

Memory Dysfunction and Word Priming in Dementia and Amnesia

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Memory performance of patients with the clinical diagnosis of Alzheimer's disease was compared with performance of patients with alcoholic Korsakoff's syndrome and patients with Huntington's disease. Although all patient groups exhibited impairment on tests of verbal memory, only patients with Alzheimer's disease exhibited impaired priming. Priming is an unconscious expression of recently encountered material, and it is intact even in severely amnesic patients. Because mildly demented patients with Alzheimer's disease exhibited impaired priming, damage to brain structures in addition to those damaged in the amnesic syndrome must occur at a relatively early stage of the disease process.

Alzheimer's disease is the most common form of dementing illness, affecting as many as 5% of persons over 65 years of age (Henderson, 1986; Huppert & Tym, 1986; Terry & Katzman, 1983). The disease is associated with progressive cortical, limbic, and subcortical brain damage (Arendt, Bigl, Arendt, & Tennstedt, 1983; Terry & Davies, 1980; Whitehouse, Price, Struble, Clark, & DeLong, 1982) and with a spectrum of cognitive changes including memory loss, deterioration of intellectual function, and personality change (Corkin, Davis, Growdon, Usdin, & Wurtman, 1982; Mohs, Greenwald, Dunn, & Davis, 1985; Weingartner et al., 1981). Because neuropathological information is obtained only at the end-stage of the disease process, little is known about the ordinary trajectory of the disease, that is, when specific brain regions become affected. Similarly, although memory impairment is recognized to be an early symptom of the disease, it is not known which aspects of memory are affected or at what point other cognitive functions become impaired.

Recent findings have shown that Alzheimer's disease prominently disrupts cortic limbic connections by damaging entorhinal and subicular cortices (Hyman, Van Hoesen, Damasio, & Barnes, 1984), thereby isolating the hippocampal formation from neocortex. This finding is critical, because damage to the hippocampal formation causes a selective amnesic disorder in humans (Scoville & Milner, 1957; Zola-Morgan, Amaral, & Squire, 1986). In such cases, amnesia occurs without impairment of other cognitive functions, such as language and intelligence. Patients with damage to the diencephalic midline (e.g., patients with Korsakoff's syndrome) also exhibit a selective memory disorder (Butters, 1984; Butters & Cermak, 1980; Mair, Warrington, & Weiskrantz, 1979; Squire & Shimamura, 1986; Talland, 1965).

Although amnesic patients perform poorly when they are asked explicitly to recall or recognize material presented only a few minutes ago, they can exhibit sparing of certain memory functions, as indicated by normal performance on tests of digit span (Baddeley & Warrington, 1970; Drachman & Arbit, 1966), skill learning (Brooks & Baddeley, 1976; Cohen & Squire, 1980), and priming (Graf, Squire, & Mandler, 1984; Jacoby & Witherspoon, 1982; for review, see Shimamura, 1986).

These facts about amnesia have suggested that memory is organized in the brain as a set of dissociable processes or systems (Cohen, 1984; Mishkin, Malamut, & Bachevalier, 1984; Squire, 1986). The term *declarative memory* has been used to describe the memory system that is impaired in amnesia (Cohen, 1984; Squire, 1982). Declarative memory is available to conscious awareness and includes the facts and specific episodes learned in everyday experience. By contrast, *procedural memory* is implicit and is available only through performance. Skill learning, simple classical conditioning, and priming have been considered to be examples of procedural memory. Because procedural memory is spared in amnesic patients, it must depend on brain regions other than the medial temporal and diencephalic structures known to be affected in amnesia.

The relative status of procedural memory in Alzheimer's disease is not well understood. Alzheimer patients exhibited good performance on a perceptual-motor (pursuit rotor) skill task (Eslinger & Damasio, 1986); yet, in another study Alzheimer patients exhibited impaired performance on a cognitive (mirror-reading) skill task (Grober, 1985). Although loss of hippocampal function in Alzheimer's disease might explain deficits in declarative memory, this impairment alone could not explain deficits in procedural memory. Neuropathological studies have demonstrated damage to neocortex as well as damage to subcortical structures that provide cholinergic innervation to the forebrain (Pearson, Esiri, Hiorns, Wilcox, & Powell, 1985; Whitehouse et al., 1982), but it is not certain when this pathology occurs in the course of the disease. Thus it is not clear whether Alzheimer's disease *begins* as a selective impairment characteristic of amnesic syndromes or whether other kinds of memory processes are also affected at a relatively early stage of the disease.

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In this study, we investigated the phenomenon of lexical priming in a group of mild to moderately impaired patients with Alzheimer's disease. Priming is the facilitation or modification of behavior by recently encountered stimuli, and it is intact even in severely amnesic patients. Lexical priming can be easily demonstrated in the word completion test (Graf et al., 1984; Squire, Shimamura, & Graf, in press). Subjects are presented a list of words (e.g., MOTEL, ABSTAIN) and then are asked simply to say the first word that comes to mind in response to three-letter word beginnings or stems (e.g., MOT, ABS). This test differs from standard tests of memory in that testing is conducted implicitly; that is, subjects are not told to use the stems as memory cues, and they apparently treat the task as a word puzzle. For amnesic patients, words appear to pop into mind in response to three-letter cues, yet the words are not recognized as familiar.

We tested both word completion priming and verbal memory ability in patients with Alzheimer's disease, patients with Korsakoff's syndrome, and patients with Huntington's disease. Patients with Korsakoff's syndrome have previously been found to exhibit intact priming ability (Graf et al., 1984; Shimamura & Squire, 1984; Squire et al., in press). Huntington's disease prominently affects the basal ganglia and impairs both motor and cognitive functions (Bruyn, Bots, & Dom, 1979; Butters, Sax, Montgomery, & Tarlow, 1978; Caine, Ebert, & Weingartner, 1977; Josiassen, Curry, & Mancall, 1983). Although patients with Huntington's disease exhibit impaired skill (mirror reading) learning (Martone, Butters, Payne, Becker, & Sax, 1984), nothing is known about their priming ability.

Method

Subjects

Patients with Alzheimer's disease. We tested a group of 8 patients with a clinical diagnosis of mild to moderate Alzheimer's disease (see Table 1). Because a definite diagnosis of Alzheimer's disease is not possible without neuropathologic evidence, we used the clinical criteria developed by the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (McKann et al., 1984). Based on these criteria, a senior staff neurologist diagnosed five patients as probable Alzheimer's disease and three others as possible Alzheimer's disease. All patients scored at or above 104 out of a possible 144 points on the Dementia Rating Scale (DRS; average DRS = 118). The DRS is a test battery that assesses a spectrum of cognitive functions, including attention, memory, construction, and verbal fluency (Coblentz et al.,

1973). In addition, the patients averaged 11.9 errors out of a possible 33 errors on the Blessed scale (Blessed, Tomlinson, & Roth, 1968), and they averaged 20.4 correct out of a possible 30 points on the Mini-Mental State (Folstein, Folstein, & McHugh, 1975). Testing occurred on the average 3.2 years after the appearance of the first symptoms (range = 1–5 years).

Patients with Huntington's disease. We tested a group of 8 patients with Huntington's disease (see Table 1). Diagnosis of Huntington's disease was made on the basis of a positive family history and the presence of choreiform movements. Their functional capacity was assessed with the Shoulson and Fahn (1979) scale, which rates functional disability on a 5-point scale (1 = *minimal*, 5 = *total*). One of the patients was rated at Functional Stage 1, two at Stage 2, four at Stage 3, and one at Stage 4.

Patients with Korsakoff's syndrome. We tested 7 patients with Korsakoff's syndrome (see Table 1) who have been studied in previous investigations (Shimamura & Squire, 1986a, 1986b). A detailed assessment of memory functions for 6 of these patients can be found in Squire and Shimamura (1986). Neuropsychological screening and independent neurological examination indicated that memory impairment was the only notable deficit of higher cortical functions.

Control subjects. Each of the three patient groups was matched to its own control group with respect to age and education level (see Table 1). The control group for patients with Alzheimer's disease consisted of 9 elderly individuals who averaged 69.6 years of age, and the control group for patients with Huntington's disease consisted of 8 younger individuals who averaged 50.1 years of age. We tested a group of 6 alcoholic individuals as control subjects for the patients with Korsakoff's syndrome. The alcoholic subjects were current or former participants in alcoholic treatment programs in San Diego County. All had abstained from alcohol for an average of 28.6 months prior to testing (range = 1–81 months).

Procedure

We used a word completion test similar to those used in previous studies of word priming in amnesic patients (see Graf et al., 1984; Squire et al., in press). Subjects were asked to read 10 words (e.g., MOTEL, ABSTAIN) and to rate how much they liked each word on a 5-point scale (1 = *dislike extremely*, 5 = *like extremely*). Two additional filler words were placed at the beginning of the list and three at the end to reduce primacy and recency effects. In this way, words were presented for study without explicitly telling subjects to expect a memory test. Following a single presentation of the words, subjects were shown 20 three-letter word stems and were asked to complete each stem with the first word that came to mind (e.g., MOT, ABS). There were always at least 10 possible words that could be used to complete each target stem, only one of which was presented for the study. Ten of the stems could be completed using study words, and the other 10 stems were used to assess baseline guessing rates. The stems used to assess baseline rates were used as target stems for other subjects. The entire procedure was then repeated in exactly the same manner using a different list of 10 words. In this way, word completion was assessed twice, using two lists of 10 words.

In addition to the word completion test, we administered the Rey Auditory Verbal Learning Test (Lezak, 1983; Rey, 1964), which tests both recall and recognition memory. Subjects were given five study/test trials to learn a list of 15 words. In the recall version of the test, subjects were asked to recall as many words as possible immediately after each of the five word-list presentations. In the recognition test, we presented a different list of 15 words and tested subjects by presenting the 15 list words intermixed with 15 new words. After each of five list presentations, subjects were asked to say whether or not a word was just presented. Recognition performance was based

Table 1
Description of Patient and Control Groups

Patient group	N	Age	ED	DRS
Alzheimer's disease	8	72.0	13.8	118
Older controls	9	69.6	13.9	140
Huntington's disease	8	48.9	13.6	128
Younger controls	8	50.1	13.5	141
Korsakoff's syndrome	7	53.9	11.7	128
Alcoholic controls	6	51.7	12.8	140

Note. DRS = Dementia Rating Scale; ED = education.

on the percentage of correct responses out of 30 (i.e., correct hits + correct rejections).

Results

Figure 1 shows memory performance of patient and control groups on the Rey Auditory Verbal Learning Test. All three patient groups exhibited impaired verbal memory on both recall ($p < .01$) and recognition tests ($p < .01$). Patients with Alzheimer's disease and patients with Korsakoff's syndrome exhibited nearly the same level of impairment, $F(1, 12) < 1.0$, $p > .50$. Although patients with Huntington's disease exhibited impaired verbal memory, they performed significantly better than the other two patient groups on tests of both recall, $F(1, 13) = 5.6$, $p < .05$, and recognition, $F(1, 13) = 6.1$, $p < .05$.

In spite of the similarity in verbal memory performance between patients with Alzheimer's disease and patients with Korsakoff's syndrome, only patients with Alzheimer's disease exhibited impaired word completion priming (see Figure 2). In fact, the mean word completion score obtained by patients with Alzheimer's disease was significantly lower than the mean score obtained by any other group ($ps < .02$). By contrast, the mean completion scores obtained by patients with Korsakoff's syndrome and by patients with Huntington's disease were not significantly different from the mean scores obtained by their respective control groups: Korsakoff patients versus controls, $t(11) = 0.41$, $p = .69$; Huntington patients versus controls, $t(14) = 1.2$, $p = .25$. Thus all subject groups except patients with Alzheimer's disease increased their tendency to complete word stems to form previously presented

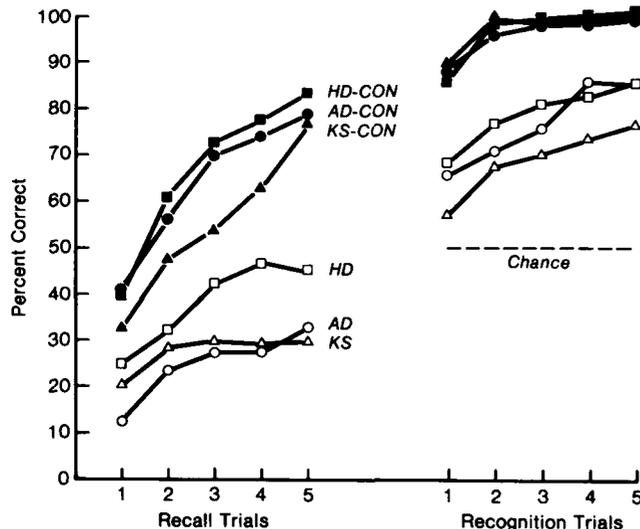


Figure 1. Recall (left) and recognition (right) memory performance across five study/test learning trials using the same 15 words on each trial. (A different set of 15 words was used for the recall and recognition trials. Figure shows performance by Alzheimer disease patients [AD; ○—○] and their controls [CON-AD; ●—●], Huntington disease patients [HD; □—□] and their controls [CON-HD; ■—■]; and Korsakoff syndrome patients [KS; △—△] and their alcoholic control subjects [CON-KS; ▲—▲].)

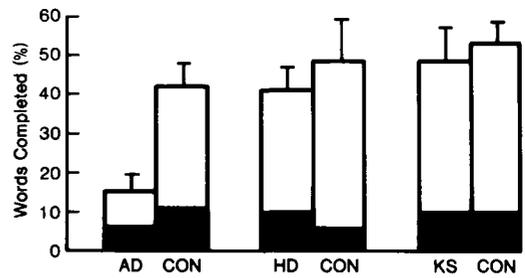


Figure 2. Lexical priming ability as measured by the word completion test. (Subjects were shown words [e.g., MOTEL] and later asked to complete three-letter word stems [e.g., MOT] with the first words that came to mind. Bars represent percentage of previously presented words that were used to complete word stems [total number of study words = 20]. Dark area represents baseline guessing performance, that is, the tendency to complete the same words under conditions when they had not been presented. Only Alzheimer disease patients [AD] exhibited impaired lexical priming. Figure shows AD patients, Huntington disease patients [HD], and Korsakoff syndrome patients [KS] adjacent to each of their respective control groups [CON].)

words by 30% to 40% above baseline (baseline = 5–11%). Alzheimer patients, however, increased their tendency to use previously presented words only 10% above baseline (baseline = 6%).

Although the ability to complete stems with previously presented words was impaired in Alzheimer patients, it is important to note that these patients were able to perform the basic task of completing word stems with words. Six patients with Alzheimer's disease completed all 20 stems with words, and the other 2 patients completed 18 of 20 stems. Thus these patients completed 98% of the stems with words—though usually not with a previously presented word.

The priming deficit seen in Alzheimer's disease was not simply related to overall dementia. Of the 8 patients with Huntington's disease, the 4 who obtained the lowest DRS scores had the same average DRS score as the Alzheimer patients (average DRS = 118 points). Yet, these 4 Huntington patients exhibited intact priming ability (44% completion, 10% baseline). Moreover, the 4 least-demented Alzheimer patients averaged 127.0 points on the DRS, which was only one point less than the average DRS scores of patients with Huntington's disease and Korsakoff's syndrome. Nevertheless, these 4 Alzheimer patients exhibited impaired priming ability (21% completion, 5% baseline).

Discussion

Explicit tests of verbal memory revealed a deficit in all three patient groups. In fact, patients with Alzheimer's disease and patients with Korsakoff's syndrome exhibited about the same level of verbal recall and recognition impairment. Nevertheless, only patients with Alzheimer's disease exhibited impaired priming ability. This is the first report of a deficit in word completion priming in any patient group. Because these findings were obtained in mild to moderately demented patients, they suggest that damage to brain regions in addition to those damaged in amnesia must occur at relatively early stages of the disease.

The deficit in priming may reflect an impairment in the ability to activate representations that store lexical memory. Accordingly, this deficit might account for problems in word finding and semantic memory that are prominent cognitive symptoms of Alzheimer's disease, particularly in later stages of the disease (Martin & Fedio, 1983; Ober, Dronkers, Koss, Delis, & Friedland, 1986; Schwartz, Marin, & Saffran, 1979; Warrington, 1974; Weingartner, Grafman, Boutelle, Kaye, & Martin, 1983). The finding of intact priming performance in patients with Huntington's disease suggests that the capacity for activation of representations in lexical memory does not depend on the brain areas damaged in this disease. The fact that a mirror reading skill was impaired in Huntington's disease (Martone et al., 1984) suggests that various forms of procedural memory can be dissociated from one another.

Several lines of evidence indicate that the word completion deficit in Alzheimer's disease reflects an impairment in memory activation rather than a global intellectual or cognitive impairment. First, patients with Alzheimer's disease were able to perform the task of completing word stems with words, so that poor performance could not be attributed simply to slowness or inability to comprehend instructions. Second, baseline guessing rates were normal in Alzheimer patients, which indicates that patients were not producing obscure or unusual words. Third, overall levels of dementia—within the range tested—were not related to impaired lexical priming. That is, even when subgroups of Huntington and Alzheimer patients were closely matched for degree of dementia, only the Alzheimer patients exhibited impaired priming.

The impairment in word priming exhibited by patients with Alzheimer's disease is robust. We have observed impaired lexical priming in a second independent sample of 13 other patients (Salmon, Shimamura, Butters, & Smith, 1987). In that study, instead of presenting words once, we presented words twice before testing for word completion. Nevertheless, the patients increased their tendency to use presented words only 15% above baseline (baseline = 11%), whereas control subjects increased their tendency to use presented words by 40% above baseline (baseline = 7%). Thus markedly impaired priming ability was observed altogether in 21 mild-to-moderately demented patients with Alzheimer's disease.

In summary, the most important finding was that patients with Alzheimer's disease exhibited impaired priming, whereas patients with Korsakoff's syndrome did not, despite the fact that these two patient groups exhibited a similar degree of impairment on explicit tests of recall and recognition memory. Patients with Korsakoff's syndrome, or patients with circumscribed medial temporal lobe damage, are impaired on explicit tests of memory, yet they exhibit intact priming. The impairment in priming must therefore reflect damage to regions other than the medial temporal and diencephalic brain structures affected in amnesia. Moreover, the finding of intact priming in patients with Huntington's disease suggests that the integrity of the neostriatum is not critical for normal priming effects. Impaired priming in Alzheimer's disease may be the result of damage to cortical representations that store lexical memory. If so, then involvement of neocortex must occur at a relatively early stage in the disease process, in conjunction with involvement of the structures responsible

for the deficit in verbal or declarative memory. Further study of this and other aspects of memory impairment may help in determining the usual trajectory of the disease and in identifying which brain systems are first affected.

References

- Arendt, T., Bigl, V., Arendt, A., & Tennstedt, A. (1983). Loss of neurons in the nucleus basalis of Meynert in Alzheimer's disease, paralysis agitans and Korsakoff's disease. *Acta Neuropathologica*, *61*, 101-108.
- Baddeley, A. D., & Warrington, E. K. (1970). Amnesia and the distinction between long- and short-term memory. *Journal of Verbal Learning and Verbal Behavior*, *9*, 176-189.
- Blessed, G., Tomlinson, B. E., & Roth, M. (1968). The association between quantitative measures of dementia and senile change in the cerebral gray matter of elderly subjects. *British Journal of Psychiatry*, *114*, 797-811.
- Brooks, D. N., & Baddeley, A. D. (1976). What can amnesic patients learn? *Neuropsychologia*, *14*, 111-122.
- Bruyn, G. W., Bots, G., & Dom, R. (1979). Huntington's chorea: Current neuropathological status. In T. Chase, N. Wexler, & A. Barbeau (Eds.), *Advances in neurology: Vol. 23. Huntington's disease*. Raven Press: New York.
- Butters, N. (1984). Alcoholic Korsakoff's syndrome: An update. *Seminars in Neurology*, *4*, 226-244.
- Butters, N., & Cermak, L. S. (1980). *Alcoholic Korsakoff's syndrome: An information processing approach*. New York: Academic Press.
- Butters, N., Sax, D. S., Montgomery, K., & Tarlow, S. (1978). Comparison of the neuropsychological deficits associated with early and advanced Huntington's disease. *Archives of Neurology*, *35*, 585-589.
- Caine, E., Ebert, M., & Weingartner, H. (1977). An outline for the analysis of dementia: The memory disorder of Huntington's disease. *Neurology*, *27*, 1087-1092.
- Coblentz, J. M., Mattis, S., Zingesser, L. H., Kasoff, S. S., Wisniewski, H. M., & Katzman, R. (1973). Presenile dementia: Clinical aspects and evaluation of cerebrospinal fluid dynamics. *Archives of Neurology*, *29*, 299-308.
- Cohen, N. J. (1984). Preserved learning capacity in amnesia: Evidence for multiple memory systems. In L. Squire & N. Butters (Eds.), *The neuropsychology of memory* (pp. 83-103). New York: Guilford Press.
- Cohen, N. J., & Squire, L. R. (1980). Preserved learning and retention of pattern analyzing skill in amnesia: Dissociation of knowing how and knowing that. *Science*, *210*, 207-209.
- Corkin, S., Davis, K. L., Growdon, J. H., Usdin, E., & Wurtman, R. J. (Eds.). (1982). *Alzheimer's disease: A report of research in progress* (Vol. 19). New York: Raven Press.
- Drachman, D. A., & Arbit, J. (1966). Memory and the hippocampal complex. *Archives of Neurology*, *15*, 52-61.
- Eslinger, P. J., & Damasio, A. R. (1986). Preserved motor learning in Alzheimer's disease: Implications for anatomy and behavior. *Journal of Neuroscience*, *6*, 3006-3009.
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). Mini-mental state. A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*, 189-198.
- Graf, P., Squire, L. R., & Mandler, G. (1984). The information that amnesic patients do not forget. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *10*, 164-178.
- Grober, E. (1985). Encoding of item-specific information in Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology*, *7*, 614.
- Henderson, A. S. (1986). The epidemiology of Alzheimer's disease. *British Medical Bulletin*, *42*, 3-10.

- Huppert, F. A., & Tym, E. (1986). Clinical and neuropsychological assessment of dementia. *British Medical Bulletin*, 42, 11-18.
- Hyman, B. T., Van Hoesen, G. W., Damasio, A. R., & Barnes, C. L. (1984). Alzheimer's disease: Cell-specific pathology isolates the hippocampal formation. *Science*, 225, 1168-1170.
- Jacoby, L. L., & Witherspoon, D. (1982). Remembering without awareness. *Canadian Journal of Psychology*, 32, 300-324.
- Josiassen, R., Curry, L., & Mancall, E. (1983). Development of neuropsychological deficits in Huntington's disease. *Archives of Neurology*, 40, 791-796.
- Lezak, M. D. (1983). *Neuropsychological assessment* (2nd ed.). New York: Oxford University Press.
- Mair, W. G. P., Warrington, E. K., & Weiskrantz, L. (1979). Memory disorder in Korsakoff's psychosis: A neuropathological and neuropsychological investigation of two cases. *Brain*, 102, 749-783.
- Martin, A., & Fedio, P. (1983). Word production and comprehension in Alzheimer's disease: The breakdown of semantic knowledge. *Brain and Language*, 19, 124-141.
- Martone, M., Butters, N., Payne, M., Becker, J. T., & Sax, D. S. (1984). Dissociations between skill learning and verbal recognition in amnesia and dementia. *Archives of Neurology*, 41, 965-970.
- McKann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, E. M. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. *Neurology*, 34, 939-944.
- Mishkin, M., Malamut, B., & Bachevalier, J. (1984). Memories and habits: Two neural systems. In J. L. McGaugh, G. Lynch, & N. Weinberger (Eds.), *The neurobiology of learning and memory* (pp. 65-77). New York: Guilford Press.
- Mohs, R. C., Greenwald, B. S., Dunn, D. D., & Davis, K. L. (1985). Assessment of cognition and affective symptoms in dementia. In J. Traber and W. H. Gispen (Eds.), *Senile dementia of the Alzheimer type*. Berlin: Springer-Verlag.
- Ober, B. A., Dronkers, N. F., Koss, E., Delis, D. C., & Friedland, R. P. (1986). Retrieval from semantic memory in Alzheimer-type dementia. *Journal of Clinical and Experimental Neuropsychology*, 8, 75-92.
- Pearson, R. C. A., Esiri, M. M., Hiorns, R. W., Wilcox, G. K., & Powell, T. P. S. (1985). Anatomical correlates of the distribution of the pathological changes in the neocortex in Alzheimer's disease. *Proceedings of the National Academy of Sciences*, 82, 4531-4534.
- Rey, A. (1964). *L'examen clinique en psychologie*. [The clinical psychology examination]. Paris: Presses Universitaires de France.
- Salmon, D. P., Shimamura, A. P., Butters, N., & Smith, S. (1987). *Lexical and semantic deficits in patients with Alzheimer's disease*. Manuscript submitted for publication.
- Schwartz, M. F., Marin, O. S. M., & Saffran, E. M. (1979). Dissociations of language function in dementia: A case study. *Brain and Language*, 7, 277-306.
- Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, and Psychiatry*, 20, 11-21.
- Shimamura, A. P. (1986). Priming effects in amnesia: Evidence for a dissociable memory function. *Quarterly Journal of Experimental Psychology*, 38A, 619-644.
- Shimamura, A. P., & Squire, L. R. (1984). Paired-associate learning and priming effects in amnesia: A neuropsychological study. *Journal of Experimental Psychology: General*, 113, 556-570.
- Shimamura, A. P., & Squire, L. R. (1986a). Memory and meta-memory: A study of the feeling-of-knowing phenomenon in amnesic patients. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 12, 452-460.
- Shimamura, A. P., & Squire, L. R. (1986b). Korsakoff's syndrome: The relation between anterograde amnesia and remote memory impairment. *Behavioral Neuroscience*, 100, 165-170.
- Shoulson, I., & Fahh, S. (1979). Huntington's disease: Clinical care and evaluation. *Neurology*, 29, 1-3.
- Squire, L. R. (1982). The neuropsychology of human memory. *Annual Review of Neuroscience*, 5, 241-273.
- Squire, L. R. (1986). Mechanisms of memory. *Science*, 232, 1612-1619.
- Squire, L. R., & Shimamura, A. P. (1986). Characterizing amnesic patients for neurobehavioral study. *Behavioral Neuroscience*, 100, 866-877.
- Squire, L. R., Shimamura, A. P., & Graf, P. (in press). Strength and duration of priming effects in normal subjects and amnesic patients. *Neuropsychologia*.
- Talland, G. A. (1965). *Deranged memory*. Academic Press: New York.
- Terry, R. D., & Davies, P. (1980). Dementia of the Alzheimer type. *Annual Review of Neuroscience*, 3, 77-95.
- Terry, R. D., & Katzman, R. (1983). *Annals of Neurology*, 14, 497.
- Warrington, E. K. (1974). The selective impairment of semantic memory. *Quarterly Journal of Experimental Psychology*, 27, 635-657.
- Weingartner, H., Grafman, J., Boutelle, W., Kaye, W., & Martin, P. R. (1983). Forms of memory failure. *Science*, 221, 380-382.
- Weingartner, H., Kaye, W., Smallberg, S. A., Ebert, M. H., Gillin, J. C., & Staram, N. (1981). Memory failure in progressive idiopathic dementia. *Journal of Abnormal Psychology*, 90, 187-196.
- Whitehouse, P. J., Price, D. L., Struble, R. G., Clark, A. W., & DeLong, M. R. (1982). Alzheimer's disease and senile dementia: Loss of neurons in the basal forebrain. *Science*, 215, 1237-1239.
- Zola-Morgan, S., Amaral, D. G., & Squire, L. R. (1986). Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *Journal of Neuroscience*, 6, 2950-2967.

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