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Speed of Lexical Activation in Nonfluent Broca's Aphasia and Fluent Wernicke's Aphasia

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Rapid, automatic access to lexical/semantic knowledge is critical in supporting the tight temporal constraints of on-line sentence comprehension. Based on findings of ''abnormal'' lexical priming in nonfluent aphasics, the question of disrupted automatic lexical activation has been the focus of many recent efforts to understand their impaired sentence comprehension capabilities. The picture that emerges from this literature is, however, unclear. Nonfluent Broca's aphasic patients show *inconsistent,* not *absent,* lexical priming, and there is little consensus about the conditions

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under which they do and do not prime. The most parsimonious explanation for the variable findings from priming studies to date is that the primary disturbance in Broca's lexical activation has something to do with *speed* of activation. Broca's aphasic patients prime when sufficient time is allowed for activation to spread among associates. To examine this ''slowed activation'' hypothesis, the time course of lexical activation was examined using a list priming paradigm. Temporal delays between successive words ranged from 300 to 2100 msec. One nonfluent Broca's aphasic patient and one fluent Wernicke's patient were tested. Both patients displayed abnormal priming patterns, though of different sorts. In contrast to elderly subjects, who prime at relatively short interstimulus intervals (ISIs) beginning at 500 msec, the Broca's aphasic subject showed reliable automatic priming but only at a long ISI of 1500 msec. That is, this subject retained the ability to access lexical information automatically if allowed sufficient time to do so, a finding that may help explain disrupted comprehension of normally rapid conversational speech. The Wernicke's aphasic subject, in contrast, showed normally rapid initial activation but continued to show priming over an abnormally long range of delays, from 300 msec through 1100 msec. This protracted priming suggests failure to dampen activation and might explain the semantic confusion exhibited by fluent Wernicke's patients.

INTRODUCTION

Several recent studies have suggested a correlation between slowed lexical activation and syntactic processing limitations in nonfluent, agrammatic Broca's aphasic patients. However, while there is indirect evidence from at least one group study examining on-line sentence comprehension (Swinney, Zurif, & Nicol, 1989), direct evidence of slowed activation in Broca's aphasia is at present restricted to one case study (Prather, Zurif, Stern, & Rosen, 1992). In the research reported here, the focus was on confirming and extending that initial finding. Of interest was determining whether the aberrant rise time in activation proposed for Broca's aphasic patients is a function of aphasia disorders generally or rather is specific to Broca's aphasia. To that end, the present research examines the time course of activation in both a nonfluent and a fluent aphasic patient, with lesions in anterior vs. posterior left hemisphere, respectively.

Examination of the temporal parameters of lexical activation depends on priming techniques. Lexical priming reflects facilitation in lexical processing. In particular, associative/semantic priming refers to the finding that response times on a dependent measure, typically a lexical decision task (e.g., deciding whether a string of letters does or does not represent a real English word), are faster for a target word when that target is immediately preceded by a prime word meaningfully associated to it (e.g., ''TABLE–CHAIR'') than when it is preceded by an unrelated prime (e.g., ''DOCTOR–CHAIR''). Facilitation effects are taken to indicate that the related prime aids in the recognition of the target word. This interpretation of facilitation effects rests on the assumption of an automatic propagation of activation

within a network of mental representations, in this case spread from the mental representation of the prime word to the representation of its near associates, which include the target word (Collins & Loftus, 1975; Neely, 1991). On these assumptions, lexical priming effects reflect something about the organization and strength of lexical/semantic connections in the mental lexicon. In that regard, available evidence from studies of lexical priming has consistently shown apparently normal priming for Wernicke's but not for Broca's aphasic patients (Prather, 1994).

Fluent Wernicke's aphasic patients routinely show lexical priming for all conditions under which control subjects show priming. Given that these patients' priming results have consistently conformed to those of control subjects, most investigators have assumed that whatever underlies fluent aphasic patients' comprehension deficits, it is *not* a disruption in initial access to that organization; rather, these patients appear to suffer from imprecision somewhere else in semantic/sentence processing routines, presumably further along or ''higher up'' in processing. Given these prior findings, we began the present research considering Wernicke's aphasia as an appropriate control condition against which to compare rate of lexical activation in Broca's aphasia.

The evidence for Broca's patients is mixed with respect to speed of initial lexical activation. In contrast to the consistent associative and semantic priming effects obtained with Wernicke's aphasic patients, for Broca's aphasic patients, there have been two studies that reported failure to find automatic lexical priming (Milberg & Blumstein, 1981; Milberg, Blumstein, & Dworetzky, 1987), one study that reported automatic priming for primary but not secondary meanings of ambiguous words (Swinney et al., 1989), our study reporting delayed rather than absent automatic priming (Prather et al., 1992), and four studies reporting priming within a temporal framework assumed to reflect automatic activation (Blumstein, Milberg, & Shrier, 1982; Hagoort, 1988; Katz, 1988; Ostrin & Tyler, 1993). These discrepancies are quite understandable, however. Both of the studies that fail to find priming (early studies by Blumstein and Milberg) and studies that claim to find ''normally fast'' priming (including recent studies by Ostrin, Tyler, and Hagoort) rely on methodologies that do not, indeed cannot, distinguish automatic from strategic influences on priming effects. When, however, strategy driven effects are minimized to provide the clearest assessment of automatic lexical activation, Broca's patients show abnormal—and more specifically, abnormally slow—priming (Prather et al., 1992; Swinney et al., 1989).

The initial study suggesting a slower-than-normal rise time in lexical activation for Broca's aphasia demonstrated that, in contrast to neurologically intact control subjects and Wernicke's patients, Broca's patients fail to provide a fully elaborated lexical data base during sentence processing (Swinney et al., 1989). More specifically, young normals and elderly and fluent aphasic subjects routinely show activation of all meanings of ambiguous words im-

mediately after hearing them, even when those words are placed in a sentence context that strongly biases them toward one meaning (Swinney et al., 1989; Swinney, 1979). Broca's aphasic patients, on the other hand, show priming only for the most frequent meaning of ambiguous words, again, even when the sentence bias contradicts that meaning. Relevant to interpretation of that finding, prior research with college students has shown a temporal advantage for primary meanings; that is, while both meanings normally are activated quickly, the more frequent meaning of ambiguous words is primed more quickly than the secondary meaning (Simpson, 1984). Given this difference, Swinney and colleagues (Swinney et al., 1989) have speculated that the Broca's patients' abnormal priming pattern reflects a slower-than-normal course of lexical activation and a corresponding failure to activate meanings beyond the most frequent one within the experimental time frame: within the narrow temporal window imposed by the experiment, Broca's aphasic patients could activate the more closely linked primary meaning probe but not the more distant secondary meaning probe. By this argument, automatic spread of activation was present, but the time course of its propagation within the semantic network was slower for Broca's than for both young and elderly control subjects.

The hypothesis of protracted rise time for *automatic* lexical activation in Broca's aphasic patients was subsequently examined directly using a technique designed to restrict priming to automatic effects. This technique, the list priming paradigm (LPP), involves presentation of letter strings sequentially and continuously, without pauses that potentially would distinguish pairs of words as ''belonging together,'' which invites intentional or strategic effects (McNamara & Altarriba, 1988; Prather & Swinney, 1988; Shelton & Martin, 1992). As in traditional priming paradigms, as each letter string in the list is presented, the subject must decide as quickly as possible whether that string represents a real English word. In the LPP paradigm used in the present study, making that decision initiates a fixed interstimulus interval (ISI) followed immediately by presentation of the next letter string, and so on, continuously. Embedded within this continuous list are experimental word pairs, e.g., ''CABBAGE–LETTUCE''; those pairs are not distinguished in any way, however, within this continuous presentation of word and nonword strings (e.g., ''DOCTOR–SRNSE–LETVY–CABBAGE–LETTUCE–HECGVY–SIMPLE–LIMPET–BUTTER–AUTO–'', etc.). Using this paradigm, the delay between stimuli can be varied in order to determine the shortest and longest intervals at which automatic priming occurs.

The LPP differs from traditional pair priming paradigms (PPPs) in one critical way relevant to charting the time course of *automatic* lexical activation. As Shelton and Martin have demonstrated empirically, the LPP minimizes, if not eliminates, the contributions of strategic effects, both anticipation and postlexical review, that are known to contribute to priming effects when sufficient processing time is allowed using PPP paradigms (Groot,

Thomassen, & Hudson, 1982; Neely, 1991; Shelton & Martin, 1992). Importantly with respect to studying the time course of activation, the LPP also offers an internal check with respect to whether and when strategic effects influence performance. Normally strategic or intentional priming effects take longer to initiate than automatic effects, but once initiated, they increase over time; automatic effects, on the other hand, peak earlier and then diminish unless they are maintained by the surrounding context or task demands (Neely, 1977). Assuming that the LPP reflects automatic priming effects, and because it is designed *not* to provide an ''encouraging'' or ''maintaining'' context, any observed priming effects should diminish rather than increase over time. Results from our initial LPP study with normal control subjects are consistent with this expectation (Stern, Prather, Swinney, & Zurif, 1991). ISI was varied from 300 to 1100 msec; priming obtained at the 500-msec ISI and subsequently declined. This overall priming curve—initial increase and subsequent decline—contributes importantly in making the argument that automatic rather than strategic priming effects are being tapped using the LPP.

Using the LPP paradigm and case study methodology, Prather et al. (1992) found that their Broca's aphasic patient did not prime until an ISI of 1500 msec, a finding that is consistent with the hypothesis of a protracted rise time. Furthermore, activation diminished at a delay longer than 1500 msec, consistent with an inference that the priming obtained at the 1500-msec ISI reflected automatic, not strategic, priming.

While this initial finding is consistent with the hypothesis of slowed rise time in Broca's aphasia, a number of questions remain unanswered by this single case study. One important question is to what extent do the initial findings generalize to other Broca's aphasic patients in terms of slowed rise time (which is expected) and, specifically, in terms of *amount* of slowing (which might reasonably vary across subjects as a function of such individual differences as severity and extent of lesion)? Another important question has to do with Wernicke's aphasia. Will a Wernicke's patient still show normally fast priming even in the LPP paradigm that, unlike paired presentations, minimizes strategic influences?

The present research is designed to address these questions of generalizability and specificity by using the LPP to examine the time course of lexical activation in two additional case studies, one nonfluent agrammatic Broca's aphasic and one fluent Wernicke's aphasic patient. The present research also, thereby, allows us to assess functional localization. The relevant fact here is that Broca's and Wernicke's aphasia are distinguishable not only clinically, but also with respect to lesion site. To be sure, the brain area associated with Broca's aphasia now seems to have a greater extent than initially proposed: Broca's area in the foot of the third frontal convolution is hardly singularly important; adjacent and deeper areas also have been routinely implicated (Alexander, Naeser, & Palumbo, 1990; Dronkers, Shapiro, Red-

fern, & Knight, 1992; Naeser, Palumbo, Helm-Estabrooks, Stiassny-Eder, & Albert, 1989). Still, the fact remains that the modal lesion site for Broca's aphasia is distinguishable from that for Wernicke's aphasia, where the associated lesion is typically in the superior temporal gyrus (Wernicke's area) (Benson, 1985; Vignolo, 1988).

METHODS

Identical materials and procedures were used for both case studies described here. We begin with a detailed description of the general methodology and subsequently present the two case studies separately, indicating within each case description any details of the procedure that were unique to that case study.

Materials. Materials included 96 related word pairs (e.g., ''cabbage–lettuce'') and 96 matched control pairs in which the prime word from the related pair was replaced by a word of the same length and frequency (Francis & Kucera, 1982) but unrelated in meaning to the target word (e.g., ''capsule–lettuce''). Associates were selected on the basis of published norms (Jenkins, 1970; Keppel & Strand, 1970; Postman, 1970) plus data obtained by polling both college-age and elderly adults for the first associations to experimental words. The final set of associations represented the experimenters' selections, based on those norms and polled associations, of a set of strongly associated lexical items.

Two lists were constructed. Each list contained 48 related and 48 control sequences. If the related sequence for a particular target word was in the first list, then its matched control was in the second list, so that a given target word occurred just once in each list. The dual-list design was used to avoid repetition of words within a list. In addition to related and control pairs, each list included 108 nonexperimental filler words and 300 pronounceable nonwords for a total of 300 words and 300 nonwords per list. Finally, the word WORD appeared randomly on average once every 15 items in both lists. Including this repeated word was intended to distract the subject from looking for or noticing the occasional related pairs by providing a salient repeated event about which a subject was more likely to generate hypotheses, if so inclined. Thus, the repeated word was intended to focus the subject's attention and strategic processing efforts on patterns that were irrelevant to the related-control pair sequences of interest.

The two lists (I and II) were further divided into three segments. Each segment included 16 related-target sequences (32 words), 16 control–target sequences (32 word), 36 filler words, and 100 nonwords, totaling 200 stimuli per segment. Within each list, these three 200–word segments were presented with different ISIs (the specific ISIs used are detailed with each case study, below). Three different versions of each list (A, B, and C) were constructed, differing only in the order in which ISIs were presented. Order of ISIs was rotated according to a 3 $\times$ 3 Latin square design. Across the three versions of the two basic lists, then, each target word appeared once at each of three ISIs preceded by its related prime and once at each of three ISIs preceded by its control prime. By presenting subjects with all versions of each list, each subject saw each target word in all of its Relatedness (2) $\times$ ISI (3) conditions. Each subject saw one version of one list per session, so that target words were seen just once per session.

Apparatus. An IBM PC compatible portable computer (Compaq II) was used to control presentation of stimuli on a Panasonic video monitor with amber phosphor, and also to record reaction times on the lexical decision task. The experiment was run under control of RTLAB v9.0 software. This software, in conjunction with a software-accessible clock card (Metrabyte CTM05), has millisecond-level accuracy in timing stimulus onset, stimulus offset, and reaction times for the lexical decision task. RTLAB synchronizes stimulus presentation with monitor raster position so that timing of responses is accurate beginning from stimulus onset. Other apparatus consisted of a set of reaction time buttons used to indicate lexical decisions, with

the right button labeled ''yes'' (to indicate ''real word'') and the left button labeled ''no'' (to indicate ''nonword'').

Design. Each of the two subjects was presented with a total of six different ISIs. Data from the two subjects were analyzed separately. Each related and control word pair was presented once (to each subject) at each ISI. To accomplish this, both lists described above were presented once with an initial set of three ISIs (500, 1100, and 1500 msec), and a second time with a different set of three ISIs. The second set of ISIs varied for the two subjects, as discussed below. The overall design, then, was a 6 (ISI) $\times$ 2 (Related vs. Control) completely ''within-subjects'' repeated measures design, with target words serving as ''subjects,'' i.e., as the random effect in analyses of variance (ANOVAs).

CASE STUDY 1: FC (NONFLUENT BROCA'S APHASIA)

Subject. FC, a 58-year-old right-handed male college graduate, sustained a left CVA with complete occlusion of the left middle cerebral artery at age 39. Speech/language evaluations have consistently indicated moderate Broca's aphasia. Recent evaluation using the BDAE indicated nonfluent output with occasional paraphasias and agrammatisms. Comprehension is good for simple sentences and commands but declines for more complex sentences and ideational material. Screening measures in our lab, consistent with diagnosis of Broca's aphasia, indicated good comprehension of sentences with active constructions (95% correct) but weaker comprehension of sentences with passive constructions (70% correct). CT scan shows a very large left dorsolateral frontal lobe lesion involving almost all of the inferior and middle frontal gyri, including all of Broca's area and the white matter deep to Broca's area. The lesion continues superiorly and includes the lower two-thirds of the premotor, motor, and sensory cortex and the white matter deep to these areas. There is no lesion in the temporal and parietal lobules.

Materials. The three versions of the two lists described above were presented initially with ISIs of 500, 1100, and 1500 msec. Preliminary analyses indicated that priming occurs at 1500 msec but not earlier. Subsequently during an additional six visits, longer ISIs were examined: specifically, the three versions of the two experimental lists were presented with ISIs of 1500, 1800, and 2100 msec. Repeating the 1500-msec ISI provided continuity across halves of the study.

Procedure. On each of the 12 visits, FC was presented with a single list of 600 words; each session lasted approximately 45 to 60 min. FC had prior experience with lexical decision tasks and so minimal practice was required in getting comfortable with the procedure. Nonetheless, each session began with a short practice list to get him into set.

FC was seated at a comfortable distance in front of the video monitor and instructed that letter strings would be presented in the center of the monitor, one at a time but continuously. He was asked to decide, for each string of letters, if it was or was not a real English word, and to make that decision as quickly and accurately as possible. Decisions were recorded by depressing one of two response buttons, the left button marked ''yes'' for a real word and the right button marked ''no'' for a nonword, using the index and middle fingers, respectively, of the left hand to depress the buttons. FC rested one finger on each button throughout the experiment. Each session began with a short practice list, followed by the experimental list. Letter strings remained on the screen until a response was made, or for a maximum of 2000 msec; as soon as a response was made to one letter string, it was removed from the screen, the appropriate ISI initiated, and then the next letter string presented. During presentation of the list, there were breaks inserted after every 40th letter string; at that point, the list would ''pause'' and mean reaction time for the preceding 40 items was presented on the video monitor. Breaks served to reduce effects of fatigue and also provide feedback that helped motivate FC to maintain fast as well as accurate responding. Breaks were generally short, but were under subject control to be certain he felt sufficiently rested and focused throughout testing.

TABLE 1
Error and Screening Data for Broca's Patient FC

ISI (msec)	Priming condition	Equipment failure	Subject error	Outlying scores (±3 *SD*)
500[a]	Related	1	6	0
500[a]	Control	1	2	3
1100[a]	Related	0	6	1
1100[a]	Control	0	7	1
1500[a]	Related	0	3	0
1500[a]	Control	0	0	4
1500	Related	0	1	1
1500	Control	0	1	2
1800	Related	0	0	3
1800	Control	0	0	2
2100	Related	0	1	4
2100	Control	0	2	5

[a] ISIs tested in the first session.

RESULTS

Of primary interest in examining the data was the initial rise time in automatic lexical activation, i.e., the earliest ISI at which lexical priming obtained. To determine at which ISIs priming obtained, FC's reaction time data were prepared for analysis as follows. First, his responses were screened both for errors and for equipment failures. Second, outlying reaction times (RTs) defined as more than 3 *SD*s above or below his mean for that ISI were removed. The total numbers of FC's errors, equipment failures, and outlying scores for each condition are provided in Table 1.

Finally, because the purpose of the study was to chart the time course of priming effects as precisely as possible, we sought to maintain experimental control over the powerful effects of word length and frequency. Recall that each target word occurred once in each of the twelve conditions formed by crossing Relatedness (2) × ISI (6). By virtue of including the same words in all conditions, all cells in the design were originally balanced for length and frequency of words. This degree of control greatly facilitates the comparison of priming effects across ISIs since the priming effects are defined in terms of the exact same words. In order to preserve this important design feature, we performed a ''list-wise'' deletion of items by removing from the analysis target words for which there was a missing or removed datum for *either* the related or control condition for a target word at *any* of the six ISIs in the study. The final set of target words for which there was complete data for all conditions at all ISIs consisted of 55 words.

The priming effect for each different ISI was calculated by subtracting the related RT from its corresponding control RT and averaging across the 55 difference scores. The average priming effects along with their 95% con-

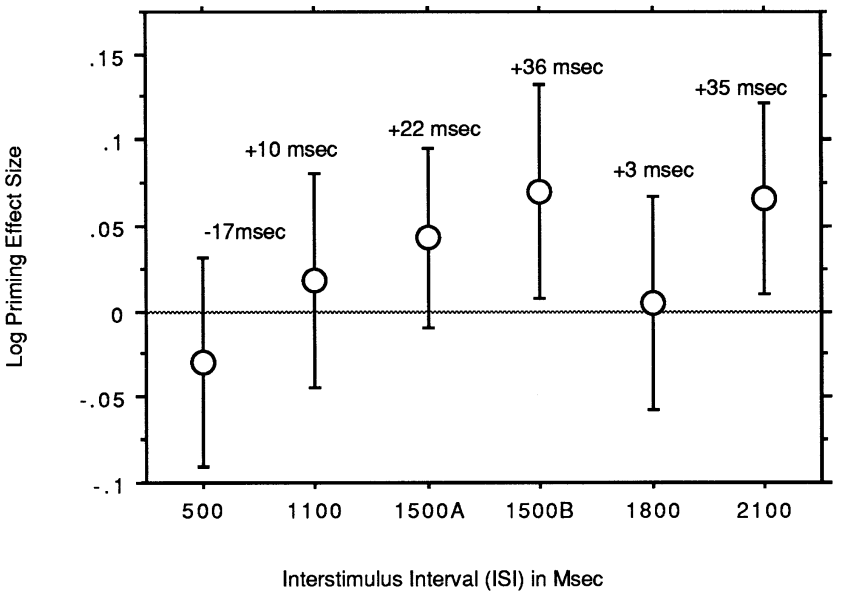

FIG. 1. Priming effects for Broca's patient FC. Error bars indicate 95% confidence intervals.

fidence intervals are displayed in Fig. 1. Where the 95% confidence interval does not overlap with 0 indicates that a priming effect is statistically significant at the $p < .05$ level. As can be seen in Fig. 1, FC shows reliable priming at the 2100-msec ISI and also at the 1500-msec ISI in the second session.

Table 2 provides additional results. First, the mean latencies are presented for each ISI. A Relatedness (two levels) × ISI (six levels) repeated measures ANOVA in which target word was treated as the random effect was performed on the data from the 55 target words. For each statistical test, we report the exact probability values, i.e., the probability of obtaining a result

TABLE 2
Mean Reaction Times and Planned Comparison Results for Broca's Patient FC

ISI (msec)	Mean RT	Related mean RT	Control mean RT	MSe	$F(1, 54)$	p
500[a]	571	581	562	8931.249	1.144	.290
1100[a]	516	510	523	7331.544	0.654	.422
1500[a,]*	510	498	523	5068.679	3.278	.076
1500*	522	503	540	7712.934	4.867	.032
1800	526	523	529	7952.720	0.155	.696
2100	542	522	562	6237.682	7.080	.010

[a] ISIs tested in the first session.

* Combined 1500-ISI priming effect: $F(1, 54) = 6.271$, $p = .0153$, MSe = 8300.878.

that extreme or more extreme, if the null hypothesis of no effect were true, rounded to three or four decimal places to provide information to support future metaanalytic studies of priming. Due to rounding, some nonzero probability values appear as ''.000'' or ''.0000,'' which indicates that the obtained probability is less than .0005 or .00005, respectively. Table 2 also presents results from the planned comparisons used to test for priming effects. Consistent with Fig. 1, Table 2 indicates reliable priming at one of the 1500-msec ISIs, a marginally reliable effect at the other 1500-msec ISI, and a reliable effect at the 2100-msec ISI.

We interpret these results as showing real priming for this patient at 1500 msec ISI. One of the effects was statistically robust, and even the statistically weaker effect was marginally reliable. Note that the 12-msec difference in the size of the priming effects at the two 1500-ISIs was not significant, $F(1, 54) = .469$, $p = .4963$, MSe = 4480.735. Most important is that when the data from the two equivalent conditions were combined to yield the best, most stable estimate of the priming effect at the 1500-msec ISI, patient FC showed a strong effect: $F(1, 54) = 6.271$, $p = .0153$, MSe = 8300.878.

Analysis also revealed other effects that are less relevant than the *a priori* tests for priming at each ISI. The data contained a strong main effect of ISI in that FC responded fastest at the 1500-msec ISI and slower at both shorter and longer ISIs, $F(5, 270) = 7.149$, MSe = 7708.341, $p = .0000$. A contrast showed no overall difference in reaction time between the first session (500-, 1100-, and 1500-msec ISIs) and the second session (1500-, 1800-, and 2100-msec ISIs), $F(1, 54) = 0.185$, $p = .6687$, MSe = 7291.178. There was an overall significant priming effect, $F(1, 54) = 5.667$, $p = .0208$, MSe = 8449.826, and a marginally reliable interaction of Relatedness × ISI, $F(5, 270) = 1.916$, $p = .0918$, MSe = 6956.997.

DISCUSSION

Consistent with the earlier case report on nonfluent patient LD (Prather et al., 1992), FC showed lexical priming at a much later ISI than the interval at which normal elderly subjects prime. This finding lends further support to the general hypothesis of slowed lexical activation in nonfluent aphasic patients.

While the general finding supports the original hypothesis, the finding of a decline in priming at 1800 msec but then rise again at 2100-msec ISIs requires further consideration. First, as described earlier, in order to claim that priming reflects *automatic* activation using the LPP, it is necessary to demonstrate a *decline* following a peak rise in activation. The decline is critical in indicating that priming effects are not strategic in origin, on the argument that strategic effects increase while automatic effects diminish with the simple passage of time. FC's priming curve clearly demonstrates delayed activation. It is also consistent with the claim that activation up to 1500 ISI

reflects largely automatic processing in that the priming effect diminishes relatively quickly and is virtually nonexistent at 1800 ISI. The lack of a priming effect at 1800 msec cannot be explained in any obvious way by unusual individual scores or odd characteristics of the distributions of scores for this ISI. In addition, the fall off of priming at 1800 msec ISI is completely consistent with our earlier findings (Prather et al., 1992). Accordingly, the lack of a priming effect at 1800 msec may be taken to signal the end of automatic, nonstrategic priming.

Granting this interpretation, however, the strong priming effect at 2100 msec still needs to be explained. Until the present study, the longest ISI that had been examined using the LPP had been 1800 msec (Prather et al., 1992); at that ISI, as noted above, findings were consistent with a decline in priming at long ISIs using the LPP. One possible, indeed likely, explanation for the ''reemergence'' of priming at 2100 msec ISI in the present study is that an ISI of that length—over 2 sec—essentially tests the limits of the LPP to inhibit or distract from strategic or conscious priming effects and consequently that the priming effect observed at 2100 msec ISI reflects nonautomatic strategic processing. Without further empirical data, that remains speculation, of course, but at present that interpretation is most consistent with the overall pattern of findings in the present and prior studies.

In sum, the present findings clearly replicate earlier findings showing priming at a much longer ISI for our Broca's aphasic subject than for elderly controls. Further, the same pattern obtains of a narrow peak of priming at 1500 msec, with no priming at either 1100 or 1800 msec. Very likely this peak reflects delayed automatic priming, although with some reservation pending further exploration of the reemergence of priming at 2100 msec.

CASE STUDY 2: JM (FLUENT WERNICKE'S APHASIA)

Subject. The subject, JM, was a 54-year-old right-handed male high school graduate 4 years post-CVA that involved occlusion of the left middle cerebral artery. JM's production was fluent with word finding difficulty and some difficulty repeating. Results of the BDAE indicated good phrase length, articulatory agility, and grammatical form, but impaired repetition and word finding difficulty and much poorer comprehension than production. Diagnosis based on language profile is fluent Wernicke's aphasia. Neuroradiological data are consistent with diagnosis of Wernicke's aphasia; based on a CT scan conducted in the same year as the CVA, there is a left temporoparietal lesion involving less than half of Wernicke's area and the white matter deep to it with superior extension into the supramarginal and angular gyrus areas and the white matter deep to these areas. A small extent of the lesion continues into the superior parietal lobule. There is no lesion present in the frontal lobe.

Materials. The three versions of the two lists described above were presented twice across 12 visits with JM. For the first 6 visits, as with subject FC, ISIs of 500, 1100, and 1500 msec were examined. Preliminary analyses indicated priming effects obtained at all three ISIs. Consequently, for the second half of the experiment, both shorter and longer ISIs were examined, specifically, 300-, 800-, and 1800-msec ISIs.

Procedure. The procedure was identical to that used with FC.

TABLE 3
Error and Screening Data for Wernicke's Patient JM

ISI (msec)	Priming condition	Equipment failure	Subject error	Outlying scores (± *SD*)
300	Related	8	0	0
300	Control	3	1	1
500[a]	Related	0	0	0
500[a]	Control	1	0	1
800	Related	0	0	1
800	Control	0	0	0
1100[a]	Related	0	0	6
1100[a]	Control	1	0	5
1500[a]	Related	0	0	1
1500[a]	Control	1	0	4
1800	Related	0	0	0
1800	Control	0	2	1

[a] ISIs tested in the first session.

RESULTS

JM's reaction time data were prepared for analysis exactly as described earlier for patient FC. First, JM's responses were screened both for his errors and also for equipment failures. Second, outlying reaction times (RTs) defined as more than 3 *SD*s above or below his mean for that ISI were removed. The total numbers of JM's errors, equipment failures, and outlying scores for each condition are provided in Table 3. In addition, a ''list-wise'' deletion of responses was performed by removing from the analysis target words for which there was a missing (or screened out) datum for *either* the related *or* control condition for a target word at *any* of the six ISIs in the study. The final set of target words for which there were complete data for all conditions at all ISIs consisted of 64 words.

The question of most interest is whether, as predicted, JM shows a ''normally rapid'' rise time in priming effects. The priming effect for each different ISI was calculated by subtracting the related RT from its corresponding control RT and then averaging across the 64 difference scores. The average priming effects along with their 95% confidence intervals are displayed in Fig. 2. Where the 95% confidence interval does not overlap with 0 indicates that a priming effect is statistically significant at the $p < .05$ level. As can be seen in Fig. 2, JM shows reliable priming at ISIs from 300 to 1100 msec and shows unreliable effects only at the longest ISIs of 1500 and 1800 msec.

Table 4 provides additional results. First, the mean latencies are presented for each ISI. A Relatedness (two levels) × ISI (six levels) repeated measures ANOVA in which target word was treated as the random effect was performed on the data from the 64 target words. Consistent with Fig. 2, Table

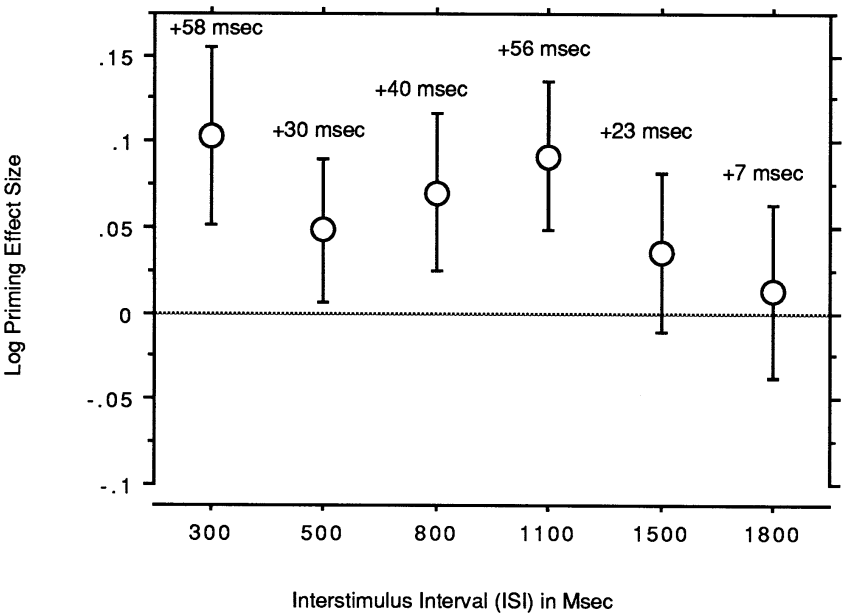

FIG. 2. Priming effects for Wernicke's patient JM. Error bars indicate 95% confidence intervals.

TABLE 4
Mean Reaction Times and Planned Comparison Results for Wernicke's Patient JM

ISI (msec)	Mean RT	Related mean RT	Control mean RT	MSe	$F(1, 63)$	p
300	572	542	603	6950.880	17.008	.000
500[a]	622	606	637	5497.532	5.599	.021
800	575	555	595	5820.474	8.879	.004
1100[a]	622	594	651	6101.554	17.453	.000
1500[a]	639	629	650	7715.617	1.840	.180
1800	575	572	578	7172.864	0.216	.644

[a] ISIs tested in the first session.

4 indicates reliable priming at 300-, 500-, 800-, and 1100-msec ISIs, and no reliable priming at the two longest ISIs of 1500 and 1800 msec.

Analysis also documented other less relevant effects. The data contained a strong main effect of ISI in that JM generally responded faster in the second session which included 300-, 800-, and 1800-msec ISIs, $F(5, 315) = 15.061$, MSe = 7677.082, $p = .0000$. A contrast confirmed that JM responded more quickly on average in the second session, $F(1, 63) = 69.865$, $p = .0000$, MSe = 7896.628. Although JM was generally faster in the second session,

he showed presence and absence of priming effects at comparable ISIs in both sessions, which suggests that no qualitative change occurred between testing sessions. There was an overall significant priming effect, $F(1, 63) = 23.556$, $p = .0000$, MSe = 10729.293, and an interaction of Relatedness × ISI, $F(5, 315) = 2.460$, $p = .0331$, MSe = 5705.926.

DISCUSSION

In one respect, JM's priming profile was consistent with predictions: priming obtained beginning at an ISI of 300 msec, which is at least as early as effects obtained with normal control subjects. In another respect, however, JM's profile was quite different from that of normal controls. For young, elderly, and nonfluent aphasic subjects, dampening, that is, loss of priming has been observed within 300 msec of obtaining priming. For JM, in contrast, priming persisted for at least 800 msec. In short, JM's decline in automatic activation is protracted relative to findings with both normal control and Broca's aphasic subjects.

GENERAL DISCUSSION

Nonfluent Aphasia

The findings reported here replicate earlier findings (Prather, Zurif, & Love, 1992) that a nonfluent Broca's aphasic patient activates lexical information in a slower-than-normal fashion. Milberg and colleagues have lately sought to account for this later-than-normal activation in terms of diminished strength rather than slowed activation (Milberg, Blumstein, Katz, Gershberg, & Brown, 1995). In light of current findings, we would argue that whether the fundamental problem is to be described in terms of a dimension of time or a dimension of strength—or alternatively some combination of the two—the hypothesis of delayed lexical activation represents a position with increasingly strong support.

There remains, however, some strong opposition to this position. In particular, Ostrin and Tyler (1993) and Hagoort (1993) argue that there is essentially no slowing to explain—that immediate lexical activation is normally rapid for Broca's aphasics. In support of this claim, they cite evidence that Broca's aphasics show priming at very short interstimulus intervals in their studies. There are, however, some fundamental methodological differences in their, relative to our, approach. As previewed in the Introduction, we believe that the difference between our findings and those of Tyler and Hagoort reflect methodological factors, in particular limitations in the ability of most pair priming paradigms to restrict priming to automatic effects. To evaluate our opposing views, it is necessary to consider those methodological concerns.

The primary goal in our investigations of the time course of lexical activa-

tion is to examine whether rapid, automatic priming is intact in Broca's aphasia. To that end, our research relies on a LPP in which words are presented continuously without breaks. The LPP was selected for two reasons: (1) As in sentence processing, it places demands on *rapid* lexical processing. The LPP is ''sentence like'' in that there are no breaks that set specific words apart as ''belonging together'' or that allow extra processing time between, for example, pairs of words. The intention is to examine lexical access in the context of continuous rather than discrete lexical processing, on the assumption that continuous processing is more demanding of—and therefore more likely to show disruptions in—rapid processing. (2) There is independent evidence that the LPP paradigm, in contrast to most pair paradigms, restricts effects to *automatic* priming. In a series of experiments, Shelton and Martin (1992) have examined empirically whether the LPP and/or PPPs reliably restrict priming to automatic effects. They did so using a paradigm in which proportion of related vs. unrelated word pairs is varied; as the proportion is higher, subjects are more likely to notice and therefore intentionally look for relations among words, that is, to show strategic priming effects. Shelton and Martin found that priming effects did vary as a function of proportion of relatedness when using a PPP paradigm but did *not* vary when using an LPP paradigm; essentially, where the PPP is sensitive to both automatic and strategic priming effects, the LPP is sensitive only to automatic effects. In our research using the LPP under conditions of different ISIs, we find an initial increase and, following ''peak'' priming, a decline in activation, consistent with an inference that strategic effects—which increase with time—do not contribute significantly to priming using the LPP (at least up to ISIs of 1800 msec). In contrast, when the same words are presented in a pair paradigm, priming continues to increase as ISIs increase, with no apparent decline (Prather & Swinney, 1988).

In short, PPPs of the type used by Ostrin and Tyler and Hagoort do *not* restrict priming to automatic effects, even when using very short ISIs. To be sure, there are conditions under which PPPs can restrict priming to automatic effects, but those conditions are quite specific; short ISIs alone are not sufficient. Neely (1977), for example, was able to disentangle the relative contributions of automatic vs. strategic priming by opposing those effects. Subjects were instructed to monitor for particular ''nonassociative'' relations that distracted from associative (automatic) priming effects. Under those conditions, Neely found that associative priming did but expectancy priming did not occur at very short (less than 300 msec) SOAs; by 750 msec, expectancy priming was increasing and associative priming was diminishing in strength. Neely's interpretations from this study focus on the relative time course of associative vs. expectancy priming under conditions where those effects are opposed. His findings at times have been interpreted as showing that only automatic effects obtain at short ISIs. That is not, however, what was demonstrated in his study, and, as noted above, empirical investigations contradict

the assumption that simply using short ISIs guarantees automaticity when using a pair paradigm. Rather, when using a PPP, the only way to ensure that priming is restricted to automatic effects is to include an internal check that allows separate examination of the contribution of automatic vs. strategic factors.

Neither Ostrin and Tyler nor Hagoort provide such an independent check in the paradigms they employed. While they used short interword delays, which arguably disallow anticipatory priming, the very long interpair/triplet delays used by both Hagoort and Ostrin and Tyler—6000-msec delays in both studies—allow more than enough time to invoke postlexical strategic priming effects (De Groot, 1984). Further, in selecting 200 to 250 msec as ISIs that are ''too short'' to allow strategic priming, both investigators cite evidence based on studies using visual presentations (e.g., Neely, 1977). There have been no independent studies to determine that temporal parameters established in visual priming studies translate directly to auditory priming studies; i.e., what is short enough visually may not be short enough auditorally. Of interest in that regard, the studies that obtain priming at short ISIs with Broca's aphasic subjects have all used auditory presentations.

It seems, then, that priming effects obtained using simple pair paradigms are more likely to reflect strategic priming or at least some combination of automatic and strategic effects. Finding that Broca's aphasic patients can prime when materials are presented auditorally under conditions of relatively low load (two or three words at a time followed by a long pause) is of interest, but is not evidence for either rapid or automatic priming. For those reasons, we think that findings using pair priming paradigms do not challenge the present or earlier findings of slowed lexical activation using LPP and cross-modal priming techniques.

In sum, the brain regions associated with Broca's aphasia seem to be crucially involved in sustaining normally rapid activation.

Fluent Aphasia

By contrast, the brain area implicated in Wernicke's aphasia does not seem to be necessary for speed of information activation. Rather, as our findings suggest, the functional commitment of this area has to do with deactivation of lexical information.

What might lead to a prolonged period of activation following posterior left hemisphere lesion? One likely possibility is failure in the normal balance of activation/deactivation processes. Of interest in that regard is recent research examining the relative contributions of the right and left hemisphere to priming effects (Beeman, 1993; Beeman, Friedman, Grafman, Perez, Diamond, & Lindsay, 1994). The left hemisphere is argued to contribute ''precision.'' When words are presented tachistoscopically to the left hemisphere, there is strong activation of a small semantic field highly related to the prime

word and a relatively rapid inhibition of ''irrelevant'' activation. In contrast, the right hemisphere appears to contribute breadth or ''coarse coding'' with weaker activation but of a broader semantic field and with more persistent activation. What the right hemisphere seems *not* to do is *inhibit* activation of any meanings (Burgess & Simpson, 1988; Nakagawa, 1991).

Speculatively, the delayed *de*activation observed with our Wernicke's aphasic subject might be accounted for by disruption of mechanisms involved in inhibition. Given the rapid decline in activation for our Broca's aphasic subjects (current study and Prather et al., 1992) and slowed decline for our Wernicke's aphasic subject, a reasonable inference is that anterior regions are involved with initiation and posterior regions with inhibition or ''focusing in'' with respect to lexical activation.

Alternatively, and not necessarily exclusive of the above hypothesis, it may be that damage to posterior temporal cortex disrupts the balance of left vs. right hemisphere contributions to lexical activation. With both hemispheres in good balance, there is both precise and coarse coding, arguably providing denotative and connotative meanings that, in context, allow flexible interpretation of intended word meanings. When one or the other hemisphere is damaged, however, that balance is likely to be lost and one or the other type of meaning activation to dominate. In support of that hypotheses, studies with right hemisphere damaged (RHD) patients show good ''literal'' or precise interpretation of words but difficulty with broader connotative intent (Brownell, Gardner, Prather, & Martino, 1995). There have been no time course studies examining rise and fall of lexical activation in RHD populations, but if this hypothesis is correct, then one would expect normal rise time and at least normally fast decline, given the left hemisphere's role in rapid inhibition of irrelevant word meanings. Damage to left posterior cortex, in contrast, would result in domination of right hemisphere coarse coding and prolonged activation—consistent with the present findings, at least with respect to prolonged activation.

Clearly, this account is speculative if for no other reason than the fact that we have data from just one Wernicke's aphasic patient to date. However, it is an appealing account in that the comprehension deficits characteristic of Wernicke's aphasics might be explained in terms of imprecise activation within the lexical/semantic network—coarse coding provides the ''sense of'' (or perhaps multiple senses of) word meanings, but disallows honing in on the exact word meaning needed for accurate comprehension of discourse. It is an account that needs detailed empirical consideration, but it does provide a framework for theoretical exploration of comprehension deficits at a lexical level that connects with a larger body of data and theory.

Connections to Sentence Level Processing

The finding of automatic but slow lexical activation for Broca's aphasia supports the more general claim that underlying the characteristic compre-

hension deficits in agrammatic Broca's aphasia is disruption of temporal relations. In particular, on-line sentence comprehension is tightly constrained temporally; if information is not available rapidly at the moment it is called for, then comprehension is compromised.

Dependency relations offer an example of a syntactic process that places demands on rapid (re)activation of lexical/semantic information, and that is uniquely disrupted in nonfluent aphasia (Grodzinsky, 1986, 1990). In examining linguistic characteristics of comprehension failures in nonfluent aphasic patients, Grodzinsky (1986, 1990) and others (Hickok, 1992; Mauner, Fromkin, & Cornell, 1993) have argued that these patients cannot represent traces and cannot, therefore, grammatically assign thematic roles to moved constituents. Faced with thematically unassigned phrases during on-line sentence comprehension, nonfluent patients rely on nongrammatical strategies, e.g., assigning the thematic role of agent to the first encountered noun phrase to try to make sense of sentences (Bever, 1970). Failure to assign thematic roles seems to be an ''on-line'' deficit; however, asked to make grammatical judgments without time constraints, nonfluent aphasics are able to detect grammatical deformations, including those that require an awareness of syntactic dependencies involving traces (Linebarger, Schwartz, & Saffran, 1983). Again, the contrast between on-line and off-line performance suggests a *processing* account. Several studies with both college age and elderly subjects have demonstrated, using cross-modal priming paradigms, that antecedents and traces are linked rapidly and automatically during the course of comprehension (Swinney & Fodor, 1989; Swinney & Osterhout, 1990). Assuming that comprehension requires not just reactivation, but *rapid* reactivation, of the antecedent in its trace position, nonfluent aphasic patients' slowed activation could explain their failed representation of traces during comprehension in the face of preserved ability to ''judge'' grammatically when there are no time constraints. Recent studies examining gap-filling on-line provide support for that hypothesis (Zurif, Swinney, Prather, & Love, 1994).

CONCLUDING REMARKS

In summary, results from the present study demonstrate abnormal patterns of lexical activation for both nonfluent and fluent aphasic subjects, but in quite different ways. The nonfluent aphasic subject showed a slow rise time in automatic activation but normally rapid decline once activation was achieved. This delayed rise time is consistent with findings of only partially successful activation during real-time sentence processing but preserved activation and comprehension when sufficient processing time is allowed. The fluent aphasic subject, on the other hand, showed at least normally rapid lexical activation, but a delay in dampening of activation. The resulting lexical activation may be broader and may reflect a ''sense'' but not a precise understanding of word meaning. Consequently, while much information is

available quickly, it is not the precise information needed, which results in generally disrupted comprehension.

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