

# Lexical–Semantic Activation in Broca’s and Wernicke’s Aphasia: Evidence from Eye Movements

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## Abstract

■ Lexical processing requires both activating stored representations and selecting among active candidates. The current work uses an eye-tracking paradigm to conduct a detailed temporal investigation of lexical processing. Patients with Broca’s and Wernicke’s aphasia are studied to shed light on the roles of anterior and posterior brain regions in lexical processing as well as the effects of lexical competition on such processing. Experiment 1 investigates whether objects semantically related to an uttered word are preferentially fixated, for example, given the auditory target “hammer,” do participants fixate a picture of a nail? Results show that, like normal controls, both groups of patients are more likely to fixate on an object semantically related to the target than an unrelated object. Experiment 2 explores whether Broca’s and Wernicke’s aphasics show competition

effects when words share onsets with the uttered word, for instance, given the auditory target “hammer,” do participants fixate a picture of a hammock? Experiment 3 investigates whether these patients activate words semantically related to onset competitors of the uttered word, for example, given the auditory target “hammock,” do participants fixate a nail due to partial activation of the onset competitor hammer? Results of Experiments 2 and 3 show pathological patterns of performance for both Broca’s and Wernicke’s aphasics under conditions of lexical onset competition. However, the patterns of deficit differed, suggesting different functional and computational roles for anterior and posterior areas in lexical processing. Implications of the findings for the functional architecture of the lexical processing system and its potential neural substrates are considered. ■

## INTRODUCTION

It is generally assumed that to access a word in the mental lexicon requires multiple stages of processing. These stages involve phonetic–phonological processing, mapping to lexical form, and accessing meaning. In particular, the auditory input from the peripheral auditory system is converted to a phonetic–phonological representation. This phonological representation maps onto the lexicon where a particular lexical entry is selected from a set of potential lexical candidates. Each lexical entry contacts a lexical–semantic network where the meaning of the lexical entry is ultimately contacted.

Evidence from neuroimaging and lesion data suggest that the processes involved in lexical access recruit a distributed neural system that includes both anterior and posterior brain structures. In particular, it has been shown that, as postulated by the classical lesion model, posterior brain structures, including the superior and middle temporal gyri, are involved in lexical–semantic

processing (Price, Moore, Humphreys, & Wise, 1997; Vandenberghe, Price, Wise, Josephs, & Frackowiak, 1996; Demonet et al., 1992; Wise et al., 1991). In addition to these structures, however, studies have shown activation in anterior brain structures, and in particular, in the inferior frontal gyrus [IFG] (Roskies, Fiez, Balota, Raichle, & Petersen, 2001; Wagner, Pare-Blagoev, Clark, & Poldrack, 2001; Poldrack et al., 1999; Gabrieli, Poldrack, & Desmond, 1998; Thompson-Schill, D’Esposito, Aguirre, & Farah, 1997; Petersen, Fox, Posner, Mintun, & Raichle, 1988; cf. Bookheimer, 2002; Binder & Price, 2001, for reviews). Much attention has been placed on these anterior structures and the role they play in processing word meaning. It has been suggested that, although long-term conceptual knowledge may be represented in the lateral temporal cortices, it is the frontal regions that play an executive role in the retrieval, selection, and recovery of word meaning. In a series of papers, Thompson-Schill, D’Esposito, and Kan (1999) and Thompson-Schill et al. (1997, 1998) have extended this proposal by hypothesizing that the left IFG is involved with selection of information among competing alternatives from semantic memory.

Data from a series of studies with Broca’s aphasic patients with lesions typically involving the left IFG and Wernicke’s aphasic patients with lesions typically involving the superior temporal gyrus (STG) and middle temporal gyrus (MTG) are consistent with the neuroimaging

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data. These studies have shown deficits in lexical processing in both groups of patients. In general, results indicate that both Broca's and Wernicke's aphasics show semantic priming in a lexical decision task, suggesting that these patients are able to map sound structure onto the lexicon and access the lexical-semantic network. However, as described below, although patients show sensitivity to phonetic category structure, lexical access deficits emerge when either acoustic or phonological structure is manipulated. Broca's aphasics have also been tested when these manipulations increase lexical competition; here, too, they exhibit deficits of lexical access.

The findings for the Broca's aphasics are of particular interest because they appear to dovetail with Thompson-Schill's hypothesis concerning the role of the IFG in selecting among competing alternatives. In particular, although Broca's aphasics are similar to normal controls, showing a reduction in the magnitude of semantic priming when the voice onset time (VOT) of the prime's initial stop consonant is shortened, and thus, is a poorer exemplar of the phonetic category, for example, *c<sup>2/3</sup>at-dog* shows less priming than *cat-dog* (Utman, Blumstein, & Sullivan, 2001), in contrast to normal controls, they *lose* semantic priming when the acoustically modified prime has a lexical competitor, for example, *p<sup>2/3</sup>ear* with the lexical competitor *bear* fails to prime *fruit* (Utman et al., 2001). Thus, Broca's aphasics show impairments in accessing the lexical-semantic network under conditions of lexical competition, suggesting that the left IFG is involved not only in the selection among competing semantic alternatives but also in the selection among competing lexical alternatives. However, increased competition may not be the entire story because, unlike controls, Broca's aphasics do not show mediated semantic priming via a lexical competitor. That is, when the prime is acoustically modified *p<sup>2/3</sup>ear*, the lexical competitor *bear* does not become active enough to prime *wolf* (Misiurski, Blumstein, Rissman, & Berman, 2005).

Although Thompson-Schill showed increased activation in the left IFG under conditions of competing semantic alternatives (Thompson-Schill et al., 1997), it is worth noting that in two of the three experimental tasks in that study, there was increased activation in the left temporal lobe including the middle and inferior temporal gyrus. What is not clear is what role the temporal lobes are playing under these conditions. Increased activation in an area does not indicate whether it plays the same role as other areas activated, nor does it indicate whether that area plays a necessary and sufficient role in the performance of the task (cf. Rorden & Karnath, 2004; Price, Mummery, Moore, Frackowiak, & Friston, 1999). Lesion data can provide a rich source of information in this regard because it is possible not only to investigate whether deficits emerge under particular experimental conditions, but it also allows for comparing potentially different patterns of performance on the same task for patients with different underlying neuropathologies.

To date, there have been no studies exploring potential lexical-semantic processing deficits under conditions of lexical competition in Wernicke's aphasics. However, a number of studies have shown that these patients do demonstrate lexical processing impairments. In contrast to normal controls, they show an equal magnitude of priming across all phonological conditions when a real word prime is changed to a phonologically similar nonword. For example, *gat-dog* and *wat-dog* show as much priming as does *cat-dog* (Milberg, Blumstein, & Dworetzky, 1988). In contrast, controls show a graded priming effect, and Broca's aphasics show priming only for *cat-dog* under these same conditions. In addition, in a case study, a Wernicke's aphasic displayed semantic priming at more timing intervals than do normal controls (Prather, Zurif, Love, & Brownell, 1997).

Although Wernicke's aphasics show lexical processing impairments, it is not clear whether they will show lexical processing impairments similar to Broca's aphasics under conditions of lexical competition. Given that the study of Thompson-Schill et al. (1997) showed increased temporal lobe activation under conditions of increased semantic competition, it is possible that Wernicke's aphasics will also show impairments. However, given that their performance has differed from that of Broca's aphasics in a number of lexical processing experiments (Prather et al., 1997; Swinney, Zurif, & Nicol, 1989; Milberg et al., 1988; Milberg, Blumstein, & Dworetzky, 1987), it is also possible that the patterns of impairments between the two groups will differ. Thus, the lesion data may provide a unique window into the computational properties of the anterior and posterior systems involved in lexical access in that the same behavioral task may give rise to different *patterns* of results as a function of clinical type of aphasia and underlying lesion. In the current study, we examine lexical access in patients with Broca's and Wernicke's aphasia in hopes of shedding light on the roles of anterior and posterior brain regions in lexical processing, and on the effects of competition on such processing.

### Computational Properties of the Lexical Access System

Most current models of auditory word recognition and lexical processing characterize the functional architecture of the speech-lexical processing system in terms of a distributed, network-like architecture with properties of activation, inhibition, and competition (Norris, 1994; Luce, Pisoni, & Goldinger, 1990; Dell, 1986; McClelland & Elman, 1986). Although this terminology suggests that these models reflect neural processes, they should not be construed as such but rather as metaphors for how information processing occurs in a cognitive system. Linguistic representations are considered as patterns of activation of either populations of units (e.g., Masson, 1995)

or as individual nodes (Dell, 1986). Every node (or population of representational units) has a resting state, rate of activation, a maximal level of activation, and a decay function over some temporal domain until it resumes its original resting state. The activation of these units may influence others through processes of spreading activation (and inhibition). The system is interactive, allowing for spreading activation to occur not only within a level of representation (e.g., within the lexical network) but also among different “levels” of representation, namely, between phonological, lexical, and semantic levels.

There are several consequences of this functional architecture. First, there is *graded activation* throughout the speech–lexical processing system. That is, activation patterns at a particular level of representation are not all-or-none but are graded. Second, there is *competition* among potential candidates. The extent of competition influences the time course and patterns of activation at each of the levels of representation, and ultimately, the performance of the entire system. Third, processing stages interact with each other such that activation patterns at one level will influence those at other levels. It is these three properties of the functional architecture (i.e., graded activation, competition, and interactivity) which guide the hypotheses that have been proposed concerning the lexical processing deficits of Broca’s and Wernicke’s aphasics.

There are two main hypotheses concerning the nature of the lexical processing deficits in Broca’s and Wernicke’s aphasics. One view is that the deficit for Broca’s and Wernicke’s aphasics lies in the *degree of activation* of lexical candidates. In this view, it is hypothesized that the overall activation in the lexicon is reduced in Broca’s aphasics and it is increased in Wernicke’s aphasics (Janse, 2006; Misiurski et al., 2005; McNellis & Blumstein, 2001; Utman et al., 2001; Blumstein & Milberg, 2000; Milberg et al., 1987, 1988). An alternative view is that the basis of the impairment for Broca’s and Wernicke’s resides in the *temporal course of lexical activation*. It is hypothesized that, for Broca’s aphasics, there is a delay in the time course of lexical activation, leading to a later than normal rise time (Swinney, Prather, & Love, 2000; Prather et al., 1997; Prather, Zurif, Stern, & Rosen, 1992; Swinney et al., 1989). For Wernicke’s aphasics, it is proposed that there is a delay in lexical *deactivation* (Prather et al., 1997).

Both hypotheses predict that semantic priming in Broca’s aphasics should be more difficult to detect than in controls. The reduced activation hypothesis also predicts that priming can be particularly vulnerable under conditions of lexical competition; because the overall activation level of a word is reduced, and thus, below some critical threshold, the system is unable to overcome activation of competitors. Thus, in this respect, reduced activation will have the same consequences as a selection impairment. With respect to Wernicke’s aphasics, both hypotheses predict that priming should occur in Wernicke’s aphasics in more circumstances than

normal. In particular, lexical competitors should remain active longer than normal.

It is difficult to distinguish between these theories using experimental methods such as lexical decision. Even with parametric manipulations of time by varying the interstimulus interval between prime–target pairs, it is difficult to quantify the time course of lexical activation as it unfolds across stimulus presentation. Recently, an eye-tracking paradigm has been developed that allows for the monitoring of lexical activation as participants listen to language input. Importantly, this paradigm allows both the activation of a lexical target, and its competitors, to be tracked over time. Several eye-tracking studies have shown that patterns of eye movements to pictures in a visual display reflect well-established lexical phenomena (for a review, see Tanenhaus, Magnuson, Dahan, & Chambers, 2000). In these studies, participants are presented with a four-picture display and asked to “pick up” (i.e., move with a computer mouse) one of the objects in the display (the *target*<sup>1</sup>). It has been found that if the name of one of the objects is an onset competitor of the target word, participants are initially more likely to fixate on this onset competitor than on objects with phonologically unrelated names. For example, if asked to “Pick up the *beaker*,” participants may fixate on *beetle* before settling on the target. Furthermore, as a word unfolds, the likelihood that a participant will fixate on its corresponding picture—and also on the picture of its phonological competitor—closely matches the word’s lexical activation as predicted by simulations using the TRACE model (McClelland & Elman, 1986) of spoken word recognition (Dahan, Magnuson, & Tanenhaus, 2001; Allopenna, Magnuson, & Tanenhaus, 1998). This correspondence suggests that when engaged in an active task (e.g., picking up an object), participants’ fixations are tightly linked to lexical activation. It has been hypothesized that this tight coupling is because the activation of a word’s representation determines the probability that a subject will shift visual attention to a picture representing that word (Tanenhaus et al., 2000; Allopenna et al., 1998). Eye movements therefore have the potential to provide detailed information about lexical activation in Broca’s and Wernicke’s aphasia, and, as a consequence, the role of anterior and posterior brain structures in lexical processing.

The current experiments explore the effects of lexical competition on spoken word recognition in Broca’s and Wernicke’s aphasia using the eye-tracking paradigm. The results may have implications both for the potential functional role of the frontal and temporal lobes in auditory word recognition under conditions of lexical competition, and for evaluating the degree of activation and time course of activation theories described above. Three experiments are conducted. Experiment 1 investigates whether Broca’s and Wernicke’s aphasics preferentially fixate on items semantically related to the uttered word, for example, given an auditory presentation of

“hammer,” will there be increased fixations to a picture of a *nail*? These findings should provide evidence about the ability of patients to map sound structure onto the lexicon and to access the lexical–semantic network. They also provide a critical baseline for Experiment 3. Experiment 2 explores whether Broca’s and Wernicke’s aphasics show competition effects from words that share their onsets with the uttered word, for instance, given the auditory presentation of “hammer,” will there be increased fixations to a picture of *hammock*? These findings should provide evidence about the ability of patients to select a word candidate under conditions of lexical form competition. Experiment 3 explores the extent to which Broca’s and Wernicke’s aphasics partially activate words semantically related to an onset competitor of the uttered word, for example, given the auditory presentation of “hammock,” will there be increased fixations to *nail* due to partial activation of the onset competitor “hammer”? These findings should provide evidence about the ability of patients to activate the lexical–semantic network of words that are phonological competitors of a heard lexical candidate. In each experiment, unimpaired, college-aged controls are tested to establish baseline effects, and unimpaired age-matched controls are tested to determine whether normal aging influences competitor effects.

### **EXPERIMENT 1: SEMANTIC RELATEDNESS EFFECT IN EYETRACKING**

Recent work has shown that normal adults show semantic relatedness effects when tested using the eye-tracking paradigm (Yee & Sedivy, 2006; Huettig & Altmann, 2005). Normal participants are more likely to fixate on a picture of an object semantically related to the target than on an object unrelated in sound or meaning. For example, when instructed to touch the “hammer,” subjects are more likely to fixate on a picture of a *nail* than on unrelated objects. This effect is not a consequence of visual similarity, lexical co-occurrence, or of attention being drawn to related items in the display (irrespective of the acoustic input). Instead, the effect appears to reflect the automatic activation of semantic information, with the probability that a subject will shift visual attention to a picture being determined by its activation.

Because most prior studies of lexical processing in patients with Broca’s and Wernicke’s aphasia have used the semantic priming paradigm, it is important to determine whether eye movements can be used to measure semantic activation in these patients. In this way, studies using the eye-tracking paradigm with Broca’s and Wernicke’s aphasics can be interpreted in the context of previous work on lexical activation. Experiment 1 was conducted to ascertain whether Broca’s and Wernicke’s aphasics show semantic-relatedness effects to an auditorily presented word. The question was whether Broca’s

and Wernicke’s aphasics would fixate more on an object that is semantically related to the auditory target stimulus than on a semantically unrelated object. This semantic eye-tracking paradigm should provide a means of exploring the time course over which aphasic patients activate words semantically related to an uttered target word.

### **Methods**

The methods described below are the same in Experiments 1 to 3, which were conducted in a single session. Any methods specific to Experiments 2 or 3 are described in their respective methods sections.

### *Subjects*

Twelve college-aged and 12 older control subjects were recruited from the Brown University community and surrounding area and were paid for their participation. The older control subjects were matched in age to the aphasic subjects (average age = 67 years).

The aphasic participants included six participants diagnosed with Broca’s aphasia and five diagnosed with Wernicke’s aphasia. Patient classification was based on performance on the Boston Diagnostic Aphasia Exam (BDAE) (Goodglass & Kaplan, 1972). The BDAE provides a profile of language abilities and impairments across a range of language functions, including measures of speech output (e.g., articulation, phrase length, articulatory agility, grammatical form), auditory comprehension (e.g., word discrimination, verbal commands, yes–no questions, word categories, and complex ideational material), naming, repetition, and paraphasia (sound substitutions and word substitutions). Diagnosis was made by review of performance on the BDAE and consensus by a team of researchers after evaluation of the patient.

The aphasic subjects all had unilateral lesions, and did not have an associated dementia or memory (e.g., Korsakoff) deficit. None had a significant history of other neurological or psychiatric illness or drug/alcohol abuse. All were literate in English, had English as the native language, and had normal hearing in the speech frequencies. All were several years poststroke. The average age of the aphasic subjects was 67 years. With the exception of one Wernicke’s subject whose data were excluded because of a right visual field neglect, all had normal or corrected-to-normal vision and no known oculomotor deficits. The response times of two patients (1 Broca’s aphasic and 1 Wernicke’s aphasic) were more than three standard deviations longer than the mean of the rest of the patients in their groups. Each also had an error rate more than two standard deviations higher than the means of the rest of the patients in his group. As a result, the data from these two participants were not included in the analyses. All of the remaining subjects

(5 Broca's and 3 Wernicke's aphasics) were able to understand the experimental task and performed well above chance on five practice trials. Further information about the aphasic participants is provided in Table 1. It is worth noting that although all five of the Broca's aphasic patients had lesions involving anterior areas, and four of these lesions involved Broca's area (the IFG), it is unclear whether the fifth patient's lesion extended into the IFG. All of the Wernicke's aphasics had lesions that

included the temporal lobe. Aphasic subjects were paid for their participation.

### Apparatus

An SMI EyeLink I head-mounted eye tracker was used to monitor participants' eye movements. A camera imaged the participant's left eye at 250 Hz. Stimuli were presented with PsyScript, a freely available language for

**Table 1.** Diagnostic Information about Aphasic Participants

<i>ID</i>	<i>Sex</i>	<i>Age at Testing</i>	<i>Years Post Onset</i>	<i>Auditory Comp. z Score</i>	<i>Fluency</i>	<i>Etiology</i>	<i>Lesion</i>
B01	F	58	6	+0.97	+0.80 (recovered Broca's)	CVA	Lesion in the anterior left MCA distribution centered on the Sylvian fissure and involving both gray and white matter; some extension into left temporal and inferior parietal area.
B02	M	67	9	+0.77	+0.50	CVA	Large left frontal infarct corresponding to occlusion of the anterior branches of the MCA (lateral frontal, frontal operculum; less severe in the motor cortex, caudate, putamen, and anterior limb of the internal capsule).
B03	M	60	18	+0.95	+0.57	CVA	Large left hemisphere lesion involving the caudate and globus pallidus, anterior internal capsule to the medial-temporal cortex and insula, and anterior PVWM. Hypodensity extending from Broca's area.
B04	M	74	18	+0.70	+0.20	CVA	Left MCA infarct involving Broca's area with deep extension involving subcallosal fasciculus. Patchy posterior lesion across temporal isthmus with superior extension to the premotor and sensory cortex.
B05	F	61	16	+0.95	+0.86 (recovered Broca's)	CVA	Large left insular lesion with possible extension into portions of the IFG and anterior temporal lobe.
W01	M	44	4	-0.39	+0.85	CVA	Cerebral infarct involving branches of the left MCA with primary involvement of the anterior left temporal lobe, adjacent frontal lobe and basal ganglia.
W02	M	70	7	+0.37	+0.93	CVA	Hemorrhagic infarct in the left temporal and parietal lobes extending into the basal ganglia-internal capsule region. Superior extension into the sensory cortex and the white matter and periventricular white matter deep to the lower premotor, motor, and sensory cortex areas.
W03	F	75	3	+0.17	+0.85	CVA	Left hemisphere lesion involves the subcortical temporal isthmus, the most posterior portion of Wernicke's area, and the white matter deep to Wernicke's area. Superior extension of lesion involves the supramarginal and angular gyri and the white matter deep to these areas.

MCA = middle cerebral artery; CVA = cerebrovascular accident; PVWM = periventricular white matter; IFG = inferior frontal gyrus.

scripting psychology experiments (Bates & Oliveira, 2003) on a 15-in. ELO touch-sensitive monitor. One Broca's and one Wernicke's aphasic were tested at Brown University. The rest of the aphasic patients were tested in their homes. The young and older control subjects were tested at Brown University.

### Materials

A female speaker (E.Y.), in a sound-treated room, read each target word in isolation with sentence-final intonation. The stimuli were recorded on a DAT tape and digitized at 20 kHz and a 14-bit quantization.

Stimuli consisted of 12 semantically related item pairs. Paired objects were related by virtue of being category coordinates and/or by having similar functions (e.g., *cherry-banana*, *battery-plug*). A complete list of experimental items is presented in the Appendix. Each critical trial display included a target picture, a semantically related picture, and two pictures that were phonologically unrelated to the target and semantically unrelated to the target and its onset and rhyme competitors.<sup>2</sup> In all critical trials, object positions, including the positional relationship between the target and the related item, were balanced so that each object type was equally likely to appear in each corner of the display. Average duration of the spoken target word was 529 msec and average number of syllables was 1.8. The related picture will be referred to as the *semantically related item*.

The names of the unrelated pictures in each critical trial were frequency-matched<sup>3</sup> with the name of the semantically related picture. To ensure that the pictures in critical trials clearly represented what they were intended to represent, picture-name correspondence pretests were conducted. Participants who did not participate in the eye-tracking study were presented with each picture and a label (either its intended name or a randomly selected name), and were asked to judge whether they matched. To ensure a high degree of picture-name correspondence, at least 15 of the 16 participants had to agree that the intended name matched the picture. A few of the pictures did not meet this criterion and were replaced with new pictures. These new pictures were presented to at least five participants (who did not participate in the experiments) who were asked to label each picture. If more than one of the participants did not provide the intended label for a picture, it was replaced with a new picture that was normed in the same way.

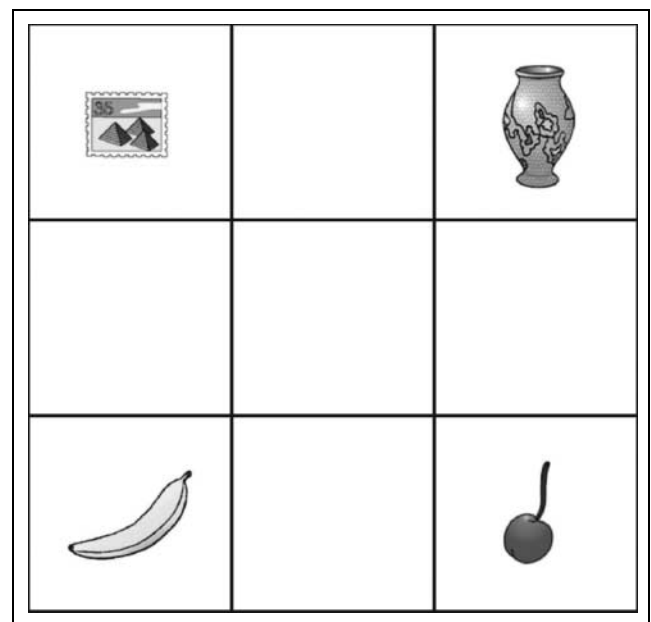
Average looking to the two unrelated pictures served as the baseline against which to compare looks to the related picture (the average of the two unrelated pictures was used in an effort to reduce the variance expected due to the small number of aphasic patients available for testing). Twelve distractor trials were included in which two of the objects in the display were semantically related, but in which neither related object was the tar-

get. Thus, even if any subjects noticed that some of the objects were related, they could not then predict that the target would be one of the related objects.

The testing session included 185 trials in all: 36 critical trials (12 for each of the three experiments), 24 distractor trials (12 for Experiment 1 and 12 for Experiment 2<sup>4</sup>), 96 filler trials, and 5 practice trials (an additional 24 trials were included in the testing session as part of a separate experiment that is described elsewhere [Yee, 2005]). Participants completed the testing in approximately 30–45 min. Fitting and calibrating the eye tracker required an additional 10–15 min. Trial order was randomized for each subject.

### Procedure

Participants were presented with a 3 × 3 array with four pictures on it, one in each corner (see Figure 1). Each cell in the array was approximately 2 × 2 in. Participants were seated at a comfortable distance (about 18 in.) from a touch-sensitive monitor, with the monitor at eye height. Therefore, each cell in the grid subtended about 6.4° of visual angle. (The eye tracker is accurate to less than one degree of visual angle.) One second after the display appeared, a red square appeared in the center of the screen. Participants were instructed to touch the red square when it appeared. Touching the red square both caused it to disappear and also triggered a sound file naming one of the objects in the display. The red square was included in the procedure to decrease the likelihood



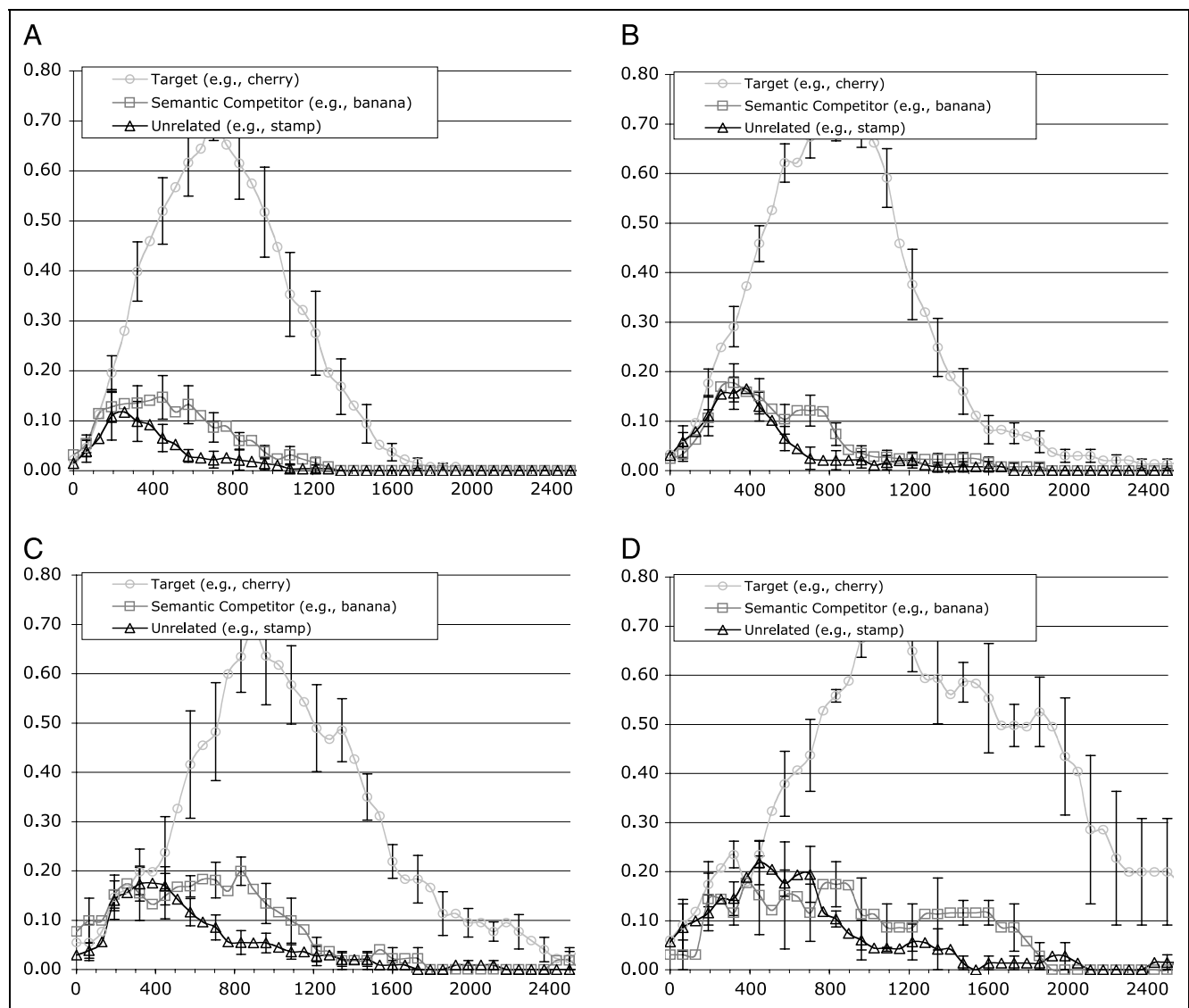
**Figure 1.** A sample display from Experiment 1 (semantic competitors). The target object (*banana*) is semantically related to one of the other objects in the display (the semantic competitor, *cherry*). The other two objects are unrelated semantically and phonologically to the target and its phonological competitors.

that participants would be fixating on one of the pictures at word onset. After the participant selected one of the pictures by touching it on the screen, the screen went blank and the trial ended. There were five practice trials, during and/or after which the instructions were repeated as necessary. Prior to any critical trials, there were also eight filler trials to further accustom subjects to the task.

## Results

Figure 2 plots the mean proportion of trials over time that contained a fixation to the target, the semantically related item, and the average of the two unrelated pictures (from target onset to 2500 msec after onset) in semantically related trials for each of the four subject groups (young controls, age-matched controls, Broca's aphasics, and Wernicke's aphasics, respectively). For the

purpose of analyzing the data, a "trial" was defined as starting 200 msec after the onset of the target and ending at 1800 msec after target onset, which is one standard deviation after the mean touch-screen response time averaged across all experiments and subjects. Fixations on the related item and the average of the two unrelated pictures at each 100 msec time bin of the trial was computed, and these differences were averaged into four time bins (200–600 msec, 600–1000 msec, 1000–1400 msec, and 1400–1800 msec). For each group, a 2 (related or unrelated)  $\times$  4 (time bins) repeated measures analysis of variance (ANOVA) was conducted on the resulting averages to determine if a relatedness effect emerged, and whether it interacted with time bin. For relatedness effects, one-tailed significance values are reported, as all groups are expected to display relatedness effects. Although main effects of time bin appeared



**Figure 2.** Experiment 1 proportion of fixations over time to the target, the semantically related object, and the average of the two unrelated objects. Standard error bars are shown for every other data point. (A) College-age controls; (B) Age-matched controls; (C) Five Broca's aphasics; (D) Three Wernicke's aphasics.

in every analysis, these effects are unsurprising and will not be discussed. When a relatedness effect interacted significantly with time bin, follow-up paired *t* tests were conducted on each time bin to determine when the effects emerged. When the assumption of sphericity was violated, a Greenhouse–Geisser correction was applied.

### Young Controls

Figure 2A plots the data from the young controls in semantically related trials. Three trials (2.1% of the 144 total trials for the 12 young controls) were excluded because the incorrect picture was selected. In each of these cases, the semantically related item was selected (two *wallet* → *purse* and one *battery* → *plug*). Eight trials (5.6%) did not provide any data because there were no eye movements after the onset of the target word (most of these were trials in which the participant was already fixating on the picture of the target object at word onset).

Results show a significant main effect of relatedness such that the average probability of fixation on the semantically related item's picture was significantly greater than the average of the two unrelated pictures [ $F_1(1, 11) = 10.5, p < .01$  by subjects, and  $F_2(1, 11) = 8.6, p < .01$  by items]. The interaction of relatedness with time bin was close to significant by subjects [ $F_1(1.7, 18.2) = 2.6, p = .10$ ; (Greenhouse–Geisser corrected *df* and *p* value)] and was significant by items [ $F_2(3, 33) = 3.4, p = .03$ ]. Follow-up paired *t* tests indicated that the difference between fixations on the semantically related item and the two unrelated pictures (henceforth, the semantic relatedness effect) was significant only in the bins from 200 to 600 and from 600 to 1000 msec after target onset ( $p \leq .05$  by both subjects and items, one-tailed).

### Age-matched Controls

Figure 2B plots the data from the age-matched controls in semantically related trials. Three trials (2.1% of the 144 total trials for the 12 age-matched controls) were excluded because the incorrect picture was selected (*tepee* → *igloo*, *muffin* → *doughnut*, *pie* → *clover*). In two of these trials, the semantically related item was selected. Eleven trials (7.6%) did not provide any data because there were no eye movements after the onset of the target word.

Similar to the young normal controls, the age-matched controls exhibited a significant main effect of relatedness; the related picture was fixated on significantly more than the average of the two unrelated pictures [ $F_1(1, 11) = 9.2, p < .01$  by subjects, and  $F_2(1, 11) = 6.1, p = .02$  by items]. The interaction of relatedness with time bin was not significant.

### Broca's Aphasics

Figure 2C plots the data from Broca's aphasics in semantically related trials. Four trials (6.7% of the 60 total

trials for the 5 Broca's aphasics) were excluded because the incorrect picture was selected (*wallet* → *purse*, *pie* → *clover*, *tepee* → *igloo*, *muffin* → *doughnut*). In three of these trials, the semantically related item was selected. Two (3.3%) trials did not provide any data because there were no eye movements after the onset of the target word.

The Broca's aphasics showed a main effect of relatedness. The semantically related picture was fixated on significantly more than the average of the two unrelated pictures [ $F_1(1, 4) = 6.2, p = .03$  by subjects, and  $F_2(1, 11) = 4.7, p = .03$  by items]. The interaction of relatedness with time bin was not significant.

### Wernicke's Aphasics

Figure 2D plots the data from Wernicke's aphasics in semantically related trials. One trial (2.7% of the 36 total trials for the 3 Wernicke's aphasics) was excluded because the incorrect picture was selected (*tepee* → *igloo*). Three trials (6.3%) did not provide any data because there were no eye movements after the onset of the target word.

The Wernicke's aphasics fixated on the semantically related picture more than the average of the two unrelated pictures, although this difference only approached significance both by subjects [ $F_1(1, 2) = 3.9, p = .09$ ] and by items [ $F_2(1, 11) = 1.4, p = .13$ ]. The interaction of relatedness with time bin was not significant.

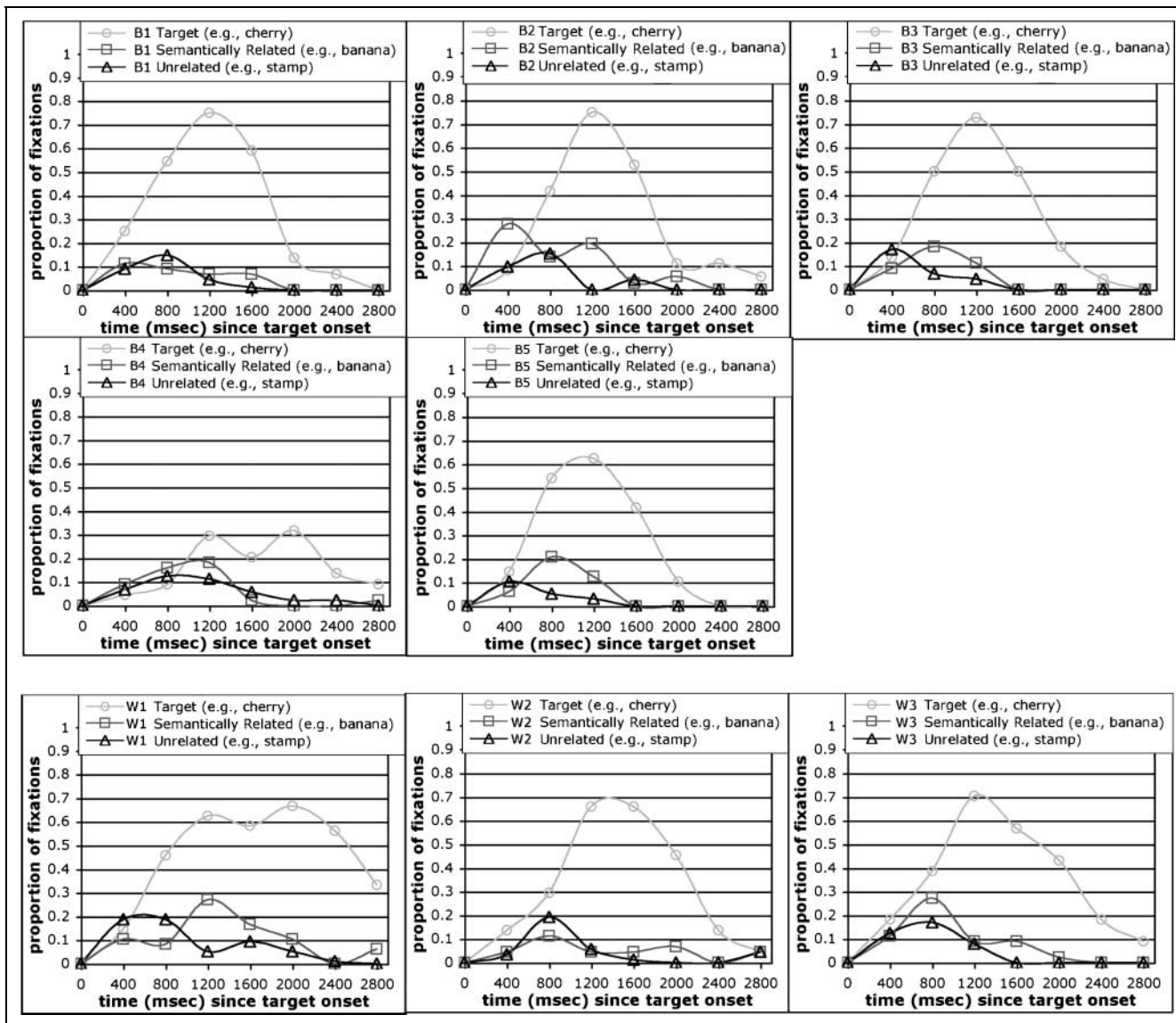
### Comparison of Groups

The relatedness effects (the differences between the semantically related item and the average of the two unrelated pictures, averaged across the entire trial) from each of the four groups were submitted to a one-way ANOVA with three planned contrasts: Broca's aphasics versus Wernicke's aphasics, and each aphasic group versus the age-matched controls. The omnibus result was not significant. The planned contrasts revealed no differences by subjects or by items. An ANOVA by items and a mixed-model ANOVA with time as a factor were not appropriate due to lack of homogeneity of variance between groups. Figure 3 shows individual aphasic patient data from Experiment 1 for both groups of aphasic patients.

### Discussion of Experiment 1

Experiment 1 demonstrates that like young controls, older controls, Broca's aphasics, and Wernicke's aphasics fixate more on pictures of objects semantically related to the target than on semantically unrelated pictures. Although the relatedness effect for the three Wernicke's aphasics was not statistically significant, there was a clear trend in the data for a semantic





**Figure 3.** Experiment 1 individual aphasic patient data. Top two rows are Broca's aphasic patients, bottom row is Wernicke's aphasic patients.

relatedness effect. Furthermore, the sole error committed in this group was in the selection of the semantically related object in lieu of the target. When eye-movement data from this trial is included in the analysis, the Wernicke's aphasics as a group do show a significant semantic relatedness effect. Taken together, these results are consistent with studies that demonstrated semantic priming in Broca's and Wernicke's aphasics using paired prime-target stimuli (Hagoort, 1997; Prather et al., 1997; Tyler, Ostrin, Cooke, & Moss, 1995; Prather, Love, Finkel, & Zurif, 1994; Ostrin & Tyler, 1993; Swinney et al., 1989; Milberg et al., 1988; Blumstein, Milberg, & Shrier, 1982). Of interest, the average size of the semantic relatedness effect did not differ significantly across groups.

It is worth noting that for the patients, the time at which they began to fixate more on the target than on the other objects in the display was delayed<sup>5</sup> and the

trial end time was later compared to normal subjects. These findings are consistent with studies of lexical processing in aphasics. Overall, aphasic patients show slower response latencies in language tasks, and although both Broca's and Wernicke's aphasics show semantic priming in a lexical decision task, their responses are typically and consistently slower than that of normal controls (Blumstein et al., 1982; Milberg & Blumstein, 1981). Nonetheless, despite their delayed fixations to the target, they still show more fixations to the semantically related than to the unrelated pictures, similar to normal controls.

## EXPERIMENT 2: ONSET COMPETITION IN APHASIA

Many words are similar in their sound shape, and thus, in order to access the appropriate lexical candidate, it is

necessary to select it from a set of potential candidates. For example, because speech is produced sequentially and a limited number of speech sounds are used to produce thousands of unique words, at a given moment a sequence may be consistent with any one of a large number of words (e.g., “ham,” could continue as “hammer,” “hammock,” “hamster,” “hamper,” “hamstring,” etc.).

An overlap in the onsets of such stimuli leads to slowed lexical decisions when a word shares three initial phonemes with a previously heard word. One explanation for this effect is that when a word with a highly similar onset competitor is heard, both candidates are initially activated. As more phonological information comes in and the correct candidate is uniquely isolated, the correct candidate’s activation is boosted while at the same time the onset competitor’s activation is inhibited. When the onset competitor is subsequently presented, responses to it are delayed, presumably because the first word is a stronger candidate (Slowiaczek & Hamburger, 1992).

The results of a recent study (Janse, 2006) show that both Broca’s and Wernicke’s aphasics display impairments under conditions of lexical onset competition in an auditory lexical decision task. In contrast to neurologically intact subjects who showed the expected inhibitory effect, both Broca’s and Wernicke’s aphasics showed impairments. Wernicke’s aphasics showed a significant priming effect. That is, unlike controls, their response latencies were not slowed down, but were instead *faster* for targets presented in the context of stimuli that had onset competitors. This pattern of results suggests a deficit in inhibiting lexical competitors. If true, then in the current eye-tracking study, this deficit in inhibiting lexical competitors should reveal itself in increased looks to the onset competitor. In contrast to the Wernicke’s aphasics and normal participants, Broca’s aphasics showed a weak, but nonsignificant, inhibitory effect. Because of the considerable variability of performance among the subjects, the interpretation of this effect is less clear. One possibility is that because of reduced activation, onset competitors do not become as active for Broca’s aphasics, and consequently, have less influence on processing. Regardless of the source, however, the findings suggest that there is a deficit in the processes of lexical activation.

The activation of a word’s onset competitors has the potential to be a valuable measure of lexical activation because it allows for distinguishing between the reduced activation and delayed time course of activation hypotheses for Broca’s aphasics. The reduced activation hypothesis (Milberg et al., 1987) predicts that Broca’s aphasics will show an abnormally small onset competitor effect (i.e., an abnormally small preference to fixate on the onset competitor rather than an unrelated object); because lexical activation is reduced, onset competitors will not become as active as they will for normal

subjects. The delayed activation hypothesis (Prather et al., 1992, 1997; Swinney et al., 1989) predicts that, for Broca’s aphasics, an onset competitor effect should emerge, but it should emerge later than it does for normal controls.

Both theories predict similar patterns of results for Wernicke’s aphasics. With increased lexical activation or a failure to inhibit lexical competitors, onset competitors will be more active than they will for normal subjects, resulting in a larger competitor effect. The slowed deactivation hypothesis proposes that onset competitors will remain active for longer than normal, resulting in an abnormally large competitor effect.

## Methods

### Materials

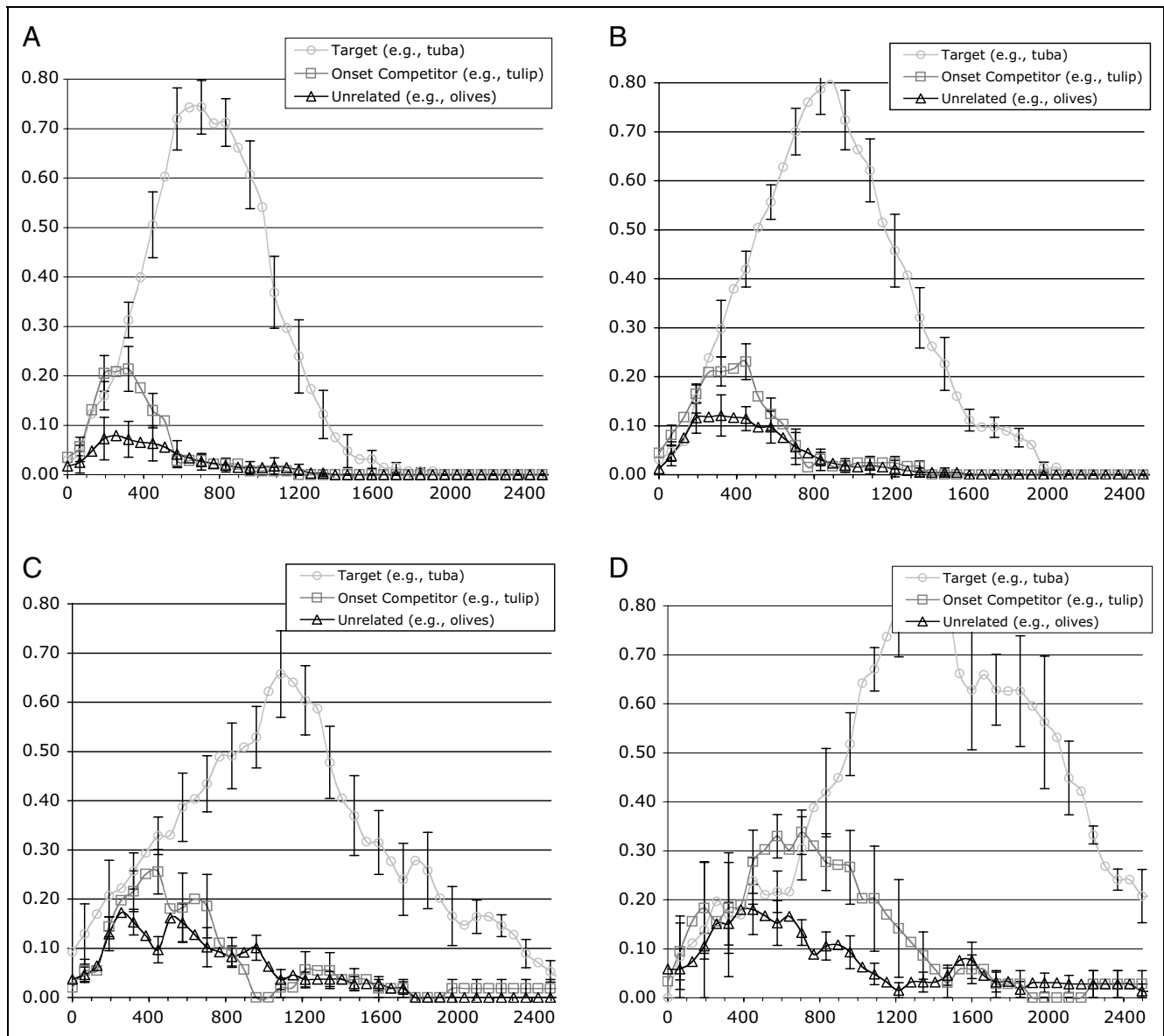
Twelve 2-syllable pictureable nouns served as target words. For each of these target words, there was a pictureable noun that was a phonological onset competitor (e.g., *hammer*–*hammock*; see Appendix). All of these onset competitors overlapped with the target by either their entire first syllable (10 of 12 items) or the onset and vowel of the first syllable (2 of 12 items). Twelve distractor trials were included in which two of the objects in the display were onset competitors, but in which neither related object was the target. Thus, even if any subjects noticed that some of the object names were onset competitors, they could not then predict that the target would be one of the related objects. Average duration of the spoken target word was 527 msec. The competitor picture will be referred to as the *onset competitor*.

## Results

Figure 4 plots the mean proportion of trials over time that contained a fixation to the target, to the onset competitor, and to the average of the two unrelated pictures (from target onset to 2500 msec after onset) in onset competitor trials for each of the four subject groups (young controls, age-matched controls, Broca’s aphasics, and Wernicke’s aphasics, respectively). For the purpose of analyzing the data, a “trial” was defined in the same way as it was in Experiment 1. The data were analyzed using the same procedures used in Experiment 1. As in Experiment 1, for each group, a 2 (related or unrelated)  $\times$  4 (time bins) repeated measures ANOVA was conducted to determine if an onset competitor effect emerged and whether it interacted with time bin.

### Young Controls

Figure 4A plots the mean proportion of trials over time that contained a fixation to the target, to the onset



**Figure 4.** Experiment 2 proportion of fixations over time to the target (e.g., *tuba*), the onset competitor (e.g., *tulip*), and the average of the two unrelated objects. Standard error bars are shown for every other data point. (A) College-age controls; (B) Age-matched controls; (C) Five Broca's aphasics; (D) Three Wernicke's aphasics.

competitor, and to the average of the two unrelated pictures in onset competitor trials for the young controls. One trial (0.7% of the 144 total trials for the 12 young normal subjects) was excluded because the onset competitor was selected instead of the target (*pillow* → *pillar*). Seven trials (4.9%) did not provide any data because there were no eye movements after the onset of the target word (most of these were trials in which the subject was already fixating on the picture of the target at the onset of the target word).

Results show a significant main effect of relatedness, such that there were significantly more fixations on the onset competitor than on the average of the two unrelated objects [ $F_1(1, 11) = 7.9, p < .01$  by subjects, and  $F_2(1, 11) = 9.7, p < .01$  by items]. The interaction

of relatedness with time bin was also significant by subjects [ $F_1(1.2, 13.0) = 8.7, p < .01$ ] and by items [ $F_2(1.6, 17.5) = 13.9, p < .01$ ; Greenhouse-Geisser corrected  $df$  and  $p$  value]. Follow-up paired  $t$  tests indicated that the difference between fixations on the onset competitor and the average of fixations on the two unrelated pictures (henceforth, the onset competitor effect) was significant only in the bin from 200 to 600 msec after target onset ( $ps < .01$  by both subjects and items, one-tailed).

#### Age-matched Controls

Figure 4B plots the data from the age-matched controls in onset competitor trials. One trial (0.7% of the 144 total

trials for the 12 age-matched controls) was excluded because the onset competitor was selected instead of the target (one instance of *basket* → *bathtub*). Eight trials (5.6%) did not provide any data because there were no eye movements after the onset of the target word.

The age-matched controls also displayed a significant main effect of relatedness; fixations on the onset competitor's picture were significantly more than fixations on the two unrelated pictures by subjects [ $F_1(1, 11) = 6.0, p = .02$ ] and by items [ $F_2(1, 11) = 5.0, p = .02$ ]. The interaction of relatedness with time bin was also significant by subjects [ $F_1(3, 33) = 7.2, p < .01$ ], but not quite significant by items [ $F_2(1.4, 15.6) = 3.4, p = .07$ ; Greenhouse–Geisser corrected *df* and *p* value]. Follow-up paired *t* tests indicated that the onset competitor effect was significant only in the bin from 200 to 600 msec after target onset ( $ps < .02$  by both subjects and items, one-tailed).

### *Broca's Aphasics*

Figure 4C plots the data from the Broca's aphasics in onset competitor trials. One trial (1.7% of the 60 total trials for the 5 Broca's aphasics) was excluded because the onset competitor was selected instead of the target (*penny* → *pencil*). Four trials (6.7%) did not provide any data because there were no eye movements after the onset of the target word.

Although fixations on the onset competitor's picture were greater than fixations on the two unrelated pictures, there was no main effect of relatedness by subjects [ $F_1(1, 4) = 0.6, p = .25$ ], and the item analysis only approached significance [ $F_2(1, 11) = 1.7, p = .11$ ]. There was also no interaction of relatedness with time bin.

### *Wernicke's Aphasics*

Figure 4D plots the data from the Wernicke's aphasics in onset competitor trials. In all trials, the correct picture was selected. Three trials (8.3% of the 36 total trials for the Wernicke's aphasics) did not provide any data because there were no eye movements after the onset of the target word.

The Wernicke's aphasics showed a main effect of relatedness; fixations on the onset competitor's picture were significantly greater than the average of fixations on the two unrelated pictures by subjects [ $F_1(1, 2) = 13.2, p = .03$ ] and by items [ $F_2(1, 11) = 6.2, p = .02$ ]. The interaction of relatedness with time bin was not significant.

### *Comparison of Groups*

The onset competitor effects (the differences between the onset competitor and the average of the two unrelated pictures, averaged across the entire trial) from each of the four groups were submitted to a one-way ANOVA with three planned contrasts: Broca's aphasics

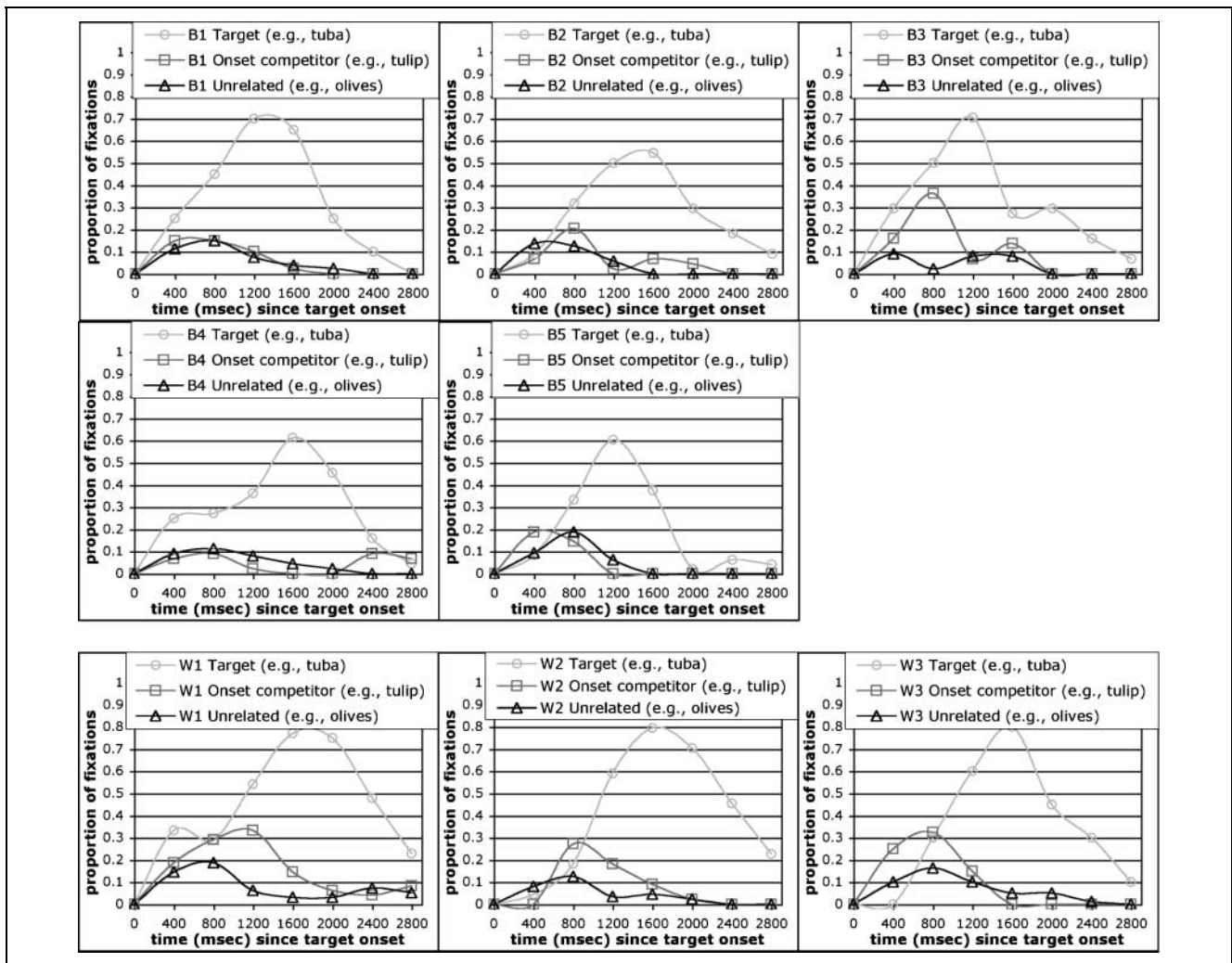
versus Wernicke's aphasics, and each aphasic group versus the age-matched controls. The overall *F* approached statistical significance by subjects [ $F_1(3, 28) = 2.6, p = .07$ ]. An ANOVA was not appropriate by items due to lack of homogeneity of variance between groups. The planned contrasts showed that the onset competitor effect was significantly larger for the Wernicke's aphasics than for the Broca's aphasics by subjects ( $p = .02$ ) and by items ( $p = .02$ ). The onset competitor effect for the Wernicke's aphasics was also significantly larger than that of the age-matched controls by subjects ( $p = .01$ ) and by items ( $p = .02$ ). No other differences were found. Figure 5 shows individual aphasia patient data from Experiment 2 for both groups of aphasic patients.

## **Discussion of Experiment 2**

The results of Experiment 2 showed that like young controls, older controls and Wernicke's aphasics fixate significantly more on pictures of objects that share their onsets with the uttered word than on unrelated objects. The onset competitor effect for the young and older controls appeared similar to the results obtained for onset competitors in Allopenna et al. (1998). For Broca's aphasics, the onset competitor effect was not statistically significant. However, their onset competitor effect was also not statistically different from that of the age-matched controls. Importantly, the Wernicke's aphasics' onset competitor effect was significantly larger than the effect in the age-matched controls and the Broca's aphasics.

Although the Broca's aphasics' onset competitor effect was not statistically significant, a visual comparison of the competitor effects for older controls and Broca's aphasics (Figure 4B and C) suggests that the Broca's aphasics' may have shown a competitor effect, but the smaller number of Broca's aphasics tested may account for the difference between the nonsignificant results obtained for the Broca's aphasics and the significant result obtained for the controls. As an informal measure of how likely it would be for an onset competitor effect of the size obtained for the five Broca's aphasics to be obtained from a group of five of our control subjects, Monte Carlo sampling was used to obtain the size of the onset competitor effect (i.e., the *t* statistic for the difference between the competitor and the unrelated stimuli) for 10,000 samples of five randomly selected subjects from among the 12 age-matched control subjects. In 86% of these 10,000 randomly selected control groups, the onset competitor effect was larger than it was for the Broca's aphasics. This result suggests that the weakness of the onset competitor effect observed in the Broca's aphasics is not due to the sample size but reflects an abnormally small onset competitor effect.

It is noteworthy that the Broca's and the Wernicke's aphasics displayed different patterns of results. Although the heightened onset competitor effect that appeared for the Wernicke's aphasics could be explained by slower



**Figure 5.** Experiment 2 individual aphasic patient data. Top two rows are Broca's aphasic patients, bottom row is Wernicke's aphasic patients.

processing in general, no increased onset competitor effect appeared for the Broca's aphasics, who also responded more slowly than the normal subjects. Similarly, although the statistically insignificant onset competitor effect that appeared for the Broca's aphasics could be due to low power, the Wernicke's aphasics group, which was even smaller, showed an onset competitor effect that was significantly larger than that of controls.

The pattern of results obtained for Broca's aphasics in this experiment (i.e., a trend toward an abnormally small onset competitor effect) cannot be explained by delayed activation; delayed activation should cause a late-appearing but normal-sized competitor effect. Instead, the results are consistent with the reduced activation hypothesis. The abnormally large onset competitor effect obtained for Wernicke's aphasics is consistent with either the increased activation hypothesis or the delayed deactivation hypothesis. Increased lexical activation (or a failure to inhibit competitors) should cause the onset competitor to remain more strongly activated (or less inhibited) than normal, and thus, cause it to draw more

fixations. Delayed deactivation should result in a large number of fixations on the onset competitor because it would cause abnormally long-lasting competition.

### EXPERIMENT 3: SEMANTICALLY MEDIATED ONSET COMPETITION

Prior research (Yee & Sedivy, 2006) has shown that, for young normal subjects, eye movements are sensitive to the activation of words semantically related to an onset competitor of the target (e.g., *hammock* → *nail*). These findings are consistent with the view that hearing a word not only activates its lexical representation, and partially activates its phonological onset competitor, but that it also, in turn, activates the onset competitor's lexical-semantic network.

Based on the results of Experiments 1 and 2, Broca's aphasics should fail to show a *semantic onset competitor effect*. That is, they should fail to show increased fixations to an object semantically related to an onset competitor of a given target. Despite the fact that Broca's aphasics

displayed a normal-sized semantic effect in Experiment 1, the underactivation of an onset competitor to a target, as shown in Experiment 2, should result in a failure to activate the lexical-semantic network of the competitor. In contrast, the normal semantic priming effect in Experiment 1 for Wernicke's aphasics, coupled with their abnormally large onset competitor effect, suggests that they will be more likely to fixate on an object semantically related to an onset competitor of a given target than on an unrelated object. Furthermore, because Wernicke's aphasics showed a larger onset competitor effect than normal subjects, they should also show a larger semantic onset competitor effect.

## Methods

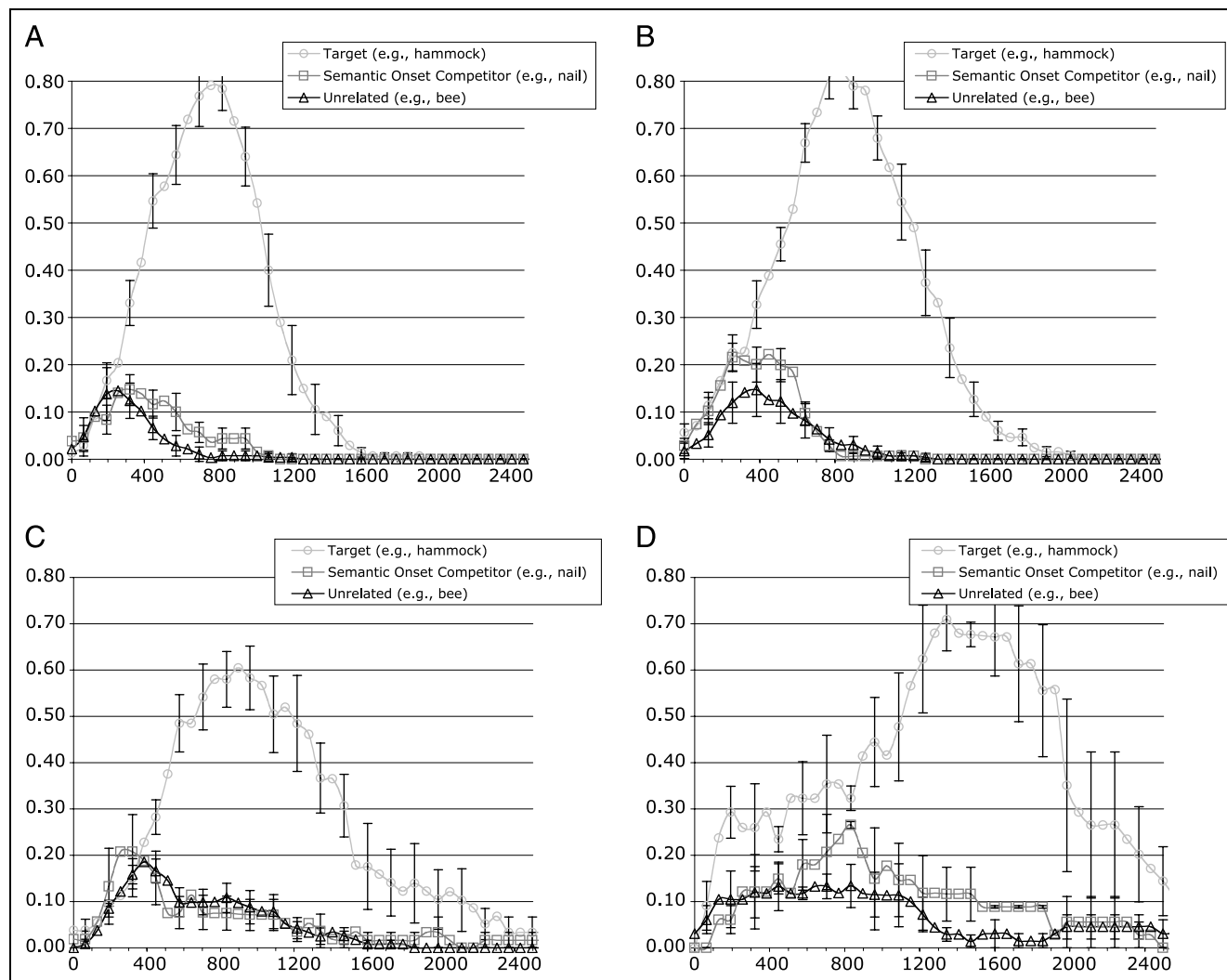
### Materials

Twelve semantically mediated onset competitor pairs were selected from the materials used in Yee and Sedivy

(2006). In most cases, the target's onset competitor and the semantically related object were related by virtue of being category coordinates and/or by having similar functions (e.g., *mattress-lighter* via *matches*, *panther-shirt* via *pants*). In the remaining cases, the objects were used together [e.g., *logs-key* (via *lock*), *hammock-nail* (via *hammer*)]. These pairs had no overlap with the pairs used in Experiment 1 or 2 (see Appendix). The average duration of the spoken target word was 615 msec and the average number of syllables was 2.2. The competitor picture will be referred to as the *semantic onset competitor*.

## Results

Figure 6 plots the mean proportion of trials over time that contained a fixation to the target, to the semantic onset competitor, and to the average of the two unrelated pictures (from target onset to 2500 msec after



**Figure 6.** Experiment 3 proportion of fixations over time to the target (e.g., *hammock*), the semantic onset competitor (e.g., *nail*), and the average of the two unrelated objects. Standard error bars are shown for every other data point. (A) College-age controls; (B) Age-matched controls; (C) Five Broca's aphasics; (D) Three Wernicke's aphasics.

onset) in semantic onset competitor trials for each of the four subject groups (young controls, age-matched controls, Broca's aphasics, and Wernicke's aphasics, respectively). For the purpose of analyzing the data, a "trial" is defined in the same way as it was in Experiments 1 and 2. The data were analyzed using the same procedures used in Experiments 1 and 2.

### Young Controls

Figure 6A plots the data for the young controls in the semantic onset competitor trials. The correct picture was selected in all trials. Ten trials (6.9%) did not provide any data because there were no eye movements after the onset of the target word.

Results show a significant main effect of relatedness such that the semantic onset competitor's picture was fixated on significantly more than the average of the two unrelated pictures by subjects [ $F_1(1, 11) = 4.4, p = .03$ ] and by items [ $F_2(1, 11) = 6.5, p = .01$ ]. The interaction of relatedness with time bin only approached significance by subjects [ $F_1(1.5, 16.9) = 2.2, p = .15$ ] and by items [ $F_2(1.4, 15.2) = 2.5, p = .13$ ; Greenhouse–Geisser corrected  $df$  and  $p$  values].

### Age-matched Controls

Figure 6B plots the data from the age-matched controls in semantic onset competitor trials. As was the case for the young controls, the correct picture was selected in all trials. Ten trials (6.9%) did not provide any data because there were no eye movements after the onset of the target word.

Similar to the young normal controls, the age-matched controls exhibited a significant main effect of relatedness; there were significantly more fixations on the semantic onset competitor's picture than the average of the two unrelated pictures by subjects [ $F_1(1, 11) = 3.9, p = .04$ ] and by items [ $F_2(1, 11) = 3.2, p = .05$ ]. The interaction of relatedness with time bin was also significant by subjects [ $F_1(1.3, 14.8) = 8.1, p < .01$ ] and by items [ $F_2(1.3, 14.2) = 4.4, p = .05$ ; Greenhouse–Geisser corrected  $df$  and  $p$  values]. Follow-up paired  $t$  tests indicated that the semantic onset competitor effect was significant only in the bin from 200 to 600 msec after target onset ( $p \leq .02$  by both subjects and items, one-tailed).

### Broca's Aphasics

Figure 6C plots the data from the Broca's aphasics in semantic onset competitor trials. The correct picture was selected in all trials. Seven trials (11.7%) did not provide any data because there were no eye movements after the onset of the target word.

Results for Broca's aphasics indicated no main effect of relatedness; fixations to the picture of the semantic

onset competitor did not differ from the average of the two unrelated pictures by subjects [ $F_1(1, 4) = 0.0$ ] or by items [ $F_2(1, 11) = 0.1$ ]. There was no interaction of relatedness with time bin.

### Wernicke's Aphasics

Figure 6D plots the data from the Wernicke's aphasics. Two trials (4.2%) were excluded because the wrong picture was selected (both were *mattress* → *lighter*). One trial (2.1%) did not provide any data because there were no eye movements after the onset of the target word.

The Wernicke's aphasics fixated on the semantic onset competitor's picture more than the average of the two unrelated pictures, although this difference only approached significance both by subjects [ $F_1(1, 2) = 4.2, p = .09$ ] and by items [ $F_2(1, 11) = 1.6, p = .11$ ]. The interaction of relatedness with time bin was not significant.

### Comparison of Groups

The semantic onset competitor effects (the differences between the semantic onset competitor and the average of the two unrelated pictures, averaged across the entire trial) from each of the four groups were submitted to a one-way ANOVA with three planned contrasts: Broca's aphasics versus Wernicke's aphasics, and each aphasic group versus the age-matched controls. We also included a linear term in the ANOVA because (as described above) the results of Experiment 2 led us to predict that Broca's aphasics should fail to show a semantic onset competitor effect, whereas Wernicke's aphasics should show an amplified effect. The linear term should capture any such monotonic pattern.

The omnibus ANOVA was not significant by subjects [ $F_1(3, 28) = 1.3, p = .29$ ], and an ANOVA was not appropriate by items due to lack of homogeneity of variance between groups. However, the linear term approached significance [ $F_1(1, 28) = 3.9, p = .06$ ], and the planned contrasts showed that the competitor effect was significantly larger for the Wernicke's aphasics than for the Broca's aphasics by subjects ( $p = .03$ ) and by items ( $p = .05$ ) (both one-tailed). The competitor effect for the Wernicke's aphasics was close to significantly larger than that of the age-matched controls by subjects ( $p = .07$ ), but not by items ( $p = .18$ ) (both 1-tailed). No other differences were found. Figure 7 shows individual aphasia patient data from Experiment 3 for both groups of aphasic patients.

## Discussion of Experiment 3

The results from Experiment 3 show that like young controls, older controls and Wernicke's aphasics are more likely to fixate on a picture of an object semantically related to an onset competitor of the target than on an unrelated object. Although the semantic onset

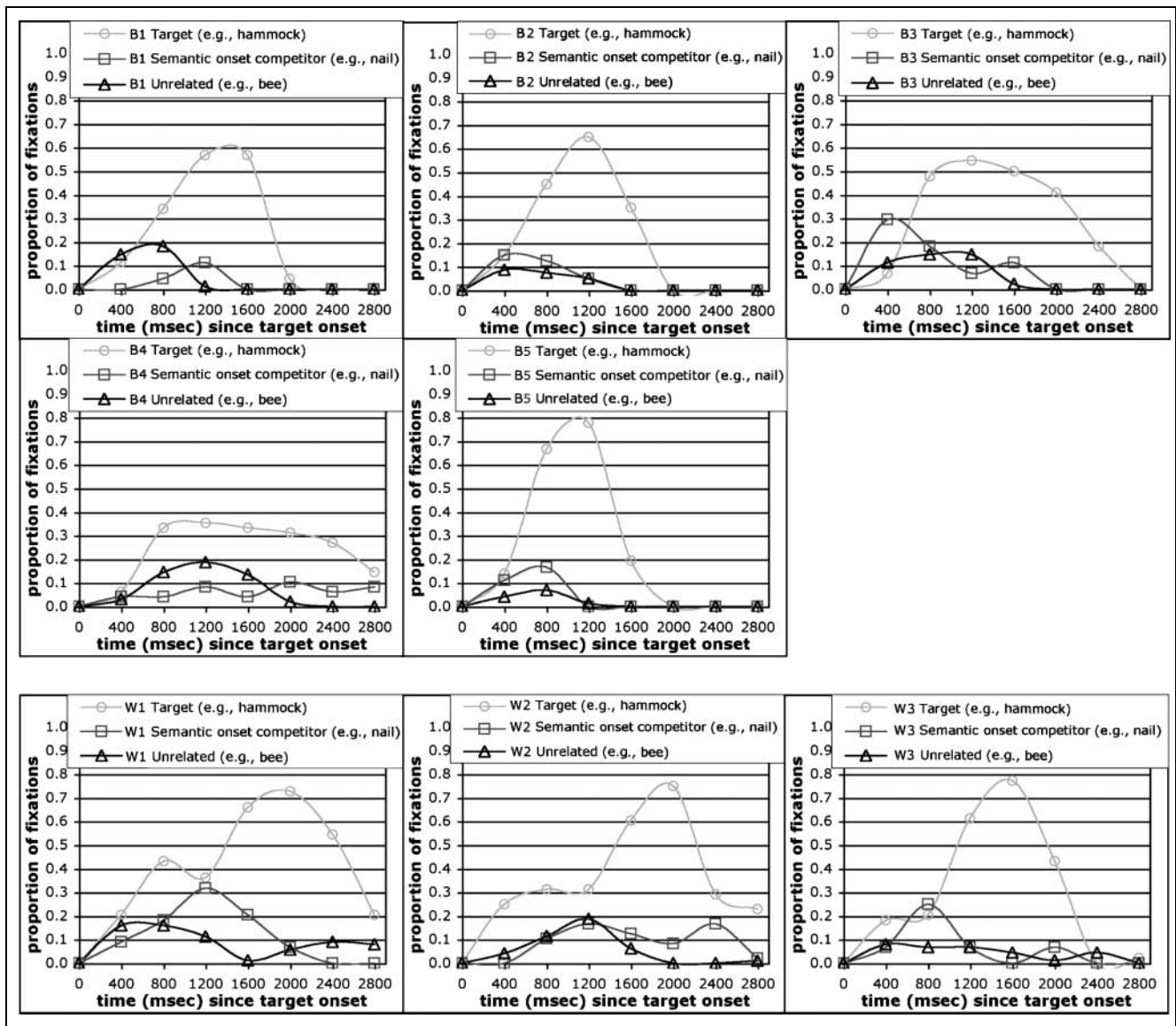


Figure 7. Experiment 3 individual aphasic patient data. Top two rows are Broca's aphasic patients, bottom row is Wernicke's aphasic patients.

competitor effect for the three Wernicke's aphasics only approached significance, there was a clear trend in the data for a semantic onset competitor effect. Moreover, the semantic onset competitor effect for Wernicke's aphasics was significantly larger than that of the Broca's aphasics and was close to being significantly larger by subjects than that of the age-matched controls. Broca's aphasics, on the other hand, showed no evidence of competition from the semantic onset competitor.

Activating the semantic onset competitor (e.g., *nail*) requires that the onset competitor (e.g., *hammer*) be partially active. In the current experiment, as in Experiment 2, Wernicke's aphasics showed a larger competitor effect than Broca's aphasics. Furthermore, in both Experiment 2 and the current experiment, the competitor effect was larger for the Wernicke's aphasics than it was for the age-matched controls (although in the current experiment the difference between the Wernicke's

aphasics and the age-matched controls only approached statistical significance by subjects).

The absence of a semantic onset competitor effect for Broca's aphasics is consistent with the reduced activation hypothesis because words semantically related to the onset competitors would fail to draw fixations from Broca's aphasics if the onset competitors themselves failed to become active or were weakly activated. Because a semantic onset competitor effect did not emerge later in the processing stream, the results for Broca's aphasics are consistent with the reduced activation hypothesis but not with delayed activation. However, it is important to acknowledge that despite the fact that Broca's aphasics failed to display a semantic onset competitor effect, paired comparisons between the effect in the Broca's aphasics and the normal controls yielded no statistically reliable differences. As an informal measure of how likely it would be for a semantic



onset competitor effect of the size obtained for the five Broca's aphasics to be obtained from a group of five of our control subjects, Monte Carlo sampling was used to obtain the size of the semantic onset competitor effect (i.e., the  $t$  statistic) for 10,000 samples of five randomly selected subjects from among the 12 age-matched control subjects. In 95% of these 10,000 randomly selected control groups, the semantic onset competitor effect was larger than it was for the Broca's aphasics. This result suggests that the absence of a significant semantic onset competitor effect in the Broca's aphasics is not due to the sample size but reflects an abnormally small onset competitor effect.

Although the difference between the Wernicke's aphasics semantic onset competitor effect and that of the age-matched controls did not quite reach the  $p = .05$  level, the large semantic onset competitor effect exhibited by the Wernicke's aphasics is consistent with both the increased activation and the delayed deactivation hypotheses. For Wernicke's aphasics, increased activation (or a failure to inhibit lexical competitors) should result in onset competitors becoming more strongly activated (or less inhibited). As a result, words semantically related to onset competitors should draw more fixations than normal. The semantic onset competitor effect displayed by the Wernicke's aphasics is also compatible with the delayed deactivation hypothesis because delayed deactivation should cause longer duration fixations and/or more fixations on the semantic onset competitor.

## GENERAL DISCUSSION

The results of this series of experiments provide a window into the nature of the neural systems underlying lexical processing. Consistent with the neuroimaging literature, lexical processing engages a distributed neural system involving both anterior and posterior structures. The results of Experiment 1 suggest that both Broca's aphasics with lesions including frontal structures and Wernicke's aphasics with lesions including temporal structures can map sound structure onto the lexicon and map a lexical entry to its lexical-semantic network. Like normal subjects, both groups of aphasic patients are more likely to fixate on an object semantically related to the target (e.g., *hammer* → *nail*) than on an unrelated object. However, the results of Experiments 2 and 3 suggest that both Broca's and Wernicke's aphasics have deficits in the dynamics of lexical activation. Both groups showed pathological patterns of performance under conditions of lexical onset competition; importantly, the patterns of deficit differ, suggesting that the functional role and the computational properties of these anterior and posterior areas differ.

In Experiment 2, unlike normal subjects, Broca's aphasics failed to fixate significantly more on an object

that shared its onset with the target (e.g., *hammock* → *hammer*) than on an unrelated object, and in Experiment 3, unlike normal subjects, they failed to fixate more on an object semantically related to a word that shared its onset with the target (e.g., *hammock* → *nail*). In contrast, Wernicke's aphasics showed increased sensitivity to onset competitors compared to age-matched controls. In Experiment 2, they showed increased looks to onset competitors compared to normal subjects and in Experiment 3, compared to normal subjects, they showed a trend toward increased looks to objects semantically related to onset competitors. Thus, although both groups showed deficits in lexical access under conditions of onset competition, patients with anterior lesions showed reduced sensitivity to onset competitors and patients with posterior lesions showed increased sensitivity to onset competitors. Taken together, these results suggest that although their roles differ, both anterior and posterior areas are necessary for normal lexical activation.

Because Broca's aphasics exhibited reduced rather than increased sensitivity to onset competitors, it appears that the functional deficit of these patients cannot be completely attributed to increased difficulty in selecting among competing alternatives because a selection deficit alone would predict more, rather than less, sensitivity to competition from onset competitors. The pattern of results for Broca's aphasics in the current series of studies is, however, compatible with the reduced activation hypothesis. Onset competitors (as shown in Experiment 2), and therefore, semantic onset competitors (as shown in Experiment 3), failed to become active, resulting in a failure to fixate on the competitors. The results of Experiments 2 and 3 are not consistent with the delayed activation hypothesis. Broca's aphasics did not show a competitor effect with a later than normal rise time; nor did they show competitor effects which emerged but were delayed in time.

With respect to the Wernicke's aphasics, the large competitor effects that were shown by these patients in Experiments 2 (onset competitors) and 3 (semantic onset competitors) are equally compatible with both the delayed deactivation and increased activation hypotheses. With increased lexical activation (or a failure to inhibit lexical competitors), onset competitors would be more active than normal, resulting in a larger competitor effect. With delayed deactivation, a failure to deactivate lexical entries over time would result in abnormally long-lasting competition, giving rise to increased and longer-lasting fixations on the competitor.

What is it about the computational properties of the anterior and posterior areas that would give rise to these different patterns? A recent connectionist computational model of word production (Gordon & Dell, 2003) provides a potential explanation. In particular, Gordon and Dell propose that lexical access and sentence production requires the convergence of activation from

two sources of information—conceptual semantic and syntactic-sequential, resulting in a continuum of dependence between these information sources. Applying a learning algorithm that takes into account these information sources, they modeled normal sentence production and lexical retrieval and then explored the effects of lesioning the model at these two levels of representation. Under normal circumstances, syntactic-sequential sources of information tend to increase overall lexical activation in the system because the semantic-conceptual representation of the entire sentence is activated. In contrast, semantic-conceptual representations tend to decrease overall lexical activation because only semantically associated lexical representations are activated. Syntactic lesions of the model (the hypothesized underlying deficit of Broca’s aphasics) resulted in lowered activation levels overall and semantic lesions of the model (the hypothesized deficit of anomic and potentially Wernicke’s aphasics as well) resulted in higher activation levels overall. The consequence of this is a dissociation in lexical access processes with an underactivation of the lexical system in Broca’s aphasics and an overactivation of the system in Wernicke’s aphasia (cf. McNellis & Blumstein, 2001).

If, indeed, Broca’s aphasics have reduced activation of lexical candidates and Wernicke’s aphasics have increased lexical activation, then why did both groups show normal semantic relatedness effects in Experiment 1? Recent neuroimaging results investigating the neural substrates of semantic priming in a lexical decision task using event-related functional magnetic resonance imaging have shown activation in both anterior and posterior regions as a function of semantic relatedness, with greater activation in the left IFG (Kotz, Cappa, von Cramon, & Friederici, 2002), left MFG (Rissman, Eliassen, & Blumstein, 2003), anterior medial-temporal cortex (Rossell, Price, & Nobre, 2003), and STG (Rissman et al., 2003) for semantically unrelated word pairs than for semantically related

word pairs. Because both anterior and posterior areas appear to be involved, it is possible that neither region is necessary or sufficient to result in abnormal performance if lesioned. Hence, one would expect to see normal semantic relatedness effects in patients with lesions involving either the IFG or the STG. However, it is also possible that because the relationship between the semantically related words was close and the task demands were easy, deficits may simply not have emerged under these conditions. In fact, behavioral studies have shown normal semantic priming for both Broca’s and Wernicke’s aphasics in lexical decision tasks (Milberg et al., 1988; Blumstein et al., 1982). Deficits have emerged for Broca’s aphasics only under conditions of lexical competition (Janse, 2006; Utman et al., 2001), when the prime–target pairs were presented in lists (Prather et al., 1992, 1997, but cf. Milberg & Blumstein, 1981) or at long interstimulus intervals (Hagoort, 1993).

Finally, the results of this series of experiments also have more general implications for current models of lexical processing. The findings for the young and old normal control subjects are consistent with models that allow for cascading activation, where the activation at one level of processing influences the activation of processing at another level further along the processing stream. As shown in Experiment 3, the presentation of the lexical candidate *hammock* drew more fixations to *nail*, the semantic associate of the lexical competitor *hammer* (replicating Yee & Sedivy, 2006). Thus, the activation of a lexical candidate not only influences the activation of a lexical competitor but also the activation of the conceptual representation of that lexical competitor (Allopenna et al., 1998; Gaskell & Marslen-Wilson, 1997; Zwitserlood, 1989; Dell, 1986). These results challenge discrete feedforward models because, in their view, a processing stage generates a single representation on the basis of its input and this single output representation is then transmitted to subsequent processing stages (Levelt, 1989).

## APPENDIX

<i>Experiment</i>	<i>Target</i>	<i>Related</i>	<i>Related’s Control 1</i>	<i>Related’s Control 2</i>
SEMANTIC	BAT	RACKET	PEAR	COIL
SEMANTIC	BATTERY	PLUG	MAP	GUITAR
SEMANTIC	MUFFIN	DONUT	DICE	CALCULATOR
SEMANTIC	PIANO	TRUMPET	LAMP	DUCK
SEMANTIC	PIE	ICE CREAM	MONKEY	CLOVER
SEMANTIC	SAW	AXE	VEST	WOK
SEMANTIC	SCISSORS	KNIFE	COAT	BREAD
SEMANTIC	TIE	JACKET	COW	GHOST
SEMANTIC	WALLET	PURSE	DRUM	ROPE

**APPENDIX** (continued)

<i>Experiment</i>	<i>Target</i>	<i>Related</i>	<i>Related's Control 1</i>	<i>Related's Control 2</i>
SEMANTIC	ROBE	SLIPPERS	LANTERN	WREATH
SEMANTIC	TEEPÉE	IGLOO	PAPERCLIP	DONKEY
SEMANTIC	CHERRY	BANANA	STAMP	VASE

<i>Experiment</i>	<i>Target</i>	<i>Competitor</i>	<i>Competitor's Control 1</i>	<i>Competitor's Control 2</i>
ONSET	ACORN	APRON	STOOL	DINOSAUR
ONSET	BANDAID	BANJO	SUNGLASSES	ASPARAGUS
ONSET	BUTTER	BUTTON	PIPE	FOX
ONSET	CAMEL	CAMERA	LEAF	FLAG
ONSET	PENNY	PENCIL	SKIRT	REFRIGERATOR
ONSET	PILLOW	PILLAR	LOBSTER	CIGAR
ONSET	PUPPY	PUPPET	AMBULANCE	KEYBOARD
ONSET	BAGEL	BABY	TRUCK	TREE
ONSET	BASKET	BATHTUB	SWAN	TOOTHBRUSH
ONSET	MUSTARD	MUSHROOM	FEATHER	CANE
ONSET	SODA	SOFA	UMBRELLA	DRILL
ONSET	TUBA	TULIP	OWL	OLIVES

<i>Experiment</i>	<i>Target</i>	<i>Competitor</i>	<i>Competitor's Control 1</i>	<i>Competitor's Control 2</i>
SEM ONSET	CATERPILLAR	MOUSE via <i>cat</i>	PUMP	PRINTER
SEM ONSET	GRAPEFRUIT	WINE via <i>grapes</i>	BELL	TRAIN
SEM ONSET	LOGS	KEY via <i>lock</i>	APPLE	DRESS
SEM ONSET	MATTRESS	LIGHTER via <i>match</i>	BONNET	PALETTE
SEM ONSET	PANTHER	SHIRT via <i>pants</i>	BRICKS	SALT
SEM ONSET	SOCCERBALL	SHOE via <i>sock</i>	BEER	BIRD
SEM ONSET	TELEPHONE	BINOCULARS via <i>telescope</i>	PUMPKIN	CROW
SEM ONSET	WINDMILL	DOOR via <i>window</i>	GUN	DOG
SEM ONSET	CANDY	LIGHTBULB via <i>candle</i>	OCTOPUS	HANGER
SEM ONSET	CARDS	BIKE via <i>car</i>	SCALE	BEAR
SEM ONSET	HAMMOCK	NAIL via <i>hammer</i>	ELEPHANT	BEE
SEM ONSET	TABLE	GLUE via <i>tape</i>	CELERY	PERFUME

**Acknowledgments**

Portions of this research were presented at the 42nd Annual Meeting of the Academy of Aphasia, Chicago, IL, October 17–19, 2004.

This research was supported in part by NIH grants NIDCD00314 to Brown University, NIDCD0081 to the Boston University School of Medicine, and a Jacob K. Javits Fellowship awarded to Eiling Yee. This material is the result of work supported with resources and the use of facilities at the Department of Veterans Affairs Medical Centers in Boston, MA and

Providence, RI. The views expressed in this article are those of the authors and do not necessarily reflect the position or policy of the Department of Veterans Affairs. We thank Paul Allopenna and Katherine White for their helpful comments and contributions to this project. We also thank Kathy Kurowski and Cara Misiurski for assistance with patient testing, and Andrew Duchon for assistance with data processing.

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## Notes

1. In the eye-tracking literature, the *target* refers to the one and only word uttered, whereas in the semantic priming literature, the *target* follows the prime and is used to gauge the prime's activation.
2. Prior studies in our lab (Yee & Sedivy, 2006; Yee, unpublished data) established that when they were unrelated to the target, the pictures that served as competitors in competitor trials did not draw more fixations than other unrelated pictures.
3. The log of each word's frequency count in the Brown corpus (Francis & Kucera, 1982), the Wall Street Journal corpus (Mitchell, Santorini, & Marcinkiewicz, 1993), and the SWITCHBOARD corpus (Godfrey, Holliman, & McDaniel, 1992), was obtained. For each word the three counts were summed; these sums were matched.
4. Distractor trials were not necessary in Experiment 3 because the semantically mediated onset competitor relationship is so subtle that subjects never report noticing it.
5. We verified this observation by comparing the average points at which participants in each group became more likely to fixate on the target than on unrelated objects (the *target's divergence point*) for 15 filler trials in which none of the other objects in the display were related semantically or phonologically to the target. Filler, rather than experimental, trials were examined to prevent any competitor effects from interacting with the target's divergence point. Submitting the average target's divergence point for each of the four groups to an omnibus ANOVA indicated that by subjects there was a significant effect of group [ $F(3,1) = 5.1, p < .01$ ]. Post hoc tests showed that the two aphasic groups did not differ from each other ( $p = .19$ ) and that the two control groups did not differ from each other ( $p = .50$ ). However, Broca's aphasics were delayed compared to age-matched controls (approaching significance at  $p = .08$ ) and young controls ( $p = .03$ ). Wernicke's aphasics were also delayed compared to age-matched controls ( $p < .01$ ) and young controls ( $p < .01$ ).

## REFERENCES

- Allopenna, P. D., Magnuson, J., & Tanenhaus, M. K. (1998). Tracking the time course of spoken word recognition using eye movements: Evidence of continuous mapping models. *Journal of Memory and Language*, 38, 419–439.
- Bates, T. C., & Oliveiro, L. (2003). PsyScript: A Macintosh application for scripting experiments. *Behavior Research Methods, Instruments, & Computers*, 35, 565–576.
- Binder, J., & Price, C. J. (2001). Functional neuroimaging of language. In R. Cabeza & A. Kingstone (Eds.), *Handbook of functional neuroimaging of cognition*. Cambridge: MIT Press.
- Blumstein, S., Milberg, W., & Shrier, R. (1982). Semantic processing in aphasia: Evidence from an auditory lexical decision task. *Brain and Language*, 17, 301–315.
- Blumstein, S. E., & Milberg, W. P. (2000). Language deficits in Broca's and Wernicke's aphasia: A singular impairment. In Y. Grodzinsky, L. Shapiro, & D. Swinney (Eds.), *Language and the brain: Representation and processing*. New York: Academic Press.
- Bookheimer, S. (2002). Functional MRI of language: New approaches to understanding the cortical organization of semantic processing. *Annual Review of Neuroscience*, 25, 151–188.
- Dahan, D., Magnuson, J. S., & Tanenhaus, M. K. (2001). Time course of frequency effects in spoken-word recognition: Evidence from eye movements. *Cognitive Psychology*, 42, 317–367.
- Dell, G. (1986). A spreading activation theory of retrieval in language production. *Psychological Review*, 93, 283–321.
- Demonet, J. F., Chollet, F., Ramsay, S., Cardebat, D., Nespoulous, J. L., Wise, R., et al. (1992). The anatomy of phonological and semantic processing in normal subjects. *Brain*, 115, 1753–1768.
- Francis, W. N., & Kucera, H. (1982). *Frequency analysis of English usage: Lexicon and grammar*. Boston: Houghton, Mifflin Co.
- Gabrieli, J. D. E., Poldrack, R. A., & Desmond, J. E. (1998). The role of the left prefrontal cortex in language and memory. *Proceedings of the National Academy of Sciences, U.S.A.*, 95, 906–913.
- Gaskell, M. G., & Marslen-Wilson, W. D. (1997). Integrating form and meaning: A distributed model of speech perception. *Language and Cognitive Processes*, 12, 631–656.
- Godfrey, J. J., Holliman, E. C., & McDaniel, J. (1992). SWITCHBOARD: Telephone speech corpus for research and development. *IEEE ICASSP*, 517–520.
- Goodglass, H., & Kaplan, E. (1972). *The assessment of aphasia and related disorders*. Philadelphia: Lea and Febiger.
- Gordon, J. K., & Dell, G. S. (2003). Learning to divide the labor: An account of deficits in light and heavy verb production. *Cognitive Science*, 27, 1–40.
- Hagoort, P. (1993). Impairments of lexical-semantic processing in aphasia: Evidence from the processing of lexical ambiguities. *Brain and Language*, 45, 189–232.
- Hagoort, P. (1997). Semantic priming in Broca's aphasics at a short SOA: No support for an automatic access deficit. *Brain and Language*, 56, 287–300.
- Huettig, F., & Altmann, G. T. M. (2005). Word meaning and the control of eye fixation: Semantic competitor effects and the visual world paradigm. *Cognition*, 96, B23–B32.
- Janse, E. (2006). Lexical competition effects in aphasia: Deactivation of lexical candidates in spoken word processing. *Brain and Language*, 97, 1–11.
- Kotz, S. A., Cappa, S. F., von Cramon, D. Y., & Friederici, A. D. (2002). Modulation of the lexical-semantic network by auditory semantic priming: An event-related functional MRI study. *Neuroimage*, 17, 1761–1772.
- Levelt, W. J. M. (1989). *Speaking: From intention to articulation*. Cambridge: MIT Press.
- Luce, P. A., Pisoni, D. B., & Goldinger, S. D. (1990). Similarity neighborhoods of spoken words. In G. T. M. Altmann (Ed.), *Cognitive models of speech processing: Psycholinguistic and computational perspectives* (pp. 122–147). Cambridge: MIT Press.
- Masson, M. E. J. (1995). A distributed model of semantic priming. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 21, 3–23.
- McClelland, J. L., & Elman, J. (1986). The TRACE model of speech perception. *Cognitive Psychology*, 18, 1–86.
- McNellis, M. G., & Blumstein, S. E. (2001). Self-organizing dynamics of lexical access in normals and aphasics. *Journal of Cognitive Neuroscience*, 13, 151–170.
- Milberg, W., Blumstein, S., & Dworetzky, B. (1988). Phonological processing and lexical access in aphasia. *Brain and Language*, 34, 279–293.
- Milberg, W., & Blumstein, S. E. (1981). Lexical decision and aphasia: Evidence for semantic processing. *Brain and Language*, 14, 371–385.
- Milberg, W., Blumstein, S. E., & Dworetzky, B. (1987). Processing lexical ambiguities in aphasia. *Brain and Language*, 31, 138–150.
- Misiurski, C., Blumstein, S. E., Rissman, J., & Berman, D. (2005). The role of lexical competition and

- acoustic–phonetic structure in lexical processing: Evidence from normal subjects and aphasic patients. *Brain and Language*, *93*, 64–78.
- Mitchell, M. P., Santorini, B., & Marcinkiewicz, M. A. (1993). Building a large annotated corpus of English: The Penn Treebank. *Computational Linguistics*, *19*, 313–330.
- Norris, D. (1994). Shortlist: A connectionist model of continuous speech recognition. *Cognition*, *52*, 189–234.
- Ostrin, R. K., & Tyler, L. K. (1993). Automatic access to lexical semantics in aphasia: Evidence from semantic and associative priming. *Brain and Language*, *45*, 147–159.
- Petersen, S. E., Fox, P. T., Posner, M. I., Mintun, M., & Raichle, M. E. (1988). Positron emission tomographic studies of the cortical anatomy of single-word processing. *Nature*, *331*, 585–589.
- Poldrack, R. A., Wagner, A. D., Prull, M. W., Desmond, J. E., Glover, G. H., & Gabrieli, J. D. E. (1999). Functional specialization for semantic and phonological processing in the left inferior prefrontal cortex. *Neuroimage*, *10*, 15–35.
- Prather, P., Zurif, E. B., Stern, C., & Rosen, J. T. (1992). Slowed lexical access in nonfluent aphasia: A case study. *Brain and Language*, *43*, 336–348.
- Prather, P. A., Love, T., Finkel, L., & Zurif, E. B. (1994). Effects of slowed processing on lexical activation: Automaticity without encapsulation. *Brain and Language*, Academy of Aphasia Conference, 326–329.
- Prather, P. A., Zurif, E., Love, T., & Brownell, H. (1997). Speed of lexical activation in nonfluent Broca's aphasia and fluent Wernicke's aphasia. *Brain and Language*, *59*, 391–411.
- Price, C. J., Moore, C. J., Humphreys, G. W., & Wise, R. J. S. (1997). Segregating semantic from phonological processes during reading. *Journal of Cognitive Neuroscience*, *9*, 727–733.
- Price, C. J., Mummery, C. J., Moore, C. J., Frackowiak, R. S. J., & Friston, K. J. (1999). Delineating necessary and sufficient neural systems with functional imaging studies of neuropsychological patients. *Journal of Cognitive Neuroscience*, *11*, 371–382.
- Rissman, J., Eliassen, J. E., & Blumstein, S. E. (2003). An event-related fMRI investigation of implicit semantic priming. *Journal of Cognitive Neuroscience*, *15*, 1160–1175.
- Rorden, C., & Karnath, H. (2004). Using human brain lesions to infer function: A relic from a past era in the fMRI age? *Nature Reviews Neuroscience*, *5*, 813–819.
- Rossell, S. L., Price, C. J., & Nobre, A. C. (2003). The anatomy and time course of semantic priming investigated by fMRI and ERPs. *Neuropsychologia*, *41*, 550–564.
- Roskies, A. L., Fiez, J. A., Balota, D. A., Raichle, M. E., & Petersen, S. E. (2001). Task-dependent modulation of regions in the left inferior frontal cortex during semantic processing. *Journal of Cognitive Neuroscience*, *13*, 829–843.
- Slowiaczek, L. M., & Hamburger, M. (1992). Prelexical facilitation and lexical interference in auditory word recognition. *Journal of Experimental Psychology: Learning Memory, and Cognition*, *18*, 1239–1250.
- Swinney, D., Prather, P., & Love, T. (2000). The time-course of lexical access and the role of context: Converging evidence from normal and aphasic processing. In Y. Grodzinsky, L. P. Shapiro, & D. Swinney (Eds.), *Language and the brain: Representation and processing* (pp. 273–292). New York: Academic Press.
- Swinney, D., Zurif, E. B., & Nicol, J. (1989). The effects of focal brain damage on sentence processing: An examination of the neurobiological organization of a mental module. *Journal of Cognitive Neuroscience*, *1*, 25–37.
- Tanenhaus, M. K., Magnuson, J. S., Dahan, D., & Chambers, C. (2000). Eye movements and lexical access in spoken-language comprehension: Evaluating a linking hypothesis between fixations and linguistic processing. *Journal of Psycholinguistic Research*, *29*, 557–580.
- Thompson-Schill, S. L., D'Esposito, M., Aguirre, G. K., & Farah, M. J. (1997). Role of the left inferior prefrontal cortex in retrieval of semantic knowledge: A reevaluation. *Proceedings of the National Academy of Sciences, U.S.A.*, *94*, 14792–14797.
- Thompson-Schill, S. L., D'Esposito, M., & Kan, I. P. (1999). Effects of repetition and competition on activation in left prefrontal cortex during word generation. *Neuron*, *23*, 513–522.
- Thompson-Schill, S. L., Swick, D., Farah, M. J., D'Esposito, M., Kan, I. P., & Knight, R. T. (1998). Verb generation in patients with focal frontal lesions: A neuropsychological test of neuroimaging findings. *Proceedings of the National Academy of Sciences, U.S.A.*, *95*, 15855–15860.
- Tyler, L. K., Ostrin, R. K., Cooke, M., & Moss, H. E. (1995). Automatic access of lexical information in Broca's aphasics: Against the automaticity hypothesis. *Brain and Language*, *48*, 131–162.
- Utman, J. A., Blumstein, S. E., & Sullivan, K. (2001). Mapping from sound to meaning: Reduced lexical activation in Broca's aphasics. *Brain and Language*, *79*, 444–472.
- Vandenberghe, R., Price, C., Wise, R., Josephs, O., & Frackowiak, R. S. J. (1996). Functional anatomy of a common semantic system for words and pictures. *Nature*, *383*, 254–256.
- Wagner, A. D., Pare-Blagoev, E. J., Clark, J., & Poldrack, R. A. (2001). Recovering meaning: Left prefrontal cortex guides controlled semantic retrieval. *Neuron*, *31*, 329–338.
- Wise, R. J. S., Chollet, F., Hadar, U., Friston, K., Hoffner, E., & Frackowiak, R. (1991). Distribution of cortical neural networks involved in word comprehension and word retrieval. *Brain*, *114*, 1803–1817.
- Yee, E. (2005). *The time course of lexical activation during spoken word recognition: Evidence for unimpaired and aphasic individuals*. Unpublished Doctoral Dissertation, Brown University.
- Yee, E., & Sedivy, J. (2006). Eye movements to pictures reveal transient semantic activation during spoken word recognition. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *32*, 1–14.
- Zwitserslood, P. (1989). The locus of the effects of sentential-semantic context in spoken-word processing. *Cognition*, *32*, 25–64.