

Word comprehension occurs with the instantaneous or automatic activation of concepts in a semantic network. This activation may continue with strategic or controlled processes for a second or two. The primed lexical decision task is one procedure that enables us to distinguish these automatic and controlled processes. The purpose of this study was to determine whether aphasia causes deficits in either of these levels of lexical-semantic activation.

The literature has been somewhat contradictory regarding whether aphasia interferes with controlled processing, automatic processing or both. Some researchers have reported that individuals with aphasia are deficient in automatic processing (e.g., Milberg, et al., 1987; Del Toro, 2000). Others have found no evidence to support impaired automatic processes (e.g., Hagoort, 1997). In priming tasks, controlled processing has been found to be spared (e.g., Bushell, 1996).

There are several approaches to the identification of automatic and controlled processes. Aphasiologists have focused on the manipulation of stimulus onset asynchrony (SOA) or the time between a prime and target in stimulus word pairs. Relatively short SOAs (≤ 250 ms) are used to measure automatic processing, while longer SOAs (> 250 ms) are used to measure controlled processing. Level of processing also is manipulated by the expectancy of the target. That is, controlled processing is more likely when expectations are high for a particular type of prime-target relationship. Based on Neely (1977), our study manipulated expectancy through instructions to participants about the nature of a relatively few categorical primes and their categorical exemplar targets.

Methods

Lexical-semantic processing was investigated through manipulation of stimulus relatedness, stimulus expectancy and stimulus onset asynchrony in a primed lexical decision task. Priming responses of ten individuals with mild to moderate aphasia were compared with responses of ten age-matched, non-brain damaged individuals from the same community (see Tables 1 and 2).

Participants were seen in a laboratory at the university or in a quiet room of their home. Experiments were conducted using Dell desktop and laptop computers with screen sizes from 16 to 17 inches. Each experiment was created using E-Prime by Psychology Software Tools, Inc (2002).

Six category superordinates (*BODY, BIRD, SPORT, BUILDING, ANIMAL* and *UTENSIL*) and 312 exemplars from four of those six categories were selected from Battig and Montague's (1969) norms. Twenty-four exemplars were used as practice stimuli and the remaining 288 exemplars were used as experimental stimuli.

The three prime conditions were related, unrelated and neutral. Each experimental set of stimuli consisted of 72 real word trials divided into 18 related prime-target trials and 18 unrelated prime-target trials. The selected neutral prime was the word *BLANK*. Neutral primes were paired with the same number of exemplars from the related and unrelated categories, thereby, generating 36 neutral prime-target trials (see Table 3).

In addition, each experimental set consisted of 72 word-nonword trials, which included 18 primes from the related condition and 18 primes from the unrelated condition. Neutral primes were paired with 36 nonword targets (see Table 4). Each

experimental set of stimuli contained an equal number of real word and nonword targets for a total of 144 trials in each experimental set. All participants saw the same target words and nonwords one time each. Target words and nonwords were not repeated within or between experimental sets to eliminate the risk of targets being primed by other identical targets (i.e., identity priming).

Within each experimental set, word pair trials were presented equally at long and short stimulus onset asynchronies of 2000 and 250 milliseconds. The interstimulus interval was consistent at 50 milliseconds for each trial. The presentation order of SOAs was randomized to discourage participants from developing a response strategy based on the rhythm of the experiment.

Before beginning the experiment, participants were told to expect specific category exemplars after a specified prime. For example, they were told to expect a type of a bird after the prime BIRD (i.e., related prime condition) and to expect a part of a building after the prime SPORT (i.e., unrelated prime condition). Therefore, both the related and unrelated primes were expected. Following written instructions on a computer screen, participants pressed the spacebar on the computer keyboard to initiate the experiment, which began with a row of asterisks alerting them to the screen. Next, they saw a prime from one of the specified categories, followed by a target word or nonword. Participants responded to each target with their left hand, by pressing either the “1” key to indicate a “yes” response for a real word or the “2” key to indicate a “no”.

Results/Discussion

In all conditions, the group with aphasia was significantly slower in responding than the control group (see Table 5). Taken by itself, this result may imply delayed processing for those with aphasia. However, in each condition, response accuracy was comparable between both groups (see Table 6). This result suggests that a motivation to be accurate may have contributed to the slower response times of the participants with aphasia.

The control group demonstrated automatic activation at the short SOA through significant overall priming and facilitation in the related condition (see Figures 1 and 5). At the long SOA, this group showed significant facilitation in both the related and unrelated conditions (see Figures 3, 9, and 11). This result suggests that the controlled process of expectancy influenced their responses. In each of the six prime condition comparisons, the control group results were consistent with the results of Neely (1977).

The group with aphasia performed in a similar priming pattern as the control group, which may suggest unimpaired automatic and controlled processing (see Figures 3, 4, 5, 6, 9, 10, 11 and 12). However, an exception to this pattern occurred in the unrelated condition at the short SOA. In this condition, the group with aphasia demonstrated no overall priming (i.e., compared with related condition) and significant facilitation (i.e., compared with the neutral condition), suggesting that expectancy influenced response times at the short SOA (see Figures 2 and 8, respectively).

High accuracy, slow responses and facilitation in the unrelated condition at the short SOA suggest that the group with aphasia was using postlexical strategies in making lexical decisions. They appeared to use the target to identify prime-target compatibility before making their lexical decision. In addition, overall priming was not observed at the short SOA, again, most likely because of postlexical processing. Therefore, previous

research that showed no overall priming effects with short SOAs may need to be reinterpreted whenever the participants with aphasia had long response times.

Strategy use at the short SOA does not imply that automatic activation was deficient or delayed. Instead, automatic processing abilities may have been masked by controlled processes. This notion also was suggested by Milberg, et al (1995) and Del Toro (2000); however, their perspective was that strategy masked automatic deficits rather than automatic abilities. The results from the group with aphasia are perceived as more compatible with Hagoort (1997), in that significant priming was observed at the short SOA. The results found here offer no evidence that automatic activation is impaired, delayed or spared.

Clinically, this study tells us something about aphasia outcomes. Like their age-matched peers, individuals with mild to moderate aphasia effectively are able to use strategies such as expectancy. However, they need additional time.

Table 1: Participant group comparison

	Aphasia	Controls
Mean age	70.9	60
Age range	64-83	40-78
Right handed pre-onset	9	10
Left handed pre-onset	1	-
Males	8	6
Females	2	4
Mean years of education	17	16.4
Range in years of education	12-22	10-22

Table 2: Aphasia profile of experimental group

	Mean	Range
Aphasia Quotient (AQ)	88.15	74.6 - 99.6
MTDD reading subtests	98.9	95 - 100
Boston Naming Test	54	31-59
Years post onset	3.4	1 - 8

Table 3: Prime condition examples with real word targets

Prime condition	Prime-target examples	Trials in each condition
Related	BIRD-robin	18
Unrelated	SPORT-door	18
Neutral	BLANK-sparrow	18
	BLANK-wall	18

Table 4: Prime condition examples with nonword targets

Prime condition	Prime-target examples	Trials in each condition
Related	BIRD-tugle	18
Unrelated	SPORT-saft	18
Neutral	BLANK-foap	36

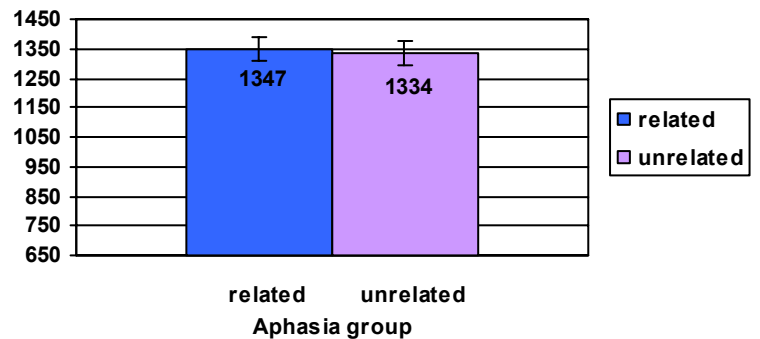
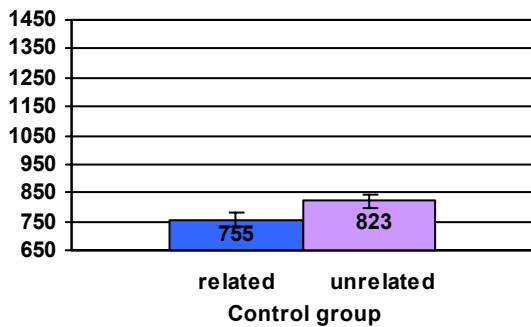
Table 5: Mean response times and ranges between aphasia group and control group

SOA	Condition	Aphasia		Control		Significance
		mean	range	mean	range	
250	Related	1347	805 - 2612	755	614 - 970	p = 0.007
	Unrelated	1334	867 - 2309	823	624 - 1144	p = 0.0102
	Neutral	1400	891 - 2547	827	677 - 1141	p = 0.008
2000	Related	1294	791 - 2399	756	613 - 960	p = 0.004
	Unrelated	1307	765 - 2366	757	589 - 931	p = 0.004
	Neutral	1387	857 - 2809	823	986 - 683	p = 0.012

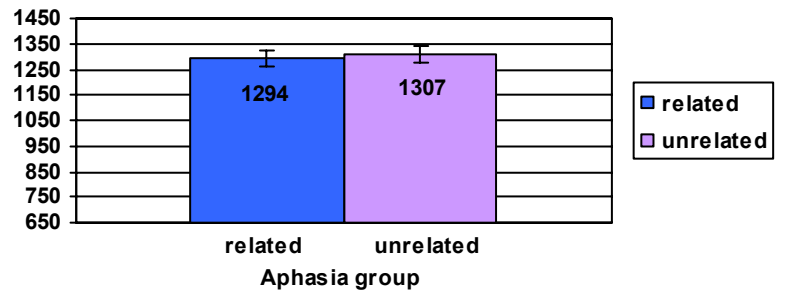
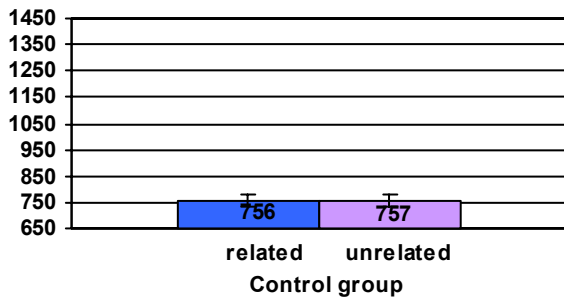
Table 6: Response accuracy (% accurate) for each group and real word target condition

	Related 250	Related 2000	Unrelated 250	Unrelated 2000	Neutral 250	Neutral 2000
Aphasia	96	95	97	97	96	95
Control	96	97	98	97	98	97

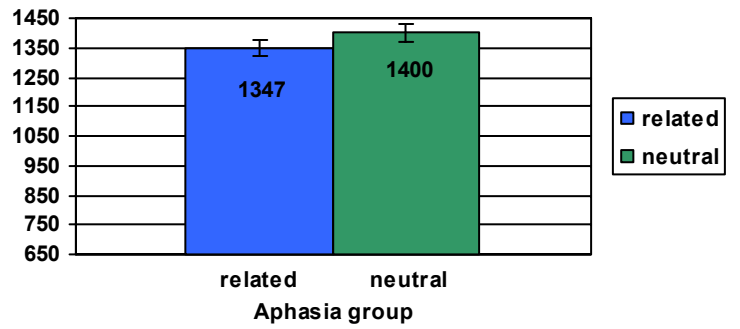
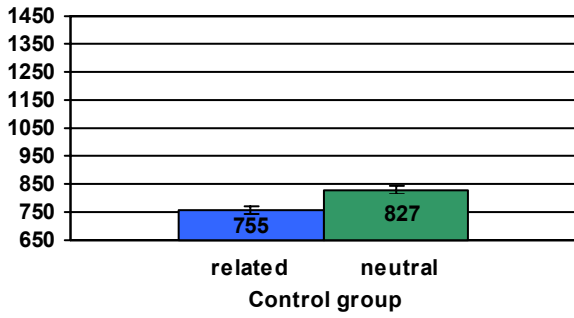
Figures 1 and 2: Control group and aphasia group response times at the 250 ms SOA



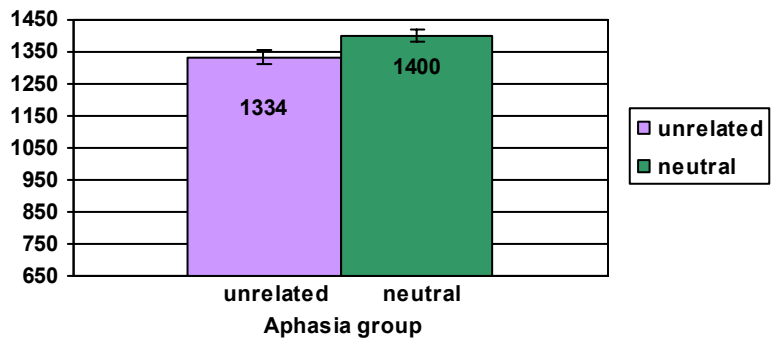
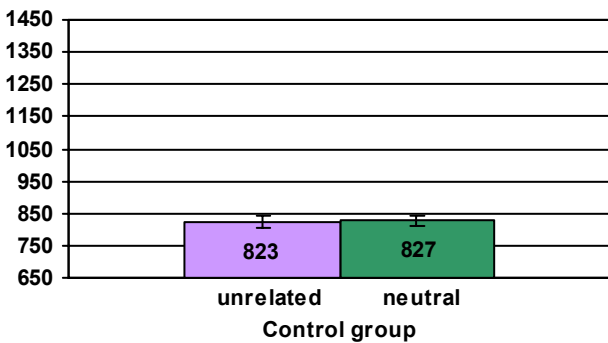
Figures 3 and 4: Control group and aphasia group response times at the 2000 ms SOA



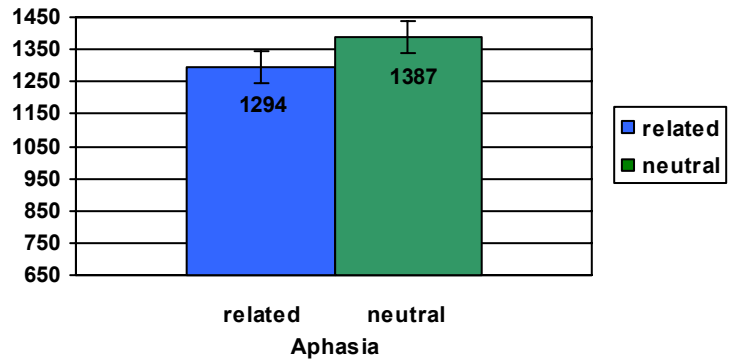
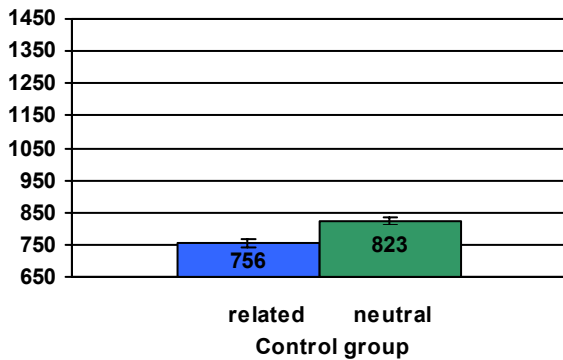
Figures 5 and 6: Control group and aphasia group response times at the 250 ms SOA



Figures 7 and 8: Control group and aphasia group response times at the 250 ms SOA



Figures 9 and 10: Control group and aphasia group response times at the 2000 ms SOA



Figures 11 and 12: Control group and aphasia group response times at the 2000 ms SOA.

