CONSTRAINTS ON THE LOCUS / LOCI OF THE ATTENTIONAL BLINK PHENOMENON

by
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ABSTRACT

The locus of the Attentional Blink (AB) was investigated using AB and Psychological Refractory Period (PRP) experimental paradigms. The first task in the AB experiments (Exps. 1 and 2) was to identify a target letter (T_1), which was an H, O, or S, presented in an RSVP stream of distractor letters. The second task in these experiments was to identify a second target letter (T_2), an X or Y. In Experiment 1, the response to T_1 was speeded and in Experiment 2 it was unspeeded. T_2 always appeared in one of the 8 positions in the RSVP stream immediately following T_1 and the response to T_2 was never speeded. Accuracy on identifying T_2 was severely attenuated when T_2 followed within approximately 500 ms after T_1. In the PRP experiments (Exps. 3 and 4), the first task was to judge whether an auditory tone (S_1) was low, medium, or high in pitch. Following the tone, at variable stimulus-onset asynchrony (SOA), a target letter (S_2) was presented and the second task was to identify the target letter, which could be an H, O, or an S. In these experiments both responses were speeded. Mean response time to S_2 slowed as SOA decreased.

The probability of the target letter, T_1 in the AB experiments, and S_2 in the PRP experiments, was manipulated so that one letter was assigned a relative frequency value of 1, another a value of 4, and another a value of 9. In the AB experiments the least frequently presented target letter produced a larger AB effect than the target letter assigned to the intermediate frequency condition, which in turn produced a larger effect than the most frequently presented target letter. These results indicate that the locus of signal probability is at, or before, the locus of the AB effect. In the PRP experiments, signal probability was additive with SOA. According to the locus of cognitive slack logic (Pashler & Johnson, 1984), additive effects indicate that the locus of probability is at, or beyond, the locus of the PRP bottleneck.

These results lead to the conclusion that a locus of the AB effect is at, or after, the PRP bottleneck. It is generally believed that the PRP bottleneck is located at a late stage of information processing, such as response selection. Thus, these results suggest that the AB effect also has its locus at a late stage of processing, possibly at, or after, the stage where responses are selected.

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"...for he dwelleth with you, and shall be in you."
John 14: 17

For Jeff, with love

TABLE OF CONTENTS

LIST OF FIGURES

When stimuli are presented in rapid serial visual presentation (RSVP) it is usual to observe an interference effect between sequential items. Raymond, Shapiro, and Arnell (1992) reported such an effect in an RSVP stream where target letters were embedded among distractor letters. A relationship was observed between correct identification of a second target (T_2) and its temporal proximity to a preceding target (T_1). The proportion of correct identifications of T_2 decreased as the number of items between T_1 decreased, except when there were no intervening items (when T_2 appeared immediately after T_1). This attenuation in performance on accurately identifying T_2 when it appears within approximately 100 to 500 ms following T_1 has been termed the Attentional Blink (AB) by Raymond et al. (1992).

Evidence suggests that interference effects like the AB are a result of attentional limitations in our ability to process information (Chun & Potter, 1995; Jolicoeur, 1998a; Luck, Vogel, & Shapiro, 1996; Raymond et al., 1992). However, our understanding of the nature of these limitations is incomplete. For example, at what processing stage(s) does interference occur, and what are the stimulus factors that modify the size of the effect?

The aim of this thesis is to understand more fully the characteristics of the AB phenomenon with specific focus on learning more about the locus, or loci, of this interference effect. Where, within the series of processing stages necessary to perform a first task, does interference on a second task occur?

The Attentional Blink

In one of the original studies designed to investigate the AB phenomenon, Raymond et al. (Exp. 2, 1992) used an RSVP stream of black uppercase letters presented on a grey background at a rate of 11 items/sec. A white target letter (T_1) was presented within the stream. T_1 was always preceded by seven to fifteen distractor letters and was followed by eight letters. On half the trials a probe letter (T_2), a black X, appeared in any one of the eight positions immediately following the target. These eight positions, subsequently referred to as T_1-T_2 lags 1 though 8, denote the positions that followed T_1 in which T_2 could appear. T_2 never appeared in any other position within the stream.

In the experimental condition the first task was to identify T_1 and the second task was to report whether T_2 had been present or absent. In the control condition, only the second task was performed and T_1 could be ignored.

Accuracy on judging whether T_2 had been present or absent dropped to below 60% when T_2 appeared at T_1-T_2 lags 2 - 5 following T_1, compared to about 90% correct, across all T_1-T_2 lags, in the control condition (T_1 was ignored).

The results show the effect is the outcome of processing necessary for T_1 that interferes with processing a second item (T_2), when the second item follows the first within a certain time frame (~100 - 500 ms). It was this deficit in processing T_2, at short T_1-T_2 lags, that Raymond et al. (1992) termed the AB. The results also suggest that the AB effect is not the consequence of sensory masking by T_1, because both experimental and control streams were identical. Rather, the effect depends on processing the target in some way. This observation in turn suggests that the AB effect is due to attentional limitations in our ability to process information.

The AB effect is not restricted to letters and has been shown to be generalizable to many types of stimuli including colour (Ross & Jolicoeur, 1999), words (Broadbent & Broadbent, 1987), orientation of T_2 (Joseph, Chun & Nakayama, 1997), location (Ross & Jolicoeur, in press), and when T_1 was in the form of a random dot pattern (Shapiro, Raymond & Arnell, Exp. 4, 1994).

Neither is it limited to the visual modality. Though less robust than that found using visual stimuli, Arnell and Jolicoeur (1998) reported a significant AB effect using rapid auditory presentation (RAP). Interestingly, there was a difference in the presentation rate necessary to produce an AB for visual and auditory stimuli in that the auditory stream had to be presented at a faster rate than RSVP. Also, when T_1 modality and T_2 modality were crossed (visual-auditory; auditory-visual) an AB was observed for both cross-modal conditions. Jolicoeur (1999c) has also reported AB effects under cross-modal conditions using visually presented distractor items and an auditory T_1 stimulus. In this study, T_1 was a 100 ms duration auditory tone presented concurrently with an RSVP stream of letters, while T_2 was a letter within the stream.

The finding of a cross-modal AB effect is consistent with the hypothesis that a central mechanism underlies the effect. If the processing limitations that produce the AB effect are modality specific then one would expect, if not an elimination, at least a large reduction in the size of the AB for cross-modal T_1 - T_2 conditions.

AB effects for auditory and cross-modal presentations are not consistently found however. Contrary to the findings of Arnell and Jolicoeur (1998), Potter, Chun, Banks, and Muckenhoupt (1998) used RAP and found that, although T_2 accuracy was poorer in the T_1-present condition compared to the control condition (T_1-absent), there was no effect of T_1-T_2 lag. Typically, AB is observed not only as a decrement in overall performance on detecting or identifying T_2, but also as a decline in T_2 accuracy as a function of the position of T_2 in relation to T_1. Accuracy on T_2 improves as the number of lag positions between T_1 and T_2 increases. In the study conducted by Potter et al. (1998), no such relationship was found. These researchers also found no AB effect, that is, no performance decrement and no function of T_1-T_2 lag for cross-modal presentations.

The discrepancy between experimental outcomes appears to be a function of the modality in which the rapid stimulus stream is presented. The magnitude of the AB effect is relatively reliable and stable in the visual realm but effects using RAP are less consistent and certainly this discrepancy requires further investigation. Possible explanations for differential effects will be discussed more fully in the next section, under the heading "Perceptual Influences."

Perceptual Influences

It does not appear that the AB effect is a consequence of processing limitations at an early sensory encoding stage. If the effect was due to masking of T_2 by T_1, then the same effect should be found in the control condition, where T_1 was ignored, because the streams were identical. Therefore, the AB effect is not simply a form of sensory masking.

Nevertheless, there are conditions under which perceptual masking appears to be a necessary component for producing an AB effect. Giesbrecht and Di Lollo (1999) reported

that, when T_2 was the last item in an RSVP stream or when it was followed by non-patterned visual noise, no AB effect was evident. Therefore, the AB effect requires pattern masking T_2.

The effect of masking T_1 seems to be more variable. Raymond et al. (1992) found no decrement in T_2 accuracy when the first (+1) item after T_1 was replaced by a blank interval. When the +1 item was present and the second item (+2) was replaced by a blank interval the AB effect was restored. Seiffert and Di Lollo (1997) also compared conditions where the +1 item was blank to where it was filled and found a reduced, though significant AB effect, in the blank +1 item condition.

Moving the +1 item 1 degree of visual angle to the right of the location of T_1 (T_1 was a letter, ~ 0.82^0 visual angle) has also been found to reduce the size of the AB, as did increasing the featural distinction between the stream and the +1 item (Raymond, Shapiro, & Arnell, 1995). The latter manipulation was accomplished by inserting an array made up of four black dots positioned randomly within the area that would typically be occupied by the +1 item in an RSVP letter stream.

Though perceptual masking, of T_2 by T_1, cannot account for the AB effect, it does appear that, under certain conditions, masking is an important factor controlling the manifestation and magnitude of the effect.

As discussed in the previous section, a larger AB effect has been observed with visual (RSVP) than with auditory (RAP) streams (Arnell & Jolicoeur, 1998) yet the underlying explanation for this differential effect is not clear. Potter et. al. (1998) hypothesize that the variation in the effect size is a result of different interference processes, one strictly visual and another that is amodal. Contrary to the findings of Arnell and Jolicoeur (1998), Potter et al. (Exps. 1 & 2, 1998) initially found a deficit in accuracy on detecting T_2 but no effect of T_1-T_2 position with auditory streams.

It is important to keep in mind that the SOA for items in the RAP stream used by Potter et al. (1998) was always longer than that used by Arnell and Jolicoeur (1998). The variations observed in the outcome of experiments using RAP could very well be due to this methodological difference. Stimulus-onset asynchronies of 120 and 135 ms, which were used

by Potter et al. (1998), may have allowed enough time between RAP items so that processing T_1 did not interfere with processing T_2. In contrast, the shorter SOA of 93.3 ms used by Arnell and Jolicoeur (1998), may have produced the necessary conditions, by reducing the time between RAP items, in which processing T_1 interfered with processing T_2. We know that masking of T_2 is a necessary condition for an AB effect to be observed (Giesbrecht & Di Lollo, 1999), and also that masking of an unspeeded T_1 stimulus is a requirement (Raymond et al., Exp. 3, 1992). Thus, variations found using RAP may be due to differential masking conditions. If the rate of presentation of items within a stream is not sufficient to provide the necessary masking conditions it is not surprising to find no AB effect.

Response Influences

A partial explanation for the divergent effects observed with perceptual masking may be that different or additional processing stages contribute to interference when a response to T_1 is speeded as opposed to when it is unspeeded. If the T_1 response is not speeded, and is made at the end of the stream, masking of T_1 seems to be necessary for an AB effect. In contrast, when the response to T_1 is speeded, in other words, when it is made as quickly as possible after detection, masking does not seem to be a requirement for AB. The difference between masking requirements for speeded and unspeeded T_1 responses and the differential effects on the AB is perhaps suggestive of the necessity for immediate processing of T_1. Under speeded, and unspeeded but masked, T_1 conditions, information must be processed immediately and on-line. In the former case it is the immediacy of the on-line response that forces processing, whereas in the latter it is immediacy of processing to a sufficient extent prior to the onset of the masking event (such as the next item in the RSVP stream). In both circumstances the decline in accuracy on T_2 identification as a function of its temporal proximity to T_1 could be a reflection of ongoing processing related to the T_1 item. Whether both situations reflect interference at the same processing stage(s) is ambiguous at this point.

In a between subjects experiment, Jolicoeur (Exp. 3, 1999a) compared speeded T_1 responses to unspeeded responses. A larger AB effect was found for the T_1 speeded condition

at shorter T_1-T_2 lags (1 - 4) but at the longer T_1-T_2 lags the unspeeded condition produced a larger AB effect. The interaction between T_1 response conditions and T_1-T_2 lag implies that different processing mechanisms may underlie speeded and unspeeded conditions, which in turn suggests that variations in more than one stage of processing may contribute to interference producing the AB phenomenon. This line of thinking will be discussed more fully later in this thesis.

Theories of the Attentional Blink Phenomenon

Attentional Gate Theory

The Attentional Gate Theory (Raymond et al., 1992) comprises two stages of processing, a preattentive detection stage during which attention is allocated, and an identification stage. The model postulates that, as visual information flows from sensory input sites to recognition centers of the brain, an attentional gate is opened if a target-defining feature is detected. The gate remains open until processing is complete and identification is achieved. Under conditions where the possibility of confusion exists, for example when stimuli are presented in rapid succession and the +1 item enters the buffer along with T_1, a suppressive mechanism is initiated in which the gate closes and is temporarily locked. Subsequent attentional allocation to the next episode takes longer to initiate when the gate is "locked" compared to when the gate is simply closed. The consequence of this delay is manifest as an AB effect.

Evidence against the Attentional Gate Theory has been provided in AB paradigms where T_1 detection, without identification, is sufficient for an AB (Shapiro et al., Exp. 2, 1994). Reasoning that letters may be identified even involuntarily (when subjects are instructed to report the presence but not the identity of T_1), Shapiro et al. (Exp. 4, 1994) obtained an AB effect in an experiment where $Task_1$ and $Task_2$ were both unspeeded and subjects were first to detect the presence or absence of a random dot pattern, followed by detection of T_2 (an X). The results demonstrate that detection of T_1 is sufficient to produce an interference effect. More recently, the finding of AB effects when T_1 and T_2 are presented in different modalities (Arnell

& Jolicoeur, 1998), does not support the Attentional Gate Model of interference. It is unlikely that the closing of an attentional gate in one modality would have detrimental effects on processing a subsequent target stimulus presented in another modality. AB effects have been observed using non-patterned target information (Ross & Jolicoeur, 1999) a finding that is also inconsistent with the Attentional Gate Theory .

Similarity Theory

Having demonstrated an AB effect for target detection without identification, Shapiro and his colleagues (1994) found it necessary to revise the two-stage Attentional Gate theory. Moreover, they had found that the AB was eliminated when T_1 was a blank interval instead of the usual letter item and thereby contained no patterned information (Shapiro et al., Exp. 5A, 1994). In this experiment subjects were instructed to make a judgment (present or absent) pertaining to T_1 which was a temporal gap in the RSVP stream. According to Shapiro et al. (1994) the observation that no AB existed when T_1 was made up of non-patterned information supported the idea that, although detection of T_1 without identification would suffice to produce an AB effect, the requirement that T_1 consist of patterned information was essential. What the researchers failed to note was that under these conditions, where T_1 was a temporal gap, masking of T_1 was also eliminated, a condition that may have caused the small observed effects in $Task_2$. As already mentioned, AB effects using non-patterned information (colour, location) have since been reported by Ross and Jolicoeur (1999).

According to the Similarity Theory (Shapiro et al., 1994) items in visual short-term memory (VSTM) compete for report based on an assigned weighting. In the initial stage of processing, structural representations of items in the RSVP stream are formed. Templates of T_1 and T_2 are compared to these representations and matching items are entered into VSTM where they are assigned a weight depending on their similarity to the templates. The similarity of the distractor items to T_1 and T_2, especially those distractors that immediately follow T_1 and T_2, is reflected in the weighting value which not only affects the probability of an item gaining access to VSTM but also the likelihood of later report. Distractor items will typically be less heavily

weighted than target items but the value of the weight assigned to them may be sufficient to create some confusion. The AB effect is a result of item confusion at the time of output from VSTM. This short-term memory bank has limited capacity and items do not remain indefinitely in VSTM. The relative absence of an interference effect when T_2 appears at the longer T_1-T_2 lags (> ~600 ms) is due to the removal of T_1, possibly through decay, from VSTM.

The Similarity Theory is limited to the VSTM and does not predict a similar pattern of T_2 interference when stimuli enter auditory short-term memory. Consequently, findings of AB effects using auditory stimuli (Arnell & Jolicoeur, 1998, Jolicoeur, 1999c) do not support the Similarity Theory.

Attentional Dwell Theory

In contrast to the preceding theories, the Attentional Dwell Theory proposes that the AB is caused by how attention is (or is not) deployed rather than by factors like perceptual masking (Duncan, Ward, & Shapiro, 1994; Ward, Duncan, & Shapiro, 1996). Ward et al. (1996) propose that attention is a sustained state, not a high-speed serial process, and that this state is necessary for the creation of object representations, which in turn will guide subsequent cognitive processing. Because attention does not proceed in a high-speed serial fashion, all items in the stream are not attended to equally. Limitations arise when items compete for capacity-limited processing resources on the basis of their match to a representation of the target. Demand increases as the number of items attended increases.

Support for this theory of attentional limitations comes from a study in which either one or two stimuli had to be identified followed, at variable stimulus onset asynchrony (SOA), by a second stimulus (Ward et al., Exp. 3, 1996). In this study either one digit was presented, to the left or right of fixation, or two digits were presented simultaneously, one to the left of fixation and the other to the right of fixation. Following the digit(s), a letter appeared either above or below fixation. SOA was varied, ranging from 0 to 900 ms. Stimulus exposure was determined for each subject during a practice session and ranged between 45 - 75 ms and each character was followed by a 250 ms masking pattern. There were four response conditions

associated with this experiment and these conditions were blocked within subjects. When only one digit was presented subjects were either to a) ignore the digit and report the letter or b) report the digit and the letter. When two digits appeared subjects were to a) ignore both digits and report the letter or b) report both digits and also the letter.

The data exhibited a similar pattern of interference as is typically found in experiments designed to produce AB effects (this study did not employ an RSVP stream). In conditions where the first item, the digit, could be ignored accuracy on reporting the second item, the letter, was relatively high (approximately 90%) and was not affected by SOA. When the digit(s) had to be reported, in the report one digit condition and in the report two digit condition, accuracy on identifying the letter dropped significantly when the letter appeared up to 300 ms after the digit(s) after which point performance improved as SOA increased. The greatest degree of attenuation in letter identification was found in the report two digits condition, which was significantly poorer than when only one digit had to be reported. Processing required for stimuli presented simultaneously (two digits) resulted in a greater degree of interference on a subsequent stimulus (a letter) than did processing only one initial stimulus (one digit). According to the Attentional Dwell Theory, when two stimuli need to be processed concurrently, capacity must be shared resulting in less availability of resources necessary for processing a third stimulus. These results suggest that interference is a function of the number of items to be attended, not the number of items to be ignored, and the temporal proximity of those items (Ward et al., 1996).

However, in an RSVP paradigm, Seiffert and Di Lollo (1997) did not find a difference in accuracy for detecting a second item as a function of identifying one or more previously presented items. Subjects were required to identify one, or two simultaneously presented, T_1 items and say whether a T_2 item, that followed at variable T_1-T_2 lags, was present or absent. In the one-T_1 condition two letters were presented beside each other (one of the letters was in the same location as the rest of the RSVP stream and the other was directly to the right). The letters differed in brightness and the brighter of the two letters was T_1 which was the letter to be reported. In the two-T_1 condition both letters were of equal luminance and both were to be

reported. No significant difference was found between the two conditions. In this experiment the +1 position was blank and accuracy for T_2 was poorest when it appeared in the second position (+2) following T_1. Performance thereafter improved steadily as a function of increasing T_1-T_2 lag. The lack of T_1 masking due to the omission of the +1 item in this experiment may have allowed two T_1 stimuli (in the two T_1 condition) to be processed to an equivalent degree, thus producing comparable effects in the one, and two, T_1 conditions.

Two-Stage Theory

A Two-Stage Theory of interference within the AB paradigm has been proposed by Chun and Potter (1995). In this theory all items within the RSVP stream are initially processed to a degree that allows them to be detected and identified briefly. Representations decay rapidly if they are not selected for further processing due to interference from other items. A second stage of processing is capacity limited and entails consolidating this early form of processed information to a more stable form, perhaps verbal short-term memory (STM). The Two-Stage Theory asserts that the consolidation process necessary for one item has to be complete before the same process necessary to consolidate another item can begin. For example, processing necessary for T_1 must be finished before processing of T_2 can proceed. If processing of T_2 has to be delayed, or postponed, the outcome is that information will be subject to rapid-forgetting resulting in difficulty in correctly reporting the identity of T_2.

In contrast to the Attentional Gate and the Similarity theories, Chun and Potter (1995) hypothesize that visually patterned information is not a necessary requirement for observing an AB. They propose however, that an AB effect is more likely to be found with visual than with auditory input and they suggest that one reason might be because VSTM has a more limited capacity and representations are stable for a shorter period of time than auditory STM, thereby providing greater opportunity for confusion or decay of items. This hypothesis can provide the basis for an explanation of the finding that the magnitude of the AB effect observed using auditory stimuli is sometimes smaller than that found for visual stimuli.

Central Interference Theory

The Central Interference Theory (Jolicoeur, 1999a) outlines an information processing model and suggests how the processing stages contained within the model are affected by interference. Moreover, the theory offers an account of the relationship between processing stages, capacity limitations, and performance.

The two earliest stages in the Central Interference Theory resemble the first stage of Chun and Potter's Two-Stage Theory (Chun & Potter, 1995). Stage one is a sensory encoding stage that is a relatively capacity-free sensory store of representations that have not yet been categorized. These low-level representations are susceptible to masking by other sensory events. Perceptual encoding follows. This stage produces post-categorical representations that are immune to masking. This stage is also capacity-free and, because representations here contain identity information, it is at this stage that items are selected for further processing. If not processed further the representations rapidly decay.

Although these early encoding stages process information to the level of identity, the form of the representation at this point is not adequate to serve as the basis for a response. According to the Central Interference Theory, in order for a response to be made information must be converted to a more stable form suitable for further processing (Duncan, 1980).

In the ensuing stage, selective control, representations produced by perceptual encoding are matched against selection criteria, such as colour and size as well as those criteria that distinguish members of a category (e.g., letters from digits; 'H' from 'S'). When a response is not immediately required (unspeeded T_1 response), a fourth stage, short-term consolidation, is required. Only items that have been selected for further processing advance to short-term consolidation and, subsequently, to a more durable form of storage in memory, called durable storage (Coltheart, 1980), which is likely akin to short-term memory (STM). Consolidation takes time and the duration of the process is dependent on the amount of information to be consolidated (Jolicoeur & Dell'Acqua, 1998). Information stored at the durable storage stage is stable for a relatively long period of time and is sufficiently processed so that a response can be made. When a response is required immediately upon detecting a stimulus within a trial,

transfer of the information to durable storage is not necessary and a response can be initiated at a separate response selection stage. The stages of short-term consolidation and response selection mutually interfere with each other so that when T_1 is speeded, short-term consolidation of T_2 is postponed due to response selection of T_1. When T_1 is unspeeded, the short-term consolidation of T_2 is delayed until short-term consolidation of T_1 is complete. The different processing stages engaged in speeded and unspeeded T_1 responding might be related to the differential effects of masking noted earlier. If T_1 is not masked and an unspeeded response is required a reduced AB is observed (Raymond et al., 1992; Seiffert & Di Lollo, 1997). But, when a speeded, on-line, response is made, masking does not seem to be a necessary requisite for the manifestation of an AB effect. The common factor seems to be that under both conditions a form of immediate, on-line processing is required and, though processing may conceivably have different predominant goals, it is the immediacy of processing, plus the capacity demands of that processing, that cause interference in subsequent processing and result in an AB effect.

Jolicoeur (1999b) proposed a general model of the AB effect, based on the Central Interference Theory, to explain the finding that an effect of increasing the number of stimulus-response alternatives was only evident when the response to T_1 was speeded. According to his account, when the response to T_1 is speeded, storage of a representation of T_1 in STM is not needed because the response occurs on-line and T_1 must be processed immediately. Response selection therefore takes place prior to the short-term consolidation of T_2. When the number of stimulus-response alternatives in $Task_1$ is increased, for example from two-alternative discrimination response to four-alternative discrimination response, the duration of the response selection stage is increased and the short-term consolidation of T_2 is postponed accordingly. The consequence of this sequence of events is a reduction in accuracy in $Task_2$. When the response to T_1 can wait until the end of the trial, the short-term consolidation of T_1 must occur so that a representation can be held in memory until a response is made. Thus, when $Task_1$ is unspeeded, short-term consolidation of T_1 and T_2 will both take place prior to response selection. Because response selection for $Task_1$ will occur after short-term

consolidation of T_2, the process of response selection will not affect $Task_2$ performance, when the response to T_1 is delayed until the end of the trial.

However, AB effects are seen when responses to T_1 are unspeeded (Jolicoeur, 1999a; Raymond et al., 1992; Raymond et al., 1994; Seiffert & Di Lollo, 1997, Shapiro et al., 1994). Jolicoeur (1999a) proposes that, under these conditions, the process of consolidating T_1 for entry in to STM interferes with the same process associated with T_2. Hence, when the T_1 response is unspeeded, and short-term consolidation of T_1 is necessary, short-term consolidation of T_2 is postponed until processing for T_1 is complete (Chun & Potter, 1995). When T_1 is speeded the process of consolidating T_1 does not occur, but response selection associated with T_1 causes the short-term consolidation of T_2 to be postponed. In either case, the delay of short-term consolidation of T_2 results in a decline in accuracy on detection and/or identification of T_2, possibly because of decay of the T_2 representation prior to the initiation of the processing necessary for entry in to STM.

Evidence, consisent with the account of interference proposed by the Central Interference Theory, shows that the process of consolidating T_1 is not exclusive to interfering with short-term consolidation of T_2, but can also postpone the process of response selection of T_2 (Jolicoeur & Dell'Acqua, 1998). When response to a first stimulus is unspeeded, to be made at the end of a trial, short-term consolidation of that stimulus is necessary. When the response to a second stimulus is speeded, the process of response selection for that stimulus is required. Thus, according to the Central Interference Theory, when an unspeeded task precedes a speeded task the process of short-term consolidation should postpone response selection of the second stimulus when SOA is short. Such was the case observed in an experiment in which the first task was to remember items (1 - 3 letters) to be reported at the end of the trial, and the second task was a two-alternative discrimination speeded response to an auditory tone (Jolicoeur & Dell'Acqua, 1998). In addition to an effect of SOA, an effect of the number of items to be remembered affected response time (RT) to the tone. Response time to the tone increased as SOA decreased, and as the number of items to be remembered increased. When no items were to be recalled there was no such effect on tone RT. Jolicoeur and

Dell'Acqua (1998) interpreted the results as evidence that the process of short-term consolidation necessary for the first stimulus postponed the process of response selection of the second stimulus.

Psychological Refractory Period

One form of dual-task interference that may be related to the AB effect is the Psychological Refractory Period (PRP) effect. In a typical PRP design two stimuli are presented with no intervening filler items. SOA is varied and a speeded response is required to both stimuli. The PRP effect is observed as a slowing in performance on a second task ($Task_2$) as a function of its temporal proximity to a first task ($Task_1$). Response times to $Task_2$ are typically delayed when the stimuli are presented at shorter SOAs compared to when they are presented at longer SOAs (Pashler, 1994b; Welford, 1952).

Found even using very simple tasks, such as detection, the PRP effect is a robust interference effect that appears to be relatively resistant to practice (Gottsdanker & Stelmach, 1971; McCann & Johnston, 1992; Van Selst & Jolicoeur, 1997). It has been observed using visual stimuli (Luck, 1998; Pashler, 1994a), as well as under cross-modal conditions (McCann & Johnston, 1992; Pashler, 1994b; Van Selst & Jolicoeur, 1997). Moreover, output modality does not need to be the same for each task. Pashler (1989, 1990) observed PRP effects when manual and vocal responses were combined.

Theories of the Psychological Refractory Period Phenomenon

Capacity Theory

In an endeavor to understand the PRP effect, theorists have pursued two distinct paths -- the capacity and postponement theories. Capacity theories propose that PRP is a reflection of limitations in available resources needed to process information (Kahneman, 1973; McLeod, 1977). When two stimuli appear in close succession to each other they may require the same processing resources. Because processing of the stimuli occurs in parallel these processors must be shared and the effect of this division is seen as a slowing in response times. The closer

in time the stimuli are to each other the more resources must be shared and the greater the slowing. Some researchers suggest that a single mental resource is responsible for the capacity limitations (Kahneman, 1973) while others favour a multiple resource view (Wickens, 1980).

Evidence against this theory lies in the fact that, if capacity is allocated equally between tasks, as might be expected when a response is required to more than one stimulus, reaction time to the first and second stimuli should show equivalent effects of SOA, slowing as SOA decreases and greater demand is placed on resource sharing. Rarely is this the case.

Most studies investigating the fundamental mechanism underlying the PRP instruct subjects to respond as fast as they can to the stimuli. If individuals are able to divide available resources unequally between tasks, so that more capacity is given to the first task than the second, they may do so in an effort to respond to the first task as quickly as possible. In such a case, bottleneck-like effects would be observed (i.e. effects of SOA on $Task_2$ but not $Task_1$), even though shared resources is the limiting factors underlying the PRP effect. However, when subjects are specifically instructed to place equal emphasis on each response they appear to be unable to do so. Instead, they typically show a bimodal distribution of RT_1 and RT_2 in that long RT_1s correlate with long RT_2s, and short RT_1s correlate highly with short RT_2s (Pashler, 1994c). This pattern of results reveals that subjects have difficulty dividing resources based on instructions. and sheds doubt on the interpretation that they do so when instructed to make speeded responses to consecutive tasks.

Manipulating $Task_2$ difficulty and SOA yields specific predictions for a limited resource account of interference. If $Task_2$ difficulty is varied so that there is more than one level, the effect of this manipulation should increase in magnitude as SOA decreases (McCann & Johnston, 1992). As SOA decreases and task overlap increases more demand is placed on shared resources and a larger effect of $Task_2$ difficulty should be observed than is seen at longer SOAs. Typically, the magnitude of the effect of $Task_2$ difficulty either remains constant across SOAs or increases as SOA increases thereby weakening the idea that we are dealing with a capacity-sharing model (McCann & Johnston, 1992; Pashler & Johnston, 1989).

Postponement Theory

An alternate, and generally more widely accepted theory of dual-task interference, is a postponement theory, which postulates a delay of processing rather than sharing of resources (Pashler, 1994b; Welford, 1952). A primary assumption of a postponement account of interference is that, if resources necessary for one task are common to both tasks, processing will not proceed in a concurrent fashion. Processing of $Task_2$ will be forced to wait until resources used for $Task_1$ become available. It is this 'wait' period that is reflected in the slowing of reaction time to the second task as SOA is reduced.

Some evidence for an information processing bottleneck underlying dual-task interference lies in the fact that, on a trial to trial basis, reaction times to both stimuli primarily show a positive correlation. If $Task_1$ takes relatively long to complete, 'wait' time for $Task_2$ will likewise be longer and, alternatively, faster $Task_1$ processing will be reflected in less 'wait' time and consequently faster $Task_2$ processing.

Locus of Cognitive Slack Logic

In addition, certain predictions can be made, based on the postponement theory and the supposition of a 'wait' period, that might help to verify the existence of such an underlying mechanism. The locus of cognitive slack logic can be used to interpret the effect of manipulating a $Task_2$ variable in relation to the PRP bottleneck (McCann & Johnston, 1992; Pashler & Johnston, 1989).

If, for example, the level of difficulty associated with $Task_2$ is varied and the effect of this manipulation occurs at a stage of processing that constitutes a bottleneck, or at a stage beyond the bottleneck, an additive data pattern across SOAs is observed.

Insert Figure 1 about here

The reasoning behind this logic can best be explained by referring to Figure 1. Stages 1A through 1C in Figure 1 refer to hypothetical processing stages necessary to complete a task ($Task_1$). Stages 2A through 2C refer to processing necessary for a second task ($Task_2$). Figure 1 also depicts two levels of difficulty for $Task_2$, easy and hard, and two levels of SOA, short and long.

Let us assume that manipulating the difficulty of $Task_2$, in this hypothetical example, affects stage 2B, in that the duration of stage 2B is longer when $Task_2$ is more difficult. Let us further assume that stage 1B of $Task_1$ involves the same processors as stage 2B, thus constituting a processing bottleneck. This means that processing involved in stages 1B and 2B cannot occur in parallel. Stage 2B processing will always have to wait until stage 1B is complete.

As the figure shows, at the long SOA there is no task overlap and all processing necessary to perform $Task_2$ (stages 2A, 2B, 2C) begins after the bottleneck stage of $Task_1$ (1B), regardless of $Task_2$ difficulty. Increasing $Task_2$ difficulty increases the duration of stage 2B, and this increase in processing time is entirely reflected in response time to the second task (RT_2). RT_2 increases as the duration of stage 2B increases.

When the two tasks overlap, that is, when SOA is short, processing for $Task_2$ begins soon after the commencement of processing for $Task_1$. Figure 1 shows that, at a short SOA, stage 2A begins shortly after the onset of stage 1A. Because stages 1B and 2B cannot be carried out simultaneously, stage 2B is postponed until 1B is complete. This postponement of stage 2B processing creates a 'wait' period, or period of cognitive slack (McCann & Johnston, 1992), allowing any $Task_2$ processing that occurs prior to stage 2B to continue uninterrupted. However, further processing for $Task_2$ (stages 2B and 2C) cannot continue until the $Task_1$ bottleneck stage is free, that is, until stage 1B of $Task_1$ is complete. Thus, an increase in the duration of stage 2B, due to varying the difficulty of $Task_2$, will again be seen as an increase in RT_2 as task difficulty increases. Even at short SOAs then, when varying the difficulty of $Task_2$ affects the duration of a stage of processing at, or beyond, the bottleneck, changes in the

duration of that stage of processing are fully reflected in RT_2. This effect will be consistent across SOAs and produce a pattern of results that is additive with SOA.

Insert Figure 2 about here

An example of hypothetical data displaying additive effects in mean RT is shown in Figure 2. Additive effects of $Task_2$ difficulty and SOA have been produced by manipulating variables that have their effects late in the sequence of processing stages, at or beyond the PRP bottleneck. S-R compatibility for example, a variable believed to affect the stage of response selection, has been found to produce additive effects with SOA (McCann & Johnston, 1992).

Insert Figure 3 about here

The outcome of manipulating a $Task_2$ variable that has its effect prior to the PRP bottleneck is somewhat different. Figure 3 is a schematic representation of the hypothetical processing stages employed in performing $Task_1$ and $Task_2$. Also depicted are two levels of SOA (long and short), and two levels of $Task_2$ difficulty (easy and hard). In Figure 3, just as in Figure 1, stages 1B and 2B require the use of common processors and hence cannot occur in parallel. In contrast to Figure 1 however, Figure 3 depicts a stage of processing affected by varying $Task_2$ difficulty that occurs prior to the bottleneck, that is, the manipulation has its effect is at stage 2A.

When SOA is long the model depicted in Figure 3 is similar to that described in Figure 1. Processing necessary for $Task_2$ can continue uninterrupted and any increase in the duration of stage 2A, brought about by increasing $Task_2$ difficulty, will be reflected in RT_2.

In contrast, at short SOAs, stage 2B must be postponed until stage 1B is complete. However, when processing associated with varying $Task_2$ difficulty occurs before the PRP bottleneck, it is not affected by postponing $Task_2$ processing. Because a period of cognitive slack is created when stage 2B is postponed, stage 2A processing can continue uninterrupted regardless of the duration associated with the level of task difficulty. Although $Task_2$ processing that involves the bottleneck and subsequent stages will be delayed until bottleneck processing for $Task_1$ is complete, these stages are not affected by the experimental manipulation. Consequently, the duration of these later stages will be similar for all levels of $Task_2$. At short SOAs then, the effect of varying $Task_2$ difficulty, when the manipulation affects a stage of processing before the PRP bottleneck, will not be reflected in RT_2.

Insert Figure 4 about here

The overall effect of manipulating a stage of $Task_2$ processing that occurs prior to the PRP bottleneck is that the difference in performance between the levels of $Task_2$ will decrease as SOA decreases. The effects of this $Task_2$ difficulty manipulation will therefore be observed as being underadditive with decreasing SOA. An underadditive pattern is shown in Figure 4. Underadditive effects with decreasing SOA have been observed for $Task_2$ variables, like stimulus contrast (Pashler & Johnston, 1989; Van Selst & Jolicoeur, 1994a), that are believed to affect early processing stages, such as encoding.

Insert Figure 5 about here

An alternative pattern, overadditivity, is predicted by a resource sharing model. According to this model, the PRP effect is caused by excessive demand on a limited amount of resources. This relationship is affected by the time between tasks in that the closer two tasks are to each other the greater the demand on resources. Additional demand is placed on resources if the level of difficulty of one, or both tasks, is increased. This effect, of increasing the level of difficulty of $Task_2$, is exacerbated at short SOA compared to long SOA because there is already a greater shortage of resources at short SOA than at long. Thus, the effect is overadditive with decreasing SOA, and data points will diverge at shorter SOAs and converge at longer SOAs. Such a pattern is shown in Figure 5.

Manipulating a variable that is likely to have an effect at a specific stage of processing (e.g., sensory encoding, response selection, etc.), and interpreting the pattern of data using the locus of cognitive slack logic described above (McCann & Johnston, 1992; Pashler & Johnston, 1989), has the potential to be a useful method for obtaining empirical evidence regarding the locus(i) of the PRP bottleneck. Underadditivity in the results reflects the effects of a variable prior to the PRP bottleneck and additivity reflects the effects of a variable that has its locus at, or beyond, the bottleneck. However, overadditivity suggests a capacity-sharing resource account of interference which would suggest concurrent processing of $Task_1$ capacity demanding stages and the stage affected by the $Task_2$ manipulation.

Locus(i) of the PRP Bottleneck

Perceptual bottleneck

Evidence against a perceptual PRP bottleneck comes in several forms. Primarily, if the bottleneck were perceptual in nature the observation of cross-modal PRP effects would not be expected. Perceptual processing of information presented to different sensory modalities should not yield interference effects like those found by McCann & Johnston (1992) using auditory and visual stimuli. In addition, a number of studies have obtained underadditive effects by varying stimulus intensity, a variable assumed to have an effect at perceptual encoding (De Jong, 1993; Pashler, 1994a, Pashler & Johnston, 1989). According to the locus

of cognitive slack logic, the interaction of stimulus intensity and SOA indicates that the effect of the variable is occurring prior to the stage comprising the processing bottleneck, and consequently the locus of the bottleneck must be somewhere after the stage at which the effects of stimulus intensity occur.

Response Selection Bottleneck

Several factors have been shown to affect the response selection stage of information processing. Varying the number of response alternatives between simple reaction time (SRT) and choice reaction time (CRT), Karlin and Kestenbaum (1968) demonstrated underadditive effects of number of stimulus-response alternatives with decreasing SOA. $Task_1$ was a two-alternative discrimination response in which subjects were to discriminate between two digits. $Task_2$ was an SRT or two-alternative discrimination response to auditory tones. Stimulus-onset asynchrony was varied and subjects responded as quickly as possible to the digit and then to the tone. The underadditive pattern suggests that the effect of manipulating the number of $Task_2$ response alternatives is occurring at a stage of processing prior to the PRP bottleneck. Varying the number of response alternatives most likely has an effect at response selection. If the locus of the PRP bottleneck is at a processing stage after response selection the most likely candidate for the bottleneck would be the stage of response execution.

The work of Karlin and Kestenbaum (1968) has not been consistently replicated however (Schubert, 1999; Van Selst & Jolicoeur, 1997). Using the same variable manipulation, Van Selst and Jolicoeur (Exp. 2, 1997) demonstrated additive effects of number of response alternatives with SOA. The additive pattern observed between SRT and two-alternative discrimination response suggests that the effect of manipulating the number of response alternatives is occurring at, or beyond, the PRP bottleneck, which in turn suggests that the bottleneck is located at, or prior to, response selection.

It should be noted, nevertheless, that the underlying response mechanism for an SRT is most likely different from that of a forced-choice response (CRT). The effect of varying the number of CRT response alternatives (e.g., two-alternative discrimination vs three-alternative

discrimination) is usually additive (Schubert, 1999; Van Selst & Jolicoeur, 1997), whereas the difference between a single response (SRT) and several possible responses (CRT) appears to be more variable. One possible difference is that a greater degree of perceptual processing is necessary for discrimination (CRT) compared to simple detection (SRT). If the effect is caused by an increase in perceptual processing, due to the nature of the task, some attenuation of the SRT and CRT RT_2 difference may be seen as SOA decreases but the difference between alternative CRT conditions (e.g., two-alternative discrimination vs. three-alternative discrimination) should remain constant across SOAs. Results consistent with this prediction have been reported (Van Selst & Jolicoeur, Exp. 1, 1997). An alternate explanation is that the underadditivity sometimes observed between SRT and CRT is a consequence of subjects anticipating S_2 in the SRT condition rather than responding to it, thereby producing anticipation errors (Van Selst & Jolicoeur, 1997). In the SRT condition a single response is repeated and can be anticipated prior to the presentation of the stimulus. Hence, anticipation error times are disproportionately fast and generally the time to respond decreases as the time between S_1 and S_2 increases. As a result the RT_2 mean for the SRT responses decreases by a greater amount at longer SOAs than at the shorter, producing an underadditive effect with decreasing SOA. Anticipation errors are unlikely in CRT because subjects must wait for the stimulus to appear in order to make the correct response. Schubert (1999) found that RT_2 response times for tasks employing single versus multiple response alternatives (SRT and CRT) were underadditive with decreasing SOA when the SRT task was performed prior to the CRT task but that the effect was less pronounced when the CRT task was performed first. Anticipation errors occurred only at the longest SOA (SRT = 17.66%; CRT = 0.3%), and were significantly higher in the SRT condition when that task had been performed first compared to when it was performed last (28.7%, 6.04%). In the CRT condition there was no difference in the frequency of anticipation errors as a function of order of condition (0.4%, 0.2%). Data such as these suggests that subjects are less likely to commit anticipation errors in an SRT condition if they have first been exposed to a task in which they were forced to wait before making their response (CRT). When the possibility of anticipation errors in an SRT task was reduced, by

including trials in which no S_2 was present, the underadditive effect of number of response alternatives with decreasing SOA was eliminated (Schubert, 1999).

Further evidence in support of a bottleneck at response selection comes from data demonstrating the effects of manipulating other variables associated with response selection. McCann and Johnston (1992) assessed the effects of overlapping tasks, when response difficulty was manipulated, by varying stimulus-response (S-R) compatibility. $Task_1$ was a two-alternative discrimination response in which subjects decided whether the frequency of an auditory tone was high or low (McCann & Johnston, Exp. 2, 1992). In $Task_2$ symbolic S-R compatibility was varied by presenting either arrows, pointing to the right or to the left and responded to using the right and left hands respectively, or the letters M and T, one to be responded to using the right hand and the other using the left hand. Spatial compatibility was also varied by presenting stimuli in either the left or right visual field so that on half the trials the stimulus position corresponded to the compatible response hand and on the remaining trials to the incompatible response hand. It was expected that response times would be faster to arrows than to letters, and faster when stimulus position and response hand corresponded spatially than when they did not. Of greatest interest was the effect of SOA on these manipulations. An additive effect with SOA was predicted for symbolic compatibility. Expectations for spatial S-R compatibility were less clear as evidence exists suggesting that this variable may be associated with stimulus identification rather than response selection (Hasbroucq & Guiard, 1991). If this is true, one would expect to observe a data pattern reflecting interference at a stage of processing prior to response selection. Thus, an underadditive effect of spatial compatibility with increasing SOA should be present. Indeed, McCann and Johnston did find additive effects with SOA for symbolic S-R compatibility and underadditive effects for spatial S-R compatibility suggesting the presence of an interference effect occurring at, or beyond, the bottleneck for the former variable and prior to the bottleneck for the latter.

The evidence accumulated thus far points to a locus of the PRP bottleneck at, or before, response selection. Research suggests that the bottleneck is not located at an early stage of processing but is more supportive of the response selection hypothesis.

Response Execution Bottleneck

Although evidence presented so far is indicative of a central processing bottleneck, possibly at response selection, some researchers have proposed a later PRP bottleneck, at response execution (Keele, 1973, McLeod, 1977). As discussed above, the underadditive data pattern sometimes revealed when response alternatives are manipulated (e.g., Karlin and Kestenbaum, 1968) has been provided by some researchers as support for a response execution bottleneck. The logic is that if underadditivity reflects interference happening prior to the PRP bottleneck, and if the SRT -- two-alternative discrimination response RT_2 difference denotes differences at response selection, then the bottleneck must be at response execution. The strongest evidence against a bottleneck at response execution is that the RT_2 difference between alternative CRT responses, such as two-alternative discrimination vs three-alternative discrimination should also reflect an equivalent decrease with decreasing SOA as the difference sometimes observed between SRT and CRT $Task_2$ response times. Karlin and Kestenbaum (1968) used only SRT and one CRT, a two-alternative discrimination, in their experimental design so it is impossible to determine any differences between alternative CRT responses using their data. Van Selst and Jolicoeur (1997) on the other hand did compare alternative CRT responses (two-alternative discrimination and three-alternative discrimination) and found additive effects with decreasing SOA, as did Schubert (1999), once again supporting a PRP response selection bottleneck.

Locus(i) of the Attentional Blink Effect

From the empirical evidence it appears then that the PRP bottleneck is located at a central stage of processing, possibly at a stage where responses are selected. We may be able

to use what we know about the locus of the PRP bottleneck to shed light on the location of the AB effect.

Manipulating the same variable in both PRP and AB experimental designs and finding an effect of the manipulation in each would allow us, first to determine the effect in relation to the PRP bottleneck by using the locus of cognitive slack logic, and then, perhaps, to ascertain the location of the AB effect, also in relation to the PRP bottleneck.

With this goal in mind, relative signal probability was manipulated in PRP and AB experimental designs. In the PRP paradigm, the signal probability manipulation was associated with $Task_2$ whereas in the AB design it was associated with $Task_1$. There is evidence to suggest that the effect of varying signal probability is additive with SOA (Luck 1998, Van Selst & Jolicoeur, 1997). According to the locus of cognitive slack logic, additive effects of relative signal probability and SOA are consistent with the hypothesis that the effect of probability is taking place at, or after, the bottleneck in the PRP. Likewise, the effect of this variable on the size of the AB is interpretable in as far as determining the locus of the probability effect in relation to the locus of the AB effect. A variable that changes the size of the AB effect must have its effect at, or before, the processing stages(s) that cause(s) the AB effect. It cannot have its effect after the latest locus contributing to the AB effect. Therefore, if signal probability affects the magnitude of the AB. this would suggest that the locus of the probability effect is at the locus of the AB effect or before it.

By the logic outlined above, the effects of probability in experiments designed to exhibit PRP and AB effects will enable us to establish more precisely the locus of the AB effect.

Locus(i) of Signal Probability Effects

Studies manipulating signal probability typically show that stimuli presented more frequently lead to faster responses than those presented less frequently (Bertelson, 1966; LaBerge & Tweedy, 1964; Miller & Hardzinski, 1981; Theios & Walter, 1974). Various views have been proposed as to the fundamental mechanism underlying this finding. Some

researchers point to the influence of perceptual factors (Bertelson & Tisseyre, 1966) while others attribute the effect to response bias (Sanders, 1970). An intrinsic difficulty with investigations of the effect of relative signal probability is that stimulus frequency is confounded with response frequency, as increases in signal frequency are coupled with comparable increases in associated responses. Variations in relative stimulus frequency may be connected with stimulus identification, frequency effects reflecting adjustments in anticipation of the presentation of a stimulus (Bertelson & Tisseyre, 1966). Such a mechanism would implicate early stimulus encoding as the locus of stimulus probability. Variations in response frequency may reflect the expectation of a particular response, thereby affecting a response selection stage of processing (Sanders, 1970). If the effect of stimulus probability is a function of response factors, then the locus(i) could be at any of the later stages, subsequent to encoding.

Several researchers have attempted to separate the contribution of perceptual and response factors in relative probability effects. Bertelson and Tisseyre (1966) investigated the issue by using a many : one stimulus-response arrangement while varying probability of stimuli and responses. Four stimuli (uppercase letters) were used, two corresponding to one response while the remaining two were associated with an alternate response. Stimulus probability was varied for each member of one stimulus pair so that one was of high probability (.55) and the other low (.15). Both members of the other pair were assigned probability values of (.15). Responses therefore had a low/high (.30 : .70) probability associated with them as did the stimuli (.55 : .15), with some decoupling between the two. If the probability effect is associated with the response, separate from the stimulus, reaction times should be fastest for high frequency responses, regardless of stimulus frequency. On the other hand, any difference between conditions where the stimulus frequency was varied but response frequency was held constant would suggest perceptual bias. Bertelson and Tisseyre found no evidence to support an effect of response probability. Relative frequency of the response did not affect reaction time whereas relative frequency of the stimulus did. They concluded that the

effect of probability is not due to response factors but more likely has an effect at an early stage of information processing such as stimulus identification.

In contrast, other researchers claim that the stimulus probability effect is a function of response frequency rather than early stimulus processing. According to Sternberg's additive factors logic (1967, 1969) variables that affect different stages of processing should exhibit additive effects whereas an interaction between variables should be evident when variables affect a common stage of processing. Sanders (Exp. 1, 1970), using two levels of stimulus frequency (low/high), found an effect of probability that interacted with the amount of motor preparation required to elicit a vocal response. In one condition (different-phoneme) response information was contained within the first phoneme of a verbal response since each stimulus-response pairing began with a different phoneme and thus each required a change in preparation state. In another condition (same-phoneme) all members of a stimulus group began with the same-phoneme, thereby equating preparation for each response. Overall, same initial phoneme responses were significantly faster than different but an effect of probability was only evident for responses to stimuli belonging to the different-phoneme response group. Sanders (1970) proposed that the effect of probability is, at least in part, due to an effect of motor preparation to the most frequent response. Response time is longer when the required response does not correspond to the expected response. If perceptual processes had been affected by probability in this study, an effect of probability should have been observed for the same-phoneme group as well. However, response times were relatively equal in this group for both levels of stimulus probability.

To determine whether or not the effect of motor preparation is independent, or works in conjunction with, response selection, Sanders introduced an S-R compatibility manipulation (Exp. 2, 1970). S-R compatibility is a variable believed to affect response selection. The same first phoneme response conditions (same-phoneme, different-phoneme) were used in this experiment but within each was a condition in which the response to the stimulus was not readily associated. For example, the letter A in the S-R compatible - different phoneme condition was responded to as A, while in the S-R incompatible condition the correct response

was E. S-R compatible and S-R incompatible conditions were created in the same-phoneme group as well as in the different-phoneme group. If motor preparation and response selection are common to one stage of processing, an interaction of S-R compatibility and type of response (same-phoneme, different-phoneme) should be observed. A significant difference in reaction time as a function of stimulus probability was found across all conditions. More frequent stimuli were responded to faster than less frequent stimuli. There was an effect of S-R compatibility, incompatible S-R pairings yielding slower responses than compatible pairing and, in the S-R incompatible condition the effect of stimulus probability was stronger in the different-phoneme condition than in the same-phoneme. As found in the first experiment, the effect of probability was only evident for the different-phoneme condition. The effects of response preparation (same-phoneme, different-phoneme) and S-R compatibility were found to be additive, suggesting that motor preparation and response selection reflect distinct stages of information processing and that each is affected by relative signal probability.

Like Bertelson and Tisseyre (1966), Hawkins, MacKay, Holley, Friedin, and Cohen (1973) investigated whether the effect of stimulus probability is due to perceptual or response bias by using a many : one S-R mapping arrangement. The stimulus set in their study consisted of 8 letters which were grouped into pairs so that each stimulus pair was assigned to one response. Probability of presentation of the stimuli was varied between members of each letter pair so that probability was high (.50) for one member of one pair and low (.10) for its partner, low (.10) for both members of another pair, and lowest (.05) for the remaining four stimuli. The responses of interest were those associated with .50 and .10 stimulus probabilities. The four stimuli associated with the lowest stimulus probability (.05) were distractors that were included to increase task difficulty. S-R compatibility was also varied so that, for some subjects, the S-R relationship was highly compatible while for others S-R compatibility was low. This was accomplished by having subjects in the S-R compatible condition report the commonly used name of a stimulus letter (e.g., D = D). Subjects in the S-R incompatible condition were to use another preassigned letter name to report the stimulus (e.g., D = G). The reaction time for vocalized responses indicated significant probability effects for stimulus as

well as response factors in the low compatibility condition, but neither of these effects was significant in the high S-R compatibility condition. Furthermore, in this study, stimulus and response frequency effects both decreased in magnitude as the number of trials increased implying that, with practice, subjects change the way they process stimuli . The finding of an effect of response frequency is in contrast to the work of Bertelson and Tisseyre (1966) who found no evidence for response factors. The fact that the effects decrease with practice, and are more likely to be found when S-R compatibility is relatively low, may provide an explanation for some of the inconsistencies coming from studies investigating the contribution of stimulus and response factors in signal probability. Bertelson and Tisseyre (1966), for example, used a relatively high S-R compatibility with their stimuli of two letters paired with one response and two paired with another response, and did not look for any change in the contribution of stimulus and response factors as a function of practice.

LaBerge and Tweedy (1964) also found effects of stimulus and response frequency when probability was varied in a design where one stimulus (coloured shape) was coupled with one response hand and two other coloured stimuli were associated with the other response hand. Only the two stimuli associated with the same response hand were involved in the probability manipulation. Since these stimuli were connected with the same response, response probability was controlled and any difference in response time could be attributed to the sole effect of stimulus frequency. The probability of the one stimulus (one stimulus: one response hand) was consistent across trials and was intermediate (.40) between the probability values assigned to the other two stimuli (.10 : .50 or .50 : .10). Subjects were instructed to press the appropriate response key as quickly as they could when the stimulus was presented. Between the stimuli connected with the same response hand, responses were faster to the more frequently presented stimulus supporting the theory that the effect of stimulus probability is a result of perceptual rather than response bias.

However, the researchers did note that, inconsistent with a theory of pure perceptual bias, the mean reaction times to the stimulus consistently presented at a frequency rate of .40, and linked with an individual response, did not lie in between the other two mean response

times (relative probability .10 : .50). In fact mean RT for this condition was slower than the response times to either of the other two stimuli. This observation can be accommodated if the probability ratio of the two stimuli associated with the same response hand are summed (.60). Thus, under these conditions the slower mean response time can be explained by including a response bias component in the probability equation. The more frequent response (.60) producing faster reaction times than the less frequent response (.40).

In a follow-up experiment LaBerge, Legrand, and Hobbie (1969), using a similar paradigm, investigated further whether it was possible that both perceptual and response frequency could contribute to the observed effect of signal probability. Again, three coloured stimuli were used, one associated with one response hand and the other two with the alternate response hand. In contrast to the previous experiment, the relative probability of one of the two stimuli associated with one response was different from the probability level assigned to the other stimulus associated with the same response. For example, in one condition, one stimulus paired with one response was assigned a value of 10 while the other stimulus, paired with the same response, was given a value of 40. The stimulus coupled with the other response was also given a value of 10. This condition was termed the 5 : 1 condition because the combination of the probability values associated with the stimuli paired with the same response was 5 times (40 + 10) that of the stimulus assigned to the other response (10). In a 9 : 1 condition, the relative probability values were 6 for the 1 : 1 S-R pairing, 6 for one of the members of the 2 : 1 S-R pairing, and 48 for the other. Perceptual probability was measured by subtracting the mean RT obtained for the high probability stimulus associated with one response from the low probability stimulus of the same response. A measurement of response probability was attained by subtracting the mean RT obtained for equal probability conditions of both response hands.

Both perceptual and response frequency were found to be factors embodied in the signal probability effect in this study. Nevertheless, the effect of response probability was greater than perceptual probability and the authors proposed that perhaps it is not possible for the effect of relative signal probability to exert an equal influence across both dimensions in

certain circumstances and that, under these conditions, response bias is more likely to dominate.

An alternate way of investigating the locus of the stimulus probability effect is to utilize Sternberg's (1967, 1969) additive factors logic and manipulate a variable associated with a specific stage of processing, observing whether there is a differential effect of that manipulation as signal probability is varied. As previously mentioned, according to the logic of additive factors, if variables affect a common stage of processing, an interaction between those variables should be evident, whereas the effects should be additive if the variables affect different stages of processing. Using this logic it is possible to determine whether signal probability affects the same stage of processing affected by some other variable. For example, varying stimulus contrast most likely has an effect at an early, encoding stage of processing. If relative signal probability has an effect at the same stage, an interaction of signal probability with stimulus contrast should be evident. On the other hand, if stimulus probability affects a stage of processing other than the one affected by varying stimulus contrast, additive effects should be observed.

Miller and Pachella (1973) varied stimulus quality and signal probability in a memory scanning choice reaction time (CRT) experiment in which subjects were to make a positive response to stimuli (digits) that were members of a previously memorized set of items and a negative response to other stimuli. Signal probability of items within both sets was varied and an interaction of stimulus quality and signal probability was found, indicating that the effect of probability was occurring at a stage common to the locus of the stimulus quality effect. Moreover, there was no interaction of probability and response type (positive/negative) implying that, in this experiment, probability did not affect a stage of processing associated with the response to a stimulus, such as response selection or execution. Additional support for the encoding stage as the locus of the signal probability effect was provided when an interaction of stimulus probability and quality was found when subjects were simply to name the stimulus.

It is very likely that the output of stimulus information, processed during the encoding stage, is in some form other than a basic physical representation, possibly in the form of a name code (Wattenbarger, cited in Nickerson, 1971). Wattenbarger designed an experiment to determine whether stimulus comparisons were based on physical identity or the names of items. Subjects were to decide whether a probe character, which was a letter, had appeared within a target set of letters. There were four conditions: - a) a control condition in which the target set and the probe were lower case letters; b) a control condition in which targets and probe were uppercase letters; c) a name identity condition where targets were presented in both upper and lowercase and the subject's task was to match the probe, which could be in either case, to a letter of the same name; d) a physical identity condition in which various letters appeared in either upper or lower case and the subject's task was to match the probe to a letter that had the same physical representation. Target letters were presented sequentially, one at a time, at a rate of one every two seconds, except in condition c) where they appeared two at a time (upper and lower case). The probe appeared after the last target letter. Subjects' task was to judge whether they had seen the probe letter in the target set using the appropriate criteria, depending on the condition, as the basis for their decision. Wattenbarger predicted that, if comparison is based on the physical image of the stimulus, the slope function relating reaction time to target set size in the physical identity condition should be the same as that obtained in the two control conditions, and the slope for the name condition should be approximately twice that of the others, owing to the fact that two target letters were presented in the name identity condition and therefore comparison time would take longer in that condition. If comparison is based on the name of the stimulus, the slope function of the name identity condition should be equal to control conditions, while the slope of the physical identity condition should be greater. Wattenbarger reasoned that the slope in the physical identity condition would be greater in this case because, if a name was used to make the comparison it would have to incorporate case information (e.g., K = 'upper Kay') and would therefore require more processing time than comparisons involved in the other conditions.

Results from this study supported the hypothesis that stimulus comparisons are based on name identity. The slope of the function for the physical identity condition was almost twice as great as that of the other conditions which were all very similar. This result however provides no evidence against the possibility that subjects base their decisions on names sometimes and on physical features at other times, perhaps using both comparison procedures and eventually relying on whichever one results in the quickest response. In the control and the name identity conditions in this experiment, if names of stimuli were the same, the physical images would also be the same, meaning that if subjects based their decision on a physical match in these conditions their response would not be incorrect. In contrast, in the physical identity condition, stimuli could have the same names but differ physically. Consequently, subjects were forced to be careful in this condition not to make a response based on name only.

Lending more support to the naming hypothesis was the finding that, in the physical identity condition where the target set contained upper and lower case letters and task was to match the probe to a target that was physically the same, time to respond was slower on trials where a target item had the same name but differed in case. If the comparison process is based on physical features there is no reason for reaction times to be longer when the names of stimuli are the same but physical representations differ. However, it is frequently found that reaction times are faster when stimuli share the same name as well as the same physical appearance compared to when only the name of the stimulus is the same (as in a name identity task when two letters have the same name but differ in case) (Nickerson, 1971). This fact again provides support for the possibility that physical feature information and name information can somehow be combined and used in the comparison process.

Wattenbarger (cited in Nickerson, 1971) concluded that, in the studies he conducted, visual representations of the target and probe items were transformed either to the actual name of the stimulus or to some other, more abstract state. He proposed that the form the stimulus takes at this stage is adequate for identification and hence is produced prior to the comparison process itself.

Theios (1975) proposed that there are at least two substages that make up the encoding stage. An initial preprocessing stage and an identification stage in which a name or abstract code for a stimulus is generated whenever the stimulus material allows.

Consistent with this theory is the observation of an interaction of stimulus probability and stimulus quality for namable stimuli (digits) in a memory scanning task, but not for nonsense symbols (a solid shape presented at various orientations) (Miller & Pachella, 1976), though main effects of stimulus quality and probability were observed for the nonsense stimuli. The nonsense symbols in this experiment did not have preassigned names attached to them and, when they were modified so that their appearance was more like that of digits (line figures rather than solid), perhaps making them easier to name, a small interactive effect (approaching significance) between stimulus quality and stimulus probability was found. These results suggest that an interaction between stimulus quality and stimulus probability only occurs when the stimuli are namable. If, during an early stage of processing certain kinds of stimulus material allow for the conversion of a physical representation to some form of code, this stage may be the locus of stimulus probability, as well as stimulus quality effects. Moreover, it appears that, if stimuli are not easily namable, an abstract code, perhaps created for a specific stimulus in an experiment, will suffice.

Pachella and Miller (Exp. 1, 1976) investigated further the nature of the effect of stimulus probability on stimulus encoding. Upper and lowercase letters were used as stimuli in a name matching task. Two letters were presented together and any pair of stimuli with the same name were to be classified as 'same' while those with different names were 'different.' Four letters were used, two of them were assigned to a high probability group (.82) and two to a low probability group (.18). High probability letters were never matched with low probability letters. The pairs were presented either as physically identical (e.g., A-A = same), same letter differing only in case (e.g., A-a = same), or different letters (e.g., A-G = different). The interaction of probability and trial type (physically identical, same letter/different case, and different letter) was significant for error data ($p < .05$) but did not quite attain statistical significance for reaction time, The effect of stimulus probability appeared to be evident for the

different case and different letter conditions but not for the physically identical condition (same letter/same case). The authors proposed that, in matching letters that were physically the same, the generation of a name code would likely have been to no advantage in performing this task. The effect of probability was not found under these conditions thereby providing further support for the locus of the probability effect at a stage of processing where some form of stimulus code is generated.

In a similar letter matching experiment, a stimulus quality variable (degradation) was introduced (Pachella & Miller, Exp. 2, 1976). A main effect of stimulus quality was found as well as effects of probability, trial type, and an interaction of probability and trial type. The effect of probability was again not evident in the physical match condition. Furthermore, an interaction of quality and probability was not detected in any condition. The authors proposed that, in this experiment, the effects of degradation had been removed from the stimulus representation early on, prior to the stage in which a name is derived. Matching tasks, like the ones used in this experiment, undoubtedly require comparison of the physical attributes of the stimulus pair and it is possible that, if this comparison process occurs prior to name code generation, the effects of stimulus degradation are removed at the physical comparison stage, before processing involved in the production of a name code begins. This theory predicts additive effects of stimulus quality and probability at each level of processing (i.e., trial type) which is what was observed here.

When instructions to the subject were modified so that letter pairs that differed in case were now to be classified on the basis of physical dissimilarity and judged as different (A-a = different), the effect of probability failed to reach a significant level for any of the three trial types (physically identical, same letter differing only in case, or different letters) (Pachella & Miller, Exp. 3, 1976). This lack of an effect of probability provides further support for the theory that signal probability has at least part of its effect at a stage of processing where a name code is generated. Once the matching tasks in this study were modified so that comparisons were based on the physical characteristics of the stimuli, the name of the stimulus may have become irrelevant for completion of the task.

Because no effect of probability was observed when the task was reduced to one based on comparing physical representations, and no effect of probability was evident in the physical match condition in their initial experiment, Pachella and Miller (1976) proposed that the differential effects of probability are due to whether or not a name code is used to perform the task. Besner (1977), however, provided an alternative explanation. Using the same stimuli (upper and lower case letters), and trials types (physically identical, same letter/different case, different letter), as Pachella and Miller (Exp. 1, 1976), Besner found that physical match trials were performed faster than same name/different case trials, but the effects of probability were evident for both conditions. The methodological modification in this experiment was the introduction of a delay of 500 ms between the stimuli to be matched. In this experiment then, probability had an effect on the comparison of physically identical stimuli suggesting that it is not the activation of a name code, per se, that is affected by probability. Besner (1977) proposed instead that the significant factor in probability effects is the interaction between memory and perception.

According to the name code hypothesis it may be possible to transfer the effects of probability from one stimulus to another if both stimuli have the same name, or abstract code. Miller first investigated the issue of probability transfer by attempting to ascertain whether probability is having an effect because of increased activation of separate features of the stimulus or whether the effect is the outcome of increased activation of the specific form of the stimulus (Miller, 1979). If the effect is at the feature detection level, then the probability manipulation might be transferable to letters that are comprised of similar features. On the other hand, no transfer would be expected if probability is having its effect on the specific form of the stimulus.

In Miller's study (Exp. 1, 1979), the stimuli used were two sets of letters pairs, each member of a pair being physically similar to its partner (e.g., I : T and K : R). The probability of presentation of each letter was varied so that one letter of one pair had a high probability of presentation (e.g., I = .46), while one letter of the other pair had a low probability (e.g., K = .04). The remaining two letters were presented equally often (e.g., T = .25, R = .25). The

high probability letter (e.g., I) and the low probability letter (e.g., K), although members of different stimulus pairs, were assigned to one response. The other two letters (e.g., T and R), with probabilities of .25, were assigned to another response. Stimulus quality was also varied so that in one block of trials, subjects saw the stimuli under normal viewing conditions; in another block, the contrast between the stimulus and its background was reduced; and in a third block, a random dot pattern array was displayed after the stimulus was presented. Probability effects were expected on the letters to which a direct low/high probability manipulation (direct condition) had been applied (e.g. K = low; I = high). Transfer of these effects to visually similar letters (T and R), that is letters that shared features common to the letters in the direct condition, was predicted if probability is having an effect at the feature detection level. Otherwise no transfer of the effect was expected. Thus, if the effect of probability is having an effect at the feature detection level the effect of probability expected to be observed for the high probability (.48) letter (e.g., I) should also be observed for the physically similar letter (T), even though the probability of that letter is considerably lower (.25). Likewise, the effect of low stimulus frequency expected to be observed for the low probability (0.4) letter (e.g., K) should be transferred to the physically similar letter (R), even though the probability of that letter is higher (.25).

Although effects of probability were evident in the direct condition, Miller found no transfer of probability to letters that were physically similar or, in another experiment (Miller, Exp. 3, 1979), to stimuli that had the same name but were physically different (e.g., *Y* and Y). These results suggest that the effect of probability is not at the feature detection level of stimulus encoding. For physically similar stimuli (e.g., I and T) an interaction of stimulus quality and probability was observed for the direct condition, but this interaction was not found for the stimuli that had the same name and were physically different (e.g., *Y* and Y). In the former case a name code may have been necessary in order to perform the task of identifying the letters and thus an interaction of stimulus quality and probability was obtained, whereas in the latter case use of a name code may have been of no benefit to performing the task since both items had the same name (Miller, 1979).

Miller and Hardzinski (Exp. 1, 1981) obtained an interaction of stimulus quality and probability in a direct probability manipulation condition in a memory scanning task (positive and negative sets). Letters contained within the memory set were upper and lower case but the probability manipulation was associated with only one case, either upper or lower. The question of interest was whether the effects of probability would transfer from one case to the other. An interaction of stimulus quality and probability was found when probability was directly manipulated. The observation of an interaction of stimulus quality and probability suggests once again that a name code was necessary to complete this task, and that the effects of stimulus degradation and probability interact under such conditions. In the probability transfer condition, a main effect of stimulus quality was found but no effect of probability. It would seem that, in this memory search task, even though the upper and lower case letters had the same name, use of a name code would be necessary in order to confirm whether or not the stimulus was a member of the positive set. Yet in this experiment, as in others (Miller, 1979) where the use of a name code would seemingly predict transfer of probability effects to stimuli of the same name (different case), no such transfer was evident. When the experimental design was modified somewhat, so that only one case size was used and direct and indirect conditions were confined to blocks of trials, rather than between trials, a small effect of probability was obtained in the transfer condition, though no interaction of quality and probability (Exp. 3, 1981). In this modified version of the experiment the same case (upper or lower) was used throughout. In the first block of trials different values of relative probability were assigned to each stimulus. In the following block, using the same stimuli (i.e., same letters/same case), stimulus frequency was equiprobable and any transfer of the effect of probability from the first to the second block of trials was assessed. Although a small effect of probability was seen here in the transfer condition, no such effect was observed in an experiment that differed only in that the stimuli presented in the second block of trials were of the alternate case to those in the first block (Exp. 2, 1981). Thus, transfer of probability effects are possible using this experimental procedure but they appear to be transient and are possibly difficult to detect (Miller & Hardzinski, 1981).

Using a stimulus matching paradigm similar to Miller's other experiments reported here (Miller, Exps 1 & 3, 1979) transfer of probability effects were found for non-alphanumeric stimuli that were visually similar and highly classifiable (Miller, Exp. 4, 1979). The four stimuli used in this experiment were an X with a line above or below it and a diamond shape with a line above or below it. An interaction of stimulus quality and stimulus probability was evident in both the direct and indirect probability manipulation conditions. Contrary to the findings of Miller and Pachella (1976), who found no interaction of stimulus quality and probability when using nonsense stimuli, these data imply that even though the stimuli used in this experiment were not designated some preassigned name, individuals were able to develop some sort of abstract code for them and thus a quality by probability effect was evident. These findings also show that the effects of a direct manipulation of probability are transferable to specific stimuli. Miller (1979) suggests that the difference between these stimuli and those used in his previous experiments where no transfer of probability was found, as well as those used by Miller and Pachella (1976) where no interaction of stimulus quality and probability was found, is that the stimuli used here are composed of a fixed set of binary features that are shared amongst them (i.e., X, diamond shape, top bar, bottom bar). An abstract code based on a combination of these features could be produced for the stimuli, which in turn would mean that the codes share common dimensions. It is because of the shared characteristics between the abstract codes that a transfer of the probability effect might have occurred. Using stimuli that are not made up of combinations of binary features likely means that the stimuli are coded on an individual basis, having no common coding dimension. In a subsequent experiment, when the stimuli were modified so that common binary features could not be used as the basis for coding, no transfer of the probability effect was observed (Miller, Exp. 6, 1979). In addition, though there was an effect of probability as well as an effect of stimulus quality in the direct probability manipulation condition there was, again, no interaction between these variables. Miller (1979) proposed that the interaction of stimulus quality and probability depends to a large degree on the conceptual or linguistic relationships between stimuli and consequently it is found using some stimuli but not others.

Subsequently Miller and Hardzinski (1981), in a memory scanning task, tested whether probability effects transferred when letter stimuli were varied on the basis of case, font, size, and visual dissimilarity (letters had the different names but differed visually from each other by one feature, e.g., O and Q). In this study, although the effect of probability was highly significant, transfer was only observed to letters that were reduced in size. There was no transfer of probability effects to letters that differed in case, font, or to different-name letters that had one different feature.

On the one hand these findings suggest that a process tied to the analysis of the physical features of stimuli is affected by signal probability. On the other hand, Miller (Exp. 4, 1979) found no evidence of transfer to visually similar letters that had a different name thereby suggesting that feature detection is not the only factor involved in the probability effect. Coupled with the findings reported by Miller and Pachella (1976) and Pachella and Miller (1976), the results suggest that the effect of probability arises in the process of producing a code associated with the stimulus and that transfer of probability effects is specific to the way this code is activated (Miller & Hardzinski, 1981).

Miller and Hardzinski (1981) proposed that stimuli are initially analyzed in terms of physical features which in turn activates a name or abstract code associated with the stimulus or with a group, possibly a category, of stimuli. Signal probability effects arise because the frequency at which a stimulus is presented affects the threshold required for the activation of the code. The direct manipulation of signal probability causes a lower threshold of activation for high probability stimuli, leading to faster responses. Stimuli presented less frequently have a higher activation threshold. When a code that is useful to performing a task is shared between stimuli, the effect of signal probability can transfer from one stimulus to another (Miller, Exp. 5, 1979; Miller & Hardzinski, Exp. 4, 1981). However, as the studies reported here suggest, transfer is not always predictable, and much remains to be understood regarding the mechanism behind the effects of relative stimulus probability, and the transfer of these effects. Though we might expect transfer of probability effects under conditions where a stimulus code is generated, caution should be taken with respect to the simplicity of this assumption. The

relationship between factors like task complexity, the type of task (e.g., memory scanning vs matching), and the specific form of the code (e.g., name vs abstract), must be considered as an important element that contributes toward the specific effects of stimulus probability. For example, the use of a name code that is highly salient to the subject may be affected by probability in a different way than an abstract code that has been generated for the sole purpose of performing an experimental task. One possibility is that the former, because of its high degree of salience, may be composed of independent stimulus and response codes, whereas the latter, having been specifically generated for the task at hand, might combine stimulus and response information into one code. The consequence of this difference might serve to explain why an interaction between stimulus quality and probability is sometimes, but not always, found. The stage at which an abstract code, that includes stimulus and response information, is generated may be later in the information processing sequence, subsequent to the stage affected by stimulus quality. Hence, additive effects of stimulus quality and stimulus probability would be observed. In contrast, the stage at which a salient name code, independent of response information, is produced, may be common to the locus of the effects of stimulus quality, thus producing an interaction between these variables.

Miller (1979) provided an account of the mechanism underlying the interaction of stimulus quality and stimulus probability that is sometimes observed under conditions where a meaningful code is used to select a response. The interaction is based on the activation threshold associated with the probability of a stimulus. Stimulus codes are activated more easily under normal viewing conditions than under degraded conditions simply because sensory information is of higher quality under normal conditions. However, high probability stimuli, having a lower threshold, are less affected by stimulus degradation than are low probability stimuli and thus an interaction of stimulus quality and probability is observed. As the probability of a stimulus increases, the effects of degradation decrease (Miller, 1979; Miller & Hardzinski, 1981).

As already mentioned, stimulus quality and probability do not always interact and this account of the effect of signal probability does not accommodate those occasions where highly

significant probability effects were found but stimulus quality and probability were additive (Miller, Exps. 2 & 6, 1979; Miller & Pachella, Exp. 2, 1976; Pachella & Miller, Exps. 2 & 3, 1976). If the interaction of stimulus quality and probability is dependent on recoding the stimulus using a particular type of abstract code, then, when stimulus quality and probability are additive, one might presume the absence of this type of coding. When the coding stage is absent, the effect of stimulus degradation may have been removed from the stimulus prior to the effect of signal probability, and consequently the locus of signal probability must come after the encoding stage. Certainly, as previously mentioned, there could be more than one locus of probability effect. If subjects do not use abstract codes to perform the task, then they may be able to create direct visuo-motor links and by-pass the intervening coding stage (as suggested by Miller & Hardzinski, 1981). In this case the effect of probability would not be due to increased activation of an associated code but may be the result of more frequent activation of the visuo-motor link (Miller, 1979). If the locus of signal probability is subsequent to the encoding stage, it is possible in situations when no code is used, or perhaps when the code contains paired stimulus and response information, that it is located at a stage of response selection as suggested by Sanders (1970).

The literature on signal probability is complex and contains a number of apparent empirical discrepancies. As it is still not entirely clear at what processing stage, or stages, signal probability has its effect, more work, using a different approach would be beneficial. Experiments 3 and 4 in this thesis use the PRP experimental paradigm and the locus of cognitive slack logic to determine the locus of signal probability effects in relation to the PRP bottleneck. We intend to use the results from these experiments to help discover the locus of the AB effect. Therefore, given that Miller (1979; Miller & Hardzinski, 1981) found evidence for a high degree of stimulus specificity in effects of signal probability, it was important to perform PRP experiments the results of which would be relevant for a pair of companion AB experiments.

Though signal probability has not been used in an identical manner to the experiments reported here, it has been employed as an independent variable in previous PRP studies (Luck,

1999; Van Selst & Jolicoeur, 1997). In these earlier studies, the effects of signal probability and SOA were additive, suggesting a locus at, or beyond, the PRP bottleneck

In the following experiments the manipulation of signal probability was applied to $Task_2$ in the PRP paradigm and to $Task_1$ in the AB design. In the AB experiments, the question of interest is whether the manipulation of signal probability modulates the AB effect. In addition, we wish to know whether the effect is dependent on having a speeded response in $Task_1$. The results from the PRP experiments will be interpreted using the locus of cognitive slack logic to evaluate where the effects of signal probability are occurring in relation to the PRP bottleneck.

Experiment 1

The initial task was to determine the effects, if any, of relative signal probability on the AB. With this in mind an AB experiment was designed in which relative signal probability was implemented as a variable associated with $Task_1$.

Method

Subjects

Seventy-eight undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All subjects had normal or corrected-to-normal vision.

Stimuli

All experimental sessions were run on a SVGA colour computer screen controlled by a 384, 486, or 586 CPU. Stimuli were uppercase letters presented in Rapid Serial Visual Presentation (RSVP) in the center of the screen for a duration of 100 ms with no blank interstimulus interval (ISI). All the letters were presented in white on a black background, except the target stimulus (T_1), which was presented in red. Background stream items were selected at random without replacement from the letters of the alphabet excluding H, O, S, X, or Y. There were 6 - 9 letters (this number was selected randomly at run time) presented prior to T_1, and 9 - 12 letters (also selected randomly at run time) following T_1. The probe stimulus (T_2) occurred in any one of the 8 T_1-T_2 positions following T_1 (henceforth referred to as T_1-T_2 lag 1-8). This procedure insured that there were always 1 - 4 letters presented after T_2, thus providing the T_2 masking conditions necessary to observe an AB effect (Giesbrecht and Di Lollo, 1999; Jolicoeur, 1999a).

Letters subtended about 1^0 of visual angle. The white letters had an approximate luminance of 25 cd/m^2 on a black background of less than 1 cd/m^2. Red stimuli had an approximate luminance of 26 cd/m^2. As the experiments were conducted on several computers, average values are reported here. Note that the luminance of the white and red letter was similar, thus reducing the possibility that T_1 would mask T_2 more than distractor stimuli.

Procedure

On two-thirds of the trials a red target letter (T_1) appeared within the stream of white letters (T_1-present trials). On the remaining trials no T_1 letter appeared (T_1-absent trials). T_1 could be one of 3 letters, either an H, an O, or an S. The relative probability of the letters was varied and had a ratio of 1: 4: 9. Thus, one letter was presented 9 times in 14 T_1-present trials, another was presented 4 times, and another 1 time. The probability of each letter was counterbalanced across every 6 subjects so that each possible assignment of each probability level to each letter was used once. Thus, each letter occurred at each probability level equally often and the probability manipulation was not confounded with letters. On all trials, a probe stimulus (T_2) appeared in the stream. On half the trials T_2 was an X and on the remainder a Y.

Reaction time to T_1 was recorded, and responses were made by pressing the assigned key on the keyboard. The following keys were assigned as T_1 response keys: ,< for H; .> for O; and /? for S. In the T_1-absent condition, subjects were instructed to wait until the end of the stream of letters and then to press the space bar. This response was not speeded.

At the end of the letter stream, a prompt for a response to T_2 appeared ("Did you see an X or a Y?"). The X key was used for an X response and the C key for a Y response. Consequently, subjects used their left hand to respond to T_2 and their right hand to make a response to T_1.

Following each trial, feedback was given in the form of plus or minus signs indicating to the subject whether the T_1 and T_2 responses made on the previous trial had been correct or incorrect. These feedback signs appeared adjacent to each other and were presented in the centre of the screen. Thus, they also served as a fixation point for the next trial. The left sign was indicative of performance on T_1, and the right of performance on T_2. Each trial was initiated by pressing the space bar, which caused the fixation symbols to disappear, and the next RSVP stream to begin 750 ms later.

At the beginning of the experimental session, subjects were required to read instructions regarding the experimental procedure. The instructions were then repeated verbally by the experimenter. The experiment began with 2 blocks of 32 practice trials each, followed

by 672 experimental trials divided equally into 8 blocks of trials. A complete crossing of the factors, T_1 probability condition (T_1-present 1 : 4 : 9, T_1-absent 7), T_2 letter (X or Y), and T_1-T_2 lag (1 - 8), consisted of 336 trials, or 4 blocks. In each complete crossing of all of the factors the T_1 letter assigned to the probability 1 condition was presented 16 times, so that each of the T_2 letters (X and Y) appeared once at every T_1-T_2 lag. The letter assigned to the probability 4 condition appeared 64 times, each T_2 letter appearing 4 times at every T_1-T_2 lag. And the letter assigned to the probability 9 condition appeared on 144 trials, so that each of the T_2 letters was presented 9 times at every T_1-T_2 lag. Thus T_1 appeared on 224 trials, according to the probability level assigned to the T_1 letter, and, in conjunction, each T_2 letter was presented at every T_1-T_2 lag. The T_1-absent condition (probability 7) was tested on 112 trials. On these trials each T_2 letter appeared 7 times at each T_1-T_2 lag. This complete crossing was presented twice throughout the experiment, making a total of 672 trials.

The first task on each trial was to identify T_1 and respond as quickly as possible by pressing the appropriate response key. On T_1-absent trials subjects were to wait until the end of the stream and then press the space bar (not speeded). Following the letter stream, or after the space bar was pressed on T_1-absent trials, a prompt for a response to T_2 appeared ("Did you see an X or a Y?"). Subjects were informed at the beginning of the experimental session that this response was not speeded. Subjects were debriefed at the conclusion of the experiment.

Results

Individual subject data was checked for $Task_2$ accuracy to ensure that all subjects could perform the task of identifying T_2 when T_1 was absent. Thirty individuals who failed to perform at or above 69% accuracy on $Task_2$, on trials where T_1 was correctly judged as absent, were eliminated from further analyses, leaving 48 subjects in the data set.

Analysis of $Task_1$ accuracy and response times

Cell means were computed for $Task_1$ response times and accuracy rates for each subject, probability level (1 : 4 : 9), and T_1-T_2 lag (1 - 8). Each cell for each subject was

screened for outliers which removed 2.5% of the correct responses (see Van Selst & Jolicoeur, 1994b). The cell means were entered in to an ANOVA which revealed main effects of probability for $Task_1$ reaction time [F (2, 94) = 118.7, p < .0001, MS_e = 16102.4] and accuracy [F (2, 94) = 23.9, p < .0001, MS_e = .031]. Subjects were fastest, with highest accuracy, on trials on which the probability of the T_1 letter was 9 and were slowest, with the lowest accuracy rate, on the probability 1 trials. On trials where the probability of the T_1 letter was 4, reaction time and accuracy were intermediate between the probability 1 and 9 values. (See Appendix A for $Task_1$ reaction times and accuracy rates for each probability at each T_1-T_2 lag.)

Analysis of $Task_2$ accuracy

Those trials on which the response to $Task_1$ was correct were analyzed for accuracy on $Task_2$. For T_1-present trials cell means for accuracy on $Task_2$ with probability (1 . 4 : 9) and T_1-T_2 lag (1 - 8) as variables were calculated and entered in to an ANOVA. Results are shown in Figure 6.

Insert Figure 6 about here

In comparison to accuracy on T_1-absent trials (0.795), accuracy on identifying T_2 when T_1 was present was severely attenuated (0.692) [F(1, 47) = 124.9, p < .0001, MS_e = .009]. Figure 6 shows mean accuracy on $Task_2$ for each T_1-T_2 lag and each probability level. In an analysis of T_1-present trials, a main effect of probability [F(2, 94) = 21.3, p < .0001, MS_e = .028] indicated that, overall, a larger AB effect was evident for trials where the T_1 letter was presented least frequently compared to trials on which the T_1 letter was presented most frequently. A main effect of T_1-T_2 lag showed that, in all probability conditions, accuracy was most reduced at the earlier T_1-T_2 lags and improved steadily as T_1-T_2 lag increased, reaching

performance equal to that of controls (T_1-absent trials) at T_1-T_2 lag 7 [$F(7, 329) = 36.6, p < .0001, MS_e = .032$]. An interaction of probability and T_1-T_2 lag revealed that performance in the probability 9 condition returned to be equal that of the control condition at an earlier T_1-T_2 lag than the other two probability conditions [$F(14, 658) = 1.9, p < .026, MS_e = .027$]. The probability 1 condition was the slowest to return to performance equal to that of the control condition. (See Appendix B for $Task_2$ accuracy for each probability at each T_1-T_2 lag.)

Analysis of T_1-not repeated and T_1-repeated trials

Because relative signal frequency was manipulated in this experiment, so that some T_1 letters were presented more often than others, it is possible to look at the effect, if any, of stimulus repetition. This effect, known as the repetition effect (Bertelson, 1961), occurs when the same stimulus is repeated on successive trials. Response time to a repeated stimulus is typically faster, and accuracy higher, than to an alternate stimulus. In the results reported here, there may be an effect of repeating T_1 on T_2 accuracy. If the T_1 letter was the same as on an immediately preceding trial (T_1-repeated), the effect on T_2 accuracy may be different from that observed when the T_1 letter was not the same (T_1-not repeated). On T_1-repeated trials, where response times to T_1 is likely to be faster, and accuracy higher, than on T_1-not repeated trials, less of an interference effect may be apparent in $Task_2$.

Finding an effect of stimulus repetition in this experiment would allow us to use this variable in a manner similar to stimulus probability, i.e. determine the locus of the effect in relation to the loci of the AB effect and the PRP bottleneck.

(N.B. stimulus repetition is a term used to refer to a variable manipulated in the experiments in this thesis. When used in this context, the term refers to the repetition effect in general and is not meant to imply that the effect of repetition is solely a function of repeating the stimulus).

As before, only those trials in which the response to $Task_1$ was correct were analyzed for accuracy on $Task_2$. The data were submitted to a new analysis in which T_2 performance was evaluated for trials on which the T_1 letter was the same as on the previous trial (T_1-

repeated) and compared to that of trials where the T_1 letter on the previous trial was not the same (T_1-not repeated). In other words, trials that were same-T_1 trials were compared to trials that were different-T_1. Because the number of observations of a repeated T_1 letter was relatively small for the probability 1 and 4 conditions, only trials that made up the probability 9 condition were used and analyzed for effects of T_1-T_2 lag and trial type (T_1-not repeated / T_1-repeated).

Insert Figure 7 about here

Analysis of the difference in overall T_2 accuracy between T_1-not repeated and T_1-repeated probability 9 trials approached, but failed to reach, statistical significance [$F(1, 47) = 3.1, p > .084, MS_e = .012$]. Figure 7 shows mean accuracy on $Task_2$ for each T_1-T_2 lag and each level of probability. The ANOVA revealed a main effect of T_1-T_2 lag [$F(7, 329) = 35.8, p < .0001, MS_e = .016$]. There was no interaction between T_1-not repeated / T_1-repeated and T_1-T_2 lag [$F(7, 329) = 0.98, p < .45, MS_e = .014$]. Though both trial types exhibited an AB effect the pattern of performance on each was a little different. (See Appendix C for $Task_2$ accuracy at each T_1-T_2 lag.)

Despite the lack of an interaction in the analysis reported above, the data pattern in Figure 7 suggests that an interaction between T_1-not repeated / T_1-repeated and T_1-T_2 lag exists. The difference between T_1-repeated and T_1-not repeated trials appears to be greater at the shorter T_1-T_2 lags than at the longer lags suggesting a difference in the size of the AB effect as a function of whether T_1 was repeated or not repeated. In an attempt to pull out this interaction we submitted the data to a new analysis. First, the difference between T_1-repeated and T_1-not repeated at each T_1-T_2 lag, for each subject, was calculated. The means of these difference scores across the early T_1-T_2 lags, T_1-T_2 lags 1 - 4, and across the later T_1-T_2 lags, T_1-T_2 lags 5 - 8, were then computed. Thus, means of the difference between T_1-repeated and

T_1-not repeated, for short and long T_1-T_2 lags, were produced. The difference between these means was statistically significant [$F(1, 47) = 5.5, p < .023, MS_e = .005$], showing a small but reliable decrease in the size of the AB when T_1 was repeated across consecutive trials.

In this experiment then, an effect of stimulus repetition was found for trials in the probability 9 condition. A T_1 stimulus that was the same as the T_1 letter on the immediately preceding trial produced less of an interference effect in $Task_2$ than a trial in which the T_1 letter was the not the same. The effect of stimulus repetition was also found to be greater when T_2 appeared at the early T_1-T_2 lags (1 - 4) than when it appeared at the later T_1-T_2 lags (5 - 8). Like stimulus probability, this variable can be used to provide evidence as to a locus of the AB effect in relation to the locus of the PRP bottleneck.

Analysis of T_1-not repeated trials

In light of the finding of an effect of stimulus repetition in this experiment it was of interest at this point to examine whether an effect of stimulus probability would be observed when only those trials classified as T_1-not repeated trials were analyzed. Thus, T_1-not repeated trials, i.e. trials on which the T_1 letter was the same as the T_1 letter on an immediately preceding trial, were excluded from the analysis. Hence, in this analysis, there are no repeated stimulus trials and therefore effects of immediate stimulus repetition have been removed.

Cell means were calculated and entered in to an ANOVA with probability (1: 4 : 9) and T_1-T_2 lag (1 - 8) as variables.

In comparison to accuracy on T_1-absent trials (0.796), accuracy on identifying T_2 on T_1-present trials was severely attenuated (0.689) $F(1, 47) = 165.6, p < .0001, MS_e = .004$].

Insert Figure 8 about here

When T_1-present trials were analyzed separately a significant main effect of T_1-T_2 lag was revealed [$F(7, 329) = 33.1, p < .0001, MS_e = .04$]. This effect can be seen in Figure 8. Accuracy on T_2 was lowest when T_2 appeared at T_1-T_2 lag 2 and improved steadily over subsequent lags reaching highest accuracy at T_1-T_2 lag 7.

Also there was a significant difference in accuracy on T_2 as a function of the probability of presentation of the T_1 letter [$F(2, 94) = 16.5, p < .0001, MS_e = .033$]. Subjects demonstrated lowest accuracy on low probability trials, were intermediate for the probability 4 condition, and accuracy was highest on trials associated with the highest probability T_1 letter. (See Appendix D for $Task_2$ accuracy for each level of probability at each T_1-T_2 lag.)

Contrary to the findings of the overall analysis (*Analysis of $Task_2$ accuracy*) in which all T_1-present trials were included, the interaction of probability and T_1-T_2 lag only approached significance for T_1-not repeated trials [$F(14, 658) = 1.6, p < .082, MS_e = .031$]. The marginal statistical interaction here is likely due to the influence of noise on trials in the lower probability conditions. When all trials were included in the analysis, the probability 1 condition consisted of a maximum of 4 trials per T_1-T_2 lag, per subject. This number is low in comparison to the probability 9 condition (<= 36 trials per T_1-T_2 lag, per subject), but the inequality of trial number between the probability conditions is a necessity of the experimental design. In the present analysis, in addition to the trials that may have been eliminated because the response to T_1 was not correct, and/or because of the RT_1 outlier procedure, the number of trials has been further reduced due to the T_1-not repeated restriction. When the reduction in trial number was investigated it was found that the greatest reduction was in the probability 9 condition which dropped from an average of about 34 per subject when all the trials were included, to approximately 19 when only T_1-not repeated trials were analyzed. In the probability 4 condition the number of trials dropped from about 14 to 11, and in the probability 1 condition, from an average of about 4 to 3. Evidence supporting the proposal that the strength of the interaction between probability and T_1-T_2 lag is affected by the low number of trials representing the probability 1 condition, when the analysis is restricted to T_1-not repeated trials, is provided by further, more detailed, examination of the interaction found in the initial

analysis, in which all trials were included (see *Analysis of Task$_2$ accuracy*). In this analysis the interaction of probability and T_1-T_2 lag reached statistical significance when all probability conditions were included in the analysis (p <.026) but was not significant when the analysis was restricted to only the probability 1 and 4 conditions [$F(7, 329) = 0.97, p < .46, MS_e$ = .036]. When the probability conditions 4 and 9 were compared, they were found to be significantly different [$F(7, 329) = 3.2, p < .002, MS_e = .011$], as was analysis of the probability 1 and 9 conditions $F(7, 329) = 2.4, p < .021, MS_e = .033$]. Comparison of the data patterns in Figures 6 and 8, serves to show that the pattern of means for the overall analysis, when all trials were included and for the analysis of T_1-not repeated trials are very similar, and that the effect of probability for T_1-not repeated trials is comparable to that observed over all trials.

These results clearly show an AB effect for trials in which T_1 was not the same on the preceding trial (T_1-not repeated), and this effect was modified by the probability of the T_1 letter. Therefore, effects similar to those found when all the experimental trials were entered in to the analysis were obtained using a data set restricted to those trials on which the T_1 letter was not the same as on an immediately preceding trial.

Task$_2$ as a function of RT$_1$

The Central Interference Theory (Jolicoeur, 1999b) predicts that Task$_1$ response times should correlate with accuracy on Task$_2$. When Task$_1$ occupies central mechanisms for a long period of time the AB effect should be larger than when those mechanisms are occupied for a shorter period of time. Also, the duration of processing when central mechanisms are occupied should be reflected in RT_1. Consequently, long RT_1s should be correlated with poorer accuracy on Task$_2$ and short RT_1s should correlate with an increase in Task$_2$ accuracy, producing a smaller AB effect. This prediction can be tested by examining the AB effect as a function of RT_1.

However, one precaution must be taken here because it is possible that a correlation between RT_1 and T_2 accuracy could reflect a change in performance over time, rather than a

causal effect tied to central processing. For example, if performance on $Task_1$ and $Task_2$ improved as the experiment progressed, a relationship between RT_1 and T_2 accuracy might be observed, as predicted by the Central Interference Theory. Subjects might be slower and less accurate at the beginning of the experiment simply because they are new to the tasks, and performance might improve over the course of the experiment as they become more expert at performing the tasks. Under these conditions, a correlation between RT_1 and $Task_1$ accuracy might be present, but only because slower responses made at the beginning of the experiment were coupled with poorer performance on $Task_2$ at the same point in the experiment. The potential for determining whether a correlation between RT_1 and $Task_2$ is a result of the fundamental mechanism stated by the Central Interference Theory, or whether it is an artifact of practice, lies in examining RT_1 and $Task_2$ performance for significant changes over trials. This can be accomplished by dividing the experimental trials into sections, representing progression through the experiment, and looking for a consistent AB effect correlational relationship between RT_1 and $Task_2$ performance across each section, rather than across the experiment as a whole.

Each subject's data was divided into four sets of trials, each corresponding to 168 sequential experimental trials, making a total of 672 trials. These trials were then divided into cells representing T_1-T_2 lag. As the probability 1 and 4 conditions resulted in empty cells, because of too few observations, only the probability 9 condition was included. Each cell was subsequently sorted into two, depending on whether RT_1 was above or below the RT_1 cell median. Mean accuracy in $Task_2$ for each cell was calculated and submitted to an ANOVA with T_1-T_2 lag, short and long RT_1, and trial set (first, second, third, and fourth) as within-subject factors. The overall means for short and long RT_1s and each T_1-T_2 lag are shown in Figure 9.

The analysis revealed an effect of trial set [$F(3, 141) = 6.9, p > .0002, MS_e = .046$]. Mean accuracy for $Task_2$ was lower in the first trial set than in the other sets (Set 1 = 0.70; 2 = 0.74; 3 = 0.74; 4 = 0.75). This effect was eliminated when the first set was not included in the analysis ($p < .55$), thereby suggesting that performance on $Task_2$ was poorer on the initial 168 trials at the beginning of the experiment, in comparison to performance throughout subsequent

trials. This difference may indicate that subjects should have been given more practice prior to the experimental trials. Most importantly, the correlation between RT_1 and $Task_2$ performance was consistent throughout the 4 sets of trials thereby providing support for the Central Interference Theory. The pattern in the 4 trial sets was similar to that shown in Figure 9 - long RT_1s produced a larger AB effect than short RT_1s.

Insert Figure 9 about here

Discussion

Relative signal probability in $Task_1$ affected the magnitude of the AB effect. $Task_1$ stimuli presented less frequently produced a larger AB effect than signals presented more frequently. This outcome tells us that, in this experiment, the effect of probability is occurring at a stage of processing that is at, or before, the locus of the AB effect.

An alternative explanation as to the effect of probability in $Task_1$ can be addressed at this point. If subjects were not performing the tasks according to the experimental instructions, and were guessing T_1 in an attempt to perform well on T_2, an effect of probability might be found for T_1. This is because, if subjects were guessing, the most frequent response would likely be the one associated with the highest probability T_1 letter, the probability 9 condition. Thus, one might expect to see an effect of probability on T_1 that is due to a guessing strategy, used to improve $Task_2$ performance, rather than an effect of the probability manipulation. However, there is evidence in the results to suggest that this was not the case. Firstly, if subjects were guessing on T_1, and their most frequent response was the one associated with the highest probability T_1 letter, accuracy on T_1 should be high for the probability 9 condition but low for the other conditions, including the T_1-absent condition. The results show, however, that T_1 accuracy was high across all probability conditions as well as when T_1 was

absent (see Appendix A). In addition, if participants were guessing on T_1 one would not expect to see an effect of probability on T_2. Clearly, such an effect is present.

Although the interaction of stimulus probability and T_1-T_2 lag only approached significance (p <.082) when the analysis was limited to those trials in which the T_1-letter was not repeated on the immediately preceding trial, probability nevertheless modified the AB effect under these conditions. The effect of probability for T_1-not repeated trials was similar to the effect observed when all trials were included in the analysis. Accuracy on $Task_2$ declined as the probability of presentation of the T_1 letter declined.

Not only was the AB effect modified by varying signal probability, but stimulus repetition was also found to have an effect on T_2 accuracy. Stimuli repeated in succession are typically responded to faster and more accurately than non-repeated stimuli. The effect of repeating a stimulus on the magnitude of the AB was observed by sorting the data into trials on which the T_1 letter was different from T_1 on the immediately preceding trial, and trials where the T_1 letter was the same as on the previous trial, and submitting these data sets to statistical analyses as before. Pashler and Johnston (1989) found, in an experiment designed to exhibit a PRP effect, that the effects of stimulus repetition were additive with SOA, suggesting that the locus of the repetition effect is at, or beyond, the PRP bottleneck. Similar findings of repetition effects found in the PRP experiments contained within this thesis are reported later.

The finding that stimulus repetition is a variable that affects the magnitude of the AB provides an additional resource to which the general procedure outlined within the objective of this thesis can be applied. At this point it is clear that both variables, stimulus probability and stimulus repetition, have an effect on the size of the AB and that the effects are therefore occurring at or before a locus of AB interference effect. It is possible now to use the same logic for stimulus repetition as originally applied to stimulus probability. Knowing these variables have an effect on the AB also makes it possible to use both variables in a PRP experimental design and apply the locus of cognitive slack logic to determine their effects in relation to the locus of the PRP bottleneck.

Experiment 2

Before determining whether stimulus probability and repetition effects are evident in a PRP design, we investigated whether such effects are observable in an AB experiment in which response to T_1 is not speeded. Most studies investigating the AB effect have not used speeded $Task_1$ conditions (Raymond, et al., 1992; Shapiro et al., 1994; Raymond, et al., 1995). Therefore, it seemed important to provide evidence that the AB effect could be demonstrated, using these variables, in a typical AB experimental paradigm under unspeeded $Task_1$ conditions. In addition, if the data from a $Task_1$ unspeeded experiment was very different from Experiment 1, in which $Task_1$ was speeded, this might help to localize the effects of stimulus probability and stimulus repetition on T_2 accuracy, thereby allowing us to establish more precisely a locus of the AB effect. Effects of stimulus probability and stimulus repetition in an unspeeded AB experiment would imply once again that the effect of these variables is occurring at, or before, the locus of the AB. In contrast, the observation of an AB effect unmodified by probability or stimulus repetition would ascertain that these effects, under T_1 unspeeded conditions, are occurring independently of the AB effect, and consequently are located at a stage of processing subsequent to the locus of the AB effect. Differential effects for both variables would suggest that the loci of these effects in relation to the locus of the AB effect are not the same.

Method

Subjects

Seventy-seven undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All subjects had normal or corrected-to-normal vision.

Procedure

Method and procedure were the same as in Experiment 1 except that response to T_1 was not speeded. Subjects were instructed to wait until the end of the stimulus stream to make their responses to T_1 and T_2. A prompt appeared at the end of the RSVP stream "Did you see a red

H, O, or S or no red letter?". After making this response, by using the same response key assignment as in Experiment 1, a similar prompt was given for the T_2 response "Did you see and X or a Y?". After making both responses subjects pressed the space bar to initiate the next trial.

Results

Individual subject data was analyzed for accuracy on $Task_2$ to ensure that all subjects could perform the task of identifying when no T_1 letter was present. Twenty-nine individuals who failed to perform at or above 69% accuracy on $Task_2$, on T_1-absent trials where $Task_1$ was correct, were eliminated from the data set. There were 48 subjects in this final data set.

Analysis of $Task_1$ accuracy

For $Task_1$ there was a main effect of probability [$F(2, 94) = 7.3$, $p > .001$, $MS_e = .025$] in that accuracy was superior in the probability 9 condition and poorest in the probability 1 condition. (See Appendix E for accuracy rates for each level of probability at each T_1-T_2 lag.)

Analysis of $Task_2$ accuracy

Those trials on which the response to $Task_1$ was correct were analyzed for accuracy on $Task_2$. As in Experiment 1, accuracy for $Task_2$ on T_1-present trials was severely attenuated (0.694) in comparison to performance on trials where T_1 was absent (0.828) [$F(1, 47) = 130.1$, $p < .0001$, $MS_e = .015$]. For T_1-present trials cell means for accuracy on $Task_2$ with probability of (1: 4 : 9) and T_1-T_2 lag (1 - 8) as variables were calculated and entered into an ANOVA. Results are shown in Figure 10.

Insert Figure 10 about here

Main effects of probability [$F(2, 94) = 35.8, p < .0001, MS_e = .032$], and T_1-T_2 lag [$F(7, 329) = 17.5, p < .0001, MS_e = .036$] were again evident, and an interaction between these variables approached significance [$F(14, 658) = 1.6, p > .07, MS_e = .026$]. A larger AB effect was observed for the T_1 letter associated with the lowest frequency (probability 1), and the smallest effect was seen for the probability 9 condition, while performance for the probability 4 condition was between the other two. When T_2 appeared at T_1-T_2 lag 1, the average accuracy across all three probability conditions was 0.715, dropping to 0.598 at T_1-T_2 lag 2. Performance progressively improved across the subsequent T_1-T_2 lags except in the probability 1 condition where it dropped at T_1-T_2 lag 4, climbing sharply to equal that of the probability 4 condition at T_1-T_2 lag 5. The interaction of probability and T_1-T_2 lag was further examined to see whether there was a contribution of noise from the probability 1 condition that was possibly weakening the interaction. When the analysis was restricted to the probability 4 and 9 conditions statistical significance remained marginal ($p > .07$). The effect was only significant when the probability 1 and 9 conditions were compared [$F(7, 329) = 2.1, p > .04, MS_e = .034$], suggesting that the number of trials in the probability 1 and 4 conditions was limiting the ability to detect a strong interaction between probability and T_1-T_2 lag. (See Appendix F for accuracy rates for each probability at each T_1-T_2 lag.)

Analysis of T_1-not repeated and T_1-repeated trials

Using trials from the probability 9 condition, the effect of T_1-T_2 lag was evaluated for trials on which the same T_1 letter had appeared on the previous trial (T_1-repeated trials) and compared to those trials on which the T_1 letter was different (T_1-not repeated).

Insert Figure 11 about here

A comparison of T_1-not repeated T_1-repeated trials in the probability 9 condition can be seen in Figure 11. In contrast to the speeded version of this AB experiment a significant difference in $Task_2$ accuracy was observed between T_1-repeated and T_1-not repeated trials [$F(1, 47) = 17.3, p > .0001$, $MS_e = .011$]. Subjects were more accurate on trials on which the T_1 stimulus had been repeated than on T_1-not repeated trials.

The pattern of data across the T_1-T_2 lags is very similar for T_1-not repeated and T_1-repeated trials and there is a significant effect of T_1-T_2 lag [$F(7,329) = 14.8, p < .0001$, $MS_e = .019$]. However, as in Experiment 1, there is no interaction between T_1-not repeated / T_1-repeated and T_1-T_2 lag [$F(7,329) = 1.2, p < .27$, $MS_e = .009$]. Accuracy on T_2 declined dramatically when T_2 was presented at T_1-T_2 lag 2, improving across the remaining T_1-T_2 lags and reaching maximum accuracy at T_1-T_2 lag 8. (See Appendix G for accuracy rates for each T_1-T_2 lag for T_1-not repeated and T_1-repeated trials.)

To examine further whether there was a difference in the size of the AB effect between T_1-not repeated and T_1-repeated trials the difference at the shorter T_1-T_2 lags was compared to the difference at the longer T_1-T_2 lags. The difference between T_1-T_2 lags 1 - 4 and T_1-T_2 lags 5 - 8, for T_1-not repeated and T_1-repeated trials, for each subject were calculated, yielding means of these scores for short and long T_1-T_2 lags. Like Experiment 1, the difference between these means was statistically significant [$F(1, 47) = 4.1, p < .050$, $MS_e = .005$], showing a small, but reliable decrease in the size of the AB when T_1 was repeated across consecutive trials.

Analysis of T_1-not repeated trials

The effects of stimulus probability and T_1-T_2 lag were evaluated for trials where the T_1 letter was the first in a sequence of same-T_1 trials or was followed by an alternate T_1 letter (T_1-not repeated). Hence, as in Experiment 1, only those trials on which the T_1 letter was different from the T_1 letter on the immediately preceding trial were analyzed.

Insert Figure 12 about here

Figure 12 shows the data for the T_1- absent and T_1-present trials. Accuracy on identifying T_2 on T_1-present trials was severely attenuated (0.681) in comparison to performance on T_1-absent trials (0.817)trials [$F(1, 47) = 131.3, p < .0001, MS_e = .017$].

In an analysis of T_1-present trials a probability effect [$F(2, 94) = 23.7, p < .0001, MS_e = .038$], and an effect of T_1-T_2 lag [$F(7, 329) = 19.5\ p < .0001, MS_e = .038$] were observed but the interaction between these variables failed to reach significance [$F(14, 658) = 1.46, p > .12, MS_e = .031$]. Accuracy on T_2 showed the greatest overall attenuation for probability 1 trials, followed by probability 4, and was highest in the probability 9 condition. The effect of T_1-T_2 lag can be seen in the dramatic drop in $Task_2$ accuracy when T_2 appeared at lags 2 and 3 compared to lag 1, and the subsequent steady improvement over the remaining T_1-T_2 lags. The probability 1 condition deviated a little from this pattern showing poorest performance at T_1-T_2 lag 4. (See Appendix H for accuracy rates on $Task_2$ for each probability level and T_1-T_2 lag.)

As in Experiment 1, there was a large reduction in the number of trials per condition for this analysis. In the probability 9 condition the number of trials was reduced from about 34 per subject to about 20. In the probability 4 condition the number dropped from an average of approximately 15 to 12, and in the probability 1 condition from about 4 to 3. The lack of an interaction between probability and T_1-T_2 lag for the T_1-not repeated trials is likely due to the influence of noise caused by the reduction in the number of trials per T_1-T_2 lag, especially in the probability 1 condition where the number of trials was already low before the T_1-not repeated limitation was applied.

Discussion

In this unspeeded AB paradigm, stimulus probability and stimulus repetition modified the size of the AB effect. As the probability of a stimulus increased, the magnitude of the AB decreased. This effect was also evident when T_1-repeated trials were removed from the data set leaving only those trials on which the T_1 letter was not the same as the T_1 letter on the previous trial. In addition to the effects of signal probability, the size of the AB effect decreased slightly, but significantly, for repeated stimuli in comparison to non-repeated stimuli in the probability 9 condition.

Clearly, there is an effect of signal probability observed in performance judging a T_2 letter when it follows a T_1 letter presented in an RSVP stream in an AB experimental paradigm. The effect of probability is apparent when $Task_1$ is speeded (Experiment 1) and when it is unspeeded (Experiment 2). The conclusion is that the probability effect in $Task_1$ is occurring at, or before, a stage of processing responsible for the AB effect regardless of whether $Task_1$ is speeded or unspeeded.

Stimulus repetition also modified the AB effect, under speeded and unspeeded T_1 conditions, though this effect was stronger when $Task_1$ did not entail a speeded response (speeded = $p > .024$; unspeeded = $p < .050$). In the probability 9 condition, a T_1 letter that was the same as the T_1 letter on an immediately preceding trial produced less of an interference effect than a different T_1 letter. Thus, a conclusion similar to that given for stimulus probability can be applied to stimulus repetition. The effect of stimulus repetition must be occurring at, or before, the locus of the AB effect.

Experiment 3

Experiments 1 and 2 determined that the effects of relative signal probability and stimulus repetition are occurring at, or before, the locus of the AB effect in $Task_1$ speeded and unspeeded AB paradigms. We now wish to discover the locus of these effects in relation to the PRP bottleneck. According to the locus of cognitive slack logic, an additive pattern to the results would indicate that the effects are occurring at or beyond the PRP bottleneck, while

underadditivity would be indicative of a locus at a stage of processing before the PRP bottleneck. Overadditivity, on the other hand, would suggest a capacity sharing model rather than a postponement model of information processing.

Method

Subjects

24 undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All subjects had normal or corrected-to-normal vision. The experiment was run on a SVGA colour computer screen controlled by a 384, 486, or 586 CPU.

Stimuli

Stimuli were auditory tones and upper-case letters. The tones were of frequencies 400 Hz, 1000 Hz and 2500 Hz and were presented using the internal speaker of the computer. The letters were H, O, and S, approximately 0.8° x 1°, presented in the centre of the screen in white on a black background.

Procedure

Each trial began with the presentation of one of the tones (S_1), for a duration of 100 ms, followed by the visual presentation of one of the letters (S_2). The letter remained on the screen until a response had been made. The SOA between the tone and the letter was varied and could be either 50 ms, 200 ms, 450 ms, or 800 ms. A probability variable was associated with the letter stimulus. The relative probability of each letter was 1: 4: 9 meaning that, within every 14 trials one letter (e.g., H) was presented 9 times, another letter (e.g., O) was presented 4 times, while the remaining letter (e.g., S) was presented once. The probability level assigned to each letter was counterbalanced across subjects so that, for every 6 subjects, all combinations of letters and probability levels were used once. Each complete crossing of SOA, letter (1 : 4 : 9), and tones, was repeated 4 times to make a total of 672 trials that were divided equally between 4 blocks. The experimental trials were preceded by 1 block of 48 practice trials.

At the beginning of the experimental session subjects read instructions regarding the procedure and were subsequently instructed verbally on the same matter. The first task ($Task_1$) was to identify the tone (low, medium, or high). The second task ($Task_2$) was to identify the letter (H, O, or S). Reaction time to both stimuli was recorded and responses to the tone and the letter were made by pressing assigned keyboard keys. The assigned keys for the tones were as follows: z for low tone (400 Hz); x for medium tone (1000 Hz); c for high tone (2500 Hz). For the letters they were: ,< for H; .> for O; /? for S. The left hand was used to respond to the tone and the right hand to respond to the letter. After each trial (except the last trial in each block) feedback was given, in the form of adjacent plus and/or minus signs, as to whether the $Task_1$ and $Task_2$ had been responded to correctly or incorrectly. The sign on the left referred to $Task_1$ and the one on the right to $Task_2$. These feedback symbols also served as a fixation point for the upcoming trial. Each trial was initiated by pressing the space bar, which caused the fixation symbols to disappear, and the trial to begin 750 ms later.

Results

Only trials on which responses to $Task_1$ and $Task_2$ were correct were used in the analyses. Response times were screened for outliers in each cell for each subject using a modified version of the procedure described by Van Selst and Jolicoeur (1994). Percent rejected were 2.7% for $Task_1$ and 1.8% for $Task_2$.

Analysis of $Task_1$ response times and accuracy

Cell means for reaction time for $Task_1$ with signal probability (1: 4: 9) and SOA (50/200/450/800 ms) as variables were calculated and entered in to an ANOVA. Significant main effects of probability [$F(2, 46) = 3.7, p < .034, MS_e = 6980.54$], and SOA [$F(3, 69) = 3.4, p < .023, MS_e = 3921.47$] were evident. As can be seen in Figure 13 (dashed lines), the effect of probability was strongest at the shortest SOA. Also, at the shortest SOA, and at all levels of probability, except the probability 9 condition, mean response time to S_1 was longer than at any other SOA. In general, RT_1 increased linearly as SOA increased from 200 ms to

800 ms, except for the probability 1 condition where RT_1 was faster at the 200 ms and 450 ms SOAs than at the 800 ms SOA. The effects of probability and SOA in $Task_1$ were eliminated when the 50 ms SOA was not included in the analysis. (See Appendix I for mean reaction time for each level of probability and SOA for $Task_1$.)

Error rates indicated a main effect of SOA [$F(3, 69) = 9.0, p < .0001, MS_e = .002$]. Error rates were slightly higher at the shorter SOAs (50 ms and 200 ms) than at the longer SOAs (450 ms and 800 ms). (See Appendix I for mean error rates for each level of probability and SOA for $Task_1$.)

Insert Figure 13 about here

Analysis of $Task_2$ response time and accuracy

A similar analysis was performed on mean response times for $Task_2$. Figure 13 (solid lines) shows the mean response time for each SOA and each probability level. A significant effect of signal probability was revealed [$F(2, 46) = 28.0, p < .0001, MS_e = 19776.99$]. Subjects were faster to respond to the letter that was most likely to appear (probability 9) and were slowest in responding to the letter that was least likely to appear (probability 1). An effect of SOA was also evident, response times decreasing as SOA increased, providing clear evidence of a PRP effect [$F(3, 69) = 455.0, p < .0001, MS_e = 7863.21$]. The effect of probability was additive across SOAs with no hint of an interaction [$F(6, 138) = 0.5, p > 0.785, MS_e = 5333.629$]. Although there was a slight fanning out of the means at the longest SOA this effect was no where near being statistically significant. (See Appendix J for mean response times for each level of probability and each SOA for $Task_2$.)

Percentage of errors for $Task_2$ was highest for the probability 1 condition (0.116) and lowest for the probability 9 condition (0.041), with the probability 4 condition intermediate between the two (0.103) [$F(2, 46) = 18.0, p < .0001, MS_e = .008$]. No effect of SOA was

evident [$F(3, 69) = 1.1, p < .371, MS_e = .003$]. (See Appendix J for mean error rates for each level of probability and SOA for $Task_2$.)

Analysis of S_2-not repeated and S_2-repeated trials

In addition, in the probability 9 condition, RT_2s for those trials on which the letter was the same as on the previous trial (S_2-repeated) were compared to trials where the letter on the preceding trial was different (S_2-not repeated).

Insert Figure 14 about here

Figure 14 shows a PRP effect for both trial types that is additive with SOA ($F(3, 69) = 1.1, p > .342, MS_e = 3205.511$]. Reaction time increased as SOA decreased [$F(3, 69) = 765.1, p < .0001, MS_e = 3278.885$], and S_2-repeated trials were faster than S_2-not repeated trials [$F(1, 23) = 42.07, p < .0001, MS_e = 4723.645$]. (See Appendix K for mean response times for all levels of probability and SOA.)

Analysis of S_2-not repeated trials

As in the AB experiments, the effect of stimulus probability was investigated for trials on which the target stimulus was not the same as on the immediately preceding trial. Trials on which the S_2-letter was not the same as the letter on an immediately preceding trial (S_2-not repeated) were entered in to an ANOVA with probability and SOA as within-subject factors. The results can be seen in Figure 15.

Insert Figure 15 about here

A significant effect of SOA was evident [$F(3, 69) = 358.3, p < .0001, MS_e = 10330.207$] in that mean response time to $Task_2$ decreased as SOA increased. In addition there was a significant effect of probability [$F(2, 46) = 14.0, p < .0001, MS_e = 21812.31$] which was additive with SOA [$F(6, 138) = 0.7, p < .669, MS_e = 6939.31$]. Letters that were presented most frequently were responded to faster than those presented least frequently, while the mean response time to letters in the probability 4 condition was intermediate between the other two probability conditions. (See Appendix L for mean response times for all levels of probability and SOA.)

Discussion

In this PRP experiment two tasks were performed in close succession to each other and performance on $Task_2$ was significantly affected by the temporal proximity of the two stimuli associated with those tasks (S_1 and S_2). Stimulus-onset asynchrony was varied and, as SOA decreased, mean response time in the second task increased. This pattern of results clearly demonstrates a classic PRP effect (Pashler, 1994b). According to the postponement account of PRP interference, interference in processing the second of two stimuli results from the inability of the processing system to accommodate concurrent processing when processing is common to more than one task. When two tasks require the same processing resources, processing necessary for the second task must wait until processing of the first task is complete, and thus, a processing bottleneck exists. When SOA is short, the degree of task overlap is greater than when SOA is long, and processing necessary for the second task can begin earlier. Nevertheless, $Task_2$ processing will be interrupted at the processing bottleneck resulting in a 'wait' period, or period of cognitive slack (McCann & Johnston, 1992), and a slowing in $Task_2$ responses. In Experiment 3 processing the auditory tone, the first task, interfered with the second task of identifying a letter, and resulted in a slowing of response times to the letter as SOA decreased.

In addition to the effect of SOA, this study clearly showed an effect of signal probability. Response times were longer to $Task_2$ stimuli, letters, that were presented less

frequently than to letters presented more frequently, and this effect was additive with SOA. As SOA decreased, RT_2 increased by about the same amount across all probability levels. According to the locus of cognitive slack logic, an additive pattern such as this indicates that the variable, in this case stimulus probability, is having an effect at, or beyond, the PRP bottleneck. If the effect of stimulus probability were occurring prior to the bottleneck stage, $Task_2$ response times should have produced an underadditive interaction of probability and SOA, as SOA was reduced. Any increase in the duration of a processing stage prior to the bottleneck would be absorbed in the period of cognitive slack.

The results from Experiment 3 indicate that the locus of the probability effect is either at, or later than, the locus of the PRP bottleneck and we found a similar effect of probability when the S_2-repeated trials were removed from the data set. When those trials on which the S_2 letter was not the same as the S_2 letter on the previous trial were analyzed separately, the effect of probability was again observed to be additive with SOA. Thus, as found in the AB experiments, the effect of probability in this experiment was not only a function of repeating the stimulus on sequential trials, but was an effect brought about by the frequency of presentation associated with a stimulus spanning multiple trials.

In this study we also found an effect of stimulus repetition. Mean response time to an S_2 stimulus that was the same as the stimulus on an immediately preceding trial, was faster than mean response time to a stimulus that was different. Like stimulus probability, the effect of stimulus repetition was additive with SOA, indicating that the locus of this effect is also at, or after, the PRP bottleneck.

Experiment 3 demonstrated that the locus (i) of the effect of two variables, stimulus probability and stimulus repetition, is at, or after, the PRP bottleneck. Evidence in the PRP literature strongly suggests that the PRP bottleneck is located at a late stage of information processing, probably response selection (McCann & Johnston, 1992; Schubert, 1999; Van Selst & Jolicoeur, 1997). Thus, the data provided by this PRP experiment leads to the inference that the locus(i) of stimulus probability, and of stimulus repetition, is at, or after, the

stage of response selection. The implication of this interpretation with respect to the locus of the AB effect will be discussed later.

Under a simple postponement theory it is usually assumed that performance on $Task_1$ should be unaffected by the temporal proximity of S_1 and S_2 (Pashler, 1994b). Contrary to this supposition however, the effect of SOA affected reaction time to the auditory tone (S_1) in Experiment 3. In the probability 4 and 9 conditions, at the three longest SOAs, the effect of SOA on reaction time to S_1 is seen as a slowing in response to S_1 as SOA increases. In the probability 1 condition, RT_1 is faster at the two intermediate SOAs than at the longest SOA. At the shortest SOA the pattern was slightly different. In the probability 1 and 4 conditions, subjects were slowest in responding to S_1 at the 50 ms SOA, but in the probability 9 condition they were slowest at the longest SOA. The general pattern of RT_1 slowing with increasing SOA could possibly result from subjects grouping S_1 and S_2 responses on some trials, rather than responding to the first stimulus independent of the second stimulus. This strategy would entail saving the first response until the second response is selected and would result in a slowing of RT_1 that is correlated with the time between onset of the two stimuli, increasing as SOA increases (Pashler, 1984).

The effects on RT_1 in this experiment are small, and not unusual in PRP data. Possible explanations might exist (e.g., grouping, see Pashler & Johnston, 1989) but, at present, the cause of the specific effects of $Task_2$ manipulations on $Task_1$ performance in Experiment 3 are unclear. The results of interest in Experiment 3 are those from $Task_2$ and, though effects on $Task_1$ were found in the data, these effects were very much smaller than those in $Task_2$ as demanded by a postponement account. Thus it seems reasonably safe to focus the analysis on the results from $Task_2$.

However, because of the influence of SOA on RT_1 in the present experiment, it seems appropriate to discuss the capacity sharing account of the PRP effect. Capacity sharing predicts, not only that the effect of SOA should be relatively equal across $Task_1$ and $Task_2$, but also that the effect should be in the same direction for both tasks. According to this account, processing resources are shared and performance on both tasks is affected by task overlap.

Consequently, reaction time for $Task_1$ and $Task_2$ should decrease as the time between the corresponding stimuli increases. Clearly this pattern is demonstrated for $Task_2$ in Experiment 3 but the effect of SOA on $Task_1$ is counter to the capacity sharing prediction. In general, $Task_1$ reaction time increased as SOA increased at all but the shortest SOA (50 ms). The observation that RT_1 increased with increasing SOA, does not support the capacity sharing account of information processing.

It should be noted that the percentage of errors on $Task_1$ decreased as reaction time slowed suggesting the presence of a speed-accuracy trade-off. As subjects responded more quickly in $Task_1$ they in turn committed more errors. In $Task_2$ there is no effect of SOA on errors, indicating that accuracy across SOAs was not affected by subjects' speed of response.

Experiment 4

Although the results from Experiment 3 provide no statistical indication of an interaction between probability and SOA (F <1), $Task_2$ performance at the longest SOA (800 ms), in the probability 9 condition, appeared to deviate slightly from strict additivity. There is a larger RT_2 difference between each probability level at this SOA than at other SOAs. To be certain that this pattern was not indicative of weak underadditivity that we might have failed to detect, Experiment 4 was conducted. The question we need to address is whether this data truly reflects additivity or whether it suggests an underadditive interaction of stimulus probability with decreasing SOA. One explanation for the slight deviation from additivity at the longest SOA is the possibility that this data point represents a state of over-preparation. Subjects are likely expecting, and are conceivably more prepared, for the most frequently presented stimulus. Accuracy is not affected (.95), but the longer SOA (800 ms) provides adequate time to prepare a response and may result in a state of over-preparation, manifest as a disproportionate decrease in reaction time. In light of this possibility it would be of interest to observe the data pattern under conditions where the longest SOA is longer than 800 ms. In addition, we decided to add a large proportion of catch trials, in which no S_2 was presented. This modification to Experiment 3 would hopefully discourage subjects from over-preparing

their response to the most likely trial stimulus. With the addition of these S_2-absent trials it was hoped that subjects would wait for S_2 and process it in an unbiased way rather than anticipating the most frequent S_2. The addition of S_2-absent trials would also serve to equate the PRP experiments with the AB experimental design in which T_1-absent trials were included as control trials in the AB experiments.

Method

Method and procedure for Experiment 4 were similar to those of Experiment 3.

Subjects

Twenty-four undergraduate students from the University of Waterloo were paid a nominal amount to participate in the study. All subjects had normal or corrected-to-normal vision.

Stimuli

Stimuli for $Task_1$ were identical to those used in Experiment 3.

Procedure

Modifications to the design were as follows. In addition to the SOAs used in Experiment 3, a 1200 ms condition was included. Also, a condition in which there was no S_2 letter was added. On half the trials an S_2 letter, H, O, or S, was presented (S_2-present trials). The remainder of the trials were S_2-absent trials. The relative probability (1: 4: 9) associated with each letter was counterbalanced, as before, between subjects. On the S_2-present trials, subjects responded using the appropriate keyboard keys, which were the same as those assigned in Experiment 3. On S_2-absent trials, an audible 'chirp' sound was presented at 1600 ms SOA to inform the subject that no letter would appear and that they should press the spacebar to continue to the next trial. The 'chirp' sound was created by presenting a sequence of four 50 ms tones (632 Hz, 1581 Hz, 632 Hz, and 1581 Hz). Reaction times for $Task_1$ were recorded on every trial, and for $Task_2$ on the S_2-present trials.

Because of the addition of the S_2-absent condition and the 1200 ms SOA condition, the number of trials increased, from 672 in Experiment 3 to 1260 in the present experiment. Due to

the length of this experiment, the trials were divided into two sessions and run over two days. In the first session subjects participated in a practice block of 100 trials. This practice session was followed by 4 blocks of experimental trials each block consisting of 105 trials. In the second session, on day two, the practice black was reduced to 40 trials and was followed by 8 blocks of experimental trials, 105 trials in each, making a total of 1260 experimental trials run over the course of the two sessions.

The procedure was identical to Experiment 3 except that subjects were informed that on some trials the second stimulus (letter) would not be presented and in those cases they would hear a 'chirp' sound at which time they were to press the space bar. To minimize the chance of subjects waiting for the chirp rather than preparing for a possible response, which might be particularly likely at the longest SOA (1200 ms), subjects were instructed to be prepared to respond to a letter right up until they heard the 'chirp' sound.

Note that the most frequent type of trial in this experiment was the S_2-absent trial, which occurred on 50% of the trials. If subjects tend to over-prepare the most frequent response they would prepare the response with the spacebar rather than to one of the critical S_2 target letters.

Results

Response time outliers for correct responses on both tasks were removed from the data set in a similar fashion to Experiment 3. Percent rejected was 2.4% for $Task_1$ and 1.6% for $Task_2$.

Analysis of $Task_1$ response time and accuracy

Cell means for reaction time to $Task_1$ with signal probability in $Task_2$ (1: 4: 9) and SOA (50/200/450/800/1200 ms) as variables were calculated and entered in to an ANOVA. Significantly slower response times at the shorter SOAs (50 ms and 200 ms) were responsible for the effect of SOA in $Task_1$ [$F(4, 92) = 31.8, p < .0001, MS_e = 4418.873$]. This pattern can be seen in Figure 16. There was no effect of signal probability [$F(2, 46) = 1.1, p > .33$,

MS_e = 3729.968] and no interaction of probability and SOA [F(8. 184) = 1.3, p < .248, MS_e = 3342.201]. (See Appendix M for mean reaction time for each level of probability and SOA for $Task_1$.)

For $Task_1$, no main effects in the error data were evident. (See Appendix M for mean error rates for each level of probability and SOA for $Task_1$.)

Overall accuracy on S_2-absent trials was 99.80% and the mean response time for S_1, when S_2 was absent, was 590 ms.

Insert Figure 16 about here

Analysis of $Task_2$ response time and accuracy

A similar analysis was performed on mean response times for $Task_2$, the outcome of which can be seen in Figure 16. Significant effects of probability [F(2, 46) = 34.2, p < .0001, MS_e = 9004.972], and SOA [F(4, 92) = 252.1, p < .0001, MS_e = 11619.403] were found, with no evidence of an interaction between these variables [F(8, 184) = 0.82, p < 0.583, MS_e = 3618.979]. Mean reaction time to the letter presented most frequently (probability 9) was significantly faster than response time to the lowest probability letter. Mean reaction time in the probability 4 condition was intermediate between the probability 1 and 9 conditions. Overall RT_2 decreased as SOA increased, being slowest at the shortest SOA and fastest at the longest SOA, although at 800 ms and 1200 ms SOAs, RT_2 was almost equivalent across the probability 4 and 9 conditions. (See Appendix N for mean reaction time for each level of probability and SOA for $Task_2$.)

Only a main effect of probability was evident when the mean error rates were analyzed [F(2, 46) = 11.12, p > .0001, MS_e = .012]. Highest accuracy was on trials that made up the probability 9 condition, followed by the probability 4 condition, and subjects were least

accurate in the probability 1 condition. (See Appendix N for mean error rates for each level of probability and SOA for $Task_2$.)

Analysis of S_2-not repeated and S_2-repeated trials

As shown in Figure 17, comparison of trials, in the probability 9 condition, on which the S_2 letter was not the same as the letter on the previous trial (S_2-not repeated), and trials on which the S_2 letter was the same (S_2-repeated), revealed, as in Experiment 3, that S_2-repeated trials were significantly faster than S_2-not repeated [$F(1, 23) = 17.3, p < .0004, MS_e = 5282.048$] and that a significant effect of SOA was present [$F(4, 92) = 309.9, p < .0001, MS_e = 6189.966$]. Overall performance was slowest at the shortest SOA and fastest at the longest SOA, and this pattern for both trial types was additive across SOAs [$F(4, 92) = 1.0, p > .40, MS_e = 2007.601$]. (See Appendix O for mean response times for S_2-not repeated and S_2-repeated trials for each SOA in the probability 9 condition.)

Insert Figure 17 about here

Analysis of S_2-not repeated

Effects of probability and SOA were calculated for trials on which the S_2 letter was not the same as presented on an immediately preceding trial.

Main effects of probability [$F(2, 46) = 25.7, p < .0001, MS_e = 9123.974$], and SOA [$F(4, 92) = 249.1, p < .0001, MS_e = 11257.436$] were found for these S_2-not repeated trials. The effect of probability was additive with SOA [$F(8, 184) = 0.8, p < 569, MS_e = 3631.794$], as can be seen in Figure 18. RT_2 was relatively equal for the 800 ms and 1200 ms SOAs across the probability 4 and 9 conditions. (See Appendix P for mean response times for each level of probability for each SOA.)

Insert Figure 18 about here

Discussion

In this experiment stimulus probability produced similar effects to those found in Experiment 3. Mean response time in $Task_2$ was shortest for the probability condition in which S_2, the letter, was presented most frequently, and slowest in the condition in which the letter was presented least frequently. Most importantly, effects of stimulus probability were additive with SOA showing that stimulus probability was having an effect at a stage of processing at, or beyond, the PRP bottleneck.

The 1200 ms SOA was added in this experiment to ascertain whether or not subjects reached asymptote in their performance on $Task_2$ at the 800 ms SOA. In the probability 4 and 9 conditions, response times were no different when SOA was 1200 ms than when SOA was 800 ms. The absence of any hint of a statistical interaction between probability and SOA suggests that the deviation from strict additivity in Experiment 3 was probably due to measurement error. The relatively flat RT_2s across the 800 and 1200 ms SOA conditions in Experiment 4 suggest that performance was near asymptote levels in the 800 ms condition. Thus, in Experiments 3 and 4, the absence of a significant interaction between probability and SOA could not have been due to a failure to test performance at an SOA producing near asymptote RT_2 as required by a postponement model of interference. The results of Experiment 4 converge nicely with those of Experiment 3 in showing the effects of probability of S_2 are additive with SOA.

In this experiment, as in the other experiments reported here, the effect of signal probability continued to be evident, and additive with SOA, when the S_2-not repeated trials were analyzed separately. The effect of signal probability for S_2-not repeated trials was similar to the effect found when all trials were included in the analysis, in that mean response time was

shortest for the most frequently presented S_2 letter (probability 9 condition) and longest for trials on which the least probable stimulus was presented (probability 1 condition).

The effect of stimulus repetition found in the present study can also be interpreted in the context of the locus of cognitive slack logic. Mean response time to a stimulus that was repeated on the previous trial was shorter than mean response time to a stimulus that was not repeated, with no interaction of stimulus repetition and SOA (F <1). As with stimulus probability, the effect of stimulus repetition was additive with SOA and, according to the locus of cognitive slack logic, must therefore be occurring at a stage of processing at, or beyond, the PRP bottleneck.

Therefore, like Experiment 3, we have identified two variables in this PRP experiment that show additive effects with SOA. Interpreting these effects using the locus of cognitive slack logic, leads us to conclude that the locus(i) of the effects of these variables is at, or later than, the locus of the PRP bottleneck. This information can be combined with the results from the AB experiments to help determine the locus of the AB effect. The contribution of the results from the AB and PRP experiments toward finding the locus of the AB effect will be discussed in more detail shortly.

Also in Experiment 4, as in Experiment 3, an effect of SOA on $Task_1$ response times was present. This effect was entirely due to the contribution of the two shortest SOAs (50 and 200 ms). There was no difference in RT_1 at longer SOAs (450/800/1200 ms), but at the shorter SOAs (50/200 ms) RT_1 increased as SOA decreased. As discussed under Experiment 3, one possibility for seeing effects of $Task_2$ variables on $Task_1$ is that subjects may group their S_1 and S_2 responses on some trials. For example, when the temporal interval between two stimuli requiring speeded responses is very short, subjects may hold off on responding to S_1 until they are also ready to respond to S_2. Under these conditions $Task_1$ response times could show effects of SOA like those found for $Task_2$. As stated already, the effects on RT_1 in this experiment, and in Experiment 3, are small, and do not warrant serious concern regarding the effects of primary interest in these studies, namely those associated with $Task_2$.

General Discussion

The research embodied within this thesis was designed to explore the locus(i) of a dual-task interference effect known as the Attentional Blink. The goal was to establish a locus of the AB effect, in relation to the PRP bottleneck, by performing manipulations in both paradigms (AB and PRP) and by using methods of analysis that provide constraints on the locus/loci of factor effects.

Relative signal probability produced the appropriate effects in both AB and PRP experimental designs. Probability of T_1 affected the size of the AB effect (Exps. 1 & 2). As the probability of presentation of a T_1 letter decreased, accuracy on identifying T_2 decreased, thereby increasing the size of the AB effect. In the PRP experiments (Exps. 3 & 4) a similar effect was reflected in reaction time to the second of two stimuli. As stimulus probability increased reaction time to S_2 decreased.

In the PRP experiments, the effect of varying the probability of S_2 was additive across SOA. According to the locus of cognitive slack logic (McCann & Johnston, 1992; Pashler, 1984; Pashler & Johnston, 1989) an additive pattern means that none of the effect of varying stimulus probability was absorbed in the period of cognitive slack, and that the effect therefore occurred at a stage of processing after the cognitive slack period.

The effect of relative signal probability was evident in the AB experiments, both when $Task_1$ was speeded and when it was unspeeded. The finding that the probability of T_1 affected the size of the AB indicates that the effect of probability must have occurred at, or prior to, the locus of the AB effect. If the locus of stimulus probability was subsequent to the locus of the AB effect then no such effect would have been seen.

The results of the PRP and AB experiments and the interpretation of the effect of stimulus probability in relation to the loci of the PRP and AB bottlenecks constrain a locus of the AB effect in relation to the PRP bottleneck. If stimulus probability is having an effect at, or beyond the PRP bottleneck, and at, or before the locus of the AB effect, then, at least one stage of processing involved in the AB effect must be located at, or beyond, the PRP bottleneck.

Locus(i) of the PRP Effect

Several studies have provided empirical evidence, by varying stimulus frequency, supporting a locus of the effect of frequency at, or after, a PRP response selection bottleneck. Pashler & Johsnton (1989) for example, using a PRP paradigm, found additive effects of stimulus repetition with SOA, underadditive effects of contrast with decreasing SOA, and no interaction between the two variables. These results show that the stages at which stimulus contrast and stimulus repetition have their effects are not one and the same. The former must be having an effect before the locus of the PRP bottleneck, whereas the effect of the latter must be at, or beyond, the bottleneck. These results suggest a late locus of the PRP bottleneck and stimulus repetition.

Manipulating a response variable, Van Selst and Jolicoeur (1997) found additive effects with SOA in a PRP experiment. The proportion of $Task_2$ go versus no/go trials was varied in this experiment so that, in one condition the proportion of go trials was 25% go and the proportion of no/go trials was 75%, and in another condition this relationship was reversed. Subjects were only to respond on the go trials. RT_2s were faster in the 75% go-trial condition than in the 25% go-trial condition, and this effect was additive with SOA. These results again support a late stage for the PRP bottleneck, and a locus of probability at, or beyond, this bottleneck.

Luck (in press), using electro-physiological data, determined the locus of the PRP bottleneck to be at a stage of processing after identification and categorization of a stimulus. In this typical PRP paradigm, probability of the second stimulus was varied and response time recorded. In addition, event-related potential (ERP) waves, that measured the amount of time to perceive and categorize a stimulus, were recorded for the second stimulus. The analysis of reaction time revealed a PRP effect. Response time to the second stimulus increased as SOA decreased and this effect was modified by stimulus frequency which was additive with SOA. However, there was no change in the ERP waves, indicating that the PRP bottleneck, as well as the locus of probability, must be located at a stage of processing subsequent to identification and categorization, possibly at response selection.

Locus(i) of the AB Effect

The evidence provided by these PRP studies is consistent with a late locus of the PRP bottleneck. Our research indicates that the locus of the AB effect is at, or after, the locus of this bottleneck. The suggestion of a late locus for the AB effect is in contrast to other theories that have attempted to provide an explanation for this interference effect. Most theories postulate an early locus for the effect, and certainly a locus separate from, and prior to, the locus of the PPR bottleneck.

The Attentional Gate Model of Raymond et al. (1992) proposes that the AB effect is caused by a gating mechanism that occurs prior to pattern recognition. Shapiro et al. (1994), in their Similarity Theory, suggest that the AB effect takes place at a stage of processing where items compete for entry and report from VSTM. The Attentional Dwell Model (Duncan, et al., 1994; Ward, et al., 1996) proposes that the interference effect is an indirect result of an inability to rapidly deploy attention to items presented in rapid succession. Attention is required to create object representations so that further processing may proceed and the AB effect occurs because capacity to encode items into a form suitable for further processing is limited. Likewise, the Two Stage Model (Chun & Potter, 1995) places the locus of the AB effect at a stage of processing that is required before representations can be processed further, namely the stage of short-term consolidation. Thus, all these theories imply an early locus for the AB effect, certainly a locus before response selection.

In contrast, the Central Interference Theory provides a theoretical framework for the AB effect that postulates a late locus of interference, possibly at response selection when $Task_1$ is speeded, and at short-term consolidation when $Task_1$ is not speeded. The theory proposes that certain stages of processing require central mechanisms, which are capacity limited and therefore, cannot occur in parallel. If central processors are occupied by $Task_1$, processing of $Task_2$ must be delayed. The Central Interference theory also proposes that more than one stage of processing is central, and that different stages are implicated in delaying $Task_2$, depending on whether the response to T_1 is speeded or unspeeded. When $Task_1$ is speeded, the stage of

response selection delays short-term consolidation of T_2, and when $Task_1$ is unspeeded, short-term consolidation of T_1 delays short-term consolidation of T_2.

Evidence supporting the theory that different stages of processing contribute to this interference effect is provided by the finding the magnitude of the AB effect increased as the number of response alternatives increased when $Task_1$ was speeded but not when it was unspeeded (Jolicoeur, 1999b). Also, the size of the AB effect has been found to be larger at the shorter T_1-T_1 lags when $Task_1$ is speeded than when it is unspeeded, but has the opposite effect at the later T_1-T_1 lags (Jolicoeur, 1999a). Together, these results suggest that different processes are at work when the response to T_1 is speeded and is made on-line, compared to when it is delayed.

RT_1, $Task_2$ accuracy, and the Central Interference Theory

Moreover, the Central Interference Theory of the AB effect is supported by the research presented in this thesis. The results from the AB experiment in which $Task_1$ was speeded (Exp. 1) can be used to test a prediction of the theory. According to the Central Interference Theory, the AB effect is caused by a delay in processing T_2 because central mechanisms are occupied by $Task_1$. The duration of the delay is related to the length of time central mechanisms are occupied, and the effect of this delay is reflected in $Task_2$ performance. When $Task_1$ is speeded, the stage responsible for the delay of $Task_2$ processing is believed to be response selection. Thus, according to the Central Interference Theory, a correlation between RT_1 and $Task_2$ performance should be evident when $Task_1$ is speeded. Long RT_1s should be associated with poorer performance on $Task_2$, and a large AB effect, and short RT_1s should correlate with better performance on $Task_1$, and a smaller AB effect. The results from Experiment 1 support this prediction. A correlation was observed between RT_1 and the size of the AB effect in that long RT_1s produced a larger AB effect than short RT_1s. This pattern was evident on a trial by trial basis and it was determined not to be a result of a general change in the pattern of performance as the experiment progressed.

An Alternate Account of Dual-Task Interference Effects

Although a response selection processing bottleneck is the most prevalent account of dual-task interference effects, another account of the fundamental mechanism underlying dual-task interference has been proposed relatively recently by Meyer and Kieras (1997a) in their computational theory of concurrent performance on cognitive tasks. The theory, Executive-Process Interactive Control (EPIC), posits that bottleneck-like effects, as found in dual-task experiments, are not induced by a structurally permanent central bottleneck, but rather, are a consequence of flexible strategies employed by individuals in order that they conform to the experimental instructions used in PRP studies. In PRP experiments subjects are instructed to respond to a primary stimulus prior to responding to a second stimulus. For example, in the PRP experiments reported here, subjects were to make their response to the first stimulus, the tone, prior to making their response to the letter. According to Meyer and Kieras (1997a), the PRP effects observed in these experiments are a result of deferring the production of a response to the letter rather than an inability to select responses for two tasks simultaneously. This response deferment strategy is flexible, implemented on a temporary basis, and does not resemble the permanent central bottleneck proposed by the postponement account of dual-task interference (McCann & Johnson, 1992, Pashler, 1989). The theoretical explanation of dual-task interference provided by EPIC is based on the application of production rules that specify that motor execution of the response associated with the first task must have progressed sufficiently before production of the response for the second task can ensue.

This computational theory (Meyers & Kieras, 1997a) is built on a production-system, called the Parsimonious Production System (PPS), made up of production rules and a production rule interpreter. The production rules and interpreter are contained within procedural memory. Actions are executed whenever the contents of working memory (i.e. symbolic information, such as goals, steps and notes used to test and apply the procedural rules), and the conditions of a production rule are satisfied. The PPS allows for parallel processing in that several production rules can be assessed by the interpreter at one time, and actions can be

executed together as long as they do not conflict with each other. In essence, processing necessary for more than one task can occur in parallel.

Stimulus input is received as sensory information by visual, auditory, and tactile perceptual processors which, in turn, send output to working memory indicating that the stimulus has been detected and providing feature information such as colour, size, loudness, etc.. From the contents of working memory, and the operation of the PPS production rule interpreter, a cognitive processor selects symbolic information about the response (e.g., left hand - index finger) and sends relevant information to vocal and/or manual motor processors. The motor processors prepare and execute responses. There is also an ocular motor processor which determines what information to send to the visual perceptual processor depending on the spatial position of the eyes. The preparation of movement features by the motor processors occurs prior to the initiation and execution of the response movement. Movement features are those characteristics of the response that are specific to the response modality, such as the hand and finger that are needed to make a response. The preparation of movement is a process that transforms the symbolic representation of a response to an output command that controls effectors. Features are processed serially and the time from onset of preparation to the start of an overt response depends on the number of response features that need to be considered during the transformation process. Thus, the number of response alternatives will affect the time to prepare a response. According to the theory, each hand is not controlled by a separate motor processor. The manual motor processor takes care of movement production for both hands and, consequently, interference can occur when more than one response needs to be made, either with different hands, or with the same hand.

Although the cognitive processor has unlimited capacity and can handle several sets of production rules in parallel, the processes performed by the production rules must be coordinated. When there is more than one task, processes necessary to perform the tasks can occur simultaneously but in order to ensure that the tasks are performed properly, and in the correct order, an executive controller is needed. In addition, the controller must monitor progress on each task, update working memory, and ensure that there is no attempt to use the

same effectors at the same time. Supervisory control is achieved by executive processes that use production rules separate from those associated with individual tasks.

To explain performance for the PRP procedure Meyer and Kieras (1997a) provide a specific model called the Strategic Response-Deferment Model (SRD). In this model there is no 'wait' period for response selection of $Task_2$ because the cognitive processor can test and apply production rules to more than one task in parallel. Response to $Task_1$ occurs immediately because participants in a PRP experiment are instructed that their primary response is to the first stimulus (i.e. $Task_1$). However, at short SOA a strategic deferment of the $Task_2$ response is applied in which the response for $Task_2$ is stored temporarily in working memory in order that the response order of the PRP paradigm can be adhered to (i.e. $Task_1$ response before $Task_2$ response). Response deferment is controlled by an executive process. After sufficient progress on the $Task_1$ response has occurred the response for $Task_2$ is released from working memory and sent to the motor processor for output.

In PRP experiments subjects are instructed to make their response to both stimuli as quickly as possible, and to make the $Task_1$ response prior to the $Task_2$ response. Meyer and Kieras (1997) propose that the capacity for cognitive processing is unlimited and that interference observed in these experiments is not caused by a permanent processing bottleneck at response selection (or anywhere else for that matter). Instead, it is due to the application of a flexible strategy that allows for the $Task_2$ response to be deferred. This strategy is adopted when processing of $Task_1$ and $Task_2$ occur in parallel so that a response to the first stimulus can be made before a response to the second stimulus. In addition, the deferred strategy ensures that there is no conflict over use of the motor processor.

Although central processes like stimulus identification, response selection, and movement production can temporally overlap, peripheral processes that are associated with the manual, vocal, and occular motor processors, are capacity limited. For example, at the level of stimulus input, the eyes cannot be directed toward two stimuli simultaneously, and, at response output, the hands cannot make responses to more than one stimulus at a time. Central

processors allocate peripheral resources efficiently so that there is no attempt to use them concurrently for different tasks which might result in confusion and attenuate performance.

What are the implications of the EPIC theory for the AB interference effect? The motivation behind using flexible strategies and executive processes is based on the necessity to respond to one task prior to another under conditions where, if the mechanism allowed, the $Task_2$ response could occur before the $Task_1$ response, or, the response for both tasks could occur simultaneously. If this is true, one would expect to find no evidence of interference when the strategy is not implemented, as when one, or both tasks do not require a speeded response. Under these conditions, when a response is not speeded, there is no pressure to assure that the $Task_1$ response is made before the $Task_2$ response. Therefore, there should be no need to employ a strategy that defers the $Task_2$ response and, furthermore, there should be no interference effect. In the AB experiments reported here (Exps. 1-2), the response to $Task_2$ is always unspeeded and subjects are free to take as much time as they wish to make their response at the end of the trial. In the second of the AB experiments (Exp. 2), both responses are unspeeded. Yet, in both experiments, where one would expect no need of deferring the $Task_2$ response, and thus no effect, interference effects are clearly apparent.

Rather than relying on the use of flexible strategies that result in deferring the $Task_2$ response, the EPIC theory posits that evidence of bottleneck-like effects in the PRP literature can sometimes be accounted for by taking into account capacity limitations on peripheral processing that occurs prior to, or after, central processing. For example, Meyer and Kieras (1997b) account for the additive effects of varying stimulus compatibility, found by McCann and Johnston (1992), by considering the influence of the capacity-limited occular motor processor. $Task_1$ in the study conducted by McCann and Johnston (Exp. 1, 1992), was a tone discrimination task to which subjects made a vocal response. $Task_2$ was a visual shape discrimination task that required a manual response. Stimulus compatibility was varied so that on some trials subjects used a highly compatible stimulus-response (S-R) mapping to make their response, while on other trials the S-R mapping was not compatible. The selection of a response is believed to be more difficult when S-R compatibility is low, thus affecting the

response selection stage of processing. In this study, a diagram was provided just below the computer screen to aid subjects in remembering the correct mapping. Meyer and Kieras (1997b) believe that the additive effects observed in this experiment are not caused by a structural processing bottleneck at response selection but are due to the effect of long occular orientation time - that is, the time taken to redirect the eyes from the S-R mapping diagram to fixation of the $Task_2$ stimulus location after the start of the trial. In order to remember the incompatible S-R mapping, subjects would presumably check the diagram frequently during the course of the experiment. Therefore, they may have been relatively slow in redirecting their eyes to the $Task_2$ stimulus location at the beginning of a trial. According to the EPIC theory, it is this long occular movement time that is reflected in additivity of RT_2 with SOAs, not a consistent 'wait' period during which $Task_2$ processing is delayed until $Task_1$ processing is completed, which is the account provided by the postponement theory of dual-task interference.

However, if this account is correct, it is not clear why the effect of S-R compatibility is not underadditive with decreasing SOA, since the locus of the effect is at an early stage of processing, prior to a stage at which a response might be deferred, i.e. the PRP bottleneck.

If an early peripheral limitation were applied to the processes involved in the AB effect it might take the form of interference in a translation process that occurs prior to response selection. Such a process might consist of translating T_1 and T_2 from visual representations to some other form of representation. This process would require verbalizing the stimulus, but, due to the limited capacity of the articulatory motor processor, verbalizing one stimulus cannot occur while verbalizing another stimulus. Thus, in the AB experiments (Experiments 1 & 2), it would not be possible to 'say' the letter 'H' (a $Task_1$ stimulus) while 'saying' the letter 'Y' (a $Task_2$ stimulus). The outcome of this interference, which occurs at an early stage of processing prior to the unlimited capacity of central processing, would be observed as the AB effect.

If this account of interference is correct, one would expect the same explanation to apply to the PRP effect. The AB experiments are similar to the PRP experiments in that stimuli are presented in close succession to each other. In addition, the response to T_1, at least in one

of the AB experiments, is speeded. Speeded responses are also required in the PRP experiments. If a process of translation is applied to stimuli in one experimental paradigm there appears to be no reason to assume that the same process would not be applied to stimuli used in the other paradigm. Either, T_1 and T_2 in the AB experiments undergo a process of translation and a similar process is applied to S_1 and S_2 in the PRP experiments, or, stimuli are not subjected to this translation process, in which case there should be no evidence of interference in either the AB or PRP experiments. It could be argued that the AB and PRP experiments reported here are not the same because in the former, T_1 and T_2 were presented in the same modality (visual) whereas in the latter, S_1 and S_2 were presented in different modalities (auditory/visual). Consequently, they may show differential effects of perceptual interference. However, Jolicoeur (1999c), showed similar AB effects to those found here, in a study where T_1 was a tone and T_2 a visual letter. Taking in to account the similarities between the AB and PRP experiments, and the comparative AB effects found in uni-modal and cross-modal designs, it seems safe to assume that, if the process of translation takes place, it is common to all target stimuli despite the modality of T_1 and T_2, and regardless of whether the experimental paradigm is one designed to exhibit an AB effect or a PRP effect.

In the PRP experiments (Experiments 3 & 4) the first task was a tone discrimination task and the second task a letter discrimination task. Thus, according to the EPIC theory, interference would occur because it would be impossible to 'say' the tone presented (e.g., 'high') while 'saying' the letter (e.g., 'H'). As was the case with the McCann and Johnston (1992) results, problematic to the interpretation of an early locus of interference provided by the EPIC theory is the observation of additive effects with SOA in the PRP experiments. According to an early peripheral capacity limitation account of interference we should have found underadditive effects with decreasing SOA in the PRP experiments because the interference effect is occurring early on in processing, prior to the PRP bottleneck. There was no hint of an interaction of probability with SOA in these experiments. Thus, the capacity limitation of early peripheral processing proposed by the EPIC theory is not supported by the results observed in these PRP or AB experiments.

Another possibility exists with respect to peripheral interference. The EPIC theory might propose that the AB effect is a consequence of interference late in processing, where a response is translated to a motor code, a code that consists of manual motor features (e.g., button-press). In the experiments reported here, the response modality was the same for each task, namely manual button-press. Consequently, the potential exists for interference in determining the features of responses, i.e. press a key. Supposedly, one cannot produce the appropriate 'press a button' code for one response while concurrently producing a similar code for another response. This account of dual-task interference is unlikely to be correct, however, because the nature of $Task_2$ in the AB experiments, eliminates the need for concurrent processing at this level of response output. As already discussed, the response to $Task_2$ in the AB experiments is always delayed until the end of a trial, and in Experiment 2, $Task_1$ is also delayed. Therefore, there should be no conflict in the process of translating response information to a motor code, as the processing necessary for each task can occur independently.

The issue of both early and late peripheral interference is addressed by the following results, demonstrated by Jolicoeur (Exp. 5, 1998d). With respect to an early locus of peripheral interference in which stimulus information cannot be transformed to articulatory codes in parallel, the stimuli in this study had no names and were therefore not easily codable in a verbal format. As far as the late locus of interference, i.e. translation into motor codes, the motor code for one of the responses in this experiment could not be determined until the end of the trial because the stimulus information was not provided until that time. Hence, the translation process for $Task_2$ was not postponed by the same process for $Task_1$.

The first task in this study was a speeded four-alternative discrimination response to an auditory tone. A random polygon was presented at variable SOA after the tone and was followed by a pattern mask. At the end of each trial, after the response to the tone, two perturbed polygons were presented. One was an exact match to the first polygon, the other was randomly perturbed. The second task was to identify which of these polygons matched the polygon initially presented by selecting the polygon that matched (a left / right response). Both

responses were made using the keyboard. The response to the tone was speeded but the response to the polygon was not speeded.

The results showed that identifying the polygon became more difficult as SOA decreased. Both the early and late accounts of peripheral interference supplied by the EPIC theory are difficult to apply as sources of interference in this experiment. First, random polygons were used as stimuli and were not easily namable, thereby providing no means of translating stimulus information into an articulatory code. In addition, the position of the polygons shown at the end of the trial could not be detected in advance, so the motor response could not be computed until the polygon appeared at the end of the trial. The design of this study, and the forthcoming results, do not support the hypothesis of an early peripheral locus of dual-task interference. Likewise, a late account of interference is not supported because the information regarding the response for $Task_2$ was not provided until the response to $Task_1$ was complete. Consequently, the condition necessary to produce interference in transforming information to a motor response code did not exist.

All in all, the results from the AB and PRP experiments reported in this thesis do not appear to be well accounted for by the EPIC theory of multiple task performance (Meyer & Kieras, 1997a; 1997b). In fact, some of the evidence provided by these results provides potential problems for the theory as it currently stands.

Locus(i) of Signal Probability

Stimulus probability has not previously been used in experiments designed to exhibit AB effects. In this thesis this variable is used in a novel way to modulate the size of the AB, as well as show effects in PRP experiments. The interpretation of the effects of stimulus probability found in the experiments reported here in relationship to the locus(i) of AB and PRP effects provides a contribution to our knowledge of the nature of signal probability.

The locus(i) of stimulus probability has not been clearly determined. Miller and Hardzinski (1981) propose that the effect of stimulus probability is to facilitate the transmission of feature information extracted from the physical representation of a stimulus to the name, or

abstract code, associated with that stimulus. They claim it is neither the sole process of extracting the stimulus information nor activation of the name code, per se, that is affected by probability, but rather, the activation of the specific route between stimulus feature information and the appropriate stimulus code. Routes that are more likely to be activated, because of high probability, develop a lower threshold of activation. Hence, a specific route that is highly probable can be activated more quickly than a route that is less probable.

If, as the data suggests, the effect of stimulus probability is occurring at, or after, the PRP bottleneck, the possibility exists that probability and the PRP bottleneck share the same locus, namely the stage of processing at which an abstract code is activated. It is possible that feature extraction and name code generation are substages of encoding. The activation of an abstract code may be the final process in encoding, immediately preceding response selection. Alternatively the stage at which a name or abstract code is generated might constitute an initial substage of response selection. If we infer that the loci of probability and the PRP bottleneck are common, and that code generation is an initial substage of response selection, this would certainly be in accordance with the findings from the PRP literature proposing the existence of a PRP bottleneck at response selection (McCann & Johnston, 1992; Pashler, 1994; Van Selst & Jolicoeur, 1997).

The modulating effect of stimulus probability on the AB effect provides evidence that the locus of the probability effect is at, or before, the locus of the AB effect. Thus, both these effects could share a common locus. It is not inconceivable to suggest that stimulus probability, the PRP bottleneck, and at least some of the AB effect (there may be more than one locus), all share a common locus and that this locus is at a stage of processing at which a name or abstract code for a stimulus is activated.

Locus(i) of Repetition Effects

In addition to the effects found with stimulus probability, stimulus repetition, another variable manipulated within the experimental design, was investigated. Trials on which the target stimulus (T_1 in the AB and S_2 in the PRP experiments) was not the same as the target

stimulus presented on the immediately preceding trial were compared to trials on which the stimulus was the same. Stimulus repetition modulated the size of the AB effect. Repeating a target stimulus produced a smaller AB effect than presenting a different target stimulus. In the PRP experiments, repeating a target stimulus produced faster responses than a different target, and the effect of stimulus repetition was additive with SOA (as also found by Pashler & Johnston, 1989).

The finding that stimuli that were repeated on an immediately preceding trial were responded to faster and more accurately than stimuli that were not repetitions is not unusual. This effect was first reported by Hyman (1953) and subsequently termed the repetition effect by Bertelson (1961). The task of determining the locus of the repetition effect yields the same problem encountered when investigating the locus of signal probability, that being that stimulus repetition is confounded by response repetition. Early research attempted to distinguish a perceptual locus from a response locus by mapping several stimuli on to one response, or one stimulus to more than one response. In this way, as with signal probability, the effects of repeating the stimulus or the response could be evaluated independently. If same-response trials showed a repetition advantage it was generally assumed that the effect was occurring at the level of the response, either response selection or execution, whereas if the RT advantage was observed only when the stimulus was repeated, the effect was assumed to be occurring at a perceptual level.

Similar to the early research in signal probability, initial studies investigating the repetition effect did not yield consistent results. Bertelson (1965) coupled two even numbered digits (2 and 4) with one response and two odd digits (5 and 7) with another response and found different stimulus/same-response trials to be faster than different-stimulus/different-response trials and only slightly slower than trials on which both the stimulus and the response had been repeated. In contrast, Smith (1968) found a greater effect of repetition on different-stimulus/different-response trials compared to trials on which the stimulus was different but the response was the same. In this study, stimulus characteristics of colour and digit were varied and subjects were instructed to press one response key if they saw a Red 1 or a Green 2 and to

use another response key if they saw a Green 1 or a Red 2. Response times were fastest when the stimulus and the response were the same as those on the immediately preceding trial, whereas trials on which only the response was repeated (different stimulus/same response) were slowest. Intermediate were those trials on which neither the stimulus nor the response were the same.

The results from these studies are contradictory with respect to the locus of the repetition effect. Bertelson's work implies that the response is the determinant of the repetition effect because the greatest difference in the size of the effect lay between the different stimulus/same response and different stimulus/different response conditions. Thus, in this study the effect of repetition appeared to be a function of repeating the response rather than repeating the stimulus. On the other hand, Smith (1968) provided evidence against the response as the sole determinant of the effect, and her results point instead to the significance of the stimulus-response pairing. Responses were faster on trials where both stimulus and response were repeated than on trials on which the response was repeated but the stimulus was different. Thus, repeating a response in this study had little effect compared to repeating a stimulus and a response.

Earlier work by Bertelson (1963) also suggested that the response component in these types of tasks is a major contributor to the repetition effect. Using a 1 : 1 S-R mapping, Bertelson found an interaction of S-R compatibility with repetition, thereby implicating response selection as a locus of the repetition effect. The effect of decreasing S-R compatibility was greater on non-repeated trials than on repeated trials. As S-R compatibility increased, the difference between repeated and non-repeated trials increased. Additional evidence in favour of a late locus for the effect of repetition comes from an absence of interactive effects found between repetition and stimulus intensity (Hansen & Well, 1984; Pashler & Johnston, 1989). These results are consistent with a locus of stimulus repetition occurring at a stage of processing that is after the stage affected by stimulus intensity, but at, or before, the stage at which S-R compatibility has its effect. Pashler and Johnston (1989) examined the effects of stimulus intensity and repetition of S_2 in a PRP experiment and found additive effects of

stimulus repetition and SOA. $Task_1$ was a two-alternative discrimination response to an auditory tone (low/high). $Task_2$ was a three-alternative discrimination response to one of three letters (A, B, or C). Stimulus intensity was found to be underadditive with decreasing SOA, while stimulus repetition was additive. According to the locus of cognitive slack logic (McCann & Johnston, 1992; Pashler, 1984; Pashler & Johnston, 1989), the effect of stimulus repetition in this experiment was occurring at, or after, the locus of the PRP effect. These results are again consistent with a locus of stimulus repetition at response selection.

Pashler and Baylis (1991) demonstrated the repetition effect in several experiments by showing that same stimulus/same response trials produced significantly faster responses than different stimulus/different response trials. They also observed a moderate benefit on different stimulus/same response trials when stimuli that shared the same response were members of a common conceptual category (e.g., letters or digits), compared to when they were not easily categorizable. For example, when the stimuli 4 and P were coupled with response 1, and stimuli 2 and V with response 2, trials on which the response was the same, but the stimulus was different, were responded to more slowly than trials on which both stimulus and response were different, or stimulus and response were the same. Thus, there was no benefit to repeating the response but not the stimulus. When the digits 2 and 4 were coupled with response 1, and the letters P and V with response 2, a small benefit of different stimulus/same response trials over different stimulus/different response trials was found. In the latter experiment, the stimuli associated with each individual response could easily be classified as members of a conceptual category (i.e., digits or letters), whereas in the former experiment this task would have been much more difficult (4 and P/2 and V). Pashler and Baylis concluded from these experiments (Exps. 1 - 3, 1991) that the effect of repetition is stimulus specific but the degree to which stimuli can be grouped into categories, such as digits versus letters, affects the size of the repetition effect when a response, but not a stimulus, is repeated. The effect of repeating the response, in their experiment, was seen as a decrease in response time, and appeared to be a function of the ability to classify same-response stimuli into members of a common group.

Although they found that categorization produced a moderate effect of response repetition, Pashler and Baylis (1991) noted that the greatest benefit to repeating a response was observed when stimuli were varied on an attribute that was not only incidental to performing the task, but was also perceptually separable from the task-relevant factor. When letters of the same name but different case (e.g., G and g) were coupled with one response, only a modest effect of repeating the response was noted (Exp. 4, 1991). These results again suggest that the repetition effect is highly stimulus specific because little benefit to repeating a response was found, even though the stimulus was varied on an attribute that was not relevant to performing the task (i.e., case). However, when letters were identical in name and case, and were varied on the basis of colour (e.g., Red G and Green G = response1; Red A and Green A = response2), a large effect of repeating the response was observed (Exp. 5, 1991). In this experiment, as in the previous one, stimuli coupled to the same response were varied along an attribute that was irrelevant to performing the task. The additional factor in this experiment was that the stimulus variation was perceptually separable from the dimensions of the stimulus that were needed to perform the task (i.e., same name/different colour) (Pashler & Baylis, 1991).

The majority of work investigating the effects of stimulus repetition, particularly the more recent research, appears to be consistent with the theory of a locus at a late stage of processing associated with the response to the stimulus, rather than early perceptual processing of the stimulus itself (Hansen & Wells, 1984; Pashler & Johnston, 1989). However, the results reported by Pashler and Baylis (1991) suggest that the repetition effect is stimulus specific. This observation links the effect of repetition to the perceptual characteristics of the stimulus and stimulus identification, which in turn suggests an early locus for the effect.

Further work by Pashler and Baylis (Exp. 6, 1991) showed that the effect of repetition disappeared under conditions where a stimulus was paired with more than one mode of response. In this experiment there were two response conditions, manual and manual/vocal, coupled with each stimulus so that one stimulus was paired with more than one response. In the manual condition subjects pressed one key to respond to two stimuli (e.g., 4 and P = response 1; 2 and V = response 2; 8 and K = response 3), and in the manual/vocal condition

they alternated between a manual response on odd numbered trials, and vocally naming the stimulus on even numbered trials. As expected, an effect of repetition was found in the manual condition. The question of interest was whether this effect would continue to occur when the stimulus was repeated, and a vocal response preceded a manual response in the manual/vocal condition. If the effect of repetition persisted across these alternating response modes, evidence would exist for a perceptual locus. If the effect disappeared, the locus of the repetition effect could be determined to be at a response stage of processing. No effect of repetition was found in the manual/vocal condition, meaning that the effect of repetition did not transfer from one response mode to another, and thereby providing further evidence in support of a response selection locus. As well as being stimulus specific, the repetition effect appears to be fairly response specific (Pashler & Baylis , Exp. 7, 1991). Pashler and Baylis proposed that the effect is caused by an increase in the strength of a link that is generated between early formed representation of the stimulus and a specific response. This pathway is a response selection short-cut and it allows for the elimination of the process of selecting a response on trials on which the stimulus is repeated.

Ells and Gotts (1977) also investigated the repetition effect under conditions where a stimulus was paired with more than one response. They found that stimulus repetition contributed more than response repetition in a simple task, but that response repetition was the greater contributor when the difficulty of the task was increased. In their first experiment subjects were assigned two target digits, and another digit was designated as a non-target. Digits could appear in any one of two locations (left or right) and subjects were to respond to the location by moving a toggle switch in the direction of the location in which the digit had appeared. Subjects were only to respond as quickly as they could to targets, not non-targets. Non-targets remained on the screen for 1500 ms, at which time the next stimulus appeared. In this experiment there were four possible stimulus-response conditions -- 1) same stimulus/ same response 2) same stimulus/different response 3) different stimulus/same response and 4) different stimulus/different response. Reaction times were faster on same stimulus/different response trials, and same stimulus/same response trials, than on different stimulus/same

response trials. Therefore, more effect of repetition was found when the stimulus was repeated compared to when the response, or neither the stimulus nor the response, were repeated. When the task was modified to be more difficult, by increasing the number of locations to three and increasing the target set size to three, and the non-target set size to 5, it was found that response repetition was more of a contributing factor than stimulus repetition. In a third experiment, in which S-R pairings were incompatible, more contribution from repeating a stimulus than repeating a response was evident. Therefore, in Experiments 1 and 3 a facilitory effect of stimulus repetition was seen, whereas in Experiment 2 a greater contribution was made by repeating the response. The authors suggested that subjects may adopt different strategies that vary the weight of the stimulus and the response depending on the task at hand. They also suggested that the effect of repeating a stimulus in this experiment was due to an decrease in the time to match a stimulus representation to items within a memory array when the stimulus was repeated. The effect of repeating a response was a facilitation of response selection processes.

All in all, stimulus repetition is similar to signal probability in that the effect is not completely understood and the locus(i), has not been empirically determined. The data from the AB and PRP experiments reported here may shed some light on this issue. Stimulus repetition can be interpreted in a similar manner as the probability data. In speeded and unspeeded T_1 versions of the AB experiment, stimulus repetition affected the magnitude of the AB, indicating that the effect of this variable manipulation must be occurring at, or before, the locus of the AB effect. The additivity of stimulus repetition and SOA, exhibited in the PRP experiments, indicates that, in relation to the locus of the PRP bottleneck, the effect of stimulus repetition is either occurring at the same location as the bottleneck, or after it. By combining the information from the AB and PRP experiments, regarding the locus of the repetition effect, it can be further established that the locus of the AB effect is occurring at, or after, the PRP bottleneck.

Thus, we have two variables, stimulus probability and stimulus repetition, that have provided information to localize the AB effect in relation to the PRP bottleneck. What does this mean in terms of the processing stages implicated in performing the tasks contained within an AB experimental paradigm? What we know about the locus of the PRP bottleneck, as well as

the locus(i) of the effects of signal probability and stimulus repetition, will allow us to determine more precisely the stage at which the AB effect occurs. Evidence from the PRP literature strongly implies that the PRP bottleneck is at, or before, the stage of response selection. The bottleneck is unlikely to be located at an early stage of processing because stimulus quality is typically seen as underadditive with decreasing SOA (De Jong, 1993; Pashler, 1984, Pashler & Johnston, 1989), meaning that the effect of stimulus quality occurs prior to the bottleneck. Evidence also suggests that, although there may be multiple loci for the effect of signal probability, one locus is at a point in the processing sequence where a physical representation of a stimulus is transformed to an abstract code of some sort. The effect of stimulus probability is not seen when a name or abstract code does not need to be generated, such as in a task where stimuli are matched on the basis of physical identity (Pachella & Miller, Exp. 1, 1976). Additional information as to the locus of the probability effect comes from the observation that stimulus quality and stimulus probability sometimes interact, indicating that these effects can share a common locus, perhaps at a final substage of encoding. However, the effects of stimulus quality and probability do not always interact (Miller, Exp. 3, 1979). The suggestion has been made that this interaction will only be evident when a name or abstract code is activated and that, if these variables do not interact, no such process has occurred (Miller, 1979). This is likely true. In experiments where no interaction was found it was often conceivable that the use of a name code would have been of no benefit to performing the task (Miller, Exp. 3, 1979; Miller & Pachella, Exp. 2, 1976; Pachella & Miller, Exp. 2, !976). However, there are studies in which stimulus quality and probability do not interact and yet the use of a name code would seem to be relevant to performing the task. One example would be a task in which stimuli were to be judged the same if they possessed the same name but were of different case (Pachella & Miller, Exp. 1, 1976). In addition, the effects of probability continue to be evident, independent of interactive effects with stimulus quality. The evidence suggests that these variables, stimulus quality and stimulus probability, are sometimes having their effects at different stages of processing, and implies more than one locus for the probability effect. Miller (1979) proposed that, when probability effects are additive with the effects of

stimulus quality, subjects have activated a pathway that, in essence, is a short-cut, visuo-motor connection, linking stimulus and response information. He suggested that this process occurs in the absence of name or abstract code activation. Adding to this view, one proposition might be that stimulus quality interacts with probability when a name code is used, but not when response information is also represented in the code. It is possible that stimulus as well as response information might be contained within a single code when an abstract code is generated for the sole purpose of performing an experimental task. The underadditive effects observed when stimulus quality alone is varied (Pashler & Johnston, 1989) indicate that the locus of this effect is early on in processing, likely at stimulus encoding. If signal probability and stimulus quality only interact when a name code is generated, it may be that name code generation, and activation, is at a final substage of encoding, immediately prior to response selection, while activation of an abstract code, that may includes stimulus and response information, occurs later in the series of processing stages, possibly at a substage of response selection.

The empirical findings regarding the effects of signal probability suggest that there is more than one locus for the effects of this variable. Possibly the type of task, and the nature of the stimulus, determine the locus. Based on the research reported here, the effects of stimulus probability appear to occur at a final substage of encoding where a name code is generated, or at an early substage of response selection, or both. Research investigating the effects of stimulus repetition is complex but implicates one locus at the stage of response selection. However, it may be that, although a greater effect is obtained by repeating a response than a stimulus, repeating a S-R code also produces an effect of repetition.

Conclusion

With respect to the locus of the AB effect we have already determined that this interference effect is occurring at, or after, the PRP bottleneck, and that the effects of stimulus probability and repetition take place at, or before, the AB locus. The possibility exists that the PRP bottleneck, the AB effect, and the effects of stimulus probability and stimulus repetition,

at least sometimes share the same locus. An alternate proposal is that the locus of the AB effect is at some late stage of processing, beyond the effect of stimulus probability and stimulus repetition, and the PRP bottleneck.

Bear in mind that the sequence of processing stages is likely not the same when $Task_1$ is speeded as when it is unspeeded. Consequently, the implications for a locus of the AB may not be the same under both $Task_1$ conditions. If we take into account the locus(i) of stimulus probability, stimulus repetition, and the PRP bottleneck, in relation to the locus of the AB, we are looking for a stage of processing that is at, or after, the effects of stimulus probability and stimulus repetition, and at, or after, response selection, the stage implicated as the locus of the PRP bottleneck. A likely candidate for the AB effect then, when $Task_1$ is speeded, is the stage of response selection. If, in the $Task_1$ speeded version of the AB paradigm, the locus of the AB effect is at response selection, it is unlikely that the same processing stage is implicated as the locus of interference when response selection is delayed until the end of a trial. It is possible that, under $Task_1$ unspeeded conditions, the AB effect is occurring at the stage of short-term consolidation.

In summary, the research presented in this thesis contributes to our knowledge of the nature of the dual-task interference effect known as the Attentional Blink. The effects of two variables, stimulus probability and stimulus repetition, were found to be additive with SOA in PRP experiments indicating that these effects occurred at a stage of processing common to the PRP bottleneck, or beyond it. These same variables modulated the size of the AB effect, thus providing evidence that their locus(i) is at, or before, a locus of the AB effect. From these results, we can conclude that at least part of the AB effect is occurring at, or after, the PRP bottleneck. The most likely candidates are the stages of response selection, when $Task_1$ is speeded, and short-term consolidation, when the response to $Task_1$ is delayed. In addition, the work presented here provides support for the Central Interference Theory by showing that, in the AB $Task_1$ speeded experiment, response times to $Task_1$ (i.e. short/long) correlated with the size of the AB effect.

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Figure Captions

Figure 1. Schematic representation of the effects of manipulating a $Task_2$ variable that has a locus at, or after, the PRP bottleneck. The effect of varying the duration of a stage of processing at, or before, the bottleneck will be reflected in mean $Task_2$ response time.

Figure 2. Hypothetical data representing the manipulation of a $Task_2$ variable that displays additive effects with SOA. The difference between the levels of the $Task_2$ variable manipulation is relatively equal across SOAs.

Figure 3. Schematic representation of the effects of manipulating a $Task_2$ variable that has a locus before the PRP bottleneck. The effect of varying the duration of a stage of processing prior to the bottleneck will be absorbed in the period of cognitive slack, and will not be seen in mean $Task_2$ response time.

Figure 4. Hypothetical data representing the manipulation of a $Task_2$ variable that displays underadditive effects with decreasing SOA. The difference between the levels of the $Task_2$ variable manipulation decreases as SOA decreases.

Figure 5. Hypothetical data representing the manipulation of a $Task_2$ variable that displays overadditive effects with decreasing SOA. The difference between the levels of the $Task_2$ variable manipulation increases as SOA decreases.

Figure 6. Experiment 1 - mean $Task_2$ accuracy for T_1-present and T_1-absent trials, as a function of T_1-T_2 lag. The effects of the probability (1 : 4: 9) of T_1 as a function of T_1-T_2 lag are shown.

Figure 7. Experiment 1 - mean $Task_2$ accuracy as a function of T_1-T_2 lag, for T_1-present trials in the probability 9 condition, where T_1 was not repeated, and T_1 was repeated. That is, trials

on which the T_1 letter was not the same as the T_1 letter on the immediately preceding trial, and trials on which the T_1 letter was the same as on the previous trial.

Figure 8. Experiment 1 - mean $Task_2$ accuracy for T_1-present trials on which the T_1 letter was not repeated, that is, was not the same as the T_1 letter on the immediately preceding trial. The effects of the probability (1: 4: 9) of T_1 as a function of T_1-T_2 lag are shown .

Figure 9. Experiment 1 - mean $Task_2$ accuracy depending on short or long response times in $Task_1$ (based on a median split of T_1-present trials only) as a function of T_1-T_2 lag.

Figure 10. Experiment 2 - mean $Task_2$ accuracy for T_1-present and T_1-absent trials, as a function of T_1-T_2 lag. The effects of the probability (1 : 4: 9) as a function of T_1 T_1-T_2 lag are shown.

Figure 11. Experiment 2 - mean $Task_2$ accuracy for T_1-present trials in the probability 9 condition as a function of T_1-T_2 lag, where T_1 was not repeated, and T_1 was repeated. That is, trials on which the T_1 letter was not the same as the T_1 letter on the immediately preceding trial, and trials on which the T_1 letter was the same as on the previous trial.

Figure 12. Experiment 2 - mean $Task_2$ accuracy for T_1-present trials on which the T_1 letter was not repeated, that is, was not the same as the T_1 letter on the immediately preceding trial. The effects of the probability (1: 4: 9) of T_1 as a function of T_1-T_2 lag are shown .

Figure 13. Experiment 3 - mean reaction time (ms) for $Task_1$ (dashed lines), and $Task_2$ (solid lines), as a function of SOA. The effects of probability (1: 4: 9) of S_2 as a function of SOA are shown.

Figure 14. Experiment 3 - mean $Task_2$ reaction time (ms) for trials in the probability 9 condition as a function of SOA, where S_2 was not repeated, and where S_2 was repeated. That is, trials on which the S_2 letter was not the same as the S_2 letter on the immediately preceding trial, and trials on which the S_2 letter was the same as on the previous trial.

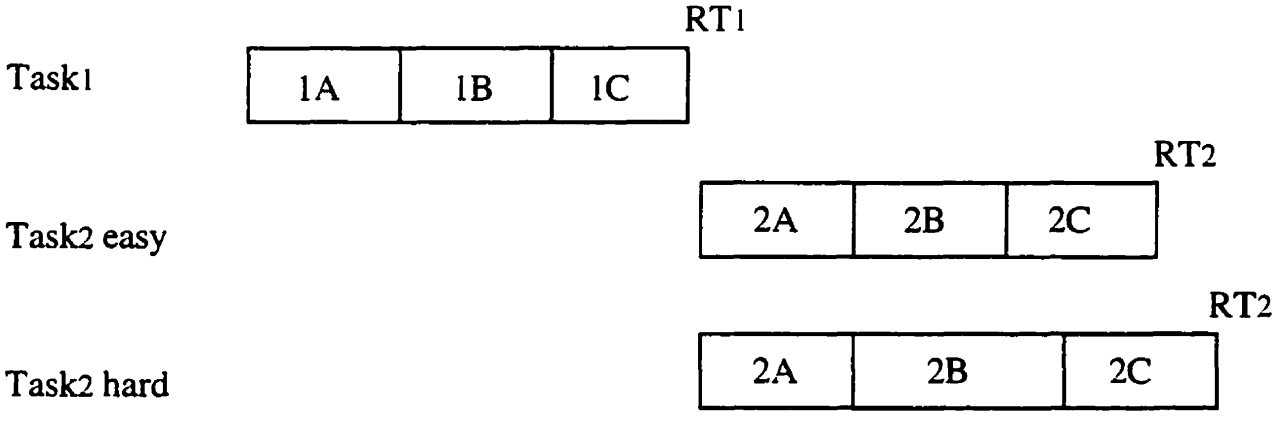
Figure 15. Experiment 3 - mean $Task_2$ reaction time (ms) for trials on which the S_2 letter was not repeated, that is, was not the same as the S_2 letter on the immediately preceding trial. The effects of probability (1: 4: 9) as a function of SOA are shown.

Figure 16. Experiment 4 - mean reaction time (ms) for $Task_1$ (dashed lines), and $Task_2$ S_2-present trials (solid lines), as a function of SOA. The effects of probability (1: 4: 9) of S_2 as a function of SOA are shown.

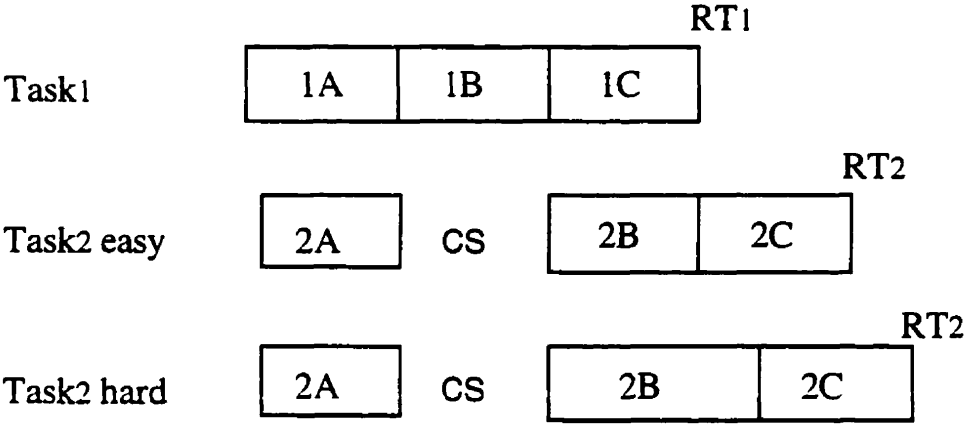
Figure 17. Experiment 4 - mean $Task_2$ reaction time (ms) for trials in the probability 9 condition as a function of SOA, where S_2 was not repeated, and where S_2 was repeated. That is, trials on which the S_2 letter was not the same as the S_2 letter on the immediately preceding trial, and trials on which the S_2 letter was the same as on the previous trial.

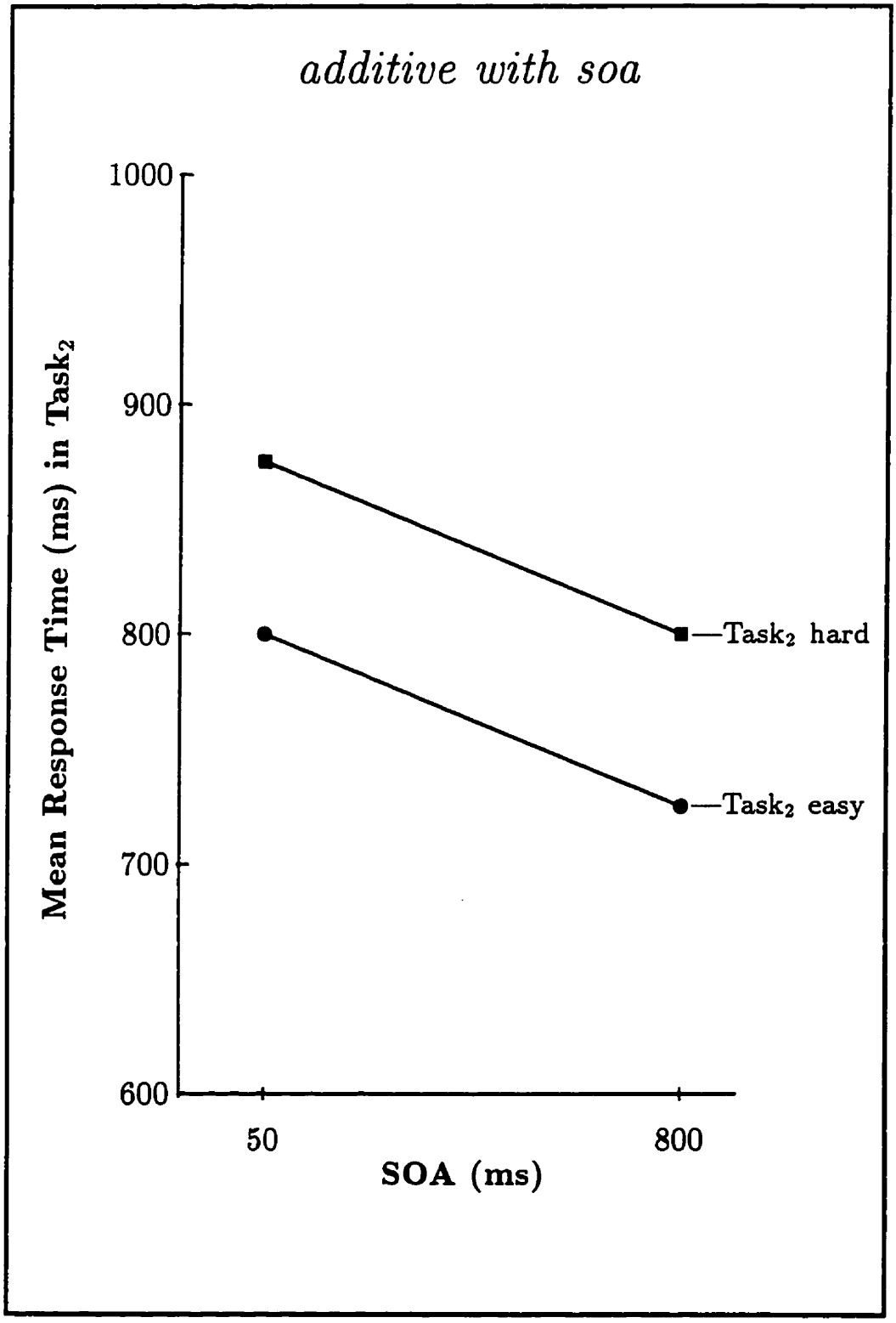
Figure 18. Experiment 4 - mean $Task_2$ reaction time (ms) for S_2-present trials on which the S_2 letter was not repeated, that is, was not the same as the S_2 letter on the immediately preceding trial. The effects of the probability (1: 4: 9) as a function of SOA are shown.

Long SOA

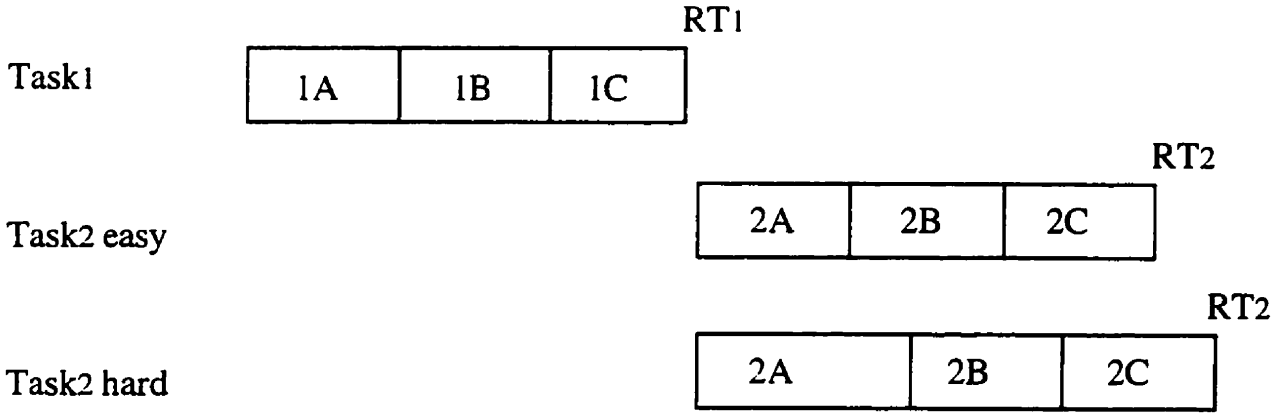


Short SOA

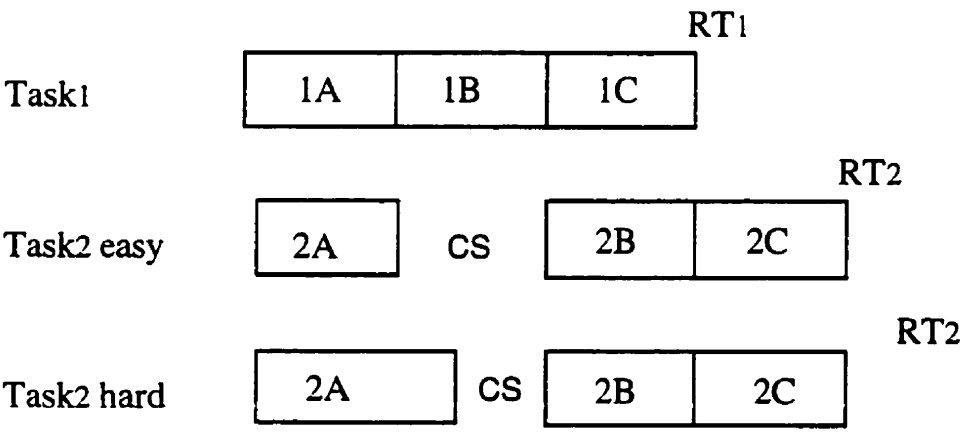


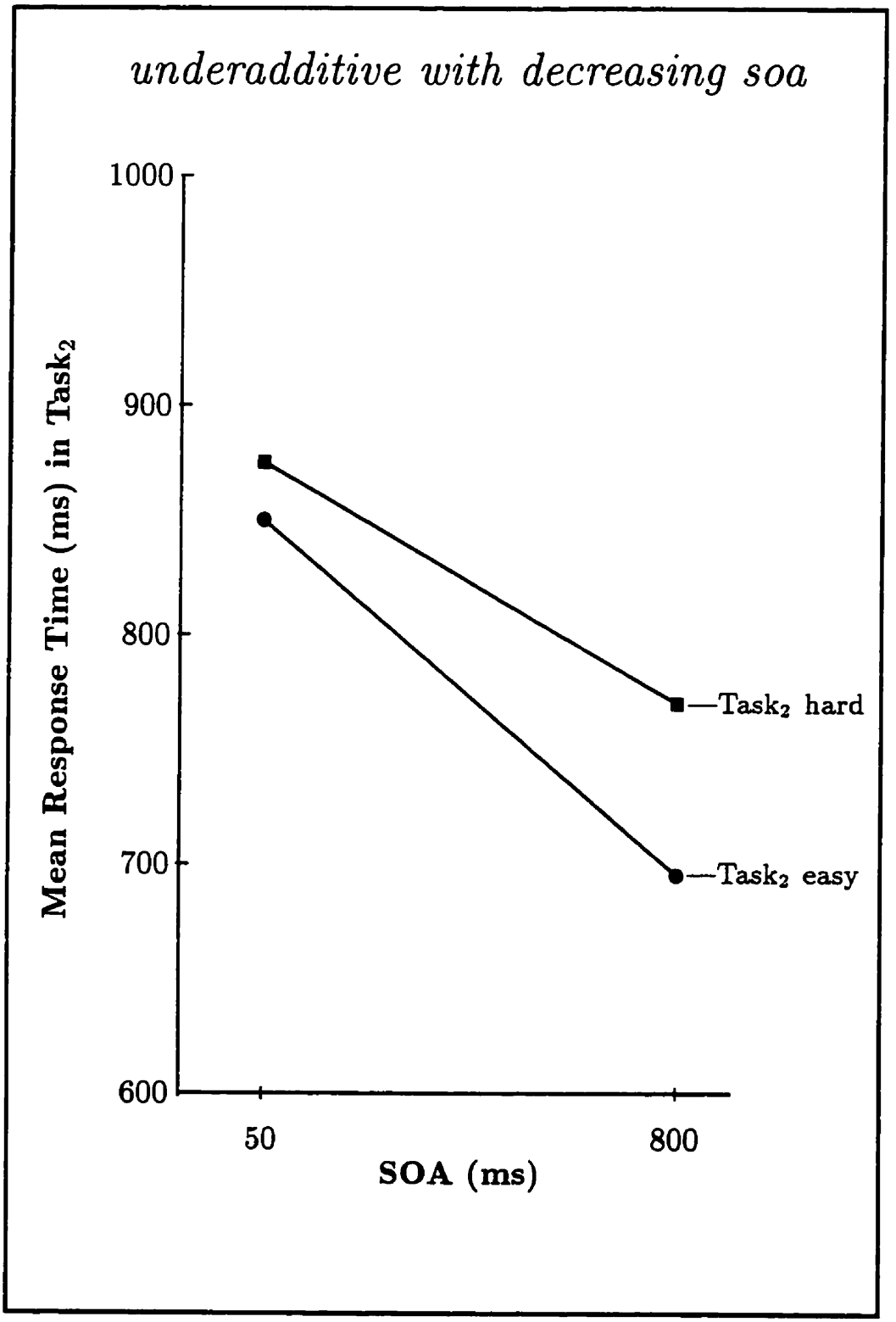

additive with soa
1000
900
800
700
600
Mean Response Time (ms) in Task2
Task2 hard
Task2 easy
50
800
SOA (ms)

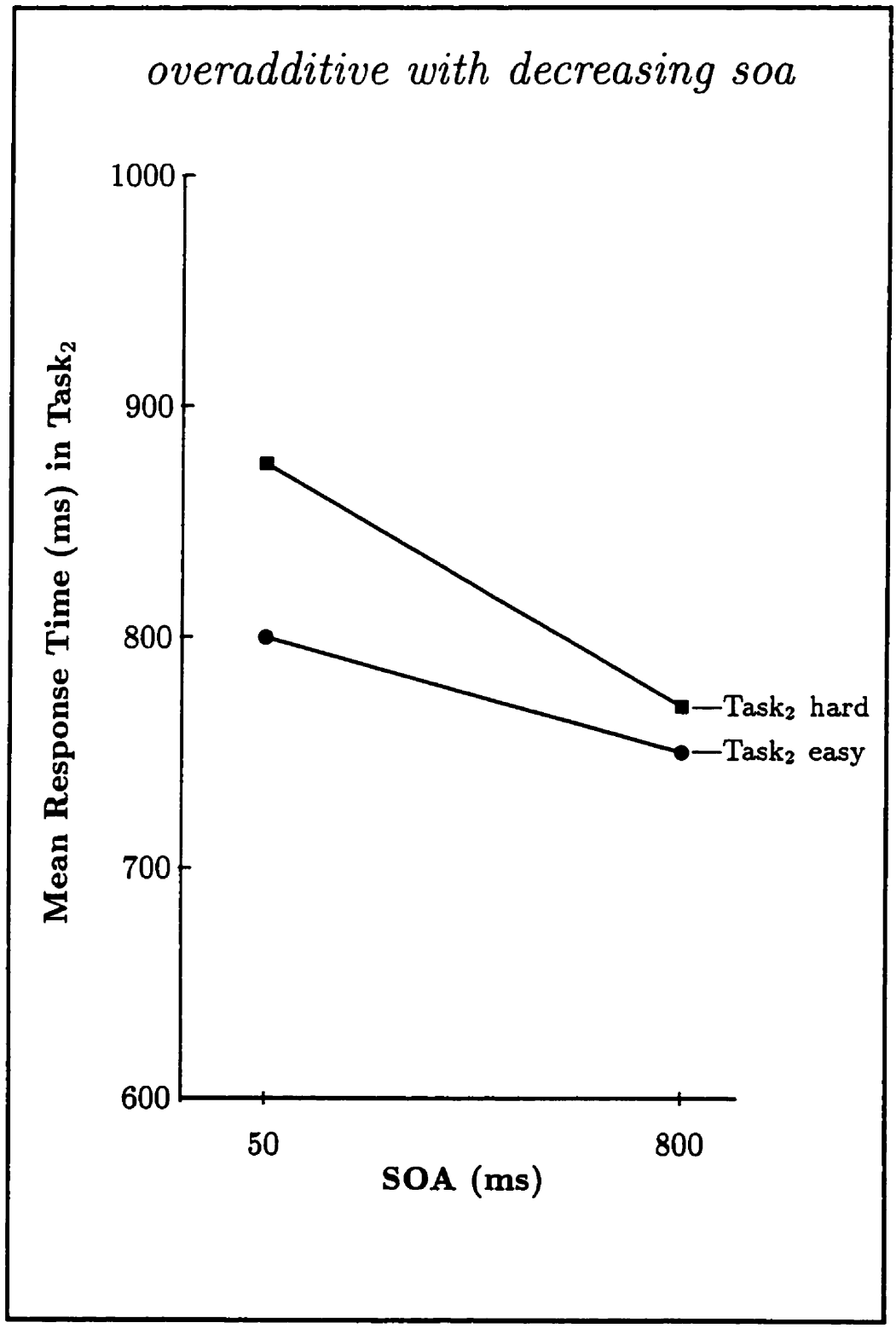
Long SOA

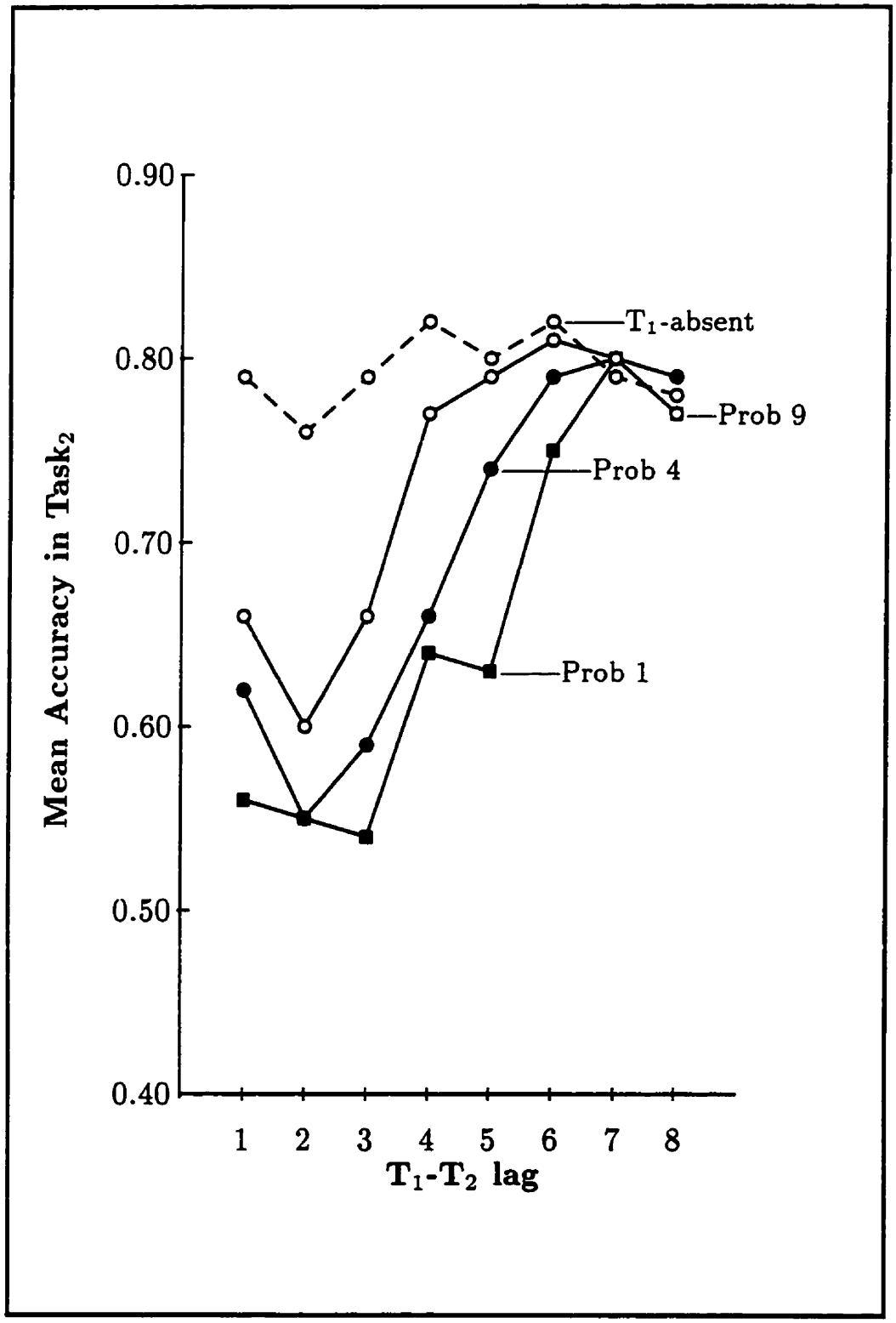


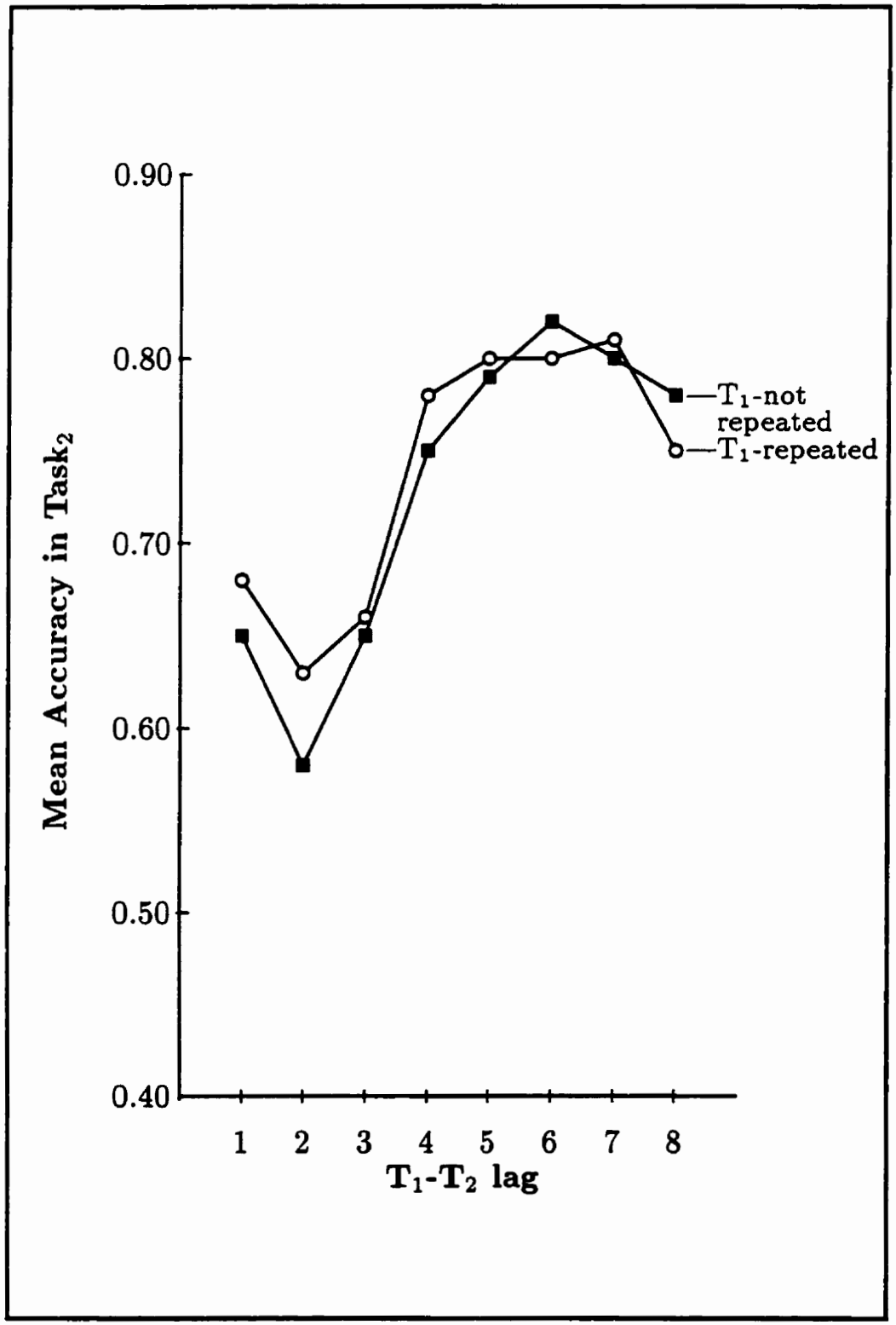
Short SOA

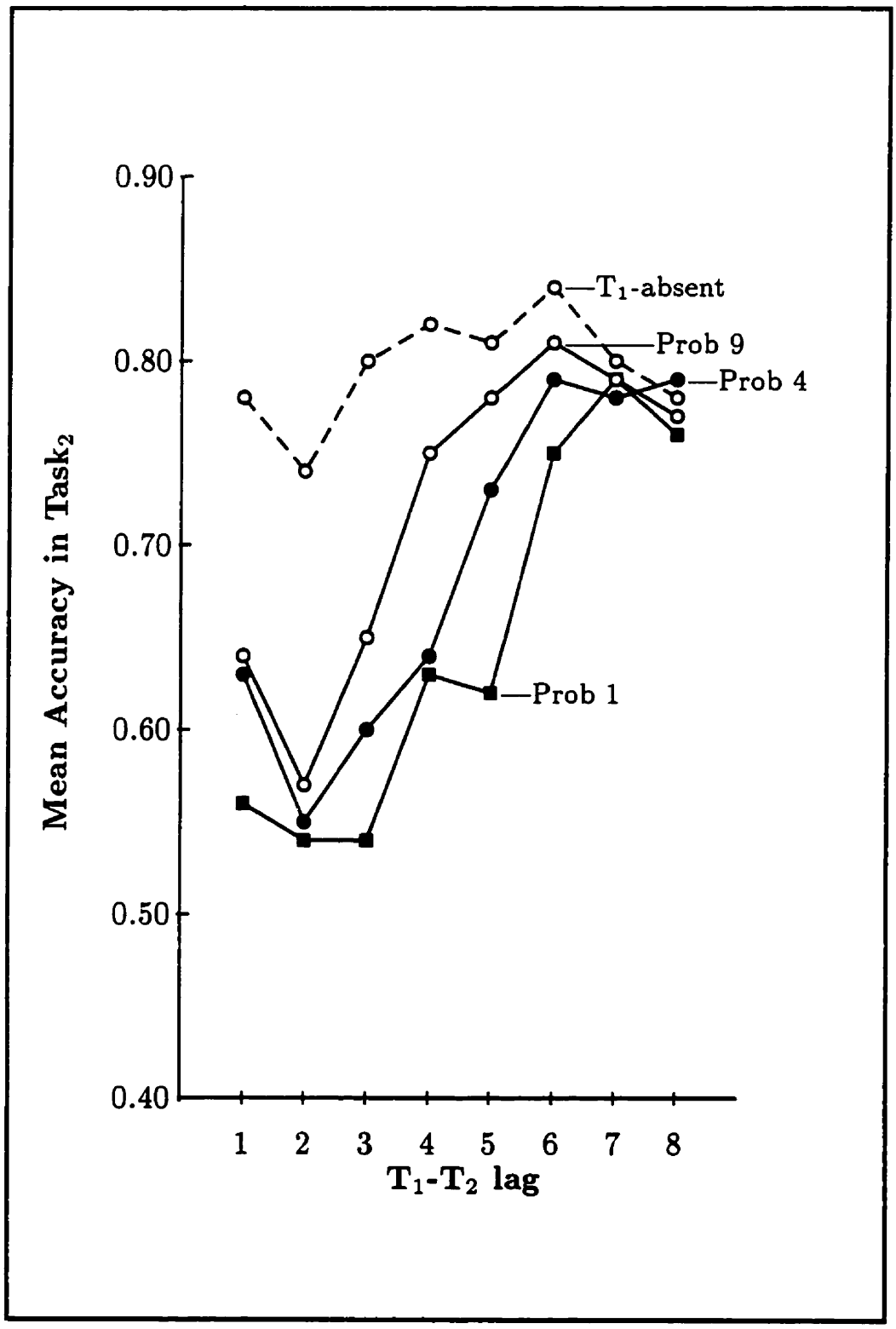


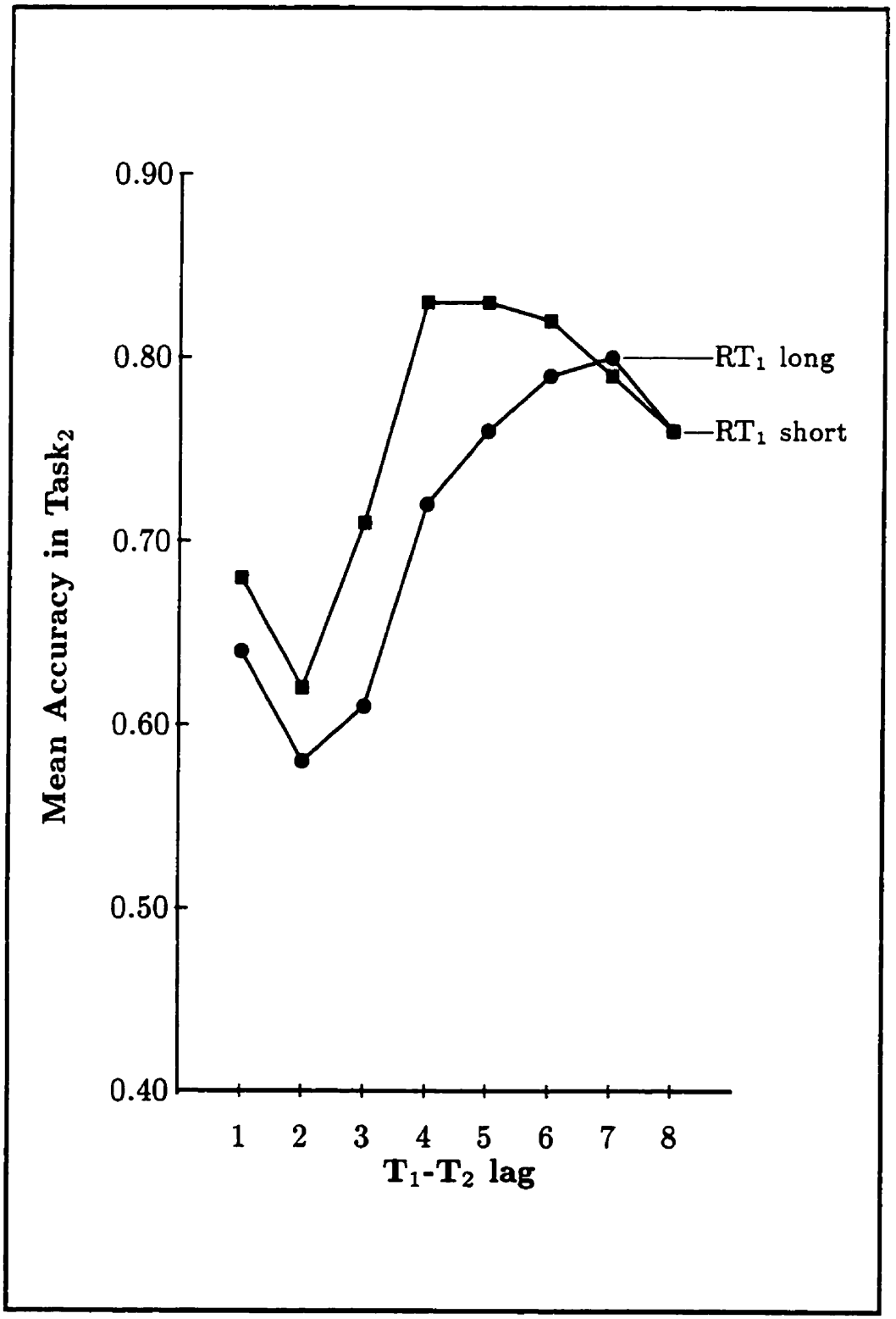

underadditive with decreasing soa
1000
900
800
700
600
Mean Response Time (ms) in Task2
Task2 hard
Task2 easy
50
800
SOA (ms)

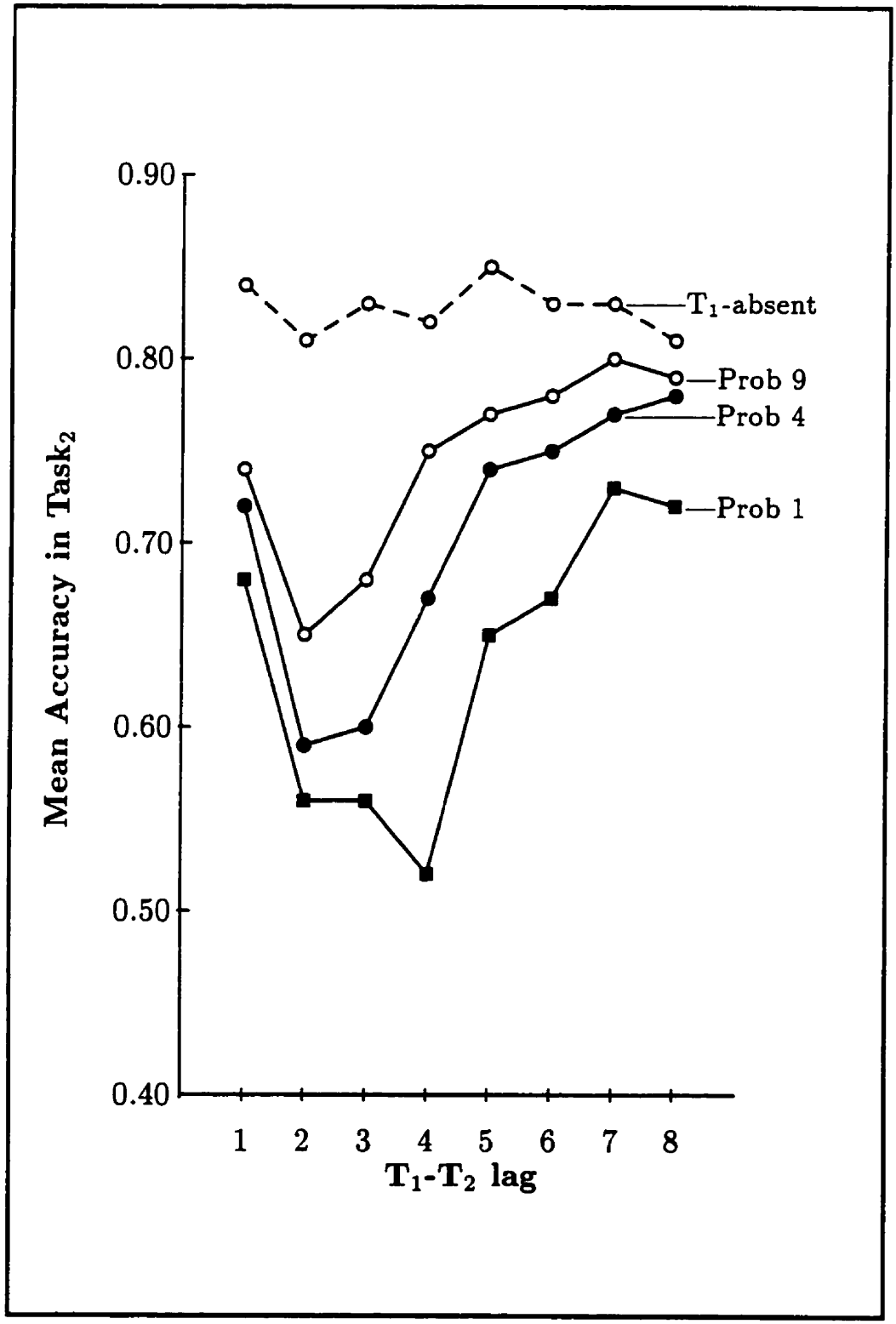

overadditive with decreasing soa
1000
900
800
700
600
Mean Response Time (ms) in Task2
Task2 hard
Task2 easy
50
800
SOA (ms)

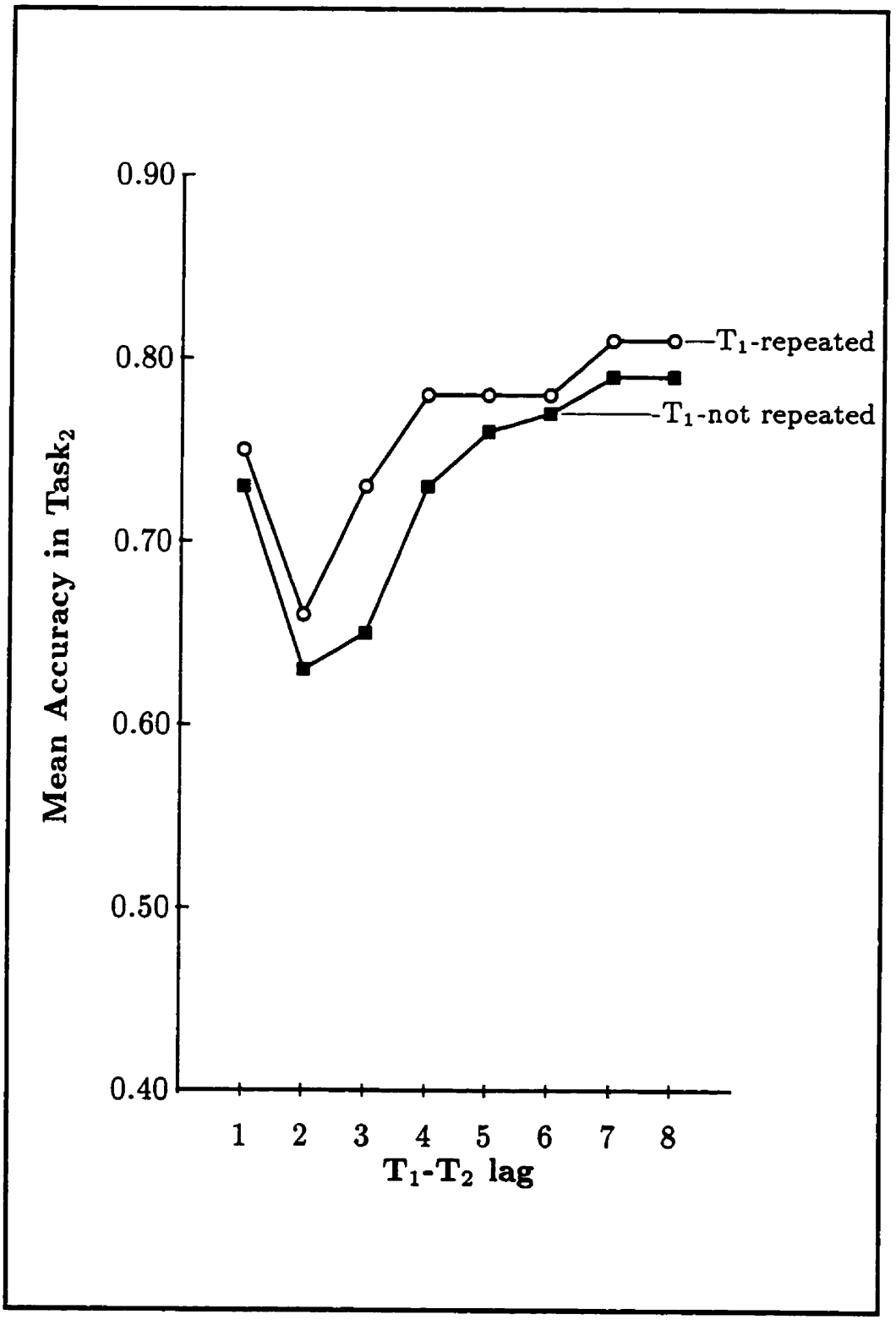

0.90
0.80
0.70
0.60
0.50
0.40
Mean Accuracy in Task2
T1-absent
Prob 9
Prob 4
Prob 1
1 2 3 4 5 6 7 8
T1-T2 lag

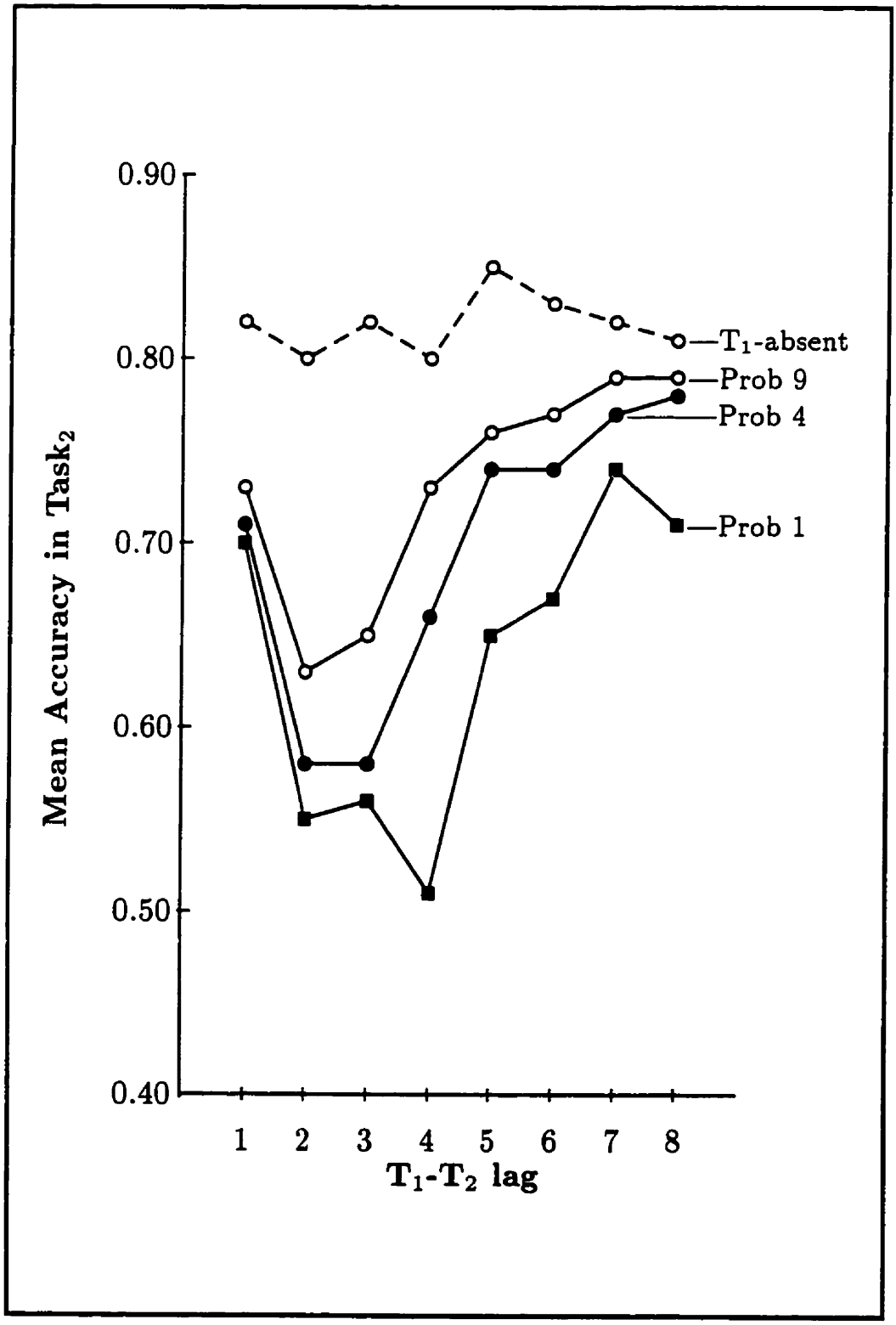

0.90
0.80
0.70
0.60
0.50
0.40
Mean Accuracy in Task2
1
2
3
4
5
6
7
8
T1-T2 lag
T1-not repeated
T1-repeated

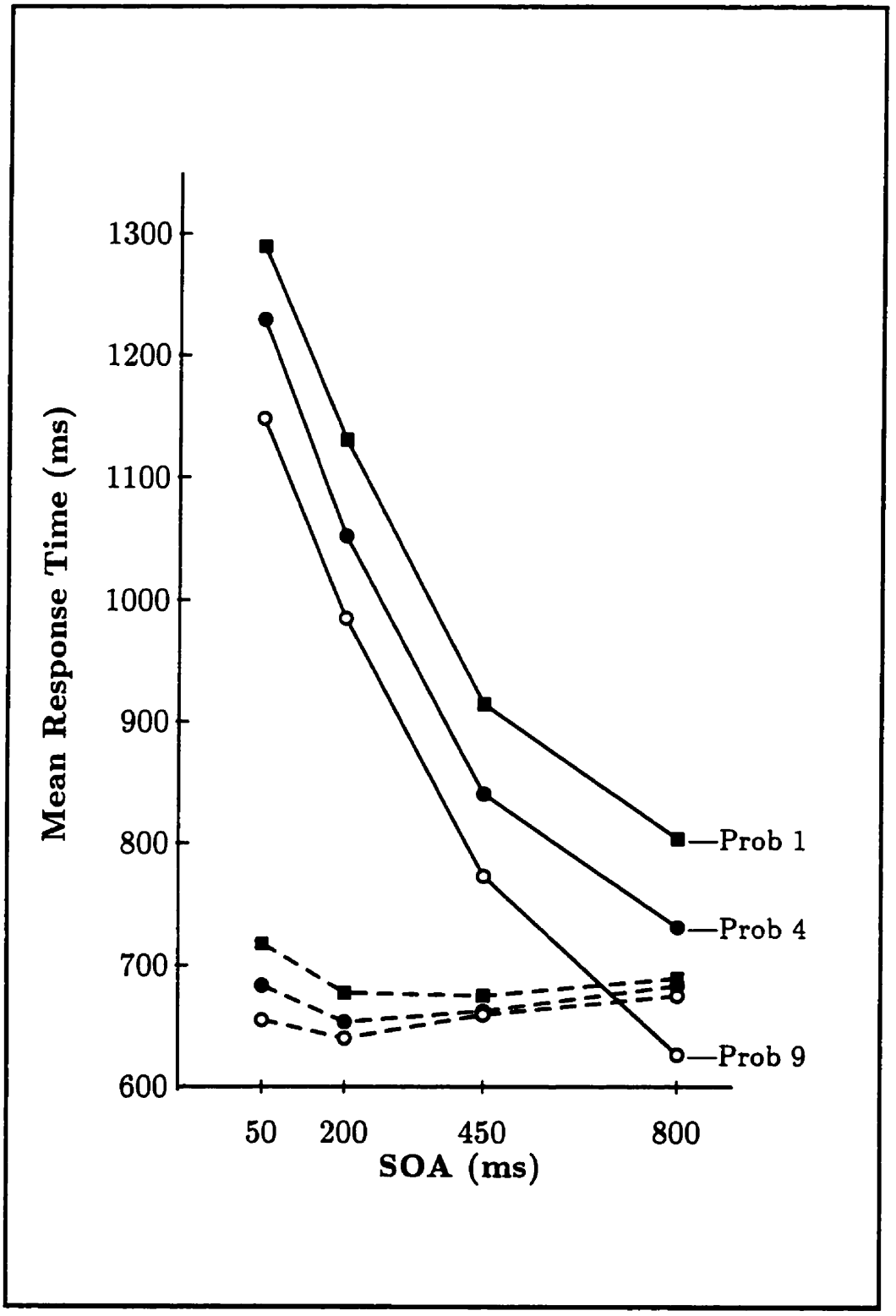

0.90
0.80
0.70
0.60
0.50
0.40
Mean Accuracy in Task2
T1-absent
Prob 9
Prob 4
Prob 1
1 2 3 4 5 6 7 8
T1-T2 lag

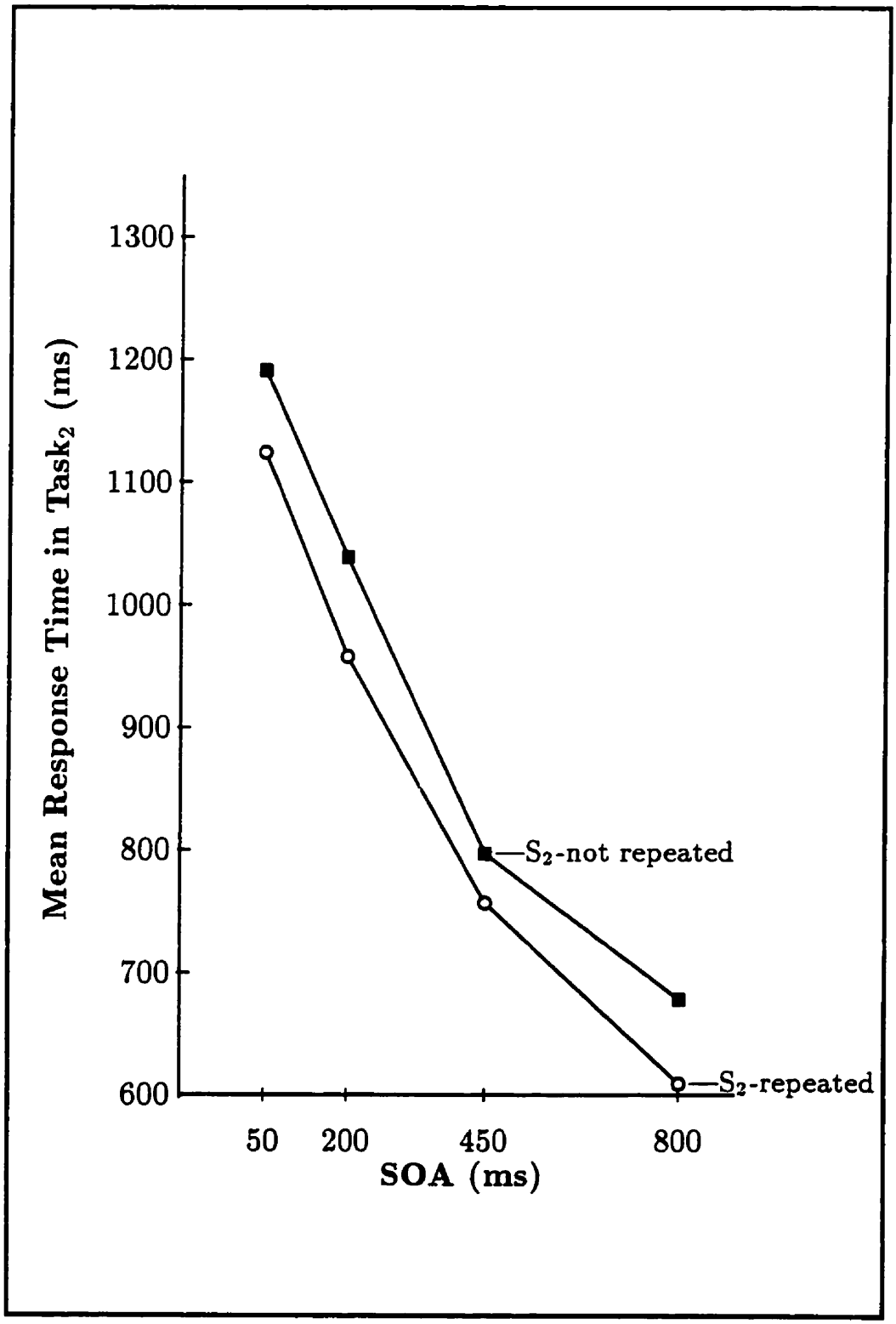

0.90
0.80
0.70
0.60
0.50
0.40
Mean Accuracy in Task2
RT1 long
RT1 short
1
2
3
4
5
6
7
8
T1-T2 lag

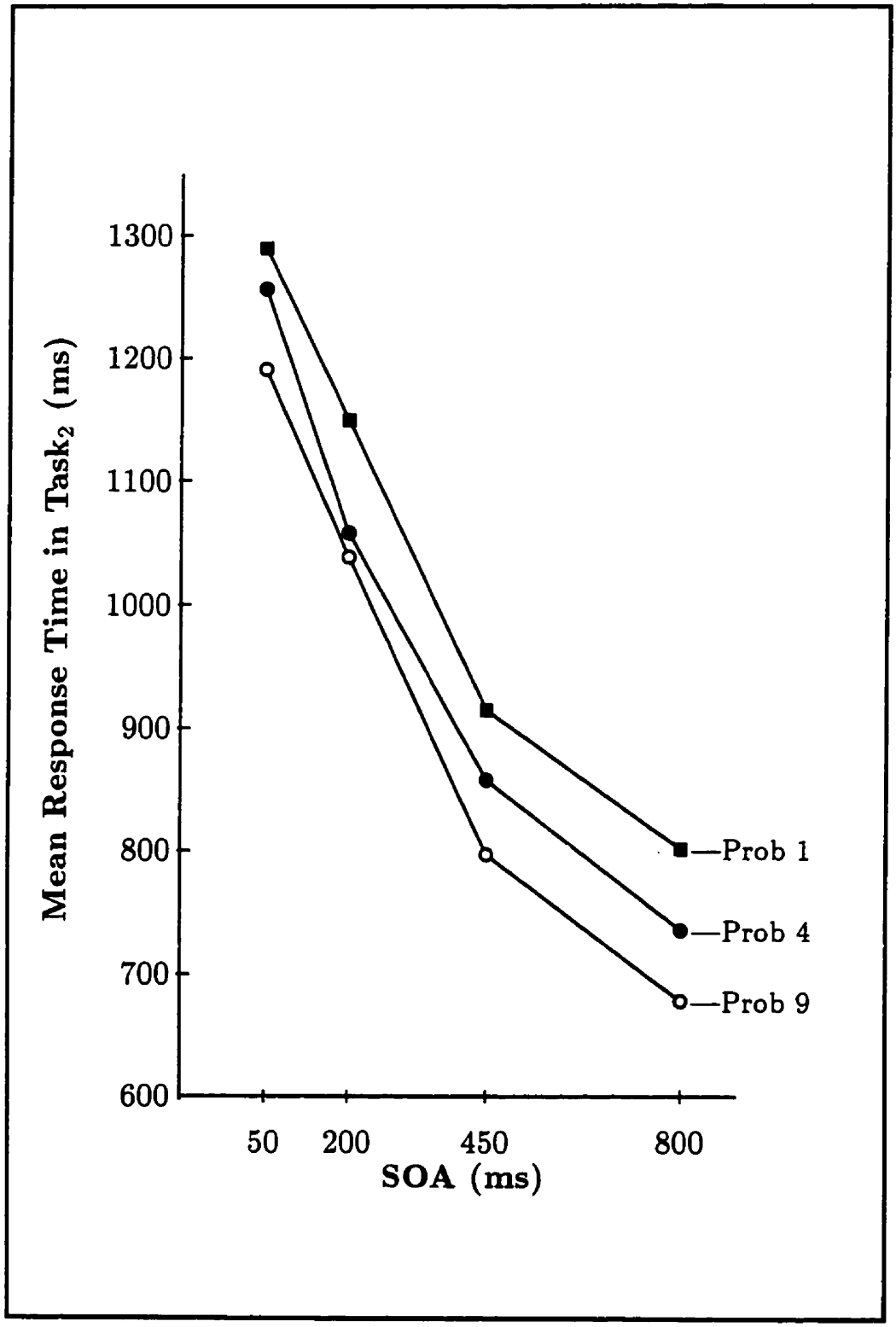

0.90
0.80
0.70
0.60
0.50
0.40
Mean Accuracy in Task2
T1-absent
Prob 9
Prob 4
Prob 1
1
2
3
4
5
6
7
8
T1-T2 lag

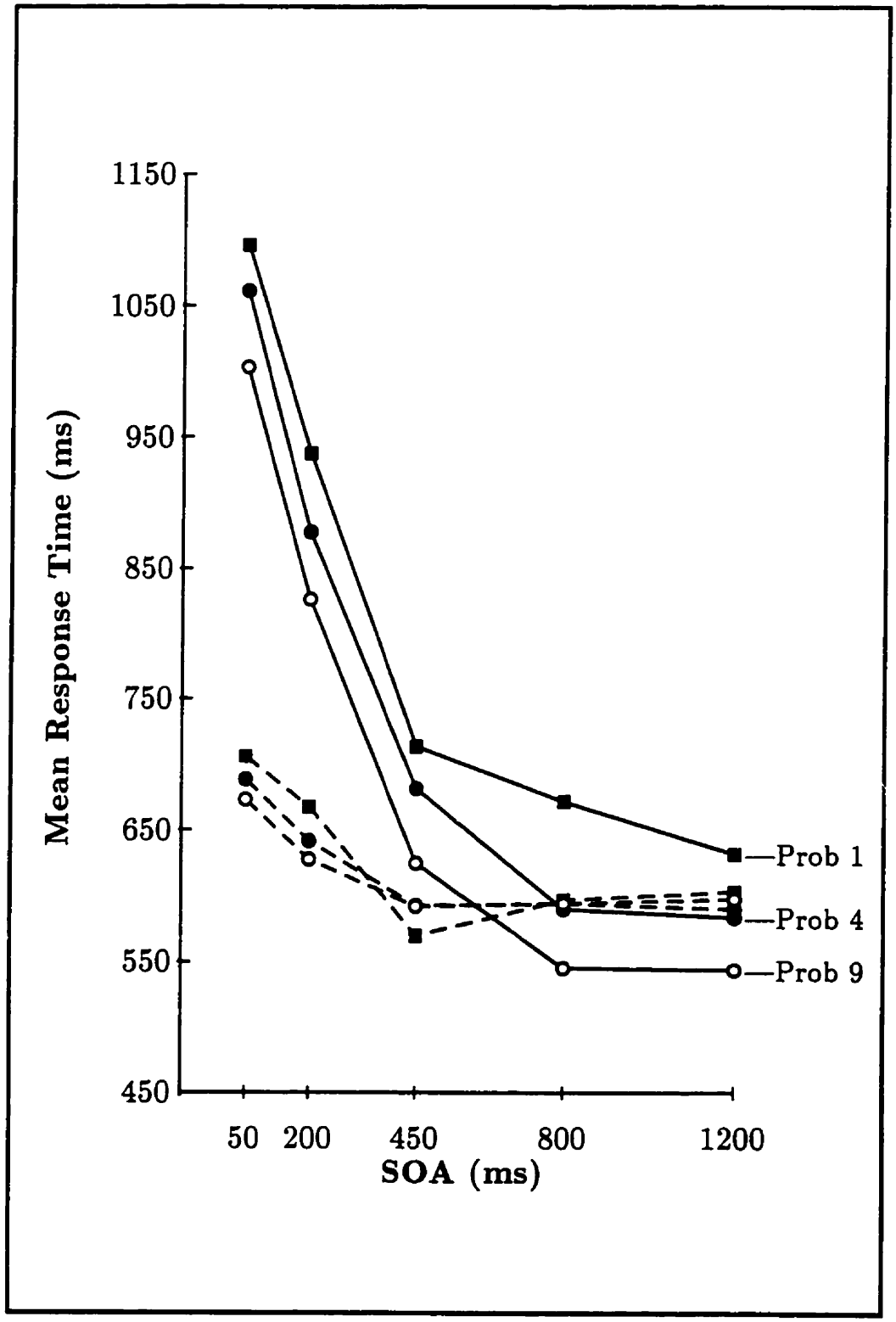

0.90
0.80
0.70
0.60
0.50
0.40
Mean Accuracy in Task2
T1-repeated
T1-not repeated
1 2 3 4 5 6 7 8
T1-T2 lag

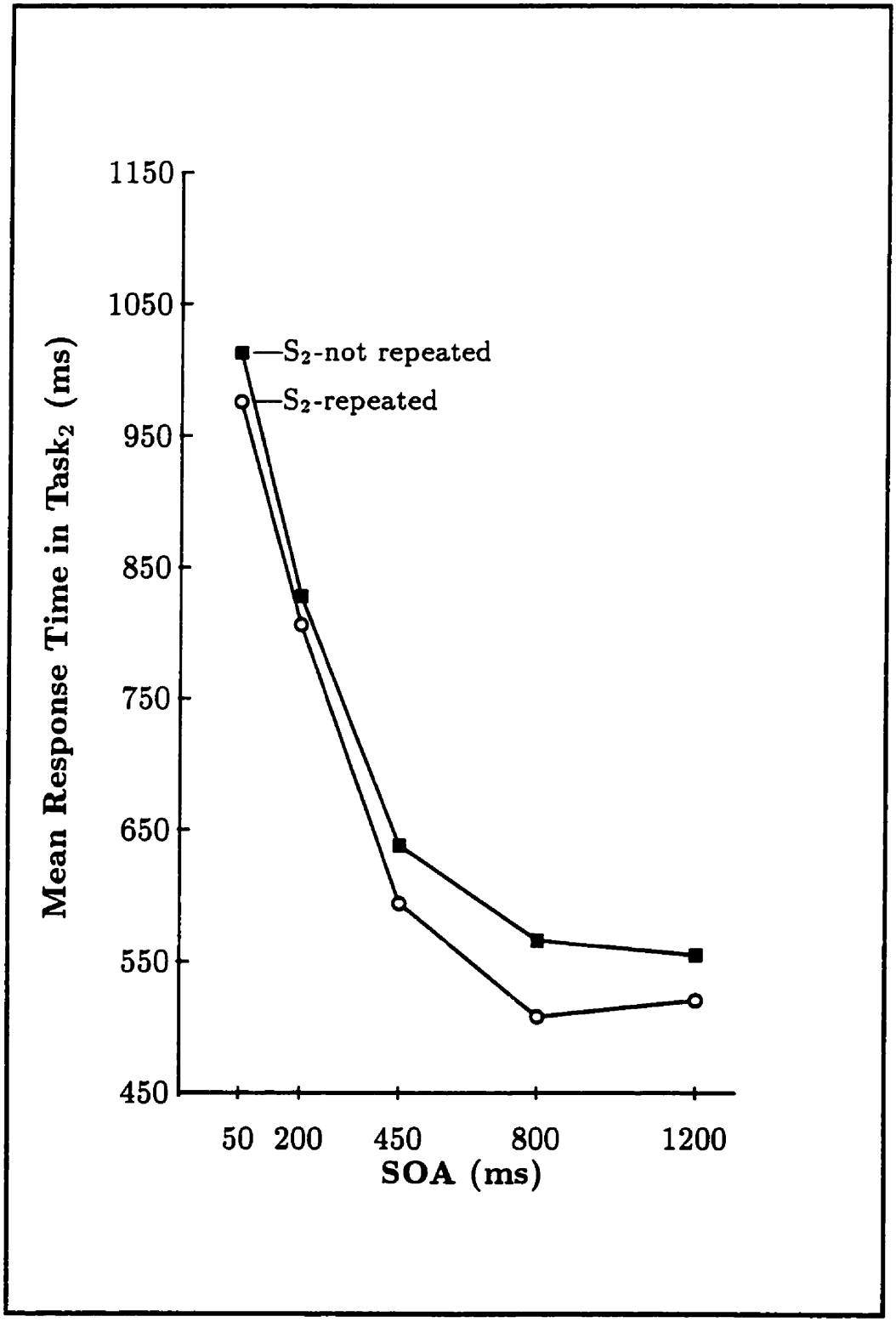

0.90
0.80
0.70
0.60
0.50
0.40
Mean Accuracy in Task2
1
2
3
4
5
6
7
8
T1-T2 lag
T1-absent
Prob 9
Prob 4
Prob 1

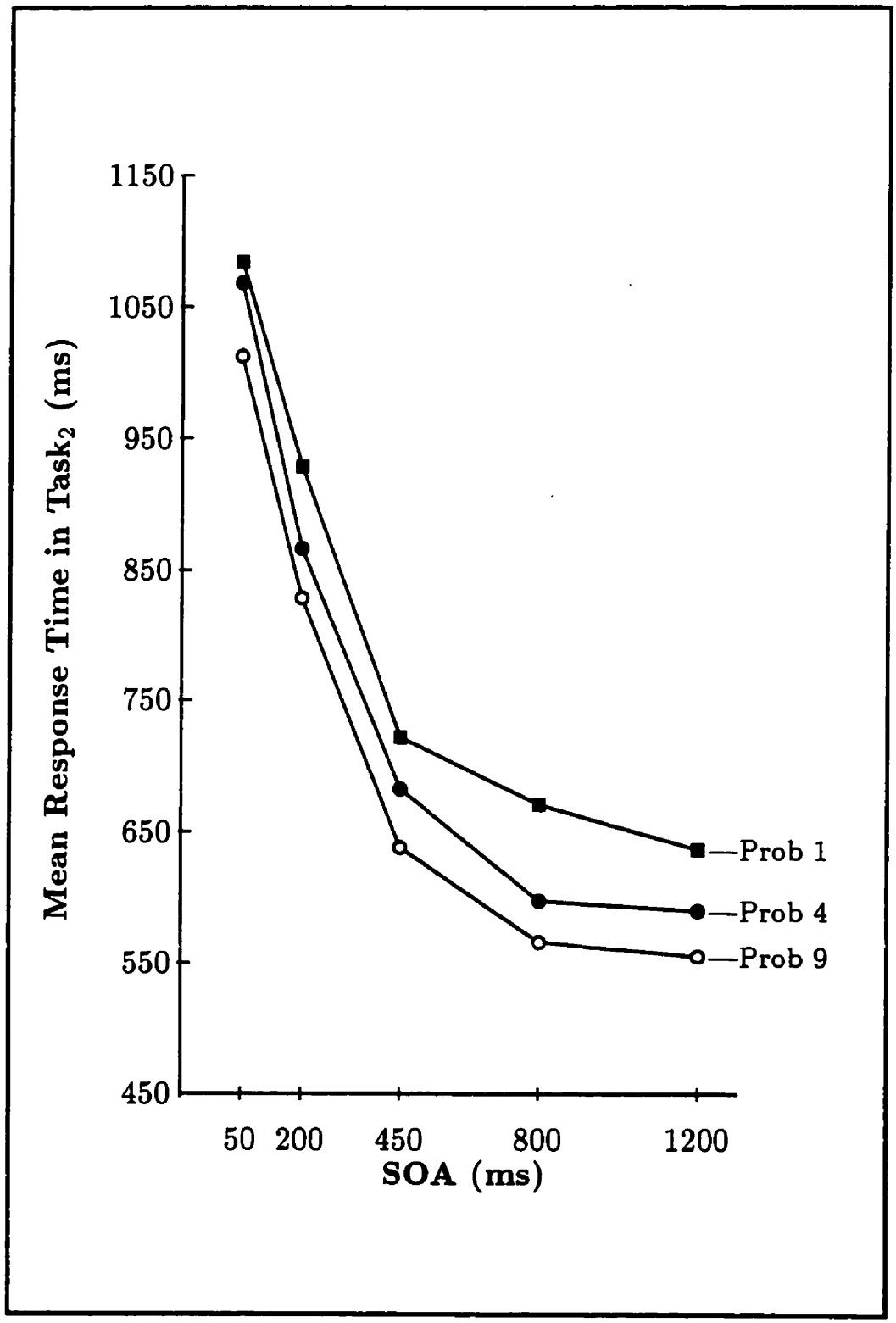

Mean Response Time (ms)
1300
1200
1100
1000
900
800
700
600
50
200
450
800
SOA (ms)
—Prob 1
—Prob 4
—Prob 9


Mean Response Time in Task$_2$ (ms)
1300
1200
1100
1000
900
800
700
600
50
200
450
800
SOA (ms)
—S$_2$-not repeated
—S$_2$-repeated


Mean Response Time in Task$_2$ (ms)
1300
1200
1100
1000
900
800
700
600
50
200
450
800
SOA (ms)
—Prob 1
—Prob 4
—Prob 9


1150
1050
950
850
750
650
550
450
Mean Response Time (ms)
50
200
450
800
1200
SOA (ms)
Prob 1
Prob 4
Prob 9


1150
1050
950
850
750
650
550
450
Mean Response Time in Task$_2$ (ms)
—S$_2$-not repeated
—S$_2$-repeated
50
200
450
800
1200
SOA (ms)


1150
1050
950
850
750
650
550
450
Mean Response Time in Task$_2$ (ms)
50
200
450
800
1200
SOA (ms)
—Prob 1
—Prob 4
—Prob 9

Appendix A

Experiment 1 - Mean $Task_1$ response times (ms) for each level of probability and T_1-T_2 lag

Probability Level	T_1-T_2 Lag							
	1	2	3	4	5	6	7	8
1	676	661	651	673	666	641	657	665
4	595	595	594	597	589	597	592	576
9	521	513	520	523	525	521	522	519

Experiment 1 - Mean $Task_1$ accuracy for each level of probability and T_1-T_2 lag

Probability Level	T_1-T_2 Lag							
	1	2	3	4	5	6	7	8
T_1-absent	0.982	0.985	0.980	0.985	0.981	0.996	0.990	0.990
1	0.887	0.905	0.889	0.844	0.873	0.870	0.880	0.893
4	0.931	0.903	0.920	0.904	0.935	0.920	0.927	0.917
9	0.965	0.964	0.968	0.969	0.973	0.968	0.969	0.966

Appendix B

Experiment 1 - Mean $Task_2$ accuracy for each level of probability and T_1-T_2 lag

Probability Level	T_1-T_2 Lag							
	1	2	3	4	5	6	7	8
T_1-absent	0.785	0.764	0.793	0.812	0.804	0.825	0.793	0.779
1	0.556	0.552	0.542	0.635	0.627	0.752	0.804	0.757
4	0.624	0.547	0.589	0.659	0.736	0.787	0.797	0.788
9	0.662	0.603	0.657	0.768	0.788	0.810	0.805	0.767

Appendix C

Experiment 1 - Mean $Task_2$ accuracy on T_1-not repeated and T_1-repeated trials for each T_1-T_2 lag in the probability 9 condition

	T_1-T_2 Lag							
	1	2	3	4	5	6	7	8
T_1-not repeated	0.648	0.580	0.650	0.753	0.786	0.815	0.799	0.777
T_1-repeated	0.682	0.629	0.663	0.782	0.796	0.800	0.813	0.755

Appendix D

Experiment 1 - Mean $Task_2$ accuracy on T_1-not repeated trials for each level of probability and T_1-T_2 lag

Probability Level	T_1-T_2 Lag							
	1	2	3	4	5	6	7	8
T_1-absent	0.777	0.744	0.803	0.821	0.807	0.840	0.797	0.782
1	0.556	0.542	0.545	0.628	0.622	0.748	0.800	0.760
4	0.626	0.547	0.602	0.642	0.734	0.794	0.780	0.792
9	0.648	0.580	0.650	0.753	0.786	0.815	0.799	0.777

Appendix E

Experiment 2 - Mean $Task_1$ accuracy rate for each level of probability and T_1-T_2 lag

Probability Level	T_1-T_2 Lag							
	1	2	3	4	5	6	7	8
1	0.922	0.896	0.891	0.896	0.917	0.938	0.885	0.922
4	0.905	0.923	0.924	0.940	0.926	0.924	0.928	0.939
9	0.951	0.949	0.950	0.955	0.952	0.953	0.950	0.955

Appendix F

Experiment 2 - Mean $Task_2$ accuracy for each level of probability and T_1-T_2 lag

Probability Level	T_1-T_2 Lag							
	1	2	3	4	5	6	7	8
T_1-absent	0.785	0.764	0.793	0.816	0.804	0.824	0.793	0.779
1	0.682	0.559	0.562	0.516	0.655	0.674	0.729	0.715
4	0.723	0.589	0.596	0.665	0.738	0.748	0.767	0.776
9	0.741	0.646	0.680	0.751	0.772	0.778	0.802	0.794

Appendix G

Experiment 2 - Mean $Task_2$ accuracy for each T_1-T_2 lag on T_1-not repeated and T_1-repeated trials in the probability 9 condition

T_1-T_2 Lag

	1	2	3	4	5	6	7	8
T_1-not repeated	0.733	0.627	0.647	0.729	0.763	0.774	0.793	0.785
T_1-repeated	0.755	0.665	0.726	0.778	0.782	0.784	0.812	0.806

Appendix H

Experiment 2 - Mean $Task_2$ accuracy on T_1-not repeated trials for each level of probability and T_1-T_2 lag

Probability Level	T_1-T_2 Lag							
	1	2	3	4	5	6	7	8
T_1-absent	0.820	0.800	0.821	0.801	0.845	0.826	0.815	0.810
1	0.696	0.545	0.563	0.507	0.651	0.672	0.736	0.707
4	0.707	0.579	0.577	0.656	0.741	0.744	0.771	0.776
9	0.733	0.627	0.647	0.729	0.763	0.774	0.793	0.785

Appendix I

Experiment 3 - Mean $Task_1$ response times (ms) for each level of probability and SOA (ms)

Probability Level	SOA (ms)			
	50	200	450	800
1	718	677	675	689
4	683	653	662	683
9	655	640	659	675

Experiment 3 - Mean $Task_1$ error rates for each level of probability and SOA (ms)

Probability Level	SOA (ms)			
	50	200	450	800
1	0.103	0.100	0.068	0.082
4	0.135	0.104	0.083	0.096
9	0.129	0.112	0.090	0.087

Appendix J

Experiment 3 - Mean $Task_2$ response times (ms) for each level of probability and SOA (ms)

Probability Level	SOA (ms)			
	50	200	450	800
1	1289	1131	914	804
4	1229	1052	841	731
9	1148	985	773	626

Experiment 3 - Mean $Task_2$ error rates for each level of probability and SOA (ms)

Probability Level	SOA (ms)			
	50	200	450	800
1	0.118	0.129	0.096	0.123
4	0.105	0.092	0.095	0.119
9	0.039	0.035	0.045	0.045

Appendix K

Experiment 3 - Mean $Task_2$ response times (ms) on S_2-not repeated and S_2-repeated trials for each SOA (ms) in the probability 9 condition

	SOA (ms)			
	50	200	450	800
S_2-not repeated	1191	1039	797	678
S_2-repeated	1124	958	757	609

Appendix L

Experiment 3 - Mean $Task_2$ response times on S_2-not repeated trials for each level of probability and SOA (ms)

Probability Level	SOA (ms)			
	50	200	450	800
1	1289	1150	915	802
4	1256	1058	858	736
9	1191	1039	797	678

Appendix M

Experiment 4 - Mean $Task_1$ response times (ms) for each level of probability and SOA (ms)

Probability Level	SOA (ms)				
	50	200	450	800	1200
1	706	668	570	597	603
4	689	642	593	594	590
9	673	628	592	594	598

Experiment 4 - Mean $Task_1$ error rates for each level of probability and SOA (ms)

Probability Level	SOA (ms)				
	50	200	450	800	1200
1	0.068	0.067	0.067	0.071	0.072
4	0.055	0.080	0.069	0.059	0.059
9	0.063	0.070	0.053	0.059	0.045

Appendix N

Experiment 4 - Mean $Task_2$ response times (ms) for each level of probability and SOA (ms)

Probability Level	SOA (ms)				
	50	200	450	800	1200
1	1096	937	714	672	632
4	1061	877	682	590	584
9	1003	826	625	545	544

Experiment 4 - Mean $Task_2$ error rates for each level of probability and SOA (ms)

Probability Level	SOA (ms)				
	50	200	450	800	1200
1	0.104	0.141	0.083	0.090	0.106
4	0.093	0.095	0.067	0.058	0.073
9	0.041	0.027	0.027	0.032	0.055

Appendix O

Experiment 4 - Mean $Task_2$ response times (ms) on S_2-not repeated and S_2-repeated trials for each SOA (ms) in the probability 9 condition

	SOA (ms)				
	50	200	450	800	1200
S_2-not repeated	1013	828	638	566	555
S_2-repeated	976	806	594	508	521

Appendix P

Experiment 4 - Mean $Task_2$ response times on S_2-not repeated trials for each level of probability and SOA (ms)

Probability Level	SOA (ms)				
	50	200	450	800	1200
1	1084	928	722	671	637
4	1068	866	683	598	590
9	1013	828	638	566	555