

The Point of No Return in Choice Reaction Time: Controlled and Ballistic Stages of Response Preparation

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A countermanding procedure and race model are used to assess separately the effects of experimental factors before and after the "point of no return" in response preparation. The results reveal details about processes that so closely precede the initiation of movement that they cannot be inhibited. These processes appear to be affected by the repetition of stimulus-response pairs, but not by the physical or semantic properties of the stimuli. A model of response preparation is supported in which (a) response inhibition depends upon the outcome of a race between independent excitatory and inhibitory processes, and (b) reaction time is the sum of the durations of at least two stages, separated by the point of no return.

Cognitive psychology has traditionally been concerned with *knowing*. However, during the past decade, many cognitive psychologists have begun to focus their attention on *doing*. One avenue that the psychological study of action has taken is to investigate the mental processes that underlie physical movement (e.g., Rosenbaum, 1980; Rumelhart & Norman, 1982; Sternberg, Monsell, Knoll, & Wright, 1978). Various processes have been postulated as occurring prior to physical movement. An important difference between these processes concerns whether, once begun, they must necessarily lead to the start of overt movement.

This difference can be characterized in terms of a distinction between *controlled* and *ballistic* processes. Ballistic processes are ones that immediately precede, and are inextricably linked to, overt movement. Once having been launched, they must proceed to completion, and, upon completion, necessitate the start of overt movement. An example of such processes might be the timing or sequencing of movements through motor commands sent down neural delay lines of different lengths (Kornhuber, 1975; Sternberg et al., 1978). In contrast, controlled processes precede ballistic processes and can occur without producing movement. Although these processes may be automatic

in other senses (cf. Jonides, 1981), they are under voluntary control with respect to whether they ultimately lead to overt movement (cf. Logan, 1981). An example of controlled processes might be the advanced planning of a movement by loading a motor program into a program buffer (e.g., Henry & Rogers, 1960; Klapp, Wyatt, & Lingo, 1974). Finally, the temporal boundary between controlled and ballistic processes can be conceived of as a "point of no return" (Bartlett, 1958) in response preparation. Once thought has passed this point, there is no turning back; action is inevitable.

In the present article we explore the possibility of separately measuring changes in the duration of mental processes before and after the point of no return. Such measurement would enable us to make inferences about the nature of the controlled and ballistic components of action, based on what kinds of experimental manipulations affect their respective durations (cf. Sanders, 1980; Sternberg, 1969). It would also be germane to a number of current issues in the study of motor control. For example, it might help us to discover what components of a movement can be prepared and stored prior to the movement's execution. The question of whether this is possible for motor programming is of particular relevance to the debate concerning whether motor programs should be studied using simple or choice reaction-time procedures (cf. Klapp, Abbot, Coffman, Greim, Snider, & Young, 1979; Sternberg et al., 1978).

It would also be especially interesting and useful to be able to study the ballistic processes in their own right. They are still little understood by cognitive psychologists, even though they contribute to reaction-time measurements in various areas of cognition, such as perception, memory, and reasoning. Several psychophysiological measures, such as reflex excitability (e.g., Bonnet, 1981) and electromyographic activity (e.g., Brunia & Vingerhoets, 1980), have previously been used to study ballistic processes. A reaction-time measure would serve as an additional converging operation and might even help to further bridge the gap between cognitive psychology and physiology (cf. Requin, 1980).

Unfortunately, classical reaction-time procedures cannot easily separate the effects of experimental factors on controlled

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and ballistic processes. Differences in total reaction time and in speed-accuracy trade-off curves might reflect changes in either or both sets of processes. Earlier methods used to partition reaction time into its components are also inadequate for solving this problem. The subtraction method (Donders 1868/1969) cannot be used to isolate either set of processes, because even the simplest reactions contain both stimulus detection and response execution. Nor can the additive factor method (Sanders, 1980; Sternberg, 1969) unequivocally reveal whether a particular stage of processing is affected by a given factor.

We attack this problem with a countermanding procedure and a race model. Both the procedure and model have been widely employed for other purposes. Our particular approach stems most directly from a line of research that began with Lappin and Eriksen (1966), who applied the countermanding procedure to study simple reaction time. Their work led Ollman (1973) to explicitly apply a race model to the results of the countermanding procedure. Logan and his colleagues (e.g., Logan, 1981; Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984) have subsequently extended the model and procedure to choice reaction time, applied them to a wide variety of problems in cognition, and done considerable work in their general theoretical development. The present research extends previous work by using the countermanding procedure and race model to precisely measure the selective effects of experimental factors on processes before and after the point of no return.

In this article, we describe our approach and demonstrate its feasibility and fruitfulness. First, we outline the procedure and model. Next, we show how they can be used together to measure separately the effects of experimental factors on controlled and ballistic processes. We then generate predictions from some of the basic assumptions underlying our approach, and test these predictions in a series of three experiments. In addition to providing support for our assumptions, the experiments confirm certain theories about the human information-processing system in general and provide some interesting insights into the nature of the ballistic processes that immediately precede overt movement.

The Countermanding Procedure

Countermanding procedures have previously been applied to a number of domains, including step tracking (cf. Welford, 1952), typing (Logan, 1982), and speech production (e.g., Ladefoged, Silverstein, & Papcun, 1973). The term *countermanding* comes from the fact that subjects are sometimes required to cancel a response to a previously issued command. The version of the procedure used as part of our work involves randomly interleaving two types of trials: go trials and stop trials. Go trials are like those used in standard simple and choice reaction-time experiments. Here, subjects must make a designated response to a go signal as quickly as possible while maintaining a high degree of accuracy. Stop trials are similar to go trials, except that they also include a stop signal following the go signal. Subjects must respond quickly to the go signal, but should attempt to avoid responding if they subsequently detect the stop signal. Of course, the subject never knows whether the current trial is a go or a stop trial until the occurrence of a stop signal.

We are concerned especially with three dependent variables

associated with the countermanding procedure: the reaction time on go trials, the reaction time on stop trials where subjects fail to withhold their responses, and *response functions*. Response functions, denoted $P_R(\text{SOA})$, give the probability of responding on stop trials as a function of the interval (SOA) between the go and stop signals. We call this interval the *signal lag*. As the signal lag increases, the probability of response inhibition $[1 - P_R(\text{SOA})]$ decreases.

The Race Model

Figure 1 shows the race model, on which we rely to interpret the results of the countermanding procedure (Osman, Kornblum, & Meyer, 1984, 1985). Similar models have previously been used to study other phenomena, including the processing of redundant signals (e.g., Meijers & Eijkman, 1977), Stroop phenomena (e.g., Morton & Chambers, 1973), subjective deadlines in simple reaction time (e.g., Kornblum, 1973b; Ollman & Billington, 1972), and speed-accuracy trade-offs (e.g., Meyer & Irwin, 1981; Meyer, Irwin, Osman, & Kounios, 1986). The particular model proposed here was suggested to us by Ollman's (1973) model, the model implicit in Logan (1981), and similar models that we have used to study simple reaction time (Kornblum, 1973b) and speed-accuracy trade-offs (Meyer & Irwin, 1981; Meyer, Irwin, Osman & Kounios, 1986). It is similar to Ollman's model (1973) and formally equivalent to Logan and Cowan's (1984).

The race model involves three independent sets of processes and a point of no return.¹ The three sets include the controlled and ballistic processes introduced previously, plus an inhibition process. They are represented by the boxes at the top of Figure 1. The point of no return is represented by the middle circle. Stop trials involve a race between controlled and inhibition processes. The race ends at the point of no return, with the winner determining whether an overt response will be executed or inhibited. If the controlled processes win, then there is a response despite the stop signal. If the inhibition process wins, then no response occurs.

Based on the race model, we may define three random variables, T_C , T_I , and T_B . These correspond to the completion times of the controlled, inhibition, and ballistic processes, respectively. The probability of responding on a stop trial (i.e., value of the response function) may thus be expressed as

$$P_R(\text{SOA}) = P[(T_C - T_I) < \text{SOA}]. \quad (1)$$

The right side of Equation 1 represents the probability that the signal lag plus the time taken by the inhibition process is greater than the time at which the controlled processes reach the point of no return. Plotting this probability as a function of signal lag

¹ It is necessary to assume independence only in the sense that the durations of each of these processes can be selectively influenced by our experimental manipulations without affecting the durations of the other two. It does not matter for our current purposes whether the random variation of their respective durations within an experimental condition is correlated over trials. Such dependencies might be caused, for example, by fluctuations in arousal or by subjects' speeding ballistic processes to compensate for slow controlled processes (cf. Danev, de Winter, & Wartna, 1971).

THE RACE MODEL

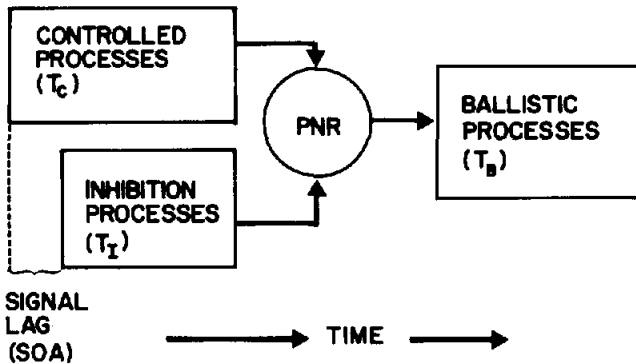


Figure 1. The race model for the countermanding procedure. (PNR = point of no return.)

(SOA, stimulus onset asynchrony) gives us the entire response function. As noted by Ollman (1973) and by Logan and Cowan (1984), the response function may be viewed as a type of psychometric and/or cumulative distribution function with its own mean (central tendency) and variance (spread) over the temporal domain. Moreover, reaction time on stop trials may be expressed as

$$RT_{\text{Stop}} \sim [T_C | (T_C < (T_I + \text{SOA}))] + T_B. \quad (2)$$

This relation represents the time taken by the controlled processes to reach the point of no return, given that they beat the inhibition processes, plus the time to complete the ballistic processes. The reaction time on go trials is simply

$$RT_{\text{Go}} \sim T_C + T_B. \quad (3)$$

Here the set of controlled processes is the only runner in the race, so reaction time equals the sum of controlled-process and ballistic-process completion times not conditioned by the presence of the inhibition process.

Measuring Controlled and Ballistic Effects

Our aim is to use the countermanding procedure, along with the race model, to measure the separate effects of experimental factors on controlled and ballistic processes. Logan (1981) previously attempted to discover which set of processes is affected by such factors. Our approach extends that of Logan (1981) by precisely specifying the magnitude of these effects and by allowing for the possibility that a factor might affect both sets of processes.

We proceed by using the race model to analyze the effects of factors on go-trial reaction time and the response function. According to the race model, any change in mean go-trial reaction time is the sum of the change in mean controlled-process time and the change in mean ballistic-process time, as shown in Equation 4

$$\Delta M_{\text{Go}} = \Delta M_C + \Delta M_B, \quad (4)$$

where ΔM_{Go} , ΔM_C , and ΔM_B denote the changes in the means

of go-trial reaction time, controlled-process completion time, and ballistic-process completion time. Similarly, any change in the mean of the response function (ΔM_R) equals the change in the mean controlled-process time (ΔM_C) minus the change in the mean inhibition-process time (ΔM_I), as shown in Equation 5

$$\Delta M_R = \Delta M_C - \Delta M_I. \quad (5)$$

If we further assume that the inhibition process remains unchanged by the factor manipulation under study, Equation 5 reduces to Equation 6

$$\Delta M_R = \Delta M_C, \quad (6)$$

where the change in the response function simply equals the change in the controlled processes alone. The change in the mean ballistic-process time would then equal the change in the mean go-trial reaction time minus the change in the mean of the response function, as shown in Equation 7

$$\Delta M_B = \Delta M_{\text{Go}} - \Delta M_R. \quad (7)$$

Thus, by observing changes in the response function and by comparing these changes with those in go-trial reaction time, we can study the separate effects of factors on controlled and on ballistic processes. Note, however, the importance of the assumption that the inhibition processes remain unchanged by these factors. The validity of this assumption will be considered later on in the article.

Empirical Predictions

Two sets of predictions follow directly from the preceding development (cf. Lappin & Eriksen, 1966; Logan, 1981; Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984). The first set is implied by the fact that changes in the response function reflect changes in controlled processes only, whereas changes in go-trial reaction time reflect changes in both controlled and ballistic processes. These predictions are presented in Table 1. The three leftmost columns of the table present a taxonomy for classifying the effects of experimental factors: (a) Only controlled processes might be affected, as shown in the first row; (b) only ballistic processes might be affected, as shown in the second row; (c) both types of processes might be affected in the same direction, as shown in the third row; or (d) both types of processes might be affected in opposite directions, as shown in the bottom row. The rightmost column of the table shows the different pattern of effects on mean go-trial reaction time and the mean of the response function implied by each of the four classes of factors on the left. If a factor affects only controlled processes, then it ought to produce equal changes in both measures. Such a pattern of effects has been reported for manipulations of stimulus discriminability and stimulus-response compatibility (Logan, 1981), and for a comparison of simple versus choice reaction time (Logan, Cowan, & Davis, 1984). If a factor affects only the ballistic processes, then, as noted by Logan (1981), it ought to change go-trial reaction time without affecting the response function. If it affects both types of processes in the same direction, then it ought to affect both measures, but reaction time to a greater degree. If it affects both types of processes in opposite directions, then it ought to produce a larger change in the response function mean than in the mean reaction

Table 1
Taxonomy of Factor Effects in the Countermanding Procedure

Factor type	Process type		Expected results Go-trial RT vs. response functions
	Controlled	Ballistic	
1	↑	—	$\Delta M_{Go} = \Delta M_R$
2	—	↑	$\Delta M_{Go} > 0, \Delta M_R = 0$
3	↑	↑	$\Delta M_{Go} > \Delta M_R$
4	↑	↓	$\Delta M_{Go} < \Delta M_R$

Note. Arrow up, arrow down, and dash indicate which sets of processes are affected and whether they are affected in the same direction; RT = reaction time; ΔM_{Go} = change in mean go-trial reaction time; ΔM_R = change in the mean of the response function.

time. Mean reaction time might even remain unchanged if effects on the controlled and the ballistic processes balanced each other perfectly.

A second set of predictions stems from the assumption that stop trials involve a race between controlled and inhibition processes. This assumption implies that the cumulative distribution functions of reaction times on stop trials and go trials should form a "fan" pattern as illustrated in Figure 2. This figure shows cumulative distribution functions for go trials and for stop trials with early, middle, and late stop signals. All four cumulative distribution functions fan out from a common minimum, and diverge increasingly at longer reaction-time values. Each stop-trial cumulative distribution function dominates those from trials with later stop signals, and all stop-trial cumulative distribution functions dominate the go-trial cumulative distribution function. Lappin and Eriksen (1966) have reported reaction-time distributions for stop and go trials that conform closely to such a pattern. Moreover, although the fastest reaction times are the same for all cumulative distribution functions, the mean reaction time on stop trials increases with signal lag and is faster than mean reaction time on go trials. Logan and his colleagues (e.g., Logan, 1981; Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984) have previously made and confirmed such a prediction regarding the ordering of mean reaction times.

One way to better understand this last set of predictions is to consider the inhibition processes induced by stop signals as a filter for go-signal control processes. On stop trials, only the controlled processes that beat the inhibition process to the point of no return activate ballistic processes, and thus contribute to reaction time. When the stop signal occurs early, only the fastest controlled processes escape being inhibited. When the stop signal occurs later, slower controlled processes also contribute to stop-trial reaction times. On go trials, all controlled processes, even the slowest, contribute to reaction time. Consequently, each cumulative distribution function in Figure 2 is "nested" within those to its right (cf. Meyer, Yantis, Osman, & Smith, 1985; Sternberg, 1973). Because the fastest controlled processes contribute to reaction time on all trial types, the minima for all of the cumulative distribution functions (assuming equal sample size) should be the same. The divergence of, and dominance order between, the cumulative distribution func-

tions results from the differential contribution of long reaction times to each trial type.

Overview of Current Experiments

Our ultimate goal is to infer the properties of controlled and ballistic processes from the properties of the factors that affect them. However, before making such inferences, it is necessary to further test the assumptions underlying our approach. In this article, we are concerned with testing three of the predictions from among those just described.

Prediction 1: If a factor affects controlled processes only, then it ought to produce equal changes in mean go-trial reaction time and in the mean of the response function (cf. Table 1).

Prediction 2: If a factor affects both controlled and ballistic processes in the same direction, then it should produce a larger change in mean go-trial reaction time than in the mean of the response function (cf. Table 1).

Prediction 3: (a) Mean reaction time on stop trials ought to increase with signal lag and ought to be faster than on go trials. (b) The cumulative distribution functions of reaction times for stop and go trials should form a fan pattern (cf. Figure 2).

Here we report three experiments in which we manipulated factors whose effects on controlled and ballistic processes could be anticipated on a priori grounds. The first experiment tests Prediction 1. It involves a factor that ought to affect only controlled processes. The second experiment tests Prediction 2. It involves a factor that ought to affect both controlled and ballistic processes in the same direction. The third experiment tests Predictions 1 and 2 simultaneously. It involves two factors: one that ought to affect only controlled processes and one that ought to affect both sets of processes in the same direction. All three experiments test Prediction 3 by comparing mean reaction

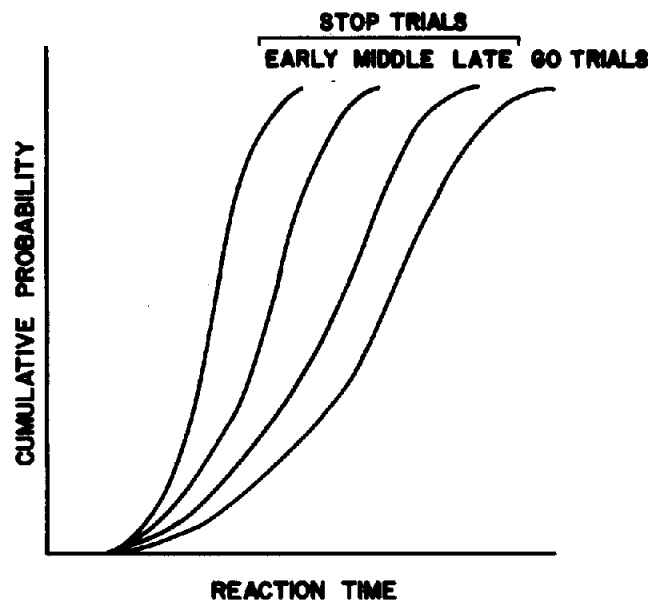


Figure 2. Idealized cumulative distribution functions for reaction times on early stop-signal trials, middle stop-signal trials, late stop-signal trials, and go trials.

times on stop and go trials and by plotting their respective cumulative distribution functions. If these experiments support the predictions, then there will be more reason for confidence in using the approach to study other factor effects whose loci are still unknown. Failure to support the above pattern of predictions would cast doubt on our efforts to use the countermanding procedure and race model to measure factor effects before and after the point of no return.

Experiment 1

To demonstrate an effect entirely before the point of no return, we chose a factor that potentially influences just perceptual or memorial processes, not motor processes. The factor was "lexical status," which distinguishes words from nonwords. Previous research has shown that lexical status influences the mean reaction time of lexical (word-nonword) decisions (Meyer & Schvaneveldt, 1971). When subjects have to decide whether or not a letter string is an English word, they are faster to respond *yes* to words than *no* to nonwords. This difference is presumably due to an effect of familiarity on stimulus encoding and/or memory retrieval, both relatively early mental processes (Sternberg, 1969). Thus, lexical status seemed like a good candidate for a factor whose effects might be revealed as occurring entirely before the point of no return, if the assumptions underlying our approach are valid.

Method

Subjects. Six undergraduate students at the University Michigan served as paid subjects. Each subject was tested individually in a single 1-hr session.

Apparatus. Subjects sat in a moderately illuminated, sound-attenuating booth throughout the session. Visual and auditory stimuli were presented with a video terminal (Hewlett Packard 2126/A) viewed at a distance of 35 cm. Manual responses with the right middle and right index fingers were made by depressing one of two keys mounted on a horizontal panel. The panel was located in front of the terminal and placed so that subjects' arms rested comfortably on the table. The keys required approximately 3.5 ounces of pressure and 1 mm of travel to register a response. Stimulus presentation and response collection were controlled by a digital computer (DEC-PDP 11/34).

Stimuli. The stimuli included a warning signal, four-letter strings for go signals, and a stop signal. The warning signal was a dashed line that appeared on the terminal immediately below the location of the go signal. The go signals each contained four capital letters, which together subtended about 1.4° of visual angle in width and about 0.5° in height at the viewing distance of 35 cm.

There were 120 different English words (e.g., *food*) and 120 different nonwords (e.g., *mafe*) used as go signals. The words consisted of familiar nouns and verbs, and their frequency of occurrence in ordinary text equaled or exceeded 32 per million (Kučera & Francis, 1967). The nonwords were pronounceable strings obtained from other English words by altering individual vowels or consonants without violating the orthographic rules of English (Venezky, 1970).

The stop signal was a 500-Hz, 10-ms tone presented at a comfortable listening level. Stop signals occurred at one of six lags (SOA), including three for words, and three for nonwords. Signal lags were adjusted individually for each subject by a staircase tracking algorithm (Levitt, 1971), yielding "early," "middle," and "late" signals, which respectively induced either 71%, 50%, or 29% of the responses to words and to non-

words to be inhibited. By adaptively adjusting the lag of each stop signal, we achieved the desired levels of response probability on stop trials.²

Design. Go signals consisted equally often of words and nonwords. Stop signals were presented on one fourth of all trials, occurring equally often after short (early stop), medium (middle stop), and long (late stop) lags for both words and nonwords.

Procedure. Subjects were tested with the countermanding procedure described earlier. Their fingers rested on the response keys at all times. Trials were presented discretely. Go trials involved normal lexical (word-nonword) decisions. At the start of each go trial, a warning signal appeared in the center of the terminal screen and was replaced 500 ms later by a go signal (four-letter word or nonword). The subject had to press the middle or index finger key, depending on whether the go signal was a word or nonword. The keypress caused the go signal to vanish from the screen. Stop trials were like go trials except that the stop signal followed the go signal, which disappeared immediately if it was still on the screen. The warning signals on successive trials were separated by an interval of 2.25 s plus feedback presentation time.

Feedback was presented on all stop trials, after errors on go trials, and after each trial block. The word ERROR appeared on go trials if the subject pressed the wrong key or did not respond within 1.5 s after the go signal. On stop trials, GOOD was presented if the subject inhibited his or her response successfully, and OOPS was presented if she or he failed to withhold the response. The trial feedback remained visible for 250 ms immediately below where the warning signals were located. After each block, the subject's mean reaction-time on go trials, number of errors on go trials, and number of inhibited responses on stop trials for that block were displayed.

The instructions to subjects stressed speed on go trials over successful inhibition on stop trials. Subjects were told to respond as quickly as possible without making more than 5% errors on go trials. They were also told that they should make a concerted effort to stop themselves from responding if they detected the stop signal but that this would not always be possible.

Subjects were tested in a single session with 18 blocks of 72 trials each. The first two trial blocks included only go trials, which familiarized subjects with the lexical decision task. The next four blocks were used to familiarize subjects with the countermanding procedure (stop plus go trials) and to let the tracking algorithm locate the right vicinity for the stop-signal lags. Only the last 12 blocks were included in the data analysis.

Results

Reaction times and error rates. Figure 3 shows mean reaction times for words and nonwords on go trials, and on stop trials with early, middle, and late signal lags for which responses were not successfully inhibited. As anticipated, go-trial responses to

² The staircase tracking algorithm was similar to those used in psychophysical experiments. It adjusted the signal lag according to a rule based on whether a subject had responded or inhibited on previous stop trials. We used three rules: (a) On early-signal trials, the signal lag was decreased each time a subject responded, and was increased when a subject inhibited the response twice consecutively; (b) on middle-signal trials, the signal lag was decreased each time a subject responded, and was increased each time a subject inhibited the response; (c) on late-signal trials, the signal lag was decreased when a subject responded twice consecutively, and was increased each time a subject inhibited the response. The three rules yield 29%, 50%, and 71% response probability, respectively. The signal lag was always adjusted by an increment of 50 ms, and early, middle, and late-signal stop trials were randomly interleaved with go trials.

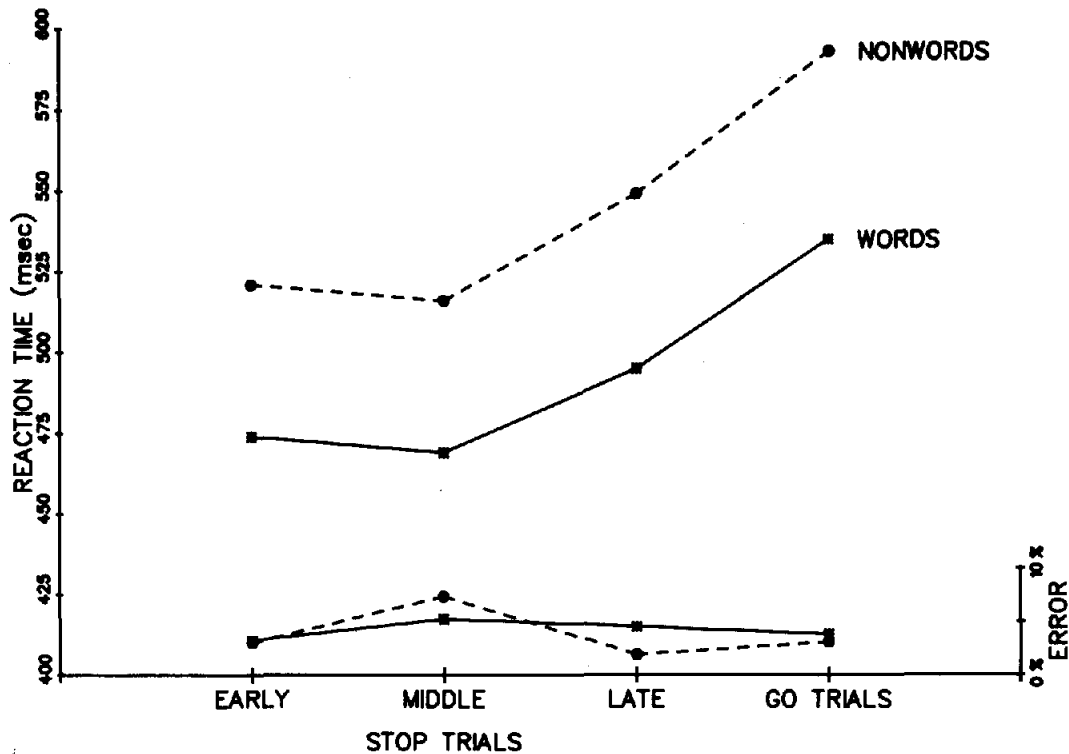


Figure 3. Mean reaction time and percent error as a function of lexical status and trial type.

words were faster than those to nonwords: mean time difference = 58 ms; $t(5) = 4.1$, $p(\text{one-tailed}) < .01$. There was also a word-nonword effect on stop trials: mean time difference = 49 ms; $t(5) = 5.18$, $p(\text{one-tailed}) < .01$. Overall, an ordering of the mean reaction times for different trial types supported the hypothesis of a race between controlled and inhibition processes on stop trials (cf. Prediction 3). That is, responses on go trials were slower than on late stop-signal trials: mean time difference = 42 ms; $t(5) = 4.22$, $p(\text{one-tailed}) < .01$, which, in turn, were slower than those on middle stop-signal trials: mean time difference = 29 ms; $t(5) = 4.30$, $p(\text{one-tailed}) < .01$. Response times did not differ significantly between middle and early stop-signal trials: $t(5) = -0.45$, $p(\text{one-tailed}) > .30$. However, it should be noted that early stop-signal trials contained very few reaction times, because most responses were inhibited on those trials.

Error rates appear at the bottom of Figure 3. They were generally low and gave no indication that the effects of lexical status or trial type on reaction time were due to a speed-accuracy trade-off. The error rates for words and nonwords were not significantly different on go trials, $t(5) = 1.87$, $p > .10$; nor on stop trials, $t(5) = 0.83$, $p > .40$. Error rates did not differ significantly between either go trials and late stop trials, $t(5) = 0.10$, $p > .90$; or between late stop trials and middle stop trials, $t(5) = 1.15$, $p > .30$.

Further support for the race model is shown in Figure 4. Group cumulative distribution functions of reaction times for each trial type are shown for words in the top panel and for nonwords in the bottom panel. These group functions were obtained by averaging (Vincentizing) the quantiles of the func-

tions from individual subjects (Thomas & Ross, 1980).³ Each panel shows the fan pattern implied by the race model. The minima on early, middle, and late stop-signal trials are virtually identical within the word and the nonword conditions. Note that the minima for go trials are smaller than for stop trials. This is understandable, because the expected value of a sample minimum decreases as sample size increases, and there were nine times as many go trials as each type of stop trial. With some minor exceptions, the four cumulative distribution functions in each panel diverge and are arranged in the predicted dominance order: early stop < middle stop < late stop < go trials. Overall, these results provide good support for using the race model to interpret other facets of the data.⁴

³ This procedure preserves the general shape of the individual functions while providing an unbiased estimate of the average of any parameter that can be expressed as a linear combination of quantiles. Consequently, Vincentizing is appropriate for the current discussion in which we are considering the average differences between quantiles. We also present Vincentized response functions to show the average time difference between experimental conditions at each quantile. Note, however, that our analyses of the means of these functions are based upon the individual functions from each subject in each condition.

⁴ Our interpretation of the results for the reaction-time minima in Experiments 1 and 2 is somewhat weakened by the fact that subjects sometimes responded before the occurrence of the stop signal. Some of the minima are associated with responses of this type and would thus not be expected to be affected by the processing of the stop signal. However, note that a different proportion of the subjects had minimum reaction times on responses that occurred before the stop signal on early, middle, and late stop-signal trials. Despite this difference, the group

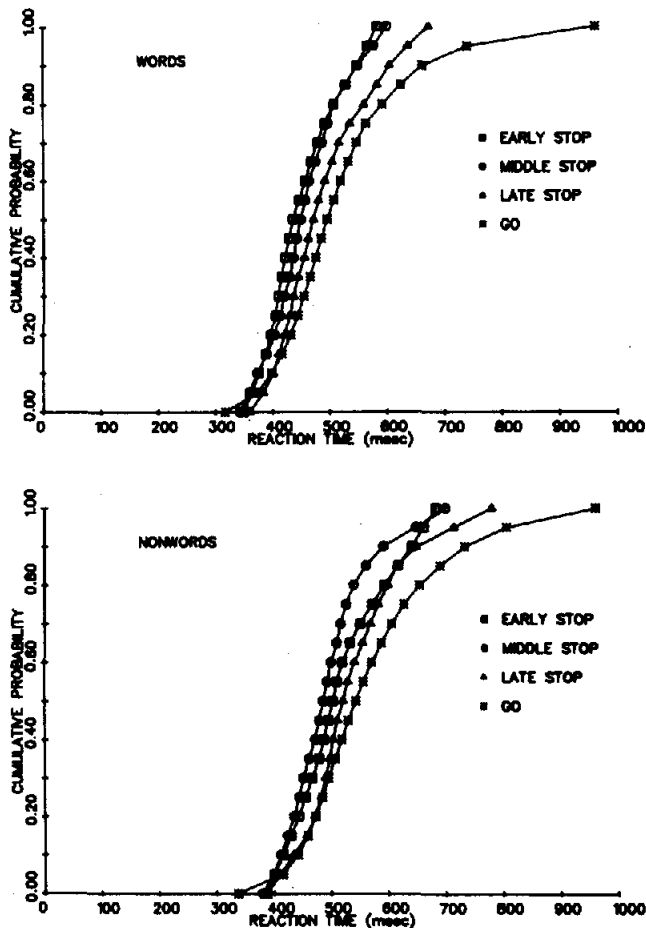


Figure 4. Group cumulative distribution functions of reaction times on each trial type for words (top panel) and nonwords (bottom panel).

Response probability and stop-signal lags. The mean signal lag and response probability on early, middle, and late stop-signal trials for words and for nonwords is shown in Table 2. We see here that the staircase tracking algorithm used to adjust the signal lags worked quite well in yielding approximately 29%, 50%, and 71% response probabilities for the respective signal-trial types. For both words and nonwords, late signals had greater lags than middle signals, which in turn had greater lags than early signals, just as we intended. Also, the average signal lags needed to produce equal levels of inhibition were less for words than for nonwords.

Group response functions appear in Figure 5. These functions were obtained by plotting response probability against signal lag, on a subject-by-subject basis, disregarding the type of stop signal (i.e., early, middle, or late) associated with each observation. The functions from individual subjects were monotized (Barlow, Bartholomew, Bremner, & Brunk, 1972) and combined into group functions by averaging their quantiles.⁵

minima, which are the average of the minima from individual subjects, are very close for all stop trial types.

Table 2
Mean Response Probabilities and Signal Lags for Experiment 1

Stimulus type	Response probability (%)			Signal lag (ms)		
	ES	MS	LS	ES	MS	LS
Words	30	50	71	276	326	413
Nonwords	31	50	70	336	389	489

Note. ES = early signal; MS = middle signal; LS = late signal.

The shapes of the group functions well represent those of their constituents, and the pattern of effects shown here was found for each subject individually. Response probability increased with signal lag, and the lag necessary to produce a given response probability was always less for words than for nonwords. The separation between these two functions reflects the effect of lexical status before the point of no return (cf. Equation 6). The separation of the functions at all percentiles shows that the effect of lexical status was distributed across controlled processes of all speeds.

Comparison of go-trial reaction times and response functions. Separately assessing the effects of factors before and after the point of no return involves comparing their effects on response functions, $P_R(\text{SOA})$, versus their effects on go-trial reaction times (cf. Equations 6–7). To make the necessary comparison, we obtained response function means for each subject by applying a discrete approximation of Equation 8 to their response functions:⁶

$$M_R = \int_{-\infty}^{\infty} t[dP_R(t)]/dt, \quad (8)$$

where M_R is the response function mean, and $dP_R(t)$ is the differential of $P_R(\text{SOA})$ evaluated at time t . Lexical status produced a 65-ms effect on the means of the response function averaged across subjects (373 ms for words and 438 ms for nonwords) and a 58-ms effect on mean go-trial reaction time (538 ms for words and 593 ms for nonwords). The response-function effect and the reaction-time effect were not significantly different from each other, $t(5) = 1.18$, $p > .25$. This is what the race model would predict if lexical status indeed has its entire effect before the point of no return (cf. Prediction 1). Because we already have some reason to believe that lexical status might influence only relatively early controlled processes, the results support both our expectations and the validity of the model.

⁵ This involved finding the monotonic function with the best least-square fit to the data. Note that the mean of the monotized function was always the same as that of the original response function.

⁶ The means of our sample response functions may be biased estimators of the population means. However, the problem of bias is reduced to some extent because we are concerned with estimating the difference between two parameters by using the difference between two estimators. If the estimators have equal biases, then the biases would be cancelled by the subtraction. Thus, our estimate of the change in the mean of the response function should be biased only to the degree that the individual estimators of the inhibition means differ in their respective bias.

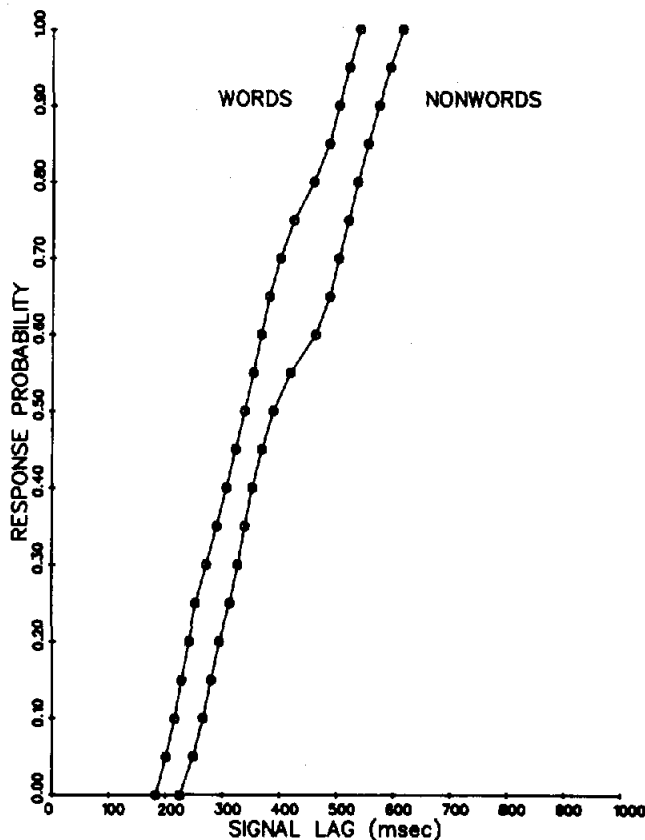


Figure 5. Group response functions for words and nonwords.

Discussion

When a factor produces equal changes in mean go-trial reaction time and the response-function means, it is evidence for the factor's effect being entirely before the point of no return. Experiment 1 displays this pattern of effects for lexical status. As indicated earlier, a similar pattern has been found by Logan (1981) for a type of stimulus discriminability and a type of stimulus-response compatibility, and by Logan, Cowan, and Davis (1984) for a comparison of simple versus choice reaction time.

Such a pattern of effects bears on our assumption that the time-course of the inhibition processes remains unaffected by our factor manipulation in this study. If the processing time for the stop signal had been prolonged in conditions with longer go-trial reaction time, we would expect a smaller effect on the response-function mean than on mean go-trial reaction time (cf. Equations 4-5). Alternately, if the processing time for the stop signal had been prolonged in conditions with shorter go-trial reaction time, we would expect a larger effect on the mean of the response function than on mean go-trial reaction time. It is possible, of course, that the processing time for the stop signal in Experiment 1 was affected by lexical status, but was compensated by a further violation of the race model that produced an equal effect in the opposite direction. However, this interpretation seems relatively implausible and unparsimonious.

An effect entirely before the point of no return, as we observed here, also supports the selective-influence assumption

upon which our approach is based. We assume that it is possible to selectively influence either the controlled or the ballistic stage of processing without necessarily affecting the other stage (cf. Sanders, 1980; Sternberg, 1969). This would not be possible if subjects compensated for effects on controlled processes by slowing or speeding ballistic processes. Likewise, if the ballistic processes were primed by partial output from the controlled processes before the latter reached the point of no return, it would not be possible to affect the controlled processes alone (cf. Miller, 1983). Fortunately, the pattern of effects for lexical status casts doubt upon these negative possibilities.

Finally, the results of Experiment 1 support the assumption that stop trials involve a race between controlled and inhibition processes (cf. Prediction 3). This assumption is essential to our approach because it enables us to treat the response function as the difference between two random variables (cf. Equation 1). As predicted by the model, the cumulative distribution functions of reaction times formed a fan pattern, and mean reaction time on stop trials increased with signal lag and was faster than mean reaction time on go trials. The time course of the ballistic processes was apparently unaffected by failed attempts at inhibition. If this were not the case, at least some of the responses on stop trials would have been prolonged. We would then expect that either (a) some of the times on late stop-signal trials might be slower than the slowest go-trial responses; (b) the stop trials with different signal lags might have different minima; or (c) the cumulative distribution functions might cross.⁷ The assumption that ballistic processes are unaffected by failed attempts at inhibition is not necessary for our approach, but confirmation of it is interesting because this suggests that the point of no return is really a point. Responses in our procedure appear to be either inhibited completely or not at all.

Now, it could be argued that lexical status does not affect processes after the point of no return because no such processes exist. Perhaps subjects can inhibit their responses up to the very moment when physical movement begins. If this were the case, then all factors would have their entire effect on reaction time before the point of no return. Thus, in order to interpret the pattern of effects for lexical status, we need to know whether ballistic processes indeed exist. Experiment 2 is an attempt to answer this question. By showing a factor effect after the point of no return, we hope to demonstrate the existence of processes intervening between the point of no return and physical movement.

Experiment 2

It is important to demonstrate an effect after the point of no return before proceeding further and applying the countermanding procedure and race model to other problems. If no factor were ever found that produced such an effect, we might take this at face value and conclude that late ballistic processes

⁷ If the processing of the stop signal affected the duration of the controlled or ballistic processes, we would expect perturbations in the cumulative distribution functions for stop trials. These perturbations would occur at different places on the functions for early, middle, and late stop-signal trials. Consequently, we might then expect these functions to cross each other, as well as crossing the function for the go trials.

are immune to the influence of any factor. Alternately, we might doubt the existence of these late ballistic processes or the ability of the paradigm and model to reveal changes in their duration. If any of these possibilities were true, the paradigm and model would be useless for partitioning reaction-time effects.

To demonstrate an effect after the point of no return we chose a factor that may conceivably have effects throughout the information processing system, including ones on motor processes: the repetition or nonrepetition of stimulus-response pairs. Subjects respond faster to a stimulus if both the stimulus and response are the same as on the previous trial. Two lines of evidence suggest that repetition affects late motoric processes. The first is that it interacts with other factors (e.g., R-R compatibility, S-R compatibility, number of alternatives, and priming) believed to affect motor processes (see Kornblum, 1973a, for a review). Secondly, an effect of response repetition, over and above that of stimulus repetition, has been found in studies where several stimuli are paired with the same response (e.g., Bertelson, 1965).

Method

Subjects. Six undergraduate students at the University of Michigan served as paid subjects. None had been in the previous experiment. Each subject was tested individually in a single 1-hr session.

Apparatus and stimuli. The apparatus and stop signals were the same as in Experiment 1. Go signals consisted of the letters *G* and *X* presented on the terminal screen. Each letter subtended about .35° of visual angle in width and 0.5° in height at a viewing distance of 35 cm. Separate stop signals were used to track 29%, 50%, and 71% inhibition probabilities for repetitions and for nonrepetitions.

Design. Half of the trials in each block involved repetitions of the go signal from the immediately previous trial (e.g., *X* after *X*), and half involved nonrepetitions (e.g., *X* after *G*). Stop signals occurred on one fifth of the trials in each of these two conditions, with equal frequencies of short, middle, and long lags.

Procedure. We again tested each subject with the countermanding procedure. On go trials, subjects pressed the right or left index-finger key, depending on which of the two go signals (letters) appeared in the center of the terminal screen. Their fingers rested on the keys at all times. After a keypress, the letter disappeared and was replaced 35 ms later by the next letter. Stop trials were like go trials except that the go signal was followed by a stop signal, and the letter disappeared if it was still on the screen. After the stop signal, there was a time-out period of 500 ms before the next letter appeared. The same feedback and instructions were used as in Experiment 1.

Subjects were tested in a single session with 15 blocks of 120 trials each. The first two blocks of trials included only go trials in order to familiarize subjects with the letter-discrimination task. The next three blocks had mixed go and stop trials to familiarize subjects with the countermanding task and to enable the tracking algorithm to find the right vicinity for the stop-signal lags. Only the last 10 blocks were included in the data analysis.

Results

Reaction times and error rates. The pattern of effects for latency and accuracy on stop trials and on go trials was very similar to the one found in Experiment 1. Figure 6 shows the effects of trial type and go-signal repetition on reaction time. As expected, repetitions yielded faster responses than did nonrepetitions on go trials: mean time difference = 58 ms; $t(5) = 4.17$,

$p(\text{one-tailed}) < .01$. This also happened on stop trials: mean time difference = 72 ms; $t(5) = 5.17$, $p(\text{one-tailed}) < .01$. Responses on go trials were slower than those on late stop trials: mean time difference = 52 ms; $t(5) = 6.0$, $p(\text{one-tailed}) < .01$, which were slower than those on middle stop trials: mean time difference = 25 ms; $t(5) = 5.49$, $p(\text{one-tailed}) < .01$, which were slower than those on early stop trials: mean time difference = 13 ms; $t(5) = 2.17$, $p(\text{one-tailed}) < .05$.

Error rates appear at the bottom of Figure 6. They were generally low and gave no evidence that the effects of repetitions or trial type on reaction time were due to a speed-accuracy trade-off. Error rates for repetitions and nonrepetitions did not differ significantly on either go trials, $t(5) = 1.55$, $p > .15$; or stop trials, $t(5) = 1.3$, $p > .25$. Neither were there significant error-rate differences between go trials and late stop-signal trials, $t(5) = 1.23$, $p > .25$; between late stop-signal trials and middle stop-signal trials, $t = 0.9$, $p > .40$; or between middle stop-signal trials and early stop-signal trials, $t(5) = 0.77$, $p > .45$.

Group cumulative distribution functions of reaction times for each trial type appear separately for repetitions and nonrepetitions in the two panels of Figure 7. Each panel again displays the fan pattern implied by the nesting of each distribution within its neighbors to the right. The minima for each stop-trial type are virtually identical within repetitions and nonrepetitions, with the exception of late stop-trial nonrepetitions. The four cumulative distribution functions in each panel generally diverge, with trial types arranged in the predicted dominance order: early stop < middle stop < late stop < go trials.

Response probability and stop-signal lags. Average signal lag and response probability for each of the six stop-signals are shown in Table 3. The tracking algorithm again worked reasonably well, yielding close to 29%, 50%, and 71% response probabilities for each response-signal type. For both repetitions and nonrepetitions, late signals occurred at greater lags than did middle signals, which in turn occurred later than did early signals. Finally, the average signal lags necessary to produce equal levels of response inhibition were less for repetitions than for nonrepetitions.

Group response functions for repetitions and nonrepetitions are shown in Figure 8. The probability of responding on signal trials increased with signal lag, and the lag necessary to produce a given frequency of responding was less for repetitions than nonrepetitions up until about the 75th percentile. It is at this stage of the analysis that the results of repetition and lexical status diverge. Beyond the 75th percentile, response functions for repetitions and nonrepetitions were very close together. This pattern, in which the repetition function dominates the nonrepetition function at early percentiles and then converges with it at later percentiles, indicates that the slower controlled processes were unaffected by the repetition factor.

Comparison of go-trial reaction times and response functions. Again we see a somewhat different picture than occurred previously for the lexical-status factor. Repetition had a 58-ms effect on go-trial reaction time (429 ms for repetitions and 487 ms for nonrepetitions), but only a 34-ms effect on the response-function mean (323 ms for repetitions and 357 ms for nonrepetitions). The difference, 24 ms, represents the size of the repetition effect on ballistic processes after the point of no return. This interaction was significant, $t(5) = 2.48$, $p(\text{one-tailed}) <$

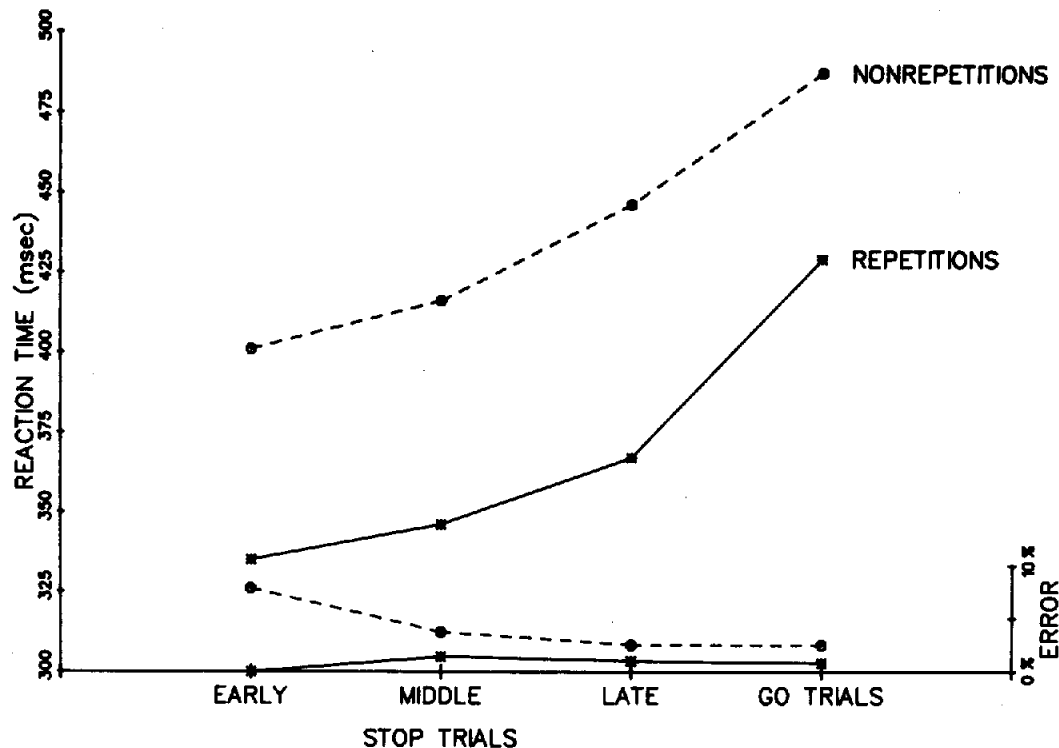


Figure 6. Mean reaction time and percent error as a function of repetitions versus nonrepetitions and trial type.

.028, and occurred in 5 of our 6 subjects. Such a result supports the hypothesis that repetition affects processes after the point of no return (cf. Prediction 2), and consequently that these processes do indeed exist.

Discussion

Experiment 2 revealed the pattern of effects that we had hoped to find for the repetition factor. A larger change occurred in the mean reaction time for go trials than in the mean for the response function. This is what would be predicted if a factor affected both the controlled and the ballistic processes in the same direction. Consequently, our results support the existence of ballistic processes and demonstrate our ability to detect changes in their duration. Furthermore, finding that the repetition factor, in particular, affects ballistic processes implies that these processes can be affected without being triggered. If this were not the case, subjects could not have prevented themselves from making more than one response to a stimulus. Presumably, making a response leaves a residual facilitation of the same response or inhibition of the alternative response. It would be interesting to investigate how the effect of the repetition factor on ballistic processes is modulated by the interval between successive responses and the number of alternative responses.

However, before definitely concluding that repetition affects ballistic processes after the point of no return, there is an alternative possibility that should be considered. The differential effects of repetition on mean go-trial reaction time and the mean of the response function found in Experiment 2 might

conceivably be an artifact of capacity sharing between the processing of the stop and the go signals (cf. Posner & Boies, 1971). As mentioned earlier, this pattern could result from the processing of the stop signal's being slowed in conditions with slower go-trial reaction time (cf. Equations 4-5). Such slowing might arise on stop trials if the mechanisms that process the two signals share a limited resource and if the go signal placed greater demand upon this resource at the expense of the stop signal in the more difficult conditions. Thus, the repetition factor might only appear to affect ballistic processes because of the violation of our assumption that the inhibition processes remain unchanged by our experimental manipulations.

Some evidence against this hypothesis comes from the results that provide support for the race model. Again we found the predicted fan pattern for the reaction-time cumulative distribution functions, and the mean reaction time on stop trials was less than on go trials and increased with signal lag. If the controlled and inhibition processes shared a limited resource, we might expect the time-course of the controlled processes to be affected by the occurrence of the stop signal and by the length of the signal lag. Signal lag could have an effect because it determines how long the controlled and inhibition processes are operating concurrently. As a result, we would expect violations of the race-model predictions similar to what would be produced if ballistic processes were prolonged by failed inhibition attempts. Some of the times on late stop-signal trials might be longer than the longest responses on go trials; stop trials with different signal lags might have different minima; or the cumulative distribution functions for different trial types might cross.

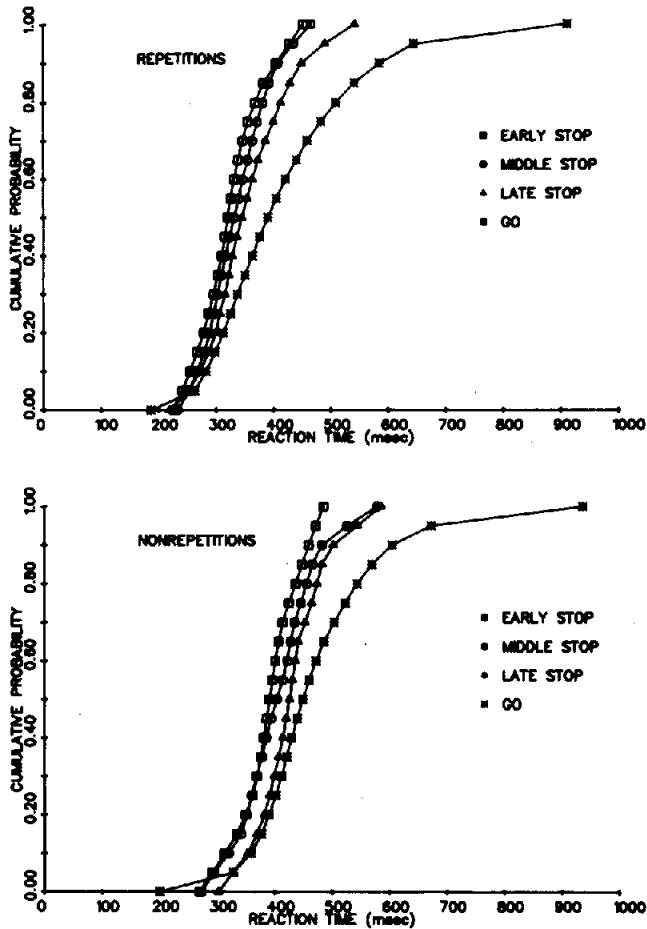


Figure 7. Group cumulative distribution functions of reaction times on each trial type for repetitions (top panel) and nonrepetitions (bottom panel).

Unfortunately, the preceding evidence against capacity sharing is somewhat indirect. It is not clear how sensitive these violations of the race model are to changes in the time course of the controlled processes. Nor is it certain that if the controlled processes prolonged the inhibition processes, such interference would be mutual. Experiment 3 was therefore designed to provide a stronger test of capacity sharing while replicating the effect of the repetition factor on ballistic processes.

Experiment 3

In Experiment 3, we attempted to replicate the effect of repetition after the point of no return, while simultaneously demonstrating the effect of a different factor entirely before the point of no return. The second factor chosen was the confusability between stimulus alternatives. Stimulus confusability seemed like a reasonable candidate for affecting only early perceptual processes and is similar to a factor that Logan (1981) found produced equal effects on reaction time and response functions.

Our rationale for including this second factor is based on the assumption that any capacity sharing between the processing of the stop and the go signals ought to be manifest in the pattern

Table 3
Mean Response Probabilities and Signal Lags for Experiment 2

Stimulus type	Response probability (%)			Signal lag (ms)		
	ES	MS	LS	ES	MS	LS
Repetitions	30	51	71	229	270	371
Nonrepetitions	30	51	73	275	323	395

Note. ES = early signal; MS = middle signal; LS = late signal.

of effects for both factors. If stimulus confusability produces equal effects on both the reaction time for go trials and the response function, the most plausible interpretation would be that its effect was on controlled processes only and that there was no capacity sharing between stop and go signal processing. This would enable us to eliminate capacity sharing as a possible explanation of a larger repetition effect on mean go-trial reaction time than on the mean of the response function.

Method

Subjects. Ten undergraduate students at the University of Michigan served as paid subjects. None had been in either Experiment 1 or 2. Each subject was tested individually in a single 1-hr session.

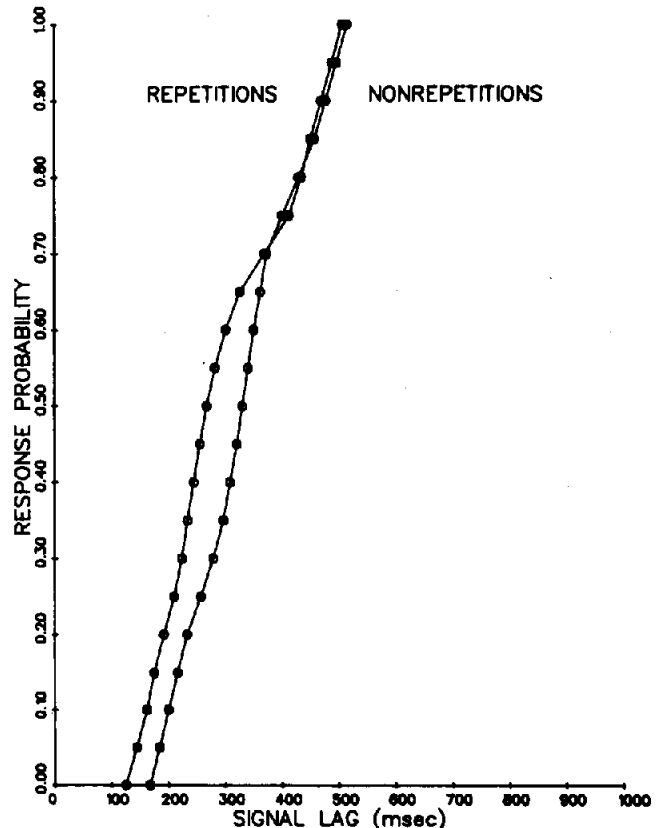


Figure 8. Group response functions for repetitions and nonrepetitions.

Apparatus and stimuli. The apparatus and stimuli were the same as in Experiment 2, with two changes. First, go signals consisted of one member from either a high- or low-confusability pair of letters. The low-confusability letter pair consisted of *G* and *X*, as in Experiment 2, and the high-confusability pair consisted of *I* and *l*. Secondly, separate stop signals tracked only a 50% response probability in each of four experimental conditions.

Design. Two independent variables, stimulus confusability and repetition, were manipulated orthogonally, yielding the four experimental conditions mentioned above. Stop signals occurred on one fifth of all trials in each of these conditions. High- and low-confusability letter pairs alternated between blocks of trials, with the order of presentation balanced across subjects.

Procedure. The trial-by-trial procedure, instructions, and feedback were the same as in Experiment 2. Subjects participated in a single session with 18 blocks of 120 trials. The first two blocks included only go trials, which familiarized subjects with the letter-discrimination task. The next four blocks included both go and stop trials, familiarizing subjects with the countermanding task and letting the tracking algorithm locate the right vicinity for the stop-signal lags. Only the last 12 blocks were included in the data analysis.

Results

Reaction times and error rates. Figure 9 shows mean reaction times and errors in each experimental condition for go trials in the left panel and stop trials in the right panel. Repetitions were again faster than nonrepetitions on both go trials, mean time difference = 51 ms; $t(9) = 7.42$, $p(\text{one-tailed}) < .0001$; and stop trials, mean time difference = 58 ms; $t(9) = 6.37$, $p(\text{one-tailed}) < .0001$. They also yielded a significantly smaller pro-

portion of errors on both go trials, mean accuracy difference = 2.3%; $t(9) = 3.02$, $p < .05$; and stop trials, mean accuracy difference = 5.5%; $t(9) = 4.03$, $p < .01$. The confusability factor also produced the desired effect. Responses to low confusable stimuli were faster than those to highly confusable stimuli on both go trials, mean time difference = 55 ms; $t(9) = 11.68$, $p(\text{one-tailed}) < .0001$; and stop trials, mean time difference = 41 ms; $t(9) = 7.44$, $p(\text{one-tailed}) < .0001$. They also contained a smaller proportion of errors on both go trials, mean accuracy difference = 1.5%; $t(9) = 4.03$, $p < .01$; and stop trials, mean accuracy difference = 3.9%; $t(9) = 3.35$, $p < .01$. Stop-trial responses were faster than go-trial responses, mean time difference = 68 ms; $t(9) = 11.81$, $p(\text{one-tailed}) < .0001$, consonant with the race model (Prediction 3). However, they also contained a larger proportion of errors, mean accuracy difference = 2.3%, $t(9) = 6.76$, $p < .0001$, indicating the contribution of a speed-accuracy trade-off to the reaction-time difference.

Figure 10 shows the group cumulative distribution functions of reaction times for go trials and stop trials in each of the four experimental conditions. The expected fan pattern was obtained in each condition. We cannot compare the minima for go and stop trials directly, because they contain unequal numbers of trials. However, after crossing at a low quantile, each pair of cumulative distribution functions diverged at longer reaction times, with stop trials to the left of go trials, as we would expect from previous considerations.

Response probability and stop-signal lags. The mean signal lag and response probability for each experimental condition are shown in Table 4. Response probabilities on stop trials were

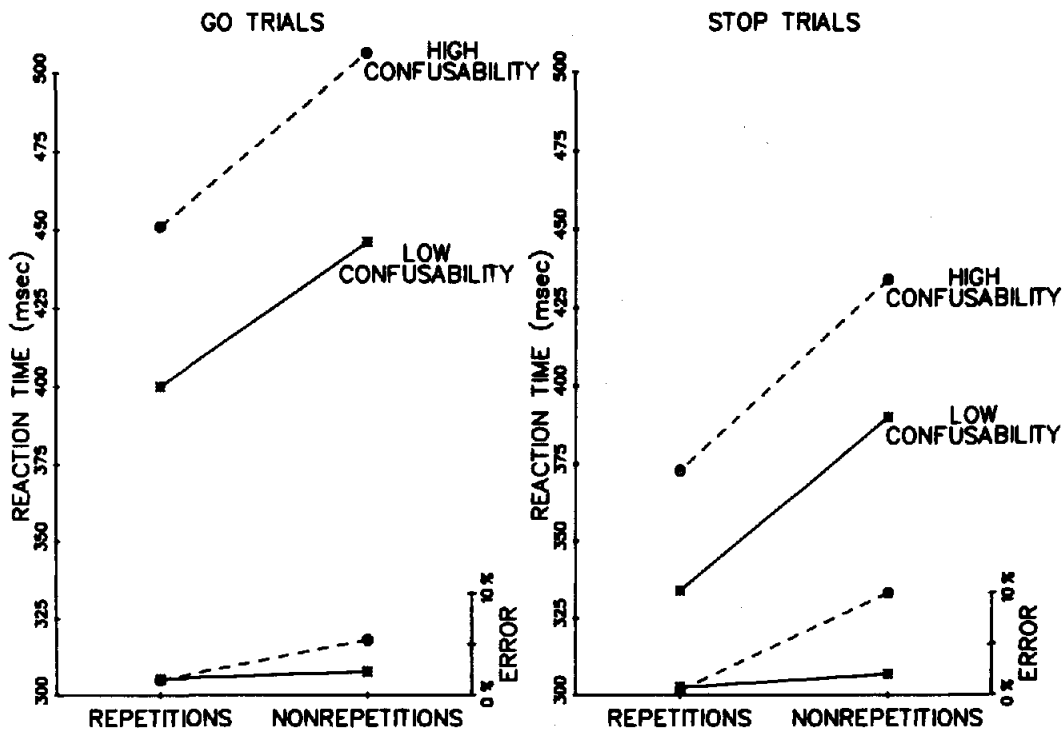


Figure 9. Mean reaction time and percent error as a function of stimulus confusability and repetition versus nonrepetition on go trials (left panel) and stop trials (right panel).

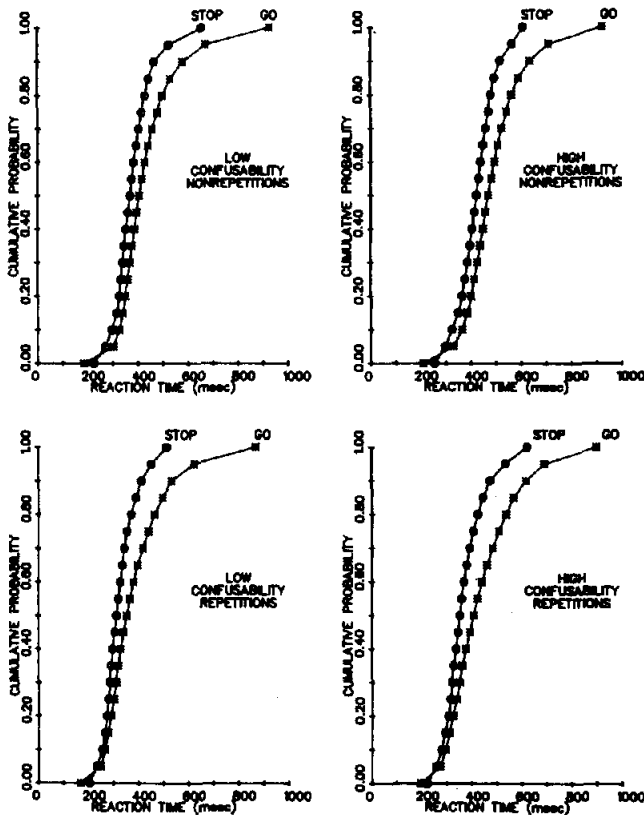


Figure 10. Group cumulative distribution functions of reaction times on stop and go trials for low-confusability nonrepetitions (top left), high-confusability nonrepetitions (top right), low-confusability repetitions (bottom left), and high-confusability repetitions (bottom right).

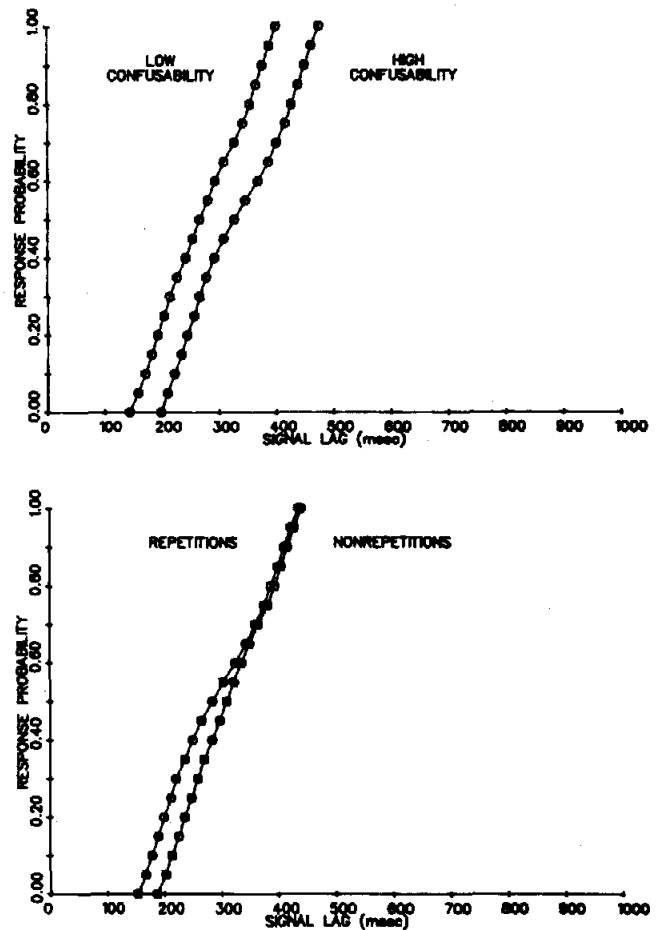


Figure 11. Group response functions for high- and low-confusability stimuli (top panel), and repetitions and nonrepetitions (bottom panel).

close to 50% in each of the four conditions. Signal lags were shorter for repetitions than for nonrepetitions and were shorter for low-confusability than for high-confusability stimuli.

Group response functions are shown in Figure 11. The top panel allows a comparison of high- versus low-confusability stimuli, and the bottom panel allows a comparison of repetitions versus nonrepetitions. The group response functions were very similar to those found in Experiments 1 and 2. The functions corresponding to the two levels of letter confusability were separated by about the same amount for all percentiles. As in the case of lexical status (Experiment 1), such a pattern indicates that the effect of confusability was distributed across con-

trolled processes of all speeds. In contrast, the functions corresponding to repetitions and nonrepetitions were separated at low percentiles but converged at higher ones. This is identical to what we found for the repetition factor in Experiment 2, indicating as before that the slower controlled processes were not affected by the repetition factor.

Comparison of go-trial reaction times and response functions. For stimulus confusability, there is a pattern similar to that found for lexical status in Experiment 1. The 55-ms effect of stimulus confusability on go-trial reaction time (423 ms for low confusability and 478 ms for high confusability) was not significantly different, $t(9) = -0.894; p > .25$; from its 62-ms effect on the mean of the response function (271 ms for low confusability and 333 ms for high confusability). The pattern for the repetition factor was the same as that found in Experiment 2. The 51-ms effect of repetition on go-trial reaction time (425 ms for repetitions and 476 ms for nonrepetitions) was significantly greater, $t(9) = 3.56, p(\text{one-tailed}) < .003$; than its 22-ms effect on the mean of the response function (291 ms for repetitions and 313 ms for nonrepetitions).

Discussion

These results replicate and extend those of Experiments 1 and 2. The pattern of effects for stimulus confusability suggests

Table 4
Mean Response Probabilities and Signal Lags for Experiment 3

Stimulus type	Repetitions		Nonrepetitions	
	Probability (%)	Lag (ms)	Probability (%)	Lag (ms)
Low confusability	50	231	50	256
High confusability	49	292	50	318

that the pattern of effects for the repetition factor was not due to capacity sharing between controlled and inhibition processes. As anticipated, stimulus confusability had its effect entirely before the point of no return and did not influence inhibition processes. Moreover, the approximately equal effects of both factors on go-trial reaction time (51 ms vs. 55 ms) suggest that the repetition factor had no more of an opportunity than did stimulus confusability to influence the capacity demands of the controlled processes. From this we conclude that the pattern of effects for the repetition factor was due to its effects on the ballistic processes, rather than to capacity sharing. Given the existence of ballistic processes, we can further conclude that these processes were not affected by either lexical status or stimulus discriminability.

However, there is still one additional possibility yet to be considered. Our conclusions are based on the assumption that if controlled and inhibition processes competed for resources, then such resources would come from a single shared pool (cf. Moray, 1967; Posner & Boies, 1971). It could be argued instead that capacity sharing was not equally reflected in the pattern of effects for both factors because they modulate the demands of controlled processes upon different resources (cf. Navon & Gopher, 1979). Repetition might produce a different pattern of effects than stimulus discriminability (and lexical status) either because it alone affects ballistic processes or because it alone affects a process that shares a resource with inhibition processes. Experiment 3 does not definitively distinguish between these two possibilities. In future research, further evidence concerning the effect of repetition upon inhibition processes could be obtained by applying the additive factor method (Sanders, 1980; Sternberg, 1969) to the response function. If repetition does not influence inhibition processes, it ought to produce effects on the mean of the response function that are additive with those of other additional factors whose effects occur exclusively on inhibition processes (cf. Equation 5). One such additional factor might be the intensity of the stop signal.

General Discussion

In this article we postulate the existence of a point of no return in response preparation, where the decision to move becomes irrevocable. Choice reaction time is considered to be the sum of two processing stages temporally separated by this point: an earlier controlled stage and a later ballistic stage. We suggest an approach for separately studying the effect of experimental manipulations on each of these two stages. The approach involves modeling subjects' ability to stop a speeded response as a race between the controlled process and an inhibition process to the point of no return. We derive three predictions from these assumptions and test them in a series of three experiments.

The first prediction is that factors whose effects occur entirely before the point of no return ought to produce equal effects on the mean reaction time for go trials and the mean of the response function. This pattern was found for lexical status in Experiment 1 and for stimulus confusability in Experiment 3. Because we already have some reason to believe that both of these factors influence relatively early controlled processes, the obtained results support both our expectations as well as the

assumptions underlying our application of the race model to the countermanding procedure. Furthermore, finding equal effects on the mean reaction time for go trials and the mean of the response function supports our assumption that the inhibition process itself remains unaffected by our factor manipulations, an assumption essential for separately measuring the effects of experimental factors on controlled and ballistic processes. It also indicates that the ballistic processes were not being used to compensate for factor effects on controlled processes or being primed by partial output from controlled processes.

The second prediction is that factors that affect both controlled and ballistic processes in the same direction will produce smaller effects on the mean of the response function than on the mean reaction time for go-trials. This pattern was found for the repetition factor in both Experiments 2 and 3. Furthermore, the confirmation of Prediction 1 for stimulus confusability suggests that the pattern of effects found for the repetition factor was not due to capacity sharing between the stop and go signals. The confirmation of Prediction 2 supports our assumption that there exists a set of unstoppable processes between the point of no return and overt movement. The finding that these ballistic processes are affected by the repetition factor, in particular, indicates that they can be influenced without being triggered.

The third prediction is that the reaction-time cumulative distribution functions should form a fan pattern and that the mean reaction time on stop trials should increase with signal lag and be faster than mean reaction time on go trials. This prediction, stemming from the assumption that stop trials involve a race between the controlled and inhibition processes, was confirmed in all three experiments. Support for the race model suggests that the processing of stop signals and go signals did not compete for limited resources and that the time course of the ballistic processes is unaffected by failed attempts at inhibition.

The overall theoretical picture that emerges from the results of the three experiments is shown in Figure 12. Here reaction time is the sum of the duration of at least two distinct processing stages separated by the point of no return. The controlled stage can be affected without indirectly influencing the ballistic stage, and the ballistic stage can be affected without being triggered. Response inhibition involves a race to the point of no return between the controlled and the inhibition processes. Controlled and inhibition processes do not compete for a limited resource, and ballistic processes are unaffected by failed attempts at inhibition. Lexical status and stimulus confusability affect only controlled processes. The repetition factor affects both controlled and ballistic processes. None of these factors affect the duration of the inhibition processes.

The above theoretical picture involves a hybrid of a two-stage model and a race model. Some question currently exists over whether human information processing is best conceived as a series of discrete processing stages or as a temporally overlapping set of continuous processes (cf. Meyer, Yantis, Osman, & Smith, 1985; Miller, 1982a). Note, however, that our model is not necessarily incompatible with continuous models of information processing. Many continuous models postulate a final discrete response stage (e.g., McClelland, 1979; Ratcliff, 1978; but see also Eriksen & Schultz, 1979). This final set of processes is separate from prior processes in that once begun it is unaffected by their activity. Hence, many continuous models may

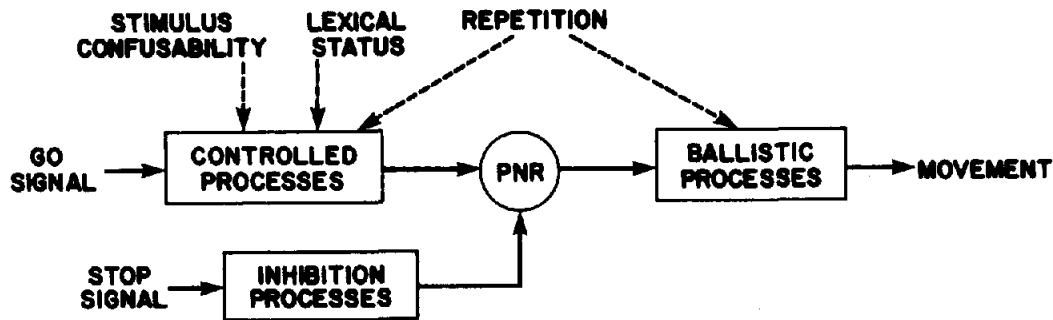


Figure 12. The effects of stimulus confusability, lexical status, and repetition versus nonrepetition on the component processes of the race model. (PNR = point of no return.)

be condensed into two global, temporally nonoverlapping, stages. Furthermore, the controlled and ballistic stages of our model could each consist of subprocesses that operate in parallel.

The use of race models is also somewhat controversial. Strong tests of such models have met with mixed success (Meyer & Irwin, 1981; Meyer, Irwin, Osman, & Kounios, 1986; Miller, 1982b). This is not surprising, because it is well known that the concurrent processing of two signals may proceed independently (cf. Shiffrin & Schneider, 1977) or may result in various types of dependence (e.g., facilitation or competition [cf. Eriksen & Schultz, 1979]), depending on the precise experimental situation. Our results, as well as those of Lappin and Eriksen (1966), Ollman (1973), and Logan (e.g., Logan, 1981; Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984), suggest that the race model may well approximate subjects' performance in the countermanding procedure under a wide range of conditions. Critical boundary conditions that lead to the model's failure may in part be determined by the perceptual relation between the stop and go signals (e.g., masking) and by the stimulus-response compatibility between the stop signals and the response ensemble (cf. Fitts & Seeger, 1953; Kornblum, Hasbroucq, & Osman, 1984).

Our most important conclusion, however, concerns the potential of the race model and countermanding procedure as a technique for separately measuring the magnitudes of factor effects on controlled and ballistic processes. The confirmation of all three predictions, as well as the general orderliness of the data, provides reasons for optimism. Future research could include other experimental manipulations designed to demonstrate independence between the processing of the stop and go signals. It would also be interesting to study experimental manipulations believed to affect motor processes, such as response complexity (Henry & Rogers, 1960). Discovering which set of processes is affected by these factors ought to provide some insight into the nature of these processes, as well as to constrain possible models of motor programming. If the point of no return continues to be a useful construct, its role in partitioning reaction-time effects will provide an important contribution to the study of the psychological processes underlying movement.

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