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# Verbal memory function in mild aphasia

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**Article abstract**—Verbal material may be processed by semantic and phonologic systems. Damage to these language systems may also impair memory. We classified 16 mildly aphasic patients according to phonologic and lexicosemantic abilities, tested them on a variety of short- and long-term memory measures, and correlated behavioral deficits with lesion location. Aphasia impaired both short- and long-term memory. Phonologic impairment affected only digit span performance. Lexicosemantic deficits impaired self-organized encoding of word lists. Memory impairment was not associated with specific lesion locations. Persistent verbal-memory impairments accompanying even mild residual aphasia may be responsible for much of the difficulty mildly aphasic patients experience returning to vocational, academic, and social life. Co-occurrence of these deficits probably reflects their underlying dependence on similar processing systems.

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Patients with aphasia often complain of memory impairment, and their families often confirm that the patients have trouble recalling previously known names and events and learning new information. Many instances of "memory" problems in these patients are a reflection of their aphasia, word-finding difficulties, poor comprehension of a request, and so on, rather than actual amnesia for events. Even though most brain lesions that produce aphasia are located outside limbic circuits believed critical for memory, there are at least two reasons to view the complaints of poor memory in aphasic patients as more important than simply a reflection of underlying aphasia. First, memory complaints often persist despite good language recovery.[1,2] Second, Ojemann et al.[3,4] demonstrated that both memory and language function can be simultaneously disrupted by electrical stimulation of left perisylvian association cortex, associated white matter, or related thalamic structures, thus illustrating a relationship between "language" and "memory" tasks. Verbal memory processes may rely on some of the same neural systems that serve language processes even though these processes are located outside limbic circuits. Colombo et al.,[5] for example, found that monkeys with lesions in auditory association cortex had impaired complex auditory memory despite unaffected auditory perception. Verbal learning deficits in humans after damage to language-specific association cortex may reflect similar processes. Furthermore, there are no good a priori reasons to believe that recall of a name as part of a "memory" task and recall of the same name as part of a "language" task should use completely different neuropsychological mechanisms. Distinction in performance in these two kinds of tasks may simply reflect factors related to task

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structure, such as context, cues, recent exposure, or instruction. Thus, in some patients, complaints of memory loss could reflect actual verbal memory deficits.

The complex interactions of memory and language have been demonstrated in several ways. First, children with acquired aphasia often have very poor academic performance (impaired acquisition of facts and skills) even when language recovery is good.[6-8] Second, using recognition tasks that avoid the necessity of verbal responses, severe aphasics show impairments in both verbal and nonverbal memory.[9-15] Third, even mildly aphasic patients may have impaired short- or long-term verbal memory when recall tasks such as the selective reminding procedure are used.[1,2,16] There have been few investigations of memory function in mildly aphasic patients and no attempts to characterize the residual language deficits in these patients in terms of anything more than classic aphasia syndromes. Thus, it is not known if specific language-processing deficits affect specific subcomponents of verbal learning. A demonstration that verbal learning deficits are functionally linked to the residual language deficit would provide further insight into the cognitive mechanisms of verbal memory.

We report an analysis of verbal learning and memory in 16 patients with mild aphasia. The aphasia is characterized in terms of their language-processing deficit rather than classic aphasia syndromes. Because aphasia was mild, memory could be tested using recall and recognition. Our hypothesis is consistent with current models of neural net architectures that postulate overlapping processing systems for language and memory. Disruption of function at any point in this overlap will affect both language and memory. The definitions of short-term memory (STM) and long-term memory (LTM) used here are principally operational: STM involves retention of information during an ongoing task over a short period of time (seconds to a few minutes). LTM involves retention of information over longer periods of time (tens of minutes) with clearly demarcated task switching. We hypothesize that phonologic deficits are associated with impaired STM and that lexicosemantic deficits are associated with encoding and possibly retrieval of information from LTM.

**Methods.** *Subjects.* Patients from the outpatient aphasia service at Braintree Hospital were recruited to participate. All patients had previously been admitted to Braintree Hospital for unequivocal and significant aphasia but had made a good recovery. Selection criteria were acute diagnosis of aphasia, single left hemisphere lesion, at least 3 months status post infarction, good language recovery, and age younger than 80. Exclusion criteria were known hearing loss, history of alcohol abuse, evidence for mental decline or CNS disease other than the aphasia-producing stroke, and nonnative English speaker.

Sixteen patients were evaluated. At the time of study their speech was intelligible, their language was fluent and grammatical (at least sentence length), and their comprehension was no worse than mildly impaired. Some patients did have speech initiation difficulties, word-finding deficits, and phonemic or semantic paraphasias in running speech. Table 1 lists the clinical profile (in terms of elementary motor, sensory, and visual field deficits) and aphasia type at the time of testing.

A control group of 16 subjects, age-matched to the study group (control subjects 68 ± 10, aphasic subjects 60 ± 12), was recruited from the population of patients with orthopedic or rheumatologic disorders at Braintree Hospital, excluding those with severe trauma, multiple fractures, amputations, and total hip replacements. None had any history of symptomatic cerebro-, cardio-, or peripheral vascular disease. Otherwise, the same exclusionary criteria used for the aphasic subjects applied.

*Language assessment.* Phonologic processing. All patients and control subjects were given a 20-item nonword repetition task. A score 2 SDs below the average of the control group was considered abnormal. Two groups were generated. Nine patients (mean score, 16 ± 2) did not differ from control subjects and were considered phonologically preserved (P+). Seven patients fell in the impaired range (mean score, 8 ± 3), and were considered phonologically impaired (P−).

Lexicosemantic processing. All patients received the Boston Naming Test.[17] To minimize phonologic influences on scoring, phonemic paraphasias were ignored if a correct target stem could be recognized. Scores 2 SDs below age- and education-matched published norms were considered abnormal. Seven patients performed within normal limits (mean z-score, −0.04 ± 0.81). These subjects constituted the lexicosemantically preserved (LS+) group. Nine subjects fell in the impaired range (mean z-score, −7.16 ± 3.30) and made up the lexicosemantically impaired group (LS−).

Phonologic and lexicosemantic status for each subject is listed in table 1.

*Lesion analysis.* All patients had CT or MRI. Lesions were classified into one of three categories: dorsolateral frontal; capsulostriatal-paraventricular; or parietal, temporal, occipital (or a mixture of posterior lesion sites). Three subjects had dorsolateral frontal lesions, four had large deep anterior lesions, and nine had posterior lesions (see table 1). Because there was considerable overlap of lesion sites in the dorsolateral frontal and capsulostriatal-paraventricular region in the first two groups, they were collapsed into a single frontostriatal group. There was *no* overlap in lesion area between the frontostriatal and posterior lesion groups.

*Memory tasks.* Short-term memory. Auditory Digit Span from the Wechsler Adult Intelligence Scale-Revised[18] served as the benchmark of immediate auditory/phonologic STM.

Recognition Span (derived from the nonmatching to sample test developed by Moss et al.[19]) was used as a measure of visual short-term recognition memory. The subject read a list of 14 words to ensure adequate reading of target items. These words then appeared, one at a time, on 14 successive sheets of paper in randomly changing spatial positions, one additional new word per sheet. The subject was asked to point to the new word on each page until all 14 words appeared on the final sheet. The total

*Table 1 Clinical profile, aphasia type, and lesion location*

| Patient | Age (yr) | Post onset (mo) | Clinical deficits | | | Recovery aphasia type | Lexicosemantics | Phonology | Lesion location |
|---|---|---|---|---|---|---|---|---|---|
| | | | Motor | Sensory | Visual | | | | |
| 1 | 76 | 4 | 0 | 2 | 0 | Conduction | + | − | PTO |
| 2 | 59 | 8 | 1 | 0 | 0 | Anomic | + | + | PTO |
| 3 | 74 | 7 | 1 | 2 | 0 | Anomic | + | + | PTO |
| 4 | 78 | 4 | 1 | 0 | 0 | Conduction | + | − | PTO |
| 5 | 48 | 4 | 1 | 2 | 0 | TCM | + | + | CS |
| 6 | 48 | 24 | 0 | 0 | 0 | Conduction | + | − | PTO |
| 7 | 40 | 36 | 0 | 0 | 0 | Conduction | + | + | DLF |
| 8 | 60 | 48 | 1 | 0 | 0 | TCM | − | − | DLF |
| 9 | 56 | 24 | 3 | 3 | 0 | TCM | − | − | CS |
| 10 | 67 | 10 | 0 | 0 | 0 | Conduction | − | − | PTO |
| 11 | 47 | 8 | 0 | 0 | 0 | None | − | + | PTO |
| 12 | 64 | 4 | 0 | 1 | 1 | Anomic | − | + | PTO |
| 13 | 76 | 3 | 1 | 2 | 2 | Anomic | − | − | PTO |
| 14 | 60 | 9 | 0 | 0 | 1 | Anomic | − | + | CS |
| 15 | 61 | 21 | 0 | 0 | 0 | Anomic | − | + | DLF |
| 16 | 49 | 42 | 3 | 3 | 0 | TCM | − | + | CS |

0 = no deficit; 1 = mild deficit; 2 = moderate deficit; 3 = severe deficit; TCM = transcortical motor aphasia; + = preserved; − = impaired; PTO = parietotemporooccipital lesion; CS = capsulostriatal-paraventricular lesion; DLF = dorsolateral frontal lesion.

number of correct pointing responses was recorded (maximum 14).

Consonant Trigrams[20,21] assessed nonlexical phonologic short-term recall and susceptibility to distraction. Trigrams were presented visually for 2 seconds on 3 × 5 index cards. The subject was instructed to read the trigram silently. Oral recall was required after 3-, 9-, or 18-second delays filled with a serial subtraction distractor task. There were five trials per delay with all trials randomly intermixed in a single block. In addition, five trials requiring immediate recall were always given at the beginning of the block and five trials with 18-second unfilled delays were given at the end. The immediate recall condition (0-second delay) served as a control of oral reading ability on this task. The number of correct consonants recalled across five trials was recorded for each condition (maximum 15).

A Recurrent Detection task[11,12] served to assess the effects of interference on auditory short-term recognition memory. In this task, strings of words, 6 to 12 items in length, were read aloud by the examiner at a constant rate of one word per 2 seconds. A pair of target words was embedded in each string separated by intervals of zero, two, four, or six intervening words. The subject was required to indicate the occurrence of the second target word later in the string. The second target word could either be identical to, a rhyme of, or a synonym of the initial target word. Target types were tested in three separate blocks of 16 trials with the different distractor intervals randomly intermixed within each block (maximum 16).

Long-term memory. The Rey Auditory Verbal Learning Test (RAVLT[22]) measures verbal learning using a list of 15 words. Immediate recall was measured after each of five learning trials and after presentation of a second 15-word list. Delayed free recall and recognition of the initial list were examined immediately after recall of the second list (short delay) and after a 20-minute delay filled with other testing (long delay) (maximum 15).

A Paired Associate Learning task[23] was used to evaluate associative learning under phonologic and lexicosemantic encoding conditions. In one condition, the stimuli consisted of rhyming word pairs, and in the other condition they consisted of synonym pairs. For each encoding condition, a list of 12 word pairs was presented over four consecutive learning trials. Subjects were required to repeat each word pair as they were presented. All aphasics were able to perform this repetition adequately. Cued recall was measured, after a 1-minute filled interval, using the first word of each pair as the cue. Cued recall was repeated after a 30-minute filled interval (maximum 12).

A Levels of Processing task[13,24] was used to assess the effect of different encoding manipulations on aphasics' LTM. In normal individuals, semantic analysis is thought to allow for "deeper" encoding that is more resistant to decay, whereas orthographically based encoding is thought to be less robust, or "shallow." Phonologic analysis represents an intermediate level of processing. In this task, subjects were asked to answer one of three types of questions regarding 60 printed stimulus words. The questions addressed the orthographic (i.e., "Is this word printed in upper case letters?"), phonologic (i.e., "Does this rhyme with ... ?"), or semantic (i.e., "Is this a type of ... ?") attributes of the target word. Response to each question was recorded as a control of accurate reading but not entered into the analysis. After a 10-minute filled interval, a four-alternative forced-choice recognition task was administered. The number of correct responses in each encoding condition was scored (maximum 20, chance 5).

The Recognition Memory Test for words[25] was used to

assess long-term forced-choice recognition memory. Fifty printed words were presented successively, with subjects indicating whether or not they found each item pleasant. A two-alternative forced-choice recognition task was given immediately after study (maximum 50, chance 25).

*Procedure.* Most subjects were tested in two sessions, each lasting approximately 1.5 hours and administered 1 week apart. Because of fatigue or logistical difficulties, testing periods were occasionally shortened and spread over three or four sessions, each at least 3 days apart. The order of test administration was randomized across subjects. Whenever possible, the performance of the aphasic subjects was compared with published normative data using z-scores. When normative data was not available, aphasics' performance was compared with that of our control group using raw scores.

**Results.** Several analyses were carried out. First, performance of the aphasic group as a whole was compared with that of control subjects. Second, to examine the effects of phonologic processing on memory, the phonologically impaired (P−) and preserved (P+) groups were compared. Third, to examine the effects of lexicosemantic processing, the lexicosemantically impaired (LS−) and preserved (LS+) groups were compared. Finally, the influence of lesion location was examined by comparing the performances of the frontostriatal and posterior lesion groups.

Optimally, subjects would have been divided into four distinct groups based on their performance on both linguistic dimensions (P+/LS+, P−/LS+, P+/LS−, and P−/LS−). This was not feasible because of the limited number of subjects in our study. Consequently, we studied the effect of linguistic performance on memory separately for each language dimension. For each of these analyses, performance on the other language dimension was used as a covariate to examine the effect of one language dimension independently of the other. Finally, age was also used as a covariate in all comparisons that examined the effects of phonology and lesion site because the groups defined according to these variables differed in terms of their average age (P+, 56 ± 11; P−, 66 ± 12; frontostriatal, 54 ± 8; temporoparietooccipital, 65 ± 12). Because of the large number of comparisons, a conservative measure of significance of $p < 0.01$ was used throughout. Data are available in table 2.

*Short-term memory.* The aphasic group as a whole was impaired on Digit Span (mean z-score = −0.90, t(15) = −3.75) and on Consonant Trigrams (F(1,28) = 7.71). As can be seen in table 2, there was a significant effect of delay on Consonant Trigrams (F(4,112) = 71.7) for aphasic patients and for control subjects. Although the group by delay interaction was not significant (F(4,112) = 1.66), simple effects analyses were performed to test the a priori hypothesis that aphasics would be more impaired after distraction. This analysis revealed that the aphasic group was impaired only at the 18-second filled delay (F(1,50) = 11.3).

Performance on Recognition Span was marginally impaired (t(28) = −2.55, $p < 0.05$). There were no significant differences between groups on the Recurrent Detection tasks (word, F(1,29) = 0.92; rhyme, F(1,29) = 0.01; synonym, F(1,27) = 1.50).

Of all probes of STM, the P+/P− groups differed only on Digit Span (P+, mean z-score = −0.22; P−, mean z-score = −1.76; F(1,12) = 21.6).

The LS+/LS− groups differed only on Recurrent Detection of words (F(1,13) = 10.6). The LS− group performed worse than the LS+ group regardless of the number of intervening items (0 items: LS+, 93 ± 12%; LS−, 64 ± 33%; 2 items: LS+, 43 ± 37%; LS−, 22 ± 26%; 4 items: LS+, 29 ± 34%; LS−, 3 ± 8%; 6 items: LS+, 39 ± 38%; LS−, 3 ± 8%).

The two anatomic groups did not significantly differ on any STM measure.

***Table 2*** *Results for aphasic group as a whole*

| Measure | Control subjects | Aphasic subjects |
|---|---|---|
| Digit Span (z-score) | 0 ± 1.0 | −0.9 ± 1.0 |
| Recognition Span (raw score) | 12.8 ± 1.2 | 11 ± 2.4 |
| Consonant Trigrams | | |
| 0-sec delay | 100 ± 0 | 96 ± 7 |
| 3-sec delay | 89 ± 15 | 75 ± 20 |
| 9-sec delay | 67 ± 18 | 59 ± 22 |
| 18-sec delay | 63 ± 17 | 43 ± 19 |
| 18-sec nonfilled delay | 99 ± 3 | 91 ± 15 |
| Recurrent Detection | | |
| Words | 82 ± 27 | 78 ± 26 |
| Rhymes | 35 ± 37 | 35 ± 38 |
| Synonyms | 59 ± 32 | 53 ± 31 |
| RAVLT | | |
| Learning Trial 1 | 35 ± 12 | 23 ± 13 |
| Learning Trial 5 | 76 ± 18 | 51 ± 17 |
| Free Recall, Short Delay | 60 ± 21 | 37 ± 24 |
| Free Recall, Long Delay | 62 ± 23 | 35 ± 27 |
| Recognition (corrected), Short Delay | 88 ± 12 | 76 ± 23 |
| Recognition (corrected), Long Delay | 89 ± 11 | 70 ± 27 |
| Paired Associate Learning | | |
| Rhyme, immediate recall | 53 ± 27 | 40 ± 20 |
| Rhyme, delayed recall | 53 ± 25 | 39 ± 21 |
| Semantic, immediate recall | 83 ± 17 | 65 ± 28 |
| Semantic, delayed recall | 88 ± 13 | 72 ± 24 |
| Levels of Processing | | |
| Orthographic | 53 ± 20 | 44 ± 18 |
| Rhyme | 68 ± 17 | 63 ± 23 |
| Semantic | 87 ± 13 | 72 ± 18 |
| Recognition Memory Test, Words (z-score) | 0.0 ± 1.0 | −0.3 ± 1.4 |

Values are mean % correct (unless otherwise noted) ± SD.

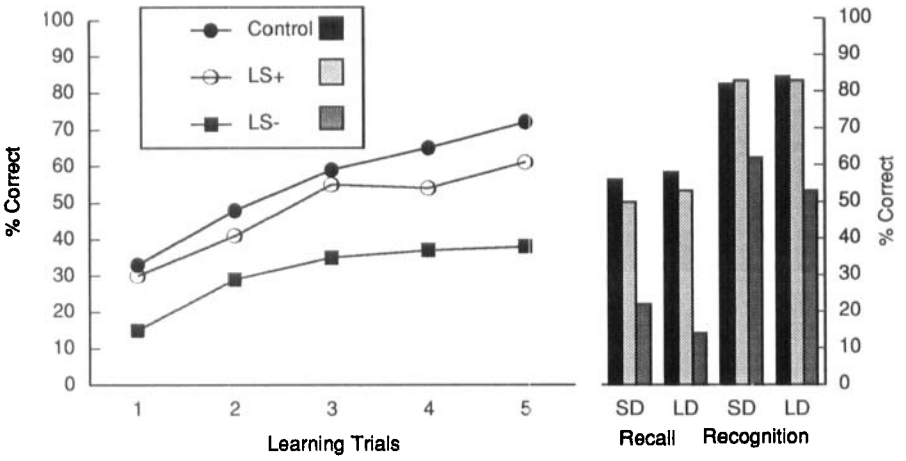


*Figure. Performance on RAVLT as a function of lexicosemantic ability. LS+ = lexicosemantically preserved; LS− = lexicosemantically impaired; SD = short delay; LD = long delay.*

*Long-term memory.* The aphasic group as a whole was impaired on all elements of the RAVLT compared with control subjects (see table 2). Learning was impaired across all five learning trials ($F(1,36) = 16.8$), as was free recall across short and long delay ($F(1,36) = 9.93$) and recognition across the same delays ($F(1,36) = 13.7$). Comparing performance at Learning Trial 5, short delay and long delay, there was a strong effect of delay on both recall ($F(2,72) = 36.00$) and recognition ($F(2,72) = 25.0$) but no interaction with group in either case ($F(2,72) = 0.70$ and $F(2,72) = 2.73$, respectively). The lack of an interaction indicates a similar decay of information over time in the two groups.

Acquisition of Paired Associates across four trials was marginally impaired in both learning conditions (rhyme, $F(1,28) = 4.56$, $p < 0.05$; synonym, $F(1,30) = 5.80$, $p < 0.05$), but there were no significant group differences in delayed cued recall (rhyme, $t(28) = -1.61$; synonym, $t(30) = -2.28$).

On the Levels of Processing task, there was a strong effect of encoding condition ($F(2,60) = 42.1$), but neither the effect of group ($F(1,30) = 2.85$) nor the group by encoding interaction ($F(2,60) = 1.50$) were significant.

The aphasic patients also performed normally on the Recognition Memory Test for words (z-score = −0.29, $t(15) = -0.93$).

There were no significant differences between the P− and P+ groups on tests of LTM.

There were numerous significant differences between the LS+ and the LS− groups on LTM tasks. On the RAVLT (figure), the LS− group was impaired compared with the LS+ group on acquisition ($F(1,14) = 18.25$), free recall ($F(1,14) = 13.88$), and recognition ($F(1,14) = 12.80$). For both groups, there was a strong effect of delay on recall ($F(2,28) = 23.19$) and recognition ($F(2,28) = 13.15$). A marginally significant group × delay interaction was found for free recall ($F(2,28) = 3.73$, $p < 0.05$) but not for recognition ($F(2,28) = 0.53$).

For Paired Associate Learning of synonyms (table 3), the LS− group showed worse acquisition across four learning trials ($F(1,14) = 11.72$) and impairment in delayed cued recall ($F(1,13) = 10.4$). For Paired Associate Learning of rhymes, the LS− group performed worse only on delayed cued recall (LS+, 53%; LS−, 28%; $F(1,13) = 12.1$).

No group differences between the LS+ and the LS− groups were found in the Levels of Processing task ($F(1,13) = 0.21$), although effect of learning condition remained strong ($F(2,28) = 14.57$). Performance on the Recognition Memory Test for words was equivalent between groups ($F(1,13) = 3.1$).

There were no significant differences between the two lesion groups on any test of LTM.

**Discussion.** Our findings suggest that patients with aphasia can have verbal memory deficits, even when the aphasia is mild. The specific form of the memory deficit is determined, at least in part, by the specific linguistic deficit. In contrast, we were not able to associate verbal learning impairments with any specific lesion site.

The aphasic group as a whole was impaired on several tasks, some involving STM and some involving LTM. They were impaired on all tasks that demanded free recall (Digit Span, Consonant Trigrams) and were particularly impaired on acquisition of information across learning trials (RAVLT). In contrast, there were fewer group differences on tasks that required recognition (Recurrent Detection and Recognition Memory for words). As we discuss below, this suggests a role of language systems in self-organized acquisition of information into verbal memory.

The various effects of mild aphasia on verbal learning were due to different language-processing deficits in different subgroups of patients. Specific impairments in phonologic functions, determined by

***Table 3*** *Paired associate learning of synonyms as a function of lexicosemantic performance*

| Condition | Preserved (LS+) | Impaired (LS−) |
|---|---|---|
| Trial 1 | 67 ± 13 | 37 ± 18 |
| Trial 2 | 86 ± 12 | 57 ± 23 |
| Trial 3 | 94 ± 9 | 58 ± 26 |
| Trial 4 | 83 ± 10 | 57 ± 30 |
| Delayed Recall | 87 ± 13 | 60 ± 25 |

Values are mean percents ± SD.

poor Nonword Repetition, had relatively little effect on verbal memory. Phonologic impairment had a strong effect only on Digit Span. Recognition Span (nonmatch to sample) and Recurrent Detection may not depend on phonologic storage but may be accomplished at a strictly lexical level. Poor performance on Recurrent Detection tasks was noted in previous investigations,[11-13] but, compared with the patients in our study, these patients were much more severely impaired in lexicosemantic and phonologic processes. All aphasic patients, with or without apparent phonologic deficits, performed poorly on the Consonant Trigrams task after a long delay filled with interference. It is unclear why the P+ group was able to perform significantly better than the P− group on Digit Span but was indistinguishable from the P− group on Consonant Trigrams. Either the Nonword Repetition task was not sufficiently sensitive to identify subtle phonologic compromise (see Gathercole[26]) or the P+ group was deficient in some nonphonologic mechanism that normally supports STM (see McCarthy and Warrington[27] for an example of lexical support of STM and Warrington and Shallice[28] for evidence of a visual short-term store). Further research on the involvement of language in different components of STM is needed to clarify this finding.

Lexicosemantic deficits had no major effect on our STM measures but very definite effects on LTM, whether measured in terms of free recall (RAVLT) or cued recall (Paired Associate Learning). In both instances, patients with lexicosemantic deficits were particularly impaired in the acquisition of information (see figure), both after single and multiple study trials. However, taking the level of initial acquisition into account, the rate of information loss in the LS− was no greater than in the LS+ group, whether on free recall or recognition. These findings suggest that lexicosemantic processes play an important role in the acquisition of verbal information, presumably because they allow for the formation of associations among study items and associations between study items and information already existing in semantic memory. These associations, in turn, may provide additional retrieval routes during recall. Because recognition performance relies less on initiation of strategic retrieval processes, lexicosemantic deficits may have a less-pronounced effect on aphasics' recognition performance. Indeed, in two of three recognition tasks (Levels of Processing and Recognition Memory for Words), the aphasic patients performed comparably with control subjects. The aphasics' performance on the recognition subtask of the RAVLT may reflect the contribution of more elaborate encoding processes on the part of normal subjects afforded by additional study trials. Thus, although both encoding and retrieval processes likely depend on limbic-mnemonic processes, language-based processes also make an important contribution to the acquisition of verbal information. Based on our findings, language mechanisms seem less important for storage and maintenance of information in the kind of verbal memory tested here. The effect of aphasia, and lexicosemantic deficits in particular, on semantic learning remains to be elucidated. The relationship of semantic memory and episodic verbal memory, in terms of interacting processing systems, needs further study.

We did not demonstrate a significant lesion site effect on any of our memory measures. With generally comparable groups, other investigators[1,2] demonstrated that parietal lesions are associated with selective STM deficits, whereas frontal lesions are associated with LTM deficits. There are at least two possible reasons for this difference in results. First, we used a much greater variety of LTM measures than the previous studies, both of which used the selective reminding paradigm.[29] Our methods may have produced poor performance in a wider variety of patients. The assumptions of the selective reminding task about compartmental distinctions between STM and LTM are quite specific to that task. Use of discrete assessments of STM and LTM may give a different picture of impairment. All tests of LTM are not equally sensitive to mild deficits. Second, although our aphasic population was quite similar to the patient groups in the studies mentioned, these other studies used lesion location as their selection criterion. Possibly, they achieved more homogeneous lesion groups for comparison.

That specific language-processing deficits have specific consequences for different aspects of verbal memory does not mean that the relationship is, at all times, bidirectional. With the exception of poor acquisition of new vocabulary,[30] there is no known effect of LTM deficits on language function. Although STM may be necessary for second pass analysis of complex verbal material[31] and for language acquisition in childhood,[32] dependence on lexicosemantic structure for acquisition of novel linguistic information is evident already in early childhood.[26] The role of phonologic STM in language processes remains controversial.

The current study is also compatible with modern concepts of neural networks, where different functions may share processing units and each function is defined by the totality of its connections and components. Language production requires, at a minimum, networks dedicated to syntax structure, grammatical morphology, word generation, and articulation. Verbal memory probably has no need for syntax, morphology, or possibly even articulation, but a word-generation system, based in a lexicosemantic network, may be essential for normal acquisition of novel verbal material. Patients with aphasia, even mild aphasia, are therefore at risk for substantial verbal memory deficits.

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