

Susceptibility to Memory Interference Effects following Frontal Lobe Damage: Findings from Tests of Paired-Associate Learning

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Abstract

■ Patients with frontal lobe lesions were administered tests of paired-associate learning in which cue and response words are manipulated to increase interference across two study lists. In one test of paired-associate learning (AB-AC test), cue words used in one list are repeated in a second list but are associated with different response words (e.g., LION-HUNTER, LION-CIRCUS). In another test (AB-ABr test), words used in one list are repeated in a second list but are rearranged to form new pairs. Compared to control subjects, patients with frontal lobe lesions exhibited disproportionate impairment of second-list

learning as a result of interference effects. In particular, patients exhibited the poorest performance during the initial trial of the second list, a trial in which interference effects from the first list would be most apparent. These findings suggest that the on-line control of irrelevant or competing memory associations is disrupted following frontal lobe lesions. This disruption may be indicative of an impaired gating or filtering mechanism that affects not only memory function but other cognitive function as well. ■

INTRODUCTION

Characterizing the underlying cognitive function associated with the frontal lobes has been a rather elusive venture. Teuber, (1964) portrayed the problem as a "riddle," because neither sensory nor motoric functions by themselves could fully describe the contribution of the frontal lobes to behavior. More recently, cognitive neuroscientists have come to realize that the frontal lobes—more specifically, the prefrontal cortices—contribute to a wide array of cognitive functions, including aspects of attention, language, memory, and problem solving (for review, see Milner, Petrides, & Smith, 1985; Moscovitch, 1992; Shallice, 1982; Shimamura, Janowsky, & Squire, 1990; Stuss & Benson, 1986). This fact may not be very surprising, because the prefrontal region comprises about 28% of the cortical mantle in humans and is intricately connected to many other neocortical as well as subcortical regions (Fuster, 1989; Goldman-Rakic, 1987).

Classically, prefrontal function has been associated with planning, problem solving, and response inhibition (for historical review, see Benton, 1991; Luria, 1966; Stuss

& Benson, 1986). Deficits in these aspects of cognition can be observed on tests of card sorting (Milner, 1964), cognitive skill learning (Shallice & Evans, 1978), and motor programming (Luria, 1966). Also, deficits in the temporal organization of information have been attributed to frontal lobe lesions. Specifically, patients with frontal lobe lesions exhibit impairment in memory for the temporal order in which stimuli are presented (Milner, Corsi, & Leonard, 1991; Shimamura, Janowsky, & Squire, 1990). Recently, an assortment of memory deficits has been observed in patients with frontal lobe lesions. These deficits include problems in free recall (Incisa della Rocchetta, 1986; Gershberg & Shimamura, in press; Janowsky, Shimamura, Kritchevsky, & Squire, 1989; Jetter, Poser, Freeman, & Markowitsch, 1986), metamemory (Janowsky, Shimamura, & Squire, 1989a; Shallice & Evans, 1978), short-term memory (Janowsky et al., 1989), and source memory (Janowsky, Shimamura, & Squire, 1989b).

Whereas some have viewed the manifold array of disorders as an indication that the prefrontal cortex cannot be characterized by a single cognitive process or mechanism, others have emphasized the importance of

this region in working memory or executive control. Specifically, prefrontal cortex is thought to contribute significantly to the on-line encoding, manipulation, and organization of information processing (Baddeley, 1986). One function of working memory appears to be the active maintenance of memory representations (i.e., short-term memory) (Fuster, 1989; Goldman-Rakic, 1987; Jonides, Smith, Koeppe, Awh, Monoshima, & Mintun, 1993). Another aspect appears to be the gating of extraneous or irrelevant information processing (see Shimamura, 1994). For example, frontal lobe lesions appear to affect the ability to gate or inhibit irrelevant stimulus information (Knight, Scabini, & Woods, 1989; Luria, 1966). Findings of greater interference effects in the Stroop test by patients with frontal lobe lesions (Perret, 1974) also suggest that the frontal lobes may be involved in suppressing irrelevant information.

Increased susceptibility to interference may also occur in tasks that require the manipulation or retrieval of information in long-term memory. That is, patients with frontal lobe lesions may exhibit increased interference from extraneous memory associations. To study such interference effects, we analyzed performance on paired-associate learning paradigms commonly used in studies of verbal learning. In the tradition of verbal learning, numerous test paradigms and detailed theoretical interpretations have been developed in the service of understanding interference effects (for review, see Bjork, 1992; Crowder, 1976; Postman, 1961; Postman & Underwood, 1973). In the present investigation, we assessed memory interference by studying the degree to which prior learning affects later learning (i.e., proactive interference). We used two simplified versions of proactive interference paradigms. In Experiment 1, we assessed AB-AC paired-associate learning. In this test, cue words used in one list are used in a second list but are associated with different response words in a second list (e.g., LION-HUNTER, LION-CIRCUS). In Experiment 2, we assessed AB-ABr paired-associate learning, in which cue and response words used in one list are rearranged to form new pairs in a second list.

EXPERIMENT 1

Interference effects in AB-AC paired-associate learning were assessed in patients with unilateral frontal lobe lesions (see Fig. 1 and Table 1) and control subjects. Twelve moderately related word pairs (e.g., RIVER-POND) were presented for three consecutive study-test learning trials. Following these trials, an additional three learning trials were administered for word pairs with the same cue words but with different response words (e.g., RIVER-BROOK). To the extent that the prefrontal cortex acts to gate or filter interference from prior memory associations, patients with frontal lobe lesion should exhibit disproportionate impairment on second-list learning. Moreover, the most significant decrement in

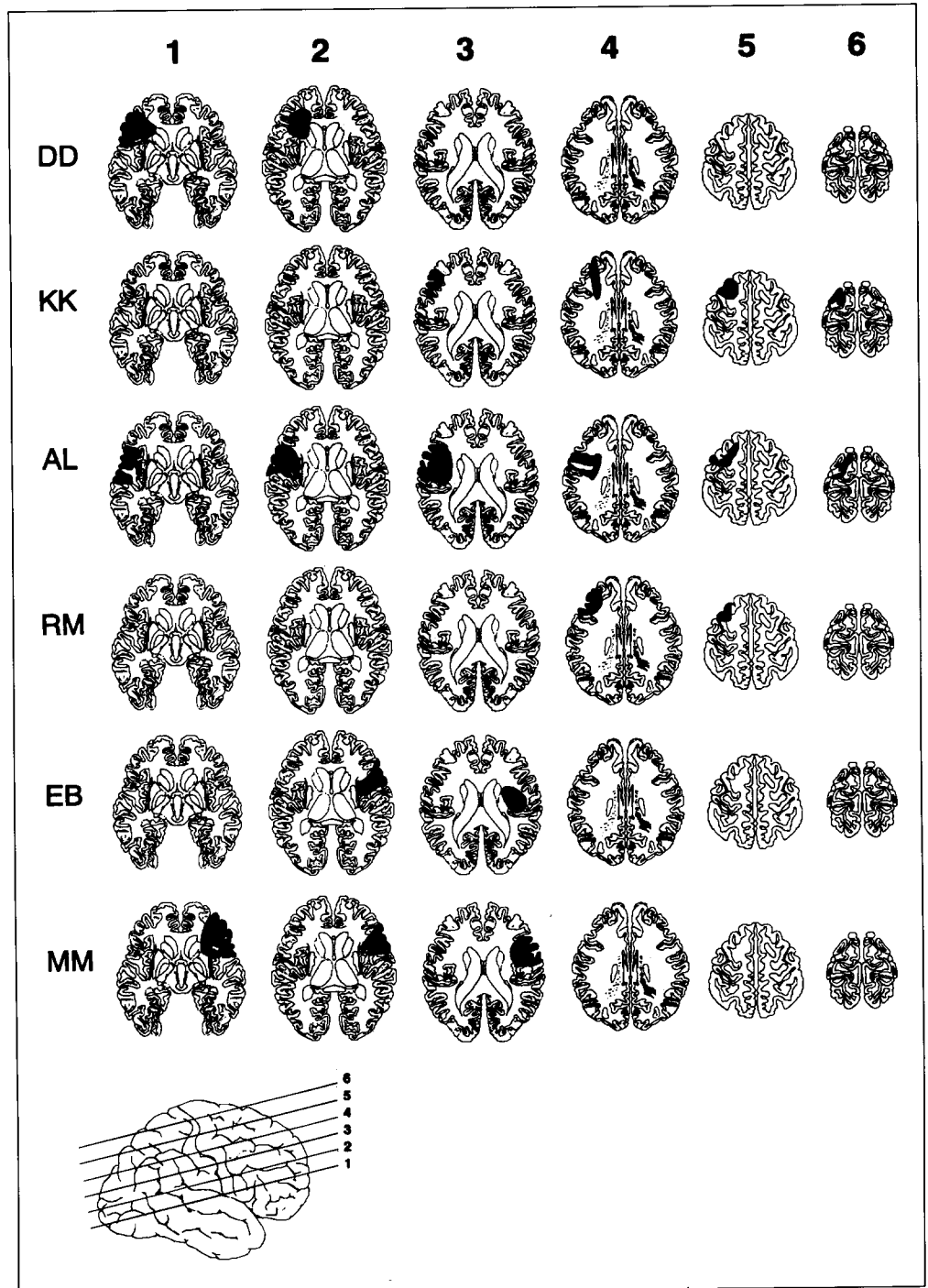
performance should occur on the first learning trial of the second list, as this trial should be most susceptible to interference from the first list. Finally, to determine if interference effects were due to problems in list discrimination, subjects were given a final cued recall test in which each cue word was presented and both of the response words were requested. If problems were simply due to a failure to remember which response words were associated with the first or second list, then subjects should be able to report both target words when list information is not required. This final cued recall test has been used in previous studies of paired-associate learning and has been called a modified-modified free recall (MMFR) test (Barnes & Underwood, 1959).

Results and Discussion

Figure 2 displays paired-associate learning by patients with frontal lobe lesions and control subjects. The data were subjected to a $2 \times 2 \times 3$ ANOVA, with subject group (frontals and controls), list (list 1 and list 2), and learning trial as variables. Significant main effects were observed for all three variables: subject group [$F(1,16) = 12.6$, $MSe = 44.5$, $p < 0.01$], list [$F(1,16) = 14.3$, $MSe = 17.8$, $p < 0.01$], and trial [$F(2,32) = 31.5$, $MSe = 41.4$, $p < 0.01$]. There was a significant subject \times list interaction, which occurred as a result of the disproportionately poor performance on the second list by patients with frontal lobe lesions [$F(1,16) = 10.8$, $MSe = 13.5$, $p < 0.01$]. No other effects were statistically significant ($p > 0.05$). It was hypothesized that a particular impairment would occur on the first trial of the second list, because it is that trial that should be most affected by interference from memory associations acquired during first-list learning. Indeed, a significant subject \times list interaction was observed in this analysis [$F(1,16) = 6.8$, $MSe = 6.7$, $p < 0.01$].

Patients with frontal lobe lesions also exhibited memory impairment on the final test of recall (MMFR test) in which cue words were presented again and both response words were requested (see Table 2). A 2×2 ANOVA with subject group and list as variables revealed a significant difference between patients and control subjects [$F(1,16) = 6.3$, $MSe = 24.5$, $p < 0.05$]. There was also a significant effect of list [$F(1,16) = 6.9$, $MSe = 9.4$, $p < 0.01$], which indicated that recall of list 1 target words was poorer than recall of list 2 words. The group \times list interaction was not significant [$F(1,16) = 3.3$, $MSe = 4.5$, $p = 0.08$], though ceiling effects prevent strong conclusions in this analysis. The results from this final recall test suggest that the impairment in paired-associate learning observed in patients with frontal lobe lesions extends beyond a problem of list discrimination. For example, if the problem was simply due to a failure to determine if a remembered response word came from the first list or the second list, the patients may have been able to recall as many words as control subjects

Figure 1. Computerized reconstructions of CT or MRI scans for six patients with frontal lobe lesions. Black areas represent site of lesion on transverse sections. A lateral view shown at bottom illustrates the level and orientation of each section from the most ventral section (1) to the most dorsal section (6).



when asked to report both response words irrespective of list order. The impairment in retrieving response words in the final recall test indicates that list discrimination cannot completely account for the heightened susceptibility to memory interference observed in the patients.

Another method by which memory interference effects can be assessed is by analysis of intrusion errors during second-list learning. Intrusion errors were scored if a subject used a response from the first list during second-list learning. Patients with frontal lobe lesions

made significantly more intrusion errors than control subjects [1.67 vs 0.17 intrusion errors; $t(16) = 2.62, p < 0.05$]. Even when errors are adjusted for total number of errors, patients with frontal lobe lesions exhibited a greater proportion of intrusion errors than control subjects (25 vs 9% intrusion errors compared to total number of errors). This finding suggests that second-list learning by patients with frontal lobe lesions may have been hampered by memory associations formed during first-list learning.

The disproportionate impairment in second-list learn-

ing by patients with frontal lobe lesions suggests that these patients had particular problems in overcoming memory interference effects. In other words, the patients appeared to exhibit increased sensitivity to proactive interference. Importantly, this disproportionate impairment occurred even when statistical analyses were restricted to performance for the first learning trial of each list. This finding is important, because differences in learning on the first trial of each list could not be attributed to ceiling effects that were observed in control subjects on second and third learning trials.

EXPERIMENT 2

To extend the findings of the first experiment, we explored memory interference effects in a different paired-associate learning paradigm. We used an AB-ABr learning paradigm in which cue and target words in the first list

are recombined to form new pairs for the second list. Learning in this paradigm is more difficult than in the AB-AC paradigm for two reasons. First, task difficulty is increased by using unrelated word pairs instead of related word pairs. Second, interference is established by unpaired repetitions of both cue and response words during second-list learning. Specifically, words learned as pairs in the first list are separated and rearranged to form new cue-response word pairings in the second list. Thus, performance on the second list requires subjects to inhibit or disregard previously learned cue-response associations.

Results and Discussion

Figure 3 displays AB-ABr paired-associate learning by patients with frontal lobe lesions and control subjects. Although the pattern of performance was generally the

Table 1. Patient Characteristics and Standardized Test Scores

Patient	Lesion		Age (years)	Education (years)	WAIS-R ^a FSIQ	WMS-R ^b General
	Side	Volume (cm ³)				
JD	Left	30.8	66	20	93	87
KK	Left	17.4	62	13	107	100
AL	Left	51.2	64	13	104	109
RM	Left	10.3	68	12	88	90
EB	Right	17.3	75	12	107	91
MM	Right	52.1	68	12	94	93
Group mean		29.8	67.2	13.7	98.8	95.0

^a Wechsler Adult Intelligence Scale—Revised, Full Scale IQ.

^b Wechsler Memory Scale—Revised, General Memory Index.

Figure 2. AB-AC paired-associate learning in patients with frontal lobe lesions (Fron) and control subjects (Con). Subjects were administered three study-test trials of list 1 followed by three study-test trials of list 2.

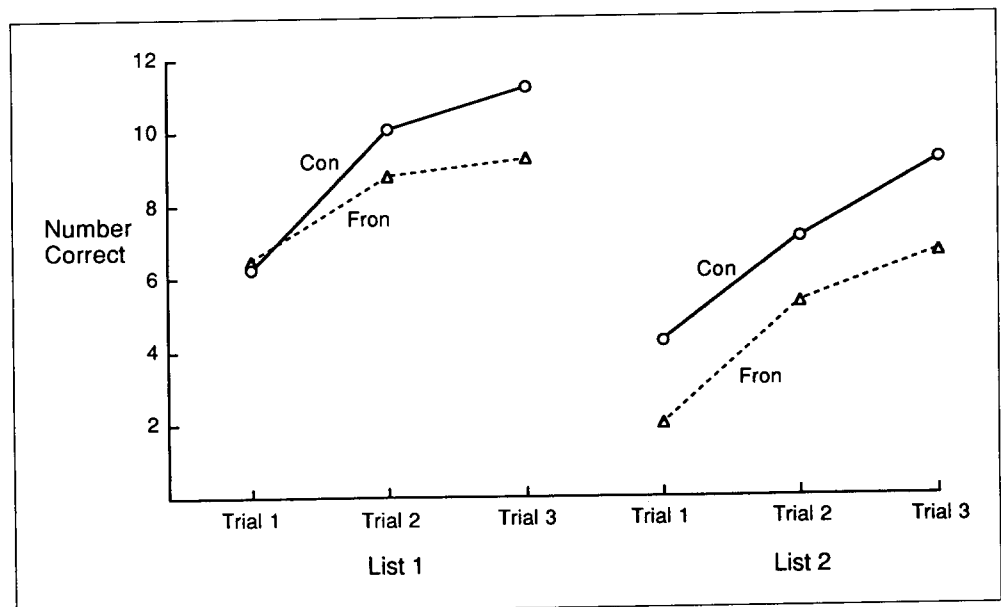
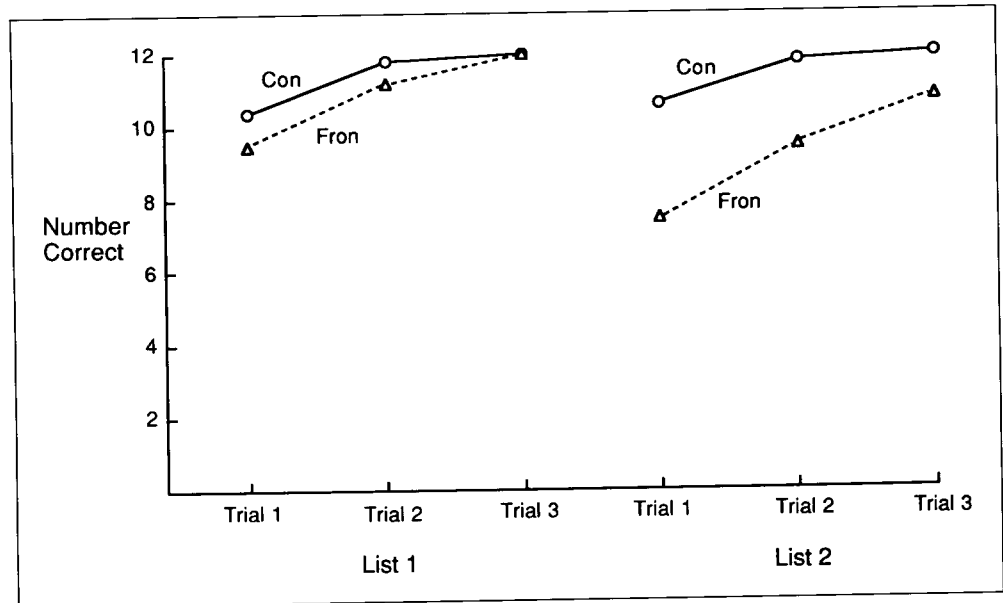


Figure 3. AB-ABr paired-associate learning in patients with frontal lobe lesions (Fron) and control subjects (Con). As in Experiment 1, subjects were administered three study-test trials of list 1 followed by three study-test trials of list 2.



same as that observed in Experiment 1, significant main effects were observed only for the variables of list [$F(1,16) = 35.8$, $MSe = 208$, $p < 0.001$] and trial [$F(2,32) = 41.0$, $MSe = 162$, $p < 0.001$]. Neither the effect of subject group [$F(1,16) = 1.7$, $MSe = 60$, $p < 0.05$] nor the group \times list interaction [$F(1,16) = 1.5$, $MSe = 9.0$, $p > 0.05$] was significant. A separate analysis of performance on the first trial of each list did reveal a significant group \times list interaction [$F(1,16) = 6.5$, $MSe = 12.58$, $p = 0.02$]. That is, the patients with frontal lobe lesions performed on the first trial of list 1 nearly as well as control subjects, but they performed significantly more poorly on the first trial of list 2. As mentioned before, it was expected that memory interference effects should be greatest on the first trial of list 2, because this trial would be expected to have the greatest degree of proactive interference effects from memory associations formed in list 1.

In this experiment, patients with frontal lobe lesions did not produce more intrusion errors on second-list learning than control subjects. Specifically, patients with frontal lobe lesions averaged 3.67 intrusion errors (19% of total errors), whereas control subjects averaged 3.58 intrusion errors (21% of total errors). Performance on the final cued recall test is shown in Table 2. Analyses of this test did not reveal statistically significant effects. In particular, the group effect and the group \times list effect were not significant ($F_s 2.3$, $p_s > 0.14$). Although these effects did not approach statistical significance, it is interesting to note that the pattern of performance on this test was *not* similar to that observed in Experiment 1. In Experiment 1, the patients with frontal lobe lesions were less able to recall words presented on the first list compared to words presented on the second list. In Experiment 2, however, the patients appeared to be less able

Table 2. Number of Correct Responses in Final Cued Recall Test

	List 1	List 2
Experiment 1		
Control subjects	11.7	12.0
Frontal patients	9.2	11.0
Experiment 2		
Control subjects	8.3	8.5
Frontal patients	7.5	5.7

to recall words from the second list compared to the recall of words presented on the first list.

GENERAL DISCUSSION

Patients with frontal lobe lesions exhibited heightened memory interference effects during paired-associate learning. In the AB-AC paradigm used in Experiment 1, performance on a second list of paired-associates was disrupted by prior learning of related pairs. In addition, patients with frontal lobe lesions committed more intrusion errors than control subjects during second-list learning. That is, patients often used word associates presented in the first list as responses during the learning of a second list. The failure to gate or inhibit first-list associations suggests that prior memory associations were actively interfering with later learning. Additional interference effects were observed on the final cued recall test. On this test, subjects were presented the cue words again and asked to produce both first- and second-list response words. The patients recalled fewer

words from the first list than words from the second list. Apparently, following three study-test trials of second-list learning, some retroactive interference effects occurred such that memory for first-list word pairs was significantly disrupted by later learning.

In Experiment 2, an AB-ABr paradigm significantly disrupted performance in both patients and control subjects. That is, second-list learning was significantly affected by rearranging cue and response words presented in the first list. Although the general pattern of results in this experiment was similar to that observed in Experiment 1, overall group differences in this experiment were not reliable. Several factors may have mitigated significant group differences. First, the control subjects exhibited large proactive interference effects in this paradigm. Previous findings have shown that older adults are disproportionately affected by interference in these paradigms (for review, see Kausler, 1991). The fact that significant frontal lobe atrophy occurs during the normal aging process (Haug, Barmwater, Eggers, Fischer, Kuhl, & Sass, 1983) suggests that mild frontal dysfunction in the control subjects may have reduced the likelihood of a reliable difference between patients and control subjects. Another factor that may have affected the results is that first-list learning in the patients did not appear to be as good as that observed in the control subjects (see Fig. 3). Poor initial learning could have reduced the interfering effect that first-list associations would have on second-list learning. Despite these factors, significant memory interference effects in patients with frontal lobe lesions were observed on the first learning trial of the second list. That is, performance by the patients on this first trial was significantly disrupted compared to performance on the first trial of list 1. Indeed, the groups performed equivalently on the first trial of list 1, whereas a significant group difference was observed on the first trial of list 2.

These findings extend the array of memory disorders observed in patients with frontal lobe lesions. Previous studies have shown that despite normal performance on standard neuropsychological tests of new learning capacity, patients with frontal lobe lesions exhibit impairment on tests of free recall, memory for temporal order, source memory, and metamemory (for review, see Shimamura et al., 1990). The present study demonstrates that heightened interference effects on tests of paired-associate learning can be added to this list of memory disorders. Indeed, it may be that heightened interference from prior memory associations is the underlying cause of impairment observed on many memory tests that are sensitive to frontal lobe damage. One similarity among these tests is the heavy reliance on organizational factors. For example, free recall, memory for temporal order, and source memory depend heavily on retrieval of the spatiotemporal context or episode in which information was presented. Metamemory—that is, the evaluation of

what knowledge is stored in memory—depends heavily on retrieval of information embedded within a semantic or knowledge-based context. Heightened interference from recently activated memory associations would likely disrupt retrieval processes, because there would be too much noise or too many aberrant associations that would interfere with access to desired information. Thus, the failure to inhibit competing memory associations may be a prominent factor in memory disorders associated with frontal lobe damage.

The interpretation that memory disorders arise from heightened interference from prior learning has been used in the past to explain memory disorders associated with amnesia (Warrington & Weiskrantz, 1974; Winocur & Weiskrantz, 1976). In particular, patients with Korsakoff's syndrome commit intrusion errors and are particularly disrupted on tests of AB-AC paired-associate learning (Winocur & Weiskrantz, 1976). However, further investigations failed to substantiate a "response competition" explanation of amnesia (Warrington & Weiskrantz, 1982). In particular, amnesic patients did not always exhibit increased interference when competition among possible alternatives was increased. Interestingly, all of these studies relied primarily on the analysis of patients with Korsakoff's syndrome. Neuroimaging findings have demonstrated that patients with Korsakoff's syndrome exhibit structural or functional (e.g., reduced glucose metabolic uptake) damage in the frontal lobes in addition to damage in the diencephalic midline (Jacobson & Lishman, 1987; Paller et al., 1993; Shimamura, Jernigan, & Squire, 1988; Squire, Amaral, & Press, 1990). Thus, frontal lobe involvement in patients with Korsakoff's syndrome may have contributed to impaired memory performance in the form of increasing proactive interference. However, the general findings from studies of amnesia suggest that the memory function associated with medial temporal and diencephalic areas involves the long-term binding, storage, or consolidation of new information rather than the mitigation of response competition (Mishkin, 1982; Squire, 1992; Warrington & Weiskrantz, 1982).

In terms of frontal lobe function, the on-line control of irrelevant or competing memory associations may be only one example of a general gating or filtering mechanism associated with this brain region. Shimamura (1994) suggested that disruption of such control processes may underlie many of the cognitive disorders associated with frontal lobe lesions. For example, attentional deficits, such as those observed on the Stroop test (Perret, 1974), may be attributed to the failure to inhibit or filter irrelevant perceptual information. Disorders of card sorting and problem solving (Milner et al., 1964; Shallice, 1982) may be attributed to the failure to inhibit irrelevant decisions or plans. Even basic sensory processes appear to be disrupted by frontal lobe lesions. Knight and colleagues (Knight et al., 1989; Yamaguchi &

Knight, 1990) demonstrated that middle latency-evoked potentials, which are generated in primary sensory cortices, were *potentiated* as a result of frontal lobe lesions. Thus, there appeared to be a *disinhibition* of posterior cortical activity as a result of frontal lobe lesions. Such aberrant activation may disrupt information processing by increasing noise levels. Based on this view, a variety of memory and cognitive disorders would occur as a result of frontal lobe lesions, not because different areas of the frontal lobe are serving different functions, but because different areas are controlling different posterior cortical regions, which themselves serve different cognitive function. The present findings indicate that the control of memory functions can be added to other aspects of control that appear to involve the frontal lobes.

EXPERIMENT 1 METHOD

Subjects

Patient with Frontal Lobe Lesions

Patients with dorsolateral prefrontal lesions were first identified by review of medical records and neuroimaging data [computed tomography (CT) or magnetic resonance (MR) scans]. Based on this review and on subsequent neurological examination, we included only patients with cardiovascular etiology—specifically, infarctions due to occlusion of the precentral branch of the middle cerebral artery. In addition, we excluded patients with evidence of multiple cerebral infarctions or other significant medical disease, such as psychiatric disorder, dementia, substance abuse, or another neurological event (e.g., head injury). To reduce the possibility that memory disorders were affected by severe language disorders, patients were excluded if they scored below 85 on the Western Aphasia Battery (Kertesz, 1982). Based on quantitative analyses of CT or MR data from the six study patients, we estimated the average size of lesions to be 29.8 cm³ (see Fig. 1 and Table 1). Two of the patients had right hemisphere lesions and four had left hemisphere lesions. They averaged 67.2 years of age and 13.7 years of education.

The patients scored within the normal range on the Full-Scale Wechsler Adult Intelligence Scale—Revised (WAIS-R) (mean = 98.8). Their mean scores on the five scales of the Wechsler Memory Scale—Revised (WMS-R) were as follows: Attention = 92.2, General Memory = 98.6, Delayed Memory = 86.9, Verbal Memory = 99.0, Visual Memory = 98.3. The patients exhibited deficits on the Wisconsin Card Sorting Test, achieving an average of 3.4 correct categories out of a total of six categories and an average of 36.3 perseverative errors. The patients also performed poorly on the FAS Verbal Fluency Test (1), averaging 17.9 words produced. Specifically, one patient with a left hemisphere lesion and another with a

right hemisphere lesion were mildly dysfluent (FAS score < 15).

Control Subjects

Twelve healthy, normal individuals (nine men and three women) participated as control subjects. These individuals were volunteers at the Martinez VA Outpatient Clinic and were matched to the patients with frontal lobe lesions with respect to age (64.7 years) and education (14.6 years). The control subjects scored similarly to the patients with frontal lobe lesions on the Information test (control subjects = 23.9; frontal patients = 18.0). However, the patients performed less well on the Vocabulary (control subjects = 55.9; frontal patients = 36.2) and Digit Symbol (control subjects = 46.9; frontal patients = 36.2) subtests of the WAIS-R and on the Digit Span subtest of the WMS-R (control subjects = 8.6; frontal patients = 6.2).

Stimuli and Design

The stimuli consisted of two lists of 12 moderately related paired-associates (e.g., RIVER-POND, LION-HUNTER). Based on norms the average associative ranking of the response words to the cue words was 26.5 (Jenkins, 1970). Based on word frequency norms, the words averaged 88.6 per million in the corpus of English usage (Kucera & Francis, 1967). Across the two lists, the cue words (i.e., first word in each pair) were the same, whereas the response words (i.e., second word in each pair) were different (e.g., RIVER-POND, RIVER-BROOK). This design conforms to typical AB-AC paired-associate learning paradigms. The stimuli were printed on 4 × 6 inch index cards using Helvetica 24-point type font.

Procedure

Three study-test trials of each paired-associate list were administered. In the study phase, subjects were shown word pairs and instructed to remember the words as pairs because later they would be asked to report the second word when presented with the first. A sample word pair was given for practice. Each word pair was presented for 4 sec, and subjects were asked to read the words aloud. In the test phase, subjects were shown index cards on which only the cue word was presented and were asked to report the word that was associated with each cue word. Subjects were encouraged to guess if a response could not be retrieved. After the first study-test trial, two additional study-test trials were administered in the same manner as the first. To reduce short-term memory effects, the last word pair presented for study was never tested during the first two test trials.

Otherwise, study words and test cues were presented in a different random order for each trial.

Following the three study-test trials for the first list, a second set of three study-test trials was administered. Subjects were told explicitly that the second list would involve the same cue words but different test words. Otherwise, the instructions and procedure for the second list were the same as those used for the first list. Across subjects, list presentation order of the word-pair associates (RIVER-POND vs RIVER-BROOK) was counterbalanced. After both sets of study-test trials were administered, subjects were presented all 12 cue words again and asked to report the two words that had been associated with each cue word. Subjects were encouraged to guess if both target words could not be recalled. Thus, in this final cued recall test, subjects were encouraged to provide two responses for each cue word.

EXPERIMENT 2 METHOD

Subjects

The same six patients with frontal lobe lesions studied in Experiment 1 were tested in Experiment 2 (see Fig. 1 and Table 1). Also, the same 12 healthy, normal individuals who participated in Experiment 1 served as control subjects in this experiment. For each subject, test sessions for Experiments 1 and 2 were separated by at least 12 weeks.

Stimuli and Design

The stimuli consisted of two lists of 12 unrelated word pairs (e.g., OFFICER-BIRD, RAILROAD-SHOES, KING-PAPER). Based on word frequency norms, the words averaged 166.1 per million in the corpus of English usage (Kucera & Francis, 1982). Stimuli were printed on 5 × 8 inch index cards using Helvetica 36-point font type. Memory interference during second list learning was established by rearranging cue-response word pairs presented in the first list (e.g., OFFICER-SHOES, RAILROAD-PAPER). This design conforms to standard AB-ABr paired-associate learning paradigms.

Procedure

The procedure for administration of the paired-associate learning tests was similar to that used in Experiment 1. Briefly, subjects were given three study-test trials of one list and an additional three study-test trials of another list. Prior to the presentation of the second list, subjects were told explicitly that the second list would include the same cue words, but the target words would be rearranged to form new pairs. After both blocks of study-test trials were administered, a final cued recall test was administered. In this test, subjects were presented the 12

cue words again and were asked to recall the two words that had been associated with each cue word.

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