-inhibit trials.[5] Thus, subjects were apparently unable to prevent the initiation of overt movement once muscle activation had started. Given the small interval between EMG onset and squeeze onset, this result could be taken to reflect the ballisticity of a short response-processing stage following EMG onset and preceding the initiation of overt movement (Osman et al., 1986). However, we prefer to interpret this result as being due to a fixed mechanical coupling between muscle activation and overt movement, with the short lag between EMG onset and the onset of the overt movement being caused by biomechanical factors and the excitation–contraction coupling in the muscles (Rozendal, 1984).

Lateralized Readiness Potential

The results discussed so far provide strong evidence against the notion of a final ballistic stage in speeded choice reaction performance or, equivalently, against the existence of a point of no return in response processing. In the following analyses we use the lateralized readiness potential to investigate two additional and related issues. The first issue concerns the actual mechanisms that underlie response inhibition and, more specifically, whether response inhibition in the present experiment might have resulted from the inhibition of central response activation processes. This latter possibility is suggested by the finding in earlier studies that EMG onset and onset of overt movement may start well before the LRP has reached its maximum amplitude (Gratton et al., 1988). This finding indicates that a response can be initiated while activation in the central motor system is still accumulating, which suggests that the progression of an overt movement may be controlled by a continuous output from ongoing central response activation processes to peripheral motor structures (Bullock & Grossberg, 1988). Interruption of this output flow

[5] This result contrasts with the results obtained in a previous study (Coles et al., 1985) in which a large number of EMG-only partial responses were found. Several differences between the two experiments may have contributed to this inconsistency. First, instructions in the earlier experiment emphasized speed, whereas those in the present experiments emphasized both speed and accuracy. The greater emphasis on speed may have resulted in a larger number of partial EMG responses. Second and most important, subjects in the earlier experiment were urged to keep their hands off the dynamometers before making a response. Thus, partial EMG responses may have resulted from small movements that did not bring the hands in contact with the dynamometers and, therefore, did not result in any measurable squeeze activity. In the present experiment, subjects had their hands continuously on the dynamometers, so that any small movement would result in measurable squeeze activity. In this experiment a large number of partial EMG responses could be obtained if the criterion for EMG detection was made more sensitive. However, in contrast to the results for partial squeeze responses, these responses occurred equally frequently on the correct and incorrect response side, and their onset was not different for the different stop-signal delays. These results suggest that the increase in the number of partial EMG responses (resulting from a lower detection criterion) was attributable to a misclassification of trials for which there was only noise on the EMG channel.

by way of inhibition of the central response activation processes, then, would provide a mechanism whereby the overt response can be inhibited or interrupted before it reaches criterion. The second issue concerns the question, discussed earlier, whether such a central inhibitory mechanism alone can account for the motor inhibition results in this experiment.

As we noted earlier, we assume that the LRP reflects the differential involvement of left and right motor cortex in the generation of motor commands specific to unimanual movements. Possible nonmotor contributions to this lateralized brain potential were removed by taking the difference between left- and right-hand responses. In the first analysis we determined whether there can be LRP activity without any EMG activity or overt movement. We computed the LRPs for signal-inhibit trials without any EMG activity or overt movement and for signal-respond trials for each stop-signal delay.

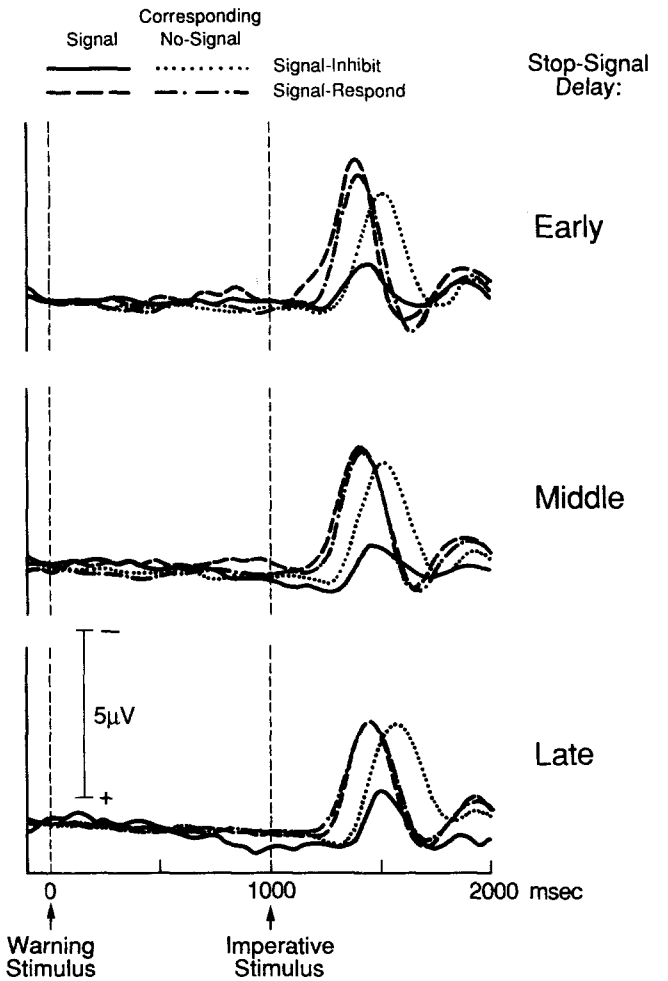

Figure 3. Waveforms of the lateralized readiness potential for signal-respond and signal-inhibit trials, and for the corresponding no-signal trials. (Upward [negative] deflections indicate a greater negativity over the hemisphere contralateral to the correct response hand. Top panel: Early stop signal. Middle panel: Middle stop signal. Bottom panel: Late stop signal.)

We also computed the LRPs for the no-signal trials from the parts of the no-signal reaction time distribution that correspond to the proportions of signal-inhibit and signal-respond trials for each delay (P(inhibit) and P(respond), see Figure 2). These LRP waveforms, averaged across subjects, are shown in Figure 3. Two main aspects of these waveforms should be noted. First, except for small and inconsistent prestimulus differences, the waveforms for signal-respond trials and those for the corresponding no-signal trials show an almost perfect overlap. This result suggests that central response activation processes remained completely unaffected by the processing of the stop signal on signal-respond trials. This is consistent with the assumption of the race model that primary-task processing and stop-signal processing are independent.

Additional evidence that response-related processes remained unaffected by stop-signal processing on signal-respond trials can be obtained from consideration of the intervals between LRP onset and EMG onset, between EMG onset and squeeze onset, and between squeeze onset and reaction time (the latency of criterion squeeze). The length of these intervals can be used to estimate the durations of subsequent stages in response processing. The mean length of these intervals for no-signal and signal-respond trials for each of the stop-signal delays is presented in Table 4. The length of the three intervals can be seen to be essentially the same for no-signal and signal-respond trials. None of the differences approached significance, suggesting once more that response processing on signal-respond trials remained unaffected by the processing of the stop signal.

The second main feature of the LRP waveforms in Figure 3 is that for signal-inhibit trials there is for each delay an initial development of the LRP that appears to be subsequently interrupted. As tested by the combined Wilcoxon *t* test, the partial LRP reached a highly significant amplitude for each delay ($ps < .0001$). It should be recalled that for these signal-inhibit trials no EMG or squeeze activity was found. These results, therefore, indicate that central response activation processes can operate without necessarily leading to any overt motor activity.

Unfortunately, the fact that the LRP waveform for signal-inhibit trials looks like an interrupted version of the waveform for corresponding no-signal trials does not provide unequivocal evidence that central response activation processes can in fact be interrupted. This is because the waveform for signal-inhibit trials could have resulted from the averaging over signal-inhibit trials on which central response activation pro-

Table 4
Mean Intervals Between LRP Onset and EMG Onset, EMG Onset and Squeeze Onset, and Squeeze Onset and Reaction Time (in ms)

Trial type	LRP–EMG	EMG–squeeze	Squeeze–RT
No signal	109.4	34.3	51.5
Early signal-respond	102.6	34.5	52.2
Middle signal-respond	107.3	34.3	52.1
Late signal-respond	112.8	34.1	51.6

Note. LRP = lateralized readiness potential; EMG = electromyogram; RT = reaction time.

cesses started relatively early and went on until completion, and signal-inhibit trials on which inhibition succeeded in preventing such processes from starting at all. Because the LRP can be obtained only as an average across a large number of trials and because we have no independent means of determining on each single signal-inhibit trial whether central response activation processes had begun, we cannot exclude accounts for the partial LRP waveforms for signal-inhibit trials in terms of a mixture of trials for which central response activation processes either did not start at all or started and went on to completion.

A possible way around this problem is to use partial response trials for which we can be certain that central activation processes must have been at least initiated. If central response activation processes, once started, necessarily go on to completion, then the LRP waveforms for partial responses and no-signal responses should be similar. On the other hand, if central response activation processes were actually interrupted in the case of partial responses, then the LRP amplitude for such responses should be smaller than for no-signal responses. Because the number of partial responses for each stop-signal delay was generally too small to obtain reliable waveforms when averaging with respect to stimulus onset, the LRP waveforms in the present analysis were obtained by averaging with respect to EMG onset. This procedure allowed us to collapse partial responses across stop-signal delays and, in general, provides more robust LRP waveforms as the time course of poststimulus LRP development has been found to be rather tightly coupled to the timing of the overt response (De Jong et al., 1988). The averaging period for partial responses and no-signal responses ranged from 200 ms before until 200 ms after EMG onset, with the 100-ms interval preceding this period serving as the baseline. The data for 1 subject were omitted from this analysis because she produced too few partial responses.

The resulting LRP waveforms, averaged across the remaining subjects, are shown in Figure 4. As can be seen from this figure, the waveforms for partial responses and no-signal responses are virtually identical until shortly after EMG onset, after which the waveform for no-signal responses increases further in amplitude and the waveform for partial responses levels off. The maximum LRP amplitude was significantly larger for no-signal responses, $t(4) = 2.97$, $p < .025$. These results demonstrate that central response activation processes were indeed subject to inhibition. We now turn to the question whether the inhibition of central activation processes alone can account for the inhibition or interruption of overt responses in the present experiment.

As can be seen from Figure 4, inhibitory effects on central activation processes for partial responses were not evident until about 30 ms after EMG onset. The question is whether these effects could have caused the timely interruption of the overt response on these trials. To answer this question, we need to estimate the minimum delay for changes in central activation processes to become evident in the overt movement. This delay consists of two components, the first concerning the delay for changes in central activation processes to result in changes in EMG activity and the second the delay for changes in EMG activity to result in changes in overt

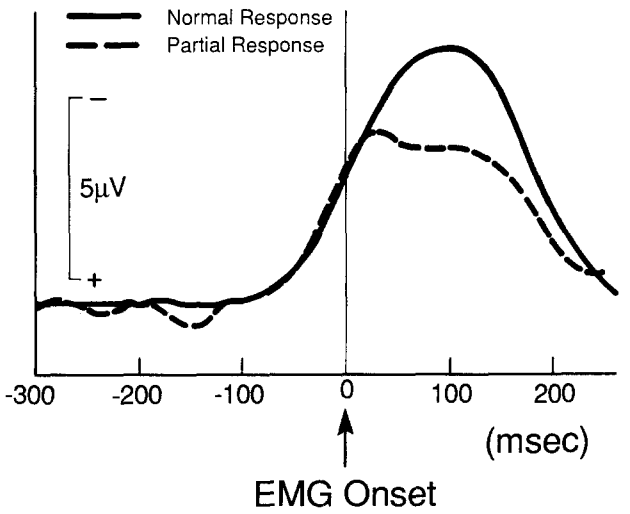

Figure 4. Waveforms of the lateralized readiness potential for partial response and for no-signal trials. (These waveforms were obtained from averaging with respect to electromyographic [EMG] onset. Upward deflections indicate a greater negativity over the hemisphere contralateral to the correct response hand.)

movement. From studies with transcranial stimulation of the cortico-spinal system in humans, the first component can be estimated to be at least 20 ms (Benecke, Meyer, Gohmann, & Conrad, 1988), and the second component can be estimated (Rozendal, 1984; see also Table 4) to be at least 30 ms. Taken together, these values indicate that interruption of the overt movement caused by the interruption of central activation processes could not occur until at least 80 ms after EMG onset on partial response trials (i.e., the 30 ms by which inhibitory effects on the LRP followed EMG onset plus an estimated 50 ms due to transmission delays). However, our previous results (Tables 3 and 4) indicate that on the majority of partial response trials interruption of the movement had already taken place within 60 ms after EMG onset. This analysis, therefore, suggests that it is unlikely that interruption of the overt movement on partial response trials was due solely to interruption of central response activation processes.

Converging evidence for the latter conclusion can be obtained from the comparison of the maximum LRP amplitude for signal-inhibit trials with the mean LRP amplitude at EMG onset for corresponding no-signal trials. In a previous study, we found that the average LRP amplitude at the time of EMG onset was invariant across experimental conditions and reaction time bins (Gratton et al., 1988). This finding of an invariant relation between LRP amplitude and time of EMG onset was interpreted to indicate that responses are initiated when central response activation reaches a relatively fixed criterion level or threshold. If this fixed-criterion hypothesis is correct, mean LRP amplitude on signal-inhibit trials should remain below the criterion amplitude on no-signal trials.

In the present analysis, we measured for each subject and each stop-signal delay the maximum amplitude reached by the LRP on signal-inhibit trials without any EMG activity, and the mean LRP amplitude at the time of EMG onset on

the no-signal trials that correspond to those signal-inhibit trials. The resulting amplitudes, averaged across subjects, are shown in Table 5. Several aspects of these results are noteworthy. First, consistent with previous findings (Gratton et al., 1988), mean LRP amplitude at EMG onset was not different for the three groups of corresponding no-signal trials ($F < 1$) despite the fact that mean EMG onset latency was different for the three groups. Second, for signal-inhibit trials the maximum LRP amplitude increased with stop-signal delay, $F(2, 10) = 13.29$, $p < .01$, $MS_e = 0.114$. This result is probably due to the fact that processing at shorter delays is more likely to have been interrupted before central response activation processes could have started, so that relatively fewer trials would contribute to the average LRP waveform at shorter stop-signal delays.

Third and most important, the maximum LRP amplitude on signal-inhibit trials exceeded the criterion LRP amplitude on no-signal trials for middle and late stop-signal delays. This occurred even though this criterion value slightly overestimates the mean LRP amplitude at which response initiation commands are sent out by the central motor system because of the lag of at least 20 ms involved in the transmission from the central motor system to the arm muscles. Maximum LRP amplitudes for signal-inhibit trials that exceeded the criterion value were found for 2 of the 6 subjects for the early, for 4 subjects for the middle, and for 5 subjects for the late delays.

These data indicate that levels of central response activation, which in the absence of the stop signal would have been associated with the initiation of the overt response, became dissociated from response initiation on signal-inhibit trials. This suggests the presence of an inhibitory mechanism that operates at a level peripheral to central activation processes and that prevents central motor processes from gaining control over the peripheral motor system, possibly by intercepting or blocking motor commands issued by the central motor system. However, alternative interpretations of these data are possible; in the next section we use the results for midline ERPs to examine some of the alternatives in more detail.

Midline ERPs

The LRP reflects central processes that differentially contribute to the activation of left- and right-hand responses. For brevity, these processes have been denoted as central response activation process, but is is possible that processes that do not distinguish between response sides may be crucially involved in central response activation. Inhibition of these activation processes, which are nonspecific with respect to response side, may have played an important role in the inhibition or interruption of responses. Furthermore, it is possible that inhibition of nonspecific activation, which would not be evident in the LRP, may have preceded the inhibition of response-specific activation. Thus, the previous results for LRP may still be compatible with a strictly central locus of response inhibition, and further evidence is required to evaluate the possible role of peripheral inhibitory mechanisms in response inhibition.

The analysis of midline event-related potentials (ERPs) was specifically aimed at the identification of components related to the processing of the stop signal and subsequent inhibitory processing. We computed mean EPRs at Fz, Cz, and Pz for signal-inhibit trials, excluding partial responses, and for signal-respond trials, separately for each stop-signal delay. Also, we computed mean ERPs for the no-signal trials from the corresponding parts of the no-signal reaction time distribution (see Figure 3). The resulting grand average waveforms are presented in Figure 5.

Of primary interest are the differences between the waveforms for signal-inhibit and signal-respond trials and those for the corresponding no-signal trials. Such differences should reflect the processing of the stop signal and, especially for signal-inhibit trials, the operation of inhibitory processes. Some of these differences can be seen in the grand average waveforms presented in Figure 5. However, these waveforms were not time-locked to the stop signal. Thus, any ERP responses to the stop signal will be smeared because of the considerable differences in stop-signal delays between subjects. Better estimates of the effects of the stop signal were obtained by deriving difference waveforms that are adjusted for differences in stop-signal delay. More specifically, these difference waveforms were obtained by subtracting from the ERPs for signal-inhibit and signal-respond trials the ERPs for the corresponding no-signal trials. These difference waveforms were computed over an interval of 600 ms, starting 100 ms before the time of onset of the stop signal. Finally, these waveforms were averaged over subjects.[6]

The resulting adjusted difference waveforms for the midline positions (Fz, Cz, and Pz) are shown in Figure 6, separately for signal-inhibit and signal-respond trials, and for each stop-signal delay. These waveforms appear to consist of two distinct deflections; an early negative deflection that is present for both signal-inhibit and signal-respond trials and a later positive deflection that is prominent for signal-inhibit trials but

Table 5
Mean LRP Amplitude at EMG Onset on No-Signal Trials and Maximum Mean LRP Amplitude on Signal-Inhibit Trials

	Stop-signal delay		
Trial	Early	Middle	Late
No signal	1.83	1.83	2.00
Signal-inhibit	1.67	2.08	2.67

Note. Amplitude is in microvolts. LRP = lateralized readiness potential; EMG = electromyogram.

[6] The use of subtraction techniques to isolate components related to one of the constituent tasks from the overall potentials during concurrent task performance requires great caution. In general, such techniques can be justified only when (a) the independence of the constituent tasks has been determined from behavioral measures and (b) the resulting components are independent of the relative timing of the constituent tasks. These criteria were met in the present application of the subtraction technique.

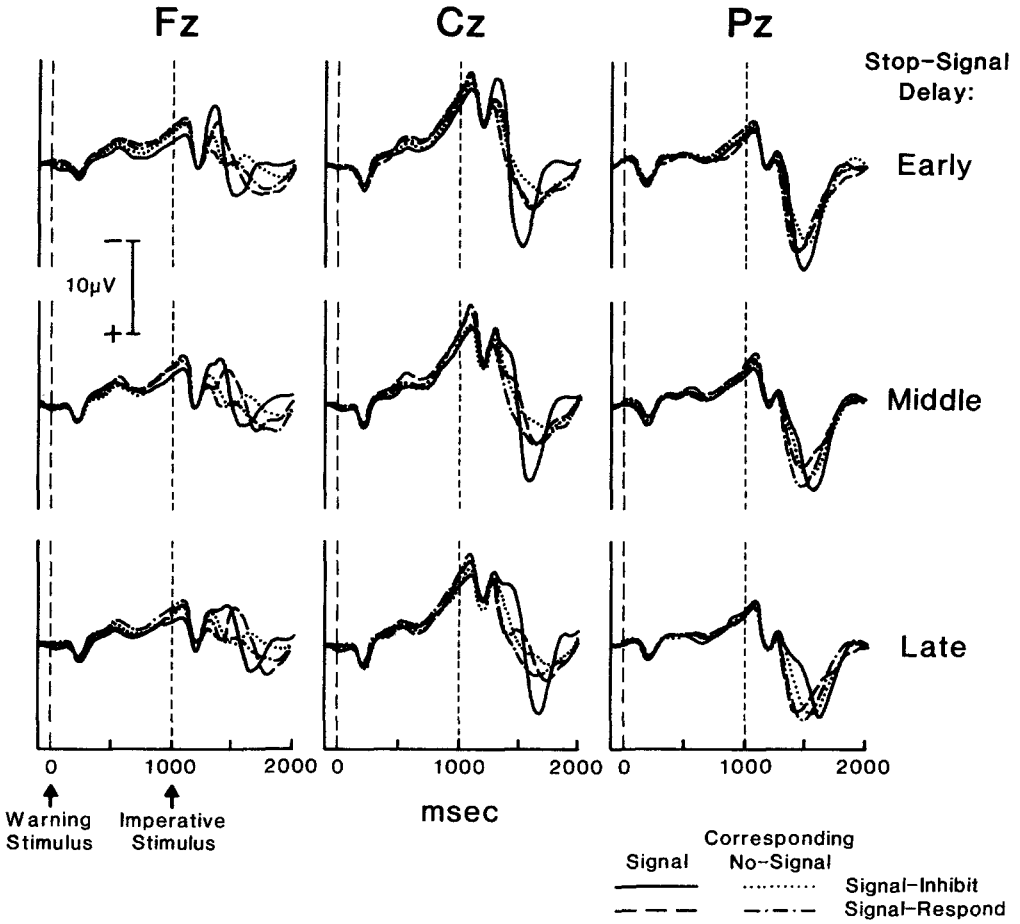

Figure 5. Grand average waveforms for Fz (left column), Cz (middle column), and Pz (right column) for signal-respond and signal-inhibit trials, and for the corresponding no-signal trials. (Top panel: Early stop signal. Middle panel: Middle stop signal. Bottom panel: Late stop signal.)

appears to be almost absent for signal-respond trials. It should be noted that the waveforms for early, middle, and late stop signals are very similar. The derivation of the difference waveforms for the three stop-signal delays involved quite different portions of the primary-task ERPs. Thus, the similarity between the waveforms for different delays both indicates the validity of the subtraction technique for obtaining potentials that are related to the processing of the stop signal and provides another indication of the independence between primary-task processing and stop-signal processing.

The fronto-central distribution of the early negative component of the difference waveforms and its peak latency of about 130 ms suggest that it can be identified with the N1 component that has been found to be elicited by presentation of auditory stimuli. The N1 component is known to have a clear fronto-central predominance and a peak latency between 100 and 150 ms after stimulus onset. Also, this component is known to be largely exogenous, reflecting physical parameters of the auditory stimulus but not its processing requirements (for a review, see Näätänen & Picton, 1987). The exogenous nature of the N1 component is consistent with the fact that the negative component of the difference waveform is present for both signal-inhibit and signal-respond trials. Indeed, there were no significant differences for peak amplitude and peak latency between signal-inhibit and signal-respond trials for this component.

The positive deflection of the difference waveforms also has a fronto-central scalp distribution. This scalp distribution suggests that this deflection is not a classical P300 component invoked by processing of the stop signal. One of the defining characteristics of the classical P300 component is a centro-parietal scalp distribution (Donchin, 1981). Indeed, as can be seen for no-signal trials in Figure 5, a P300 component with a centro-parietal maximum and a peak latency of about 500 ms *was* found to be invoked by primary-task processing. Moreover, it is not clear why a P300 should be invoked by processing of the stop signal on signal-inhibit trials but not on signal-respond trials; according to the race model, processing of the stop signal should proceed in the same way on signal-inhibit and signal-respond trials. Thus, the positive component would appear to be related to processes that differentiate between signal-respond and signal-inhibit trials, that is, to those processes related to the actual inhibition of the response.

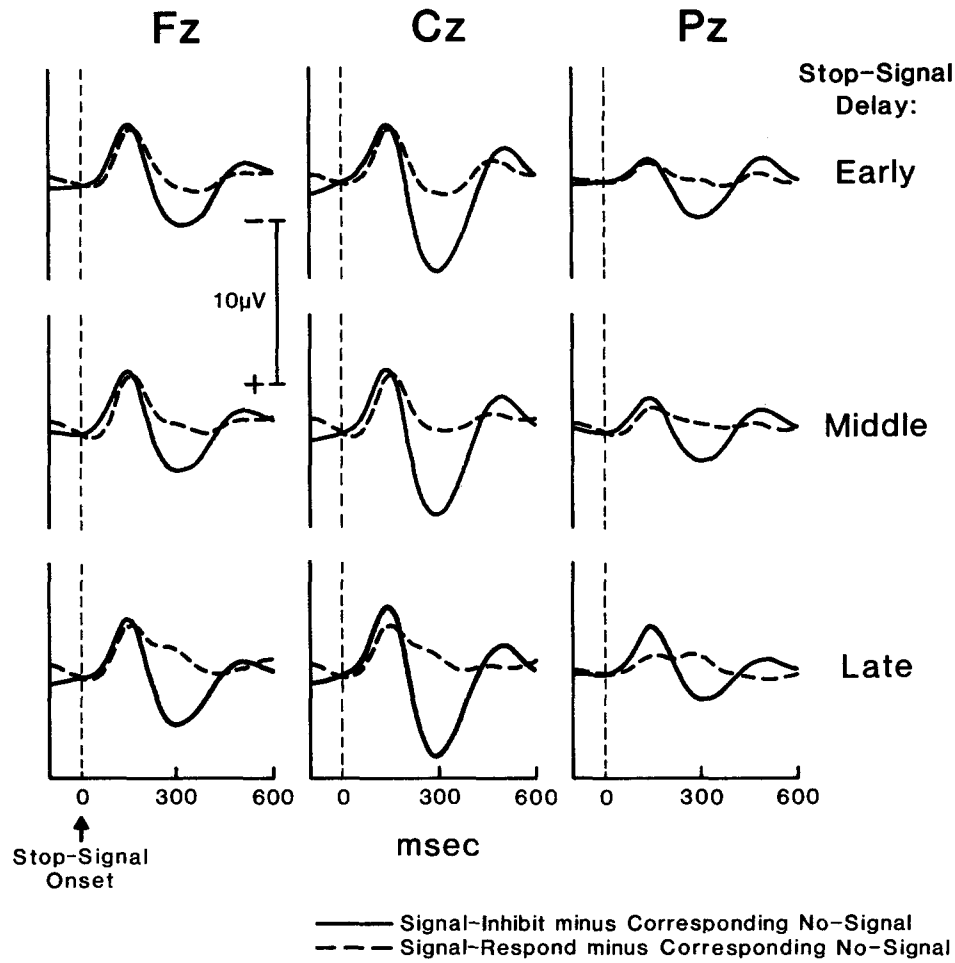

Figure 6. Difference waveforms for Fz (left column), Cz (middle column), and Pz (right column). (These waveforms represent the difference between the averaged waveforms for signal-respond and signal-inhibit trials and the averaged waveforms for the corresponding no-signal trials in Figure 5. Upward deflections correspond to a greater negativity on signal-inhibit or signal-respond trials. Top panel: Early stop signal. Middle panel: Middle stop signal. Bottom panel: Late stop signal.)

An increase in positivity at fronto-central locations has also been observed for no-go trials in go/no-go reaction time tasks (Kok, 1986; Pfefferbaum, Ford, Weller, & Kopell, 1985). As the response has to be withheld on no-go trials, it seems reasonable to assume that this effect is related to the positive deflection we observed on signal-inhibit trials. An increase in fronto-central positivity has also been observed on no-go trials when the task was to count specific target stimuli (Pfefferbaum et al., 1985). Thus, the positive deflection seems to occur whenever a cognitive task is interrupted, independently of particular motor sets.

That the positive component of the difference waveform was present only for signal-inhibit trials indicates that it is not related to stop-signal processing per se but rather to the successful inhibition of the response. An important question is whether the inhibition-related processes that are reflected by the positive component may have caused the actual inhibition of the response. This question can again be addressed by considering the timing of this component in relation to the timing of inhibitory effects on the overt response.

The onset of the positive component of the difference waveform could not be accurately determined because it is overlapped by the N1 component. However, it does not seem to start until at least 150 ms after onset of the stop signal. Stop-signal reaction time, representing the completion time of inhibitory processing was estimated to be about 200 ms (see Table 1). These considerations suggest that the positive component might be directly related to the actual inhibition of the response. However, partial responses were excluded in the analysis of midline ERPs, so that the criterion for successful inhibition in this analysis was the absence of any EMG activity rather than the absence of a criterion response. Consequently, one should compare the time of onset of the positive component with the estimated inhibitory processing time with respect to the onset of EMG, this latter time representing the mean time it takes stop-signal processing to

inhibit the onset of muscle activity. To estimate this time, we followed the same procedure as for the criterion response, but now with the onset of the EMG, instead of the criterion response, serving as the overt response measure. Thus, in this analysis, partial responses were counted as full responses. Mean estimated stop-signal reaction times with respect to EMG onset were 140, 129, and 132 ms for early, middle, and late stop signals. These values are smaller than the estimated onset time for the positive component of the difference waveform, 150 ms. Furthermore, as mentioned before, the manifestation of changes in central response activation processes in muscle activity involves a transmission delay of at least 20 ms so that the effect of the processes reflected by the positive component would not become apparent in the EMG response until at least 20 ms after its onset. Consequently, it seems necessary to assume that on at least some of the signal-inhibit trials processes other than those reflected by this component were responsible for the inhibition of the response.

It is interesting to observe that the timing of the positive midline difference potential and of the inhibitory effects on the LRP appear to be very similar. This suggests that the positive component may reflect the interruption of nonspecific response activation processes that occurs at about the same time as the interruption of response-specific activation processes. This interpretation of the positive component is consistent with the evidence from other studies that the occurrence of this component does not depend on particular motor sets. The similar timing of the two central inhibitory manifestations suggests that they may reflect a general inhibitory effect on central response activation processes. Our analyses suggest that this central inhibitory effect cannot fully account for the inhibitory effects on more peripheral response processes. The results do not conclusively rule out the possibility of a strictly central locus of response inhibition in the present experiment because of the possibility of some other central inhibitory process that may have been crucially involved in response inhibition but did not become evident in the scalp-recorded brain potentials. However, our results are at least consistent with the existence of a more peripherally operating inhibitory mechanism that was instrumental in the actual inhibition of the response on a considerable part of the trials.

General Discussion

Response interruption can be modeled as a race between two sets of processes: (a) response activation processes that involve the planning, the initiation, and the execution of the response and (b) inhibition processes that disrupt normal response processing and result in the withholding or interruption of the response. The finishing times of the two sets of processes are assumed to be independent random variables. As a consequence, response processing should remain unaffected by inhibition processes if these processes fail to actually inhibit the response. The validity of this assumption is indicated by several aspects of the data. First, the race model provided an excellent fit to the experimental data. Using a series of simulations, we showed that systematic deviations from the predictions of the race model should have been expected if there had been stochastic dependence between the durations of response activation and response inhibition processes. Second, we found that the relative timing of several consecutive stages in the activation and execution of the response, as indicated by LRP onset, EMG onset, movement onset, and the final reaction time, was the same for no-signal and signal-respond trials. Finally, we used a subtraction technique to estimate the contribution of inhibition-related processes to the overall scalp-recorded brain potentials. This contribution was found to be the same for the different stop-signal delays despite the fact that the subtraction involved quite different pairs of the overall brain potential for the different delays. This finding indicates that inhibitory processing proceeded in the same way regardless of the current stage of concurrent primary-task processing. Taken together, these results provide strong evidence for the independence of response activation and inhibition processes.

The finding that response activation and inhibition processes operated independently is important, because it rules out an alternative to the race model. It is conceivable that inhibition processes might result in a gradually accumulating inhibitory activity that, over time, becomes increasingly powerful in counteracting response activation. If this is the case, inhibition might be expected to affect response activation processes even in those cases in which inhibitory activity did not accumulate fast enough to prevent activation processes from resulting in an overt response. The fact that no evidence for such braking effects was found suggests that, instead, inhibition processes produce a discrete and powerful effect on response activation processes that, if it occurs in time, is highly effective in disrupting such processes. Limitations on the effectiveness of inhibition processes became evident only when the response was close to criterion and had presumably attained considerable momentum.

One of the main questions in the present research concerned the notion of the "point of no return": In which stage is response processing controlled so that it can be interrupted, and in which stage is response processing ballistic so that, once initiated, it always leads to overt movement? We found evidence that central response activation processes, as reflected by the lateralized readiness potential, can be interrupted. Furthermore, we found that the overt response could be interrupted up to the very moment at which it reached criterion. Thus, the evidence suggests that a response can be inhibited at any time during its activation and execution and argues strongly against the notion that there is a final ballistic stage, or a point of no return, in response processing.

The conclusion that there is no ballistic process immediately prior to the response does not imply that there are no ballistic processes earlier on. Retinal processes, for example, are almost certainly ballistic, as may be many other perceptual processes. Highly automatic cognitive processes may be ballistic as well.[7] The stop-signal methodology applies to the

[7] It is not necessary that processes be highly automatic to be ballistic. Processes are ballistic if they run on to completion without intention; they need not begin without intention. By contrast, automatic processes may either begin without intention, or run on to completion without intention, or, in case of highly automatic, autonomous processes, both begin and run on to completion without intention (see Zbrodoff & Logan, 1986). Thus, automaticity is the more general concept that includes ballisticity as a special case.

overt response and so would be insensitive to possible prior ballistic processes. Other methods must be developed to assess the ballisticity of prior perceptual or cognitive processes. This task is difficult because these processes are not as directly observable as motor processes. One possibility is to look at aftereffects of processing, such as memory and repetition effects, to infer whether processing could be inhibited (e.g., Logan, 1983; Zbrodoff & Logan, 1986).

The second main question in the present experiment concerned the nature and locus of response inhibition processes. Previously we have introduced the notion of a response channel (Coles & Gratton, 1986; Gratton et al., 1988). In this conception, a response channel involves a complex of motor structures whose activities are related to the overt response. Different degrees of involvement of response structures are viewed as different degrees of response channel activation, such that at specific, and relatively invariant, levels of activation the response is initiated. In this conception of a response channel, it seems natural to assume that the progression of response execution depends critically upon a continuous input from central to peripheral motor structures (Bullock & Grossberg, 1988).

Within this theoretical framework, a possible mechanism for response inhibition involves the inhibition of central response activation processes. Thus, response initiation might be inhibited by preventing central response activation from reaching the criterion level, and interruption of an already initiated response can be achieved by discontinuing the output from central to peripheral motor structures. However, three findings suggest that inhibition of central response activation processes was not the only mechanism for response inhibition in this experiment. First, on trials with a partial response the interruption of the LRP occurred too late to be directly responsible for the interruption of the overt response. Second, on trials on which subjects succeeded in withholding the response without any muscle activity or overt movement, the LRP was found to reach amplitudes that under normal conditions would have been sufficient for muscle activity and overt movement to be initiated. Finally, when the response was successfully inhibited, a large fronto-central positivity was found that was hypothesized to reflect the interruption of general central activation processes. However, this positivity appeared to develop only *after* the time at which the earliest inhibitory effects on muscle activity had become evident.

Taken together, these results suggest that response inhibition did not always depend on the inhibition of activity in central motor structures. These findings can be explained by assuming that even if central inhibition processes do not succeed in preventing central motor outflow, the overt response can be inhibited or interrupted by preventing the transmission of such outflow to peripheral motor structures. Consistent with the evidence obtained in this and related experiments (for a review, see Logan & Cowan, 1984), such a peripherally operating inhibitory mechanism may be assumed to operate in a fast and highly effective way and to interfere little with central processing structures that are involved in primary-task processing. The distinction between a central and a peripheral inhibitory mechanism is also consistent with the distinction between central processes, concerned with the programming of the movement, and more peripheral processes, involved in the initiation of the movement and the control of its speed, proposed by Bullock and Grossberg (1988) in their model for the control of limb movements.

The most obvious difference between central and peripheral inhibitory mechanisms is the speed at which the two mechanisms can achieve motor inhibition; if central activation has progressed too far to be interruptible by central inhibition, interruption or inhibition of the overt movement may still be achieved by means of peripheral inhibition. Further study is required to assess other possible functional differences between the two mechanisms. Of special interest is the degree of selectivity of motor inhibition that can be achieved by these mechanisms. In the model of Bullock and Grossberg (1988), peripheral processes multiply or shunt outputs of central processes in a largely nonspecific way. Thus, it is possible that the peripheral inhibitory mechanism may be useful only when, as in the present study, actions have to be inhibited or interrupted nonselectively whereas slower but possibly more flexible central inhibitory mechanisms may become crucially involved when selective motor inhibition is required; for example, in a choice reaction time task, when the incorrect response has been initiated, a selective inhibitory mechanism would permit the simultaneous interruption of the incorrect response and initiation of the correct response (e.g., Gratton et al., 1988). A nonspecific inhibitory mechanism would lead to the inhibition of both incorrect and correct responses, so that initiation of the correct response would have to be postponed until the inhibition of the incorrect response has been completed. These issues will be addressed in future research.

To summarize, the combined use of reaction time and psychophysiological measures has allowed us to explore response inhibition in the stop-signal paradigm in considerable detail. A race model provides a very good account of both overt behavioral and psychophysiological data. The data suggest that response processes can be inhibited at any time before the response reaches criterion, a result arguing against the notion of a final ballistic stage in response processing. The evidence also suggests that response inhibition involved both the inhibition of central response activation and a more peripherally operating mechanism.

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Appendix

Monte Carlo Simulations of the Horse Race Model

The assumption of constant stop-signal reaction time (RT) is likely to be violated in practice. Given some degree of variability in this RT, there exist several ways in which stochastic dependencies between primary-task RT and stop-signal RT may violate the independence assumption made by the race model. We performed a series of Monte Carlo simulations to study the effects of violations of these assumptions on the performance of the race model.

In the general case of stochastic stop-signal RTs, the independence assumption of the race model holds that the primary-task RT distribution is the same for signal and no-signal trials and that primary-task and stop-signal processing times are stochastically independent. Under these assumptions, the distribution function of stop-signal RTs [$F_s(t)$] can be shown to be given by

$$F_s(t - t_d) = 1 - P(\text{respond}) \times f_{sr}(t) / f_n(t),$$

where t_d is the stop-signal delay, P(respond) is the probability of a response on signal trials, and $f_{sr}(t)$ and $f_n(t)$ are the probability-density functions of signal-respond and no-signal RTs, respectively. All terms on the right side of this equation can be estimated from the data so that the stop-signal RT distribution function can be estimated as well. If the actual distribution of stop-signal RTs is assumed to be the same for all values of the stop-signal delay, then the estimated distribution function should also remain invariant across different stop-signal delays. Thus, differences between the estimated distribution functions at different stop-signal delays would indicate violations of the independence assumptions of the race model. We did not follow this approach for two reasons. First, reliable estimation of the stop-signal RT distribution function requires a very large number of trials. Second, in addition to testing whether the assumptions underlying the race model are strictly valid, we wanted to estimate the effects of possible violations of these assumptions on the accuracy of the predictions and estimates derived from the race model.

In the simulations, we used convolutions of a normal and exponential distribution to represent the distributions for primary-task RTs and stop-signal RTs. Such convolutions has been shown to provide an excellent fit to RT distributions in a variety of paradigms (e.g., Ratcliff, 1978, 1979). The convolution parameters for the primary-task distribution used in these simulations, the mean and standard deviation of the normal distribution, and the time constant of the exponential distribution were 400 ms, 40 ms, and 100 ms, respectively; these values were chosen to given an overall mean and standard deviation similar to those obtained experimentally. For the stop-signal RT distribution, we used two sets of convolution parametes. For the first set, the mean and standard deviation of the normal distribution were 175 ms and 10 ms, and the time constant for the exponential distribution was 25 ms. For the second set, the parameter values were 134 ms, 20 ms, and 66 ms, respectively. The mean and standard deviations for the resulting stop-signal RT distributions were 200 ms and 27 ms for the first and 200 ms and 69 ms for the second distribution. It should be noted that standard deviations for simple RTs with variable foreperiods are commonly found to fall in the range of 25 ms to 82 ms (Luce, 1986). Finally, we ran a simulation in which stop-signal reaction was fixed at 200 ms.

For variable stop-signal RTs, we considered three cases. In the first case, primary-task and stop-signal RTs were negatively correlated (−.5). Such a negative correlation would result if primary-task processing and inhibitory processing competed for the same resources, so that speeding up one process by devoting more resources to it would slow down the other process (Navon & Gopher, 1979). In the second case, primary-task and stop-signal RTs were uncorrelated. In the third case, these times were positively correlated (.5). A positive correlation might be caused by fluctuations in general arousal that have similar effects on primary-task processing and inhibitory processing. For each of the cases, the simulation involved 30,000 trials, on 40% of which a stop signal occurred at one of three equiprobable delays. The results of each simulation were analyzed by the same method used in analyzing the experimental data.

The results of the simulations are presented in Table A1. Of primary interest is the extent to which these results exhibit the two major characteristics of the experimental data: the close correspondence between observed and predicted RT and the relative invariance of estimated stop-signal RT as a function of signal delay. It is not surprising that when the assumptions of the race model are met exactly (i.e., constant stop-signal RT), these two features are present. For the case of negatively correlated RTs, an almost perfect correspondence between observed and predicted RTs is obtained. However, unlike the experimental data, the simulation data for this case exhibit a strong and consistent decrease of estimated stop-signal RT with increasing delay, both for small and large variance in stop-signal RT. When RTs are uncorrelated and the variance of stop-signal RT is relatively small, a close correspondence between observed and predicted RTs is found as well as an almost constant estimated stop-signal RT as a function of delay. When stop-signal RT variance is relatively large, differences between observed and predicted RTs are evident, as well as a consistent tendency for estimated stop-signal RT to decrease with longer delays. Finally, when RTs are positively correlated, clear differences between observed and predicted RTs are apparent, especially when stop-signal RT variability is relatively large. Furthermore, estimated stop-signal RT is found to increase with increasing stop-signal delay, especially when variability in stop-signal RTs is relatively small.

The results of these simulations show that the race model can accommodate some degree of variability in stop-signal RT, but much less so negative or positive dependencies between primary-task pro-

Table A1
Stop-Signal Delays (in ms), Percentage of Responses, Mean Observed and Predicted Reaction Times for Signal-Response Trials, and Estimated Stop-Signal Reaction Times: Simulation Results

		Variable					
		$r = -.5$		$r = .0$		$r = .5$	
Delay/measure	Constant	S1	S2	S1	S2	S1	S2
230							
% responses	26.4	36.1	37.1	27.1	28.6	18.2	17.3
Observed RT	396	408	413	401	421	393	434
Predicted RT	394	408	409	396	399	382	383
Stop-signal RT	200	219	220	201	205	182	181
280							
% responses	53.0	50.8	49.1	52.8	45.3	51.5	42.4
Observed RT	425	422	423	426	432	431	447
Predicted RT	425	424	422	425	418	423	414
Stop-signal RT	201	199	193	202	187	198	179
330							
% responses	69.4	63.2	56.2	70.7	64.4	75.8	74.9
Observed RT	444	438	430	447	444	460	456
Predicted RT	443	438	429	445	439	456	450
Stop-Signal RT	200	178	159	201	181	225	218

Note. All times are in milliseconds. RT = reaction time. Constant = constant stop-signal RT. Variable = variable stop-signal RT (r = across-trial correlation between primary-task RT and stop-signal RT). S = simulation. Distribution parameters (M, SD, and time constant) were 175, 10, and 25 ms for Simulation 1 and 134, 20, and 66 ms for Simulation 2.

cessing and inhibitory processing. Therefore, although the good fit between the data and predictions derived from the race model in the present experiment does not depend on the assumption of constant stop-signal RT, it does suggest that, relative to the variance in primary-task RTs, the variance in stop-signal RTs is small. Thus, the assumption of constant stop-signal RT, although unlikely to be correct in a strict sense, appears to provide a reasonable approximation. Furthermore, the good fit strongly suggests stochastic independence between the duration of primary-task and inhibitory processing.

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