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Semantic Priming in Anomic Aphasia: A Focused Investigation Using Cross-Modal Methodology

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Word count: 6,900

Acknowledgements

The authors wish to express gratitude Dr Asad Khan for his advice regarding data preparation and statistical analysis. The authors would also like to acknowledge the contribution of all participants that made this research possible.

Abstract

Background: Semantic priming in individuals with anomic aphasia has never been the primary focus of an investigation. To date, one study investigated the effects of semantic priming in individuals with fluent aphasia (including anomic aphasia), revealing an inconsistency in semantic priming in the anomic group. Parallels from Broca's aphasia and Wernicke's aphasia literature may be drawn. However, due to the heterogeneity of anomic aphasia, a focused investigation was necessary.

Aims: Semantic priming effects were investigated using a cross-modal pairwise paradigm. It was hypothesised that participants with anomic aphasia would demonstrate priming patterns at a stimulus onset asynchrony (SOA) of 500 msec with slower overall reaction times (RTs) than the control participants. It was further hypothesised that the participants with anomic aphasia might show less inhibition effects than the control participants.

Methods & Procedures: Participants with anomic aphasia (n = 11) and healthy control participants (n = 16) completed a lexical decision task where prime-target pairs were present in equal proportions (related, unrelated and nonwords). A neutral prime condition was also incorporated. Using a cross-modal pairwise paradigm, participants heard a spoken word then 500 msec later, a written word appeared on screen (for 4000 msec). Participants were required to make a decision as to whether the written word was real and RTs were recorded.

Outcomes & Results: Linear mixed model analysis was undertaken and revealed no significant two-way interaction effect, indicating both groups showed priming patterns. A main effect of group~~,~~ was evident, showing faster RTs by the control participant group, confirming our hypothesis that people with anomic aphasia primed at an SOA of 500 msec in a similar manner to the control participants, with slower RTs.

Conclusions: Semantic priming effects were present in anomic aphasia at relatively short SOAs and may be contributed to by automatic processes. Several parameters are proposed

that should be adopted for further investigation into semantic priming in anomic aphasia including electrophysiological measures and manipulation of SOAs and relatedness proportions (RPs) to more precisely measure the effects of controlled vs. automatic processes. Such investigation has the potential to inform new assessment and management techniques.

Keywords: spreading activation, semantic priming, anomic aphasia, cross-modal, lexical decision

SEMANTIC PRIMING in ANOMIC APHASIA: A focused investigation using CROSS-MODAL methodology

Anomic aphasia is a subgroup of aphasia characterised by significant lexical retrieval difficulties (Andreetta, Cantagallo, & Marini, 2012) which can arise at a number of different sources, from meaning representation (Bandur & Shewan, 2008) to phonological encoding (Ralph, Sage, & Roberts, 2000). This word retrieval deficit involves difficulty naming objects and pictures, and can also affect a person's ability to produce nouns, verbs, and other words that convey meaning in connected speech (Goodglass, 1993; Jonkers & Bastiaanse, 2007). The speech produced by people with anomic aphasia is fluent with regard to rate, syntactic form, and articulation. However, because of the word retrieval impairment, hesitations at content word boundaries emerge and circumlocutions, and fillers are common (Hallowell & Chapey, 2008). Individuals with anomic aphasia typically have good overall comprehension skills (LaPointe, 2005) although syntactic comprehension deficits are known to be present (e.g., Bates, Marangolo, Pizzamiglio, & Dick, 2001; Caplan & Waters, 1999; Cardell, 2006).

Anomic aphasia accounts for 29% of all aphasia subgroups, making it the largest aphasia subgroup (Pederson, Vinter, & Olsen, 2004). A plethora of data exists on how assessment and intervention may be planned for this population. Key therapeutic tasks involve offline methods (i.e., tasks that require conscious reflection on a decision and often involve problem solving by the person with aphasia such as word-picture matching) (Boyle, 2010; Hickin, Best, Herbert, Howard, & Osborne, 2002; Nickels, 2002; Shapiro, Swinney, & Borsky, 1998). Performance during offline tasks represents a response to the end point of processing and all events that have contributed to this. However, experimental data regarding the use of online methods (i.e., tasks that allow investigation of the temporal effects of specific processes as they occur such as priming, eye-tracking) (Shapiro et al., 1998) in this population are relatively scarce and biased towards sentence-level processing. For example, it

has been demonstrated that people with anomic aphasia have some qualitative similarities in their syntactic comprehension abilities and well-formedness-judgement performances to that of people with Broca's aphasia, but with fewer errors present (Cardell, 2006; Chen, West, Waters, & Caplan, 2006; Dick, Bates, Wulfeck, Utman, Dronkers, & Gernsbacher, 2001; Wilson & Saygin, 2004). Hence, whilst online sentence-level enquiry has been undertaken, little research has been conducted into lexical-semantic processing in people with anomic aphasia, despite the apparent difficulties at the word level.

The use of online tasks has become central to the investigation of linguistic processing in neurologically-impaired adults. Online tasks allow a system to be locally probed and can measure processing at specific temporal points and are sensitive to fast-acting processes and usually involve some type of timed paradigm or lexical decision (Shapiro et al., 1998). Offline tasks, on the other hand, measure end points of processing (Shapiro et al., 1998) and are therefore a response to an interplay of several processes, including extralinguistic factors such as rehearsal, conscious reflection, and short-term memory. Therefore, online tasks are less affected by memory and attentional demands (Cardell, 2006; Shapiro et al., 1998), and offer a clearer view into the core linguistic deficits of people with aphasia and its time-course. One online task, semantic priming, has received much attention in the psycholinguistic research literature and has been studied extensively in healthy populations and in neurologically-damaged populations, including different subgroups of aphasia (McNamara, 2005).

Semantic Priming

Language processing relies on interplay of processing amongst lexical, semantic and phonological representations (Milberg, Blumstein, Giovanello, & Misiurski, 2003). The semantic priming paradigm has been used to investigate aspects of perception and cognition, such as word recognition, language comprehension, and knowledge representations

(McNamara, 2005; Neely, 1991). The semantic priming effect refers to the fact that processing of a lexical item is faster for target words when they are immediately preceded by a semantically associated word, compared to when preceded by an unrelated word (Rissman, Eliassen, & Blumstein, 2003; Hutchison et al., 2013). The stimulus to which a response is made is known as the target while the preceding stimulus is the prime (McNamara, 2005). In essence, the activation of the prime word facilitates retrieval of the target word (Swinney & Zurif, 1995). For example, the word "nurse" will be recognised faster when preceded by a related word (e.g., "doctor") compared to when preceded by an unrelated word (e.g., "tree").

The "Spreading Activation" theory (Collins & Loftus, 1975; Meyer, Schvaneveldt, & Ruddy, 1974) accounts for such semantic priming effects, stating that activation spreads from one concept to a semantically related concept as they share some of the same information nodes. Furthermore, residual activation of related nodes facilitates their subsequent retrieval if they are part of another shared representation (McNamara, 2005). Thus, the extent to which the first concept primes with the second concept will determine the reaction time (Collins & Loftus, 1975). Some theorists claim that semantic priming results from a dual-process model, first put forward by Posner and Snyder (1975), which states that priming is determined by both automatic and controlled mechanisms (Mummery, Shallice, & Price, 1999). Studies, using electrophysiological measures, have furnished support for this position (e.g., Copland, de Zubicaray, McMahon, Wilson, Eastburn, & Chenery, 2003).

As per the work of Posner and Snyder (1975) outlined above, the presence of semantic priming effects can be the result of automatic and/or controlled processes (McNamara, 2005; Neely, 1991). Automatic processes are typically fast-acting and are not able to be consciously manipulated (Ostrin & Tyler, 1993). Neurophysiological research has found that in non-brain damaged individuals, lexical decision may occur between 300-400 milliseconds (msec) and further to this, McNamara (2005) suggests a semantic priming effect

can be present after just 100 msec. In contrast, controlled processes, such as expectancies (i.e., when predictions are made for the target based on the prime and in response to experimental manipulation) and post-lexical checking (i.e., reflecting and/or using information as a checking mechanism following lexical access to assist decision making), are slower to act and require conscious attention or intention (Harley, 2013; McNamara, 2005; Neely, 1991). McNamara (2005) states that controlled processes can start at a stimulus onset asynchrony (SOA) of 200 msec, although Neely (1977) found spreading activation was still having an effect at 700 msec. Whilst some discrepancy exists, which may partially reflect different paradigm's sensitivities (e.g., neurophysiological research is "real time" and arguably the most sensitive to timing alterations), the fact remains that short SOAs are more reflective of automatic processing. Three parameters that can be manipulated as experimental factors include the SOA, relatedness proportions (RPs) and the inclusion of a neutral prime in the experiment.

The SOA refers to the amount of time between the presentation of the prime and the onset of the target word and is a means through which the influence of automatic and controlled processes can be measured, to an extent (McNamara, 2005). The longer the SOA time is, the more likely it is that controlled processes will emerge, and in particular, post-lexical checking mechanisms. Similarly, at short SOAs, processing is more likely to be automatic (Bushell, 1996; de Salles, Holderbaum, Parente, Mansur & Ansaldo, 2012; Del Toro, 2000). In a meta-analysis of semantic priming studies in Broca's aphasia, Del Toro (2000) concluded that long SOAs are typically greater than 500 msec while short SOAs can range from 100 msec to 500 msec.

Relatedness proportions can also be manipulated to investigate the relative contributions of automatic vs. controlled processes (Bodner & Masson, 2003; Bushell; 1996; Del Toro, 2000; McNamara, 2005). RPs are the proportion of related trials out of all prime-

word target trials. A high RP may build a higher expectation that word pairs will be related. Thus, responses are more rapid because of the controlled process of expectancy-priming. A low RP has the opposite effect; creating a reduction in expectancy-based behaviour and an increase in RT (Bodner & Masson, 2003; McNamara, 2005).

A final parameter that addresses automatic vs. controlled processing is the use of a neutral prime condition as it purportedly provides a baseline of comparison for investigating inhibition and facilitation effects during priming (see studies reported by Bell, Chenery, & Ingram, 2001; Del Toro, 2000; Milberg, Blumstein, Katz, Gershberg, & Brown, 1995; Neely, 1977). When a prime and a target are unrelated, reaction time to the target is slowed, in relation to a neutral condition (Bell, Chenery, & Ingram, 2000). When this occurs, an inhibition effect is said to exist and controlled processing is considered to have occurred. Conversely, when a prime and a target are related, reaction time to the target is enhanced and a facilitation effect is said to exist, resulting in automatic processing (Bell et al., 2000; McNamara, 2005).

Some contention does exist for whether the inclusion of a neutral prime results in accurate measurement of facilitation and inhibition (Dien, Franklin, & May, 2006). Several commentators suggest that if a neutral prime, in any way, speeds RTs to the target word then facilitation will be underestimated and inhibition will be overestimated while the opposite effect would occur if the presence of a neutral prime slows RTs to the target word (Dien et al., 2006; McNamara, 2005).

Semantic Priming in Broca's & Wernicke's Aphasia

The semantic priming paradigm has been used extensively to examine the nature of semantic representations and lexical access in people with Broca's aphasia and, to a lesser extent, in Wernicke's aphasia. Over 20 semantic priming studies in Broca's aphasia were reported in a meta-analysis by Del Toro (2000) and findings of five studies which have

investigated semantic priming effects in Wernicke's aphasia are noted in Milberg et al. (2003). All these studies used lexical decision. Typically, individuals with Broca's aphasia present with non-fluent speech but relatively normal comprehension abilities (Hallowell & Chapey, 2008). In comparison, individuals with Wernicke's aphasia are able to produce fluent speech (not unlike individuals with anomic aphasia) but their comprehension is significantly impaired (Hallowell & Chapey, 2008). Therefore, one might predict that differences exist between patterns of semantic activation and processing, which might then be reflected in their semantic priming patterns. Indeed, such differences have been obtained. Semantic priming research in Broca's aphasia has tended to show a delay or absence of semantic priming, thus suggesting impaired rapid activation and/or short-lived activation effects (e.g., Bushell, 1996; Del Toro, 2000; Milberg et al., 2003) or an over-reliance on controlled processing mechanisms (Del Toro, 2000). Paradoxically, individuals with Wernicke's aphasia have been shown to exhibit strong priming effects, indicating intact mechanisms which sustain activation of associated pairs, despite these individuals having poor comprehension (Milberg et al., 2003).

It should be noted that research into priming in "fluent aphasia", including anomic aphasia, at a more general level, has produced mixed results. Research conducted by Gordon and Baum (1994) and Milberg and Blumstein (1981) found no consistent priming effects exist in fluent aphasia. In contrast, another study by Milberg, Blumstein, and Dworetzky (1988) found a greater degree of priming in fluent aphasia than in their non-brain damaged counterparts, which supported the overactivation hypothesis. Importantly, the focus on fluent aphasia means that it is unclear whether these results relate to individuals with anomic, Wernicke's, or even conduction aphasia. As such, this fluent classification may be responsible for the conflicting findings, particularly given the later evidence for priming

effects in Wernicke's aphasia (Blumstein & Milberg, 2000) when focused enquiry was undertaken.

Semantic Priming in Anomic Aphasia

To date, only one study by Baum (1997) has documented semantic priming effects, specifically, in individuals with anomic aphasia. **S**emantic priming was studied across 11 individuals with non-fluent aphasia and 10 individuals with fluent aphasia by recording mean RTs to auditory real word targets. Whilst not the specific focus, six participants with anomic aphasia were investigated. In addition to semantic priming, the effects of phonological and mediated priming also were studied. Accordingly, an auditory pairwise paradigm was constructed using an SOA of 500 msec with no neutral condition. The results showed semantic priming patterns for half of the fluent participants. Of the six participants with anomic aphasia, three were shown to demonstrate semantic priming (i.e., faster RTs to related targets vs. unrelated targets). Further, one of the remaining participants with anomic aphasia recorded a negative semantic priming pattern of -135msecs. Importantly, five out of the six participants with anomic aphasia were classified as having mild anomic aphasia, with one classified as being moderate. Notably, the three individuals who showed semantic priming were all classified as being mild. These overall results suggest some lack of consistency among semantic priming results in anomic aphasia, but raise the interesting possibility that the presence of semantic priming effects may be somewhat dependent on the severity of the anomic aphasia.

A major limitation to the Baum (1997) study was that while semantic priming effects were investigated in 21 participants with aphasia, the participants with anomic aphasia were not analysed as a separate group, making the results difficult to interpret. Furthermore, the unequal balance related to aphasia severity may have further affected the results, and the author, herself, acknowledged that the fluent individuals who showed inconsistent semantic

priming had more severe comprehension deficits. A second limitation from the Baum (1997) study was that only 6% of word pairs presented to the participants were semantically related, while a much higher number of phonologically related pairs were used. Hence, there is a possibility that the reduced percentage of semantically related items, coupled with the presence of a higher number of phonologically related items, created some processing interference that influenced the response latency patterns and the overall results.

A final issue relates to the semantic priming paradigm employed. Baum (1997). This study used an auditory pairwise paradigm, which has been used in the Broca's aphasia priming literature (e.g., Milberg et al., 1995; Ostrin & Tyler, 1993). Other paradigms that have been used in the aphasia literature include visual pairwise and list priming (auditory & visual) (see review by Del Toro, 2000). However, the cross-modal pairwise paradigm is noticeably under-utilised in the aphasic literature. Cross-modal priming is said to occur when the prime and the target are processed via two different modalities (e.g., an auditory prime and a visual target) (Harley, 2013; Scherer & Larsen, 2011). The cross-modal paradigm has been extensively used in the normal language processing literature since Holcomb & Anderson (1993; 1995) first contrasted unimodal and cross-modal semantic priming paradigms to investigate whether semantic information was organised along modality-specific lines, according to the input, or whether all modalities converged on a single amodal semantic system. Their results supported the presence of an amodal semantic system and, more recently, Carter, Hough, Stuart, and Rastatter (2011) provided further evidence in support of an amodal semantic system, when they concluded that automatic spreading activation occurred across both auditory and visual modalities. To add to this, Sass, Krach, Sachs, and Kircher (2009) used a cross-modal paradigm and affirmed neurophysiologically that, irrespective of the modality of input, an amodal semantic system was being accessed.

The lack of studies that have used a pairwise cross-modal paradigm with participants with aphasia is interesting, as a related methodology has been used in much of the seminal online sentence comprehension research (e.g., Swinney, Zurif, & Nicol, 1989). Specifically, to investigate the integrity of gap-filling and its relationship to asyntactic comprehension, sentences are auditorily presented and, at a designated experimental point within the sentence, the person must make a "Yes/No" lexical decision about a written word that appears on a computer screen (e.g., Shapiro et al., 1998). In the future, direct comparisons of priming at a lexical level with sentence level priming will contribute much to our understanding of the impact that disturbances in lexical processing may play during online sentence comprehension and lexical access. Hence, a good starting point to investigate semantic priming in anomic aphasia would be with a cross-modal pairwise paradigm.

Therefore, the aim of the present study was to conduct a focused investigation of semantic priming effects in anomic aphasia in an effort to understand more about the underlying semantic operations in this population. The outcomes from previous semantic priming studies need to be taken into account when planning a new line of research to ensure that a reasonable starting point is first established. As such, a targeted enquiry with a balance of semantically-related and unrelated words was undertaken. In addition, because of the future value that information about cross-modal semantic priming at a lexical level might have for the interpretation of results from sentence comprehension studies that use cross-modal methodology, a cross-modal pairwise lexical decision paradigm was implemented.

In view of the literature, it was hypothesised that both the healthy control participants and the participants with anomic aphasia would show semantic priming at an SOA of 500 msec. In addition to semantic priming effects, facilitation and inhibition effects were also of interest to investigate controlled vs. automatic processing contributions. Therefore, in consideration of findings that have suggested that lexical activation might be operating under

a protracted time course in some types of aphasia (Bushell, 1996; Milberg et al., 2003; Prather, Zurif, Love, & Brownell, 1997), it was further hypothesised that at an SOA of 500 msec, the participants with anomic aphasia might show less inhibition effects than the control participants. Finally, it was predicted that the overall RTs would be slower for the participants with anomic aphasia.

Methods

Ethical clearance to conduct the study was obtained through The University of Queensland's Human Research Ethics Committee.

Participants

Included in this study were 11 participants with a clinical classification of anomic aphasia (6 females, 5 males) and 16 neurologically-intact healthy control participants (7 females, 9 males). The classification and severity of anomic aphasia was determined following administration of the Western Aphasia Battery (WAB) (Kertesz, 1982) on which all the participants with aphasia were classified as having mild anomic impairment (i.e., Aphasia Quotient of 76 or above). All the healthy control participants and the participants with aphasia were right handed, spoke English as a first language, had no known history of drug or alcohol abuse, no psychiatric history, and had normal (or corrected) vision and hearing. All participants with aphasia had experienced a single neurological event and were more than 6 months post-event. The biographical and neurological information for the participants with aphasia can be seen in Table 1.

"(Table 1 about here)"

The mean age of the participants with aphasia was 56 years (*SD* = 9.93) while the mean age of the healthy control participants was 56.5 years (*SD* = 12.97). The mean years of education of the individuals with aphasia was 12.27 years (*SD* = 1.95) while the mean years of education of the healthy control participants was 13.06 years (*SD* = 2.52). Two-sample equal variance t-tests were conducted to assess comparability of the groups and yielded no significant difference in education, $p = .371$, and no significant difference in age, $p = .909$.

Stimuli

In total, 180 prime-target word pairs were presented to all participants, which formed two experimental blocks of 90 words each (i.e., 45 prime-word target pairs and 45 prime-nonword target pairs), with different words in each block. Each block comprised three cohorts, each of which contained 15 pairs. The 15 pairs were made from 5 prime-word targets for each of three prime conditions. The three prime conditions comprised unrelated primes (e.g., hat-animal), related primes (e.g., dog-animal), and a neutral prime condition which employed the word “blank” in line with other priming studies (e.g., Bell et al., 2001; Del Toro, 2000). The 5 prime-word targets from each of the three prime conditions were counterbalanced across each cohort. In addition, 15 prime-nonword pairs were presented in each cohort, ensuring an equal number of real word and nonword targets. These **primes** included 10 real words (e.g., dishes – dimp) and 5 instances of the word “blank”, thereby having the same 1:3 ratio as the prime-word pairs in an attempt to reduce expectancy-based effects.

All real words were predominantly nouns with some verb primes (related). The distribution of related verb-noun pairs across blocks was not controlled. Related prime-targets were derived from the Edinburgh Associative Thesaurus (Kiss, Armstrong, Milroy, & Piper, 1973) as having high associative values. Therefore, related primes relationships were super-ordinate, co-ordinate or associative. All nonwords were between 3-8 letters in length

and were orthographically and phonologically plausible variations of the English language, created by substituting 1-2 letters of real words not selected as word targets. No pseudohomophones were included.

All prime words were presented auditorily and had been recorded by a trained male English speaker. These recordings were digitised onto a notebook computer using a sampling frequency of 22 kHz. The written target words were presented in the centre of the computer screen in size 64 lower case letters in Arial font.

Procedure

Each participant was seen individually over three sessions with each session lasting 45 minutes to one hour. Each session was separated by a minimum of one week (and up to three weeks) to account for possible learning effects. Participants were seated in front of a First Lite notebook computer in either a quiet university-based testing room or at his/her place of residence. Sony headphones were worn throughout experiment

The two experimental blocks were created on SuperLab Pro (version 2.0) (Cedrus Corporation, 1996) experimental software and each block was divided into three experimental cohorts of 30 items. In each session, one cohort from Block 1 and one cohort from Block 2 were presented. Therefore, no target was repeated in any session. The blocks were presented to each participant one block at a time and each block was presented at least one week apart. The items in each cohort were identical, but were randomised across the two blocks to eliminate order, learning and sequencing effects. To further reduce these effects, the order in which participants were presented the blocks was counterbalanced so that half of the participants received a cohort from Block 1, then one from Block 2, while the other participants received a Block 2 cohort, and then Block 1 one. The order in which cohorts were presented to individual participants was randomly selected.

Participants were familiarised with the task through an initial training block consisting of nine practice trials which did not appear in the experiment. These initial training blocks comprised of computerised simultaneously presented auditory/spoken and visual/written instructions (presented centre-screen on 1-2 lines in size 64 Arial font). The instructions were presented in two stages, namely, (1) teaching the participant to respond to real and nonwords, that is lexical decision, and (2) teaching the concept of an auditory 'prime' and responding to words (see Appendix for specific instructions). Participants confirmed that they were ready for the experiment to begin following the trial items, by pressing a green button on a serial mouse which was accurate to within one millisecond. Once the experiment had begun, a visual attention cue (of 500 msec), represented by $$$$$ positioned centrally on the screen, was simultaneously presented with an auditory "ding" attention cue to alert the participants. Participants then heard a spoken word and 500 msec later, from the point of the offset of the spoken word, a different written word appeared on screen (for 4000 msec). Participants were told to respond only to the written word and to decide whether the written word was real or not, by pressing a green button for a "yes" response or a red button for a "no" response. Response accuracy and reaction times were recorded directly onto the computer for later analysis. Figure 1 shows an example of one complete presentation trial for each cohort.

Following the presentation of the selected 30-item cohort from Block 1, there was the opportunity for the participant to take a break. Upon resuming the experiment to collect Block 2 data, four practice items preceded the 30 experimental trials, but data from the practice trials was not collected.

"(Figure 1 about here)"

Results

Data Preparation & Analysis

The RTs of interest were those from correct identification of real word targets. Hence, all nonword target responses were removed, comprising 621 points of data for the anomic participant group and 880 points of data for the healthy control participant group. Removal of error responses to the real words accounted for 4.97% of data for the anomic participant group, and 1.46% for the healthy control participant group. These low error rates contraindicated further analyses of error patterns.

Prior to statistical analysis, RTs above 1500 msec were removed in the healthy control group, concurrent with other semantic priming studies that take1500 msec as the upper limit for measuring priming (Angwin, Chenery, Copland, Murdoch, & Silburn, 2005). After conducting normality tests, both participant groups were found to be positively skewed and the assumption of approximation to a normal distribution was not applicable. Accordingly, both data sets were transformed using inverse transformation and this resulted in approximation to normal distribution (Tabachnick & Fidel, 1996). Following the inverse transformation, no extreme values were apparent in either group's data set. In total, 1363 values in the healthy control group and 995 values in the aphasic group were analysed. It is important to note that data was analysed using transformed values however, values reported in figures and tables are untransformed real RTs, for ease of interpretation.

Between Group Analysis

Linear mixed model analysis using a two-factor design (Group X Prime) was undertaken. Group was set as a between participants factor, prime was set as a within-participants factor, and participants was set as a random factor. No significant two-way interaction effect was present, $F(2, 2359) = .193$, $p = .825$, indicating both groups exhibited the same overall priming pattern. A main effect of group, $F(1, 1339) = 87.61$, $p < .001$, was evident, showing faster RTs by the healthy control participant group when compared to the

anomic participant group. A main effect of prime condition, $F(2, 2359) = 11.79$, $p < .001$, also was present. Table 2 shows the mean RTs and standard deviations for the anomic aphasic participant group and the healthy control group across all three prime conditions.

"(Table 2 about here)"

Within Group Analysis

Of particular interest was each group's individual performance profiles related to the extent to which inhibition, facilitation and overall priming occurred (see Table 3). Accordingly, additional pairwise comparisons, using t-tests, were undertaken to further investigate these issues.

"(Table 3 about here)"

For the healthy control participant group, comparison of the related vs. unrelated primes (overall priming) showed significance, $t = -3.00$, $p = .003$, as did the unrelated vs. neutral primes (inhibition), $t = 4.19$, $p < .001$. Comparison of the related vs. neutral primes (facilitation) did not yield significance, $t = 1.01$, $p = .311$. Similarly, for the anomic participant group, comparison of the related vs. unrelated primes (overall priming) showed significance, $t = -2.72$, $p = .007$, as did the unrelated vs. neutral primes (inhibition), $t = 2.67$, $p = .008$. Comparison of the related vs. neutral primes (facilitation), however, showed no significance, $t = .043$, $p = .966$. Overall, both groups demonstrated patterns of inhibition without facilitation.

Discussion

The present study has provided new evidence that semantic priming effects can be observed in people with anomic aphasia when focused investigation is undertaken. In order to further understand online lexical-semantic processing in this population, a cross-modal pairwise paradigm with an SOA of 500msec was employed. Additionally, the extent to which

automatic and controlled processes impact on lexical decision at an SOA of 500msec in the anomic and non-brain damaged populations was of theoretical interest. It was hypothesised that both the people with anomic aphasia and the control participants would demonstrate semantic priming at an SOA of 500 msec and, indeed, this hypothesis was supported. It also was hypothesised that the overall RTs of the anomic participants would be slower in comparison to the healthy control participants, and again the results supported this proposal. In relation to the presence of automatic vs. controlled processing, it was hypothesised that, at an SOA of 500 msec, the participants with anomic aphasia might show less inhibition effects than the healthy control participants; however, greater inhibition effects in the control participant group were not found. These results will now be discussed in more detail.

In spite of the global heterogeneity of anomic aphasia and the novel use of a cross-modal pairwise paradigm, robust priming effects were present in both participant groups, as predicted. Such a result might suggest that people with anomic aphasia are accessing semantic representations in a similar manner to non-brain damaged individuals, at least when there is a 500 msec window available for processing. Thus, on the surface, it appears that the mechanism of spreading activation is occurring in the healthy control participant group and also in the anomic aphasia participant group.

However, an important theoretical and methodological point related to the use of an SOA of 500 msec warrants discussion. As described, semantic priming can result from automatic and/or controlled processes. Some commentators have argued that an SOA of 500 msec is representative of automatic processes (Del Toro, 2000) and consider 500 msec to be a short SOA. However, neurophysiological studies utilising event-related potentials and magnetoencephalography have found that in non-brain damaged individuals, lexical decision and its associated semantic operations occur between 300-400 msec, suggesting that the present study's use of a 500 msec SOA will contribute to controlled processing (Copland et

al., 2003; Pylkkänen, Stringfellow & Marantz, 2002; Sass et al., 2009). Therefore, it is perhaps possible that at an SOA of 500 msec, the healthy control participants were using the dual process of spreading activation and controlled processes, as were the participants with anomic aphasia.

It also could be argued that the priming effects seen in participants with anomic aphasia in the present study did not necessarily mean that spreading activation or the controlled processes that contribute to priming were proceeding in a normal manner. Interestingly, in support of this proposition, much of the literature suggests that people with Wernicke's aphasia typically display priming patterns across a variety of SOA conditions (Milberg et al., 2003). However, people with Wernicke's aphasia, nonetheless, were found to be impaired in terms of their lexical access abilities which are either increased (overactivation) or their inhibitory processes are decreased (Blumstein, 2001; Milberg et al., 1988).

The 500 msec time-frame also is important in aphasia priming research. Strong priming effects have been found across the majority of priming studies with people with Broca's aphasia at 500 msec SOAs (see review by Del Toro, 2000). As previously discussed, however, semantic priming patterns have been more mixed when looking beyond or earlier than the 500 msec time-frame, thus indicating that semantic operations can be disrupted under certain conditions. Some studies have shown no priming occurs at either short or long SOAs (e.g., Bushell, 1996; Milberg & Blumstein, 1981) while other studies have found priming could occur at long SOAs (up to 2000 msec) as well as at short SOAs (as little as 150 msec) (e.g., Milberg et al., 1995*).* Further to this, Bushell (1996) and Prather et al. (1997) concluded that people with Broca's aphasia exhibited a slower rise time of semantic activation, as it was shown that priming was absent at a short SOA but became present when the SOA was longer (e.g., Milberg et al., 1995).

To restate, in the current study, the participants with anomic aphasia were seen to prime at an SOA of 500 msec. Therefore, it is possible that, in adapting the views of Bushell (1996), people with anomic aphasia may be seen to prime at 500 msec, but not a lot sooner (e.g., at 200 msec), unlike their non-brain damaged counterparts for whom consistent earlier priming has been found (Pylkkänen et al., 2002; Sass et al., 2009). However, without further evidence, it is not possible to make any firm conclusions regarding this issue from the present experiment's results.

In addition to semantic priming effects, the present experiment aimed to gain further insights into the role of automatic vs. controlled processes by considering patterns of inhibition and facilitation across the two participant groups, through the inclusion of the neutral prime condition. It was predicted that the participants with anomic aphasia might show less inhibition effects than the healthy control participants at an SOA of 500 msec, however, this prediction was not confirmed. The overall patterns of facilitation and inhibition were found to be the same between the anomic participant and the healthy control participant groups. That is, both groups demonstrated inhibition effects; however, no facilitation effects were present.

Most commentators put forward the view that inhibition effects are indicative of controlled processing and that the presence of controlled processing can be exemplified when priming patterns of inhibition occur, with or without facilitation (Angwin, Arnott, Copland, Haire, Murdoch, Silburn, & Chenery, 2009; McNamara, 2005; Neely, 1977). However, a state of pure automatic processing can only be claimed when facilitation exists without inhibition (Del Toro, 2000; McNamara, 2005; Neely, 1991). The presence of inhibition in the present investigation speaks to the use of controlled processing, across both groups, **as a** mechanism for mediating priming in the present experiment. The lack of facilitation effects could be interpreted as meaning that, (1) no spreading activation has occurred, (2) spreading

activation is compromised or, (3) spreading activation has been completed in an earlier phase, which is likely for the control group, at least, given the results from Copland et al. (2003) and Sass et al. (2009).

When trying to establish the role of automatic vs. controlled processing in the present experiment, a methodological issue pertaining to the use of the neutral prime "blank" emerges. While some commentators argue that the use of a neutral prime is central to the investigation of facilitation and inhibition (e.g., Bell et al., 2000; Milberg et al., 1995), it has also been suggested that the presence of the word "blank" may result in overestimation or underestimation of these factors. McNamara (2005) comments that the use of "blank" (and other similar neutral primes) may result in repetition priming, which ultimately leads to a decrease in the time needed to process the neutral prime (in relation to the related and unrelated primes). In this instance, facilitation is underestimated and inhibition is overestimated. Hence, in the current study, it is possible that when inhibition was present without any effects of facilitation, the source of this pattern was in the use of the neutral prime condition "blank". Nonetheless, the strong presence of inhibition in the current study indicates that controlled processing was occurring. One must assume the healthy control participants used normal spreading activation mechanisms; however, the question remains about what occurred in the early automatic stages for the participants with anomic aphasia. Even in spite of this possible methodological confound, priming was found and some commentators would argue that priming is the real measure, rather than the facilitation and inhibition effects (McNamara, 2005).

The final prediction for this study was that the overall RTs of the anomic participants would be slower in comparison to the healthy control participants and this prediction was supported by the results. It is possible that the presence of slowed response times in the anomic group may have been an indicator of slowed lexical retrieval or spreading activation,

as some commentators have suggested (e.g., Del Toro, 2000; Milberg et al., 2003; Prather et al., 1997).. However, also possible was that these RTs were slower as a result of external factors, such as an increased susceptibility to distraction or an increased time to involve attentional or controlled mechanisms. Importantly, these factors are known to be affected in aphasia, even with mild impairment (Murray, Holland, & Beeson, 1997). Also, it is not unreasonable to suggest that the slower response times of the anomic participants may have been reflective of slowed motor responses, which are common in people with aphasia (Hallowell & Chapey, 2008). Therefore, no firm conclusions can be made regarding these slowed response times and their relationship to semantic operations. However, if people with anomic aphasia are operating under a slower time-course, there are implications for sentence processing as well as for therapeutic management.

The significant semantic priming patterns found in the present study for the participants with anomic aphasia contrast, somewhat, to those found by Baum (1997), who found variable patterns. Baum (1997) speculated that this priming pattern variation may be attributable to the severity of the anomic aphasia; that is, the more severe the aphasia, the less likely that priming would occur. Concurring with this proposition, the participants in the present study all had an Aphasia Quotient range of 79 to 93.4 and, according to the WAB, a score of 76 or above indicates mild impairment (Kertesz, 2007). All anomic aphasic participants showed priming effects to semantically-related words with the same patterns as the healthy control participants, albeit with slower response times. Therefore, although the current results are positive for making conclusions about the fact that robust priming effects can be observed in individuals with anomic aphasia, global assumptions cannot be made about this population, as only those with mild impairment were investigated and the extent of their semantic difficulties was not assessed in detail.

Accordingly, the degree to which greater levels of impairment severity may impact on semantic priming effects in anomic aphasia is unable to be concluded from the current investigation, although Baum's (1997) work suggested that severity might be influential.

Limitations and Future Directions

As the current investigation represented the first targeted study into semantic priming effects in individuals with anomic aphasia, a number of limitations and new lines of enquiry emerged. That cohorts within the blocks were randomly selected meant that it was possible that some participants may have received the same cohort order across the blocks. Despite the present experiment controlling overall order effects by strict counterbalancing at the block level and for items within cohorts, this further control should be considered when designing future priming studies.

The use of electrophysiological measures, that tap into real-time processing, to complement behavioural measures is the future of the semantic priming research field (e.g., Copland et al., 2003; Copland, de Zubricaray, McMahon, & Eastburn, 2007; Kotz, Cappa, von Cramon, & Friederici, 2002; Mummery et al., 1999; Pylkkänen et al., 2002). Indeed, with the advances in technology in the past decade, it is now possible to measure both behavioural and neurophysiological correlates of various language processes through simultaneous measurement of RTs and brain activity. Incorporating electrophysiological measures with the current behavioural measures would have provided a means of better understanding the neurophysiological correlates of semantic priming in anomic aphasia. Using such measures also would have revealed, more definitively, if the semantic priming effects observed were, in fact, the same as for the healthy control participants, as real-time data pertaining to the strength and timing of semantic processing would have been furnished. A further limitation was the use of just the one 500 msec SOA. The inclusion of both a shorter SOA condition and a longer SOA condition would have added valuable information to assist with

interpreting the role of automatic and controlled effects during priming, and might have resulted in some greater insights into the time-course of semantic operations in anomic aphasia. Specifically, it is suggested that manipulation of the SOA using shorter SOAs of 200 msec and longer SOAs greater than 1000 msec (e.g., see Angwin et al., 2009 for examples), would yield particularly useful information. Increasing the RPs also would offer some insights into how expectancy-based processes affect priming in anomic aphasia, as high RPs are a signature of measuring controlled processing (McNamara, 2005; Neely, 1991).

An additional line of enquiry pertains to the assessment of the semantic abilities of participants with anomic aphasia. In future studies, it would be important to conduct detailed semantic assessment to generate a comprehensive semantic profile for each participant with anomic aphasia. It is known that subgroups exist in anomic aphasia (LaPointe, 2005; Ralph et al., 2000), so the extent of semantic influences could well be of significance to how priming occurs across different subgroups. Variances, such as this, however, did not seem to impact on the current investigation's outcomes, as all participants with anomic aphasia had high comprehension quotients, ranging from 17.8 to 20. In relation to investigating differences in anomic aphasia subgroups, it may also be useful for future studies to include participants with a range severity levels. As this study only had individuals with mild anomic aphasia, the conclusions regarding semantic priming abilities relate to this cohort. The impact that different severity levels may have on semantic priming abilities is not yet known.

Conclusion

This study represents a starting point for further exploration into the underlying operating characteristics of lexical and semantic processing in anomic aphasia and affirms that such investigation is possible in this subgroup, despite its heterogeneity. Whilst the present results showed semantic priming effects in anomic aphasia at an SOA of 500 msec, this does not necessarily mean that lexical access and semantic processes are operating

normally and across the entire time-course. At a behavioural level, lexical and semantic deficits are present in anomic aphasia. If it can be shown more directly when lexical processing is disrupted, and how these disruptions manifest, such findings will have implications for understanding the underpinnings of both word retrieval deficits and subtle sentence comprehension disturbances in anomic aphasia. Hence, more semantic priming investigation is required, at earlier and later time-points, to establish the time course of lexical and semantic activation in anomic aphasia. Most importantly, however, is that the more that is known and understood about the contributions of semantic processing to anomic aphasia, the more informed we will be in the move towards developing more efficacious and, potentially, online treatments.

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Appendix

Instructions given to participants prior to commencement of experiment

1. Welcome to our experiment
2. A row of letters or words will appear on the screen
3. It could be a word like ‘clap’
4. Or it could be a word like ‘plab’
5. If a real word appears, press the green YES button
6. If a nonword appears, press the red NO button
7. Respond as fast as you can
8. A get ready signal will appear just before each item
9. It looks like this $$$$$
10. Let’s have a practice
11. Here comes the first item
12. Now let’s introduce a small change
13. This time you will hear a SPOKEN word just before the written word appears
14. Just listen to the SPOKEN word
15. Respond to the WRITTEN word
16. Is it a word or a nonword?
17. If a word appears, press the green YES button
18. If a nonword appears, press the red NO button
19. Are you ready?
20. Here is the first item
21. Press the YES button when you’re ready to start the experiment
22. $$$$$

Table 1

Biographical and Neurological Characteristics for Each Participant with Aphasia (n = 11)

Participant Number	Gender	Age	Years of Education	Previous Occupation	Time Post-event	Lesion Information	Aphasia Quotient (AQ)
1	M	59	9	Caravan Park Manager	3 years	Left anterior branches of MCA	92
2	M	65	15	Civil Engineer	15 months	N/A	82.4
3	M	68	12	Clerk	8 years	N/A	93
4	F	58	10	Secretary	18 months	Left MCA	91.6
5	F	60	12	Home Duties	5.5 years	Left caudate nucleus & internal capsule	89.2
6	M	59	16	English Lecturer	32 months	Left parieto-occipital, left basal ganglia and caudate nucleus	85.6
7	F	59	12	Home Duties	**6 months**	Left basal ganglia	94
8	F	33	12	Secretary	18	Striato-capsular	89.2

					months	infarct	
Participant Number	Gender	Age	Years of Education	Previous Occupation	Time Post-event	Lesion Information	Aphasia Quotient (AQ)
9	F	59	12	Business Woman	4.2 years	Left temporo-occipital infarct	90.8
10	M	53	13	Economics Lecturer	14 months	Left parietal	79
11	F	43	12	Shop Assistant	10 years	Left temporo-parietal and basal ganglia	93.4

Note. F = female; M = male; MCA = middle cerebral artery; N/A = not available.

Table 2

Mean Reaction Times (msec) and standard deviations for the three prime conditions as a function of Group

Control Group (n = 16)			Anomic Aphasia Group (n = 11)		
Conditions	Mean	*SD*	Conditions	Mean	*SD*
Neutral	637.00	143.94	Neutral	969.33	415.90
Related	652.66	163.40	Related	955.82	371.81
Unrelated	686.02	207.16	Unrelated	1040.66	488.42

Note. SD = standard deviation.

Table 3

Reaction Time (RT) Priming Effects Summary

	Controls (n=16)	Anomics (n=11)
Facilitation	-15	+13
Inhibition	-49	-71
Overall Priming	+34	+84

Note. Facilitation= neutral minus related; Inhibition= neutral minus unrelated; Overall Priming= unrelated minus related.

Figure 1. Example of one complete trial for each prime-target condition using a cross-modal paradigm where ISI= interstimulus interval.