Pergamon

PII: S0028-3932(97)00036-5

Neuropsychologia, Vol. 35, No. 8, pp. 1087–1092, 1997

Printed in Great Britain
0028-3932/97 $17.00+0.00

Impaired word-stem priming in patients with temporal–occipital lesions

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(*Received* 5 *May* 1996; *accepted* 17 *January* 1997)

Abstract—In the word-stem priming test, words are presented (e.g., MOTEL, PARADE), and later subjects are shown three-letter word stems (e.g., MOT, PAR) and asked to complete each stem with the first word that comes to mind. Word-stem priming, as well as other aspects of implicit memory, are intact in amnesic patients with medial temporal lesions. However, this form of priming has been shown to be impaired in patients with Alzheimer's disease, suggesting that damage to neocortical areas outside the medial temporal lobe contributes to impaired priming in these patients. To examine the role of posterior cortical areas on word-stem priming, we administered the test to patients with unilateral temporal–occipital lesions. Patients with temporal–occipital lesions exhibited significantly impaired priming on this test. The findings suggest a critical role of the inferior posterior neocortex in the expression of this form of implicit memory. © 1997 Elsevier Science Ltd

Key Words: implicit memory; posterior neocortex; lexical memory.

Introduction

Implicit memory concerns a class of memory functions important for the facilitation or bias of recently presented information [8, 18, 20, 21, 24]. In contrast, explicit memory is exemplified by conscious recollection and can be assessed by standard tests of memory, such as recall and recognition. The distinction between implicit and explicit memory has played an important role in understanding the brain mechanisms underlying memory performance. Specifically, the diencephalic midline and medial temporal areas damaged in amnesia are critical for explicit memory but do not contribute to the expression of implicit memory.

A benchmark measure of implicit memory is the word-stem completion test [9, 18, 21]. In this test, subjects are presented words for study and then asked to complete each three-letter word stem with the first word that comes to mind (e.g., MOT for MOTEL). This test necessitates lexical or word knowledge and has been shown to involve both perceptual and non-perceptual (i.e. cross-modality) components [6, 8, 12]. Amnesic patients exhibit entirely normal performance on this word priming test, despite severe impairment on tests of recall or recognition [9]. Using the word-stem completion test, intact priming has been observed in other patient groups, including patients with Huntington's disease [17, 23] and patients with frontal lobe lesions [22]. These findings suggest that the basal ganglia and anterior neocortex do not contribute significantly to this form of implicit memory.

To date, the only patient samples that have exhibited impaired performance on the word-stem completion test are patients with Alzheimer's disease [17, 23]. These findings have been replicated and extended [2, 5]; however, it should be noted that others have demonstrated intact word-completion performance in patients with Alzheimer's disease [4, 10]. Equivocal findings may be attributed to subtle differences in test procedures and heterogeneity of dementia observed in patients with Alzheimer's disease [5]. Importantly, patients with Alzheimer's disease have demonstrated impaired word-stem completion performance using identical procedures and word stimuli that have been used to demonstrate intact priming in patients with frontal lobe lesions and in amnesic patients with medial temporal lesions [17, 22, 23]. Taken together, these findings implicate the contribution

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of posterior neocortical areas outside of the medial temporal region for the expression of word-stem priming.

Support for the role of the posterior neocortex in lexical and perceptual priming has been provided by recent neuroimaging studies. Word-stem priming has been associated with decreased positron emission tomography (PET) activation in inferior cortical regions [1, 26]. Decreased PET activity is consistent with the view that prior presentation of a word reduces the metabolic demands of processing the same word at a later time. The posterior areas that mediate priming may actually be the storehouses of lexical and perceptual knowledge about words. Support for this notion comes from neuroimaging studies in which temporal–occipital regions become particularly active on tasks involving word knowledge [14, 16]. Also, neuropsychological studies have shown that patients with temporal lobe atrophy exhibit impaired access to lexical and conceptual knowledge [11].

In the present study, we assessed priming in the word-stem completion test in a group of patients with temporal–occipital lesions. Prior findings from single-case studies of patients with posterior cortical lesions have produced somewhat mixed findings. In one study [19], a letter-by-letter reader with a left temporal–occipital lesion exhibited evidence of word priming on a perceptual identification task. In this task, the ability to identify words that are briefly presented is facilitated by prior presentation of the words. In that study, control subjects were not tested because, in order to observe word identification priming in the patient, the presentation of the stimuli had to be slowed to such an extent that control subjects would have performed at ceiling levels for the same exposure durations. Thus, it could not be determined whether the priming effect exhibited by this patient was normal. Impaired lexical or perceptual priming has been observed in a patient with a right occipital lesion [6], in a patient with a left temporal–occipital lesion [3] and a patient with bilateral temporal–occipital lesions [12]. We sought to clarify and extend these single-case studies by assessing a group of patients with inferior cortical lesions involving temporal–occipital regions. To the extent that this brain region mediates priming effects, such patients should exhibit impairment on tests of word-stem completion.

Methods

Participants

Patients with temporal–occipital lesions. Seven patients (one woman, six men) with unilateral temporal–occipital lesions were tested. Based on computerized reconstructions of the lesions using computer tomography (CT) or magnetic resonance scans, the average lesion volume was estimated at 43.5 cm^3 (see Fig. 1, Table 1). Patients were selected on the basis of a single neurological event. Patients with psychiatric disturbances, multiple neurological events or dementia were excluded. All lesions were at least six months old. Four patients had left hemisphere lesions and three had right hemisphere lesions. The lesions of five patients (four left- and one right-hemisphere patients) occurred as a result of infarction of the posterior cerebral artery. In the other two right-hemisphere patients, one lesion was due to surgical resection of an arterial–venous malformation and the other was due to a congenital defect which may have been resulted from neonatal or perinatal vascular occlusion. The cortical lesions extended into the medial temporal region, including the hippocampus, in six patients, and one other patient had a fornix lesion with possible hippocampal sparing. The patients had an average age of 63.3 years and an average of 13.9 years of education.

On neuropsychological tests (see Table 1), the patients scored within the normal range on the Full-Scale Weschler Adult Intelligence Scale—Revised (WAIS-R; mean = 97.3). Their mean scaled scores on the Weschler Memory Scale—Revised (WMS-R) were as follows: Attention = 95.7, General Memory = 97.3, Delayed Memory = 91.6, Verbal Memory = 95.0, Visual Memory = 101.3. Performance on the Warrington Recognition Memory Test [29] showed that left-hemisphere patients were particularly impaired for word recognition (number of words recognized: control = 45.7, right = 46.3, left = 33.3) as seen by a significant group × recognition interaction [$F(2,11) = 13$, $P < 0.005$]. This finding could be related directly to the temporal–occipital lesions or to the presence of hippocampal damage in the left-hemisphere group. All patients, however, exhibited normal recognition memory for faces compared to control subjects (number of faces recognized: controls = 40.9, right patients = 40.7, left patients = 38.0). Two of the left-hemisphere-lesioned patients were mildly to severely alexic, yet both were able to read the word stimuli and complete the stems with words.

Control group. Eight healthy, normal control subjects (six men and two women) were recruited form the Martinez VA Medical Center volunteer service and the Martinez Senior Center. The control subjects were matched to the patients with respect to age (64.7 years) and education (12.9 years). The control subjects scored comparably to the patients with temporal–occipital lesions on the Information (control subjects = 22.0, patients = 20.7), Digit Symbol (control subjects = 42.9, patients = 36.0) and Vocabulary (control subjects = 47.9, patients = 51.9) subtests of the WAIS-R.

Stimuli and procedure

The words and stems used in this experiment were the same as those used in previous neuropsychological studies of word-stem completion [17, 23]. The words had an average frequency of occurrence of 72 per million [13], and each three-letter stem could be completed with at least 10 possible solutions, only one of which was presented at study [9]. In the study phase, subjects were shown 10 words one a time (e.g., MOTEL, PARADE) and asked to rate how much they liked each word on a five-point scale (1 = dislike very much, 5 = like very much). Words were printed on 5 × 8 in. index cards (Helvetica type, 18 pt). Subjects were given enough time to produce a rating for each word (approximately 3–5 sec), and the liking scale was displayed continuously during the rating task. After all words were presented, the list was presented again using the same incidental learning task, but in a different order. For each presentation, two filler words were placed at the beginning of the list, and three were placed at the end to reduce primacy and recency effects, respectively.

Immediately following the two presentations of the words, subjects were asked to participate in a different task. Twenty word stems (e.g., MOT, PAR) were printed on a sheet in a random order, and subjects were asked to complete each stem with the first word that came to mind. Ten of the stems could

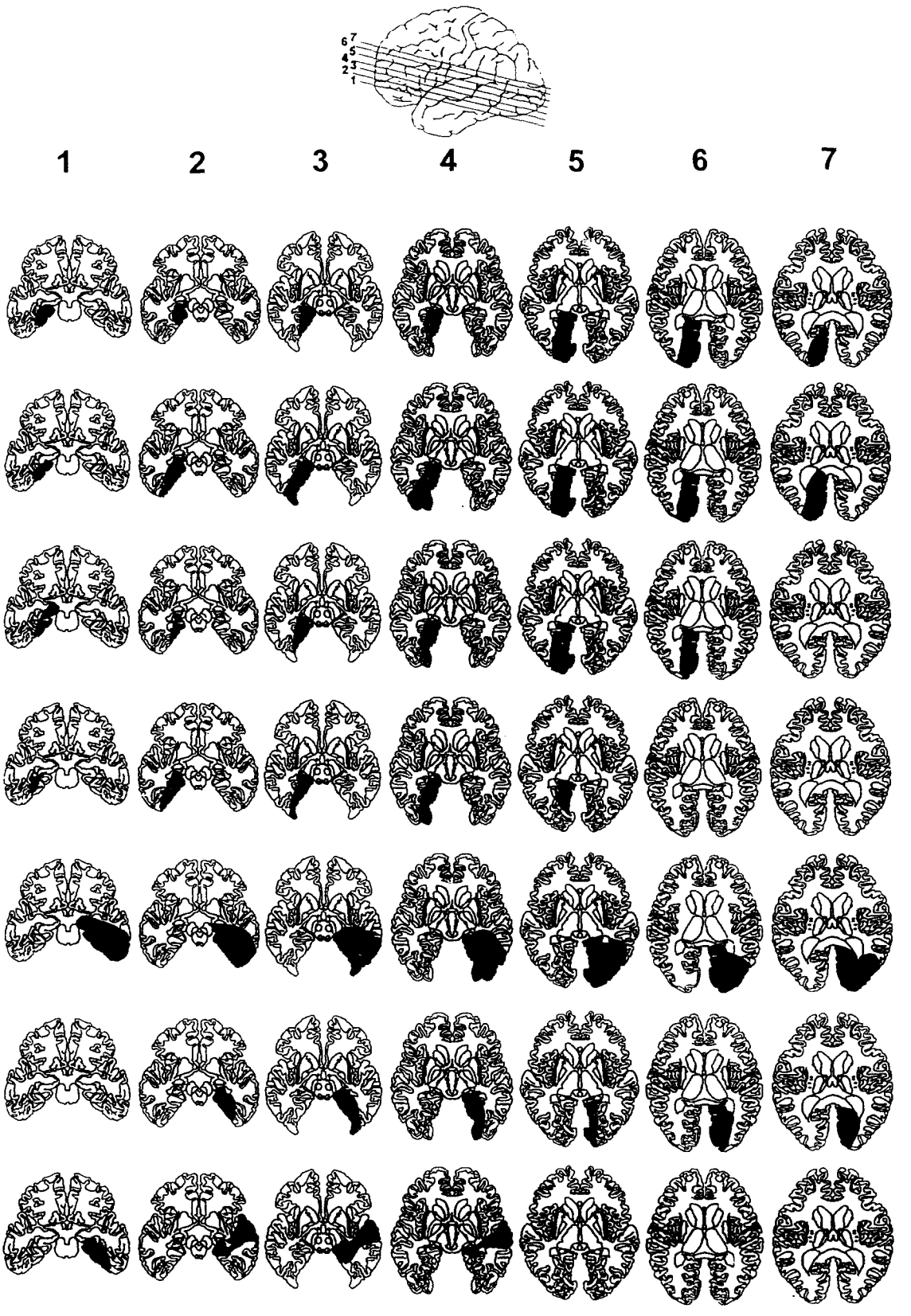

Fig. 1. CT reconstructions of lesioned areas in each of the seven patients with temporal–occipital lesions (average lesion volume 43.5 cm^3). Four patients had left unilateral lesions and three had right unlateral lesions. Lateral view above indicates position of axial cuts (1 = most inferior, 7 = most superior).

Table 1. Patient characteristics and word-stem completion data*

Patient	Lesion	Vol.	Age	ED	IQ	MQ	RMT-W	RMT-F	Test 1	Test 2	Mean	BL
J.S.	Left	17.4	72	13	103	79	36	30	60	40	50	0
A.L.	Left	29.9	63	14	92	81	25	37	40	10	25	5
E.M.	Left	61.7	78	18	112	81	38	46	20	20	20	10
W.M.	Left	27.6	71	16	117	102	32	36	20	40	30	10
F.N.	Right	26.0	58	12	120	126	46	42	30	0	15	10
D.R.	Right	27.9	45	12	97	111	47	33	30	10	20	10
J.C.	Right	114.0	56	12	185	101	46	46	30	20	25	10
Patient mean	4	3.5	63.3	13.9	103.7	97.3	38.6	38.7	32.9	20.0	26.4	7.9
Control mean			65.7	13.6	—	—	45.8	40.3	41.1	45.6	43.3	7.8

*Vol., lesion volume (cm^3); ED, years of education; IQ, WAIS-R Full Scale score; MQ, WMS-R General Memory Index; RMT-W, number correct for word recognition on RMT; RMT-F, number correct for face recognition on RMT; Test 1, first test of word-stem completion (%); Test 2, second test of word-stem completion (%); Mean, mean of Tests 1 and 2 (%); BL, mean baseline guessing score (%); —, score not available.

be completed using previously presented study words, and the other 10 stems could be completed with non-presented words and were used to assess baseline word completion rates. Words and word stems used to assess priming performance for one subject were used to assess baseline completion rates for another subject. Each word stem could be completed with at least 10 possible words. Subjects were instructed to use English words; proper nouns were not accepted as correct responses. To reduce the effects of perceptual anomalies or response slowing, subjects were given ample time (up to 10 sec) to complete the stems. Immediately following the stem completion test, the study and test procedure was then repeated using a different set of word stimuli and stems.

Results

Baseline guessing rates were calculated by the percentage of target words completed by subjects when these words were not presented in the study phase. As shown in Fig. 2 and Table 1, baseline guessing rates by patients with temporal–occipital lesions and control subjects were comparable (patients = 7.9%, control = 6.3%). Moreover, no statistical difference was observed between left and right lesion groups (baseline; left = 6.3%, right = 10.0%). Thus, the patients were able to make suitable responses to the word stems. This finding is consistent with the finding that patients with Alzheimer's disease can also exhibit baseline guessing rates that are comparable to control subjects [17, 23].

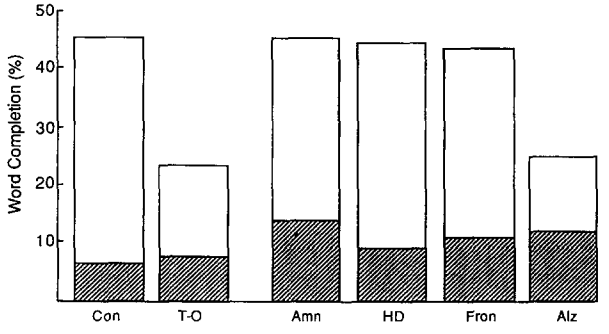

Fig. 2. Word-stem completion performance by control subjects (Con) and patients with temporal–occipital lesions (T-O). Also shown are data from amnesic patients (Amn), patients with Huntington's disease (HD), patients with frontal lobe lesions (Fron) and patients with Alzheimer's disease (data reprinted from [17] and [22]). Shaded area within each bar represents baseline guessing rates.

We subjected the word-stem completion data to a 2×2 analysis of variance with group (patients vs controls) and test condition (completion vs baseline) as factors. There was a significant group main effect [$F(1,13) = 5.24$, $P < 0.05$, $MSe = 118$] and a significant group × test condition interaction [$F(1,13) = 5.39$, $P < 0.05$, $MSe = 159$]. That is, despite comparable levels of baseline performance, the patients exhibited impaired word-stem priming compared to control subjects. As a group, the patients exhibited priming effects above baseline levels [$t(6) = 3.2$, $P < 0.05$], but these effects were not as large as those observed in the control group. No significant difference was observed between the left and right lesion patients (left = 31.3%, right = 20.0%), though these findings must be tempered by the small sizes of the subgroups. Another way to assess priming performance is to score the subjects on the percentage of words completed above baseline levels (e.g., percentage studied words completed minus percentage baseline words completed). Using this priming score, patients with temporal–occipital lesions again exhibited reduced priming compared to control subjects [lesion patients = 18.6%, control = 39.3%; $F(1,13) = 5.0$, $P < 0.05$].

Also shown in Fig. 2 are data from previous neuropsychological studies using the same stimulus material and test procedures [17, 22, 23]. The findings suggest that the impairment observed in the present study of patients with temporal–occipital lesions is comparable to the impairment observed in patients with Alzheimer's disease.

Discussion

The findings provide evidence that word-stem priming is impaired in patients with temporal–occipital lesions.

Although these patients generally exhibit lesions that extend into the medial temporal region, it has been demonstrated in numerous studies that circumscribed lesions of the medial temporal lobe do not produce impairment in word-stem priming [20, 21, 25]. Thus, the findings suggest that adjoining temporal–occipital areas that extend posteriorly are involved in the mediation of these priming effects. Prior to the present study, the only patient groups to exhibit impaired word-stem priming were patients with Alzheimer's disease [2, 5, 17, 23]. However, in Alzheimer's disease cortical atrophy is rather widespread, and it is difficult to isolate neural systems that may be involved in priming. The present findings suggest a critical role of the inferior temporal–occipital area in the expression of word-stem priming.

The findings are consistent with case studies of word-stem priming in patients with lesions involving the temporal–occipital regions [3, 6, 12]. The findings are also consistent with neuroimaging studies, which suggest that the inferior posterior cortex contributes to word priming and the retrieval of lexical information [1, 14, 16, 26]. Also, a recent study of event-related potential (ERP) reported reductions in the amplitude of a late positive ERP component associated with stimulus repetition in right temporal–occipital lesioned patients performing a priming task [27]. These patients also exhibited poor priming performance on behavioral measures.

One advantage of the word-stem test is that subjects are given ample time to form completions, and thus impairment cannot be attributed to general slowing or to gross perceptual impairment. Furthermore, the finding of comparable baseline levels between patients and control subjects suggests that the patients could elicit words for the three-letter stems. Thus, compared to control subjects, the impairment in word-stem completion occurred because the words that were elicited during the test were less likely to be words that were presented at study. Importantly, the use of the same materials and procedures used in previous studies of neurological patients suggests that such deficits are not observed in patients with medial temporal lobe lesions or frontal lobe lesions [17, 22], are observed in patients with Alzheimer's disease [17], and are now observed in patients with temporal–occipital lesions. The present findings, however, cannot determine if impaired word-stem priming reflects problems in lexical access or problems in visual word perception. Based on other reports, there is evidence that impaired performance on this task is mediated by problems in modality-specific processing, as indicated by particular impairment in occipital–temporal patients when both study and test are presented in the visual modality [6, 12].

In the present study, there did not appear to be differences in the level of impaired priming in right- and left-hemisphere patients, though these conclusions must be tempered by the small numbers in each subgroup. Previous studies have shown that patients with alexia as a result of left temporal–occipital lesions exhibit impaired performance on tests of verbal memory, whereas patients who do not exhibit alexia (left- or right-hemisphere lesions) do not exhibit these verbal impairments [28]. These and other findings have led to the hypothesis that patients with alexia exhibit impairment in the processing of letter strings as lexical wholes [7, 15, 30]. Evidence for the role of the right posterior hemisphere on tests of priming comes from case studies [6] and PET studies [1, 26], in which word-stem completion appeared to be mediated by inferior temporal–occipital areas. Taken together, these findings raise the possibility that both left and right temporal–occipital regions are involved in word-stem priming, though these two regions may be contributing to priming in different ways. Further studies using cross-modality conditions and conceptual priming tasks may help sharpen the kind of processing operations that contribute to implicit memory.

Acknowledgements—This research was supported by NIH Program Project grant NS17778. We would like to thank Diane Swick, Clay Clayworth and Lucretia Fairchild for their assistance in this research.

References

1. Buckner, R. L., Petersen, S. E., Ojemann, J. G., Miezin, F. M. *et al.* Functional anatomical studies of explicit and implicit memory retrieval tasks. *Journal of Neuroscience* **15,** 12–29, 1995.
2. Carlesimo, G. A., Fadda, L., Marfia, G. A. and Caltagirone, C. Explicit memory and repetition priming in dementia: evidence for a common basic mechanism underlying conscious and unconscious retrieval deficits. *Journal of Clinical and Experimental Neuropsychology* **17,** 44–57, 1995.
3. Carlesimo, G. A., Fadda, L., Sabbadini, M. and Caltagirone, C. Visual repetition priming for words relies on access to the visual input lexicon: evidence from a dyslexic patient. *Neuropsychologia* **32,** 1089–1100, 1994.
4. Christensen, H. and Birrell, P. Explicit and implicit memory in dementia and normal ageing. *Psychological Research/Psychologische Forschung* **53,** 149–161, 1991.
5. Downes, J. J., Davis, E. J., Davies, P. D. M., Perfect, T. J. *et al.* Stem-completion priming in Alzheimer's disease: the importance of target word articulation. *Neuropsychologia* **34,** 63–75, 1996.
6. Gabrieli, J. D., Fleischman, D. A., Keane, M. M., Reminger, S. L. and Morrell, F. Double dissociation between memory systems underlying explicit and implicit memory in the human brain. *Psychological Science* **6,** 76–82, 1995.
7. Geschwind, N. Disconnexion syndromes in animals and man. Part II. *Brain* **88,** 585–645, 1965.
8. Graf, P., Shimamura, A. P. and Squire, L. R. Priming across modalities and priming across category levels: extending the domain of preserved function in

amnesia. *Journal of Experimental Psychology: Learning, Memory and Cognition* **11,** 386–396, 1985.
9. Graf, P., Squire, L. R. and Mandler, G. The information that amnesic patients do not forget. *Journal of Experimental Psychology: Learning, Memory and Cognition* **10,** 164–178, 1984.
10. Grosse, D. A., Wilson, R. S. and Fox, J. H. Preserved word-stem-completion priming of semantically encoded information in Alzheimer's disease. *Psychology and Aging* **5,** 304–306, 1990.
11. Hodges, J. R., Patterson, K., Oxbury, S. and Funnell, E. Semantic dementia. *Brain* **115,** 1783–1806, 1992.
12. Keane, M. M., Gabrieli, J. D. E., Mapstone, H. C., Johnson, K. A. *et al.* Double dissociation of memory capacities after bilateral occipital lobe or medial temporal lobe lesions. *Brain* **118,** 1129–1148, 1995.
13. Kucera, M. and Francis, W., *Computational Analysis of Present-day American English.* Brown University Press, Providence, RI, 1967.
14. Martin, A., Haxby, J. V., Lalonde, F. M., Wiggs, C. L. and Ungerleider, L. G. Discrete cortical regions associated with knowledge of color and knowledge of action. *Science* **270,** 102–105, 1995.
15. Patterson, K. and Kay, J. Letter-by-letter reading: psychological descriptions of a neurological syndrome. *Quarterly Journal of Experimental Psychology* **34A,** 411–441, 1987.
16. Petersen, S. E., Fox, P. T., Snyder, A. Z. and Raichle, M. E. Activation of extrastriate and frontal cortical areas by visual words and word-like stimuli. *Science* **249,** 1041–1044, 1990.
17. Salmon, D. P., Shimamura, A. P., Butters, N. and Smith, S. Lexical and semantic priming deficits in patients with Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology* **10,** 477–494, 1988.
18. Schacter, D. L. Implicit memory: history and current status. *Journal of Experimental Psychology: Learning, Memory and Cognition* **13,** 501–518, 1987.
19. Schacter, D. L., Rapscak, S. Z., Rubens, A. B., Tharan, M. and Laguna, J. Priming effects in a letter-by-letter reader depend upon access to the word form system. *Neuropsychologia* **28,** 1079–1094, 1990.
20. Shimamura, A. P. Priming in amnesia: evidence for a dissociable memory function. *Quarterly Journal of Experimental Psychology* **38,** 619–644, 1986.
21. Shimamura, A. P. Neuropsychological analyses of implicit memory: recent progress and theoretical interpretations. In *Implicit Memory: New Directions in Cognition, Development, and Neuropsychology*, ed. P. Graf and M. E. Masson. Erlbaum Associates, Hillsdale, NJ, 1993, pp. 265–285.
22. Shimamura, A. P., Gershberg, F. B., Jurica, P. J., Mangels, J. A. and Knight, R. T. Intact implicit memory in patients with frontal lobe lesions. *Neuropsychologia* **30,** 931–937, 1992.
23. Shimamura, A. P., Salmon, D. P., Squire, L. R. and Butters, N. Memory dysfunction and word priming in dementia and amnesia. *Behavioral Neuroscience* **101,** 347–351, 1987.
24. Squire, L. R. *Memory and Brain.* Oxford University Press, New York, 1987.
25. Squire, L. R. Memory and the hippocampus: a synthesis from findings with rats, monkeys, and humans. *Psychological Review* **99,** 195–231, 1992.
26. Squire, L. R., Ojemann, J. G., Miezin, F., Petersen, S., Videen, T. and Raichle, M. Activation of the hippocampus in normal humans: a functional anatomical study of human memory. *Proceedings of the National Academy of Sciences, U.S.A.* **89,** 1837–1841, 1992.
27. Swick, D. and Knight, R. T. Inferior temporal–occipital and hippocampal contributions to word priming and recognition memory. *NeuroReport*, in press.
28. Von Cramon, D. Y., Hebel, N. and Schuri, U. Verbal memory and learning in unilateral posterior cerebral infarction. *Brain* **11,** 1061–1077, 1988.
29. Warrington, E. K. *Recognition Memory Test.* NFER-Nelson, Windsor, 1984.
30. Warrington, E. K. and Shallice, T. Word-form dyslexia. *Brain* **30,** 99–112, 1980.