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The Time-Course of Lexical Activation During Sentence Comprehension in People With Aphasia

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Abstract

Purpose—To investigate the time-course of processing of lexical items in auditorily presented canonical (subject–verb–object) constructions in young, neurologically unimpaired control participants and participants with left-hemisphere damage and agrammatic aphasia.

Method—A cross modal picture priming (CMPP) paradigm was used to test 114 control participants and 8 participants with agrammatic aphasia for priming of a lexical item (direct object noun) immediately after it is initially encountered in the ongoing auditory stream and at 3 additional time points at 400-ms intervals.

Results—The control participants demonstrated immediate activation of the lexical item, followed by a rapid loss (decay). The participants with aphasia demonstrated delayed activation of the lexical item.

Conclusion—This evidence supports the hypothesis of a delay in lexical activation in people with agrammatic aphasia. The delay in lexical activation feeds syntactic processing too slowly, contributing to comprehension deficits in people with agrammatic aphasia.

Keywords

aphasia; syntax; slow rise-time; rate of speech; online; priming; sentence processing; neurolinguistics

Complex sentence types are often used as a means to examine the operations underlying sentence processing in people with normal comprehension and to examine how comprehension goes awry in people with agrammatic (Broca's) aphasia (hereafter referred to as agrammatic aphasia). These constructions, which may contain long-distance dependencies, have been characterized as complex because they do not follow canonical word order (subject–verb–object, or S–V–O, in English). Listeners with agrammatic aphasia often demonstrate difficulty comprehending these types of sentences. Several syntactic accounts have attempted to explain this difficulty, including proposed deficits in assigning thematic roles to displaced arguments (e.g., Friedmann & Shapiro, 2003; Grodzinsky, 2000) or slowed syntactic processing routines (Burkhardt, Avrutin, Piñango, & Ruigendijk, 2008). In contrast, the delayed lexical access (DLA) hypothesis suggests that delayed lexical access feeds syntactic processing too slowly, leading to breakdowns of automatic structure building and contributing to the "syntactic" comprehension disorder that is observed in people with

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agrammatic aphasia (Love, Swinney, Walenski, & Zurif, 2008; see also Myers & Blumstein, 2005).

The DLA hypothesis suggests that delayed lexical activation should also be observed in relatively simple, canonical constructions, and it is this hypothesis that we investigate in the current study. Unlike some of our previous efforts, however, we charted lexical activation across several probe points during sentence comprehension to examine the time-course of activation and decay without the additional processing required for complex syntax. Our approach was two-pronged: First, we examined the time-course of lexical access in a group of young, neurologically unimpaired control participants. There is a surprising lack of information about what occurs after a lexical item is activated in sentences during comprehension of simple canonical constructions in unimpaired populations. Second, we examined the time-course of lexical access in a group of participants with agrammatic aphasia to observe if lexical access is indeed delayed relative to normal, unimpaired processing and to further examine its time-course.

We begin with a brief review of the relevant literature, first probing lexical access in young, neurologically unimpaired participants. Previous research has demonstrated that when an unimpaired listener encounters a lexical item (e.g., a noun) in a sentence, its various properties, including its meaning(s), are activated. Consider the following (from Swinney, 1979):

Rumor had it that, for years, the goverment
building had been plagued with problems.
The man was not surprised when he found
several insects*1 in the cor*2ner of his room. (1)

Using the cross modal priming (CMP) technique, Swinney (1979) presented sentences, and during the temporal unfolding of the sentence, Swinney visually presented letter strings momentarily, either at the lexical point of interest (at the offset of *insects*; at *1) or three syllables downstream from the lexical item (*2). The participant was required to listen to the sentences for meaning and to make a lexical decision on the visually presented probes. These probes either were associatively related to the lexical item (e.g., ANT – related to *insect*) or were unrelated control probes (e.g., SEW). Results showed that participants' reaction times (RTs) to the lexical decision were significantly faster to the related probe relative to the unrelated probe at both probe positions. Thus, the lexical item of interest (in this example, *insects*) primed its associate, suggesting that when a word is encountered in a sentence, its meanings are activated, and this activation continues for (at least) approximately three syllables.

There have been very few studies like this examining the time-course of lexical access in simple, canonically ordered sentences that use the CMP technique with neurologically unimpaired participants when the lexical item of interest was not ambiguous. Most of the studies have been concerned with how lexical ambiguities are processed given a neutral or biasing context (and so are not discussed here). However, there have been several studies examining lexical priming of unambiguous words in more complex noncanonical sentences with both neurologically unimpaired populations and populations with language impairments. Consider an example from one of the more recent studies (Love et al., 2008; Experiment 1):

The audience liked *the wrestler**1 that the*2
parish priest condemned___*3 for*4foul*5 language. (2)

In the object-extracted relative clause in Example 2, the direct object of the verb *condemned* (i.e., *the wrestler*) has been displaced to a position occurring well before the verb, leaving behind a "gap" or trace of the moved noun phrase. Using the CMP technique (described above), Love et al. (2008) measured priming at five positions (as shown above). Based on the observed priming patterns, unimpaired participants evinced access of the meaning of the filler of the gap (*the wrestler*) at the filler's offset (*1) and again at the gap (*3), with no priming found in between (*2).

This typical time-course of activation—immediate access, decay, then re-access at particular syntactic positions (e.g., gaps)—has not been observed in studies exploring online processing in individuals with agrammatic aphasia. Specifically, individuals who are agrammatic appear to show delayed activation of a noun's meanings during sentence processing, with lexical access not occurring until ~½ s past the appearance of the noun. This pattern has been observed using the CMP task (Love et al., 2008) as well as with the visual world eye-tracking method (Thompson & Choy, 2009). Using sentence (2) as an example, Love et al. (2008) found that participants with Broca's aphasia did not evince priming at the offset of the target noun (*1) but did 300 ms later (*2), and did not evince re-access at the gap (*3) but did 500 ms later (*5). Thus, the patterns observed for these participants with aphasia support the DLA hypothesis because the initial activation of the noun serving as the filler is delayed, as is its syntactically driven reactivation.

In addition, the findings (from Love et al., 2008, for example) further suggest that the deficit in understanding complex sentences with displaced arguments has its roots in a disorder of lexical access (see also Blumstein & Milberg, 2000; Myers & Blumstein, 2005; but see Ostrin & Tyler, 1993). Consistent with this suggestion, participants with Broca's aphasia evinced the normal access/re-access pattern (and improved off-line comprehension) when speech input was slowed, reinforcing the hypothesis that delayed lexical access underlies syntactic-like deficits and showing that the deficits could be overcome when the normal time constraints for re-access were relaxed (Love et al., 2008; Experiment 2). However, Thompson and colleagues (Choy & Thompson, 2010; Thompson & Choy, 2009) suggested that their findings from experiments using the visual world eye-tracking paradigm indicate delayed lexical access but relatively normal and on-time gap filling in participants with agrammatic aphasic. If Thompson and colleagues are correct, then the DLA hypothesis would suggest a disorder that is independent from any putative disorder in understanding complex sentences. We return to this point in our Discussion.

There are three important limitations to the work showing delayed lexical access, which suggest that we may have put the cart before the horse. First, as far as we are aware, there has been no priming study that has investigated the time-course of lexical activation with more than two probe positions. Second, the bulk of the evidence in people with aphasia has come from investigations of complex filler-gap constructions, as described above. Third, even in canonical sentences, the nature of the lexical access deficit in people with agrammatic aphasia is not clear. For example, Swinney, Zurif, and Nicol (1989) examined whether or not participants with agrammatic aphasia show the normal pattern of access of multiple meanings of ambiguous nouns during sentence comprehension. They found that their participants with agrammatic aphasia activated only the most frequent interpretation for an ambiguous noun in the immediate vicinity of the noun; only the secondary meaning was delayed. Given this result, we would assume that, when an unambiguous noun is encountered in a sentence, on-time lexical access should occur. Yet, in the Love et al. (2008) study described earlier, access of the meaning of an unambiguous noun did not occur until at least 300 ms after the noun for their Broca's participants.

These limitations of past work yield the following questions about the time-course of "normal" lexical access that we address in the current study:

- Does activation of a noun's meaning decay?
- If so, is this decay linear or is it nonlinear and U-shaped, essentially showing reactivation in sentences without syntactic dependencies?

The latter question comes from the possibility that a lexical item is cyclically activated throughout the time-course of processing, making it available for further operations of the sentence processor. Further, we noted above that reactivation effects have been ubiquitously observed in constructions that have displaced arguments (with, for example, object-extracted relative clauses) in neurologically unimpaired participants. It is thus reasonable to also ask whether such re-access effects will be observed in canonical-ordered sentences that do not contain displaced arguments. Adding to these is a question about agrammatic aphasia: Do participants with agrammatic aphasia evince delayed activation of the meaning of an unambiguous noun in canonical sentences?

These questions are critical to accounts of sentence processing (both in adults without brain damage and in adults with aphasia) for the following reason: Several studies have suggested that, through a process called "integration, " a word's meaning is merged into the context of the sentence (Cairns, Cowart, & Jablon, 1981; Friederici, Steinhauer, & Frisch, 1999; Marslen-Wilson, 1987; Myers & Blumstein, 2008; Swaab, Brown, & Hagoort, 1997). And perhaps not surprisingly, there are several accounts that suggest that one limitation in aphasia is a disorder in lexical integration (Thompson & Choy, 2009). However, if there is such a lexical integration problem, its source might very well be the initial delay in lexical access (or, alternatively, in reduced activation of the lexical item; see Yee, Blumstein, & Sedivy, 2008). We consider delayed lexical access and its possible relation to a disorder of lexical integration in the Discussion section.

Experiment 1: Neurologically Unimpaired Participants

We examined these issues of the time-course of lexical access in simpler, canonical constructions in a group of college-age neurologically unimpaired participants in order to (a) reveal the time-course of lexical processing in canonical sentences, and (b) determine a standard of comparison for our participants with left-hemisphere damage (LHD) and agrammatic aphasia (Experiment 2).[1]

Method

Participants—One hundred and fourteen college-age students (*M* = 20.9 years, *SD* = 3.4, range: 18–33 years; 17 male, 97 female) from San Diego State University participated in this study. All participants were neurologically unimpaired right-handed native English speakers with no history of learning disability, head injury, or neurological disease, and no uncorrected vision or hearing impairments. Participants received course credit for their participation in the study.

Task—We used a cross modal picture priming paradigm (CMPP; Swinney & Prather 1989). Here, participants listened to uninterrupted auditory sentences and made binary, *human (yes)/not human (no),* decisions regarding visually presented black-and-white line drawings (visual probes). Each experimental sentence had two visual probes (each requiring

[1]Prior published reports have shown no differences in priming patterns in CMP tasks when comparing college-age and older age-matched control participants (Love et al., 2008 and references therein; Stern, Prather, Swinney, & Zurif, 1991). Even so, we also include a small group of age-matched controls and describe our results in footnote 3.

a *yes* response): a related probe that was a representation of the noun phrase in the direct object position of the sentence, and a control probe that was unrelated to any word in the sentence (Figure 1). In this task, the visual probes are presented at key times during the ongoing sentence, and priming effects are measured by comparing RTs to the related and control probes at that point; faster RTs to the related probes indicate a priming effect. Importantly, priming effects in cross modal tasks reflect activation of the sentential prime at that point in the ongoing auditory sentence, not the integration of the visual probe into the sentence (Nicol, Swinney, Love, & Hald, 2006).

Materials—We created 40 experimental sentences with the following canonical sentential structure:

Subject	Verb	Object	Prepositional Phrase	
The boxer	punched	he golfer^{*1}	after^{*2}the tre^{*3}mendou^{*4}sly antagonistic discussion about the title fight	(3)

In these sentences, the direct object noun (Object) was the lexical item of interest (e.g., *golfer* in Example 3 above) and was followed by a prepositional phrase that was designed to avoid the occurrence of another noun, and its lexical activation, within 2,000 ms of the direct object noun.

The experiment employed a switched target design in which the related probe for one sentence appeared as a control probe for a different sentence, whose related probe served as the control probe for its paired sentence (Figure 1). Thus, over all of the sentences, the set of related probes was identical to the set of control probes, minimizing the possibility that any observed priming effects are due to unimportant aspects of the pictures that might influence participants' RT (e.g., visual complexity differences).

Probe pictures were created by a cartoonist who drew black-and-white line drawings, each depicting the critical nouns for each of the 40 experimental sentences. The cartoonist also drew black-and-white line drawings for the filler sentences (described in more detail later). To ensure that the drawings accurately depicted the appropriate noun, all of the pictures were pretested on a group of 38 University of California, San Diego undergraduates (*M* age = 20.3 years, *SD* = 1.4; 15 male, 23 female) who participated for course credit. Participants were asked to label each of the drawings. Any drawing for which there was <75% agreement in labeling was removed and was modified by the cartoonist for clarity.

In order to establish the time-course of activation of the direct object noun, the related and control visual probes were presented at four positions in the ongoing auditory sentence (indicated approximately by the superscript numerals in Example 3 earlier). Probe position (PP) 1 occurred immediately at the offset of the direct object noun. Each subsequent probe position was 400 ms downstream from the previous probe position. This allowed us to chart a pattern of lexical activation of the direct object noun across 1,200 ms (PP4) after it was heard in the ongoing auditory stream.

In addition to the 40 experimental sentences, 60 filler sentences were created. Forty of the filler sentences were similar in length and structure to the experimental sentences but were paired with an animal (nonhuman) picture probe (requiring a *no* response). The other 20 filler sentences were of a different syntactic structure, to provide a variety of sentence forms. Of these 20 sentences, 10 were paired with a human picture probe, and 10 were paired with an animal picture probe to balance the number of "human" and "non-human" responses across the full set of items. As is standard practice in CMP tasks, the position of the visual probe varied for the filler sentences, with probes appearing equally often near the beginning, middle, and end of the sentences. This was done to prevent the anticipation of probe positions. Finally, 10 practice sentences (also balanced for the number, order, and type of response) were added to the beginning of each script to familiarize the participant with the task as well as to allow the experimenter the opportunity to monitor the participants to ensure that they understood the task.

After the 10 practice items, the remaining 100 sentences (40 experimental; 60 filler) were intermixed and were pseudo-randomly ordered such that no more than three sentences in a given condition or with a particular response occurred in a row. The sentences were recorded by a female native English speaker at a normal rate of speech (4.6 syllables per second). Recording and editing were performed using Adobe Audition 3.0 software.

Design—We used a mixed between-within factors design to counterbalance all eight conditions (4 probe positions × 2 related/control probes). Given the number of factors in this study, we wanted to ensure that each participant contributed data to more than one probe position and to both related and control probes, so that each participant provided his or her own control data. This was accomplished by randomly assigning an individual participant to any two probe positions (e.g., PP1 & PP2, PP2 & PP3, etc.), with related/control probes counterbalanced across two visits.

Procedure—At the beginning of each session, participants were instructed that they would be performing a dual task—listening to sentences for comprehension and responding to a picture probe that would appear centrally on a screen at a given point during the unfolding of a sentence. When they saw the picture, they were to make a yes/no decision as quickly and accurately as possible (using a two-button response box) as to whether the picture was human (*yes* response) or not human (*no* response).

To encourage attention to the sentences and keep participants on task, participants were told that it was important for them to listen carefully to each of the sentences as they would be asked comprehension questions at various points during the session. These questions bore only on the setting or general topic of the sentence and were intended only to reinforce the need for the participants to listen to the sentences. Tempo (Version 2.1.5) software was used to control the timed presentation of auditory and visual stimuli and the collection of participant responses (both yes/no decisions and millisecond accurate RTs for each decision). Each visual probe was presented for 1,000 ms, or until a response was made. Responses were recorded for up to 1,000 ms following the appearance of the picture probe, for a total time of 2,000 ms. An interstimulus interval (ISI) of 2,000 ms followed each sentence.

Analysis—All participants performed above chance on the task-related comprehension questions. Before analysis, two sentences were found to have script errors; all data from these two sentences and their paired sentences were removed (four sentences total). Data were first reviewed for button-press accuracy. Participants who responded incorrectly (wrong button press or no response) to >10% of the experimental sentences in either session were excluded ($n = 7$). For the remaining 107 participants, incorrect responses (2.03% of all data), RTs <300 ms, or overall outliers >1,150 ms (1.5 times the interquartile range, based on all correct responses) were removed (3.9% of the data), distributed roughly equally across participants and conditions. Finally, we screened out responses for each participant that were >2 *SD*s from his or her individual mean for each session (4.3% of the data). After completing this screening protocol, we eliminated seven of the 107 participants who were found to have mean RTs that were >2 *SD*s from the average of the participant means.

Analysis of the RTs from the remaining 100 participants was conducted using restricted maximum likelihood (REML) in a mixed-effects regression model. Included in this model were the crossed random effects on the intercept of participant and sentence and fixed effects of probe position (1 vs. 2 vs. 3 vs. 4), relatedness versus control, and their interaction.[2] The models were fit with an unstructured covariance matrix for each random effect. Follow-up models examined the interaction of probe position and relatedness separately for each pair of probe positions and are presented in the Results section accordingly. Type III *F* tests are reported for main effects and interactions. All analyses were conducted using SAS Version 9 software.

While the fixed and random effect terms were entered into the model per our design and hypotheses, we also examined the justification of the crossed-random effect structure using the Bayesian information criterion (BIC; Schwarz, 1978) to evaluate model fit (a relative measure in which smaller values indicate better fit). With no random effects, the overall model (i.e., including all four levels of probe position) had BIC = 83,591.6, which improved to BIC = 80,129.2 when the random effect of participant was added, and improved again to BIC = 80,027.8 when the additional random effect of sentence was added, thus justifying the inclusion of both random effects. The follow-up models on pairs of probe positions showed similar improvements to model fit. Note that differences in BIC values >10 constitute very strong evidence of a better fit for the model with the smaller score (Kass & Raftery, 1995).

We predicted a priming effect at PP1, at the offset of the lexical item of interest in the sentence. We also predicted a null effect at PP2, given the Love et al. (2008) findings that activation decays at ~300–400 ms. Our predictions for the remaining two positions were less concrete, but we entertained the possibility that reactivation would occur at one of these positions; for example, if a working memory system requires the availability of a lexical item for further operations of the sentence processor. Thus, and central to the hypotheses under investigation, for a priori planned comparisons of related and control probes at each probe position, we computed *t* tests of the differences of the least square means from the full model. All *p* values are reported two-tailed. Degrees of freedom (reported in the *t* tests below) were computed using the Satterthwaite approximation. Note that the degrees of freedom are large because in these models, they are based on the number of data points, not the number of participants or items. For further discussion of these statistical methods, see Baayen (2004, 2008) and Littell, Milliken, Stroup, Wolfinger, and Schabenberger (2006); for studies with similar analyses, see Love, Walenski, and Swinney (2009) and Walenski, Mostofsky, and Ullman (2007).

Results

Mean RTs and standard errors for each condition are shown in Table 1. A priori planned comparisons revealed significantly faster RTs for picture probes when related to the sentence prime than when unrelated to the prime, at PP1, 13 ms control – related difference; $t(6386) = 2.33$, $p = .02$, but not at PP2, −4 ms control – related difference; $t(6387) = 0.78$, $p = .43$, PP3, 5 ms control – related difference; $t(6387) = 0.96$, $p = .34$, or PP4, −8 ms control – related difference; $t(6386) = 1.24$, $p = .21$. Overall, there was a significant interaction between probe position and relatedness, $F(3, 6387) = 2.66$, $p = .047$, which is consistent with changes in related/control differences across the sentence. Neither the main effect of probe position, $F(3, 639) = 0.63$, $p = .60$, nor relatedness, $F(1, 6387) = 0.29$, $p = .59$, reached significance.

[2]This model combines traditionally separate *F*1 and *F*2 analyses into a single statistical test.

In order to further pin down the time-course in the decay of activation across the sentence, we examined the change in activation (i.e., the interaction between probe position and related/control differences) across each pair of probe positions beginning with PP1, the point just at the offset of the lexical item in the sentence. The Probe Position × Relatedness interaction was significant for PP1 versus PP2, $F(1, 3301) = 4.84$, $p = .03$, not significant for PP1 versus PP3, $F(1, 3309) = 0.84$, $p = .36$, but again significant for PP1 versus PP4, $F(1, 3155) = 6.15$, $p = .01$.[3] These results will be discussed after we present Experiment 2.[4]

Experiment 2: Participants With LHD and Agrammatic Aphasia

We examined whether individuals with agrammatic aphasia evince a similar pattern of lexical activation and decay as neurologically unimpaired individuals. According to the DLA hypothesis (Love et al., 2008), we expected a delay in the rise-time of a priming effect for participants with agrammatic aphasia, suggesting that significant priming should not be observed at PP1. Other hypotheses, such as the slowed-syntax hypothesis (Burkhardt et al., 2008), would not predict such a delay in canonical sentences.

Method

Participants—A total of eight participants with unilateral LHD and agrammatic aphasia met criteria for participation (mean age at time of testing: 56.7 years; range: 32.7–86.7 years). Demographic information for these participants is presented in Table 2. All participants had experienced a single, unilateral left-hemisphere stroke and were native English speakers with normal or corrected-to-normal visual and auditory acuity. They were right-handed before their stroke. Diagnosis of agrammatism was made based on the administration of standardized language testing to determine each participant's severity of agrammatic language impairment; specifically, fluency and auditory comprehension ability. Testing included the Boston Diagnostic Aphasia Examination—Third Edition (BDAE–3; Goodglass, Kaplan, & Barresi, 2000), Western Aphasia Battery (WAB; Kertesz, 2006), and SOAP (Subject-relative Object-relative Active and Passive) Test of Auditory Sentence Comprehension (Love & Oster, 2002).

As the DLA hypothesis makes the specific claim that a lexical deficit underlies comprehension deficits in people with agrammatic aphasia, participants were considered for inclusion if they showed evidence of such comprehension deficits, which we defined here as at or below chance on comprehension of sentences with noncanonical order (object-relative and passive sentences) from the SOAP. All participants were neurologically and physically stable (i.e., at least 6 months post onset), with no history of active or significant alcohol and/or drug abuse, active psychiatric illness or intellectual disability, and/or other significant brain disorder or dysfunction (e.g., Alzheimer's/dementia, Parkinson's, Huntington's, Korsakoff's). Participants were tested at the Cognitive Neuroscience Laboratory at San Diego State University and were paid $15 per session.

Materials—The same materials as those used in Experiment 1 were used.

Design—Unlike the mixed-factorial design that was used in Experiment 1, here we employed a fully within-subjects design for the participants with aphasia. The four probe

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[3]Additional interactions were not significant for PP2 versus PP3, $F(1, 3136) = 1.56$, $p = .21$, PP3 versus PP4, $F(1, 2996) = 2.38$, $p = .12$, or PP2 versus PP4, $F(1, 2984) = 0.15$, $p = .70$).

[4]We also tested a group of four age-matched controls (relative to our group of participants with aphasia tested in Experiment 2) to corroborate our past work suggesting no differences in activation patterns based on age. And indeed, even with this small *N*, we observed a 15-ms priming effect at the first probe position that approached significance ($p = .07$), with no priming at the other three positions.

positions, combined with the related/control variable, yielded eight conditions. In this design, each participant heard every sentence and saw every picture in every condition, and these were counterbalanced across sessions so that no one sentence or picture was repeated in any given session. The order of presentation of the eight conditions was counterbalanced across participants. Sessions were separated by at least 1 week, and most often, 2 weeks.

Procedure—The general procedure was the same as that used in Experiment 1, except that (a) responses were recorded for up to 3,000 ms from the onset of each picture and (b) a more extensive training session was implemented.

Training protocol: To ensure that our participants with aphasia were performing reliably on the binary picture decision task and that they understood this dual-task paradigm, a training session was given before the experimental script at each of the eight visits. For this training, participants were trained on the binary picture decision. Participants were told that a picture would appear in the middle of the screen and they were to identify whether the picture was human or not by pressing a button labeled *yes* for human or *no* for not human, as quickly as possible. The list consisted of 20 items: 10 human and 10 nonhuman. This picture-only training list was repeated as many times as needed until the participant was able to reliably perform the task. None of the pictures used in the training task was repeated in the scripts. Participants were instructed to make their responses using the hand ipsilateral to their lesion (left hand). Once the experimenter felt that the participant understood the binary decision task and was ready to move on to the dual-task experiment, participants were then given the instructions as described in Experiment 1.

Analysis—All participants performed above chance on the task-related comprehension questions. All participants responded to the button-press decision with >90% accuracy (M = 98.8%; SD = 2.2%; range: 93%–100%). Before analysis, incorrect responses (1.2% of all data) were removed. There were no RTs <300 ms or >2,000 ms; thus, no overall outliers were removed. A 2-SD participant screening was then performed for each participant separately for each condition (5.1% of the data). In all other respects, the data were analyzed the same way as for Experiment 1. The inclusion of crossed random effects was examined as in Experiment 1, with model fit improving from BIC = 30,725.8 with no random effects, to BIC = 28,353.4 adding the random effect of participant, and to BIC = 28,333.5 adding the random effect of sentence, thus justifying the inclusion of both random effects.

Results

Mean RTs and standard errors for each condition are shown in Table 3. As for Experiment 1, the a priori comparisons of the related and control probes for each probe position are of primary importance to address the question of when (i.e., at which probe position) any priming effects reach significance. A priori planned comparisons of the RTs for related compared to control picture probes revealed a nonsignificant priming effect at PP1, 10 ms control – related difference; $t(2347) = 1.37$, $p = .17$, a significant priming effect at PP2, 18 ms control – related difference; $t(2347) = 2.41$, $p = .02$, and nonsignificant control – related differences at PP3, 0 ms; $t(2347) = 0.16$, $p = .87$, and PP4, 6 ms; $t(2358) = 0.49$, $p = .62$. The overall interaction between probe position and relatedness did not reach significance, $F(3, 2349) = 1.02$, $p = .38$, though main effects of probe position, $F(3, 2347) = 12.1$, $p < .0001$, and relatedness, $F(1, 2353) = 4.9$, $p = .03$, did. Despite the nonsignificant overall interaction, we also examined the change in priming over the course of the sentence, as in Experiment 1.

To examine whether there was any change in activation across the sentence, we looked at the interaction between probe position and related/control differences across each pair of probe positions. Unlike what we found for the control participants, none of these interactions reached significance. However, the change in related/control differences between PP2 and PP3 and PP4 combined yielded an interaction with trend significance, $F(1, 1752) = 2.88$, $p = .086$, suggesting that the priming effects are indeed decaying for the participants with agrammatic aphasia as the sentence continues.

Discussion

The primary intent of this study was to investigate the time-course of lexical activation and decay in simple, canonical constructions both in young, neurologically unimpaired control participants and in participants with agrammatic aphasia. Our first aim was to map the normal time-course of lexical access during ongoing sentence processing. Our second aim was to examine the accounts that have been proposed in the literature regarding the underlying comprehension deficit in agrammatic aphasia; specifically, the DLA hypothesis (Love et al., 2008; Prather, Zurif, Shapiro, & Swinney, 1991; Swinney, Zurif, Prather, & Love, 1996), the slowed syntax hypothesis (Avrutin, 2006; Piñango, 2000), and the lexical integration deficit (Thompson & Choy, 2009).

We first discuss the data from Experiment 1 exploring lexical access in our young, neurologically unimpaired control group. We predicted that unimpaired participants would activate the meaning of an unambiguous noun at that word's offset and that the activation would decay soon after. What was unknown were details about the time-course of decay; whether or not, for example, the decay would be gradual and linear across the sentence, or whether the activation of the noun's meaning would decay rapidly and then reappear further downstream.

The results from Experiment 1 revealed significant priming at the offset of the target noun and no further significant priming effects for the remainder of the probe positions. These patterns suggest that the meaning of an unambiguous noun in a sentence is activated immediately when that noun is encountered, and then decays rapidly (by 400 ms). This rapid decay is buttressed by the finding of a significant interaction between the first two probe positions.[5]

For our participants with agrammatic aphasia, we predicted that the initial access of a noun's meaning during sentence comprehension would be delayed relative to the normal pattern. The results indeed showed no significant priming at the offset of the noun, but significant priming was observed 400 ms from the initial position. Even so, there was a 10-ms priming effect at the offset of the noun, suggesting that activation of the meaning of the noun had begun but had not reached threshold. Furthermore, we observed a remarkable similarity of the activation curves between the two participant groups (see Figure 2), with the patterns for our participants with agrammatic aphasia pushed downstream relative to the patterns of the neurologically unimpaired participants. These patterns strongly support the DLA account of agrammatic aphasia described in the introduction.

One important corollary of the DLA hypothesis is that it is the source of the syntactic comprehension deficits that are evinced by some participants with agrammatic aphasia (Love et al., 2008). Recall from the introduction that these individuals not only show delayed lexical access, but they also show delayed re-access effects *downstream* from the

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[5]There is an upward tick at the third probe position after the rapid decay found at the second position, and there is no interaction between the first probe position at the offset of the lexical item and the third probe position (800 ms past the initial activation). This pattern might suggest that the meaning of the noun is becoming active again well after its initial activation. The functional role of such reactivation would be to make the item available for further operations of the sentence processor. Although our results are empirically too thin to allow any strong conclusions regarding this hypothesis, these data hint that re-access effects in canonical structures remain a possibility; additional experiments should be able to resolve this issue.

gap position, when such effects are typically found in the immediate vicinity of the gap for healthy participants in sentences that have displaced arguments. Though in principle there could be two independent disorders—delayed lexical access and a syntactic comprehension disorder—a more parsimonious explanation is that there is a single processing disorder, and that the delay in lexical access disallows the on-time construction of long-distance dependencies, resulting in an easily observable off-line comprehension disorder. This account is buttressed by the finding that slowing down speech input for participants with Broca's aphasia results in both on-time lexical access as well as on-time gap filling. Furthermore, slowing down input yields better off-line performance as well (Love et al., 2008).

However, we noted in the introduction that recent evidence from eye-tracking studies has suggested on-time gap filling for participants with agrammatic aphasia (Thompson & Choy, 2009), contrary to what we have found using cross modal methods (Love et al., 2008; Love, Swinney, & Zurif, 2001; Swinney et al., 1996). The off-line comprehension deficit, according to this view, stems from an additional deficit—a "lexical integration" problem. The evidence for this account comes from data that purport to show delayed looks to a relevant picture of a filler noun phrase when it is mentioned in the sentence, but on-time looks to the picture of the filler when the listener encounters a gap. Although our present study only examined lexical access during the comprehension of canonical structures, it is critical to the evaluation of the DLA hypothesis— and its corollary of delayed lexical *re*-access in syntactically complex structures—that the evidence for intact gap filling in agrammatic aphasia be closely scrutinized.

Consider, then, the results reported in Thompson and Choy's (2009) important effort, where they examined processing of object-extracted *who* questions (Experiment 2) and object-extracted relative clauses (Experiment 3). An example from Experiment 2 reported in Thompson and Choy is shown below:

$$\begin{array}{c}\text{This story is about a boy and a girl.}\\ \text{One day, they were at school.}\\ \text{The girl was pretty, so the boy kissed the girl.}\\ \text{They were both embarrassed after the kiss.}\\ \textit{who did the boy kiss that day at school}?\\ (\textit{wh}-\text{question probe})\end{array} \qquad (4)$$

A visual array was presented to the participants as they were listening to the story, and the eye movements to the pictures in the array were recorded. In the current example, a picture of a "girl" (the relevant item that should be "activated" in the *who*-question above), a "boy" (a relevant distractor that is mentioned in the sentence), "schoolhouse," and "door" were presented in a four-picture square array. For analysis, the auditory input for the *who*-questions was divided into regions:

$$\text{who did } \frac{\text{Region 1}}{\text{the boy}}\ \frac{\text{Region 12}}{\text{kiss}}\ \frac{\text{Region 3}}{\text{that day at}}\ \frac{\text{Region 4}}{\text{scholl?}} \qquad (5)$$

Note that the *who*-question involves a displaced direct object argument (i.e., *who,* displaced from its canonical position that occurs directly after the verb *kiss*), which gets its reference from the preceding context ("the girl was pretty, so the boy kissed the girl"). Thus, the idea behind this experiment was to examine if participants with agrammatic aphasia look to the referent when encountering the gap (i.e., region 2) from which the object noun phrase (*who,* linked to *the girl* in the discourse) has been displaced. If these participants evince timely looks to the picture of the noun phrase "girl," then according to Thompson and Choy (2009), this would be evidence for an intact syntactic processor. If so, this result would be evidence against the corollary of our DLA hypothesis that delayed lexical access underlies the syntactic processing disorder, and would contradict prior findings in people with aphasia not only of disrupted syntactic processing (see introduction), but also of delayed re-access effects at structurally defined gaps (Burkhardt et al., 2008; Love et al., 2008).

Thompson and Choy (2009) reported that control participants and those with agrammatic aphasia evinced a significant theme preference at the verb (region 2; e.g., when hearing the verb *kiss,* they were significantly more likely to look at the picture of the direct object than the subject). This pattern strongly suggested that the participants with agrammatic aphasia were showing normal, on-time gap filling in such *wh*-questions, given the visual world paradigm. This finding suggested that agrammatic aphasia does not involve a deficit in computing syntactic representations, even though these participants may have delayed lexical access.

However, the finding of on-time gap filling with *who*-questions does not generalize to other question types, those that more clearly fit the deficit pattern observed in people with agrammatic aphasia. Consider the results from Thompson and Choy's (2009) Experiment 3, where object-extracted relative clauses were examined. Thompson and Choy found a significant theme preference only in the region *following* the gap (see Figure 10, p. 277), unlike what was found for the object-extracted *who*-questions from Experiment 2 (p. 271; see also Figure 7, p. 272). Given the exquisite temporal sensitivity of the eye-tracking method, why should there be a distinction in the time-course for the two construction types? That is, why should object-extracted *who*-questions be understood within a normal time-course, when the comprehension of other question types appears to be delayed?

Before suggesting an answer, we note that there are additional data that support such a distinction between question types, including data from participants with normal development (Friedmann, Belletti, & Rizzi, 2009), participants with specific language impairment (Friedmann & Novogrodsky, 2011), and adult psycholinguistics (De Vincenzi, 1991), and, more relevant to the present study, data from participants with aphasia. Hickok and Avrutin (1996), Thompson, Tait, Ballard, and Fix (1999), and Salis and Edwards (2008) found evidence for a distinction between object-extracted *who*-questions and *which*-noun phrase questions, with the former yielding unambiguous evidence for intact comprehension in participants with Broca's aphasia and the latter for the most part yielding significantly worse comprehension (see also van der Meulen, 2004).

Given these intriguing results from intact as well as disordered populations, several hypotheses regarding a distinction between question types have emerged. Here we briefly consider only one, an account that refers to the linguistic underpinnings of the questions, generalizes to multiple question types, and is buttressed by findings from several literatures (as noted below). Though the details of this account take us beyond the scope of the present paper, the intervener hypothesis suggests that when the filler (i.e., displaced argument) and an intervening noun phrase are similar in linguistic properties, interference occurs, disallowing normal thematic role assignment to the displaced argument. Consider the following:

Which student did the soldier push _ _ _ _ into the street? (6)

who did the soldier push _ _ _ _ into the street? (7)

In the *which*-noun phrase construction in Example 6, the noun phrase *the soldier* intervenes between the displaced argument (which student) and its gap that occurs immediately after the verb. Both of these noun phrases are referential; that is, they both refer to an individual, and thus the intervener interferes with thematic role assignment to the displaced argument. However, in the *who*-question in Example 7, the intervener (the soldier) is a distinct linguistic object from the displaced argument, who, the latter of which is nonreferential, and thus no interference occurs, rendering *who*-questions easier to understand. Such an account has been suggested in several studies of comprehension in people with agrammatic aphasia, including those reported in Friedmann (2008), Friedmann and Shapiro (2003), and Grillo (2005). Furthermore, this account has strong support from the linguistic literature (e.g., relativized minimality; see Rizzi, 1990, 2004) as well as from the psycholinguistic literature (see, for example, Gordon, Hendrick, Johnson, & Lee, 2006; Warren & Gibson, 2005). Thus, not all complex constructions turn out to be problematic in people with agrammatic aphasia; those that are problematic (e.g., object-extracted relative clauses) reveal delayed processing using the visual world eye-tracking method in Thompson and Choy (2009); those that are not (e.g., object-extracted *who*-questions) reveal on-time processing. The results of the former are consistent with the findings reported using the CMP methodology.

The interaction of lexical and syntactic deficits found in people with agrammatic Broca's aphasia suggests some unresolved issues. First, though Love et al. (2008) claimed that a lexical access deficit underlies the purported syntactic deficits in people with aphasia, it is not presently clear how delayed lexical access can account for the patterns of sparing and loss found with specific syntactic structures that we have described in this paper (e.g., object-extracted *who* and *which*-noun phrase constructions). Second, Thompson and colleagues (Choy & Thompson, 2010; Thompson & Choy, 2009) suggested that the deficit in syntactic comprehension is best explained by a lexical integration deficit. If lexical integration is indeed problematic in people with agrammatic aphasia, an initial delay in lexical activation may still be its origin. Further work exploring lexical access, integration, and syntactic comprehension using other complex constructions should be able to resolve these issues.

To conclude, the results of our study show that, for young, neurologically unimpaired participants, the meaning of a lexical item is activated at the immediate temporal offset of the relevant lexical item in the sentence and then rapidly decays. For our participants with agrammatic aphasia, a different pattern emerged, suggesting that lexical activation may begin when the noun is encountered but does not reach threshold until later in the speech stream (in our study, 400 ms). We suggest that this lexical delay may feed syntactic processing too slowly, compromising the ultimate comprehension of some complex constructions that contain displaced arguments.

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References

Avrutin, S. Weak syntax. In: Grodzinsky, Y.; Amuts, K., editors. Broca's region. Oxford, UK: Oxford University Press; 2006. p. 49-62.

Baayen RH. Statistics in psycholinguistics: A critique of some current gold standards. Mental Lexicon Working Papers in Psycholinguistics. 2004; 1:1–45.

Baayen, RH. Analyzing linguistic data. A practical introduction to statistics using R. Cambridge, UK: Cambridge University Press; 2008.

Blumstein, SE.; Milberg, W. Language deficits in Broca's and Wernicke's aphasia: A singular impairment. In: Grodzinsky, Y.; Shapiro, L.; Swinney, D., editors. Language and the brain: Representation and processing. New York, NY: Academic Press; 2000. p. 167-184.

Burkhardt P, Avrutin S, Piñango MM, Ruigendijk E. Slower-than-normal syntactic processing in agrammatic Broca's aphasia: Evidence from Dutch. Journal of Neurolinguistics. 2008; 21:120–137.

Cairns HS, Cowart W, Jablon AD. The effect of prior context on integration of lexical information during sentence processing. Journal of Verbal Learning and Verbal Behavior. 1981; 20(4):445–453.

Choy J, Thompson CK. Binding in agrammatic aphasia: Processing to comprehension. Aphasiology. 2010; 24(5):551–579. [PubMed: 20535243]

De Vincenzi, M. Syntactic parsing strategies in Italian. Dordrecht, The Netherlands: Kluwer Academic; 1991.

Friederici AD, Steinhauer K, Frisch S. Lexical integration: Sequential effects of syntactic and semantic information. Memory & Cognition. 1999; 27(3):438–453.

Friedmann, N. Agrammatic aphasia and the psychological reality of the syntactic tree. In: Hatav, G., editor. Theoretical Hebrew linguistics. Jerusalem, Israel: Magnes; 2008. p. 339-368.

Friedmann N, Belletti A, Rizzi L. Relativized relatives: Types of intervention in the acquisition of A-bar dependencies. Lingua. 2009; 199(1):67–88.

Friedmann N, Novogrodsky R. Which questions are most difficult to understand? The comprehension of *wh* questions in three subtypes of SLI. Lingua. 2011; 121:367–382.

Friedmann N, Shapiro LP. Agrammatic comprehension of simple active sentences with moved constituents: Hebrew OSVand OVS structures. Journal of Speech, Language, and Hearing Research. 2003; 46:288–297.

Goodglass, H.; Kaplan, E.; Barresi, B. Boston Diagnostic Aphasia Examination. 3. Philadelphia, PA: Taylor & Francis; 2000.

Gordon PC, Hendrick R, Johnson M, Lee Y. Similarity-based interference during language comprehension: Evidence from eye tracking during reading. Journal of Experimental Psychology: Learning, Memory, and Cognition. 2006; 32:1304–1321.

Grillo, N. Unpublished doctoral dissertation. Universiteit; The Netherlands: 2005. Generalized minimality: Syntactic under specification in Broca's aphasia.

Grodzinsky Y. The neurology of syntax: Language use without Broca's area. Behavioral and Brain Sciences. 2000; 23(1):1–71. [PubMed: 11303337]

Hickok G, Avrutin S. Comprehension of *wh*. Brain and Language. 1996; 52:314–327. [PubMed: 8811962]

Kass R, Raftery A. Bayes factors. Journal of the American Statistical Association. 1995; 90:773–795.

Kertesz, A. Western Aphasia Battery. San Antonio, TX: Pearson; 2006. (rev. ed.)

Littell, RC.; Milliken, GA.; Stroup, WW.; Wolfinger, RD.; Schabenberger, RD. SAS system for mixed models. 2. Cary, NC: SAS Institute; 2006.

Love T, Oster E. On the categorization of aphasic typologies: The SOAP (a test of syntactic complexity). Journal of Psycholinguistic Research. 2002; 31(5):503–529. [PubMed: 12528429]

Love T, Swinney D, Walenski M, Zurif E. How left inferior frontal cortex participates in syntactic processing: Evidence from aphasia. Brain and Language. 2008; 107:203–219. [PubMed: 18158179]

Love T, Swinney D, Zurif E. Aphasia and the time-course of processing long distance dependencies. Brain and Language. 2001; 79:169–170.

Love T, Walenski M, Swinney D. Slowed speech input has a differential impact on on-line and off-line processing in children's comprehension of pronouns. Journal of Psycholinguistic Research. 2009; 38(3):285–304. [PubMed: 19343495]

Marslen-Wilson WD. Functional parallelism in spoken word-recognition. Cognition. 1987; 25:71–102. [PubMed: 3581730]

Myers EB, Blumstein SE. Selectional restriction and semantic priming effects in normal and Broca's aphasics. Journal of Neurolinguistics. 2005; 18(3):277–296.

Myers EB, Blumstein SE. The neural bases of the lexical effect: An fMRI investigation. Cerebral Cortex. 2008; 18(2):278–288. [PubMed: 17504782]

Nicol J, Swinney D, Love T, Hald L. The on-line study of sentence comprehension: Examination of dual task paradigms. Journal of Psycholiguistic Research. 2006; 35(3):215–231.

Ostrin RK, Tyler LK. Automatic access to lexical semantics in aphasia: Evidence from semantic and associative priming. Brain and Language. 1993; 45:147–159. [PubMed: 8358594]

Piñango, M. Canonicity in Broca's sentence comprehension: The case of the psychological verbs. In: Grodzinsky, Y.; Shapiro, L.; Swinney, D., editors. Language and the brain. San Diego, CA: Academic Press; 2000. p. 330-350.

Prather P, Zurif E, Shapiro L, Swinney D. Real-time examinations of lexical processing in aphasics. Journal of Psycholinguistic Research. 1991; 20(3):271–281. [PubMed: 1880765]

Rizzi, L. Relativized minimality. Cambridge, MA: MIT Press; 1990.

Rizzi, L. Locality and left periphery. In: Belletti, A., editor. Structures and beyond: The cartography of syntactic structures. Vol. 3. New York, NY: Oxford University Press; 2004.

Salis C, Edwards S. Comprehension of *wh*-questions and declarative sentences in agrammatic aphasia: The set partition hypothesis. Journal of Neurolinguistics. 2008; 21(5):375–399.

Schwarz G. Estimating the dimension of a model. The Annals of Statistics. 1978; 6(2):461–464.

Stern C, Prather P, Swinney D, Zurif E. The time-course of automatic lexical access and aging. Brain and Language. 1991; 40:359–372. [PubMed: 2054592]

Swaab T, Brown C, Hagoort P. Spoken sentence comprehension in aphasia: Event-related potential evidence for a lexical integration deficit. Journal of Cognitive Neuroscience. 1997; 9(1):39–66.

Swinney D. Lexical access during sentence comprehension: (Re) consideration of context effects. Journal of Verbal Learning and Verbal Behavior. 1979; 18:645–659.

Swinney, D.; Prather, P. On the comprehension of lexical ambiguity by young children: Investigations into the development of mental modularity. In: Gorfein, D., editor. Resolving semantic ambiguity. New York, NY: Springer-Verlag; 1989. p. 225-238.

Swinney D, Zurif E, Nicol J. The effects of focal brain damage on sentence processing: An examination of the neurological organization of a mental module. Journal of Cognitive Neuroscience. 1989; 1:25–37.

Swinney D, Zurif E, Prather P, Love T. Neurological distribution of processing operations underlying language comprehension. Journal of Cognitive Neuroscience. 1996; 8(2):174–184.

Thompson CK, Choy J. Pronominal resolution and gap filling in agrammatic aphasia: Evidence from eye movements. Journal of Psycholinguistic Research. 2009; 38(3):255–283. [PubMed: 19370416]

Thompson CK, Tait ME, Ballard KJ, Fix SC. Agrammatic aphasic subjects' comprehension of subject and object extracted *wh* questions. Brain and Language. 1999; 67(3):169–187. [PubMed: 10210629]

van der Meulen, I. Unpublished doctoral dissertation. ULCL Leiden University; Leiden, The Netherlands: 2004. Syntactic movement and comprehension deficits in Broca's aphasia.

Walenski M, Mostofsky SH, Ullman MT. Speeded processing of grammar and tool knowledge in Tourette's syndrome. Neuropsychologia. 2007; 45(11):2447–2460. [PubMed: 17493643]

Warren T, Gibson E. Effects of NP-type on reading English clefts. Language and Cognitive Processes. 2005; 20(6):751–767.

Yee E, Blumstein SE, Sedivy JC. Lexical-semantic activation in Broca's and Wernicke's aphasia: Evidence from eye movements. Journal of Cognitive Neuroscience. 2008; 20(4):592–612. [PubMed: 18052783]

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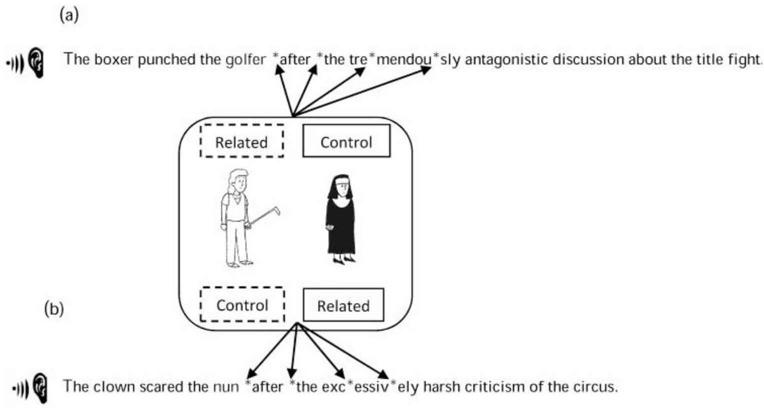

FIGURE 1.

(a) A sample sentence and corresponding probe pictures from the online cross modal picture priming task in Experiments 1 and 2. The sentence was presented auditorily at a normal speech rate. Probe pictures were presented at the offset of the direct object noun in each sentence (italics) and at three subsequent test points at 400-ms intervals. Approximate probe positions in each sentence are indicated by *. Probe pictures for the related and control conditions are depicted, though only one probe picture was presented on each individual trial. (b) The paired sentence that had the same probe pictures to depict counterbalancing of related and control probes. In this matched sentence design, the pictures related to the direct object noun for one sentence were used as the unrelated control pictures for the direct object noun of another sentence (e.g., as indicated by the dashed box around animal trainer), so that over all items, the related and control sets of pictures were identical, avoiding response time confounds due to differences between pictures.

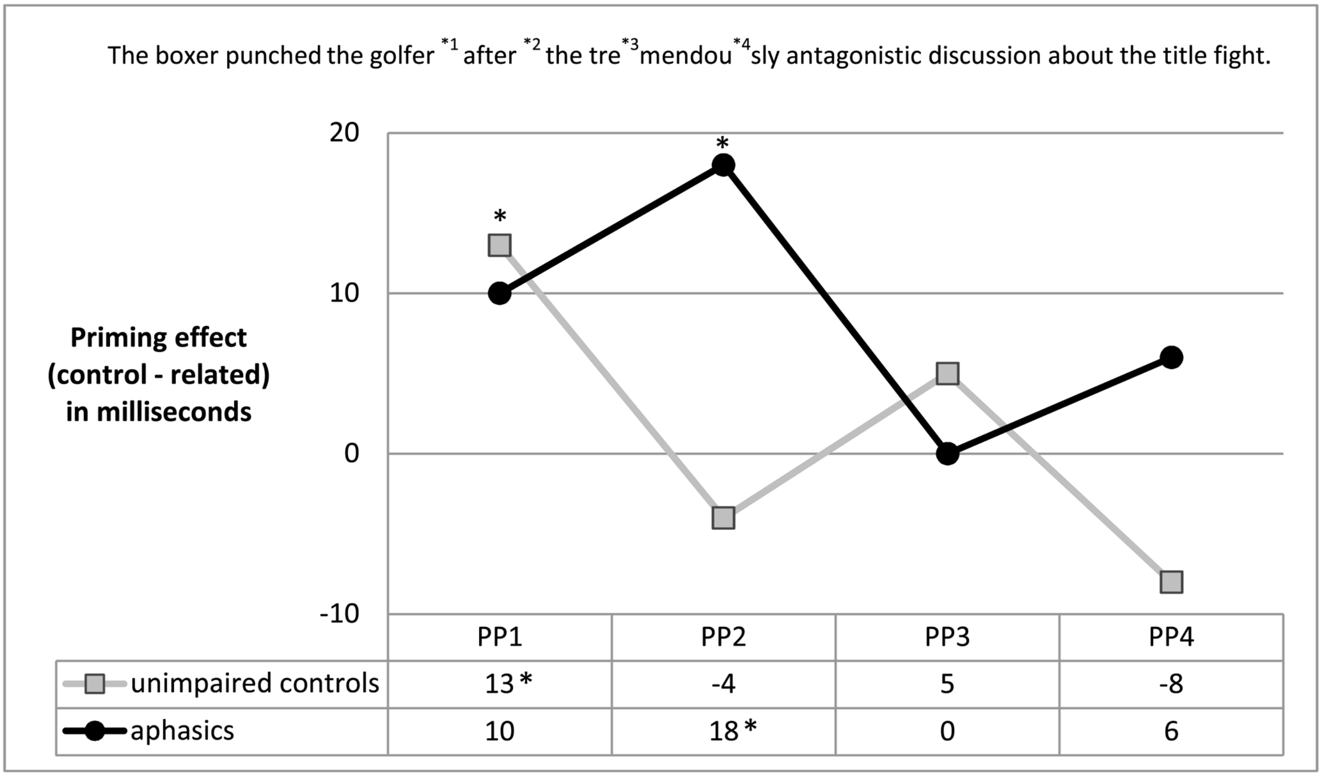

FIGURE 2.

Priming effects (control – related reaction times) for unimpaired control participants (from Experiment 1) and participants with aphasia (from Experiment 2) across four probe positions (PP).

*$p < .05$.

TABLE 1

Mean reaction times (and standard error) in ms to control and related probes at each probe position (PP) for the young, neurologically unimpaired control participants.

	PP1	PP2	PP3	PP4
Control	647 (4.9)	639 (5.3)	650 (4.7)	649 (5.5)
Related	634 (4.8)	643 (5.2)	645 (4.9)	657 (5.8)
(Control – related)	13 ms*	−4 ms	5 ms	−8 ms

* $p < .05$.

TABLE 2

Demographic and lesion information for the participants with aphasia.

Participant	BDAE severity	WAB aphasia quotient	SOAP canonical	SOAP noncanonical	Gender	Age at testing (years; months)	Time post stroke	Education	Lesion location
LHD009	3	76.3	75%	55%	M	59;11	11;7	5 years college	Large L lesion involving inferior frontal gyrus (BA 44,45)
LHD019	1	54.1	90%	20%	F	60;2	15;1	High School	L MCA embolic stroke; distribution encompasses broad left frontal lobe region
LHD040	2	76.7	60%	60%	M	61;1	5;4	BA	Small L subcortical lesion involving the basal ganglia
LHD043	2	79.6	80%	55%	M	86;8	5;8	MA	L MCA lesion involving the frontal lobe with extension to the insula and caudate nucleus
LHD101	2	82.4	95%	35%	M	60;10	4;5	PhD	Large L lesion involving posterior inferior frontal gyrus (BA44) with posterior extension
LHD130	4	81.1	95%	65%	M	57;3	1;9	BA	L IPL with posterior extension sparing STG
LHD138	2	N/A	70%	25%	M	32;8	11;7	Some college	L MCA infarct
LHD140	2	72.9	80%	30%	F	35;10	10;3	BA	L MCA infarct with distribution encompassing broad left frontal lobe region

Note. BDAE = Boston Diagnostic Aphasia Examination—Third Edition (Goodglass, Kaplan, & Barresi, 2000); WAB = Western Aphasia Battery (Kertesz, 2006); SOAP = Subject-relative, Object-relative, Active and Passive Test of Auditory Sentence Comprehension (Love & Oster, 2002). L = left; MCA = middle cerebral artery; IPL = inferior parietal lobule; STG = superior temporal gyrus.

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TABLE 3

Mean reaction times (and standard error) in ms to control and related probes at each probe position for the participants with aphasia.

	PP1	PP2	PP3	PP4
Control	787 (9.0)	789 (9.6)	771 (8.2)	758 (8.0)
Related	777 (8.1)	771 (8.4)	771 (8.7)	752 (8.2)
(Control – Related)	10 ms	18 ms*	0 ms	6 ms

*$p < .05$.