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Intramodal and crossmodal processing delays in the attentional blink paradigm revealed by event-related potentials

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Abstract

In the attentional blink (AB), processing of a second target (T2) is impaired if it is presented shortly after the onset of a first target (T1), leading to a decrease in accurate report of T2 if T2 is masked. Some prominent theories of the AB suggest that an amodal bottleneck in working memory consolidation underlies the AB. We investigated this by factorially manipulating T1 and T2 modalities (visual or auditory) using equivalent stimuli and tasks in both modalities to minimize task switching. T2 was not masked. In all modality combinations, the electrophysiological P3 component to T2, obtained by subtracting T1 only trials from T1+T2 trials, was delayed and reduced in amplitude when T2 was presented soon after T1 relative to when T1 and T2 were presented farther apart. Results provide support for a common amodal bottleneck that underlies the AB effects observed in all visual/auditory modality combinations.

Descriptors: Human electrophysiology, Working memory, Bottleneck, Task switching

The attentional blink (AB) refers to a widely studied effect in which accurate report of a masked second target (T2) often suffers a severe decline if it is presented while a first attended target (T1) is undergoing processing by the cognitive system (Broadbent & Broadbent, 1987; Raymond, Shapiro, & Arnell, 1992). According to one class of models, central bottleneck models, limited capacity attentional resources are theorized to be the main cause of the AB. For example, Chun and Potter (1995) proposed a two-stage bottleneck model in which a high-level representation is created and meaning information is extracted in a first stage, and sustained attention resulting in consolidation of stimulus identity into working memory occurs in a second stage. This first, unconscious, stage is thought to have a very large processing capacity and can therefore process multiple stimuli in parallel. In contrast, the second, central, stage is thought to have severe capacity limitations in that targets must be processed serially. Many authors (Arnell, Helion, Hurdelbrink & Pasieka, 2004; Chun & Potter, 1995; DiLollo, Kawahara, Shahab, Ghorashi, & Enns, 2005; Jolicœur, 1998, 1999a, 1999b; Jolicœur & Dell'Acqua, 1998; Nieuwenstein, 2006) suggest that the bottleneck occurs at this second, postperceptual stage (i.e., after stimulus representation and at conscious identification and/or consolidation stages). Jolicœur (1998) argued that the second stage takes inputs from multiple sensory modalities and that the AB is thus caused by an amodal central bottleneck.

Interestingly, the AB is only observed when T2 is masked by interruption (e.g., by a backward pattern mask). Indeed, when T2 is not masked, there is no drop in report accuracy, no matter the time elapsed between T1 and T2 presentation (Giesbrecht & DiLollo, 1998). To explain this finding, central bottleneck models postulate that when T2 closely follows T1, and T1 is still undergoing central processing, T2 must wait for limited-capacity central mechanisms to become available. If T2 is trailed by a mask, the early representation of T2 will be overwritten by this mask before it can be consolidated in stage 2. However, when T2 is not trailed by a mask, the early representation of T2 can persist long enough to outlast the period of time during which T1 undergoes central processing. In this view, there are two key factors that lead to the behavioral manifestation of the AB. The first is a delay of processing of T2, in stage 2, that is caused by processing of T1 by limited-capacity central mechanisms. The second is the masking of T2 during this period of delay. If T2 is trailed by a mask, the initial representation of T2 can be overwritten during the delay, leading to a decrease in accurate report. However, if T2 is not trailed by a mask, the representation of T2 in stage 1 can survive the delay and be consolidated when central resources are freed from T1 processing.

The P3 component of the event-related potential (ERP) has been used to evaluate predictions made by central bottleneck

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models. The P3 is a large, positive, event-related potential (ERP) component, generally peaking between 300 ms and 400 ms after stimulus presentation. It attains maximum amplitude in response to infrequent and unpredictable stimuli (Donchin, 1981). However, it does not seem to be the probability of the physical stimuli per se that matters, but rather the probability of the class defined stimulus category (see Donchin & Coles, 1988; Kutas, McCarthy, & Donchin, 1977). Because P3 amplitude is sensitive to this probability of class-defined stimulus category, it logically ensues that it cannot be generated before the stimulus has been categorized. Moreover, the P3 latency and amplitude are relatively insensitive to factors that influence response selection processes (Magliero, Bashore, Coles, & Donchin, 1984). Consequently the P3 indexes central processes, existing in a rather narrow window between categorization and response selection, and has been suggested by many to reflect consolidation into working memory (e.g., Donchin, 1981; Luck, 1998). Furthermore, the larger amplitude of the P3 elicited by infrequent stimuli enables us to create infrequentfrequent difference waves (frequency related P3). This procedure allows for the elimination of all activity that is not sensitive to T2 frequency, including any overlapping T1 activity. Thus, in the context of the AB, the P3 is ideal to validate the central locus of interference postulated by many extent models of the AB. Indeed, Vogel, Luck, and Shapiro (1998) demonstrated that although perceptual components (P1, N1) for T2 were unaffected during the AB, the frequency-related P3 for T2 was virtually abolished at the shortest lag when T2 was masked, but only when both T1 and T2 required a response (in dual-task conditions). Indeed, in single-task conditions, when both T1 and T2 were presented but only T2 required a response, the P3 was unaffected by lag, strongly suggesting that the P3 effect observed in dual-task conditions was directly linked to T1 processing inducing the AB. In another experiment, Vogel et al. found that the N400 difference wave (sensitive to semantic match/mismatch) to T2 was as large during the AB as that observed at a longer T1–T2 interval (although the N400 was clearly reduced in dual-task conditions relative to single-task conditions). Vogel et al. interpreted the overall patterns of results as evidence that T2 was normally perceived (intact P1, N1) and even interpreted at a semantic level (no lag effect on N400), but not stored in working memory (abolished P3).

In a subsequent experiment, Vogel and Luck (2002) studied the P3 wave time-locked to the presentation of both masked and not-masked T2s. For a masked T2, when T2 was presented during the AB interval, both report accuracy for T2 and the size of the P3 were reduced, replicating earlier findings by Vogel et al. (1998; see also Dell'Acqua, Jolicœur, Pesciarelli, Job, & Palomba, 2003). For a T2 that was not masked, report accuracy for T2 was not reduced during the AB interval, replicating findings by Giesbrecht and Di Lollo (1998), nor was the amplitude of the P3 component. Importantly, however, the onset latency of the P3 was clearly delayed at short T1–T2 lags compared to long lags, suggesting that T1 processing (underlying the AB) caused a delay in the time at which T2 could be categorized (see also Arnell, 2006). The P3 delay to a T2 that was not masked, due to T1 processing, provides strong confirmation of a key claim of central bottleneck accounts of the AB (Jolicœur, 1998), namely, that central processing of T2 is delayed when T1 must be attended.

Most of the afore-cited studies were all conducted in the visual modality. There is mounting evidence that the AB can also be observed in the auditory (Arnell & Jenkins, 2004; Arnell & Jolicœur, 1999; Arnell & Larson, 2002; Duncan, Martens, & Ward, 1997; Mondor, 1998; Shulman & Hsieh, 1995; Soto-Faraco & Spence, 2002; Tremblay, Vachon, & Jones, 2005; Vachon & Tremblay, 2005, 2006) and tactile (Dell'Acqua, Jolicœur, Sessa, & Turatto, 2006; Hillstrom, Shapiro, & Spence, 2002) modalities, as well as across visual and auditory modalities (Arnell & Jenkins, 2004; Arnell & Jolicœur, 1999; Arnell & Larson, 2002; Jolicœur, Tombu, Oriet, & Stevanovski, 2002; Shulman & Hsieh, 1995), auditory and tactile modalities, and visual and tactile modalities (Soto-Faraco et al., 2002). Several studies in which a speeded response to T1 was required have similarly found evidence of an AB in various modalities (e.g., Dell'Acqua, Turatto, & Jolicœur, 2001; Jolicœur, 1998, 1999a, 1999c). Finding an AB in these various modalities, and importantly in cross-modal situations, provides support for the notion that at least part of the AB is due to a central bottleneck, given that such a central locus of interference should be independent of the sensory modality in which the stimuli were presented.

However, some results suggest that the AB effect may be reduced or even absent in auditory and cross-modal paradigms, as opposed to visual paradigms (e.g., Arnell & Jenkins, 2004; Soto-Faraco & Spence, 2002). Two differing hypotheses can explain this reduction or absence of the observed AB effect in auditory and cross-modal paradigms. The first does not exclude the possibility of a single, amodal, bottleneck on working memory consolidation to explain all AB effects, regardless of modality. Rather, the reduced or absent AB effect is explained by the fact that masking targets may be more difficult in some modalities than others (e.g., in audition; Arnell & Jenkins, 2004). The second hypothesis rejects the possibility of an amodal bottleneck and explains any observed cross-modal AB-like effect on a switch in task set required to process T1 and T2 (Potter, Chun, Banks, & Muckehoupt, 1998).

Using electrophysiological measures, Arnell (2006) showed that the categorization/identification of T2, as indexed by the frequency-related P3, was delayed across and within the auditory and visual modalities, in a design that eliminated differential masking as a possible explanation. This study included four experiments, each using a different combination of T1 and T2 modalities (visual–visual, auditory–auditory, visual–auditory, and auditory–visual), the first of which replicated Vogel and Luck's (2002) experiment with T2 stimuli that were not masked. In each experiment, a rapid serial visual presentation (RSVP) stream and a rapid auditory presentation (RAP) stream were presented concurrently. Independent of modality, the task always required unspeeded responses reporting the identity of a masked first target letter (T1) and the presence or absence of an unmasked X as the last item in the stream (T2). Participants were informed at the beginning of the experiment as to which stream (auditory or visual) would contain each target. T2 accuracy was at ceiling in all conditions; however, all four experiments yielded a P3 to T2 that was delayed at short lags compared to long lags, consistent with the hypothesis of an amodal bottleneck. However, the fact that the T1 and T2 tasks differed from each other raises the possibility of task switching as the underlying cause of the delayed P3. Moreover, the fact that participants knew in advance which streams were to be monitored, and in what order, raises issues related to differential preparation and possible modality switch costs. Consider, for example, the auditory–visual modality combination in the Arnell study. At the beginning of each trial, participants could have been more prepared for an auditory target and less prepared for a visual target. Differential preparation can sometimes have substantial effects on performance

(De Jong, 1995; De Jong & Sweet, 1994; Pashler, 1994). After the encoding of T1, participants might have shifted preparation to the visual stream. Presumably, the shift of preparatory state would follow the detection and encoding of the auditory T1 by some time. Consequently, the probability that preparation had become optimal for the visual target (T2) would have been greater at a longer T1–T2 lag than at a shot T1–T2 lag. Accuracy for T2 would increase as the interval between T1 and T2 increased and allowed preparation to shift from the auditory to the visual modality. Thus, the observed effects of T1–T2 lag on the T2-locked P3 component could have been due to changes in preparation rather than to an amodal central AB bottleneck.

The present study was designed to rule out the possibility of task-switching and differential preparation across modalities as the cause of the P3 delays observed within and across different modalities. To do so, our paradigm used an entirely within-subject within-session approach in which the task to be performed for T1 and T2 was identical in all sensory modalities. Both T1 and T2 were digits presented among letter distractors, and an unspeeded report of both targets was required at the end of the trial. This procedure eliminated the possibility that task-switch costs could contaminate the observed AB effects. Moreover, targets could be randomly presented in the visual or auditory modalities, unpredictably from trial to trial, so as to have all four combinations (visual–visual, auditory–auditory, visual–auditory, and auditory–visual) intermixed randomly in every test session. As the modality of T1 did not predict that of T2, subjects had to monitor both streams at all times, eliminating planned modality switches or differential modality preparation as possible contributors to any observed AB deficit.

Isolation of T2 ERP from Overlapping Activity

Dual-task experiments with short stimulus onset asynchronies (SOAs) between targets pose a special problem for the analysis and interpretation of electrophysiological data because of overlapping brain activity associated with rapidly presented targets and distractors. Vogel et al. (1998) provided an elegant solution to this problem by creating two types of RSVP sequences that were meant to be identical in all ways, except for the probability of presentation of T2. Because the P3 component is sensitive to the probability of a target stimulus, it was expected that a larger P3 would be elicited by T2 when the less probable target was presented relative to the P3 expected when T2 was the more probable target (e.g., Donchin, 1981). All other ERPs from T1 and distractors should summate in the ERPs to T2 to the same extent, regardless of the probability of T2. Subtracting the ERP to T2 for the frequent target from the ERP to T2 for the infrequent target should reveal just that portion of the ERP that was affected by the probability of T2, and this would be mainly the P3 component. This method allows researchers to pull out the frequency-related P3 response for a target embedded in a complex sequence of stimuli.

The subtraction method employed by Vogel et al. (1998) was also used by a number of other researchers working with AB paradigms involving short SOAs between stimuli (Arnell, 2006; Arnell et al., 2004; Dell'Acqua et al., 2003; Dell'Acqua, Jolicœur, Vespignani, & Toffanin, 2005; Sessa, Luria, Verleger, & Dell'Acqua, 2007; Vogel & Luck, 2002). One disadvantage of the method is that the frequency manipulation requires a large number of trials for each experimental condition in which one wishes to estimate the P3 response to T2, because each condition now has two subconditions (frequent T2, infrequent T2). Assuming a desired 3:1 ratio for the relative frequency of targets, each condition requires four times as many trials as required for the reliable estimation of the P3.

In our context, we wished to measure the P3 to T2 in eight conditions: four combinations of T1 and T2 modality (visual–visual, visual–auditory, auditory–visual, and auditory–auditory), crossed with two lags (a short lag in the region of high AB interference and a long lag outside AB interference). The novel aspect of our experiment was the within-subject, within-session manipulation of all factors, which was critical for avoiding task- and modality-switch costs. Use of a frequency-related P3 design (where frequent and infrequent P3 categories are used to isolate a P3 difference wave) would have required either extremely long recording sessions and/or multiple sessions. To address this concern, the frequency method to isolate the P3 was modified to reduce the number of trials and thus the overall length of the experiment. This was done by presenting two types of trials: T1 only trials (single-target trials, in one third of trials) and T1+T2 trials (dual-target trials, in the remaining two-thirds of trials). Single-target, T1-only trials produced ERPs to T1 and distractors in the absence of ERPs related to T2. Dual-target trials produced ERPs just like those in T1-only trials, plus activity produced by T2. By subtracting the ERP from single-target, T1-only trials from the ERP from dual-target, T1+T2 trials, we were able to isolate the ERP to T2.

Presentation of T1 on T1-only trials was identical in modality and stream position to its presentation in T1+T2 trials to ensure that the amount and timing of the T1 plus distractors ERPs would be identical to that in the T1+T2 ERP waveforms except for T2-related activity. This method of subtraction is generally more efficient than the frequency-related method because it (roughly) requires doubling the number of trials for any experimental condition, rather than quadrupling the number of trials to get the 3:1 frequency manipulation. Thus, generally, our approach would require about half as many trials, making it possible for us to estimate more conditions within a single experimental session.

As with any such subtraction method, the interpretability of the final results depends on the validity of the assumption that the ERPs in the two estimated conditions are in all ways identical, except for the desired difference (the ERP to T2, in this case). A slightly more relaxed criterion would be that the ERPs, should they not be completely identical (except for the response to T2), be constantly different across all experimental conditions, so as not to introduce differences confounding the main experimental manipulations in the experiment. In the present design, it was certainly the case that the two stimulation sequences were physically identical and processed under equivalent attentional states, which would equate their sensory ERPs. Perhaps the main issue concerns the impact of the presence versus absence of T2 on ERPs following the presentation of T2 (dual-target trials) or following the distractor we substituted for T2 (single-target trials). One third of the trials in the present design were single-target (T1-only) trials, and two thirds were dual-target (T1+T2) trials. Thus, the absence of T2 was a relatively frequent event, and this event was as frequent as the presence of T2 in the visual modality (one third of trials) or the auditory modality (one third of trials). Even if we were to suppose that the absence of T2 would be associated with a small P3 due to the surprise of not detecting T2 (this possibility is reduced because T2 was missed on a significant proportion of dual-target trials, bringing the functional relative

frequency of T2-absent trials closer to 50%), this effect would be the same, regardless of the modality of presentation of T1 and so could not differentially affect the subtraction waves for different T1 modality conditions. The same argument holds for different T2 modality conditions or for combinations of T1 and T2 modality conditions, which are the most critical for present purposes. Finally, one might wonder whether our subtraction method could distort results in different ways for short-lag versus long-lag trials, assuming, for example, that the AB phenomenon affects the P3 response of any stimulus, including the absence of a stimulus, in the same general way. In this case, one would expect a delayed P3 to the absence of T2 in short-lag trials relative to long-lag trials. Any such difference across lag, however, would be constant for the different combinations of T1 and T2 modality. Furthermore, any such effect would not cause the spurious appearance of a delay in the P3 observed when T2 was presented given that both the P3 to T2-present, and the P3 to T2-absent would be delayed by the AB. Thus, any small P3 associated with the absence of T2 would be subtracted from the P3 to T2-present trials, to the same extent for all conditions, including both SOA conditions. In summary, any small P3 response specifically associated with the absence of T2 in single-target trials would affect the overall measured amplitude of the P3 to the same extent in all conditions, but could not produce differential effects of modality or lag on the latency or amplitude of the P3 in the subtraction waveforms.

Method

Participants

Data were collected from 40 participants, 29 women and 11 men, with a median age of 20.4 years. All participants reported having normal hearing and normal (or corrected to normal) vision. Prior to analysis, data obtained from six of the participants were excluded because they displayed no discernible P3 wave in at least one condition.

Stimuli

Each trial was comprised of simultaneous RSVP and RAP streams, each having 18 randomly generated items with an SOA of 90 ms and no blank interstimulus interval. Stimuli in the visual stream were independent of the stimuli in the auditory stream, so that one stream could not be used to assist with performance on the other stream.

Distractors were letters (all except W in the auditory stream and I and O in the visual stream) and targets were the digits 1, 2, 3, or 4. T1 and T2 were chosen independently so that all combinations of targets (even T1–T2 repeats) occurred equally often. This ensured that the identity of T1 revealed nothing about the identity of T2 and that the nature of the processing and expectations about T2 would be identical to those for T1, again eliminating possible task differences.

All visual items were presented in bold 18-point black Courier New font (approximately 1.1° of visual angle) in the center of a light gray background.

Auditory items were recordings of spoken letters presented in compressed speech. Digitized vocal recordings of a male voice were collected using an Apple microphone and a Power Macintosh AV computer. Recordings were done using 16 bits of resolution for amplitude at a sampling rate of 47 kHz with the aid of SoundEdit 16 software. Each letter was then edited and compressed to 90 ms. Auditory stimuli were presented using speakers placed immediately to the left and right of the computer monitor.

Design

There were 12 conditions in this experiment. There were 8 dual-target conditions where both T1 and T2 were presented, created by a factorial combination of T1 modality (visual or auditory), T2 modality (visual or auditory), and lag (short or long). Thus, the two targets could be presented in the same sensory modality (visual T1 and T2 or auditory T1 and T2) at either short or long lags or in different modalities (visual T1–auditory T2 or auditory T1–visual T2) at either short or long lags. There were 4 single target conditions in which only T1 was presented (T1-only trials). These were created from a factorial combination of T1 modality (visual or auditory) and short or long lag (i.e., temporal distance of T1 from the end of the stream).

In single-target trials, the target position was matched with T1 position in the dual-target trials at both short and long lags, so that the lag in this case represented the temporal interval of the target from the end of the stream (i.e., where T2 would have been). In dual-target trials, T2, when it did appear, always appeared as the very last (18th) item in the trial. When the lag between T1 and T2 was short, T1 appeared two items before T2 (16th position). When the lag between T1 and T2 was long, T1 appeared eight items before T2 (10th position).

Each participant completed 960 trials in a single session, with a break after the first 576 trials. Trials from all 12 conditions were randomly presented so that the modality of T1 did not predict the modality, identity, or presence of T2. The only constraints were that two thirds of the trials were dual-target conditions (640 trials or 80 for each target modality and lag combination), one third of the trials were single target conditions (320 trials, or 80 for each modality and lag combination), and each condition occurred twice in every 24 subblock of trials.

As all T1+T2 combinations were possible and given that there were four possible targets (digits 1, 2, 3, or 4), 25% of all of the dual-target trials had the same digit for T1 and T2. We did not include repeated-digit trials in the analyses because of the possible influence of exact target repetitions at short lags, over and above expected AB effects, as found in the repetition blindness phenomenon (Chun, 1998). Because these repetitions were removed, there remained 60 dual-target trials to analyze for each modality and lag combination. Because there could be no repetitions for T1-only trials, there were 80 trials to analyze for each combination of single-target modality and lag.

Procedure

Each trial began with the presentation of a black fixation cross at the center of the screen for 500 ms, followed by a 500-ms blank interval before the start of the RSVP and RAP streams. The visual and auditory streams began at the same time, ran concurrently, and had 18 items each. The task was to identify two target digits among letter distractors. Participants were instructed to identify the targets as soon as possible after they were presented, but to hold their response until prompted at the end of the stream. Instructions stressed accuracy rather than speed. At the end of each trial a prompt for the identity of T1 was presented. Following this response, another prompt, for the identity of T2, was presented. A separate response key was to be used when T2 had not been presented (for T2-absent trials).

Electrophysiological Recordings

A Sony VIAO desktop PC with a 17-in. color monitor, running E-Prime (Schneider, Eschman, & Zuccolotto, 2002) was used to present stimuli and record behavioral responses. Neuroscan software running on a separate Sony VIAO dektop was used to acquire and analyze electroencephalographic (EEG) data recordings from 64 sites (cap by Electrocap International) referenced to linked earlobes. Electroocular (EOG) recordings were taken by affixing electrodes to the outer canthi of each eye and the top and bottom of the orbit of both eyes. Signals were amplified with a band-pass of 0.15 to 30 Hz, and digitized at a rate of 500 Hz. ERPs were time-locked to the onset of T2. Epochs began 200 ms prior to T2 presentation and ended 1000 ms after T2 presentation. Epoched waves were low-pass filtered with a value of 30 Hz.

Trials with eyeblink artifacts were corrected off-line during analysis using Neuroscan software. The algorithm calculates the amount of covariation between each EEG channel and a vertical EOG channel and removes the EOG from each EEG electrode on a sweep-by-sweep, point-by-point basis to the degree that the EEG and EOG covaried. If this correction appeared insufficient for a given trial, then the trial was removed by hand prior to averaging. For the 34 participants included in the analyses, 2.65% of all trials were rejected during our trial by trial visual inspection. Trials with incorrect T1 and/or T2 responses were also removed (14.9% of all trials).

Results

Behavioral Results

Accuracy for T1, dual-task trials. The mean accuracy and standard errors of report of the identity of T1 are shown in Table 1 for dual-target trials for each possible combination of T1 modality and T2 modality and lag. These means were submitted to a repeated-measures analysis of variance (ANOVA) with T1 modality, T2 modality, and lag as within-subject factors. The three-way interaction (T1 modality × T2 modality × lag) was significant, $F(1,33) = 15.70$, $p < .001$, $MSE = .003$. As can be seen in Table 1, mean accuracy for T1 was reduced at the short lag, relative to the long lag, but only for the auditory T1–visual T2 modality combination. Given that the joint effects of T1 modality, T2 modality, and lag were only prominent in the T1 auditory–T2 visual condition, we chose not to interpret this result as a general effect of modality switching on T1 accuracy. Indeed, T1 accuracy was similarly reduced in the auditory T1-only condition at the short lag, suggesting that lone auditory targets were more difficult to identify when they were presented earlier in the RAP stream.

The results shown in Table 1 also produced a variety of subsidiary main effects and two-way interactions, resulting largely from the three-way interaction pattern. There was a small, but significant, reduction in accuracy as lag was reduced, $F(1,33) = 11.72$, $p < .001$, $MSE = .047$. Accuracy was slightly lower, overall, when T2 was in the visual stream than when T2 was in the auditory stream, $F(1,33) = 25.56$, $p < .001$, $MSE = .112$. There was also a two-way interaction between T1 modality and T2 modality in which T1 accuracy was lower when T1 was visual than when T1 was auditory, when T2 was auditory, but the opposite was found when T2 was visual, $F(1,33) = 22.89$, $p < .001$, $MSE = .138$. Finally, there was an interaction between T2 modality and lag in which there was no effect of lag when T2 was in the auditory stream, but T1 accuracy was reduced for the short lag when T2 was in the visual stream. No other effects approached significance (all p's $> .283$).

Accuracy for T1, single-target trials. The mean accuracy and standard errors of T1 identification on single-target trials are shown in Table 2 for each T1 modality and each lag (although there was no second target, lag was coded for where T2 would have been presented on a dual-target trial). These means were submitted to a repeated-measures ANOVA with T1 modality and lag as within-subject factors. As in the analysis of dual-target trials, there was a small decrement in T1 accuracy at short lag (mean = .821) relative to long-lag trials (mean = .892), $F(1,33) = 13.43$, $p < .001$, $MSE = .013$, and no main effect of T1 modality, $F(1,33) = 3.129$. There was an interaction between T1 modality and lag, in which the effect of lag was larger when T1 was in the auditory modality than when T1 was in the visual modality, $F(1,33) = 15.875$, $p < .001$, $MSE = .005$.

Accuracy for T2. Mean accuracy for T2 was also computed for each combination of T1 modality, T2 modality, and lag and submitted to an ANOVA with these variables as within-subject factors. The means and standard errors can be seen in Table 3. Again we observed a three-way interaction between T1 modality, T2 modality, and lag, $F(1,33) = 9.26$, $p < .005$, $MSE = .017$. As can be seen in Table 3, although there was a tendency for lower accuracy for T2 at the shorter lag relative to the longer lag, this effect was particularly marked for the auditory–T1, visual–T2 condition where the lag effect is about twice as large as for any other combination of T1 and T2 modalities. Indeed, there was no lag effect when T1 and T2 were both in the auditory stream. These results match those found for T1 accuracy and suggest that the auditory–visual condition was particularly prone to produce T1 and T2 accuracy deficits at the short lag.

Several effects resulted from collapsing over the three-way interaction. Overall T2 accuracy was significantly lower at the short lag than at the long lag, $F(1,33) = 32.01$, $p < .001$, $MSE = .004$. Mean T2 accuracy was also higher when T2 was in the auditory modality than when T2 was in the visual modality, $F(1,33) = 16.43$, $p < .001$, $MSE = .019$. Again, there was a two-way interaction between T1 modality and T2 modality in which accuracy for T2 was generally better for an auditory T2 than a visual T2, but this difference was especially large when T1 was auditory, $F(1,33) = 24.53$, $p < .001$, $MSE = .004$. Finally, there was a significant interaction between T2 modality and lag, reflecting a larger effect of lag in accuracy for T2 when T2 was in

Table 1. *T1 Accuracy (Percentage Correct) for Dual-Task Trials*

	Visual T1–visual T2	Auditory T1–visual T2	Visual T1–auditory T2	Auditory T1–auditory T2
Lag 2	88.2 (0.017)	81.5 (0.029)	88.9 (0.019)	96 (0.011)
Lag 8	91.4 (0.017)	92.1 (0.13)	89.8 (0.017)	94 (0.012)

Standard error of the mean in parentheses.

Table 2. *T1 Accuracy (Percentage Correct) for Single-Task Trials*

	Visual T1	Auditory T1
Lag 2	85.9 (0.022)	78.3 (0.033)
Lag 8	88.1 (0.019)	90.2 (0.017)

Standard error of the mean in parentheses.

the visual stream than when T2 was in the auditory stream, $F(1,33) = 38.79$, $p < .001$, $MSE = .002$.

Electrophysiological Results

Figure 1 shows the grand average ERPs for each of the T1–T2 modality combinations. The ERPs displayed are from the midline parietal electrode site (Pz), where the P3 is typically maximal. Each panel shows the waveforms for either the long or short lag, time-locked to T2 on dual-task trials and to the last item in the stream (where T2 would have been) on T1-only trials. Notice in each panel that the initial portions of the waveforms are very similar for single-target and dual-target waveforms, supporting our assumption that single-target waveforms provide an unbiased estimate of brain activity generated by T1 and the distractors.

Figure 2 shows the difference waveforms obtained by subtracting the single-target waveform from the dual-target waveform for each combination of T1 and T2 modality and lag. These subtraction waves reveal the P3 response to T2, with the ERPs to T1 and to the distractors in the two stimulus streams removed. Several key results are immediately apparent by looking at these difference waveforms. First, the onset of the P3 to T2 was delayed in the short-lag condition relative to the onset in the long-lag condition by about the same amount in all four combinations of T1 and T2 modality. The return to baseline of the P3 was also delayed in the short-lag condition relative to the long-lag condition for all four combinations of T1 and T2 modality. These results suggest that the P3 component was shifted to a later time when T2 was presented at a short lag following T1, compared to the component latency when T2 was presented at a long lag following T1. This result replicates previous research using a different method to extract the P3 to T2 from overlapping activity associated with T1 and distractors (e.g., Arnell, 2006; Sessa et al., 2007; Vogel & Luck, 2002).

For each participant, separately for each condition, the P3 component was isolated using the subtraction waveform. Because we expected the latency of the P3 difference wave to differ for the short and long lag conditions, we did not use a common time window to delineate the P3 in all conditions. Instead, P3 isolation was done by finding the largest positive component within a window 250 ms to 700 ms after the T2 onset and marking the onset and offset of this component. Thus the onset, offset, and duration of the P3 window varied from participant to participant and condition to condition. These windows were used to calculate all P3 latency and amplitude measures used here. We examined these component latency differences by computing the 50% fractional area latency of the subtraction waveform for each lag and each modality condition for each participant. The fractional area latency is the latency at which a given proportion of the area of the component is reached. Thus, the 50% fractional area latency is the latency at which half of the area has been reached. The fractional-area latency measure is generally more accurate and yields higher statistical power to detect true component latency differences, without inflation of Type I error, relative to the peak latency of individual subject waveforms (Hansen & Hillyard, 1980; Kiesel, Miller, Jolicœur, & Brisson, 2008; see also Luck, 2005). However, all results reported here were also observed when peak latency was used as the dependent variable instead of the fractional area latency measure.

The 50% fractional area latency measure, for the dual-target minus single-target subtraction waves, was submitted to an ANOVA with T1 modality, T2 modality, and lag as within-subject factors. The mean component latencies can be found in Table 4. First, across all modalities, the latency of the P3 was longer for the short-lag condition (mean = 552 ms) than for the long-lag condition (mean = 472 ms), $F(1,33) = 63.62$, $p < .001$, $MSE = 6822.6$). Importantly, this lag effect did not interact with T1 modality, with T2 modality, or with the combination of T1 and T2 modality, $p > .28$ in all cases.

The ANOVA revealed a T1 modality × T2 modality interaction, $F(1,33) = 7.21$, $p < .01$, $MSE = 4561.6$. The mean latency was 505.6 ms for the visual–visual condition, 497.2 ms for the auditory–auditory condition, 533.4 ms for the visual–auditory condition, and 513.3 ms for the auditory–visual condition. Thus, the overall mean latency of the P3 (averaging across lag) was slightly shorter in the within-modality conditions (visual–visual, auditory–auditory) than in the between-modality conditions. Critically, however, this effect did not vary with lag, $F(1,33) = 1.16$, $p > .29$, $MSE = 3444.567$.

The grand average difference waves also suggest that there were differences in the amplitudes of the P3 waves to T2 across the various experimental conditions (see Figure 2). Component amplitude was estimated by computing the mean amplitude of the P3 component for each condition for each participant. P3 amplitudes were submitted to the three-factor ANOVA as above. The mean amplitude of the P3 was larger at the long lag (mean = 4.24 μV) than the short lag (mean = 3.01 μV), $F(1,33) = 43.13$, $p < .001$, $MSE = 102.416$. The P3 was also significantly smaller when T2 was in the auditory stream (mean = 2.89 μV) than when T2 was in the visual stream (mean = 4.36 μV), $F(1,33) = 73.82$, $p < .001$, $MSE = 146.758$. There was also an interaction between T2 modality and lag, where the lag effect on P3 amplitude was larger when T1 was in the visual modality (short-lag mean = 3.62 μV; long-lag mean = 5.09 μV) relative to the difference when T1 was in the auditory modality (short-lag mean = 2.40 μV; long-lag mean = 3.38 μV), $F(1,33) = 8.49$, $p < .006$, $MSE = 4.157$. Finally, the amplitude of the P3 also depended on the joint effects of T1 modality and T2 modality: Mean P3 amplitude was generally higher when T2 was in the visual stream than when T2 was in the auditory stream; how-

Table 3. *T2 Accuracy (Percentage Correct) for Dual-Task Trials*

	Visual T1–visual T2	Visual T1–auditory T2	Auditory T1–visual T2	Auditory T1–auditory T2
Lag 2	88.5 (0.023)	93.3 (0.011)	81.9 (0.028)	97.6 (0.007)
Lag 8	93.6 (0.020)	94.7 (0.011)	91.8 (0.020)	97.6 (0.004)

Standard error of the mean in parentheses.

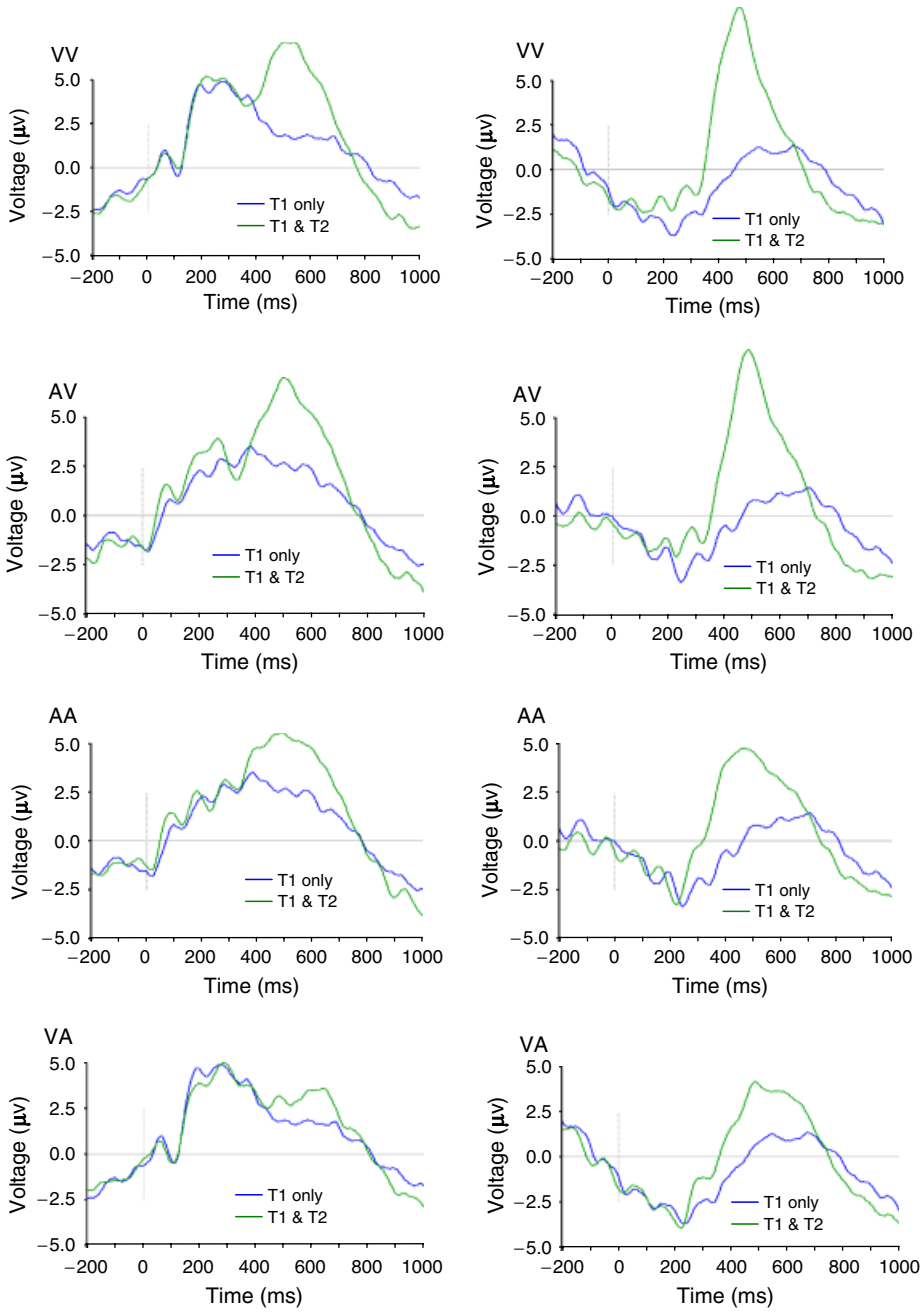

Figure 1. Grand average ERPs to T2 in dual-target trials and the last stream item for T1-only trials recorded from the midline parietal electrode site (Pz). The left column shows the short lag conditions and the right column shows the long lag conditions. The top row shows data from the visual T1–visual T2 condition, the second row shows the auditory T1–visual T2 condition, the third row shows the auditory T1–auditory T2 condition, and the bottom row shows the visual T1–auditory T2 condition.

ever, this difference was larger when T1 was in the visual stream (T2 visual mean = 4.66 μV; T2 auditory mean = 2.68 μV) than when T1 was in the auditory stream (T2 visual mean = 4.05 μV; T2 auditory mean = 3.09 μV), $F(1,33) = 15.61$, $p < .001$, $MSE = 17.572$. There were no other significant effects, and in particular, no three-way interaction between T1 modality, T2 modality, and lag, all $ps > .47$. The same pattern of results was observed when peak amplitude was used as the measure of P3 amplitude instead of mean amplitude.

Discussion

In the present study, we investigated whether an amodal bottleneck in working memory consolidation underlies the AB by measuring the P3 to the second of two targets, T2, under four combinations of modality of presentation of T1 and T2 (visual–visual, visual–auditory, auditory–visual, and auditory–auditory). In the present paradigm, differential effects of task switching and differential preparation for presentation modality of T1 and T2 were minimized. These goals were achieved by presenting T1 and T2 unpredictably in one of two concurrent streams of information, one visual and the other auditory. Because the modality of presentation of T1 was unknown at the beginning of each trial and because the modality of presentation of T1 provided no information concerning the modality of presentation of T2, participants were required to monitor both in-

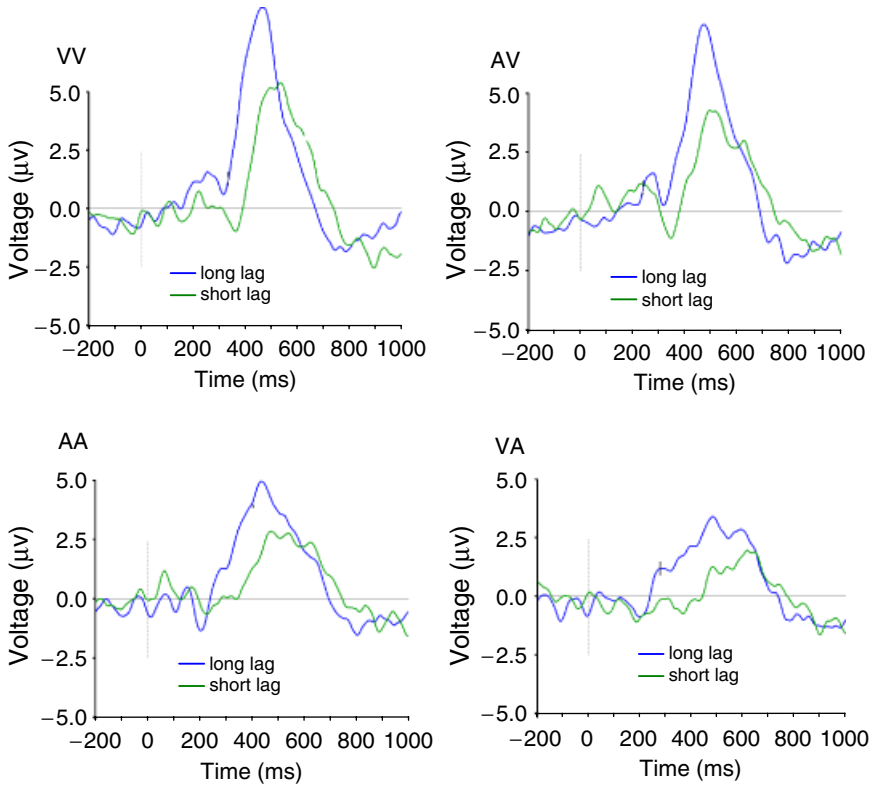

Figure 2. T2-locked P3 difference waveforms obtained by subtracting the single-target waveform from the dual-target waveform for each lag. The upper left panel shows the results for the visual T1–visual T2 modality combination; the upper right panel shows the results for the auditory T1–visual T2 modality combination; the lower left panel shows the results for the auditory T1–auditory T2 modality combination; the lower right panel shows the results for the visual T1–auditory T2 modality combination.

formation streams at all times in all trials. Hence, our paradigm eliminated differential preparation effects across the various T1 and T2 modality combinations. Both T1 and T2 targets were always digits presented in a background of letter distractors. Given that the same task was used for both T1 and T2 and the identity of T1 and T2 digits were independent, the paradigm also minimized any differential effects of task switching given that the same task had to be performed for T1 and T2 (i.e., the task was always to monitor both streams for the presence of a digit).

As pertains to the choice of the P3 as a measure of the attentional blink phenomenon, it is due to the fact that P3 delays attributable to T1 processing can be observed in the absence of a behavioral manifestation of the AB when T2 is not masked. Given that efficient masking is a critical factor for the observed amplitude of the behavioral AB and that masking targets may be more difficult in some modalities than others (e.g., in audition; Arnell & Jenkins, 2004), to avoid confounds, we chose to forgo this behavioral manifestation in favor of an electrophysiological counterpart, and thus we never masked T2.

The P3 results were clear-cut in that the latency of the P3 difference wave to the unmasked T2 was significantly delayed at short T1–T2 lags relative to the latency at long lags, by about the same amount, regardless of the T1–T2 modality combination. The present findings, in the visual–visual condition, replicate those reported by Vogel and Luck (2002). The present findings also corroborate and extend those of Arnell (2006) by showing that delays in the P3 wave for T2 can be obtained in all four modality combinations even when the paradigm is not compromised by task switching or preparation for modality switches. Potter et al. (1998) have argued that instances of cross-modal AB are artifacts of task switching. In contrast, using behavioral measures only, Arnell and Larson (2002) showed that reliable ABs could be observed for all four combinations of visual and auditory T1s and T2s under conditions that did not promote task or modality switching. The present results extend those of Arnell and Larson by providing electrophysiological evidence for T2 processing delays in all four modality conditions also under conditions that do not promote task or modality switching.

The fact that the P3 to T2 was delayed in all combinations of T1 and T2 modality and that there was no evidence that these delays were generated by different neural mechanisms brings us to the conclusion that there is a common underlying mechanism

Table 4. *Mean 50% Fractional Area Latencies (in Milliseconds) of the T2-Locked P3*

	Visual T1–visual T2	Visual T1–auditory T2	Auditory T1–visual T2	Auditory T1–auditory T2
Lag 2	547.4 (9.78)	574.6 (19.63)	543.4 (12.97)	543.0 (18.39)
Lag 8	463.4 (7.89)	491.3 (16.48)	483.2 (9.33)	451.4 (10.95)

Standard error of the mean in parentheses.

both within visual and auditory modalities and across these modalities. Indeed, the P3 component is sensitive to stimulus identification and categorization operations (e.g., Donchin & Coles, 1988; Luck, 1998). Therefore, the presence of significant P3 delays for T2s at short lags in all modality combinations argues for an amodal delay in operations occurring at or before stimulus identification and categorization. This amodal delay in T2 identification/categorization, in the absence of task-switch and modality-switch costs, supports theories postulating an amodal bottleneck in working memory as an important contributor to the AB phenomenon, (Arnell & Jolicœur, 1999; Arnell et al., 2004; Jolicœur, 1998, 1999a; Jolicœur & Dell'Acqua, 1998). According to this model, identification of a masked T1 results in a bottleneck of identification operations that delays conscious identification of T2 if T2 is presented soon after T1. If T2 is masked by a trailing stream item, the T2 representation is overwritten by the masking item, T2 accuracy is reduced, and an AB is observed. If T2 is not masked, then the stage 1 representation of T2 can often outlast the bottleneck, resulting in postponed, but accurate, identification of T2. Indeed, studies asking participants to make speeded identification responses to T2 have shown longer T2 response times at short lags relative to long lags, even when T1 and T2 have been presented in different stimulus modalities (Arnell & Duncan, 2002; Arnell et al., 2004; Jolicœur & Dell'Acqua, 1998, 1999). However, the use of speeded responses in these studies makes it difficult to know whether T2 response times were delayed at short lags because of identification/consolidation delays or later response selection delays. By using unspeeded T2 responses in the present study, but isolating the P3 ERP component to T2, we have shown that at least some of the processing delay occurred at or before stimulus identification/consolidation.

The electrophysiological approach allowed us to reveal not only large and consistent processing delays due to the AB effect, despite rather modest effects in terms of accuracy of report of T2, but also P3 amplitude effects. Although we found significant effects of lag on accuracy of report of T2, despite the absence of a mask following T2, these effects were relatively small in absolute terms (only 4%, compared with typical AB deficits of about 20% to 40% when T2 is masked). Nonetheless, it could be proposed that the significant drop in accuracy for T2 could explain, in part, the significant reduction in the amplitude of the P3 at the short lag relative to the amplitude at the long lag. However, the drop in accuracy was mainly observed when T2 was presented visually and therefore could not explain the reduction in P3 amplitude when T2 was presented in the auditory modality. Another possible account for the reduced P3 amplitude at the short lag could be formulated on the basis of a reduction of available resources (e.g., Arnell, 2006).

Finally, we note that we used a subtraction method that had not been used previously in the context of the AB paradigm. The method consisted of measuring the ERP to T1-only trials as well as to T1+T2 trials. T1-only trials reflected activity related to the processing of T1 and ERPs associated with the distractors, but not activity caused by T2. In contrast, ERPs from T1+T2 trials contained summed activity from all three sources of activation (T1, T2, and distractors). The subtraction of T1-only ERPs from T1+T2 ERPs allowed us to isolate activity specifically related to the processing of T2. For other successful uses of difference waves, see Brisson and Jolicœur (2007), Luck, Fan, and Hillyard (1993), Vogel and Luck (2002>), or Vogel et al. (1998). We note that the method used here, consisting of the addition of matched T1-only trials, was significantly more efficient that the method consisting of varying the relative frequency of T2 targets, which requires a significant imbalance in the number of trials in the frequent and infrequent conditions, and hence a larger number of trials overall, in order to estimate the conditions with the infrequent targets. The fact that our results converge nicely with those of Vogel and Luck (2002), Arnell (2006), and Sessa et al. (2007) provides further support for the particular subtraction method we used in the present experimental design.

The P3 latency results revealed a small but significant delay (and reduction in amplitude) when T2 was in a different modality from T1. We suspect that the detection of a target in one modality may have primed further processing of targets presented in that stream. This effect was small, however, and importantly, it did not interact with lag, suggesting that modality priming was independent of the AB per se, perhaps operating before or after the AB bottleneck. This interesting effect should probably be studied in future research.

Although the P3 results strongly indicate that an amodal bottleneck is a significant contributor of the AB phenomenon, part of the behavioral data suggests that the AB effect cannot be entirely accounted for by modality-independent factors. Indeed, a significant effect of lag on T2 accuracy was present for visual T2 items but absent for auditory T2 items. Although this does not contradict the importance of the amodal bottleneck, it may possibly suggest that visual and auditory targets have differential access to central resources such that the representation of a visual target is more likely to be lost as a result of a processing delay than that of an auditory target.

In conclusion, the present electrophysiological results provide strong evidence for the existence of an amodal bottleneck in working memory consolidation as a significant cause of the AB within and across the visual and auditory modalities.

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