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Memory performance on the California Verbal Learning Test–II: Findings from patients with focal frontal lesions

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Abstract

Numerous studies have suggested that frontal cortex plays a strategic, rather than an absolute, role in memory performance. Typically, frontal patients are reported to have impaired recall but normal recognition memory. A recent meta-analysis, however, has questioned this conclusion. To further investigate the role of frontal cortex in long-term memory, patients with focal frontal lesions and age- and education-matched controls were tested on a new version of the California Verbal Learning Test (CVLT–II). Frontal patients exhibited a number of deficits on this test, including overall poorer recall, an increased tendency to make intrusions, reduced semantic clustering, and impaired yes/no recognition performance. Further analysis of the error rates in the yes/no recognition task revealed that frontal patients were most likely to mistakenly endorse 2 types of distractors: semantically related words and words from an interference list. These findings are discussed with respect to the role of frontal dysfunction in false recollections and poor source memory, as well as the distinction between the roles of frontal and temporal cortex in long-term memory. (*JINS*, 2002, *8*, 539–546.)

Keywords: Frontal cortex, Long-term memory, Free recall, Source memory

INTRODUCTION

A number of studies have suggested that frontal cortex plays a role in long-term memory (Jetter et al., 1986; Mangels et al., 1996). Evidence for this idea comes in part from studies of free recall in patients with focal frontal lesions (Gershberg, 1997; Gershberg & Shimamura, 1995; Incisa della Rocchetta & Milner, 1993; Vogel et al., 1987). Generally, such patients are not characterized as amnesic, because their recognition is relatively intact. However, a recent meta-analysis (Wheeler et al., 1995) reviewed a large number of studies that assessed recognition and recall performance in patients with frontal pathology. Most of the individual studies included in this meta-analysis failed to find significant differences between frontal patients and controls on recognition measures. However, as a group of studies, there was a clear indication that even recognition memory is impaired in frontal lobe patients. This meta-analysis revealed that patients with circumscribed frontal lesions show significant impairments not just on free recall, but also on recognition and cued recall, though to a lesser degree.

A number of studies have shown that frontal lobe patients exhibit deficits on tasks such as free recall, in part due to a failure to utilize strategies and cues (Gershberg & Shimamura, 1995; Hirst & Volpe, 1988; Stuss et al., 1994). Gershberg and Shimamura found that patients with unilateral, focal frontal lesions were impaired on free recall tasks but that their performance improved with strategy instruction. Interestingly, such instruction improved performance when given at both study and recall, suggesting that frontal lesions impair both encoding and retrieval processes by way of their impact on strategy formation. Similarly, Incisa della Rocchetta and Milner (1993) reported that both left medial temporal lobe and left frontal lobe patients exhibited impaired free recall performance. However, when given strategies at encoding and retrieval, the frontal lobe patients

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performed normally, whereas the temporal lobe patients were still impaired. Such a dissociation between patient groups suggests that frontal cortex plays a strategic role in memory, whereas the medial temporal lobe plays a more absolute role in memory formation/consolidation.

One strategic aspect of memory performance is the tendency to utilize semantic information inherent in list material. For example, when given a list of words made up of different categories (e.g., animals, tools, furniture, etc.), normal individuals will often note this relationship and make use of it in their encoding and recall of the material (Delis et al., 1987). Patients with focal frontal lesions, however, are impaired at this ability. Hirst and Volpe (1988) showed that while frontal patients performed comparably to controls on free recall for an unrelated word list, patients were impaired on free recall for a categorizable list. On a subsequent task in their study, frontal patients were able to make use of the categories when they were told explicitly about organizing the words into semantic categories. Similarly, Gershberg and Shimamura (1995) found that frontal patients exhibited reduced semantic clustering, compared to normal controls on a series of free recall tasks. That is, frontal patients failed to effectively utilize the semantic organization present in the word lists. Such a failure to utilize a clustering strategy has also been noted in frontal lobe patients on other types of tasks, such as verbal fluency (Troyer et al., 1998).

It has been hypothesized that the use of memory strategies such as semantic organization relies on working memory and attention, which are affected in frontal lobe patients (Baldo & Shimamura, 2000; Chao & Knight, 1996; Ptito et al., 1995; Shimamura et al., 1991). For example, Incisa della Rocchetta and Milner (1993) showed that interference during the delay period of a recall task (in the form of part-list cueing) disrupted performance in left frontal patients but not in medial temporal lobe patients. Also, frontal lobe patients demonstrate increased interference on AB–AC paired-associate learning (Shimamura et al., 1995) and under certain conditions, demonstrate cross-list interference on free recall (Gershberg & Shimamura, 1995). In a similar vein, Vogel et al. (1987) found that aids that increased depth of processing improved recall performance in frontal patients but did so to a lesser extent in diencephalic and medial temporal patients. Again, these data suggest that frontal cortex is critical for attentional and strategic aspects of memory formation.

PRESENT STUDY

A new version of the California Verbal Learning Test (CVLT) has recently been developed (CVLT–II, Delis et al., 2000), and the present study reports on the use of this test with focal frontal patients. The CVLT–II differs from the original CVLT in that new word stimuli are used, new norms were collected, and there is a forced choice recognition procedure at the end (see *CVLT–II Manual* for other optional procedures and modifications; Delis et al., 2000). The aims of this study were (1) to further elucidate the role of frontal cortex in long-term memory (and in particular, its role in recognition memory) and (2) to provide normative data from a group of focal frontal lesion patients.

The CVLT (Delis et al., 1987) has been used extensively in clinical neuropsychology to assess for a number of different aspects of long-term memory and executive functioning (Delis et al., 1991). The test is constructed in a similar way to experimental studies of free recall described above. The new CVLT–II was designed in part to better assess frontal contributions to memory performance by incorporating new measures and analyses. To our knowledge, there has been no published report of focal frontal patient performance on either the CVLT or CVLT–II. Given that many clinicians describe performance on the CVLT as being "frontal," it seemed important to actually document performance on this test in a group of focal frontal patients.

METHODS

Research Participants

Eleven patients (7 men and 4 women) with focal frontal lobe lesions were recruited for this study based on review of computed tomography (CT) and magnetic resonance imaging (MRI) scans. Patients with lesions extending into non-frontal regions were excluded. One patient's lesion (M.K.) may have affected a very small strip of superior temporal cortex, although this was difficult to discern from scan review. Five patients had focal left lateral prefrontal cortex lesions, 5 had focal right lateral prefrontal cortex lesions, and 1 patient had a left, orbital frontal lesion (see Figure 1 for lesion reconstructions). In 6 of the patients, lesions were due to an infarct of the anterior branch of the middle cerebral artery. In the other patients, lesions were due to surgical treatment for an aneurysm, arterial-venous malformation, cyst, or meningioma. The average time since onset of injury was 9.9 years (range 1–17 years), and patients had an average lesion volume of 41.2 cc (range 12.9–200.4 cc; see Table 1). All patients were in the normal range on the Western Aphasia Battery (Kertesz, 1982). One patient with moderate aphasia was originally tested but then excluded due to his verbal output impairment. All patients were right-handed, except for 1 patient (C.L.) who was ambidextrous.

The control group was 11 healthy controls (8 men and 3 women) recruited from the same community as the patients. Controls were all right-handed. Patients and controls did not differ in terms of age [$M \pm SE = 63.82 \pm 3.90$ and 68.09 ± 1.82, respectively; $F(1,20) = .99$, $p = .33$] or years of education [$M \pm SE = 14.27 \pm .70$ and $14.64 \pm .69$, respectively; $F(1,20) = .14$, $p = .72$]. All testing was conducted at the Veterans Affairs Northern California Health Care System in Martinez, CA. Patients and controls were screened for history of dementia, drug abuse, and psychiatric illness, and controls were additionally screened for prior

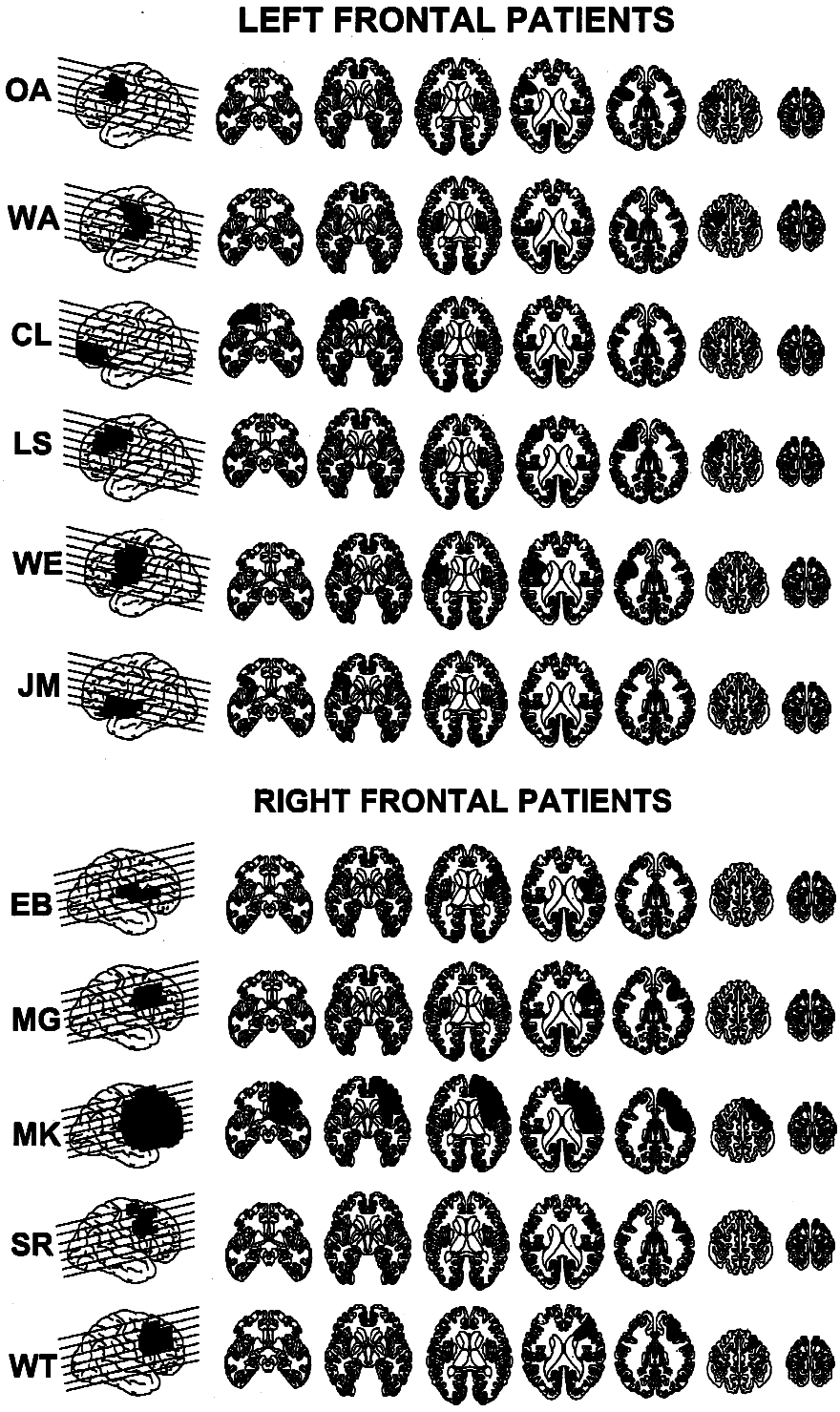

Fig. 1. Reconstructions of patients' lesions based on CT and MRI scans.

neurologic history. All participants read and signed consent forms prior to participating in the study.

Materials

The word lists used in the CVLT–II were made up of 16 words from four different categories. In List A, there were four animals, four vegetables, four ways of traveling, and four pieces of furniture. In List B, there were four animals, four vegetables, four instruments, and four parts of a house. Two of the four categories (animals and vegetables) were common to both lists.

In the long-delay yes/no recognition task, 48 words were presented: 16 target words from List A, 16 distractors from List B (eight of which were from the same categories as A—animals and vegetables), eight novel distractors from

Table 1. Participant characterization

Patients	Gender	Lesion site	Volume (cc)	Age at test	Years post	Lesion etiology	Educ. (years)	WAIS–R PIQ
O.A.	M	L	17.5	65	13	Stroke	14	134
E.B.	F	R	17.3	79	14	Stroke	12	114
S.R.	F	R	12.9	77	2	Stroke	12	93
W.A.	F	L	26.2	75	11	Stroke	14	132
W.T.	M	R	25.9	53	9	Cyst	18	—
M.G.	M	R	24.5	34	13	AVM	12	—
M.K.	M	R	200.4	66	17	Aneurysm	17	—
L.S.	F	L	27.9	68	16	Meningioma	16	—
W.E.	M	L	41.1	69	2	Stroke	15	104
J.M.	M	L	18.8	54	1	Stroke	11	91
C.L.	M	L Orb	40.3	63	10	Meningioma	16	—
					Means			
Frontal	7M,4F	6L,5R	41.2	63.8	9.9	—	14.3	113.6
Control	8M,3F	—	—	68.1	—	—	14.6	—

Note. Dashes indicate data that are not applicable or were not obtained due to patient availability. L = left hemisphere; R = right hemisphere; Orb = orbital; AVM = arterio-venous malformation; Educ. = education; WAIS–R PIQ = Wechsler Adult Intelligence Scale–Revised Performance IQ.

the same four categories as List A, and eight novel distractors that were unrelated to any categories from List A or B.

In the long-delay forced choice recognition task, there were 16 items, in which each target word was paired with a distractor word. All of the distractor words were unrelated to any categories used in the rest of the CVLT–II. This procedure was included in the CVLT–II as a means to evaluate whether an examinee is exerting full effort (i.e., to assess motivation and malingering).

Procedure

The California Verbal Learning Test–II (CVLT–II) was administered to patients and controls and was embedded in a larger neuropsychological battery. The experimenter read a list of words (List A), and participants were asked to recall as many of the words as they could, in any order (Immediate Free Recall: List A). This procedure was repeated four times (Trials 2–5), so that there were five trials total. Each time, the experimenter read the list of words, and the participant attempted to recall the words in any order.

Following the list-learning procedure in Trials 2 to 5, participants heard a second list of words (List B) and subsequently tried to name as many of these words as possible (Immediate Free Recall: List B). Then, the participants were asked to name as many words as possible from the first list again (Short-Delay Free Recall: List A). Following this free recall task, participants were given a cued recall task, in which they were asked to name all the words from the first list that belonged to each of the four categories (Short-Delay Cued Recall).

Following a delay of approximately 20 to 30 min during which participants engaged in other executive function tasks (e.g., design fluency and Stroop tasks), they were asked to recall words from the first list that was read to them (Long-Delay Free Recall). Participants were not expecting this free recall condition, as they were not told that there would be further tests with the word lists. The categories were then provided as cues (Long-Delay Cued Recall). Following this, a yes/no recognition task was given, in which participants had to respond yes or no as to whether a word had been on the first list (Long-Delay Yes/No Recognition). After another delay of approximately 15 min, during which participants performed another executive function task, they were given a two-choice, forced choice recognition task (Long-Delay Forced Choice Recognition).

RESULTS

List Learning (Trials 1–5)

Patients' and controls' performance on learning the list of words (List A) across five trials was assessed with a 2×5 mixed analysis of variance (ANOVA) with Group (patients *vs.* controls) as a between-subjects factor and Trial (1–5) as a within-subjects factor. (On one trial, a control participant mistakenly thought he was only to name those words that he had not already said, and thus, his performance on that trial was excluded.) There was a main effect of Group, as controls recalled more of the words in the list across the five trials compared to frontal patients [78 *vs.* 52%, respectively; $F(1,19) = 24.21$, $p = .0001$]. There was also a main effect of Trial [$F(4,76) = 59.87$, $p = .0001$], as all participants tended to recall more items on the later trials (see Figure 2). The Trial $\times$ Group interaction was not significant [$F(4,76) = 2.03$, $p = .10$], as the pattern of learning across trials was similar for patients and controls (see Figure 2).

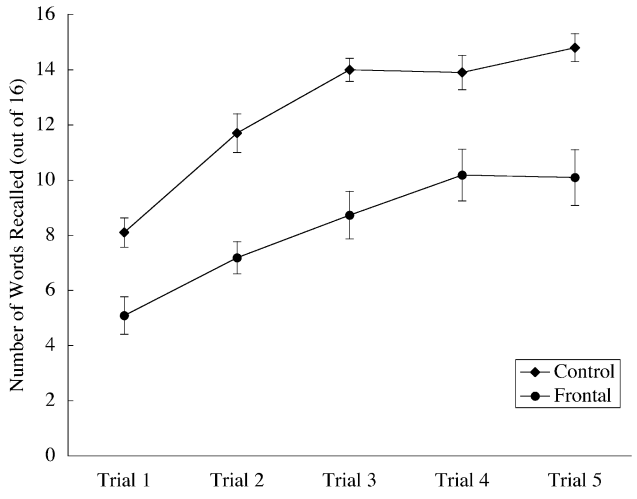

Fig. 2. Learning curves for frontal patients and controls across five trials of List A.

A separate analysis of slope confirmed that the overall learning curves for the two groups were not different [1.30 in patients and 1.59 in controls; $F(1,20) = 1.19$, $p = 2.89$]. One part of the learning curve that did approach a significant difference for patients and controls was improvement from Trial 1 to Trial 2 [slope of 2.09 *vs.* 3.54, respectively; $F(1,20) = 4.04$, $p = .06$]. In general, however, the pattern across trials was very similar for the two groups, suggesting that patients with focal frontal lesions demonstrate a normal learning curve, although they recall fewer words overall.

Interference (List B) and List A Short-Delay Recall

Immediate free recall on List B also showed a large discrepancy between patients and controls. Patients with frontal lesions recalled significantly fewer items from List B, compared to controls [4.36 *vs.* 7.64 items out of 16, respectively; $F(1,20) = 19.29$, $p = .0003$].

As a measure of proactive interference, performance on List B was compared to Trial 1 of List A (using the ratio of List B minus Trial 1, divided by Trial 1). Patients and controls showed almost identical levels of proactive interference [$F(1,20) = .0002$, $p = .989$].

The subsequent attempt to recall items from List A (Short-Delay Recall, Free and Cued) also distinguished the groups. A 2 × 2 ANOVA with Group (patients *vs.* controls) and Condition (free recall *vs.* cued recall) was used to analyze these results. There was a main effect of Group, as frontal lobe patients recalled fewer items across both conditions, compared to controls [8.59 *vs.* 13.73 out of 16; $F(1,20) = 18.18$, $p = .0004$; see Figure 3]. There was also a main effect of Condition [$F(1,20) = 8.29$, $p = .009$], as more words were recalled in the Cued Recall condition in both groups. There was no interaction between Group × Condition, as both patients and controls improved comparably from the Free to Cued Recall condition (adding an average of 1.1 words to their score).

Separate analyses assessed participants' tendency to generate intrusions on recall trials. Frontal lobe patients exhibited an elevated intrusion rate across all recall trials (including both short and long delays), relative to controls [$F(1,20) = 14.50$, $p = .001$]. This difference was mostly due to category (semantically related) intrusions, as frontal patients made significantly more category intrusions than controls [$F(1,20) = 30.73$, $p = .02$], but made a comparable number of non-category (unrelated) intrusions [$F(1,20) = 1.20$, $p = .28$].

Long-Delay Recall and Recognition

Long-delay recall performance was analyzed with a 2 × 2 ANOVA with Group (patients *vs.* controls) and Condition

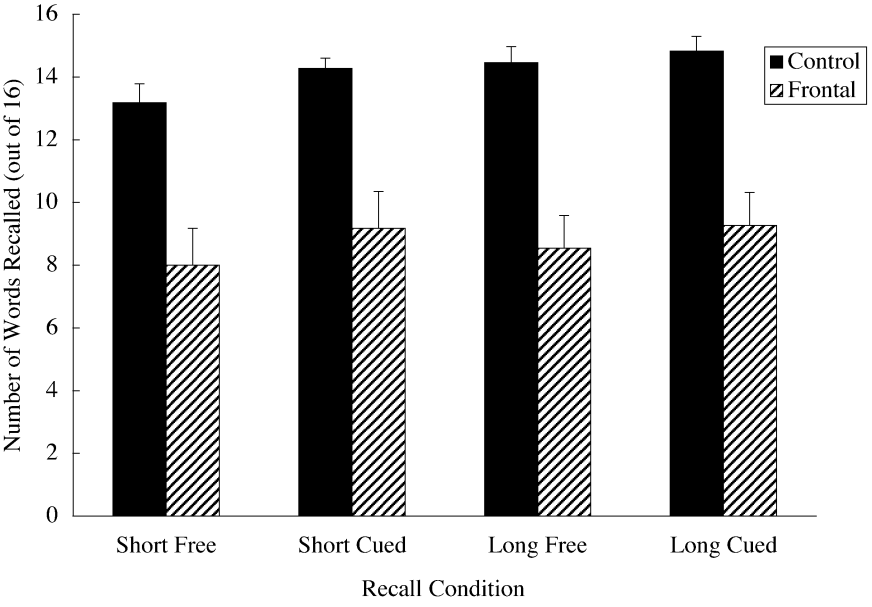

Fig. 3. Performance in frontal patients and controls on free and cued recall following short and long (20–30 min) delays.

(free recall *vs.* cued recall) as factors. As in the Short Delay conditions, there was a main effect of Group, with patients recalling fewer items than controls [8.91 *vs.* 14.64 out of 16, respectively; $F(1,20) = 25.33$, $p = .0001$; see Figure 3]. There was a main effect of Condition, as more words were recalled in the Cued condition [$F(1,20) = 6.32$, $p = .02$]. However, the Group × Condition interaction was not significant, as both patients and controls improved similarly when given category cues to prompt them.

Retention or savings was compared between the two groups by analyzing performance on Long-Delay Free Recall *versus* Short-Delay Free Recall. There was actually an increase in recall performance following the long delay that was significant across both groups [$F(1,20) = 9.35$, $p > .01$], but the interaction of Group × Delay was not significant. Therefore, while controls continued to recall more words after the long delay, both groups showed a comparable amount of savings and actually improved their recall performance to a similar degree. This increase in performance was not expected and may be due to the Short-Delay Cued Recall condition given prior to the delay.

The yes/no recognition procedure distinguished between frontal lobe patients and controls. Controls had a higher hit rate than frontal patients [97.2% *vs.* 88.1%, respectively; $F(1,20) = 5.10$, $p < .05$], and a higher correction rejection rate [97.7% *vs.* 84.7%; $F(1,20) = 4.84$, $p < .05$]. We did not correct recognition performance for the degree of initial learning, and thus reduced learning on initial trials in frontal patients may have played a role in their reduced recognition performance.

A series of d' analyses of yes/no recognition performance were conducted to tease out different mechanisms that might account for poorer recognition performance in frontal patients. d' is a measure of discriminability that combines both false positive and hit rates into a single statistic, in order to take into consideration response biases, for example, to always respond "yes." A d' value of zero means that a person cannot discriminate between two classes of items (in this case, List A *vs.* non-List A items), and larger d' values signify greater discriminability. The first d' analysis simply confirmed the main comparison, namely, that frontal patients were worse than controls in their overall discrimination between List A items and distractors [$d' = 2.57$ *vs.* 3.68, respectively; $F(1,20) = 15.43$, $p = .0008$]. A second analysis revealed that frontal patients were significantly worse than controls at discriminating between List A target words and List B distractor items [$d' = 2.28$ *vs.* 3.43, respectively; $F(1,20) = 15.21$, $p = .0009$]. This difference represents a deficit in source memory in frontal patients, an impaired ability to distinguish between two different sources of information (in this case, List A *vs.* List B). A third analysis showed that frontal patients were also worse than controls at discriminating between List A target words and novel distractors (including eight unrelated words and eight prototypical words from the same categories as List A) [$d' = 2.68$ *vs.* 3.48, respectively; $F(1,20) = 14.44$, $p = .001$].

In order to better understand what types of distractors frontal patients were most likely to mistakenly endorse, a series of *post-hoc* comparisons was carried out (see Figure 4 for a breakdown of yes/no recognition error rates). False positive rates were separated into three categories: endorsing semantically unrelated distractors, endorsing distractors from List B, and endorsing novel, semantically related distractors (that is, novel words belonging to a category from List A or B). While frontal patients were just as likely as controls to mistakenly endorse semantically unrelated distractors [$F(1,20) = 2.22$, $p = .15$], patients were more likely than controls to endorse List B distractors [$F(1,20) = 4.96$, $p = .04$], and semantically related distractors [$F(1,20) = 3.62$, $p = .07$ (trend)].

The forced choice recognition task did not discriminate between the groups, because all participants performed per-

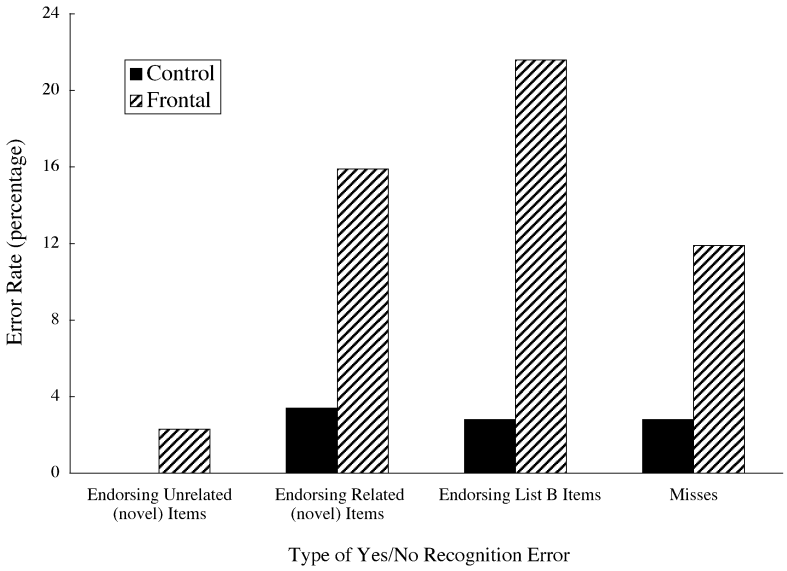

Fig. 4. Yes/no recognition error rates in frontal patients and controls.

fectly on this task (i.e., there were no errors on this procedure for any participant).

Semantic and Serial Clustering

Participants' recall data were analyzed for evidence of two types of clustering: semantic clustering (the tendency to group items according to semantic category) and serial clustering (the tendency to group items according to their order in the list). These measures were corrected for chance clustering with a list-based clustering index that uses the target word list as a baseline for calculating chance expected values. This correction measure is not influenced by the number of words recalled and has been verified using simulations (see Stricker et al., in press, for a complete description of clustering formulas and rationale).

A measure of total semantic clustering across learning Trials 1 to 5 revealed a significant difference between frontal patients and controls, with frontal patients exhibiting a reduced degree of semantic clustering [$F(1,20) = 30.73$, $p = .02$]. This reduced semantic clustering was still present in the Long-Delay Free Recall condition, as frontal patients were still inferior in their use of semantic clustering, compared to controls [$F(1,20) = 17.78$, $p = .0004$].

To assess whether the cues provided in the Short-Delay Cued Recall condition directed participants to utilize the semantic categories to support later recall, we compared semantic clustering for frontal patients and controls on Trial 5 *versus* Long-Delay Free Recall. The interaction was not significant [$F(1,20) = .78$, $p = .39$], as both controls and participants increased their semantic clustering comparably (and only marginally) from Trial 5 to Long-Delay. Therefore, the Short-Delay Cued Recall condition did not serve as a cue for frontal patients to significantly enhance their performance on a later recall task.

In terms of serial clustering in the initial list-learning phase, there was no significant difference between patients and controls [$F(1,20) = .244$, $p = .63$], as both groups were comparable in their tendency to recall words in the same order as they appeared in the original list.

DISCUSSION

Using the CVLT–II, a newly revised clinical measure of learning and memory, we showed that focal frontal patients performed significantly worse than age- and education-matched controls in the following ways: (1) frontal patients learned fewer words in the initial list-learning phase; (2) frontal patients recalled fewer words from a second, interference list; (3) patients recalled fewer items at short and long delays; (4) patients made more intrusions at recall; (5) patients exhibited reduced semantic clustering in the initial list-learning phase and at free recall; and (6) patients made more errors on a yes/no recognition task. Patients and controls did *not* differ in the following ways: (1) the slopes of their learning curves across the initial five learning trials were comparable; (2) the groups benefited similarly from category cues (cued *vs.* free recall); (3) the groups showed comparable amounts of serial clustering in the learning phase; (4) the groups showed comparable retention rates following a long delay; and (5) both groups performed perfectly on a forced choice recognition measure.

Thus, in the current study, we confirmed earlier findings that patients with frontal lobe lesions are impaired on list-learning tasks and at free recall (Gershberg & Shimamura, 1995; Incisa della Rocchetta & Milner,1993; Vogel et al., 1987). However, unlike many earlier findings, we found that frontal patients also showed a deficit on a yes/no recognition measure. In the past, normal recognition in frontal patients was taken to suggest that they were not amnesic but simply had difficulty retrieving information due to poor retrieval strategies and the like. We certainly confirmed this latter notion in the current study, as frontal patients were less likely to use semantic clustering, compared to controls. However, this strategy should not have played a direct role in yes/no recognition performance. Nonetheless, frontal patients made a significantly greater number of errors on the yes/no recognition task of the CVLT–II. Interestingly, an analysis of the types of false positives made showed that the greatest number of errors was due to source memory difficulties, that is, distinguishing words from List A and B. Frontal patients were much more likely to mistakenly endorse words that were heard in List B. The second most frequent recognition error type in frontal patients was endorsing semantically related distractors. This tendency was true, even when we considered only novel, semantically related distractors (i.e., words that had not appeared in List A or B), although this effect was only a trend.

Both of these phenomena have been reported in the literature with respect to frontal functioning, namely, both source memory deficits and false recollections due to semantic confusion have been attributed to frontal dysfunction (Janowsky et al., 1989; Schacter & Curran, 1995). These phenomena are consistent with a dynamic filtering view, in which prefrontal cortex is crucial for the selection of relevant activations and the inhibition of irrelevant activations. With damage to prefrontal cortex, the irrelevant activations rise above threshold and are thus not appropriately ignored (Shimamura, 2000).

A recent meta-analysis of memory performance in frontal patients suggested that a recognition deficit may be present in frontal patients but usually goes undetected due to low power (e.g., due to few participants, etc.; Wheeler et al., 1995). Even though the current study did not test a very large patient group, the CVLT–II was sufficiently sensitive to detect recognition deficits in this group of focal frontal patients. This may be in part due to the types of distractors used in the yes/no recognition procedure, namely, semantically related words and words from an interference list. Indeed, on a final forced choice recognition procedure in which the distractor words were highly unrelated, frontal patients did not make any errors. Thus, frontal patients'

memories were reliable as long as the distractor information was sufficiently distinct from the study material.

The current study provides an indication of the type of performance to expect in patients with frontal dysfunction. Our study would suggest that one can expect to see the deficits outlined above, namely overall reduced learning and recall (even with cued recall), reduced semantic clustering, reduced yes/no recognition performance, but normal forced choice recognition. The patients in the current study had relatively circumscribed lesions, and it is likely that more pronounced learning and memory deficits may be observed in patients with bilateral and/or more extensive frontal lesions. Also, it must be kept in mind that our study included a small sample of patients, and thus there are some limitations in the generalizability of the findings. Moreover, as our study did not include a neurologic control group, the specificity of these findings to frontal dysfunction alone was not tested. Testing with other neurologic groups (e.g., focal temporal lobe patients) on the CVLT–II is needed to further clarify the roles of these brain regions in memory and to provide normative data for comparison when the locus of injury is unclear (e.g., in mild traumatic brain injury).

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