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Direct estimates of processing delays in the attentional blink

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Abstract When two targets (T1 and T2) are presented in rapid succession, identification of T2 is often impaired (*attentional blink*: AB). The two-stage model accounts for the AB deficit by assuming that: (a) T2 is delayed in Stage 1 while Stage 2 is busy processing T1, and (b) T2 is vulnerable to masking while delayed. We report converging evidence for the model by evaluating these assumptions independently of the AB deficit itself. The results show that: (a) response times for T2 identification decreased as the lag between T1 and T2 was increased; (b) response times for T2 decreased across lags only if T1 was masked; and (c) accuracy of T2 identification increased as the stimulus-onset asynchrony between T2 and the trailing mask was increased.

Introduction

Brief stimuli presented in rapid sequence may exceed the processing capacity of the visual system, thus giving rise to perceptual deficits. One such deficit is the *attentional blink* (AB), in which identification of the second of two targets is impaired as a function of the temporal lag between them. At short lags (e.g., 200–300 ms), identification of the second target is maximally impaired. However, as the lag between the first (T1) and second (T2) target is increased, identification of T2 improves.

A theoretical account of the AB deficit has been proposed by Chun and Potter (1995), based on two sequential processing stages. Although, as suggested by Shapiro, Arnell, and Raymond (1997), all current models of the AB possess broad common characteristics (Duncan, Ward & Shapiro, 1994; Shapiro, Raymond & Arnell, 1994; Jolicoeur & Dell'Acqua, 1998, in press), Chun and Potter's two-stage model makes some specific predictions which are evaluated in the present work.

In the first stage of Chun and Potter's (1995) two-stage model, stimulus features such as colour, orientation and form are rapidly analyzed in order to identify potential targets. Because representations at this stage are subject to rapid decay and interferences by trailing stimuli, they must be transferred to a second stage where they are processed to a level appropriate for response planning and execution. Stage 2 is said to be capacity-limited in that it can process only a single target at a time. Therefore, if T2 arrives while Stage 2 is busy, it is delayed in Stage 1 where it is vulnerable to interference by trailing stimuli. Such interference may take the form of overwriting (Chun & Potter, 1995) or, equivalently, backward masking (Giesbrecht & Di Lollo, 1998). At short temporal lags, identification of T2 is impaired because T2 is delayed in Stage 1 and thus remains vulnerable to masking for an extended period while Stage 2 is busy processing T1. At longer lags, the probability is higher that T1 has been processed before the arrival of T2. Therefore, the probability that T2 will be delayed in Stage 1 is reduced, and performance improves.

Two critical assumptions are made in the two-stage model. The first is that T2 is delayed in Stage 1 while Stage 2 is busy. The second is that, while delayed, T2 is vulnerable to masking by trailing stimuli. In earlier work, support for these assumptions was obtained indirectly, mainly in the context of the AB deficit itself. For example, Chun and Potter (1995) found that increasing the discriminability of T1 reduced the magnitude of the AB. From this, they concluded that when T1 was more discriminable, was processed more rapidly, thus reducing the delay of T2 in Stage 1. The present work was designed to test these assumptions directly and independently of the AB deficit itself.

The period for which T2 is delayed in Stage 1 can be assessed directly by estimating how long observers take to identify T2 over a range of T1–T2 lags. To this end,

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we recorded response times (RTs) to T2 as a function of the temporal lag between the targets. At short lags, the delay of T2 in Stage 1 should be relatively long because Stage 2 is likely to be busy with T1. This means that a relatively long interval will elapse from the time T2 is presented to the time it is identified in Stage 2. Because of this delay, response times should be relatively long. In contrast, at longer lags there is a higher probability that T1 has been processed; thus, the delay of T2 in Stage 1 should be shorter. Therefore, T2 response times should decrease as a function of T1–T2 lag.

In the same experiment, we tested the model's assumption that T2 is vulnerable to masking by a trailing stimulus while delayed in Stage 1. This was done by varying the stimulus-onset asynchrony (SOA) between T2 and the trailing mask at a fixed T1–T2 lag. We reasoned that at short SOAs the probability would be relatively high that T2 would still be in Stage 1 and therefore vulnerable to masking, when the trailing mask was presented. As a result, accuracy of T2 identification should be low. At longer SOAs, the probability should increase that T2 will have gained assess to Stage 2 before the arrival of the mask, thus escaping masking. Therefore, T2 accuracy should improve as a function of the SOA between T2 and the trailing mask.

The assumptions made by the two-stage model are similar to those made in a dual-task interference model proposed by Jolicoeur and colleagues (Jolicoeur, 1998, 1999; Jolicoeur & Dell'Acqua, 1998, in press). Indeed, Jolicoeur (1999) argues that the dual-task model is a more general case of the two-stage model, applicable to a variety of tasks in which two targets must be identified or detected sequentially (e.g., Pashler, 1994; Welford, 1952). The dual-task model consists of a series of stages in which stimuli are processed in parallel, followed by a limited-capacity serial stage. As in the two-stage model, the parallel stages, referred to as sensory and perceptual encoding, process such attributes of incoming stimuli as form and colour in order to identify potential targets. Because representations in these stages decay rapidly, they must be transferred to a limited-capacity, serial stage for consolidation of information into short-term memory and response planning. Within this dual-task framework, the AB is said to occur because T2 arrives before the limited-capacity stage has completed processing T1. In this case, T2 is delayed at the perceptual encoding stage where it is vulnerable to masking (Jolicoeur, 1999).

To test the accounts of the AB given by both the two-stage and dual-task models, Jolicoeur and Dell'Acqua (1998; Exp. 6) conducted an experiment that measured T2 response times as a function of lag. In that experiment, T1 was a letter presented on the computer screen and T2 was a high- or low-pitched tone. The temporal lag between T1 and T2 was varied from 50 to 650 ms, and participants were required to make a speeded identification of the tone pitch and then identify the letter at leisure. Consistent with predictions from both models, response times to the tone decreased as a function of lag. This suggested that by increasing the temporal interval between the targets, more processing of T1 occurred before the presentation of T2. Consequently, the period of delay for T2 was decreased, and response times improved accordingly.

In Exp. 1, we replicated and expanded upon the work of Jolicoeur and Dell' Acqua (1998) in two ways. First, while Jolicoeur and Dell' Acqua presented targets cross-modally, we presented only visual targets. This allowed a test of their explicit claim that the cross-modal effects should generalize to the single-modality case. Such a test is important because it has been suggested that different mechanisms may be responsible for the AB deficits found with cross-modal and unimodal targets (see Arnell & Jolicoeur, 1999; Potter, Chun, Banks, & Muckenhoupt, 1998). Second, while Jolicoeur and Dell'Acqua employed a control condition in which T1 was ignored, this was done only for lags of 300 ms or longer. In the present work, we employed a similar condition over a wider range of lags from 100 to 500 ms.

Experiment 1

Experiment 1 comprised two conditions. In the *Lag-RT* condition, observers were required to identify T1 and to make a speeded response to T2, which was never masked. Response times to T2 were recorded over a range of T1–T2 lags. We expected response times to decrease as lag was increased, reflecting the decreasing delay of T2 in Stage 1. In the *Mask-SOA* condition, observes were required to identify T1 and T2, both of which were followed by masks. However, while the SOA between T1 and its mask was fixed at 100 ms, T2 was followed by a pattern mask at varying SOAs between 100 and 200 ms. We expected the accuracy of T2 identification to improve as a function of SOA, reflecting the increasing probability that T2 had entered Stage 2 before the arrival of the mask.

Method

Participants. Ten undergraduate psychology students (eight female) at the University of British Columbia participated for course credit. All participants reported normal or corrected-to-normal vision.

Apparatus and stimuli. All stimuli had a luminance of 25 cd/m^2, as measured by a Minolta LS-100 luminance meter, and were displayed on a Tektronix 608 oscilloscope, equipped with P15 phosphor. At a viewing distance of 57 cm, set by a headrest, all stimuli subtended approximately 1° of visual angle. The background and surrounding visual field were dark, except for dim illumination of the keyboard.

The first target was a digit from one to eight. The second target was either a vowel (A, E, I, O, U) or a consonant (V, T, L, P, C). The masks consisted of geometric shapes, constructed from letter segments. Although the masks shared visual features with letters, they were not confusable with any English letter.

Procedure. Stimulus presentation in both conditions was based on the two-target paradigm used by Duncan et al. (1994). Items were displayed for 32 ms and were followed by a 68-ms blank inter-stimulus interval (ISI). In the Lag-RT condition, a trial began with the presentation of a fixation point in the centre of he screen. Participants were instructed to press the space bar to begin a trial. Following a 500-ms blank screen, the T1-digit was presented randomly at one of four locations – 1° above, below, left, or right of the centre of the screen. A mask was presented in the same location as T1 at an SOA of 100 ms. The second target, which was a letter, followed T1 at one of five lags corresponding to T1–T2 SOAs of 100, 200, 300, 400, or 500 ms. The location of T2 depended on that of T1. If T1 had been presented above or below the centre, T2 was presented to the right or left of centre. Alternately, if T1 had been presented to the left or right of centre, T2 was presented above or below centre. T2 was never masked. Participants were asked to indicate as quickly as possible whether T2 was a vowel or a consonant by pressing one of two appropriately-marked keys on the keyboard. After the speeded response to T2, participants made an unspeeded identification of the T1-digit by typing it on the keyboard.

The sequence of events in the Mask-SOA condition was identical to the Lag-RT condition with the following exceptions. The T1–T2 lag was fixed at 200 ms on every trial. In addition, the second target was followed by a mask at one of five SOAs, either 100, 125, 150, 175, or 200 ms. Participants were required to indicate whether T2 was a vowel or a consonant, but this decision was not speeded.

All participants received both the Lag-RT and Mask-SOA conditions. The order of these conditions was counterbalanced such that half the participants received the Lag-RT condition first. Each condition began with 15 practice trials, during which no data were recorded. The experimental trials then followed. In the Lag-RT condition, there were 200 randomly-sequenced trials, 40 at each of the five lags. Similarly, in the Mask-SOA condition, there were 200 randomly-sequenced trials, 40 at each of the five T2-mask SOAs.

In a separate experiment, ten additional participants were tested as a control for the Lag-RT condition. The sequence of events for the control group was identical to that of the Lag-RT condition except that participants were told to ignore T1 and respond only to T2. The control condition was designed to check on the possibility that faster responses to T2 at longer lags may be due not to greater availability of attentional resources, but to an alerting signal triggered by T1 (Posner, 1980). Differences in RTs between the Lag-RT and the control conditions can be ascribed to the requirement to attend to T1.

Results

In both the experimental (Lag-RT) and the control conditions, trials on which errors were made on either T1 or T2 were discarded from the reaction-time anlaysis. This amounted to a total of 10.6% of trials in the experimental group and 4.5% in the control group. Accuracy of T2 identification, given correct T1 identification, did not vary across lags, $F(4,45) = 0.49$, $p > 0.05$, $MSE = 18.18$. This is consistent with earlier findings obtained when T2 was not masked (e.g., Giesbrecht & Di Lollo, 1998).

Reaction times from all other trials were screened using the outlier procedure described by Van Selst and Jolicoeur (1994), which employs a floating criterion based on sample size to determine outliers. This resulted in the removal of a further 2.6% of RTs from the experimental group and 2.4% from the control group. The remaining RT data were used to calculate means for each of the five T1–T2 lags. The means and average standard error for the experimental condition are shown in Fig. 1A. The corresponding means and average standard error for the control condition are displayed in Fig. 1B.

Individual results were analyzed in a 2 (Experimental Group vs. Control Group) × 5 (T1–T2 Lag) analysis of variance. The analysis revealed significant effects of Group, $F(1,18) = 9.71, p = 0.006, MSE = 669337.00$, and of Lag, $F(4,72) = 10.27$, $p < 0.001$, $MSE = 4681.90$. The Lag × Group interaction effect was also significant, $F(4,72) = 3.02, p = 0.023, MSE = 4681.90$.

In the Mask-SOA condition, mean accuracy was calculated for each of the T2-mask SOAs, excluding all

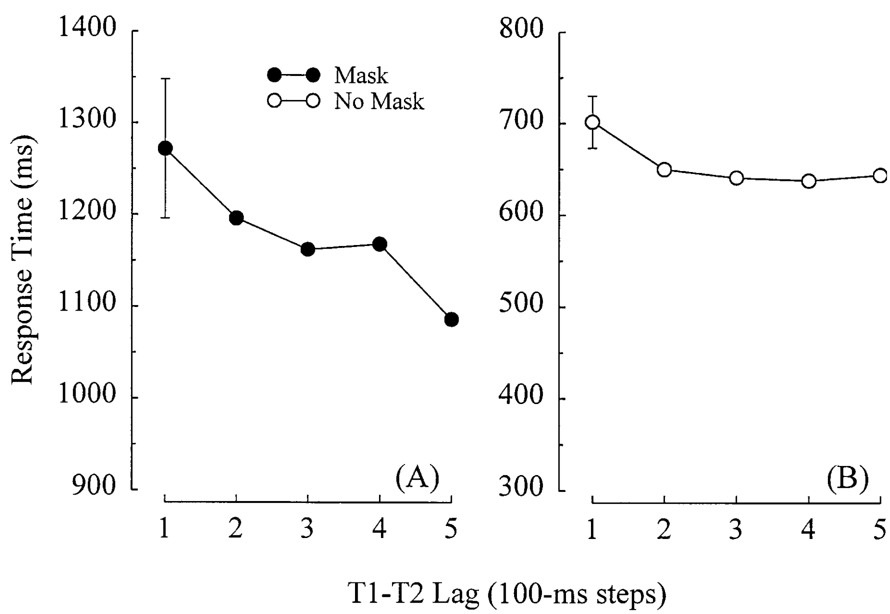

Fig. 1 **a** Mean response time for T2 classification as a function of the temporal lag between the first and second targets in the Lag-RT condition. **b** Mean response time for T2 classification as a function of the temporal lag between the first and second targets in the control condition. Trials on which errors were made on the first target were excluded. In each graph, the *error bar* represents the average standard error of the mean. Note that the response scale is the same in **a** and **b**, but the vertical axes cover different parts of the range

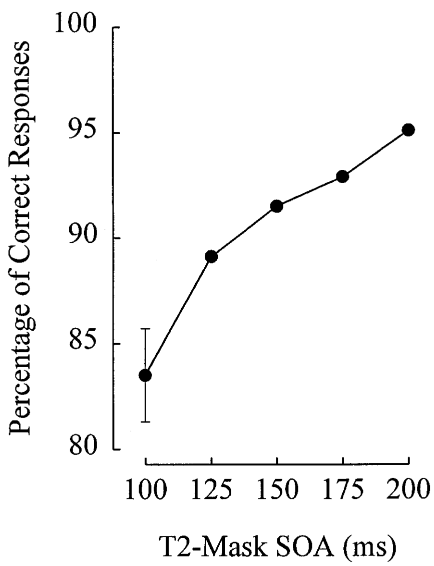

Fig. 2 Mean percentage of correct classifications of the second target, as a function of the stimulus-onset asynchrony (SOA) between the second target and the trailing mask. Trials on which errors were made on the first target were excluded. The *error bar* represents the average standard error of the mean

trials on which errors were made on T1. These means and the average standard error are shown in Fig. 2. A within-subject analysis of variance indicated a significant effect of SOA, F (4,36) = 8.64, $p < 0.001$, MSE = 22.81.

Discussion

Response times in both the experimental and the control conditions became progressively faster as lag was increased (Fig. 1). However, the temporal course of the decrement differed in the two conditions, as confirmed by the Lag × Group interaction effect. At the two shortest lags, both conditions exhibited similar declines in RT. Beyond Lag 2, however, RT continued to decline in the experimental condition, but remained steady in the control condition.

The similarities between the groups at the two shortest lags can be interpreted in two ways. One is that performance was governed only minimally by the main factor responsible for the AB, namely the requirement to attend to T1. Instead, T1 may have served an alerting function, heralding the onset of T2 and resulting in faster response times (Posner, 1980). Alternatively, the initial decline in RTs in both groups may have stemmed from attending to T1. On this option, observers in the control group may have been unable to ignore T1 completely. As a result, some processing of T1 occurred, with a resulting delay for T2 at the shortest lag and correspondingly slower response times.

Beyond Lag 2, RTs continued to decline in the experimental condition while remaining steady in the control condition. This difference must be regarded as stemming from the requirement to attend to T1 in the experimental condition. With reference to the two-stage model, the continued decline in RT is consistent with the assumption that increasing the temporal lag between T1 and T2 reduces the delay of T2 in Stage 1, thus reducing the magnitude of the AB deficit.

The Lag-RT condition replicates and extends earlier findings obtained by Jolicoeur and Dell'Acqua (1998; see also Arnell & Duncan, 1997). By including a control condition in which T1 was ignored, we were able to establish that some of the decrement in RT across short lags (i.e., 100–200 ms) might be attributable to alerting effects triggered by the presentation of T1. This control condition was not employed by Arnell and Duncan (1997) and was employed only for lags longer than 300 ms by Jolicoeur and Dell'Acqua (1998). Second, the decline in RTs across lag found in the Lag-RT condition is similar to that found by Jolicoeur and Dell'Acqua. This confirms their suggestion that both cross-modal and uni-modal presentation of targets will produce a similar delay for T2.

Results in the Mask-SOA condition show that accuracy of T2 identification increased as the SOA between T2 and the trailing mask was increased (Fig. 2). This result is consistent with the assumption that T2 is vulnerable to masking while delayed in Stage 1. By increasing the SOA between T2 and the mask, the probability increases that T2 will have entered Stage 2 before the arrival of the mask, thus obviating an AB deficit on that trial. Homologous results have been reported by Giesbrecht and Di Lollo (1998, Exp. 3), who studied the effect of mask delay on AB magnitude with an RSVP paradigm, and by Jolicoeur and Dell'Acqua (in press, Exp. 2). The present results supplement these earlier findings by demonstrating the effect within a single modality using the two-target paradigm. In addition, the present results confirm the equivalence of the RSVP and the two-target paradigms as techniques for investigating the AB deficit (e.g., Ward, Duncan & Shapiro, 1997).

In summary, the findings of Exp. 1 are consistent with the assumptions of the two-stage model that T2 is delayed in Stage 1 while Stage 2 is busy and that, while delayed, T2 is vulnerable to masking. In Exp. 2, we expanded upon the findings of Exp. 1 by examining the effect of T1-masking on response times to T2 across a range of temporal lags.

Experiment 2

Two experimental paradigms have been used to study the AB deficit. One is the *two-target* paradigm used in Exp. 1 (Duncan et al., 1994). The other is the *rapid serial visual presentation* (RSVP) paradigm in which T1 and T2 are inserted in a stream of distractors, with all items presented in the same location at a rate of about 10 items/s (Raymond, Shapiro & Arnell, 1992). The form and temporal course of the AB deficit is approximately the same in the two paradigms (Ward et al., 1997).

A reliable finding obtained with the RSVP paradigm is that the AB deficit is much reduced, or even absent, if the item directly following T1 (the "+1 item") is blank (Chun & Potter, 1995; Raymond et al., 1992; Seiffert & Di Lollo, 1997). Within the two-stage model, this result can be understood if it is assumed, as did Seiffert and Di Lollo (1997) and Ward et al. (1997), that the +1 item acts as a mask, thereby increasing the difficulty of processing T1. Because it is more difficult to process, T1 keeps Stage 2 busy for longer when the +1 item contains a masking stimulus than when it is blank. As a consequence, the period for which T2 remains vulnerable to masking in Stage 1 is longer, with correspondingly greater probability of an AB deficit on that trial.

Experiment 2 was designed as a direct test of this hypothesis. The rationale was similar to that of Exp. 1: if it is the case that the processing of T2 is delayed when T1 is masked, then RTs to T2 should be longer when the +1 item contains a masking stimulus than when it is blank. We tested this prediction using the two-target paradigm as in Exp. 1. Observers made speeded responses to a non-masked T2 under two conditions. In one condition, T1 was masked; in the other, T1 was not masked. This was done on the assumption that the masking function served by the +1 item in the RSVP paradigm corresponds to that of the T1 mask in the two-target paradigm. As expected on the basis of the two-stage model, RTs to T2 showed a progressive decline over T1–T2 lags when T1 was masked, but not when T1 was not masked.

Method

Participants. Fifteen undergraduate psychology students (12 female) at the University of British Columbia participated for course credit. All participants reported normal or corrected-to-normal vision.

Apparatus and stimuli. Apparatus and stimuli were identical to those in Exp. 1.

Procedure. The sequence of events in Exp. 2 was the same as in the Lag-RT condition in Exp. 1, except that there were two blocks of trials. In one block (*Mask* condition), T1 was followed by a mask, as in Exp. 1, while in the other block T1 was never masked (*No Mask* condition). The order of the blocks was counterbalanced such that half the participants received the Mask condition first.

Results and discussion

Trials on which errors were made on either T1 or T2 were discarded from the analysis. This amounted to a total of 10.6% of trials. Response times from all other trials were screened using the outlier procedure described in Exp. 1 (Van Selst & Jolicoeur, 1994), which resulted in the removal of a further 2.3% of RTs. The remaining RT data were used to calculate means for each of the five T1–T2 lags in both conditions. These means and the average standard errors are shown in Fig. 3. Individual results were analyzed in a 2 (Mask vs. No Mask conditions) × 5 (T1–T2 lags) analysis of variance. The analysis revealed a significant effect of Lag, $F(4,56) = 7.09$, $p < 0.001$, $MSE = 8208.57$, and a significant interaction effect between Condition and Lag, $F(4,56) = 3.72$, $p = 0.009$, $MSE = 3840.92$. No other effects reached significance.

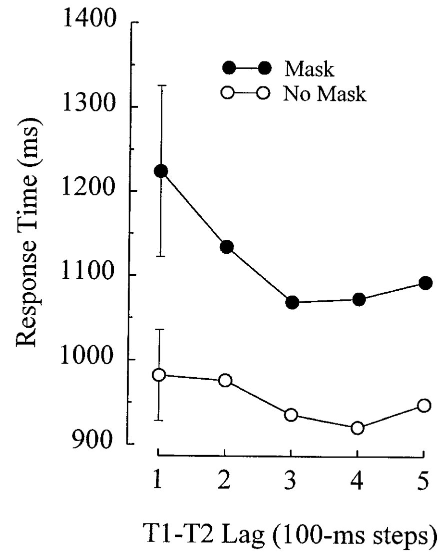

Fig. 3 Mean response time for T2 classification as a function of the temporal lag between the first and second targets for the Mask conditions. *Error bars* represent the average standard error of the mean

The results in Fig. 3 are entirely consistent with expectations based on the two-stage model. The decline in response times over lags was far more prominent in the Mask than in the No Mask condition, as confirmed by the significant Condition × Lag interaction effect. The steeper decline in the Mask condition verifies the assumption in the two-stage model that masking increases the difficulty of processing T1 and therefore increases the time for which Stage 2 is kept busy before T2 can be processed. When the T1–T2 lag is short, T2 is delayed in Stage 1 for a relatively long period, and the time required for its identification (RT) is correspondingly longer. At longer lags, on the other hand, more processing of T1 can be accomplished before the arrival of T2, the delay of T2 in Stage 1 is reduced, and RTs are reduced correspondingly. These contingencies do not apply when T1 is easy to process, as in the No Mask condition. In this case, Stage 2 becomes available at relatively short lags. This enables T2 to escape masking sooner, and the slope of RTs reaches asymptote at correspondingly shorter lags.

In earlier work, evidence that the +1 item acts as a mask for T1 was gathered exclusively with the RSVP paradigm (e.g., Chun & Potter, 1995; Raymond et al., 1992; Seiffert & Di Lollo, 1997). In the present experiment, the same masking action was demonstrated with the two-target paradigm. This buttresses earlier claims that the two paradigms are equivalent ways of studying the AB deficit (Ward et al., 1997).

General discussion

The principal objective of Exp. 1 was to examine two assumptions made in the two-stage model of the AB: that T2 is delayed in Stage 1 while Stage 2 is busy, and that T2 is vulnerable to masking by trailing stimuli while delayed. These assumptions are similar to those made in the dual-task model of Jolicoeur and colleagues (e.g. Jolicoeur, 1998, 1999; Jolicoeur & Dell'Acqua, 1998, in press) and reflect the close relationship of the two theories (Jolicoeur, 1999). Both assumptions had been examined in earlier studies in which the magnitude of the AB deficit itself was used as a dependent measure (Chun & Potter, 1995; Giesbrecht & Di Lollo, 1998). Namely, variables were manipulated that were presumed to modulate the delay of T2 or its vulnerability to masking, and their effects on the magnitude of the AB served as the basis for validating the assumptions. Experiment 1, however, examined the two assumptions independently of the AB deficit itself and, in so doing, provided converging evidence for their validity.

In addition, Exp. 1 confirmed the prediction of Jolicoeur and Dell'Acqua (1998) that both cross-modal (one visual target, one auditory target) and uni-modal (two visual targets) presentation will produce similar delays for T2. This suggests that a single, central mechanism may be responsible for producing an AB deficit in both auditory and visual modalities.

In Exp. 2, we tested the prediction of the two-stage model with respect to the effect of T1 masking on response times to T2. Within the two-stage framework, masking is thought to affect the difficulty of T1 processing. When T1 is not masked, processing is relatively easy, meaning that Stage 2 is kept busy with T1 for only a short time. This allows T2 to gain quick access to Stage 2 and thus avoid a period of delay in Stage 1. In contrast, when T1 is masked, it is more difficult to process, so Stage 2 is kept busy for longer. During this period, T2 remains delayed in Stage 1. Consistent with this account, when T1 was not masked, response times to T2 did not vary as a function of lag. This suggests that T2 did not experience a significant period of delay, even at the shortest lag. When T1 was masked, however, T2 response times declined as a function of lag. This pattern of results implies that processing of T1 delayed the processing of T2 and that this delay decreased as inter-target interval increased, resulting in faster response times to T2.

The relationship between T1-masking and the delay of T2 is clearly predicted by the two-stage model. However, with certain assumptions, it can also be accommodated within the dual-task model of Jolicoeur and colleagues. Arnell and Jolicoeur (1999) suggested that masking of T1 increases the time it takes the serial consolidation stage to store T1 in short-term memory. On this account, when T1 is presented without a mask, consolidation is relatively fast, and T2 gains access to the limited-capacity resources with little delay. In contrast, when T1 is masked, consolidation occurs more slowly, delaying T2 at the perceptual encoding stage, where it is vulnerable to decay. This decay occurs indirectly as a result of masking, which over-writes the representation of T2 at the sensory encoding stage (Jolicoeur, 1999).

One last point needs to be made. It is known that the AB deficit depends critically on backward masking of T2. That is, no AB deficit is obtained unless T2 is followed by a mask at an appropriate SOA (Giesbrecht & Di Lollo, 1998). On the face of it, this may suggest that there is no cost to processing T1 unless T2 is masked. In fact, the cost remains, but it is nor reflected in the *accuracy* of T2 identification. Rather, it is reflected in *increased delay* of T2 identification, as shown in Exp. 1 (Fig. 1A). On these considerations, it can be said that the primary consequence of processing T1 is to introduce a delay in the processing of T2. Mediated by this delay is the conventional AB deficit in T2 identification, which occurs when the mask is presented during the critical period.

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