Source Amnesia in Patients with Frontal Lobe Damage

Catherine Ann Gow

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Acknowledgments

It was the best of times, it was the worst of times,
it was the age of wisdom, it was the age of foolishness,
it was the epoch of belief, it was the epoch of incredulity...
Charles Dickens *A Tale of Two Cities*

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Abstract

Frontal lobe lesions do not produce a classical amnesia syndrome, although such damage has been associated with some forms of disordered memory processing. This study examined the phenomena of source amnesia in patients with frontal lobe damage. Patients with focal lesions to the frontal lobe, and age- and education-matched normal control subjects were compared on two memory tasks for their recall of both fact and source. In the first experiment, subjects learned fictitious facts about famous and nonfamous individuals and were tested for their recall of both the fact and the source following a 15 minute, and one week delay. In the second experiment, subjects were tested for their recall of the placement of objects on two surfaces under both plain and enhanced conditions. Subjects' ability to recall the board, as well as the exact placement of the objects on the board was tested. Additionally, subjects were required to discriminate experimental objects, from those not seen during the learning session. In both experiments, patients' ability to recall the facts, and the overall placement of objects was as good as that of normal control subjects, but the patients tended to commit more source errors than the normal control subjects. Results from experiment 1 indicated that some patients with lesions of the right hemisphere tended to have more difficulty recalling the presenter source (intra-experimental source) while some patients with left hemisphere damage tended to have more difficulty recalling where they had learned a fact (extra-experimental source). Subtle language impairments were found to be associated with acquisition but not delayed recall of the verbal material. Intra-experimental source errors were related to poor performance on the WCST, while extra-experimental source errors were related to non-verbal measures such as WMS-R, Visual Memory Index, and WAIS-R, PIQ. The data suggest that recall of contextual features such as the

modality of presentation and time and place of a learned event can be dissociated from the contents of the learning episode, and that source recall may be disproportionately impaired in some patients with frontal lobe damage. The second experiment revealed deficits in the ability of patients, particularly those with left hemisphere damage, to determine the exact placement of objects on the board to the left. The use of color to enhance learning did not result in increased recall for any of the subjects, however, the patients with damage to the left hemisphere were more likely to incorrectly attempt to place an object under these conditions. Recall of object placement was associated with classical frontal lobe measures, while discrimination of the sets of objects, and placing an object on the wrong board were related to the BNT in the enhanced condition. In fact, although significant correlations were found between the spatial memory measures in the plain condition and the Visual Memory Index, these measures were correlated with the Verbal Memory Index. The results suggest that enhanced context does not increase the accuracy of recall in either patients or normal control subjects. However, the addition of color to the learning situation increased erroneous attempts at placement in the group of patients with left hemisphere damage. This propensity was attributed to a combined effect of use of verbal strategies in the enhanced condition, and poor language decoding abilities in the left hemisphere damaged-patients.

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Compelling evidence exists to suggest that the frontal lobes support some forms of memory functioning. Early lesion studies in animals have demonstrated consistent impairments on tasks that appear to have some mnemonic components. It has been suggested that patients with frontal lobe damage do not have impaired memory functions. Rather such deficits are the indirect result of other disordered processes such as attention and motivation. Yet numerous studies provide evidence of disproportionately compromised ability to learn and recall a variety of materials in these patients. A number of questions about the precise nature of the disorder remain unanswered. In the following review I outline the history of this research leading to hypotheses about one particular type of memory disorder that has been associated with frontal lobe damage - source amnesia.

First, I describe the classical amnesia syndrome to facilitate comparisons with deficits highlighted in subsequent sections. For simplicity, the review is arbitrarily divided into four sections: 1) The historical review begins with an overview of early research in the area of memory functions following frontal lobe damage and includes a synopsis of work using delayed response and delayed alternation-type tasks which provided much of the impetus for research in the area of memory function in patients with frontal lobe dysfunction. 2) Evidence refuting the postulate that damage to frontal cortical regions results in classical amnesia follows. 3) An examination of the types of impairments exhibited by patients with focal frontal lobe lesions will illustrate the dissociation between disordered memory processing following frontal lobe damage and classic amnesia. 4) Finally, conclusions drawn from this research will be advanced to support the role of the frontal lobes in source memory.

Classic Amnesia

Amnesia is typically defined by four major characteristics: 1) intelligence, as assessed by standard tests such as the Wechsler Adult Intelligence Scale-Revised (WAIS-R), is unaffected, 2) short-term memory, exemplified by tasks such as digit span, is intact, 3) there is usually a period of retrograde amnesia (a loss of premorbidly established memories), and 4) there is poor acquisition and retention of new episodic and semantic memories (anterograde amnesia) (Mayes, 1988). The most widely accepted criterion, however, is the observation of a severe deficit in acquiring new information in the face of relatively preserved intellectual functioning and immediate memory (Cermak, 1982; Hirst, 1982; Milner, 1972; Schacter, 1987; Squire, 1986; Weiskrantz, 1987). In humans, two regions of the brain, the medial aspect of the temporal lobe, and the diencephalic midline have been most consistently implicated in classic amnesia as defined above (Squire, 1987). The appropriateness of applying this definition to the impairments typically seen in patients with damage to the frontal lobes will be examined below.

Role of the Frontal Lobes in Memory Functioning

Historical Review

The role of the frontal lobes in encoding and retrieval is controversial. Early studies of behavioral disruption following frontal lobe damage revealed surprisingly little change in such functions as intelligence and memory (Feuchtwanger, 1923). A large body of evidence has been amassed implicating frontal lobe dysfunction in problems of planning (Milner, 1982; Shallice, 1982), regulation of verbal behavior (Luria, 1969), perseveration (Hecean and Albert, 1975; Sandson and Albert, 1984), verbal and non-verbal fluency (Benton, 1968; Jones-Gotman and Milner, 1977), hypothesis testing (Milner, 1963), arousal (Luria, 1973), and motivation (Eslinger and Damasio, 1985). These are but a few

of the more frequently cited areas of compromised functioning following damage to a variety of regions within the frontal lobes. Yet the ability of these patients to perform traditional memory tests has been repeatedly tested, often with seemingly contradictary results. A brief historical review of the significance of the frontal lobes to memory functioning is presented.

Delayed Response

The notion that the frontal lobes are involved in memory functioning came from studies that tested monkeys with large prefrontal cortex lesions on delayed response and delayed alternation tasks (Jacobsen, 1935; 1936; Jacobsen and Nissen, 1937). As a number of excellent reviews of this literature exist (Goldman-Rakic, 1987; Oscar-Berman et al., 1991; Squire, 1987; Stuss and Benson, 1986; Warren and Akert, 1962), the work is briefly summarized. In these tasks, the subject watches while food is placed in one of two locations and, after a delay of a few seconds, is given the opportunity to select between these two locations, obtaining the reward if the location indicated is the correct one. In the delayed response paradigm, the animal must choose the well in which the food was hidden; in the delayed alternation paradigm, the location of the food reward alternates from trial to trial, with the correct response being the response opposite to that made on the previous trial. Jacobsen (1935) was the first to demonstrate that lesions of the prefrontal cortex produce profound deficits on the delayed response and delayed alternation tasks (Jacobsen and Nissen, 1937). Subsequent testing showed that the crucial locus of damage within the prefrontal cortex was the area in and around the principal sulcus, Brodmann's area 9 (Blum, 1952; Butters et al., 1972; Goldman and Rosvold, 1970; Gross and Weiskrantz, 1962; Goldman, 1971; Mishkin, 1957). The corresponding area in humans is Brodman's area 46 (Oscar-Berman et al., 1991). Performance on the delayed response task is not affected by

damage to other areas of cortex such as the posterior parietal cortex (Harlow et al., 1952; Jacobsen, 1936; Pohl, 1973). Lesions of the inferior and superior temporal cortex, and periarcuate or premotor areas also have no effect on delayed response performance (Goldman and Rosvold, 1970; Goldman et al., 1971; Jacobsen, 1936) Finally, lesions of dorsolateral frontal cortex produce delayed response deficits that increase in severity as a function of delay (Miller and Orbach, 1972; Treicher et al., 1971).

The principal sulcus appears to mediate specific functions. Deficits of delayed response suggest that this area mediates memory processes specifically for the location rather than for the distinct features or quality of objects in the environment. It has been shown, for example, that lesions of principal sulcus do not disrupt performance on a variety of tasks involving spatial processing which do not require retention of experimentally acquired information (Goldman and Rosvold, 1970; Oscar-Berman, 1978), including visual pattern-discrimination (Goldman, 1971; Passingham, 1972), object learning set (Mishkin, 1964), object discrimination reversal (Goldman, 1971), matching to sample (Mishkin and Manning, 1978; Passingham, 1978), and cross-modal matching (Petrides and Iversen, 1976).

Monkeys with principal sulcus lesions can also succeed at other tasks that require short-term memory, such as object alternation and discrimination tasks (Mishkin et al., 1969; Passingham, 1972). Animals with lesions in this area have also demonstrated intact conditional discrimination performance, wherein a particular response to a stimulus is conditional upon, for example, its colour (Goldman and Rosvold, 1970; Passingham, 1972, 1985; Petrides, 1982). Principal sulcus-lesioned monkeys are not impaired on go, no-go tasks, in which the animal is reinforced for alternately responding and withholding a response to a food well (Mishkin and Pribram, 1965; Goldman et al., 1971). Finally, monkeys with these

dorsolateral frontal lesions have been shown to be capable of completing the trial unique, delayed non-matching to sample task with delays up to two minutes and with as many as 10 objects to be remembered concurrently (Bachevalier and Mishkin, 1986).

Thus, in animals, lesions of the principal sulcus reliably produce deficits on such tasks as delayed response and delayed alternation, while leaving animals capable of performing such tasks as delayed matching to sample and go, no-go. Lesions of the prefrontal cortex in the region of the principal sulcus do not disrupt spatial perception, long-term memory, or the execution of motor responses, and the impairment following these circumscribed lesions is not due to sensory or motor difficulties, motivational deficits, or to task difficulty.

While the integrity of the prefrontal cortex appears to be essential for a number of delayed-response-type tasks in monkeys, a review of the work in this area with human subjects yielded less consistent results. No deficit on single- and double-delayed alternation, or on Goldstein's stick task were found in a patient with partial frontal lobectomy (Hebb, 1945). A deficit in delayed alternation was reported in lobotomized psychiatric patients (Pribram et al., 1964). However, patients with frontal lobe lesions showed no impairments on a more difficult series of tasks designed to demonstrate that deficits in delayed response performance were not due to impaired use of positional and orientational cues (Ghent et al., 1962). The authors interpreted these results as evidence that damage to the frontal lobes does not necessarily produce a specific recent memory deficit. Chorover and Cole (1966) were unable to demonstrate any impairment on a delayed response task in a group of patients with frontal lobe pathology. They compared the performance of patients with unilateral and bilateral lesions with that of patients with lesions in non-frontal regions and normal control subjects on a task requiring a response opposite to the one made on an immediately preceding trial. Lesion

localization in some of the human studies may not have been comparable to that of the animal research.

Yet several authors have found evidence for impairment on delayed response tasks in human subjects with frontal lobe damage. Freedman and Oscar-Berman (1986) have demonstrated in humans with bifrontal damage that performance on the delayed response task is dependent upon intact functioning of the dorsolateral frontal cortex. They tested patients with damage to frontal-cortical regions (confirmed by CT scan) at delays of 0, 10, 30, and 60 seconds and compared their performance to that of Korsakoff syndrome patients, or patients with ruptured ACA aneurysms. The authors found that patients with frontal lobe damage performed more poorly on the delayed response task than either of the control groups. Furthermore, a correlation was found between performance on this task and the Wisconsin Card Sorting Task (WCST), a measure that has been shown to be sensitive to frontal lobe impairment (Milner, 1962).

Amnesic patients with frontal lobe signs have also been shown to be impaired on a task modeled after Piaget's $A\overline{B}$ Task (Schacter et al., 1986). The $A\overline{B}$ task closely resembles the delayed response task, as both require the subject to relate two temporally separated events. Monkeys with lesions of the dorsolateral prefrontal cortex perform in an analogous fashion on $A\overline{B}$ as monkeys with the same lesion on delayed response (Diamond and Goldman-Rakic, 1989). Schacter et al. also tested adults with damage to the medial frontal cortex and found that they were not impaired on the $A\overline{B}$-like tasks and did not make as many perseverative errors as the first group. Therefore, patients with damage specifically to the dorsolateral frontal cortex appear to be impaired on some tasks that resemble the delayed response task used with monkeys. While disparate lesion size and localization parameters account for some of the variability, the divergent results apparent in the human literature may be a function of widely

differing task requirements across studies. Where substantial changes were made from the delayed-response paradigm used with animals to that used with human subjects, results tended not to coincide with animal work; where the paradigm remained essentially unchanged, similar findings were revealed (Oscar-Berman et al., 1991).

A number of different explanations of the delayed response/delayed alternation deficit have been advanced including impaired short-term memory, spatial information processing, attention, response inhibition, temporal chunking, and feedback system (Goldman-Rakic, 1987; Oscar-Berman et al., 1991; Stuss and Benson, 1986; Warren and Akert, 1962). While none of these explanations have proven entirely satisfactory, and some are clearly inadequate, the task requires, at a minimum, attention to and memory for spatial-location cues. In general, these tasks appear to be sensitive and specific measures of the functions mediated by the prefrontal cortex. Some form of memory processing figures prominently in successful performance.

Intact Memory Following Frontal Lesions

Feuchtwanger (1923) systematically studied 200 patients with frontal lobe lesions and 200 patients with lesions in other brain regions caused by gunshot wound in World War I. He concluded that lesions of the frontal lobes had little effect on intellectual or memory functions, but such damage had a severe impact on a patient's mood, attitude and planning abilities. Luria (1973) held a similar view based on years of work with patients with large frontal lesions resulting from strokes, tumors, and closed and open head injuries. He stated that the frontal lobes were essentially the brain center involved in planning, preferentially when the activity involved non-routine operations requiring the initiation and maintenance

of a plan, as well as a feedback system to check the effectiveness of the plan (Luria, 1973). Although Luria's patients had memory impairments, he attributed these to a more general failure of processing of non-routine material which he felt was essential in the encoding and retrieval of complex memory. The quintessential feature of the disorder, according to this view, is an impairment in the ability to perform tasks which are not routine, and would be expected to affect many domains of functioning, including memory.

It has also been repeatedly demonstrated that frontal lobe lesions in humans do not typically cause the kinds of classical memory deficits found in patients with medial temporal lobe or diencephalic lesions (Milner, 1964; Jones-Gotman and Milner, 1977; Kolb and Milner, 1981; Petrides and Milner, 1982). Milner (1967) examined the performance of patients with unilateral, left frontal-lobe damage on delayed recall of paired associates and stories that were presented in the auditory and visual modalities. She found that performance of the patients with frontal lobe lesions was normal on all tasks. Similarly, patients who underwent bilateral orbito-frontal resections in an attempt to ameliorate schizophrenic symptomatology showed good recall of prose and nonverbal material (Stuss et al., 1982). Patients with focal, unilateral lesions of the frontal lobes performed normally when asked to recall the spatial location of an array of 16 objects both immediately and after a 24 hour delay (Smith and Milner, 1984a). Normal performances were also achieved on tests of visual and tactual memory, digit span, and non-verbal object span after 15 second delays in patients who had incurred either left, right, or bilateral frontal-lobe injuries from missile wounds (Ghent et al., 1962; Teuber and Weinstein, 1956). There was no evidence of differential impairment amongst these three subgroups. Patients with focal lesions of the frontal lobes performed within normal limits on all four memory indices of the Wechsler Memory Scale-Revised (WMS-R), were not significantly different from

normal controls on paired-associate learning, story recall or complex figure recall, and had normal recognition scores on list learning tasks (Janowsky et al., 1989, Shimamura et al., 1990). Finally, these patients have been shown recently to be just as capable of performing such implicit memory tasks as word stem completion as their normal control counterparts (Shimamura et al., 1992).

When more general memory failure has been described in patients with frontal lobe damage, it can usually be demonstrated that brain regions outside the frontal lobes have incurred damage. For example, Baddeley and Wilson (1988) examined the nature of the amnesic syndrome shown in a patient who sustained bilateral frontal lobe damage following a severe head injury. The patient (R.J.) showed many of the deficits seen typically in the classic frontal syndrome, or dysexecutive syndrome, as coined by Baddeley (1986). R.J. was also impaired on a number of memory tasks which measure a wide variety of mnemonic abilities. The authors compared the performance of R.J. on a series of memory tasks to that of classic amnesics and concluded that frontal amnesia does not constitute a qualitatively different form of amnesia, but that it results from the existence of the typical pattern of memory deficits in conjunction with secondary problems typically seen to occur in the frontal dysexecutive syndrome (Baddeley and Wilson, 1988). It has been shown, however, that traumatic brain injury often results in at least mild memory disturbance as temporal lobe involvement is common. Moreover, the type and extent of injury sustained by R.J. has been shown in humans, and non-human primates to affect widespread areas cortically, subcortically, and in the brainstem (see Stuss and Gow, 1992 for a review). Indeed, R.J. performed in the clearly impaired range relative to his other test scores (and presumed premorbid level of ability) on the performance subtests of the WAIS-R suggesting impaired visuo-perceptual and/or visuo-construction abilities, likely as a result of parietal lobe involvement. The demonstration of "classic amnesia" in *this* patient does not

speak to the issue of the pattern of memory disorder in patients with focal frontal lobe damage, because it is unclear to what extent R.J.'s memory impairment is due to frontal lobe damage/dysfunction, or to damage of the medial diencephalic and/or temporal regions.

Impairments on Memory Tasks After Frontal Lesions

A review of the literature on memory functioning in patients with focal lesions of the frontal lobes revealed few studies that suggest that these patients are classically amnesic; that is, patients with frontal lobe lesions do not exhibit anterograde amnesia. In general, it can be demonstrated that they have acquired the to-be-learned material in a manner that is not statistically different from their normal control counterparts, although their mean scores are usually numerically lower. They appear to be less proficient than normal controls at encoding under some conditions and to have particular difficulty with certain aspects of the recall of previously learned material.

Free Recall, Cued Recall, and Recognition

Damage to the frontal lobes appears to result in consistent deficits on multiple-trial list learning tasks. Jetter, Poser, Freeman and Markowitsch (1986) found that patients with frontal lobe damage were significantly impaired in the number of words remembered under free recall conditions following a 24 hour delay relative to normal controls. The patients and normal controls performed similarly in their recall of the word list following a 15 minute delay, as well as in their cued recall and recognition of the material. Additionally, the patients used the strategy of semantical clustering of the words to the same extent that the normal controls did, suggesting that the impairment was one of poor retrieval and not due to insufficient encoding. Similar findings of impaired free recall in patients with

frontal lobe damage have been reported by other investigators (Smith and Milner, 1984; Janowsky et al., 1989).

Confabulation

There have been a number of reports of patients with lesions of the frontal lobes who exhibit an unusual form of disordered retrieval. Confabulation is the production of incorrect and sometimes bizarre responses to standard questions. Talland (1965) reported confabulation in the acute phase of the Wernicke-Korsakoff syndrome which typically subsided, and usually disappeared in the chronic amnesic phase. When persistent, the confabulations were not strongly related to the severity of the memory disorder. Confabulations were viewed as recollections of the patient's actual experiences that may be distorted and which are reproduced in an incorrect or modified context. These accounts are produced without awareness of the distortions or appreciation of the implausibility (Talland, 1965).

Kopelman (1987) has suggested that there may be two types of confabulation: spontaneous and provoked. In the latter form, a confabulated response is produced by amnesic patients on memory tests, and healthy subjects at prolonged retention intervals. Provoked confabulation is postulated to represent a normal response to a faulty memory (Kopelman, 1987). Confabulations can also be produced spontaneously (Stuss and Benson, 1986). Although confabulation is frequently coexistent with amnesia in patients with frontal lobe lesions (Barbizet, 1963; Kapur and Coughlan, 1980; Stuss et al., 1978), it has also been demonstrated in patients with frontal lobe lesions that the degree of confabulation is not closely correlated with either the severity of the memory deficit or the suggestibility of the patient, but is instead strongly related to the individual's ability to self-correct (Mercer et al., 1977), to control and monitor retrieval (Baddeley and Wilson,

1988), or to organize and evaluate retrieval information (Moscovitch, 1989). Moreover, the quantity of the confabulation decreases as patients' performance on frontal lobe tests improves (Kapur and Coughlan, 1980). As noted earlier, amnesia is usually the result of damage to structures within the temporal lobe, or in the region of the diencephalic midline such as the hippocampus, mammillary bodies, or dorsomedial nucleus of the thalamus, whereas frontal lobe damage or dysfunction was common to a number of studies of confabulation. However, confabulation has been demonstrated in patients with damage to cortical regions outside the frontal lobes (DeLuca and Cicerone, 1991; Gainotti, 1975; Weinstein and Lyerly, 1968). It appears that while a constellation of deficits may be present in patients who confabulate, decreased ability to organize, monitor and/or carry out strategic retrieval are key factors of the disorder.

Encoding

Despite demonstrating intact capabilities on many classical memory indices, patients with frontal lobe lesions are clearly impaired when task demands are manipulated in specific ways. For example, Signoret and Lhermitte (1976) presented frontal lobe patients with weakly associated word pairs and found that they learned this material very poorly; however, when instructed in visual imagery techniques, their performance was greatly improved. The authors attribute these findings to an inability in their patients to utilize elaborative encoding strategies spontaneously. They were capable of making use of these strategies if given instruction and guidance. In a similar vein, patients with frontal lobe lesions appear to be impaired relative to patients with temporal lobe damage in the categorization and subsequent recall of pictures. Della Rochetta (1986) examined two groups of patients who underwent unilateral (left or right) cerebral excision for relief of focal seizures. Patients were asked to categorize 36 pictures of

common items into groups of their own choosing and were told that they would be tested for memory of this material. Patients with either left or right frontal lobe lesions did poorly, both on the categorization task and on the recall of this material. Della Rochetta interpreted this difference as reflective of poor elaborative encoding in the group with right frontal lesions, and poor retrieval strategies in the group with left frontal lesions. One problem with this study is that it confounds the ability to categorize material with the ability to recall that material. It is an often-cited finding that patients with frontal lobe damage perform poorly on a wide variety of categorizing and sorting tasks (Drewe, 1974; Heaton, 1981; Milner, 1963; Nelson, 1976; Robinson et al., 1991; Delis et al., 1992). Nevertheless, these findings suggest that some form of strategic organization or elaboration of to-be-remembered material may be required to elicit impaired memory in patients with damage to the frontal lobes.

Proactive Interference

Patients with frontal lobe damage have been shown to be abnormally sensitive to the effects of interference during learning. Stuss et al. (1982) tested a group of patients who had undergone prefrontal leucotomy on a variety of memory tests, including the Wechsler Memory Scale, and found little evidence of impaired memory. Nevertheless, these patients performed very poorly on the Brown Peterson paradigm, a task that involves recalling a series of three consonants following a delay period filled with a distractor task (counting backwards by three). A similar sensitivity to interference was shown in a list-learning paradigm. Moscovitch (1982) presented four lists containing 12 words each to patients with unilateral frontal or temporal lobe lesions. The words from the first three lists were selected from the same semantic categories, while those from the last list were drawn from a new category. Normal subjects would be expected to show

improved recall of the final list, a phenomenon known as release from proactive interference. The author found that while patients with temporal lobe lesions showed poor recall of the words, they showed the expected release from proactive interference. The frontal lobe patients showed far less release from proactive interference. This was particularly true of those patients who performed poorly on the Wisconsin Card Sorting Test (WCST), and most marked in patients with left hemisphere lesions.

Freedman and Cermak (1986) also observed failure to release from proactive interference in a study designed to examine the role of encoding in mnemonic abilities in patients with frontal lobe lesions. They reasoned that if poor memory is a result of faulty encoding, then patients with frontal lobe lesions with poor memory should show little or no release from proactive interference following category shifts (by virtue of being unable to improve their performance by noticing or encoding the shift), while with good memory patients would be expected to show an improvement. The authors demonstrated that patients with good memory profited from a change in categories while poor-memory patients were unable to profit from or learn to use the expectation of this shift on multiple release trials. Further, failure to release from proactive interference in these patients was found to be correlated with performance on tests of anterograde memory. The authors postulate that combined damage to frontal and diencephalic regions may be necessary to prevent release from proactive interference.

In a recent study examining the nature of memory impairment in three groups of patients, Janowsky et al. (1989b) found that a group of patients with frontal lobe lesions performed comparably to a group of non-Korsakoff amnesic patients and normal controls in a test of release from proactive interference. In contrast, patients with Korsakoff syndrome did not benefit by the shift to a new category. Both the non-Korsakoff and Korsakoff amnesic patients showed poorer overall

recall of the lists, whereas the patients with damage to the frontal cortex were not significantly different from the normal controls. Korsakoff syndrome comprises a constellation of deficits which includes severe amnesia, as well as impairments on certain tasks sensitive to frontal lobe functioning, such as the WCST (Janowsky et al., 1989; Schacter, 1987). The finding of a dissociation in the performances of patients with frontal lobe lesions and those with Korsakoff syndrome on release from proactive interference tasks has prompted the postulate that combined frontal lobe dysfunction and amnesia is required to produce this type of encoding deficit (Shimamura et al., 1991).

Prisko (1963; reported in Milner, 1964) demonstrated a susceptibility to interference from the effects of preceeding trials using a modified delayed-comparison task. She examined the performance of a group of patients following frontal leucotomy on a task which involved the presentation, in succession, of two easily discriminable stimuli in the same sensory modality. The patient was asked to report whether the second stimulus was the same as, or different from, the first stimulus, which had been presented 60 seconds earlier. Thc patients with frontal lobe lesions, in contrast to those with temporal lobe lesions, were impaired on those tasks in which a few stimuli recurred in different pairings throughout the test. In contrast to this poor performance, they made virtually no errors on the one task in which new stimuli were used on each trial.

Milner (1968) has suggested on the basis of these findings that frontal lobectomy may interfere with the ability to structure and segregate events in memory. According to this view, in situations without strong contextual cues, patients with lesions of this type are impaired in the ability to discriminate between stimuli presented only 60 seconds before and a previous stimuli occurring in the same series of trials. Other researchers have provided evidence in support

of the hypothesis that the frontal lobes are involved in the temporal ordering of events. This work will be reviewed below.

Temporal Sequencing

Lewinsohn et al.(1972) found that patients with unilateral left, unilateral right, and bilateral frontal lobe lesions were substantially impaired when required to recall the temporal order of recently presented pictures and words. In contrast, these patients were much less impaired on memory tasks that did not demand ordered recall. Patients with unilateral frontal lobe damage were found to be impaired when required to produce, in the correct temporal order, a series of arm and facial movements (Milner and Kolb, 1985). Additionally, patients with damage to frontal-cortical regions have been shown to experience difficulty with tasks requiring various types of sequential organization, such as recollection of autobiographical episodes with temporal dates (Baddeley and Wilson, 1988; Moscovitch, 1989).

Corsi (cited in Milner 1971) devised three operationally similar tasks (long lists of concrete words, representational pictures, or abstract paintings) to test the assumption that the frontal lobes are involved in the temporal ordering of events. For the verbal discrimination task, he presented the subject with a deck of 184 cards, each of which depicted two high-imagery words, with instructions to read each word aloud and then turn to the next card. Periodically, a card would appear with a question mark between two words, at which point the subject had to indicate which of the two words had been presented most recently. Typically, both words on the card would have previously appeared (perhaps 8 trials ago as opposed to 16), but in one condition the word was new, in which case the task became one of simple item recognition. A similar procedure was followed for the pictorial tests. Patients with left frontal lobe lesions were impaired at judging the

order of the verbal material, but performed within normal limits on either of the pictorial tasks. Conversely, patients with right frontal lobe lesions were incapable of determining the relative recency of the abstract paintings, and were impaired on recency judgments for representational drawings. Their performance on the verbal task was only slightly better than that of the left frontal patients. The patients with lesions of the frontal lobes had no difficulty on simple item recognition. The above mentioned task on which the patients with frontal lobe lesions were impaired has a number of different components. A subject must first recognize the stimulus as being part of the to-be-recalled materials, *and* discriminate it from those not previously seen. The patients had no difficulty with this aspect of the task (simple item recognition was intact). However, when subjects were also required to make a temporal judgment - which of the two stimuli was presented most recently - patients with lesions of the frontal lobes were impaired. If the ability to order events or materials on the basis of their temporal characteristics is subserved by frontal cortical regions, these patients should have difficulty with other types of temporal judgments. This has been shown to be the case.

Milner et al. (1985) and Smith and Milner (1984b) provided evidence demonstrating that patients with frontal-lobe lesions are impaired at making frequency judgments. In this study, 20 abstract designs or words were presented to subjects as parts of a series in which each design or word appeared 1, 3, 5, 7, or 9 times, with the repetitions spread uniformly throughout the series. Following presentation of the entire series, subjects were shown an array of eight designs, four not previously been shown, and four which had been presented a variable number of times. Subjects were asked to estimate the frequency of occurrence of each design. The authors found that both patients and controls were reasonably good at estimating item frequencies up to three; however, patients with frontal lobe lesions were unable to estimate frequencies higher than this. In keeping with

Corsi's findings of hemispheric asymmetry of frontal lobe function, patients with lesions of the right frontal lobe were relatively more impaired in estimating the frequency of the abstract designs, whereas lesions of the left frontal lobes affected patients' ability to estimate the frequency of the occurrence of words. Recognition of the materials was normal for both groups. Unilateral temporal lobe patients showed poor recognition, but unimpaired frequency judgments.

Petrides and Milner (1982) also reported impairments in patients with frontal lobe lesions on a self-ordered pointing task. In this study subjects were given a stack of cards which depicted an array of stimuli (concrete words, abstract words, representational drawings, and abstract designs) whose position varied from card to card. The subjects were required to go through the cards and touch one item on each card without touching the same item on any of the cards. Patients with lesions of the left frontal lobe were impaired at pointing to a sequence of greater than six items, regardless of the nature of the material. Conversely, patients with right frontal lobe lesions were impaired on only the non-verbal tests.

Milner (1985) noted that the deficits on the self-ordered pointing tasks appeared to be most prominent when damage was in the area of the dorsolateral frontal cortex. Although many of the patients' lesions extended beyond the boundaries of one particular recognized division, she observed that lesions of the orbitofrontal cortex had little or no effect on tasks of this nature.

Summary

Damage to frontal cortex does not result in a classic amnesia syndrome; such mnemonic functions as paired associate and paragraph learning, and memory for spatial location of objects are spared. However, compromised frontal lobe functioning does appear to disrupt memory in very specific ways. The findings from the available literature suggest that when to-be-remembered information must

be organized, categorized, ordered, or monitored, disproportionately large deficits in recall become apparent. Damage to the frontal cortex also renders a subject more susceptible to effects of interference, and less able to learn more difficult associations (i.e. weakly-associated word pairs). Additionally, these patients are impaired relative to normal control subjects on list learning tasks when tested under conditions of free recall, but performance can be normal under conditions of cued recall or recognition. Finally, damage to the frontal cortex impairs a subject's memory for temporal order.

Memory for Context

Research with both normal subjects and amnesic patients provoked the development of theoretical models which distinguish between memory for facts or semantic knowledge, and episodic or personally experienced events (Cermak, 1984; Hirst, 1982; Jacoby and Dallas, 1981; Tulving, 1972; 1983). The encoding of context relevant to an event or item of information has been hypothesized to be the domain of the episodic memory system (Tulving, 1984). A variant of this postulate is that disorders of memory for the context of learned information are related to impaired memory for the temporal order of events. The type of information in episodic memory includes episodes or events in an individual's personal past which are temporally organized in memory. Semantic memory is generally understood to refer to factual information that has no necessary connections to the rememberer's individual identity. The episodic system, however, is the one said to be directly responsible for keeping track of the temporal order of occurrence of personal events (Tulving, 1984). It has been suggested that episodic memory is selectively impaired in amnesia (Cermak, 1984; Kinsbourne and Wood, 1975,1982; Schacter and Tulving, 1982; Shimamura et al., 1991).

In normal subjects, greater frontal and temporal lobe activity, as measured by increased cerebral blood flow to these areas, was associated with remembering episodic events, while recall of semantic material resulted in an increased activity in more posterior (parietal and occipital) regions (Tulving et al., 1988a; reported in Tulving, 1989). This preliminary demonstration of frontal lobe involvement in normal episodic retrieval is in agreement with much of the work on temporal and spatial episodic memory tasks in individuals with frontal lobe damage.

Source Amnesia

Numerous memory experiments have demonstrated that the ability to recall the contents of a recent episode is not inextricably linked to the ability to recall the personal event, the temporal sequence of the contents of an event (Bjork and Healy, 1974; Healy, 1982; Murdock, 1976), or the physical attributes of the event such as modality of presentation (Bray and Batchelder, 1972; Hintzman, Block, and Inskeep, 1972; Light and Berger, 1974), sex of speaker (Geiselman and Bellezza, 1977), and location (Mandler, Seegmiller and Day, 1977; Schulman, 1973). Some amnesic patients have been shown to be unable to recall where or when they acquired specific information, a deficit which has been referred to as source amnesia (Schacter et al., 1984). Shimamura and Squire (1987) have demonstrated amnesic subjects can show both impaired fact recall and impaired source recall; however, these are dissociable. Shimamura et al. (1991) have suggested that source amnesia is an impairment of memory for the spatial-temporal context. This dissociable deficit can occur in addition to a more general impairment in memory, and may be attributable to frontal lobe pathology (Shimamura and Squire, 1987; Shimamura et al., 1991).

A typical source memory paradigm involves presenting subjects with to-be-learned material (words, names of individuals, previously unknown facts, or

fictitious facts) and following a delay period, testing subjects for their recall of both the material and the source of the material. The source is usually defined as the time and place in which the material was presented (i.e. in the experimental setting, as opposed to on T.V., radio, etc.) but "source" has also been used to refer to which of two individuals, screens, or stereo speakers (modality) presented the information. A review of the populations that have been tested, the paradigms used, and the nature of source memory examined is presented below.

Schacter et al. (1984) presented amnesic patients characterized by diverse etiologies (early stages of Alzheimer's disease, anoxia, ruptured anterior communicating artery aneurysm, and closed head injury) with fictional characteristics about well-known and unknown people. The facts were presented by one of two experimental sources (the source being which of the two experimenters presented the facts). Subsequently, these patients were tested for retention of the facts and for the source of this material. The amnesic patients exhibited more source amnesia than their controls, and the frequency of forgetting the source was positively correlated with performance on tasks sensitive to frontal lobe pathology such as the WCST and the Benton Word Fluency Test, two commonly cited tests of "frontal lobe function".

In order to examine the contribution of the frontal lobes to this deficit more directly, Schacter et al. (1984) also tested four patients with frontal lobe pathology (two after ruptured anterior communicating artery aneurysms and two after left sided stroke) on a modified version of the task. They found that in the patients with lesions of the frontal lobes, level of fact recall was below that of the amnesic group, but the group with damage to frontal cortex did not exhibit evidence of source amnesia. The authors concluded that both severe memory impairment and frontal dysfunction is necessary in order to observe significant source amnesia levels. These last data are somewhat confusing. Little information was provided

regarding the memory functioning of the patients with frontal lobe damage, although it was suggested they did not have severe memory impairment. In fact, their level of item recall was .17 (less than an average of 2 of the presented 14 items); below that of the amnesic patients. Consequently, there was little opportunity for source errors to be made, and it is not surprising that there were no differences between the groups. In addition, the parameters of the lesions of the patient's with frontal lobe damage in the Schacter et al. study are not provided. It is entirely possible that the cortical areas damaged within the frontal cortex in this study are not the requisite areas for the production of source memory deficits.

Source memory impairments have also been demonstrated in normal older subjects. McIntyre and Craik (1987) assessed source memory in a group of normal older and younger adults using the paradigm developed by Schacter et al. (1984). They found that older adults exhibited considerable source amnesia when tested for retention of new facts and their source after a delay of one week. The authors suggest that there is evidence indicating that the normal aging process may resemble frontal lobe pathology by virtue of the fact that both are associated with difficulty in integrating experienced episodes with their temporal and spatial contexts.

Older adults also had greater difficulty than their young counterparts when asked to monitor the source of previously read, nonfamous names (Dywan and Jacoby, 1990). The authors used a task initially developed to measure unconscious influences of memory (the feeling of familiarity derived by reading a list of nonfamous names) on their conscious recall of the source of that material. When tested under these conditions, the older subjects were more likely to call nonfamous names famous. They also tended to perform generally more poorly on list-learning tasks. Recently, Ferguson et al. (1992) provided further evidence that older subjects are impaired relative to younger subjects in their ability to recall the

source of presented material, even when multiple source cues were available at the time of study.

Craik et al. (1990) demonstrated a relationship between normal aging, source amnesia, and frontal lobe dysfunction. Using the Schacter et al.(1984) source amnesia paradigm, they tested 24 older adults between the ages of 60 and 84 on their ability to recall fact, and source, and also administered two measures of frontal lobe functioning (WCST and the Verbal Fluency Test). The authors found no evidence of general intellectual impairment in their sample; however, the degree of source amnesia shown by normal elderly people correlated reliably with the Verbal Fluency Test, and WCST - number of perseverative errors, number of categories achieved, and total errors. Furthermore, the authors found that age correlated reliably with the measures of source amnesia and perseverative errors, suggesting that source amnesia and frontal lobe dysfunction, as measured, increase with age. These results were interpreted to suggest that the normal aging process may include a reduction in the efficiency of frontal lobe functioning and that this is manifested as an increased tendency to commit source errors. Source amnesia has also been shown to occur in patients with more focal frontal lobe damage. This research will be reviewed below.

If source memory deficits can be primarily attributed to compromised frontal lobe functioning rather than to impaired memory *per se*, then isolated damage to the frontal cortex should produce source memory impairments in the absence of any significant amnesia. This has recently been found to be the case. Patients with frontal lobe damage were shown to be impaired relative to age- and education-matched controls (older group), and to a younger group of control subjects, in their ability to recall the source of an experimentally learned fact (Janowsky et al., 1989b). The group of patients with frontal lobe damage included two with right dorsolateral, prefrontal damage (one with additional damage to the

orbital surface), one with damage to the right medial prefrontal area, two with left dorsolateral prefrontal damage, and two with bilateral damage to the prefrontal cortices. Janowsky et al. (1989b) hypothesized that, if there is a relationship between source memory and memory for temporal context, then patients with frontal lobe lesions might be expected to have impaired source memory. This assumption was examined by testing all subjects for item and source memory in two ways.

They first tested subjects' to explicitly recall both facts and the source of those facts by presenting them with 20 general information questions that they did not know the answer to for subsequent recall. Following a 6-8 day retention interval, subjects were tested for recall of the fact, the source, and for recognition of the materials. No reference was made to the study phase during the test phase.

Recall and recognition of the facts were similar across all groups. Thus, the patients with frontal lobe damage showed comparable background knowledge to both control groups and were as capable of learning and remembering new material as these subjects. However, they were impaired in their ability to recall the source of these facts.

In order to examine whether frontal lobe damage would result in the same type of source memory errors following incidental presentation of material, a second experiment was conducted. Subjects were presented 30 obscure facts that were divided into two sets of 15. Each set contained three facts from each of five categories: books and comics, movies and plays, history, geography, and sports. The first set was presented in the study phase - the second set was used in the test phase to assess baseline level of recall. Subjects were also presented with a series of easy factual questions to ensure that some material could have been acquired outside the experimental setting. The testing procedure used differed from the first experiment in that subjects were told that they were being tested on their ability to

categorize information, with no instructions to learn or remember the information. Fact recall, source recall, and fact recognition were tested on two separate occasions, at 5 minute and 2 hour retention intervals. Patients with frontal lobe lesions were shown to be as capable of learning and remembering new material as the control groups at both retention intervals. Under these incidental testing procedures, however, both the patients with frontal lobe lesions and the older control group were shown to commit more source errors than the young control group at both retention intervals. At a delay of 2 hours, the patients with frontal lobe lesions committed significantly more source errors than the young control group, but did not differ significantly from the older control group.

The authors concluded that the frontal lobes associate information stored in memory to various aspects of its context. Because the patients with frontal lobe damage were capable of demonstrating both fact knowledge and source knowledge, it was suggested that frontal lobe pathology disconnects fact memory from context memory rather than causing amnesia for the context itself (Janowsky et al., 1989b). They (Shimamura et al., 1991) describe the memory disorders exhibited by patients with frontal lobe damage under the rubric of prospective memory. According to this view the frontal lobes are involved in such strategic aspects of learning and memory as developing, planning, and initiating efficient encoding and retrieval of information.

The patients with frontal lobe damage tested in the above mentioned study generally performed well on memory tests demonstrating that source amnesia can occur in the absence of a classic amnesia syndrome. This is in contrast to the findings of Schacter et al. (1984), who found no source memory impairment their group of four patients with frontal lobe pathology. However, differences in subject selection and task demands may account for these divergent findings.

Four out of the five patients with unilateral frontal lobe damage in the Janowsky et al. (1989b) paradigm had definite dorsolateral frontal cortex involvement, and at least one of the patients with bilateral damage appeared to have some extension into the dorsolateral cortex on the left. It has been previously suggested that many of the deficits in organizing, sequencing, and temporal ordering, in a variety of tasks, seen in patients with frontal lobe damage are most prominent following damage to the dorsolateral frontal cortex (Milner et al., 1985). Similarly, deficits on delayed response and delayed alternation have been shown to be associated with bilateral damage to dorsolateral frontal cortex in humans, and damage to homologous cortical regions in monkeys. If one views delayed response and delayed alternation as tasks which rely heavily on a system responsible for maintaining the correct temporal order of a sequence of events, the similarities between many of the previously discussed tasks becomes apparent. It seems plausible that the frontal cortex, specifically dorsolateral prefrontal cortex, is responsible for the mediation of operations necessary to ensure that memories are laid down in a temporally contiguous fashion. According to this theory the frontal cortex plays an integral role in source memory.

The extent to which differences in experimental procedures between the Schacter et al. (1984) paradigm and the one used by Janowsky et al. (1989b) account for the discrepant findings remains to be determined. It is possible that the different task demands may have tapped into distinct functional systems. For example, in the Janowsky et al. study, information was presented by one individual to all subjects. The "source" or "context" for the purposes of that paradigm was the experimental material versus material never presented during the test session (including some material possibly experienced prior to the experimental session). Consequently, the task required a temporal discrimination between the contexts in which the test items were learned (6-8 days or 5 min. to 2

hours earlier) and the context in which non-presented items were learned (if known, these would have occurred at some point in time before the experimental session, or during the intervening delay period in explicit memory condition, and should be attributable to some source outside the experiment).

The procedure used in the Schacter et al. (1984) paradigm differs in several important ways. Subjects were presented high-knowledge, and no-knowledge name-characteristic pairs (false facts), as well as seven buffer items (true facts), by one of two experimenters. They were tested for retention of fact and source after .5 hour delay. Consequently, the source for this material would be one of the two experimenters, as well as the context within which the facts were learned. It is unclear whether or not the processes necessary to establish this kind of contextual memory are the same as or related to temporal context memory. Additionally, the overall item recall of the patients with frontal lobe damage was low in the Schacter et al. (1984) study. The possibility exists that the extremely low recall scores made it difficult to demonstrate clearly whether source memory errors would have occurred had item recall been higher. Finally, fact recall and source recall have been shown to be dissociable some amnesic patients when tested at short delay intervals (Shimamura and Squire, 1987). In the Janowsky et al. (1989b) study, the commission of source errors was examined only under explicit memory conditions, following a 6-8 day delay. If fact and source recall are truly dissociable, recall of both types of information would be expected to decline as the length of the retention interval increases, although fact recall should be superior to source recall.

Spatial Context Memory

Research with monkeys on delayed response tasks has demonstrated that the impairment following prefrontal lesions is specific to memory for the location of

objects in space over a given delay period. A number of experimenters have examined the ability of human subjects to perform a variety of spatial tasks. The right hippocampal system has been shown to be crucial for spatial learning and memory (Corkin, 1965; Smith and Milner, 1981). Patients with frontal lobe disease have been shown to be capable of simple spatial memory tasks, such as recall of object location (Milner, Petrides, and Smith, 1985), but they do have difficulty with certain spatial problems, such as spatial associative tasks. For example, in one task patients were taught to associate six identical blue lights with six identical white cards placed in front of the subject. The examiner determined the location of the correct association. In the non-spatial version, patients were taught to associate each of the lights with different hand postures (Petrides, 1985a). Patients with frontal lobe lesions of either the right or left hemisphere were impaired on both of these tasks. Kolb and Milner (1985) demonstrated that patients with frontal lobe damage were impaired relative to control subjects when asked to copy a variety of hand postures as demonstrated by an examiner. Consequently, it appears that damage to the frontal cortex results in a reduced ability to use a variety of spatial cues, and in associating and remembering these cues.

Patients with lesions of the frontal lobes have also been shown to be impaired on a spatial orientation task. Semmes et al. (1963) devised two tests of orientation; personal orientation (egocentric spatial relations), and extra-personal orientation (allocentric spatial relations). In the egocentric task, subjects were to point to the location on his or her own body corresponding to that illustrated in a diagram. In the allocentric task, the subjects carried a series of maps, one at a time, in a room where nine dots were painted on the floor in an evenly spaced manner. The subjects were required to pace out on the floor the route laid out on the map. Patients with frontal lobe lesions were impaired at the egocentric test and not the

allocentric; parietal lobe patients were impaired on the allocentric test, and only slightly impaired on the egocentric test. Performances of both groups were more severely impaired by lesions on the left side. Semmes et al. interpreted this as a difficulty with behaviors that depend on accurate assessment of one's body orientation in space. Pohl (1973) was able to demonstrate similar egocentric orientation deficits in monkeys with dorsolateral frontal cortex lesions. These studies support the role of the frontal lobes in certain types of spatial tasks where an association must be made between incoming chunks of information with spatial cues.

Indirect evidence also exists to support the notion that an association is made between contextual or environmental cues and the information presented within that environment. Winocur and Kinsborne (1978) found that enhancing the surroundings of a learning setting using coloured light and classical music had a beneficial effect on the recall scores of amnesic subjects. Conversely, the normal control subjects performed no better under these conditions than under regular, unenhanced conditions. The finding of improved recall scores in amnesic subjects following such a manipulation suggests that poorly developed associations between the context of a learning situation and the study material may be partly responsible for impaired memory. While other interpretations of this phenomena exist (i.e. environmental enhancement increases one's attention to the task), the conditions contributing to these associations have yet to be fully explored.

Definition of Context

A review of studies addressing the role of the frontal lobes in memory tasks delineated several important variables which must be considered in addressing the issue of source amnesia (Schacter, 1987). First, two types of information in memory should be considered: item and contextual. "Contextual information" or

"spatiotemporal context" is defined as "global information about when and where a specific event took place, such as the room in which a learning episode occurred, the temporal relation of one episode to another, and so forth." (Schacter, 1987, p.5). In contrast, item memory or information refers to what occurred during the particular episode, the to-be-remembered information, or contents of a learning session. Further, the demonstration of relatively poorer memory for the spatio-temporal contexts of recent episodes in amnesic patients is only compelling when item recall and recognition performance of the amnesic patients has been equated to that of normal controls by appropriate experimental manipulations.

Pickering et al. (1989) also suggest making a distinction between the to-be-learned information (target events), which are the focus of attention, and contextual information, which is not directly attended to. These authors describe contextual information as including the time and place of occurrence, the source, the modality, and the format of presentation of the target material. They maintain that for target information to be truly distinct from contextual information, the above mentioned features should have virtually no effect on the conceptual interpretation of target information.

In a recent commentary on Schacter's (1987) article, Lewis (1989) has suggested that there is no compelling evidence to suggest that the frontal lobes are involved in spatial contextual aspects of memory. While agreeing with Schacter that the frontal lobes do appear to be involved in the processing of temporal contextual information, he raises a number of interesting points about what has been termed spatial context. He points out that the task used by Schacter et al. (1984) confounds temporal and spatial information making it difficult to assess the relative individual contribution to the noted impairment. Similarly, he questioned Schacter's (1987) interpretation of Winocur and Kinsbourne's (1978) study examining the role of contextual cueing as supporting the notion that amnesic

patients with frontal pathology (Korsakoff) show deficient use of spatial context features to aid memory performance. Lewis postulated that these results support a difficulty in temporal discrimination between the tests which was reduced by placing subjects in a different, distinct environmental context.

Finally, he argues against grouping spatial and temporal context memory together, proposing both conceptual and functional distinctions. He provides support for the latter in an experiment carried out by Lori Shaler (Lewis, 1989). She examined the rates of forgetting for temporal order, and spatial location. Subjects were tested for recognition of slides of objects, buildings and landscapes which were presented on one of three screens to the left, right or center of the subject. Memory was tested at three delays; 5 min, 4 days or 1 week after presentation. Upon correctly recognizing a slide subjects were asked in which third of the list it had been presented and on which of the three screens it had appeared. In relation to the accuracy of the recall of location and time of stimulus presentation, a significant interaction was found between delay and the nature of the contextual condition, offering support for the notion of functional independence between temporal and spatial contexts. Spatial contexts, according to Lewis are more likely to be processed in the medial temporal lobe area.

Summary

The foregoing studies provide evidence that the frontal lobes play a role in associating contextual information about a specific event or fact to the information in memory. It has been previously demonstrated that patients with frontal pathology have difficulty with memory for temporal context (Corsi, cited in Milner, 1971). A similar deficit in temporal context memory has been demonstrated in Korsakoff syndrome patients, but not in other amnesic patients who exhibited a deficit in item memory similar in magnitude to that shown by

Korsakoff syndrome patients. It has been suggested that source amnesia may be a special case of impaired memory for temporal context in which the frontal lobes play an important role (Janowsky et al., 1989b). It remains to be demonstrated whether the frontal lobes are involved in processing primarily the temporal aspects of contextual information, or whether memory for spatial information is dependent on specific spatial contextual cues which also rely upon intact functioning of the frontal lobes. Additionally, the available evidence suggests that damage to the dorsolateral frontal cortex is predominantly responsible for the source memory impairments seen. It remains to be determined whether the left and right hemispheres contribute differentially in attributing context to information in memory.

The present studies were designed to investigate these issues by comparing the performance of patients with damage to either the right or left frontal cortex with that of normal control subjects on two different memory tasks; a verbal, and a nonverbal, or spatial memory task. Each of the experiments tested subjects' recall of both the experimentally presented items as well as their recall of the contextual cues associated with these items (more generally referred to as source in previously discussed research). One of the goals of the present research was to discover if dissociations exist between fact recall and different types of contextual cues in patients with damage to the frontal cortex and normal control subjects. This was achieved in two ways. A replication of the paradigm used by Schacter et al. (1984), McIntyre and Craik (1987) and Craik et al. (1990) was carried out to demonstrate that impairments in source memory do occur in patients with frontal lobe lesions who do not demonstrate significant impairments in fact recall. Two types of source were examined, modality of presentation, and the learning environment (temporal context). Inclusion of facts that relate to famous and unknown individuals makes it possible to examine the influence of prior

familiarity with a topic on recall of a new association (false fact) and its source. Previous research has demonstrated that variables such as prior level of knowledge are associated with the ability to acquire and retrieve new information in normal controls and some amnesic subjects, but the relationship has yet to be demonstrated in patients with frontal lobe lesions. Manipulation of the delay period made it possible to examine the effect of the passage of time on the ability of normal control subjects and patients with frontal lobe damage to recall both the fact and the source. An additional goal of the present study is the documentation of the area and extent of lesion necessary to produce source amnesia errors. Although conclusions will be limited due to the small sample size, the inclusion of patients with unilateral left and right frontal lesions will permit preliminary analysis of both the functional and anatomical parameters contributing to the above mentioned deficit.

Finally, considerable evidence suggests that the prefrontal cortex is prefentially involved in tasks which require the use of certain spatial cues, although the role of such cues in memory functioning remains unclear. An attempt was made to elicit source errors in spatial memory paradigm by creating two separate "learning environments" that required the use of spatial information for successful performance. The inclusion of this second task provided information about the processing of spatial versus temporal contextual cues and their relationship to memory functioning. The goal was to elicit source memory errors by requiring subjects to recall the distinct placement of similar classes of objects in two separate locations. Given that patients with damage to the frontal cortex have poor memory for source under some conditions, it was hypothesized that they would make more source errors than the normal control subjects. Also, it was postulated that the typically reported relationship between task demands and hemispheric specialization would be in evidence, with the result that a greater number of source

errors would occur in the group with right hemisphere damage. A second goal of the present study was to examine the effect of enhanced context on both the fact and source memory abilities of subjects to better delineate the roles that such cues play in normal and disordered memory processing.

Hypotheses to be Tested

Specific hypotheses to be tested in the present study are stated in the expected direction as follows:

1. The number of source errors committed on either the verbal (temporal) or nonverbal (spatial) context tasks will be significantly greater in the group of patients with frontal lobe damage than in the normal control subjects (NC), although these groups will not be significantly different in their recall of the materials.
2. The patients with lesions of dorsolateral frontal cortex will commit more source errors than patients with lesions in other areas of frontal cortex or normal control subjects.
3. There will be significant differences between the performance of patients with unilateral left frontal lobe lesions (LF) versus unilateral right frontal lobe lesions (RF). On the verbal context task LF patients will perform more poorly than RF patients, while RF patients will perform more poorly than LF patients on the spatial context task LF > RF.
4. A significant relationship will be found between classical measures of frontal lobe functioning (WCST, FAS) and the number of source errors made by any of the groups under study.
5. There will be a significant difference between the patients and the normal control group when enhanced context is introduced to the temporal and spatial context tasks RF = LF > NC.
6. Performance on the enhanced context task will be positively correlated with memory performance as assessed by WMS-R.

Method

Subjects

General Description

Patients meeting inclusion criteria for the present study were identified in three ways: 1) by a review of medical records and brain computerized tomography scans (CT) and magnetic resonance image scans (MRI) at the Ottawa General Hospital completed during the period 1990-1992; 2) by referrals from Ottawa and Toronto area neurologists; 3) subjects who had previously participated in a study in our laboratory (Della Malva, 1990, doctoral dissertation). They were divided into groups on the basis of localization of lesion as documented by MRI or CT scan. Inclusion criteria included evidence that 75% of the lesion resided within one hemisphere and did not significantly encroach upon another cortical region (i.e. patients with frontal lobe lesions extending posteriorly into the temporal lobe, or subcortically into neostriatum, were excluded). The presence of significant psychiatric disease, alcoholism, or previous significant neurotrauma served as exclusion criteria. Several of the patients suffered from pre-existing medical conditions (e.g. hypertension); only subjects whose medical conditions were adequately controlled were included in the present study. Additionally, subjects were required to meet a pre-established standard on several neuropsychological measures assessing functions considered to be relevant to perform the experimental tasks. These criteria are discussed under "Neuropsychological Assessment".

The protocol was approved by the Baycrest Hospital/University of Toronto Ethics Committee, the Ottawa General Hospital Human Research Ethics Committee, and the University of Ottawa Ethics Committee. Subjects were fully

informed, signed consent was obtained, and full debriefing followed completion of the experiment.

Patients with frontal lobe lesions

Sixteen patients with focal lesions of the frontal lobes were identified by review of medical records and CT scans at the Ottawa General Hospital. An additional four were referred by Toronto area neurologists; five other patients had participated in a previous study (Della Malva et al., 1993). Of these 25 patients, nine could not be contacted (last known address was incorrect), and two declined participation. The 14 remaining patients were considered first as one group with frontal lobe lesions, and were also classified into one of three groups on the basis of locus of lesion, right (RF-N=9) or left (LF-N=4) or bilateral (BF-N=1). One subject from the RF group withdrew from the study following an initial session due to poorly controlled seizures. The data from this subject were not entered into any analyses.

Neurological reports and CT brain scans were obtained for each of the patients. These records were examined in consultation with a neurologist and used to determine a subject's suitability for participation in the present study. Patients were excluded with lesions due to tumor causing midline shift, severe traumatic injury, or other insults suspected of causing damage to areas outside the frontal lobe. An attempt was made to select patients with cerebral infarction or hemorrhagic stroke in order to ensure inclusion of subjects with focal rather than more diffuse brain damage. However, due to limited patient availability, four patients with well defined tumor excision boundaries were accepted (one with left hemisphere, three with right hemisphere damage). One patient with a left frontal lesion had incurred the damage following an open skull traumatic brain injury. This latter patient was accepted on the basis of CT scan evidence confirming that

he met eligibility requirements for the present research. Patients were tested at least 8 months post-onset. Table 1 lists the characteristics of the 13 subjects who completed all experimental procedures (See Appendix 1 for more detailed lesion parameters).

			WAIS-R			
Group	**Age**	**Educ**	**FSIQ**	**VIQ**	**PIQ**	**Lesion**
Patients						
J.G.	18	11	109	98	110	Right dorsolateral prefrontal
B.C.	52	10	115	110	109	Right dorsolateral & orbital prefrontal
L.B.	47	10	115	110	109	Right medial prefrontal
R.M.	43	17	128	121	125	Right dorsolateral prefrontal & parietal
T.H.	52	19	125	120	127	Right medial & orbital prefrontal
I.M.	44	5	79	83	75	Right dorsolateral & orbital prefrontal
A.F.	68	9	97	100	93	Right dorsolateral prefrontal
E.B.	50	11	96	104	98	Right medial & orbital prefrontal
K.R.	27	17	117	113	115	Left dorsolateral prefrontal & temporal
B.H.	48	14	115	114	113	Left medial & orbital prefrontal
E.A.	51	13	104	99	111	Left medial & orbital prefrontal
C.C.	67	17	113	114	108	Left dorsolateral prefrontal
D.S.	60	9	94	90	101	Bilateral orbital prefrontal

Table 1. Lesion characteristics and WAIS-R VIQ, PIQ and FSIQ scores of the patients with frontal lobe lesions.

Lesions were identified and classified by a neurologist on the basis of CT brain scan analyses. Lesion parameters examined included size, extent and quadrant occupied, with the Fissure of Rolando demarcating the anterior-posterior divisions. This procedure is comparable to that used by other researchers (Benson and Barton, 1970; Black and Strub, 1976; Della Malva et al., 1993). CT brain scans were available for schematic reconstruction of the patients' lesions. Following the method of Damasio and Damasio (1988), the radiological images for each patient

were compared to templates. The set that best corresponded to the angle and size of the slices was selected and the lesion was reconstructed on these templetes (Appendix 2).

Normal control Subjects

Eleven control subjects were tested. They comprised volunteer friends or relatives of the patients, as well as community dwelling volunteers. They were matched to the patients with frontal lobe lesions with respect to age (± 5 years), education (± 2 years), and two subtests of the Wechsler Adult Intelligence Scale-Revised (Information and Block Design subtests of the WAIS-R). These subtests were chosen for both their neuropsychological and psychometric properties.

The Information subtest is considered a "hold" test as it is the least sensitive to neurological deficit of the WAIS-R subtests (Anastasi, 1982; Blatt and Allison, 1968; Boll, 1974; Groth-Marnat, 1984; Reitan, 1974a). Furthermore, it is a good measure of general intelligence and is highly correlated with the WAIS-R Full Scale IQ (Wechsler, 1981). The Block Design subtest is a good measure of nonverbal problem solving ability, is highly correlated with general intelligence, and is fairly sensitive to effects of brain damage, especially of the right hemisphere, and maximally with parietal lobe involvement (Groth-Marnat, 1984). Consequently, these two subtests provide baseline measures of general intelligence on which to match subjects.

Statistical Analyses

The nature of the patient population and the inclusion criteria adopted in the present study resulted in small sample sizes and unequal cells. Nevertheless, the sample size is equivalent or superior to many other published studies using similar patient groups (Della Malva et al., 1993; Janowsky et al., 1989; Shallice and

Burgess, 1991; Eslinger and Grattan, 1993). Specifically, the patient group with left frontal cortex lesions had fewer subjects than the group with right frontal lesions due to the high incidence of aphasia in patients with left anterior lesions. However, the number of subjects in any one group did not exceed that in any other group by a ratio of more than three to one (only one subject with bilateral frontal damage participated in the present study and was included in "all patients" analyses exclusively). This ratio was reported to be the maximum one acceptable for performing non-biased, two-way analysis of variance (Ferguson, 1981).

Data were analyzed using the Statistical Package for the Social Sciences 4.1 (SPSS 4.1). One and two-way univariate analyses of variance (ANOVA) were conducted. The verbal "fictitious" facts task was a 4 x 2 x 2 (group x knowledge x delay) split-plot design, with repeated measures on knowledge and delay. The spatial context memory task was a 4 x 2 x 2 (group x cue x context) with repeated measures on cue and context.

The level of significance for the repeated measures analysis was determined using the Geisser-Greenhouse correction (Keppel, 1982). All post-hoc comparisons were performed using the Tukey-Kramer method (Kirk, 1982) for unequal sample sizes. Results were considered significant at $p = .05$.

Neuropsychological Assessment

All patients were administered a neuropsychological battery of tests selected to establish premorbid functional levels and to ascertain that subjects had intact cognitive abilities necessary to meet the present experimental demands as well as for use in subsequent statistical analyses. All testing took place between February 1991 and August 1992. A full description of the tests follows.

Wechsler Adult Intelligence Test-Revised (WAIS-R) (Wechsler, 1981)

The following subtests were administered: Information, Digit Span, Arithmetic, Similarities (prorated Verbal IQ), Picture Completion, Picture Arrangement, Block Design, and Digit Symbol (prorated Performance IQ). Estimated IQ scores were calculated on the basis of these subtests according to age-adjusted norms (where applicable) provided in the manual [estimated Full Scale IQ (FSIQ)]. This information provided a measure of intellectual functioning on which subjects and their controls were compared. The WAIS-R yields a mean score of 100 and a standard deviation of 15.

Wechsler Memory Scale-Revised (WMS-R) (Wechsler, 1987)

The revised scale allows assessment of new learning ability in both the verbal and visual domains and results in a profile composed of the following five index scores: Verbal Memory, Visual Memory, General Memory Attention-Concentration, and Delayed Memory. The mean score for each index is 100 with a standard deviation of 15. Thus, the index scores can be directly compared with the WAIS-R IQ scores. Severity of memory loss has often been defined in terms of the numerical difference between IQ scores and and memory quotients obtained from the Wechsler Memory Scale (Bowden, 1990; Butters and Cermak, 1980; Butters et al., 1988), with approximately 68% of normal adults obtaining scores between 85 and 115 on both tests (Wechsler, 1981; 1987). Recently, the relationship between IQ scores and the WMS-R has been shown to as sensitive to memory loss as the IQ-MQ difference (Oscar-Berman, Papadopoulos-Clancy and Altman-Weber, 1993). The absolute difference between the WAIS-R Full Scale IQ (FSIQ) and the General Memory Index of the WMS-R was computed and used as an index of amnesia in the present study. WMS-R scores were correlated with measures of source amnesia to document any relationship between these two phenomena.

Western Aphasia Battery (Kertesz, 1982)

The WAB was administered to all patients in order to rule out the possibility of a significant language or comprehension deficit.

Boston Naming Test (Kaplan, Goodglass, and Weintraub, 1983).

The 60 item version of large, black ink drawings of objects ranging from common items (tree) to relatively uncommon items (abacus) was administered to evaluate the extent of naming deficits. The total number of items spontaneously correct was used.

Verbal Fluency Test (FAS) (Benton and Hamsher, 1976)

This test requires the production of words beginning with a particular letter (excluding proper nouns) within a one minute time limit. The procedure is repeated for the letters F, A, S. In addition, subjects were asked to generate exemplars from the semantic category of animals. The total number of words produced was recorded. It is a well established fact that patients with damage of the frontal lobes, especially those with lesions of the left hemisphere, evidence decreased word fluency (Milner, 1982). The total number of words produced in each one minute segment, the total number produced in three minutes (F, A, and S), and the total number of animal names produced were correlated with measures of source amnesia to determine if any relationship exists between commonly recognized neuropsychological measures of frontal lobe functioning and source amnesia.

Wisconsin Card Sorting Test (Heaton, 1981)

A test of problem solving, the WCST has been shown to be sensitive (although not specific) to frontal lobe dysfunction (Milner, 1962), particularly if other factors such as language are intact. The possibility that inability to recall the source of an event (item) is related to abilities tapped by WCST (total number of

categories, number of perseverative errors) was examined through correlational analyses.

Separate analyses were completed to compare the patient group as a whole to the normal control group, as well as to examine the difference between the two patient groups when compared to the normal control group for each of the demographic and neuropsychological variables. There were no significant differences between the groups in age, years of education or scores on the Information and Block Design subtests of the WAIS-R (Table 2).

Variable	Patients					All Patients	Normal Controls
	Right Frontal Lesions (N=8)		Left Frontal Lesions (N=4)		Bifrontal Lesions (N=1)	N=13	N=11
	Mean	S.D.	Mean	S.D.	Mean	Mean (S.D.)	Mean (S.D.)
AGE	47.0	14.2	47.3	16.3	60.0	48.1(14.0)	44.2 (15.2)
EDUC	11.3	3.8	15.0	1.8	9.0	12.2 (3.7)	12.6 (3.2)
WAIS-R							
Information	9.1	4.3	10.0	3.6	5.0	9.1 (3.9)	10.5 (3.2)
Digit Span	8.6	2.1	10.8	6.6	8.0	9.2 (3.8)	11.9 (3.6)
Arithmetic	9.0	3.4	9.8	3.6	9.0	9.2❖(3.2)	12.8 (4.0)
Comprehension	10.0	5.5	10.8	4.1	9.0	10.2 (4.7)	12.5 (3.1)
Similarities	9.1	3.3	10.0	3.5	7.0	9.2 (3.1)	10.2 (2.5)
VIQ	98.5	17.3	97.3	13.6	90.0	97.5❖ (15.1)	112.8 (17.0)
Picture Compl.	8.4	4.3	9.0	2.4	9.0	9.8 (3.0)	11.1 (1.8)
Picture Arrange	8.1	4.5	9.3	3.1	7.0	8.3 (3.9)	10.0 (2.9)
Block Design	8.9	3.9	9.5	4.1	9.0	9.0 (3.6)	10.6 (3.0)
Digit Symbol	7.0	2.4	6.0	2.0	5.0	6.5❖(2.2)	9.6 (2.6)
PIQ	96.3	19.9	75.0	45.3	101.0	90.1 (29.2)	103.1 (13.3)
FSIQ	98.1	20.0	98.0	12.8	94.0	97.8❖ (16.6)	113.0 (14.9)

❖ significantly different from Normal Control Group (p <.05)

Table 2. Means and standard deviations for demographic and WAIS-R variables for LF, RF, the combined patient group (all patients including those with right, left and bifrontal lesions), and the normal control group.

The patients as a group achieved IQ scores in the average range, although considerable individual variability is evident. Significant differences were found between the patient group (all patients) and the normal control group on the WAIS-R VIQ, $F(1, 22) = 5.5$, $p = .03$, and overall FSIQ, $F(1, 22) = 5.5$, $p = .03$. The groups did not differ significantly in terms of PIQ, $F(1, 22) = 1.0$, $p = .32$. The patients also performed significantly more poorly on the Arithmetic subtest, $F(1, 22) = 6.0$, $p = .02$, and on the Digit Symbol subtest, $F(1, 22) = 6.7$, $p = .02$. The finding of relatively preserved IQ in the face of deficits on specific subtests of the WAIS-R is consistent with previous studies of patients with lesions of the frontal lobes (Drewe, 1974; Janowsky et al., 1989; McFie and Thompson, 1972; Stuss and Benson, 1986).

Table 3 shows the means and standard deviations for the neuropsychological variables for the patients with lesions of the right and left frontal lobes and the normal control group. The groups were not significantly different in terms of the four WMS-R memory index scores. All groups had mean General Memory Index, and Delayed Memory Index scores that were in the average range or higher indicating that none of the groups were classically amnesic as defined (i.e. had anterograde learning deficits). There were also no differences between the groups on the WAIS-R FSIQ minus WMS-R General Memory Index. However, differences were apparent between the patients with right frontal lobe lesions (RF) and the normal control group on the Attention/concentration index.

There were also significant differences between the normal control group and the patients with lesions of the left frontal lobe (LF) on the Boston Naming Test, and between the LF and RF groups on the WAB indicating that although the LF group was not aphasic, they displayed some subtle language impairments, notably a mild anomia. Finally, group differences were apparent between the RF and

normal control groups when the number of perseverative errors made on the WCST was examined.

Variable	Patients				Normal Controls
	Right Frontal Lesions N=8*	Left Frontal Lesions N=4	All Patients N=13	Bifrontal Lesion	N=11
WMS-R	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	Mean	Mean (S.D)
Verbal Memory	96.9 (22.5)	93.3 (22.3)	95.2 (20.6)	89	95.7 (11.7)
Visual Memory	109.3 (19.1) N=7*	100.0 (23.4)	106.3 (19.2)	110	116.3 (12.8)
General Memory	104.6 (22.0) N=7*	94.3 (26.3)	100.3 (21.9)	95	101.5 (12.0)
Attention Concentration	86.4❖ (12.2) N=7*	96.3 (20.4)	91.8❖ (15.9)	111	107.3 (14.8)
Delayed Recall	102.9 (25.8) N=7*	102.5 (30.3)	101.9 (24.9)	93	105.0 (21.0)
WCST					
Categories	3.5 (2.4)	3.0 (2.2)	3.5 (2.3)	6	5.0 (1.8)
Perseverative Errors	35.5❖ (29.3)	17.0 (8.8)	28.5❖ (24.6)	19	12.2 (9.2)
Perseverative Responses	44.0 (41.0)	17.5 (8.3)	33.9 (34.3)	19	15.8 (11.6)
FAS					
F	11.1 (3.8)	13.3 (6.8)	11.7 (4.6)	10	13.9 (3.6)
A	7.4❖ (2.8)	7.5 (4.8)	7.3❖ (3.3)	6	13.0 (4.2)
S	12.3 (5.4)	10.3 (5.0)	11.3❖ (5.0)	9	15.2 (3.8)
Total	30.8 (10.3)	31.0 (15.1)	30.4❖ (11.0)	25	42.1 (10.1)
WAB-AQ	99.2✱ (.73)	95.2 (5.4)	97.9 (3.4)	99	99.6✱ (.67)
BNT	51.6 (6.9)	43.0❖ (15.0)	48.9❖ (10.1)	51	56.2 (3.9)

✱ significantly different from the group with left frontal lesions ($p < 0.05$)
❖ significantly different from the normal control group ($p < 0.05$)

Table 3. Neuropsychological and demographic variables for RF and LF patient groups and normal controls. Abbreviations: WMS-R=Wechsler Memory Scale

Index scores, FAS=Verbal Fluency Test, WAB-AQ=Western Aphasia Battery (Aphasia Quotient), BNT=Boston Naming Test.

Experimental Paradigms

General Description

Source amnesia was measured in two different paradigms. The first was essentially a replication of previous work examining source amnesia in normal young and elderly people (Craik et al., 1990; McIntyre and Craik, 1987), and in amnesic subjects and patients with frontal lobe lesions (Schacter et al., 1984). This study examines subjects' ability to recall as distinct one of two sources of verbally presented material, and to attribute the learning of such material to the experimental session. Failure to recall the source of a successfully remembered item would be an example of a source error. Recall was tested at two delay intervals, short and long.

The second paradigm examined memory for spatial location using two spatial contexts. The latter paradigm is a modified version of one used to examine the manipulation of context on memory performance in normal young and elderly subjects. The ability of subjects to replace an object correctly on one of two study surfaces was examined for the occurrence of source errors in a plain and enhanced context condition.

Patients and control subjects received 4 tests: 2 formats of the verbally presented material, one tested following a 15 minute delay, and one tested following a 6-8 day interval; and two formats of the spatial context paradigm, plain and enhanced context conditions, administered at least one week apart. The order of administration for the two verbal tests was randomized within blocks of 6

subjects (blocks were repeated for N>6 subjects in each group). The order of the spatial context tasks also followed the randomization procedure mentioned above. Each of the experimental paradigms will be discussed in more detail in the upcoming sections.

Experiment 1

Materials

Studies by McIntyre and Craik (1987) and Schacter et al. (1984) have established that memory for new facts is strongly dependent upon subjects' knowledge of the real or fictitious personalities involved. McIntyre and Craik (1987) have established familiarity ratings for 60 names of famous personalities with subjects from the Toronto, Ontario region. Although some of the patients in the present study were drawn from the same region as those in the McIntyre and Craik (1987) study, it could not be assumed that the above-mentioned personalities would have been rated in the same manner three years later. Therefore, in constructing our stimuli, we selected names and facts using the same procedure as that used by McIntyre and Craik (1987). The names were selected by having 42 neurologically intact individuals between the ages of 22 and 81 rate 60 names of public figures on a scale of 1 to 10 representing "how famous or well-known she/he is to you", with a score of 10 for "extremely well known". Subjects were subsequently given 1 min to generate as many facts as possible about each of the 60 public figures. If two or more subjects had never heard of a name it was eliminated. This procedure resulted in 12 names with the highest fame rating and the greatest number of generated facts, and 12 names with the lowest fame rating and the fewest number of generated facts being accepted as the high and low knowledge famous names, respectively. The 12 fictitious (nonfamous) names were those used by Schacter et al. (1984) and McIntyre and Craik (1987). The

fictitious facts (coupled with the famous and nonfamous names) used in the present study were those prepared by Schacter et al. (1984) and used by McIntyre and Craik (1987) and Craik et al. (1990).

Each fact was paired once with each of the 12 high knowledge, and 12 low knowledge famous names and once with each of the 12 nonfamous names (See Appendix 3). This procedure resulted in three different combinations of 36 fictitious facts that were felt to be believable but not obviously true (Format 1, 2 or 3). These sets were rotated across study conditions (short delay and long delay) so that every six subjects formed a completely counterbalanced group. As some groups were not numbered in multiples of six, the design was partially counterbalanced for those groups (See Appendix 4 for complete randomization of design). Preliminary analyses of this data revealed insufficient separation of the high and low knowledge famous name responses (that is, low knowledge famous names were well known to most subjects and this initially became apparent during debriefing). These two knowledge categories were collapsed to form "famous" names (24) and compared to the "nonfamous" names (12) in all further analyses.

Twenty four buffer items were also included. These comprised both fairly obvious statements (e.g. George Burns likes to smoke cigars) and less obvious statements (e.g. Lucille Ball was once a night club performer). These items were included to increase the overall credibility of the facts presented. This procedure resulted in a total of 60 facts; 36 "made-up" statements, and 24 true buffer statements presented to each subject at the study session.

The retrieval test included all 36 "fictitious facts" (24 famous and 12 nonfamous), 12 buffer items, and 36 "lure" questions. The lure questions were included as general control questions and to make extra-experimental source errors possible. Twelve lures were well-known facts about well-known people, 12 were concerned with fictitious characteristics of well-known people, and 12 dealt with

fictitious characteristics about fictitious people (See Appendix 3). In total, the retrieval test included 84 questions, with the three types randomly interspersed (See Appendix 5).

Procedure

The 60 facts used in the study session were printed on index cards, one fact per card. The deck was divided into four blocks of 15 cards with the various types of items being evenly distributed among the four blocks. A completely randomized presentation order of the four blocks, ABCD was used. Two different presenters (one female, one male) were videotaped reading the facts. This method of presentation was used in order to ensure consistency of presentation across subjects. Half of the facts were read aloud by a male presenter (first and third or second and fourth blocks of cards); each fact was read twice in succession. The remaining 30 facts (alternate blocks) were read aloud by a female presenter. All facts were presented at a rate of one item per 10 seconds. Experimenter presentation and presentation of the four blocks of facts (ABCD) were completely counterbalanced for groups of six subjects (See Appendix 4).

An attempt was made to render the two presention modes (female or male) as distinctive as possible by selecting individuals with contrasting physical characteristics (e.g. voice timber, height, and appearance). The position of the presenter in the room, as well as the distance each stood from the camera, was held constant. The presenter stood in a corner approximately three meters from the camera. The top of the viewing frame was aligned to the wall-ceiling junction and centered on the corner junction. This was done in order to maximize the presenter characteristic of height, which was differentially represented in this viewing frame (female presenter height 5' 1"; male presenter height 6' 1").

Subjects were all individually tested and read the following instructions:

" You will be learning trivial facts about famous and not-so-famous people. Please try to remember as much as you can. Following the presentation of all of the facts, you will be asked to complete a questionnaire which requires you to remember this information. Do you have any questions?"

Subjects were then allowed to seat themselves at a comfortable distance from a 21" television screen and the volume was individually adjusted for each subject prior to the beginning of the experiment. Four video-tapes, each approximately ten minutes in duration, containing the four randomized blocks of facts (A,B,C,D) of Format 1, 2 or 3 were played in succession. For example, Subject 1 in any of the groups viewed Format 1 with Tape 1 containing Block A (female), Tape 2 containing Block B (male), Tape 3 containing Block C (female), and Tape 4 containing Block D (male). The administration of the second delay condition was separated from the first by at least six days.

At the appropriate retention interval following the presentation of Tape 4 (15 minute - short delay or 6-8 day - long delay), subjects were administered the 84 item retrieval test booklet. No reference to the tape viewing session was made during the test phase; however, all subjects eventually realized that at least some items had been presented to them in the testing session. Subjects were encouraged to provide an answer to each fact that they were questioned about. As well, they were asked to recollect where the information had been learned even if the correct answer could not be recalled. This latter information was collected in order that source recall could be examined independent of fact recall. If a subject indicated that an item was learned in the experimental session, they were requested to remember whether it was presented on Tape 1, 2, 3, or 4, and whether it had been presented by a woman or a man. If a subject indicated that an item was learned outside the experiment, they were required to state the specific extra-experimental source (i.e. newspaper, television). Finally, subjects were required to provide a

familiarity rating (very familiar, somewhat familiar, not all familiar) for each of the 84 items that they were questioned about. Most subjects did not understand the nature of this request, and it could not be clarified without revealing the fact that many of the items had been presented at the testing session. Therefore, there was incomplete collection of the familiarity data. The test was self-paced, and on average took one-half hour.

Source errors

Dependent measures were calculated for item recall. and the two possible types of source error, that is, extra-experimental, intra-experimental, as well as for the ability to recall the order of tape presentation. These terms will be defined below.

It was postulated that the patient groups would be less proficient at attributing a recalled item to its correct experimental source; that is, to recall that a fact was learned in the experiment and not from an external source such as the newspaper, TV, etc. Such an erroneous attribution was referred to as an *extra-experimental* error in keeping with the nomenclature used by Craik et al. (1990). Also a focus of study was the ability of patients to recall the correct *intra-experimental* (presenter) source (Craik et al., 1990) given a correct response. It was hypothesized that patients with damage to frontal cortex would be less able to recall the gender of the individual who had presented an item to them than would normal controls. Finally, the ability of subjects to recall the order of the materials presented to them was investigated by asking subjects which tape an item was presented on. This measure was considered to be an index of an individual's ability to recall the temporal order of a series of items. The number of correct tape attributions was conditionalized on the total number of items correctly recalled. Calculations for each of the measures were as follow:

1. Fact Recall - The total number of items correctly recalled. In most cases a response was considered correct only in cases where the response corresponded exactly to the item presented on the tape (e.g. "What does Bob Hope grow in his garden?" correct answer "roses", incorrect "flowers"); In some cases, several responses were essentially correct ("What does Jane Fonda eat for breakfast?" correct answer "oatmeal", accepted "porridge", incorrect "cereal"), and an alternative response was accepted. These "alternate" correct responses formed less that five percent of the total correct responses. The total *fact recall* possible was 24 in the "famous" condition, and 12 in the "nonfamous".

2. Correct Source - The total number of items correctly answered, correctly attributed to the experiment, *and* correctly attributed to the correct presenter (male or female).

3. Extra-experimental error - The total number of items correctly recalled but attributed to the wrong source (TV, radio, or any source other than the actual experiment), divided by the total number of facts recalled correctly.

4. Intra-experimental error - The total number of items correctly attributed to the experiment but attributed to the wrong presenter, divided by the total number of facts recalled correctly.

5. Tape misattributions - The number of items correctly recalled but incorrectly attributed to Tape 1, 2, 3, or 4, divided by the total number of tape responses made.

Results

Table 4 shows the mean proportion of facts recalled by patients with lesions of the right (RF) and left (LF) frontal lobes, the entire patient group (all 12 patients), and the normal control group at the two delays (short and long) and for the two levels of prior knowledge (famous and nonfamous) of the associated personality. An ANOVA examining the interaction between group and response was performed to determine whether a dissociation existed between the groups in their ability to recall facts and source. This analysis collapsed across level of knowledge and type of error made. No significant interaction was revealed in the group by response type analysis.

In order to examine the performance of subjects in the different learning and delay conditions, separate univariate analyses of variance were conducted on the proportion of correct facts recalled and each of the different classes of source recall listed above. Results of each of these analyses are presented first for the patients versus the normal control group comparison. Additional comparisons between the groups of patients with right and left frontal lobe lesions, and the normal control group will follow.

An ANOVA of the number of items correctly recalled (fact recall) within each of the conditions revealed no significant differences between the patient and normal control group in terms of overall number of items recalled, and no significant interaction. There is a consistent trend for the patients to perform numerically lower across the delay and knowledge conditions, however, there is considerable variability within and across groups. Predictably, there was a significant main effect of delay, $F(1, 22) = 90.4$, $p=.00$, and of knowledge, $F(1, 22) = 62.4$, $p=.00$ reflecting the general trend for subjects to recall more items at the short delay, and more famous than nonfamous items, $p=.05$.

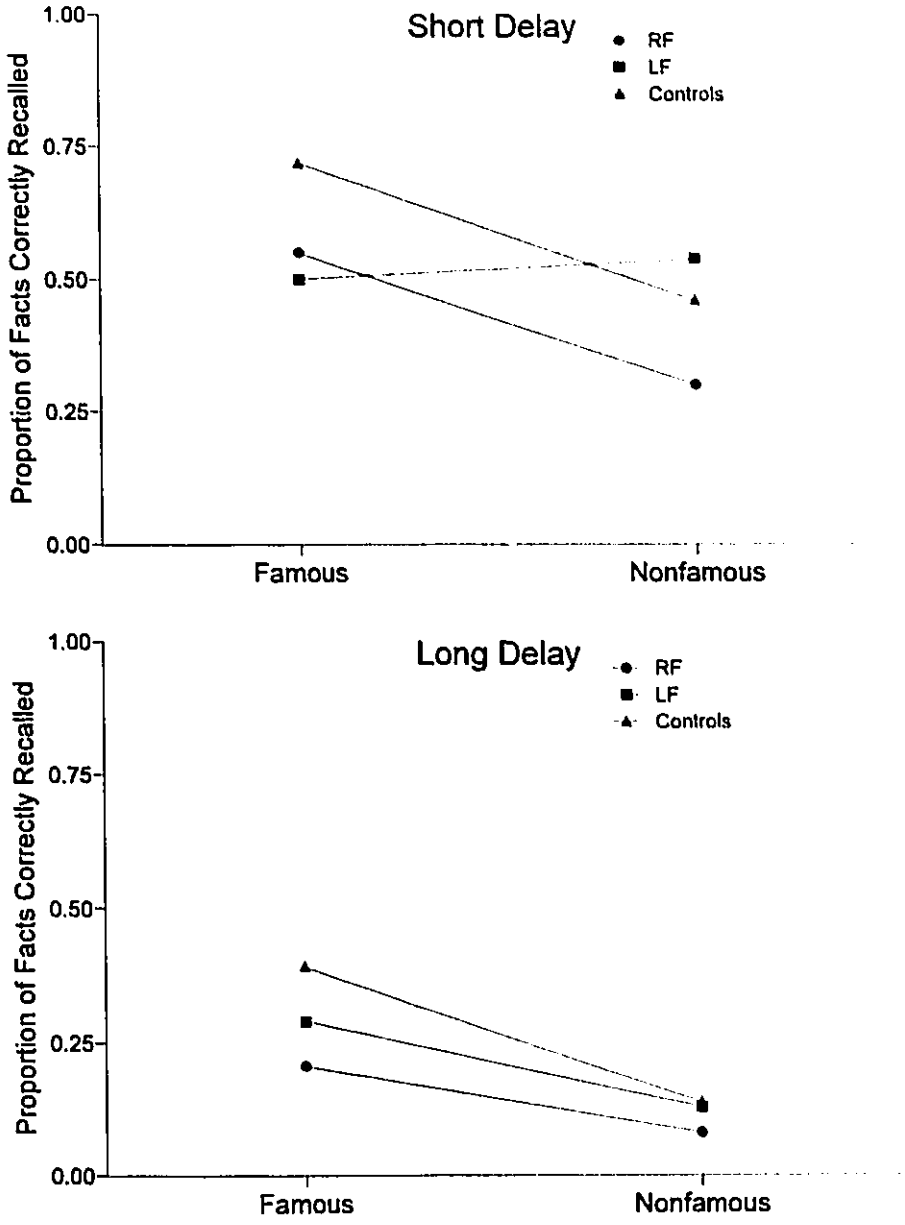

Figure 1. Mean proportion of famous and nonfamous facts correctly recalled at the short and long delays for the RF (N=8) and LF (N=4) patient groups and normal control subjects (N = 11).

A significant two-way interaction of group by knowledge was revealed when the performance of the RF, LF and normal control groups was compared,

F (2, 20) = 3.78, p=.04. Post-hoc analysis revealed that whereas both the RF and the NC groups recalled more famous than non-famous facts, the LF group's recall of famous and non-famous facts was not significantly different, p=.05. Figure 1 shows the level of fact recall of the three groups for the two levels of knowledge at the short and long delays. The three-way interaction of group, knowledge and delay failed to reach significance, F (2, 20) = 2.6, p = .10.

Results (in proportions) for Experiment 1

Delay	Knowledge	Group	Fact Recall	Source Recall		
				Correct source	Intraexp. error	Extraexp. error
Short	Famous	Right F.L.	.55 (.21)	.23(.21)	.70(.22)	.07(.08)
		Left F.L.	.50(.36)	.52(.23)	.47(.23)	.01(.03)
		All Patients	.55(.25)	.30(.26)	.65(.26)	.05(.07)
		N. control	.72(.12)	.45(.18)	.55(.18)	.00(.00)
	Nonfamous	Right F.L.	.30(.23)	.29(.38)	.50(.44)	.21(.36)
		Left F.L.	.54(.37)	.54(.37)	.47(.37)	.00(.00)
		All Patients	.37(.28)	.34(.38)	.53(.41)	.13(.29)
		N. control	.46(.25)	.34(.29)	.66(.29)	.00(.00)
Long	Famous	Right F.L.	.21(.12)	.25(.28)	.70(.35)	.06(.11)
		Left F.L.	.29(.26)	.19(.22)	.22(.28)	.44(.49)
		All Patients	.24(.16)	.21(.25)	.57(.40)	.17(.32)
		N. control	.39(.22)	.21(.20)	.64(.30)	.06(.13)
	Nonfamous	Right F.L.	.08(.06)	.44(.50)	.31(.46)	.00(.00)
		Left F.L.	.13(.20)	.20(.40)	.05(.10)	.25(.50)
		All Patients	.09(.12)	.33(.45)	.21(.38)	.08(.28)
		N. control	.14(.14)	.18(.41)	.46(.52)	.09(.30)

Table 4. Mean proportion (S.D.) of facts recalled and source attributions proportioned among completely correct items for the patients with right frontal lobe (RF) and left frontal lobe (LF) lesions, all patients (including the subject with bilateral frontal lobe damage), and the normal control group.

The focus of this experiment is the attribution of source for the correctly recalled facts. These items should all be attributed to the experiment, and then to the appropriate presenter within that general source. Table 4 shows how the source attributions for correctly recalled facts were proportioned among completely correct items, intra-experimental errors, and extra-experimental errors for the various conditions. Correct source in Table 4 refers to items that were correct both in terms of the general source and the specific (presenter) source. Intra-experimental errors are those items correctly attributed to the experiment but attributed to the wrong presenter. Extra-experimental errors are those items that were correctly answered but whose source was given as guessing or as TV, newspaper, school, etc. (Schacter et al., 1984; McIntyre et al., 1987) and corresponds to source amnesia as defined by Schacter et al. (1984). Separate univariate repeated measures of analyses of variance were conducted on the three sets of source recall data.

With respect to the proportion of correctly recalled items that were completely correct (correct source as defined above), there were no significant differences between the overall patient group and the normal control group. When the two patient groups were compared to each other and to the normal controls, there was a significant main effect of delay, $F(1, 20) = 4.6$, $p = .04$; of those facts that subjects correctly recalled the response to, they were completely correct in terms of source attributions more often at the short delay. However, these differences did not reach statistical significance in post-hoc analysis, $p=.05$. The two-way interaction of group and delay approached significance, $F(2, 20) = 3.11$, $p = .07$. There was a great deal of individual variability in the data. This is particularly problematic in the long delay condition where some subjects failed to recall any facts. Consequently, correct source attributions could not be made.

Figure 2 illustrates the general trend for source recall to decline as the level of fact recall and knowledge of a personality (famous to nonfamous) declines in the normal control group. In the group of patients with lesions of the left frontal lobe, ability to recall the source deteriorates markedly from the short to the long delay for both famous and nonfamous items. At the short delay, unlike the normal control group, the LF group's ability to recall the source (and the fact) is roughly equivalent regardless of level of knowledge. Conversely, the patients with right frontal lobe lesions appear to be generally poor at recalling the source, even at the short delay. In fact, none of the groups, including the control group, were very good at recalling the source of a learned fact.

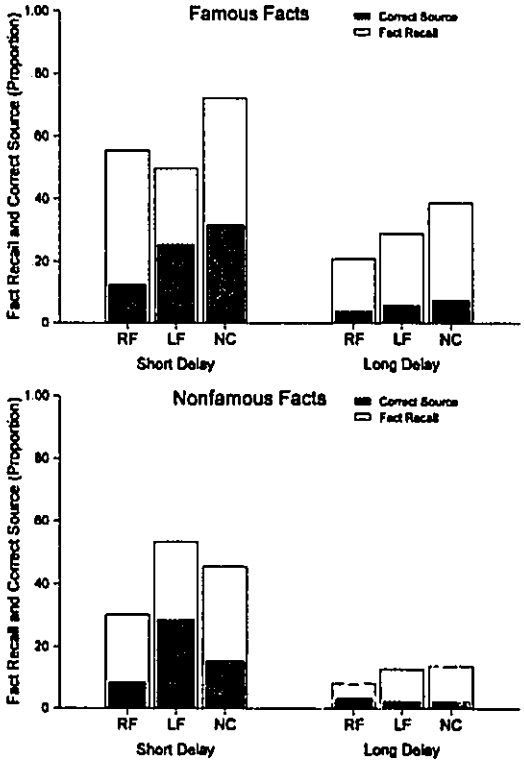

Figure 2. Mean proportion of correct source responses (correct fact, general source, and presenter source in hatched bar) shown as a function of the mean proportion of the total facts recalled (open bar) for the patients with right frontal lobe lesions (RF), left frontal lobe lesions (LF), and normal control subjects (NC).

Although Table 4 appears to suggest that the group of patients with lesions of the right frontal lobes were reasonably proficient at recalling the source of nonfamous facts at the long delay, it is important to note that this is based on a fact recall of .08, or roughly one fact (.08 x 12= .96).

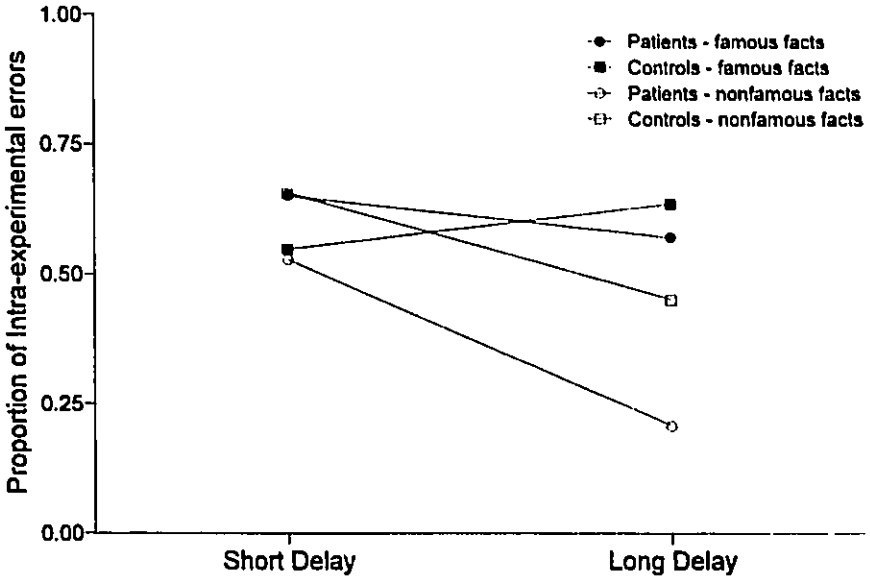

Figure 3. Mean proportion of intra-experimental errors for famous and nonfamous facts at the short and long delays for patients (N = 13) and normal control subjects (N = 11).

An analysis of the number of intra-experimental errors made by the patient group and the normal control group yielded a significant two-way interaction of delay by knowledge, $F(1, 22) = 5.73$, $p=.03$, reflecting the fact that patients and normal control subjects made more intra-experimental errors on famous facts at both the short and long delays, and on non-famous facts at the short delay, than they did on non-famous facts at the long delay, $p=.05$ (Figure 3). This effect is largely attributable to the floor effect evident for the recall of nonfamous facts at the long delay, which resulted in little opportunity for intra-experimental errors.

The group by knowledge interaction approached significance, $F(1, 20) = 3.87$, $p = .06$; although there was considerable variability, the patients tended to make the fewest intra-experimental errors at the long delay on nonfamous facts and the greatest amount on famous facts at the short delay. The normal controls displayed an opposite pattern tending to make the most intra-experimental source errors on nonfamous facts at the short delay and famous facts at the long delay. There was also a significant main effect of knowledge, $F(1, 22) = 7.1$, $p = .01$, reflecting that, in general, more intra-experimental source errors were made on famous facts. This trend was confirmed in post-hoc analysis, $p < .05$.

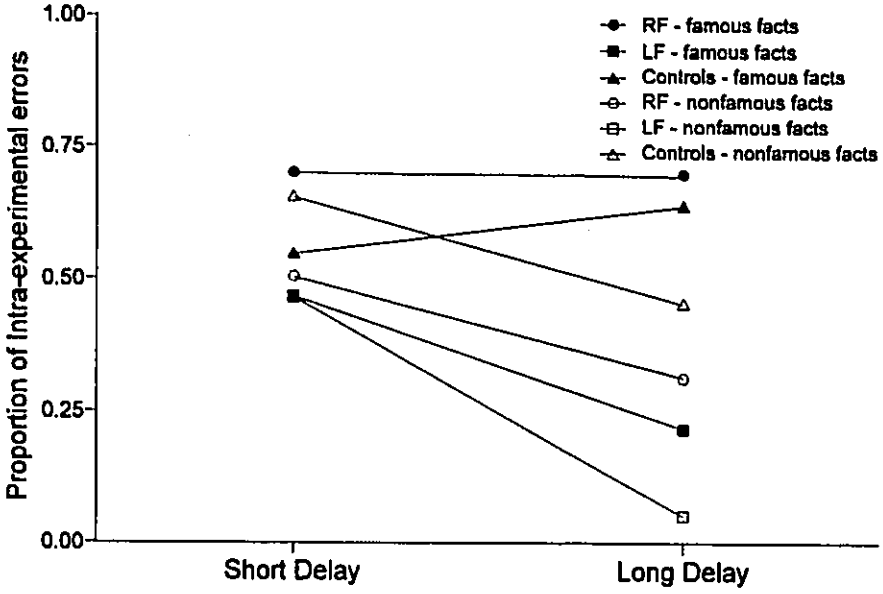

Figure 4. Mean proportion of intra-experimental errors for the two levels of knowledge (famous and nonfamous) at the short and long delays for the RF and LF patient groups and the normal control subjects.

The ability of each of the two patient groups and the normal control group to recall the presenter is depicted in Figure 4. An analysis of the proportion of intra-experimental errors made on famous and nonfamous facts across the two delay

periods revealed significant main effects of delay, $F(1, 20) = 4.46$, $p = .05$, and knowledge $F(1, 20) = 5.77$, $p = .03$. There was a trend for subjects to make more intra-experimental errors in the short delay condition, and for famous facts, but these results were not significant in post-hoc analysis. Figure 4 shows that at the short delay all subjects were performing at or below chance level (50% chance of responding correctly given a male or female presenter). While the normal control subjects and the RF group continued to perform at this level for the famous facts at the long delay, the LF group's level of intra-experimental recall was relatively good, suggesting that if they recalled a fact, on roughly 80% of those trials they also recalled the presenter source. Very few nonfamous facts were recalled at the long delay, but here again, the LF group was more likely than the other two groups to recall the presenter.

In order to examine the pattern of forgetting presenter source, an analysis of the absolute number of correct presenter responses (without regard to whether the item was correctly recalled) was performed. In other words, does the pattern of presenter recall parallel that of fact recall? This analysis yielded significant main effects of delay, $F(1, 19) = 18.62$, $p = .00$, and knowledge, $F(1, 19) = 44.11$, $p = .00$, indicating that, like their recall of the facts, subjects correctly recalled the presenter source more often on famous than nonfamous facts, and at the short than at the long delay. There was a significant two-way group by delay interaction, $F(2, 19) = 4.78$, $p = .021$, reflecting the tendency for the LF and normal control groups to recall the gender of the presenter more often at the short than the long delay. The RF group's recall of this information was similar across the delays and roughly equivalent to that exhibited by the other two groups at the long delay; however, these findings fell short of significance in post-hoc analysis (Figure 5).

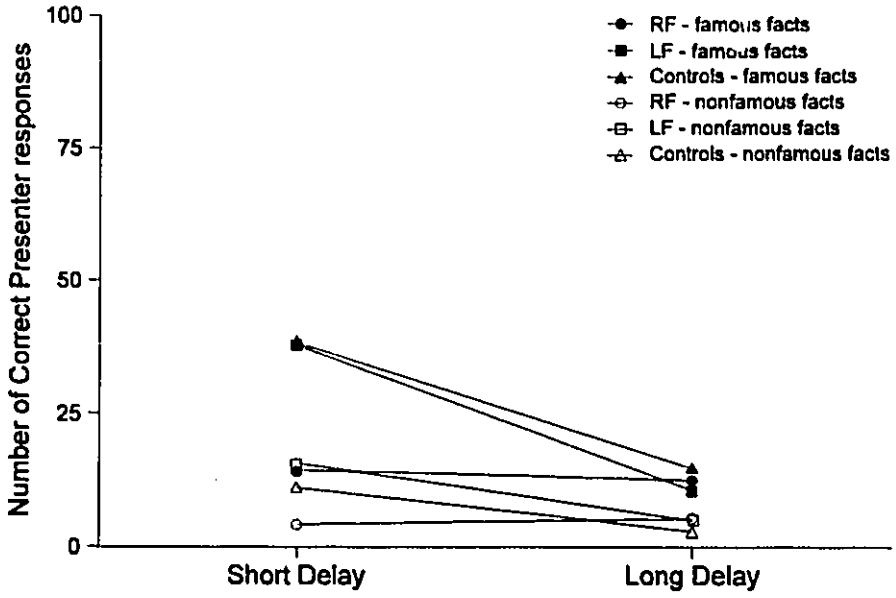

Figure 5. Mean number of absolute presenter responses for the RF and LF patient groups and normal control subjects.

Most subjects rarely attributed an experimentally learned fact to an external source such as television or the newspapers etc. An analysis of the proportion of extra-experimental errors made by the total patient group and the normal control group revealed no significant main effects or interactions. Only patients with lesions of the left frontal lobes made an appreciable amount of these errors. When the two patient groups were compared to each other and the normal control group there was a significant two-way interaction of group and delay, $F(2, 20) = 5.01$, $p = .02$. Figure 6 shows the proportion of extra-experimental errors made by each of the groups at the short and long delays. Although this finding fell short of significance in post-hoc analysis, the normal control group never made any of these errors at the short delay regardless of the level of knowledge of the associated fact. When the RF group did commit these errors, it tended to be made on a nonfamous fact. At the long delay, only the LF group made extra-

experimental errors at a level much above zero, and more of these on the famous facts.

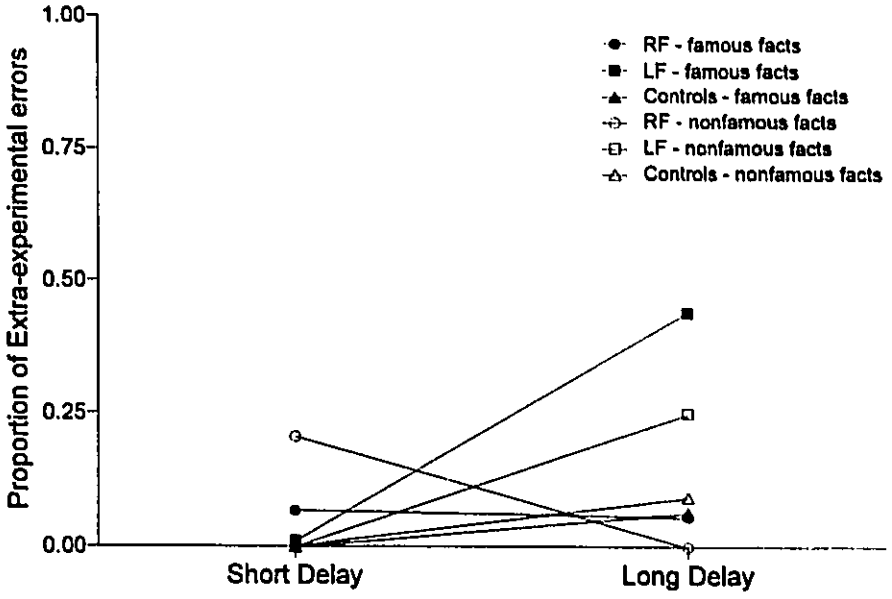

Figure 6. Mean proportion of extra-experimental errors at the short and long delay for famous and nonfamous facts for the RF and LF patient groups and the normal control subjects.

Also of interest for the present experiment was the number of subjects who correctly recalled the video tape on which the item in question had appeared. As subjects often recalled that an item had been presented to them in the experiment (via one of four audio-visual tapes), even though they could not recall the correct response, this measure was analyzed using the absolute number of correct tape responses (i.e. without regard to whether the associated fact was correct). A score of "1" was recorded if a subject recalled correctly that a particular fact had been presented on Tape 1 (for example), and the total number of such correct responses was divided by the total number of tape responses made. In general, subjects performed at about the chance level (1/4 probability of correct response by

guessing) across all conditions, although the LF and normal control groups performed at slightly higher level in the famous, short-delay. Nevertheless, the only analyses to reach significance were the main effects of delay, $F(1, 20) = 10.75$, $p = .004$, and knowledge, $F(1, 20) = 18.16$, $p = .000$, reflecting the fact that subjects' recall was better at the short than at the long delays, and was better for famous facts, $p = .05$. No other effect or interaction was reliably significant.

It is clear that considerable variability is evident in the performances of the patient and normal control subjects. Visual inspection of the data revealed that some subjects rarely or never made either type of source error, while others made a number of one or the other type. In order to more fully explore trends that might be present, especially considering the heterogeneity of lesions areas and sizes represented in the patient group, chi square analyses were performed using the following definitions to classify an individual. A subject was placed in the "poor performance" group if the number of intra-experimental errrors made was greater than two standard deviations above the mean of the normal control group. All other patients were classified into the "good performance" group. This cut-off was chosen somewhat arbitrarily, but it is representative of decisions made using standardized tests to facilitate clinical diagnoses.

Using these criteria, there were no significant differences in the group classification for intra-experimental source errors. Nine of 13 patients and 9 of 11 normal control subjects were placed in the "good performance" group, while four patients and two normal controls were classified into the "poor performance" group at the short delay for the number of intra-experimental errors made on famous facts. There were also no significant differences for the short delay, nonfamous, or either of the long delay conditions.

Extra-experimental source errors were made rarely by subjects. Consequently, an individual making any of these errors was classified into the "poor

performance" group. If none of these errors were made, a subject was placed in the "good performance" group. This classification significantly differentiated the groups at the short delay for the recall of famous facts (Fisher, $p = .05$). None of the normal control subjects made these errors, in either knowledge condition at the short delay. At the long delay, no significant differences were revealed for either famous or nonfamous fact conditions. Table 5 shows the frequency counts for the patient and normal control groups falling into the "poor performance" group for the classification of extra-experimental errors at the short delay for famous facts.

Group	Good Performance	Poor Performance
Patients	8	5
Normal Controls	11	0

Table 5. Number of individual subjects who made extra-experimental errors ("poor" performance group) when reclassified as defined above.

Visual inspection of the characteristics of individual subjects who made source errors revealed that of the patients, only those with right hemisphere damage (including the patient with bilateral frontal lobe damage) made intra-experimental source errors. Extra-experimental source errors were made by patients in both the RF and LF groups in all conditions but one, but only one patient with left hemisphere damage made these errors consistently. In the long delay condition for recall of famous facts, two patients with right hemisphere damage and three with left hemisphere damage made extra-experimental errors, as did three of the normal control subjects.

Pearson product-moment correlations were calculated between the neuropsychological measures and the recall measures including the source memory variables for all subjects. Although the alpha level of .001 more accurately represents the experiment-wise error, correlations at the .05 level will be reported in order to examine possible trends in the data. It was originally predicted that source memory would be correlated with classical measures of frontal lobe functioning including verbal fluency (F, A, S) and the Wisconsin Card Sorting Test. No significant correlation was found between measures of verbal fluency and source amnesia. The only significant WCST correlation was a negative correlation between intra-experimental errors made at the long delay for nonfamous facts and the number of nonperseverative errors made, $r = -.52$, $p = .01$. As the ability to recall the presenter source decreased, the number of nonperseverative errors increased. The number of intra-experimental errors made at the long delay for famous facts was correlated with the WAB-AQ, $r = .52$, $p = .01$. Hence, good performance on a test of language ability was positively related to the number of intra-experimental source errors.

Extra-experimental source errors made at the long delay for famous facts were negatively correlated with the Visual Memory Index, $r = -.53$, $p = .01$, indicating that committing a greater number of extra-experimental errors was associated with lower scores on the non-verbal or visual subtests of the WMS-R. This type of poor source memory was also negatively correlated with PIQ, for both famous, $r = -.64$, $p = .001$, and nonfamous facts, $r = -.84$, $p = .001$, again suggesting a relationship between extra-experimental source errors and presumed non-verbal processes.

Predictably, the ability to recall correctly the famous facts following a 15 minute delay (short delay) was correlated with all four of the WMS-R memory indices, Verbal Memory, $r = .69$, $p = .001$, Visual Memory, $r = .62$, $p = .001$,

General Memory, $r = .73$, $p = .001$, and Delayed Recall, $r = .65$, $p = .001$. Fact recall of familiar (although previously unknown) material appears to be related to classic anterograde memory processes. Famous fact recall at the short delay was also significantly correlated with VIQ, $r = .52$, $p = .01$, the Boston Naming Test, $r = .75$, $p = .001$, the WAB-AQ, $r = .63$, $p = .001$, and the Verbal Fluency Test, and FAS-A, $r = .49$, $p = .01$, suggesting that verbal abilities were heavily weighted in famous fact recall.

Recall of nonfamous facts at the short delay revealed a similar pattern of correlations among the WMS-R indexes (Verbal, $r = .62$, $p = .001$, General Memory, $r = .62$, $p = .001$, and Delayed Recall, $r = .64$, $p = .001$), although the Visual Memory Index was not significantly correlated, nor were any of the language or fluency measures. These differences may relate to disparate processing demands of familiar (famous) versus nonfamiliar (nonfamous) material. Fact recall at the long delay correlated only with the Delayed Recall Index, for nonfamous facts, $r = .52$, $p = .01$, and negatively with WCST-total number of cards correctly sorted. No significant correlations were revealed between recall of the famous facts at the long delay.

It was predicted that the performance of the patients would differ from that of the normal control subjects on several of the experimental and neuropsychological variables. Therefore, Pearson product-moment correlations were calculated for the patient group and the normal control group individually. In this directed analysis, the prediction of a relationship between intra-experimental source errors and a classic index of frontal lobe functioning was found. In the group of patients, the number of intra-experimental errors made at the long delay for nonfamous facts was negatively correlated with the WCST number of cards correctly sorted, $r = -.60$, $p = .05$, and positively correlated with the number of perseverative responses, $r = .80$, $p = .001$, and the number of perseverative errors, $r = .74$, $p =$

.01. The number of cards correctly sorted was negatively correlated with fact recall for the famous facts at the short, $r = -.60$, $p = .01$, and long delays, $r = -.84$, and with nonfamous facts at the long delay, $r = -.73$, $p = .01$.

The predicted relationship between the source memory measures and the FAS was not found in the patient group. However, the number of famous facts recalled at the short delay was significantly correlated with FAS-A, $r = .63$, $p = .05$. Interestingly, higher BNT scores were significantly related to a higher number of intra-experimental errors at the long delay for famous facts, $r = .67$, $p = .05$.

The ability to recall famous facts at the short delay was significantly correlated with the Verbal Memory Index, $r = .69$, $p = .01$, General Memory Index, $r = .80$, $p = .01$, and the Delayed Recall Index, $r = .69$, $p = .01$. The ability of the patients to recall the nonfamous facts at the short delay was also significantly correlated with the Delayed Recall Index, $r = .60$, $p = .05$.

In the normal control group, the number of intra-experimental errors made at the long delay for nonfamous facts was negatively correlated with the WCST number of perseverative errors, $r = -.64$, $p = .05$. The number of famous facts recalled at the short delay was also negatively correlated with the number of cards correctly sorted, $r = -.64$, $p = .01$.

In terms of their performances on classical memory measures, significant correlations were found between the number of famous and nonfamous facts recalled at the short delay, the number of nonfamous facts recalled at the long delay, and the Verbal Memory Index, $r = .72$, .74 and .82, $p = .01$. Additionally, famous and nonfamous fact recall was at the long delay was correlated with the Delayed Recall Index, $r = .64$ and .68, $p = .01$. The General Memory Index correlated only with the ability to recall nonfamous facts at the long delay, $r = .65$, $p = .03$.

Overall, significant correlations were found between classical measures of memory functioning and the measures of fact recall used in the current study, but these were most apparent at the short delay. The ability to learn (Verbal, Visual and General Memory Indexes) and remember (Delayed Recall Index) information on a standardized neuropsychological test was related to the ability to recall the famous facts after a 15 minute delay. Following a one week delay, only the Delayed Recall Index was reliably correlated with recall of the experimentally presented facts, and only for the nonfamous facts. Intact language abilities figured prominently in good famous fact recall at the short delay for all subjects, but good language abilities (as indexed by the BNT) were associated with a greater number of intra-experimental source errors for these facts at the long delay in the patient group. Intra-experimental errors were also associated with poorer performance on the WCST in the patient group at the long delay for nonfamous facts, particularly with perseverative errors and perseverative responses. This was not the case for the normal control subjects. Finally, extra-experimental source errors showed an inverse relationship with performance on nonverbal tests of intellectual functioning and memory, but only at the long delay.

Discussion

Deficits in the ability to recall the source of learned information have been reported to be associated with damage to the frontal cortex, and have also been shown to be related to frontal system dysfunction as indexed by performance on tests thought to be sensitive to frontal lobe functioning. The present study examined the ability of patients with relatively circumscribed damage to the frontal cortex to recall two different types of source of experimentally learned

information under several conditions. The first pertained to the ability of subjects to learn and remember fictitious facts about famous versus unknown, or nonfamous individuals. Second, performance was examined at two delay periods, one-half hour and seven days after the presentation of the facts. It was predicted that the patients as a group would be able to recall the famous and nonfamous facts at a level approximately equivalent to that of normal control subjects, but that they would have greater difficulty recalling the source of the material.

Results generally indicated that there were no statistically significant differences between the patient and normal control groups in terms of their ability to recall the experimentally presented facts. Subjects tended to recall more facts at the short delay than the long delay, and more famous than nonfamous facts, a pattern comparable to that shown by both young and old subjects in McIntyre and Craik's (1987) Experiment 2. These findings are also in keeping with several previous studies showing that patients with lesions of the frontal lobes are about as capable of aquisition and retention of information in memory studies as normal control subjects (Milner, 1971; Janowsky et al., 1989). However, as has been noted by other researchers (Shimamura et al., 1991), the level of recall of the patients with damage to the frontal cortex is consistently numerically lower than that of their control counterparts. Moreover, in a recent study examining recall and recognition in patients with focal lesions of the frontal lobes, significant recognition deficits were found in patients with left hemisphere and bilateral lesions of the frontal lobe. Some of these patients were shown to have mild decrements in language performance as indexed by the BNT. The authors postulated that the recognition impairment may have been the result of a verbal encoding deficit stemming from reduced ability to elaborately encode such material (Stuss, in press).

In the present study, compromised recall was most apparent in the short delay condition for the recall of famous facts (presumably the easiest condition), suggesting that the patient's are slightly less proficient at either encoding or retrieving the experimentally presented information. As subject's were not tested for the ability to recognize the correct response, it is not possible to determine which of these two alternatives is responsible; nevertheless, with the exception of paradigms utilizing list learning tasks (one of the most demanding memory tasks), previous research has demonstrated that when given the opportunity to choose among alternative responses, the recognition memory of patients with frontal lobe damage is not significantly different from that of normal control subjects (Janowsky et al., 1989), particularly if the lesions does not extend to the basal forebrain region, or if there is no language disturbance. There is no reason to suspect those findings in the present study.

Another important finding to emerge with possible relevance to the encoding deficit hypothesis relates to the ability of the patients to use level of knowledge (famous versus nonfamous) to learn and recall a fact. When the patients were compared as a group to the normal control subjects, overall fact recall was generally superior for all subjects for famous facts at the short delay; however, considerable variability was evident. In comparing the RF, LF and normal control groups, an interesting pattern emerged. Both normal control subjects and patients with lesions of the right frontal lobe recalled more famous than nonfamous facts overall. The recall of the subjects with left frontal lobe lesions was approximately the same for the two levels of knowledge. In addition, patients with lesions of the left and right frontal lobes recalled about as many famous facts at the short delay as the normal controls did in the nonfamous condition. However, while the level of recall of both normal control subjects and patients with right frontal lobe damage declined from famous to nonfamous facts, the recall of the group with left

frontal lobe damage remained at the same level-roughly equivalent to that of the normal controls for the nonfamous condition.

While it is possible that the patients as a group may have been less able to use knowledge of a personality to encode a fact, resulting in generally poorer recall of information in the famous condition, this seems unlikely. In fact, although there is considerable individual variability, especially within the group with left frontal lobe lesions, this group appears to be noticablely different from the normal control group only at the short delay for famous facts. Conversely, the patients with lesions of the right frontal lobes show the same pattern of decline as the normal control group, but have consistently lower scores.

Several factors may account for the unexpected finding of lowered recall specifically in the famous fact recall condition at the short delay in the left frontal lobe group. These patients were signficantly different from the normal control group in their performance on the Boston Naming Test, and significantly different from both their right-hemisphere-lesioned counterparts and the normal control group on the Western Aphasia Battery. Both of these measures were significantly correlated with correct fact recall for famous facts at the short delay. Numerous previous studies have attested to the preeminence of the left anterior cortical region in language abilities. Left frontal lobe pathology has been reported to result in deficits on verbal fluency tasks (Benton, 1968; Milner, 1963, 1964; Perret, 1974), as well as reduced word-finding ability, and depending on the area and extent of damage, a more complete aphasia syndrome (Alexander, Benson and Stuss, 1989; Stuss and Benson, 1986). Only one of the four subjects in the group with left frontal lobe damage scored in the aphasic range on the Western Aphasia Battery (WAB), and this was largely attributable to his reduced verbal output. With one exception, the remainder of his scores on this test were perfect or near perfect. All of the subjects in the left frontal lobe group had reduced verbal

fluency scores on the WAB, and two of the four scored in the impaired range on the Boston Naming Test. Although small subject numbers preclude drawing firm conclusions, deficits in the ability to make use of knowledge of, or familiarity with a personality at the time of encoding a fact may be related to subtle language deficits like those manifested in the present study. In fact, in the present study, the ability of the patients to recall famous facts at the short delay was related to both naming in response to a picture cue (BNT), and phonemic word fluency abilities (FAS-A).

Such a postulate is supported by the results of Stuss et al. (in press) who found a significant relationship between the ability to recognize previously presented word-list items and confrontation naming scores. Consequently, mild residual aphasia may result in decreased ability to profit from externally provided encoding aids (familiarity with an individual's name) which in turn impairs the ability to recall such material, even when the name is provided as a cue. Stuss et al. also found that their patient group demonstrated the greatest level of impaired recall for list learning on the list with the most intrinsic organization (that is, semantically related words which were presented consecutively, in blocks). Taken together, these results suggest that lesions of the left frontal cortical region may impair a subjects' ability to make use of semantic associations at the time of learning resulting in subtle, but appreciable compromises in overall level of recall.

Other evidence, however, points to a relationship between verbal abilities and the level of initial recall. Recently, both visual object naming (a task similar to the BNT) and a measure of auditory comprehension were found to be predictive of the number of words head-injured subjects initially learned in a list-learning paradigm. However, when recall on the delay trials was equated for initial learning, the verbal measures had little predictive power (Crosson, Cooper, Lincoln, Bauer and Velozo, 1993). The authors suggest on the basis of these findings that basic verbal

memory mechanisms may be intact, although verbal input and output channels might be dysfunctional. If such a postulate is correct, it suggests that subtle encoding deficits for verbal material should be understood as decoding deficits that result in a lower acquisition rate, but normal verbal memory abilities. In the Signoret and Lhermitte (1976) study which found that patients with frontal lobe lesions learned weakly associated word pairs poorly, improved performance was found when these patients were instructed in visual imagery. Such a manipulation may have helped these subjects overcome verbal decoding impairments, resulting in an absolute increase in their recall scores. Visual inspection of the data for the present group of patients with left frontal lobe lesions revealed that three of the four subjects had difficulty learning the unrelated word pairs from the WMS-R, but that those same subjects had no difficulty recalling what they had initially learned following a 20-30 minute delay. Clearly, in all of the work to date, acquisition of verbal material relies heavily on intact language abilities. But there is some suggestion that disordered language processes do not result in a more rapid rate of forgetting verbal material.

It has been suggested that patients with frontal lobe damage fail to encode information semantically (Moscovitch, 1982), although others (Freedman and Cermak, 1986; Shimamura et al., 1991) have suggested that combined frontal lobe dysfunction and amnesia are necessary to produce semantic encoding deficits. The sample of patients in the present study as a group were not amnesic; there were no significant differences between any of the groups on the four memory indexes on the WMS-R. However, as with level of fact recall in the experimental task, the group of patients with left frontal lobe lesions scored consistently lower than the normal control group, and the group of patients with right frontal lobe damage, on the Verbal Memory, Visual Memory and General Memory Indexes, but not on the Delayed Recall Index. The fact that the Delayed Recall Index scores of the

patients approached the absolute value of the normal controls suggests that frontal lobe damage may result in a slower rate of consolidation in memory which may, in turn, eventually result in a weaker memory trace. Future work could examine this issue by testing subjects' memory for such materials across a series of delay periods in order to better characterize the rate of consolidation versus decay.

The patients with right frontal lobe lesions were not significantly different from the normal control group in terms of the number of famous or nonfamous facts that they recalled either at the short or the long delay. In addition, they exhibited the same pattern of forgetting as the normal control group, although their scores were consistently lower across the conditions. Thus, they performed in a manner not unlike McIntyre and Craik's old subjects, some of whom were shown in a later study to exhibit impairments on two measures of frontal lobe functioning. Like the patients with left frontal lobe lesions, the RF group scores on the four WMS-R Indexes were consistently lower (though not significantly different) than those of the normal control subjects. However, as a group, the RF patients were not impaired on the either the BNT or the WAB. Further, of the eight patients with right frontal lobe damage, only two had difficulty learning and recalling the unrelated word pairs from the WMS-R. Differences were evident, however, in their Attention/Concentration Indexes from the WMS-R on which they were significantly different from the normal control group. Here again, small sample size prevents drawing firm conclusions, although the relatively greater numerical discrepancy between the patients with right frontal lobe lesions and the normal control subjects on fact recall, may be a result of a lower overall ability to attend, especially over a long period of time. As subjects were required to attend to the presented facts for a half an hour, deficits not apparent on classical memory tasks requiring attention over relatively short periods may have become more obvious. Damage to the right frontal cortex has been shown to be related to decreased

ability to direct attention, impaired selective attention mechanisms, and reduced capacity for sustained attention (Stuss, Eskes and Foster, in press). Of particular relevance to the present study, impaired ability to sustain attention, especially when the stream of information is relatively slow and uninteresting was shown to be related to damage to the frontal cortex, especially to the right hemisphere (Wilkins, Shallice and McCarthy, 1987). In fact, the patients with right hemisphere damage performed significantly more poorly on the Attention/Concentration index than the normal control subjects. Nevertheless, as differences in overall fact recall were not statistically meaningful, and no significant correlations emerged between the Attention/Concentration Index and measures of recall in the present study, future work will be required to investigate and substantiate such hypotheses.

The main focus of the present experiment was to explore differences in source memory between normal control subjects and patients with documented frontal lobe lesions who were not classically amnesic. The pattern of fact recall demonstrated by the patients in the present study was similar to that exhibited by McIntyre and Craik's young subjects as noted above, yet their level of fact recall was lower than their sample of old subjects. Examination of their recall of source (conditionalized on fact recall) revealed no statistically signigicant differences between the patient group as a whole and the normal control group. Subjects were better at recalling both the intra- and extra- experimental sources at the short delay and for famous facts, than for either famous or nonfamous facts at the long short delay. Although the finding failed to reach significance, there was a trend for the patients with right frontal lobe lesions to do worse at the short delay than either the patients with left frontal lobe lesions or the normal control group. In comparing the pattern of source errors between the patient group and the normal control group, there were significant main effects of delay and knowledge, indicating that

subjects made more intra-experimental errors at the long delay, and for nonfamous facts, but there were no significant group effects.

The present results yield some interesting information about the patterns of forgetting between patients and normal subjects. The normal control subjects in the present experiment, like those in previous, similar studies (Schacter, 1984; McIntyre and Craik, 1987) tended to make the most intra-experimental errors when they had no prior knowledge of a subject (nonfamous facts) and when tested at long delays; a pattern of forgetting which parallels their forgetting of fact information. In contrast, the patients tended to make the greatest number of intra-experimental errors on the famous facts, even when tested after a relatively brief delay (30 minutes). In other words, even when their memory for facts is relatively good, and equivalent to the normal subjects, disproportionate deficits in source recall become apparent. While this finding failed to reached conventional significance levels in the present study, the means are in the same directions as those found by Janowsky et al. (1989) for their group of patients with lesions of the frontal lobes. They too reported considerable individual variability in their subjects. As in the present research, these performance differences may be related to discrepancies in lesion size and boundary. Due to small sample size, it was not possible to address the issue of intra-hemisphere functional specialization.

In an attempt to understand the variability, individual profiles of response were analyzed. Nonparametric analysis revealed that only four of patients made intra-experimental errors to a significant degree on the famous facts at the short delay. While none of these patients were classified as "good performers", four of these patients had right hemisphere lesions. Moreover, the patients with right hemisphere lesions made these errors when recalling both famous and nonfamous facts, at both the short and long delays. Normal control subjects, on the other hand, rarely made these errors for famous and nonfamous facts at the short delay.

Consequently, in the normal control subjects, forgetting of the contextual information parallells forgetting of the facts themselves, although the absolute value differs. However, some of the patients with right hemisphere lesions recall more facts than sources.

Due to small sample sizes, it is difficult to address the issue of hemispheric specialization. It appears that only a few of the eight subjects comprising the group of patients with right frontal damage were impaired in terms of source recall of modality (intra-experimental source). Despite the largely verbal nature of the task, the LF group did not commit a meaningful number of intra-experimental source errors.

Extra-experimental errors were made only rarely by normal control subjects (.00 at the short delay for both levels of knowledge; .06 and .09 for famous and nonfamous, respectively at the long delay). In contrast, a group by delay interaction revealed the patients with lesions of the left frontal lobes made more of these errors at the long delay than any other group at either delay (.44 for famous facts, and .25 for nonfamous facts, at the long delay). Nonparametric analysis revealed that patients with frontal lobe damage could be classified as "poor performers" on the basis of their poor extra-experimental source recall. Visual inspection of the data suggests that damage, particularly to the right frontal lobe, is associated with poor recall of the source of a learning experience, even when the fact is successfully recalled. The considerable individual variability apparent in the data suggests that not all subjects with damage to frontal cortex make these errors. Those subjects who did commit source errors did not appear to be noticably different in a consistent fashion when compared to other subjects on any of the neuropsychological measures under study. Five of the patients made extra-experimental errors at the short delay when recalling famous facts. However, of these five patients, four had right hemisphere damage, and only one left

hemisphere damage. Interestingly, this individual made extra-experimental source errors in all conditions except for the recall of nonfamous facts at the short delay. Descriptively, he did not appear to be systematically different in his performance on any of the other neuropsychological measures collected. A systematic analysis of the factors contributing to this small finding was not possible in the present study.

As in previous source memory studies, source recall performance was extremely variable in all subjects, but most notably in the patient group. No clear pattern of disordered cognitive processes emerged when the subjects were divided into groups on the basis of those making source errors and those who generally performed well. However, four of the patients made more intra-experimental errors than other subjects at the short regardless of level of knowledge, and only one of these made any extra-experimental errors, and only at the short delay for famous facts. These subjects all had right hemisphere lesions. Although they were not the most impaired individuals on any of the recall or neuropsychological measures, they tended to be impaired on the Attention/Concentration Index of the WMS-R, and as a group they were significantly different from the normal control subjects. As previously mentioned, damage to right frontal cortex has been shown to result in compromised attentional processes (Stuss et al., in press). Recently, increased activation of regional cerebral blood flow in mesial and bilateral frontal regions was found during performance of a sustained attention task (Continuous Performance Test) in normal control subjects (Rezai, et al., 1993). While it seems plausible that reduced attentional capacity is related to poorer ability to encode information, and particularly to the encoding of context, which is poorly recalled even by healthy, young control subjects. However, in the present study, no relationship emerged between attention as measured and source memory errors, perhaps due to the small sample size, and the degree of variability.

It was hypothesized that significant relationships would be found between the source memory measures and classsical measures of "frontal lobe functioning", the WCST and the FAS. No relationship was found between the WCST or the FAS and measures of source amnesia in the entire current subject population, or in the patient groups alone. However, when examining zero-order correlations within the patient group, it was found that intra-experimental source errors at the long delay for famous facts correlated positively with the WCST-number of perseverative errors. The opposite pattern emerged for the normal control subjects. In this group, the greater the number of intra-experimental errors, the fewer perseverative errors made. While the reason for the differential relationship of WCST variables to source memory performance between the patient and normal control group is unclear, it does suggest a partial relationship between classic frontal lobe functioning and source memory.

Experiment 2

Method

It has been previously shown that patients with lesions of the frontal lobes perform poorly in tasks which involve the use of spatial cues to encode such as reproducing a series of hand movements and arm positions in the correct order (Milner and Kolb, 1985), or when associations must be made between spatial cues. Patients with damage to either the right or left frontal lobes perform poorly on these types of tasks (Petrides, 1985a). However, little is known about the ability of these subjects to remember the source(s) of a learning situation which is largely spatial in nature and whether such processes are dissociable from those observed in verbal tasks. The procedures and materials used in the present experiment were modified from studies examining memory for spatial item and spatial location in normal elderly and young subjects (Cherry et al., 1990; Sharp and Gollin, 1987; 1988). In these studies, forty objects were presented on one surface for subsequent item recall and relocation. The aim of the present study was to attempt to elicit source errors when memory for the spatial location of a series of objects was tested. To achieve this, two study surfaces were used instead of one, and the forty objects were evenly distributed between the two study surfaces. Subjects were required to recall the placement of a series of objects on these two similar but spatially distinct study surfaces.

As little is known about the conditions under which such errors might occur, or about the precise factors that contribute to spatial-temporal context, an attempt was made to control for non-spatial factors known to affect the performance of patients with frontal lobe damage. For example, requiring subjects to recall the distinct placement of objects in two separate rooms would necessarily introduce other cues, such as time (to get from one room to the other), in addition to global spatial cues about the location of each of the arrays of stimuli. To control for

these factors while utilizing two separate study surfaces, each of the surfaces selected for use in the present experiment were approximately one half the size of those used in the studies mentioned above (i.e. two study surfaces instead of one room), and half of the number of objects were placed on each surface. In this manner, subjects in the present study were required to learn the same relative amount and type of material, but had an additional task in associating half of the to-be-learned material to the left-half of the experimental location, and the other half to the right.

Materials

The materials consisted of a stimulus set of items to be recalled and two separate surfaces on which the stimulus items were placed for study. The stimulus set consisted of 50 small objects (40 to be used at the learning session, and ten new objects to be used at the time of recall) which could be found in most households (e.g. lipstick, small light bulb). Objects were selected on the basis of two criteria. The first was that they be difficult to categorize according to any meaningful classification system. Second, to ensure that the objects used in the present study were easily identifiable, 60 objects were rated initially for recognizability on a scale of 1 to 10 by a pilot group. A rating of "1" represented "completely unfamiliar", and a rating of "10" represented "immediately recognizable". The pilot group comprised the same 42 individuals who established familiarity ratings for the "famous" names used in experiment 1. In this manner, 50 objects with ratings of "8" and over (deemed to be immediately recognizable to all subjects, were included in the present study. Forty of these 50 objects were randomly divided into two groups and were situated on one of two study surfaces. The remaining 10 objects were used at the time of recall to ensure that some items were available for replacement which were not used at the encoding phase.

The study surfaces were two matte boards measuring approximately .60 m X .60 m. Ten photocopied pictures of furniture and appliances were placed on each of the boards. These articles were evenly spaced over the entire surface of the board. An attempt was made to achieve two distinct groupings of articles; the board on the left of the subject contained furniture and appliances typically found in the kitchen/dining area and bedroom, while the board to the right contained living/entertainment room articles.

The placement of the objects was studied under two conditions. In the plain condition, objects were placed on either of two white matte boards. Subjects were also tested in an enhanced condition in which colour was used to attempt to improve subjects' memory for the correct placement of objects. In this condition, the matte boards were identical in every respect to those in the plain condition, with the exception that one was bright yellow and one was bright blue. The same schematic pieces of furniture were placed in the same positions in the plain and enhanced conditions.

Procedure

Subjects were tested individually by the same experimenter. They were read the following instructions prior to viewing the boards:

" You will be shown a series of objects on two surfaces. I am going to place a number of objects on two different surfaces and you should try to remember the specific location of each of these objects-that is, the exact position on the board, as well as which board it was placed on. Do you have any questions?"

The two boards were placed side by side on a table measuring 3m X 5m, separated by a distance of 1 meter. Subjects were asked to stand at the center of

the table. The experimenter was situated across from the subject at the center of the table so that each of the matte boards was one-half meter to either her left or right. An object was placed by the examiner beside a schematic piece of furniture on one of the two boards, named aloud, and pointed to for five seconds, following which it was removed from the board. This procedure was repeated for each of the 40 objects. The order of presentation, and placement of objects on either the left or right board, was randomized across subjects. Following the acquisition phase, subjects performed a distractor task (Digit Span and/or Digit Symbol subtests of the WAIS-R) for five minutes before recall.

The enhanced condition was identical to the plain with the exception that the two boards were coloured as described above. The placement of objects upon these surfaces followed the same procedure as above, and was randomized for each subject. At least six days separated testing in the plain and enhanced conditions.

Two types of recall were tested. In the no-cue condition, (following the five minute delay) out of subjects' view, the two boards were replaced with identical boards without schematic furniture, and the 40 experimental objects, as well as the 10 new objects were placed randomly on the table below the boards. Subjects were informed of the presence of the 10 new objects. Subjects were then asked to replace the objects they had just seen the experimenter place, in as close to the original positions as possible.

The cued-condition immediately followed the no-cue condition for all subjects. In this condition, the boards with the schematic furniture were replaced, out of the subjects view, and they were requested to replace all objects a second time. The 50 objects were randomly arrayed below the boards as in the no-cue condition. No time limit was imposed, but all subjects completed the replacement within ten minutes.

Scoring was completed by devising two 8½" x 11" replicas of the boards and furniture. Each of the boards was divided into eight equal squares, and the arrangement was reproduced on 8½" x 11" sheets of paper. These grids were used at the recall session by locating the subject's placement of an object on the board in the exact position on the 8½" x 11" grid. The subjects' responses were scored at a later date using transparent grids duplicating the placement of schematic furniture as scoring keys. An object was considered correct if positioned on the correct board, and also not more than half the distance from the correct piece of schematic furniture and the nearest position of another piece of schematic furniture.

Source errors

An intra-experimental error was defined as an object which was placed on the correct surface, but in the wrong position within the board. Extra-experimental errors were considered to be those occurring when a subject placed one of the ten not-previously seen objects on one of the two boards. A third type of error was classified as an experimental error and occurred if a subject replaced one of the forty original objects in a completely incorrect position-that is, on the wrong board.

Results

This task proved to be a difficult one for all subjects. Table 6 shows the mean number of items placed on the correct board and in the correct position for each group in the no-cue and cue conditions and in the plain and enhanced conditions. Overall, it appears that subjects recalled the placement of roughly half the objects, regardless of whether cues were presented, or whether the learning situation was

enhanced through the use of colour. These results are similar to those reported by Cherry et al. (1990) and Sharp and Gollin (1987; 1988) for overall level of recall.

			Correct Responses			
			Experimental Objects (N=40)		New Objects Not Placed (N=10)	
Condition	**Board**	**Group**	**No Cue**	**Cue**	**No Cue**	**Cue**
Plain	**Right**	Right F.L.	11.5 (4.1)	10.1 (5.5)	10.0 (.00)	9.8 (.46)
		Left F.L.	8.3 (4.3)	8.0 (4.8)	9.8 (.50)	9.8 (.50)
		All Patients	10.4 (4.1)	9.6 (5.0)	9.9 (.23)	9.8 (.44)
		N. Control	12.0 (4.1)	10.3 (4.4)	9.7 (.65)	9.8 (.60)
	Left	Right F.L.	9.5 (5.4)	10.0 (5.3)	--------	--------
		Left F.L.	8.0 (4.6)	7.3 (4.!)	--------	--------
		All Patients	8.8 (4.8)	9.0 (4.7)	--------	--------
		N. Control	11.0 (4.4)	11.2 (4.3)	--------	--------
Enhanced	**Right**	Right F.L.	11.0 (5.3)	10.9 (5.6)	9.8 (.46)	9.9 (.35)
		Left F.L.	10.3 (6.2)	10.0 (6.7)	9.3 (1.5)	9.3 (1.5)
		All Patients	10.5 (5.2)	10.5 (5.5)	9.6 (.87)	9.7 (.86)
		N. Control	10.2 (3.8)	10.5 (3.4)	9.9 (.30)	9.8 (.41)
	Left	Right F.L.	10.4 (3.6)	9.4 (5.3)	--------	--------
		Left F.L.	9.8 (7.1)	10.8 (7.3)	--------	--------
		All Patients	9.7 (4.8)	9.4 (5.8)	--------	--------
		N. Control	10.6 (4.4)	11.2 (4.4)	--------	--------

Table 6. Mean number of objects (SD) placed on the correct board and in the correct position, and "new objects" placed on either of the boards, for both the plain (white matte boards) and enhanced (coloured boards) conditions, with and without cues (schematic furniture) for the patients with right frontal lesions (Right F.L.), and left frontal lobe (Left F.L.) lesions, the patients as a group (including the subject with bilateral frontal lobe damage) and the normal control group.

These observations were confirmed in repeated measures analysis of variance. Separate analyses were conducted on each of the six possible response types (See Appendix 6). There were no significant differences between the patient groups (RF, LF, all patients) or the normal control group in terms of the number of items

they placed on the boards correctly irrespective of learning or testing conditions. Thus, the patients were as capable of correctly recalling the position of an object on each of the boards as the normal control subjects, whether cued or uncued, or in the plain or enhanced condition. Figure 7 illustrates the level of recall for each of the groups in each of the two learning and recall conditions.

Figure 7. Mean number of objects placed in the plain and enhanced conditions for the two groups of patients and normal control subjects.

In order to determine whether the patient and the normal control subjects differed in their pattern of forgetting the placement of objects, the number of experimental objects not placed on either the left or the right board was compared. This analysis yielded a significant group by board interaction, $F(1, 22) = 4.32$, $p=.50$. The patients did not attempt to place a greater number of experimental objects from the board to their left, $p=.05$. Additionally, there was a significant three-way group by context by cue interaction, $F(1, 22) = 6.05$, $p = .02$. The patients left a significantly greater number of objects unplaced in the plain condition regardless of whether or not they were cued, and in the enhanced condition when they were cued, than did the normal control group, $p=.05$.

In comparing the performance of the RF and LF patient groups individually to the normal control group, the group by board interaction remained significant $F(2, 20) = 3.97$, $p = .04$, confirming the fact that the RF and LF groups left more unplaced objects from the left board than the normal controls did from either the right or left boards. The LF group left a significantly greater number of objects unplaced from the left board than from the right board, and from the normal controls on either the left or right boards, $p=.05$. Additionally, the three-way group by context by cue interaction remained significant, $F(2, 19) = 3.51$, $p = .05$. Figure 8 shows that the LF group left more experimental objects unplaced in the plain condition, with or without cues, than the normal control subjects did in all conditions except the plain, cued condition. The RF group only left a significant number of objects unplaced in the plain, no cue condition, when compared to the normal controls. The two patient groups did not differ from each other in their performance on this measure; the post-hoc analyses were not significant.

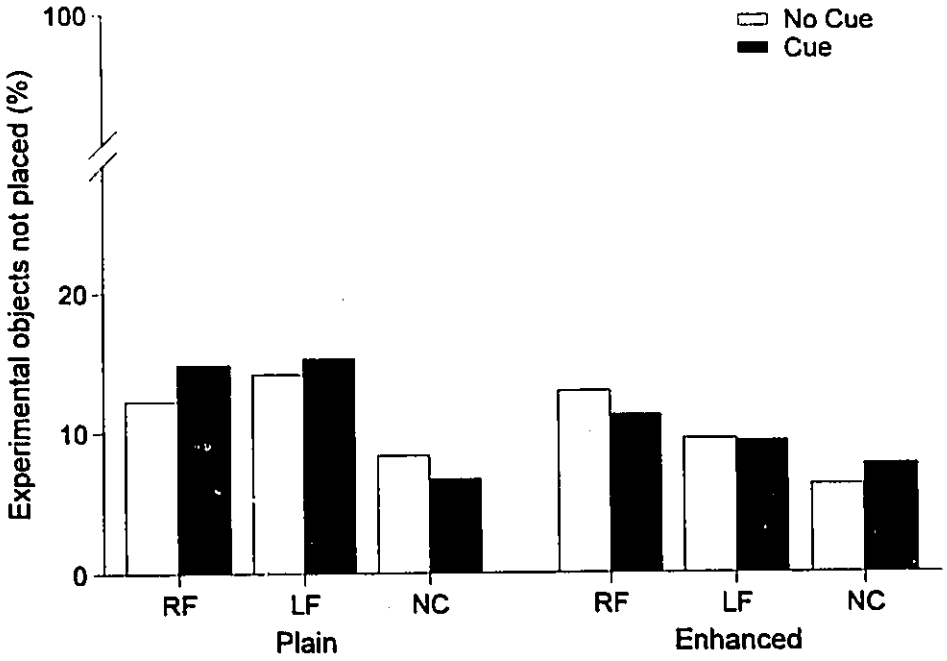

Figure 8. Mean number of experimental objects that subjects did not attempt to place in the plain and enhanced conditions, with and without cues.

Subjects were informed at the time of recall that ten *new* objects had been added to the array of experimental objects that they were asked to replace. A correct response was scored for any of these ten new objects not placed on either of the boards. The only significant comparison between the patient group and the normal control group for this contrast was the group by context by cue interaction $F(1, 22) = 5.66$, $p = .03$, which failed to reach the .05 significance level in post-hoc analysis.

Figure 9 shows the differential effects of cue and context (collapsed across the right and left boards) on the ability of the RF, LF, and normal control groups to reject the ten new objects, $F(2, 20) = 4.12$, $p=.03$. Post-hoc analysis confirmed that in the plain condition, in both the uncued and cued trials, the groups were equally proficient at rejecting the new objects. When colour was added to the learning situation in the enhanced condition, the performance of the RF and

normal control groups was significantly better than the performance of the LF group in both the cued and uncued conditions. In fact, while none of the groups benefited from the addition of colour, the LF group performed significantly more poorly in this condition than they did in the plain condition. Furthermore, their mean scores in the enhanced condition were lower than either of the other two groups irrespective of condition or cue. The use of the schematic furniture to aid recall did not improve subjects' performance.

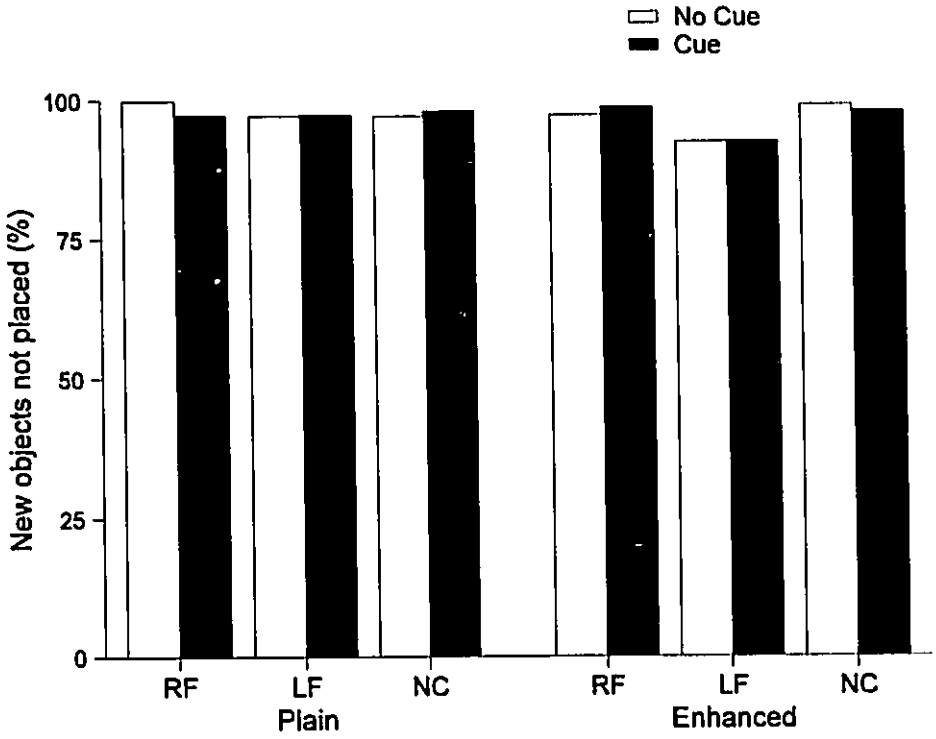

Figure 9. Percentage of new objects subjects did not attempt to place on either the right or left board, under both plain and enhanced conditions.

Roughly half of the experimental objects were not correctly placed. These objects could have been placed on the correct board in the wrong position (intra-experimental error), placed on the wrong board (experimental error), or left unplaced (forgetting). Placing a new object on the board constituted an extra-

experimental error. The mean scores of each of the groups on the four possible types of errors are presented in Table 7.

			Incorrect Responses							
			Experimental Objects						New Objects	
			Not Placed (forgetting)		Intra-Experimental		Experimental Errors		Extra-Experimental Errors	
Condition	Board	Group	No Cue	Cue	No Cue	Cue	No Cue	Cue	No Cue	Cue
Plain	Right	Right F.L.	5.4 (4.7)	4.3 (4.1)	2.0 (1.3)	3.0 (2.6)	1.1 (2.1)	2.6 (2.1)	.00 (.00)	.25 (.46)
		Left F.L.	4.8 (2.5)	4.0 (2.9)	4.0 (.82)	4.5 (1.7)	3.0 (2.4)	3.5 (1.9)	.25 (.50)	.25 (.50)
		All Patients	5.1 (3.8)	4.0 (3.6)	2.8 (1.6)	3.6 (2.3)	1.6 (2.2)	2.8 (2.0)	.08 (.28)	.23 (.44)
		N. Control	2.7 (2.1)	2.7 (2.1)	3.4 (2.3)	3.6 (1.8)	1.9 (1.9)	2.3 (1.8)	.72 (1.6)	.18 (.60)
	Left	Right F.L.	6.5 (4.8)	5.6 (5.6)	2.6 (2.8)	2.0 (1.4)	1.4 (1.8)	2.4 (2.4)	------	------
		Left F.L.	7.5 (4.2)	7.3 (4.2)	3.0 (.82)	4.0 (1.4)	1.5 (1.0)	1.5 (1.3)	------	------
		All Patients	6.3 (4.6)	5.7 (5.1)	3.2 (2.8)	3.2 (2.4)	1.6 (1.7)	2.2 (2.0)	------	------
		N. Control	2.5 (2.3)	2.6 (2.3)	3.9 (2.8)	3.9 (3.0)	2.5 (2.0)	2.3 (1.8)	------	------
Enhanced	Right	Right F.L.	4.3 (4.2)	5.1 (5.7)	2.9 (1.9)	2.9 (1.9)	1.9 (1.8)	2.0 (1.5)	.25 (.46)	.13 (.35)
		Left F.L.	3.5 (5.1)	3.0 (5.4)	4.5 (1.0)	4.5 (1.0)	1.8 (1.7)	1.8 (1.7)	.75 (1.5)	.75 (1.5)
		All Patients	4.1 (4.1)	4.4 (5.1)	3.4 (1.7)	3.0 (2.4)	2.0 (1.7)	2.2 (1.7)	.39 (.87)	.30 (.86)
		N. Control	3.4 (2.0)	2.9 (1.5)	3.6 (2.6)	3.8 (2.5)	2.8 (1.4)	2.7 (1.6)	.09 (.30)	.18 (.41)
	Left	Right F.L.	4.8 (4.2)	5.1 (5.3)	2.1 (2.3)	2.1 (2.3)	2.8 (1.8)	2.6 (1.8)	------	------
		Left F.L.	4.0 (7.3)	4.8 (6.9)	4.3 (1.5)	4.3 (1.5)	2.0 (1.6)	3.3 (.50)	------	------
		All Patients	4.5 (4.9)	5.0 (5.3)	3.1 (2.3)	3.1 (2.3)	2.7 (1.8)	2.7 (2.5)	------	------
		N. Control	7.7 (2.0)	2.5 (1.6)	4.5 (1.8)	4.5 (1.8)	2.1 (2.4)	2.6 (2.5)	------	------

Table 7. Mean number of source errors (SD) for the patients with right frontal lesions (Right F.L.), and left frontal lobe (Left F.L.) lesions, the patients as a group (including the subject with bilateral frontal lobe damage) and the normal control group.

When subjects placed one of the ten new objects on either the left or the right board an extra-experimental error was committed . Subjects made, on average, fewer than one of these errors, regardless of condition. No effect was reliably significant when the patients were compared to the normal controls, or to themselves, although the group by context by cue comparison approached significance F (1, 22), = 3.97, p = .06. The pattern of errors committed by each of the groups is represented in Figure 10.

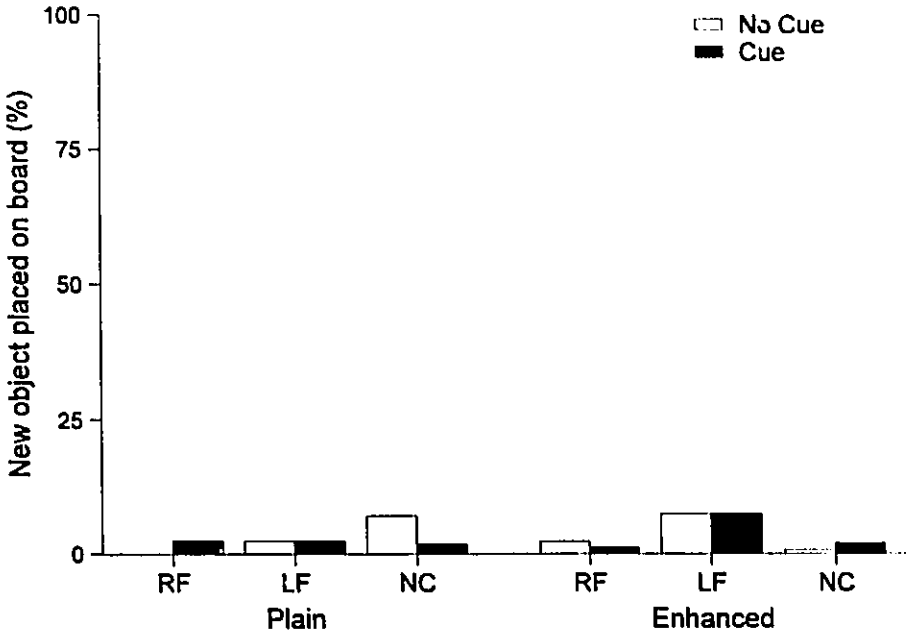

Figure 10. Percentage of extra-experimental errors made by the two patient groups and the normal control subjects.

The remainder of the experimental objects would either have been placed in the wrong position on the correct board (intra-experimental error), or on the wrong board (experimental error). For the contrast of intra-experimental errors between the total patient group and normal control subjects, there was a significant interaction of context and cue, $F(1, 22) = 6.38$, $p = .02$. Post-hoc analysis revealed that subjects placed more objects on the correct board, but in the incorrect position in the plain, cued condition than in the no cue condition or in the enhanced cue condition. Also, more of these errors were made in the enhanced no cue condition than in either enhanced cued, or plain no cue condition. Subjects incorrectly attempted to determine the exact placement of an object within a board more often in the plain condition when furniture cues were present, and more often in the enhanced condition when they were not. When all patients were compared to the control group, no group effects were found for this measure.

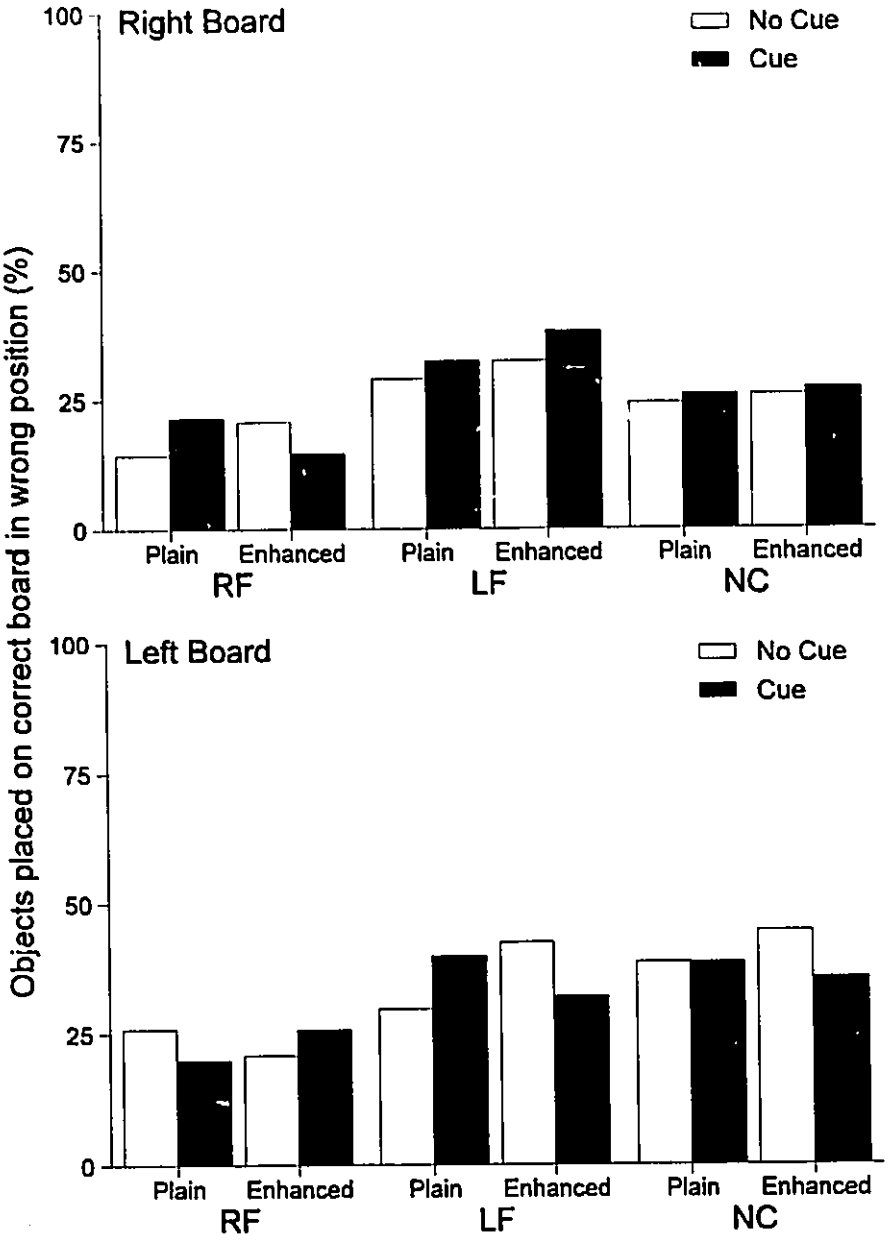

Figure 11. Percentage of intra-experimental errors in the plain and enhanced conditions on the right and left boards.

In comparing the two patient groups (RF and LF) and the normal control group, the four-way interaction of group, context, cue and board was significant, F (2, 20)

= 4.53, p=.02. Post-hoc analysis demonstrated that in the enhanced cued condition, the LF group incorrectly placed more objects within the board to the right of their bodies than the RF group did in either the plain, uncued, or the enhanced, cued conditions. Additionally, the number of incorrect right board placements made by the LF group in the enhanced, cued condition was greater than the number incorrect placements made by the RF group in the plain, cued condition, or the enhanced uncued condition to the left of their bodies. The LF group made no more errors placing objects within the board to the left of their bodies in any of the conditions than either of the other two groups (Figure 11). The significant interaction of context and cue did not achieve significance in post-hoc analysis.

Finally, the fourth type of source error investigated was defined as an experimental error. An object from the board positioned to the right of a subject placed on the board positioned to the left of subject (and vice versa) was scored as an experimental error. The total number of such errors committed in each learning and recall condition was entered into the analysis of experimental errors. Analysis of this data yielded a significant main effect of cue for the patient versus control comparison, $F(1, 22)$ =6.44, p=.02; however, the means were not reliably different when post-hoc analysis was completed. A significant three-way interaction between group, context and board, $F(1, 22)$ =5.31, p=.03, also failed to yield significant comparisons between the means in post-hoc analysis. There was a significant three-way interaction between context, cue and board, $F(1, 22)$ = 5.2, p=.03. Post-hoc analysis revealed that all subjects generally made fewer experimental errors on the right board in the plain, uncued condition than in the plain, cued or enhanced, cued conditions on the right board, or in the enhanced cued or uncued conditions on the left board. Additionally, fewer such errors were made in the plain, uncued condition on the left board, than in the enhanced cued

condition on the left board, $T(8, 22) = .60$, p=.05. Thus, in general subjects performed better in the plain conditions, especially without cues and somewhat better in the uncued conditions overall (Figure 12).

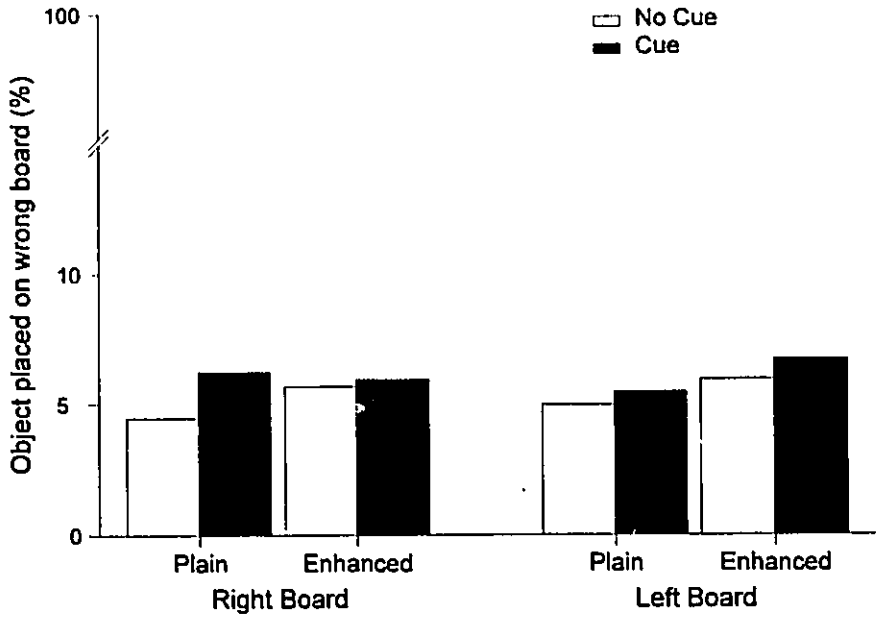

Figure 12. Percentage of experimental errors made by all subjects in the plain and enhanced condition with and without cues.

In comparing the RF group to the LF group and the normal controls, there was significant three way interaction between group, context and board $F(2, 20) = 4.24$, $p = .03$. Figure 13 shows the pattern of experimental errors made by subjects in the plain and enhanced conditions on the left and right boards and illlustrates that the LF group made more experimental errors on the right board in the plain condition, than they did on the left board, or on the left and right boards in the enhanced condition, $p = .05$. The three way interaction of context by cue by board was significant, $F(1, 20) = 4.54$, $p = .05$, as was the group by context by cue interaction, $F(2, 20) = 3.60$, $p = .05$; however, the means were not significantly different in post-hoc analysis.

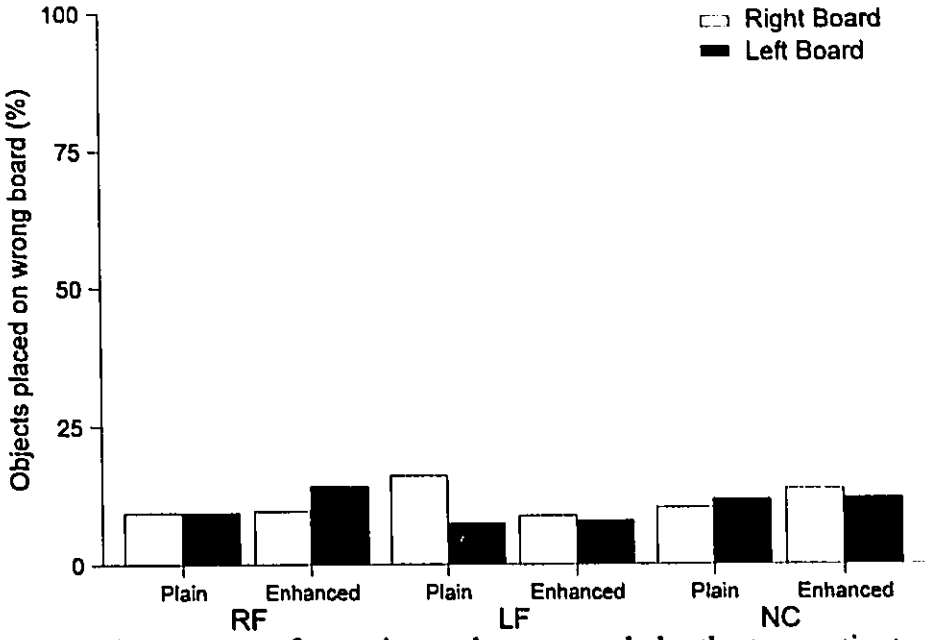

Figure 13. Percentage of experimental errors made by the two patient groups and the normal control subjects.

Pearson product-moment correlations between the experimental and neuropsychological variables revealed that the ability to recall the correct placement of an object was negatively correlated with the WCST-number of perseverative responses and perseverative errors in both the plain and enhanced conditions, $r = -.49$ to $-.59$, $p = .01$. In the plain condition, object recall was positively correlated with FAS, F, FAS, S, and FAS total score, $r = .49$ to $.59$, $p = .01$.

The ability to discriminate the ten new objects from the experimentally placed objects was correlated with the BNT in the enhanced condition only, $r = .51$ to $.56$, $p = .01$. Intra-experimental errors (placing an object on the correct board, in the wrong position) were not correlated with any of the neuropsychological measures. Experimental errors (placing an object on the

wrong board) were negatively correlated with the BNT, only in the plain condition, r = -.48 to -.59, p = .01.

The number of experimental objects left unplaced (forgetting) was negatively correlated with the WCST-number of correct category sorts, and positively with the number of perseverative errors and responses both in the plain and enhanced conditions.

There were also a number of interesting correlations between the classical memory measures (WMS-R) and the spatial memory variables. Recalling the placement of the objects was significantly correlated with the Visual Memory, General Memory and Delayed Recall Indexes in the plain conditions, r = .51 to- .54, p = .01, and with the Verbal Memory, General Memory and Delayed Recall Indexes in the enhanced conditions, r = .66 to .75, p = .001. Conversely, the number of intra-experimental errors made was negatively correlated with the Verbal Memory and General Memory Indexes only in the plain conditions, r = -.51 to -.59, p = .01.

Discriminating a new object from the experimental objects was correlated with the Visual Memory General Memory Indexes only in the enhanced conditions, while forgetting to place an object was negatively correlated with the Visual Memory and Delayed Recall Indexes in the enhanced conditions, and in the plain condition only when cues were provided.

Due to the large number of variables and conditions, the patient and normal control group's pattern of correlations was not examined individually.

Discussion

The ability of subjects to recall the spatial location of objects on two spatially distinct environments was examined for overall fact recall (object placement) and source recall (correct placement within the board, as well as discrimination between experimental objects and new objects). It was predicted that there would not be differences between the patient and normal control groups in terms of overall correct placement, but that they would differ in their pattern of errors in that they would have more difficulty recalling the source of more of the objects than the normal control group.

In general, all subjects recalled the correct placement of approximately one half of the objects. These data compare well with those obtained by Cherry et al. (1990), and Sharp and Gollin (1987; 1988) in their group of normal control subjects. In the present study, two learning surfaces were used instead of one, and the overall number of correctly placed objects were approximately evenly distributed across each of the two boards. The lack of significant group differences indicates that there is no appreciable spatial memory deficit in the patient group, and that the groups are reasonably equivalent in this regard. These findings compare well to previous studies describing no spatial memory deficits in patients with damage to the frontal cortex (Smith and Milner, 1984b).

In order to examine the relative contribution of spatial location and enviromental cues, subjects were first asked to replace the experimental items without the aid of the schematic furniture which had been used at the initial learning session (no cue condition). Both patients and normal controls recalled the distinct placement of roughly the same number of objects without the use of the schematic furniture as cue as when they were able to use it as an aid. As all subjects received the no cue condition first, it is possible that the use of schematic

furniture was not beneficial because the additional passage of time had degraded the memory of the object layout. However, it seems equally plausible that when verbal cues are absent, or not particularly useful (as in the present study where there were no obvious associations between items of furniture and study objects), successful recall of this material will rely heavily on the spatial attributes of the environment. Consequently, the addition of the schematic furniture as cues did not improve subjects' recall scores, and may have been in some cases mildly, though not significantly, detrimental.

The context of the learning environment was also manipulated by adding colour to the study surfaces. This contextual enhancement did not result in any increase in subjects' memory for the placement of objects. The normal control subjects and the patients with frontal lobe lesions recalled as many objects when they were initially presented on the white matte boards as when they were presented on the distinctly coloured boards. These findings are somewhat at odds with those of Winocur and Kinsbourne (1978) who found that enhancing the learning environment using coloured lights and classical music did little to enhance the recall of normal control subjects, but did improve the recall of amnesic subjects. The group of patients with frontal lobe lesions in the current study were not amnesic on the basis of their scores on the WMS-R, nor were significantly different from the normal control group on most of the recall measures used in the present study. The finding that enhanced context did little to improve their recall of the placement of objects in the present study may indicate their ability to encode this type of contextual feature is comparable to that of normal subjects. Other authors (Mayes, Meudell and Som, 1981) have also found that testing normal control subjects in unusual environments did not enhance their performance, even when memory was tested at long delays.

"Forgetting" refers to the objects which subjects did not attempt to place on the boards. Examining the pattern of forgetting across the left and right boards between the patients and the normal control group showed that the patients did not attempt to place more of the objects from the left board, and more objects remained unplaced in the plain condition, regardless of whether they were cued with schematic furniture. The converse observation is that more incorrect attempts were made in the enhanced condition. Both the RF and LF groups contributed to this finding, although the LF group tended to be slightly more likely to leave objects unplaced. If these results are compared to the recall scores, it becomes apparent that enhancing the learning situation did improve recall, but rather invited more attempts, albeit incorrect ones.

The LF group also had difficulty in discriminating between not-previously seen objects and those used in the experiment, primarily when colour was added to the learning situation. This finding adds additional support to the above postulate that colour enhancement make these subjects more likely to attempt a placement, that is, it resulted in a slight "yea saying" bias. As the number of new objects they attempted to place on either the right or left board was small (an average of about 1), it was not possible to discover what learning and recall conditions affected their performance.

Two types of placement errors for the experimental objects were possible. If an object was placed on the correct board in the wrong position, an intra-experimental error was committed. Placing an object on the wrong board constituted an experimental error. Examining what types of errors were committed when subjects attempted to determine where an experimental object should be placed revealed an interesting interaction between cued recall and enhanced context. There were no significant group (patient versus normal control comparison) differences. All subjects incorrectly attempted to determine the exact placement of

an object within a board more often in the plain condition when furniture cues were present than when they were not, or than in the enhanced condition when cues were present. Conversely, when colour was used to enhance learning, more intra-experimental errors were made without the use of schematic furniture than with, and more than in the plain condition when no cues were used. This suggests that in the plain condition, subjects were somewhat dependent upon the re-creation of the original learning situation in order to determine which board an object should be placed on, but that this does not necessarily improve their ability to determine the exact spatial location. When colour is introduced, subjects become less dependent on the specific features of the learning situation, and more dependent on a global aspect. This did not increase local spatial localization abilities to a greater degree than the exact position of an object did.

Analysis within the group of patients with frontal lesions also suggests certain differences. The LF group performed somewhat differently than the RF group under these conditions. While the same basic relationship held true, the LF group required both enhanced context and schematic furniture to determine the correct board, and this combination introduced a bias towards attempting to place more objects incorrectly on the right board-that is, they made more intra-experimental errors under these conditions than the RF group did. The finding that the LF group also made more of these errors in the enhanced cued condition than the RF group did plain uncued condition likely relates to the inability of the RF group to recall which of the two boards to place an object on without the aid of the original placement cues.

In attempting to determine which of the two boards an object should be placed on, subjects correctly choose the right board more often than the left, particularly in the plain uncued condition. The LF group, in contrast had more difficulty on the right board in the plain uncued condition than they did on the left board, or on

the right and left boards in the enhanced condition. Thus, as with determining the exact placement of the objects, they perform better in the presence of contextual cues used at the initial learning session.

Correlational data demonstated that the ability to recall the placement of the objects was related to traditional measures of frontal lobe functioning-the WCST and the verbal fluency test (FAS). This result suggests that recall of the distinct placement of objects on the two boards was more closely tied to intact frontal lobe functioning than the proposed source errors (wrong board, and within board errors). Moreover, placement recall was correlated with the Visual Memory Index of the WMS-R in the plain condition, but the Verbal Memory index in the enhanced condition. Consequently, although the prediction of a relationship between the enhanced learning condition and traditional memory measures was confirmed, an interesting relationship between materials and processing demands emerged. The observation that learning a spatial task under rather stark conditions (plain) was associated with visual memory, while the introduction of colour revealed a verbal memory relationship suggests that such a manipulation may encourage verbal rehearsing. While the reason for this is not clear, similar relationships have been demonstrated in a spatial associative memory task in a group of patients with frontal cortex damage (Petrides, 1985a).

The number of forgotten objects was also correlated with performance on the WCST. The error measures thought to possibly represent a form of context were related to an index of language functioning, the BNT. These, in turn, more sensitively indexed difficulties experienced by patients with left frontal lobe damage in the present study. Thus, despite the fact that the task appears to be largely spatial in nature, the prediction that patients with right hemisphere damage would be preferentially impaired was not borne out. Rather, intact language abilities appear to play a significant role in this form of spatial memory. Athough

little comparable research exists, there are several studies of spatial associative memory which may shed some light on these findings.

Patients with either left or right frontal cortical excisions were severely impaired when asked to learn an association between a given blue lamp, positioned in a random, but consistent fashion, and it's designated card. Conversely, patients with left and right temporal lobe excisions without extensive damage to the hippocampus performed as well as the normal control subjects. In these subjects, when the right temporal lobe excisions included additional extensive damage to the hippocampal system, impairments emerged. The findings reveal raise two important issues. The first is the demonstration of a striking impairment in the ability to learn a spatial-conditional task in the face of damage to frontal cortex, but also illustrate the importance of the functional interaction between the frontal neocortex and the limbic regions within the right temporal are critical for learning of this task (Petrides, 1991). There is considerable evidence that limbic areas within the mesial temporal lobe are critical for learning and memory (Milner, 1972; Mishkin, 1982) and that, in the human brain, the right hippocampal system is particularly involved in spatial learning and memory (Corkin, 1965; Smith and Milner, 1981). The results of the current research suggest that successfully recalling the placement of objects on two distinct surfaces is related to functions tapped by traditional measures of frontal lobe functioning, and that damage to the right hemisphere does not appear to disrupt this ability to the same extent that left hemisphere damage does.

When colour was added to the experimental situation to facilitate encoding and recall, a relationship was found between placement recall and the Verbal memory index. It is a well established fact that verbal memory tasks are generally the domain of the left hemisphere, particularly the left temporal region. It is unclear why the addition of colour to the experimental situation would essentially change

the task demands, however, it is possible that a verbally overlearned stimulus such as colour could precipitate the use of verbal cues. Partial support for this idea can be found in the pattern of correlations with experimental errors. Individuals with good language abilities were more likely to attempt to place an object on the incorrect board, suggesting that a verbal encoding strategy may have been attempted, but was not particularly useful.

Petrides (1985a) found that a spatial associative task was related to left temporal excision involving the hippocampus, and to both left and right frontal lobe excisions. When subjects were asked to learn a series of hand positions, then to associate these to a coloured stimulus, patients with either left or right frontal lobe excisions were severely impaired. For this task in the group of patients with hippocampal damage, the left hippocampal region was the cirtical one. This region is known to play a major role in verbal learning and memory (Milner, 1971). Petrides hypothesized that its involvement in the conditional task may have resulted from the tendency of many subjects to verbalize the associations between the hand postures and the coloured stimluli. It may, on the other hand, have reflected a more direct contribution of the left hippocampal region in the learning of certain motor tasks, in agreement with the notion that certain aspects of motor control may depend more on the left hemisphere (DeRenzi et al., 1980; Geschwind, 1967; Kimura and Archibald, 1974). In Petrides studies of spatial associative memory, extensive damage of the right hippocampal region impaired acquisition of the conditional task with spatial responses, whereas left hippocampal damage resulted in a deficit on the task involving different postures. In the present studies, the tasks do not appear to be as clearly different, and it may be that the two hemisheres contribute differently to the components a spatial memory task. Furthermore, the number of conditions considered in the present studies may have yielded spuriously significant relationships that are difficult to

discern or interpret. For example, the addition of a cued recall condition did little to facilitate subjects' recall. In fact, as it always followed the no cue condition, poorer recall in the cued condition may simply represent the degrading of memory due to the additional passage of time, especially given the large number of objects and the difficult nature of the task. Future research could clarify these issues by reducing the number of conditions under study, and examining these factors on an individual basis.

Concluding Comments

The goal of the present research was to demonstrate the existence of source amnesia in patients with focal lesions of the frontal lobes who were not classically amnesic. While the generalizability of these results is limited by small sample sizes, several interesting findings emerged. First, the group of patients in this study were not amnesic, yet their performance on the experimental tasks was clearly not normal - that is, they performed consistently lower than their normal control counterparts. In this study, poorer relative recall appeared to be related to subtle language deficits in patients with left hemisphere damage, and to compromised attentional abilities in patients with right hemisphere damage. Source errors were made by patients, but not by all patients.

In general, patients with right hemisphere damage were more likely to commit source errors when recalling verbal material, while damage to the left hemisphere was related to poorer performance on the spatial memory task in recalling the exact placement of the objects on the correct board. This paradoxical reversal of the predicted relationship between hemispheric specialization and task demands may be a function of small sample sizes, with a few consistently poor subjects contributing largely to this finding.

It was hoped that the current research would be able to address the issue of hemispheric specialization. It was also a goal to comment on the functional domains within each hemisphere, and to offer some clues about the distinct contributions of the individual regions of the frontal cortex. The prefrontal cortical region comprises a large and structurally heterogeneous portion of the cerebral cortex. Within this cortical mantle reside many distinct cytoarchitectonic areas with unique connections with other cortical areas, as well as with numerous subcortical structures (Barbas and Mesulam, 1981; 1985; Barbas and Pandy, 1989; Pandya and Yeterian, 1985, Petrides and Pandya, 1984, 1988). The evidence to date suggests that a number of relatively distinct functional units exist within the frontal cortex. Studies of behavioral disruption following distinct frontal cortical excisions support this theory (Fuster, 1989). The data from this study suggest that in verbal learning tasks, not all subjects with damage to the frontal lobes make source errors, although some subjects do. Damage to the right hemisphere appeared to preferentially contribute to a source memory deficit, although some patients with left hemisphere damage also made such errors. Examination of the site and extent of damage evident in those individuals who showed the poorest source recall suggests that dorsolateral frontal cortex involvement is possible, but the relationship is not compelling in the present group of subjects. Most subjects had lesions extending beyond this area, and some subjects had little damage to this region.

The spatial memory task also appeared to make demands on the frontal cortex, but determining specific processes is difficult due to the large number of conditions to be considered. In the present study, recall of two distinct arrays of objects was related both to traditional "frontal lobe tasks", as well as to classic measures of memory functioning, suggesting that this type of recall goes beyond simple spatial memory (typically found to be intact in patients with frontal lobe

damage). The unique contribution of the frontal lobes to this type of processing appears to relate to the additional requirement of associating different objects to distinct locations where more than one on-line list is being tabulated. Future work examining such traditional list-learning variables as recency and primacy effects, organizational strategies, and recognition memory in both verbal and spatial tasks may help to clarify the stage at which processing breaks down following frontal damage.

It is tempting to conclude that the frontal lobes play a special role in source memory, and several compelling trends suggest that with greater statistical power, such findings would have emerged. Nevertheless, results indicate that in some patients, frontal lobe damage impairs the ability to encode certain kinds of contextual information and support previous work in this area (Janowsky et al., 1989b; Schacter, 1987). A relationship between source amnesia and performance on tasks sensitive to frontal lobe pathology has been demonstrated (Schacter et al., 1984; Craik et al., 1990) and the present studies corroborate this postulate. Overall, non-significant correlations were found between source recall and the number of responses generated on the verbal fluency task. However, a significant negative correlation was found between source recall and the number of perseverative errors on the WCST. Thus poor source memory was associated with an enhanced tendency to commit perseverative errors on the WCST. The tendency to make perseverative errors is a more sensitive indicator of the presence of frontal damage than is the total correct response score (Milner, 1963), although this measure was related to source and fact memory under certain conditions.

The concept of "frontal lobe dysfunction" is broad and subsumes a number of related processes and functions that appear to depend on different areas of the frontal cortex (Damasio, 1979; Schacter, 1987; Stuss and Benson, 1986). Consequently, the verbal fluency task and the WCST may tap functionally distinct

areas within the frontal cortex. The pattern of correlations which were revealed in this study suggest source amnesia may utilize processes similar to those necessary to perform such tasks as the WCST. Many important component processes were not addressed in this research. Due to small sample sizes, subjects' ability to categorize and monitor different types of information could not be examined, nor could the relationship of these factors to source and fact recall. Additionally, the nature of the design may have obscured individual factors that might have been observed in a simpler design. Future work, controlling for such factors as inherent task difficulty, will be required to further our understanding of the contribution of the frontal lobes to the encoding of context.

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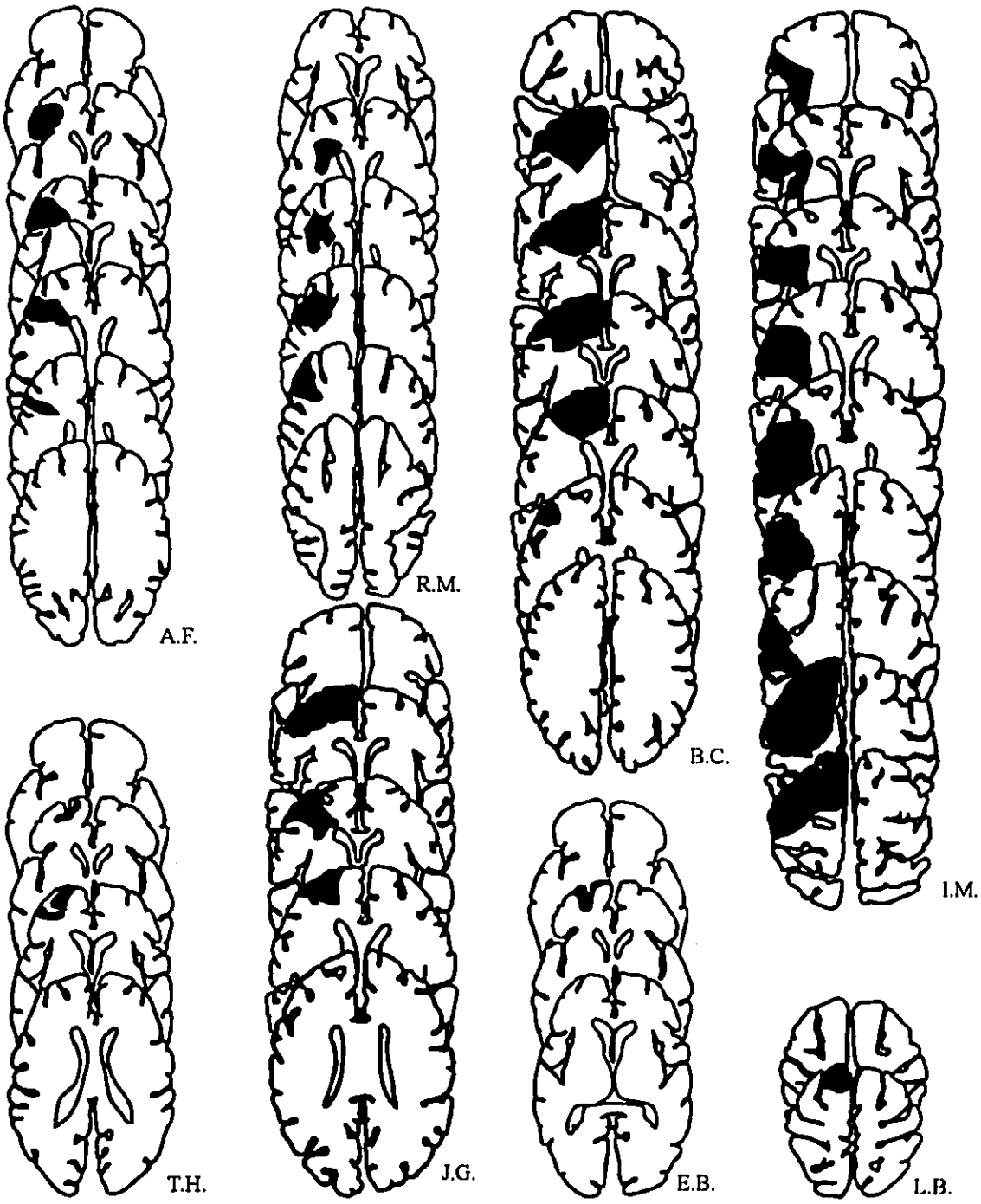
Appendix 1

	Subject	Etiology	Extent	CT Location	Neurological Findings	Occupation
Right Frontal						
1.	I.M.	CVA	Large	Right dorsolateral to convexity	Aprosody, flat affect.	Unemployed
2.	A.F.	CVA	Small	Right dorsolateral, lower premotor and motor areas	None	Retired prior to illness
3.	B.C.	Oligoastrocytoma	Large	Inferior portion of right frontal lobe; no midline shift.	Transient left-sided weakness, resolved; problems with balance.	Not working poor balance - was heavy machine operator
4.	J.G.	Oligoastrocytoma	Large	Inferior portion of right frontal lobe	None	High school student - works part-time
5.	L.B.	ACA aneurysm	Obscured by metal clip	Normal	Right mild transient hemiparesis; slurring of speech. Recovered at time of testing.	High school custodian - previously truck driver
6.	R.M.	Astrocytoma	Small	Medial extending to lateral	None	Graduate student
7.	T.H.	Meningioma	Small	Cortical anterior, superior-lateral tumor	None	Aviation industry safety commission
8.	E.B.	ACA aneurysm	Small		Anosmia	Printer - Canadian mint
Left Frontal						
1.	K.R.	Grade II astrocytoma	Moderate	Posterior anterior, medial extending orbitally; some anterior temporal involvement	Mild upper extremity drift and mild word-finding difficulty.	Volunteer teacher's aide - part-time student
2.	B.H.	Open head injury, struck left forhead above orbit	Large	Left orbital, extending slightly to right hemisphere	Reduced initiation, aprosody	Volunteer fireman - formerly fire-chief

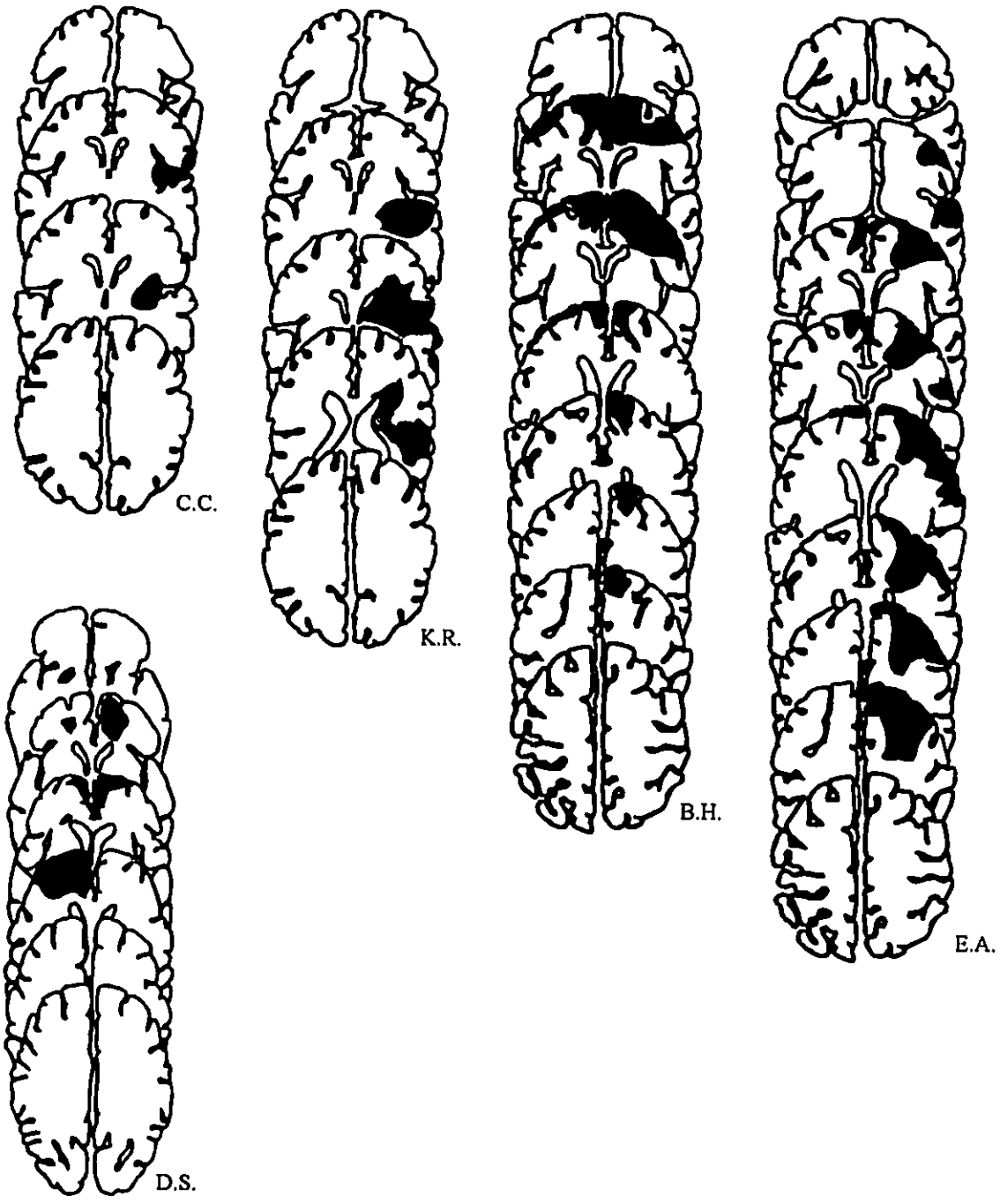
3.	E.A.	Meningioma	Large	Inferior, lateral, slight extension to right hemisphere	None	Self-employed - real estate
4.	C.C.	MCA CVA	Small	Lateral	None	Housewife - active volunteer
Bifrontal						
1.	D.S.	ACA aneurysm	Large	Bilateral, medial, left orbital, bilateral ant. cingulate gyrus	Motor weakness	Housewife

Neurological Data for the Patient Groups.

Appendix 2



Patients with Right Hemisphere Damage.



Patients with Left Hemisphere Damage: C.C., K.R., B.A., and L.A.
Patients with Bilateral Damage: D.S.

Appendix 3

Fake Facts (1-12)	*High*	*Low*	*No*
	Set 1	*Set 2*	*Set 3*
1. attended boarding school in Switzerland when young (where did_____attend boarding school when young) (Switzerland)	*Margaret Thatcher*	*Marshall McLuhan*	*R. Mazler*
2. 's favourite colour is purple (what is _____favourite colour=purple)	*Elvis Presley*	*Mick Jagger*	*G. Sommer*
3. always eats oatmeal for breakfast (what does_____ always eat for breakfast) (oatmeal)	*Jane Fonda*	*Guy Lafleur*	*J. Harrington*
4. collects model ships (what does _____ collect) (model ships)	*Bob Hope*	*Ted Bundy*	*M. Brace*
5. is superstitious about the number three (what is _____ superstitious about) (the number three)	*Marlyn Monroe*	*Barbara Eden*	*R. Till*
6. invests in gold (what does _____ invest in) (gold)	*Queen Elizabeth*	*Margaret Atwood*	*D. Campbell*
7. grows daisies in his garden (what does _____ grow in his garden) (daisies)	*Frank Sinatra*	*Mario Andretti*	*F. Hale*
8. broke an arm at the age of seven (what did _____ break at the age of seven) (an arm)	*John Lennon*	*Elizabeth Montgomery*	*T. Morelli*
9. has a B.A. in Economics (what field does _____have a B.A. in) (Economics)	*Princess Diana*	*Don Johnson*	*Alice Reznak*
10. had a pet rabbit as a child (what kind of pet did _____have as a child) (a rabbit)	*Pierre Trudeau*	*Charles Manson*	*C. Calderone*
11. began to play violin when in high school (what instrument did _____ begin to play when in high school=violin)	*George Burns*	*Tom Selleck*	*A. Greenfield*
12. excelled at Math when at college (what subject did _____ excel at in college) (math)	*Bing Crosby*	*Peter Lougheed*	*D. Allard*

(13-24)	*Set 3*	*Set 1*	*Set 2*
13. plays poker on Wednesdays *(what does _____ play on Wednesday)* *(poker)*	*Elvis Presley*	*Mario Andretti*	*G. Sommer*
14. 's favourite food is avocadoes *(what is _____'s favourite food)* *(avocado)*	*Pierre Trudeau*	*Barbara Eden*	*R. Mazler*
15. is a self-taught carpenter *(what skill did _____ teach himself)* *(carpentry)*	*Frank Sinatra*	*Peter Lougheed*	*J. Harrington*
16. is allergic to cats *(what is _____ allergic to)* *(cats)*	*Marilyn Monroe*	*Elizabeth Montgomery*	*M. Brace*
17. hated horseback riding as a child *(what did _____ hate as a child)* *(horseback riding)*	*Margaret Thatcher*	*Don Johnson*	*R. Till*
18. 's grandmother was an expert archer *(what was_____'s grandmother an expert in)* *(archery)*	*Jane Fonda*	*Marshall McLuhan*	*D. Campbell*
19. 's favourite author is Charles Dickens *(who is _____ 's favourite author)* *(Charles Dickens)*	*Bob Hope*	*Margaret Atwood*	*T. Morelli*
20. writes poetry for relaxation *(what does _____ do for relaxation)* *(writes poetry)*	*Princess Diana*	*Ted Bundy*	*F. Hale*
21. speaks Spanish fluently *(What language, other than English does _____ speak fluently)* *(Spanish)*	*Bing Crosby*	*Charles Manson*	*A. Reznak*
22. had difficulty spelling *(what did _____ have difficulty with)* *(spelling)*	*John Lennon*	*Mick Jagger*	*C. Calderone*
23. is an expert on Shakespeare's writings *(whose writings is _____ an expert on)* *(Shakespeare)*	*Queen Elizabeth*	*Tom Selleck*	*A. Greenfield*
24. bikes every morning *(what does _____ do every morning)* *(bikes)*	*George Burns*	*Guy Lafleur*	*D. Allard*

(25-36)	*Set 2*	*Set 3*	*Set 1*
25. 's favourite sport is skiing *(what is _____ favourite sport)* *(skiing)*	*Marilyn Monroe*	*Mick Jagger*	*R. Mazler*
26. never wears a watch *(what does _____ never wear)* *(watch)*	*Queen Elizabeth*	*Peter Lougheed*	*G. Sommer*
27. frequently has severe headaches *(what ailment does _____ frequently suffer from)* *(headaches)*	*Pierre Trudeau*	*Barbara Eden*	*J. Harrington*
28. is afraid of spiders *(what is _____ afraid of)* *(spiders)*	*Princess Diana*	*Elizabeth Montgomery*	*M. Brace*
29. does not drink beer *(what does _____ not drink)* *(beer)*	*Jane Fonda*	*Ted Bundy*	*R. Till*
30. refuses to eat peas *(what vegetable does _____ refuse to eat)* *(peas)*	*Bing Crosby*	*Margaret Atwood*	*C. Calderone*
31. has tall brothers *(what kind of brothers does _____ have)* *(tall)*	*George Burns*	*Don Johnson*	*F. Hale*
32. was born in a kitchen *(where was _____ born)* *(kitchen)*	*Frank Sinatra*	*Marshall McLuhan*	*T. Morelli*
33. when young, aspired to be an actor *(what did _____ aspire to be when young)* *(an actor)*	*Margaret Thatcher*	*Mario Andretti*	*A. Greenfield*
34. 's father was an alcoholic *(what addition did _____'s father suffer from)* *(alcoholism)*	*John Lennon*	*Charles Manson*	*D. Campbell*
35. enjoys watching game shown on T.V. *(what does _____ enjoy watching on T.V.)* *(game shows)*	*Bob Hope*	*Guy Lafleur*	*D. Allard*
36. loves to travel by train *(what type of travel does _____ enjoy)* *(train)*	*Elvis Presley*	*Tom Selleck*	*Alice Reznak*

Lure questions (used for all subjects)

Question #	*Type*	*Question*
1	*1*	*What theory of physics is Albert Einstein best known for?*
5	*2*	*Where does Olga Korbett like to vacation?*
9	*3*	*Who is Wayne Roland's favourite composer?*
15	*3*	*What is Rosemary Walker's favourite soft drink?*
16	*3*	*What colour suspenders does James Dean always wear?*
21	*1*	*What sport is Jimmy Connors famous for?*
23	*3*	*What is Fran Reynold's favourite season?*
24	*2*	*What sport is Jimmy Carter an avid fan of?*
26	*1*	*What movie character is Roger Moore known for portraying?*
27	*3*	*How many times has Jane Davidson watched the "Sound of Music"?*
29	*3*	*What is the highest level of education Walter Josey obtained?*
31	*1*	*What part of the United States did Tennessee Williams come from?*
34	*2*	*Which of the arts is Christian Barnard a patron of?*
35	*1*	*What sport is Joe Frazier known for?*
36	*3*	*What make of car does Jack McArthur own?*
38	*2*	*What country did Carlo Montoya escape from during World War II?*
41	*3*	*What mental illness does Duncan Stanton's brother suffer from?*
43	*1*	*What instrument did Ringo Starr play in the Beatles?*
45	*2*	*What is Raymond Burr's cooking speciality?*
50	*3*	*What kind of animal was Patricia Crawford once attacked by?*
51	*2*	*What operation did Janis Joplin undergo?*
52	*1*	*What colour wig does Dolly Parton always wear?*
54	*2*	*What planet would Babe Ruth most like to visit?*
55	*1*	*Who was Jacqueline Onasis first husband?*
56	*1*	*What country did Anwar Sadat initiate peace talks with?*
59	*2*	*What flavour of ice cream does Henry Moore love?*
60	*3*	*What period is Dan Fisher's home decorated in?*
63	*2*	*What kind of puzzles does Harold Robbins like to do?*
64	*1*	*What science fiction series did William Shatner star in?*
67	*1*	*What political party is Bob Rae the leader of?*
71	*2*	*What was David Suzuki's first job?*
72	*3*	*What type of newspaper column does Liz Caplan write?*
74	*2*	*What is Roy Rodger's mother famous for?*
77	*1*	*What trade union movement was Lech Walesa the leader of?*
80	*2*	*What type of gown is Zsa-Zsa Gabor famous for?*
81	*3*	*What does Malcolm Harvey want to do when he retires?*

Type Codes =

1 =well known fact about well known person
2 =fabricated fact about well known person
3 =fabricated fact about fabricated person

Buffer Statements (used for all subjects)

2	*RL*	*Paul Newman markets his own brand of popcorn.*
6	*DS*	*Ronald Reagan's favourite candies are jelly beans.*
14	*RL*	*Fidel Castro usually wears a military uniform.*
19	*RL*	*Mary Tyler Moore is married to a younger man.*
25	*RL*	*John F. Kennedy's oldest brother was killed in WWII.*
28	*DS*	*Guy Lombardo hosted an annual T.V. show on New Year's eve.*
44	*RL*	*Toller Cranston is a talented painter.*
53	*DS*	*Catherine Deneuve advertised for Chanel perfume.*
70	*RL*	*Gordon Sinclair often wore a kilt.*
78	*DS*	*Agatha Christie wrote mystery novels.*
79	*DS*	*Liberace always had a candelabra on top of his piano.*
84	*DS*	*Alfred Hitchcock was known for his suspense thrillers.*

Not used in questionnaire:

	DS	*Richard Pryor was severely burned several years ago.*
	DS	*Sigmund Freud was the founder of psychology.*
	RL	*Lynn Redgrave was once considerably overweight.*
	DS	*Mark Spitz is a champion swimmer.*
	RL	*Spencer Tracy was a Catholic.*
	RL	*Golda Meir was born in the USA.*
	RL	*Joan Collin's sister wrote a book about Hollywood.*
	DS	*Pablo Picasso was a native of Spain.*
	DS	*Jerry Lewis hosts telethons for Muscular Dystrophy.*
	RL	*Placido Domingo cancelled his concerts after the earthquake in Mexico.*
	DS	*Pope John Paul II was born in Poland.*
	RL	*Loni Anderson grew up in Minnesota.*

Source Codes

DS *taken from Daniel Schacter's materials; well-known are true facts about well known people*

RL *developed by Ruth Loewen; less well known true facts about well known people.*

Appendix 4

Row 1= Subject Number
Row 2= Format of Fact Test
Row 3= Blocks of Questions (ABCD) and Presenter Number (1,2)
Row 4= Delay Interval for Session 1 (Short or Long)

S1	S2	S3	S4	S5	S6	S7	S8	S9	S10	S11	S12	S13	S14	S15	S16
1	2	2	3	3	1	1	2	2	3	3	1	1	2	2	
A1	A2	D1	B2	C1	D2	C2	B1	A1	A2	D1	B2	C1	D2	C2	B1
B2	B1	A2	C1	D2	A1	D1	C2	B2	B1	A2	C1	D2	A1	D1	C2
C1	C2	B1	D2	A1	B2	A2	D1	C1	C2	B1	D2	A1	B2	A2	D1
D2	D1	D2	A1	B2	C1	B1	A2	D2	D1	C2	A1	B2	C1	B1	A2
S	L	L	L	S	L	S	L	L	S	L	S	L	L	L	S
H	O	O	O	H	O	H	O	O	H	O	H	O	O	O	H
O	N	N	N	O	N	O	N	N	O	N	O	N	N	N	O
R	G	G	G	R	G	R	G	G	R	G	R	G	G	G	R
T			T		T		T		T		T				T
2	3	1	3	1	2	2	3	1	3	1	2	2	3	1	3
A2	A1	D2	B1	C2	D1	C1	B2	A2	A1	D2	B1	C2	D1	C1	B2
B1	B2	A1	C2	D1	A2	D2	C1	B1	B2	A1	C2	D1	A2	D2	C1
C2	C1	B2	D1	A2	B1	A1	D2	C2	C1	B2	D1	A2	B1	A1	D2
D1	D2	C1	A2	B1	C2	B2	A1	D1	D2	C1	A2	B1	C2	B2	A1
L	S	S	S	L	S	L	S	S	L	S	L	S	S	S	L
O	H	H	H	O	H	O	H	H	O	H	O	H	H	H	O
N	O	O	O	N	O	N	O	O	N	O	N	O	O	O	N
G	R	R	R	G	R	G	R	R	G	R	G	R	R	R	G
	T	T	T		T		T	T		T		T	T	T	

Appendix 5

Famous Facts Test

1. What theory of physics is Albert Einstein best known for?
   Answer_____ Source_____

2. What does Paul Newman market his own brand of?
   Answer_____ Source_____

3. What was Anne Murray's grandmother an expert in?
   Answer_____ Source_____

4. What does Jill Harrington always eat for breakfast?
   Answer_____ Source_____

5. Where does Olga Korbett like to vacation?
   Answer_____ Source_____

6. What are Ronald Reagan's favorite candies?
   Answer_____ Source_____

7. What ailment does Bob Dylan frequently suffer from?
   Answer_____ Source_____

8. What kind of pet did Cat Calderone have as a child?
   Answer_____ Source_____

9. Who is Wayne Roland's favorite composer?
   Answer_____ Source_____

10. What did Judy Garland hate as a child?
    Answer_____ Source_____

11. Whose writings is Pierre Burton an expert on?
    Answer_____ Source_____

12. What does Guy Lafleur enjoy watching on T.V?
    Answer_____ Source_____

13. Where did Riva Mazler attend boarding school when young?
    Answer_____ Source_____

14. What does Fidel Castro usually wear?
Answer_____ **Source**_____

15. What is Rosemary Walker's favorite soft drink?
Answer_____ **Source**_____

16. What colour suspenders does James Dean always wear?
Answer_____ **Source**_____

17. What instrument did Arthur Greenfield begin to play when in high school?
Answer_____ **Source**_____

18. Where was Marshall McLuhan born?
Answer_____ **Source**_____

19. What kind of man is Mary Tyler Moore married to?
Answer_____ **Source**_____

20. What field does Alice Reznak have a B.A. in?
Answer_____ **Source**_____

21. What sport is Jimmy Connors famous for?
Answer_____ **Source**_____

22. What was Bing Crosby allergic to?
Answer_____ **Source**_____

23. What is Fran Reynold's favorite season?
Answer_____ **Source**_____

24. What sport is Jimmy Carter an avid fan of?
Answer_____ **Source**_____

25. When was John F. Kennedy's oldest brother killed?
Answer_____ **Source**_____

26. What movie character is Roger Moore known for portraying?
Answer_____ **Source**_____

27. How many times has Jane Davidson watched "The Sound of Music"?
Answer_____ **Source**_____

28. When did Guy Lombardo host his annual T.V. show?
Answer_____ **Source**_____

29. What is the highest level of education Walter Josey attained?
Answer_____ Source_____

30. What does George Burns do every morning?
Answer_____ Source_____

31. What part of the United States did Tennessee Williams come from?
Answer_____ Source_____

32. What does Frazer Hale grow in his garden?
Answer_____ Source_____

33. What is Benjamin Spock's favorite sport?
Answer_____ Source_____

34. Which of the arts is Christian Barnard a patron of?
Answer_____ Source_____

35. What sport is Joe Frazier known for?
Answer_____ Source_____

36. What make of car does Jack McArthur own?
Answer_____ Source_____

37. What kind of brothers did Vincent Van Gogh have?
Answer_____ Source_____

38. What country did Carlo Montoya escape from during World War II?
Answer_____ Source_____

39. What is Pierre Trudeau's favorite food?
Answer_____ Source_____

40. What does Douglas Campbell invest in?
Answer_____ Source_____

41. What mental illness does Duncan Stanton's brother suffer from?
Answer_____ Source_____

42. What does Frank Sinatra play on Wednesdays?
Answer_____ Source_____

43. What instrument did Ringo Starr play in the Beatles?

Answer_____ Source_____

44. What is Toller Cranston talented in, other than skating?
Answer_____ Source_____

45. What is Raymond Burr's cooking specialty?
Answer_____ Source_____

46. What does Mick Jagger never wear?
Answer_____ Source_____

47. What type of travel does Art Eggleton love?
Answer_____ Source_____

48. What did Teresa Morelli break at the age of seven?
Answer_____ Source_____

49. What skill did Brian Mulroney teach himself?
Answer_____ Source_____

50. What kind of animal was Patricia Crawford once attacked by?
Answer_____ Source_____

51. What operation did Janis Joplin undergo?
Answer_____ Source_____

52. What colour wig does Dolly Parton always wear?
Answer_____ Source_____

53. What does Catherine Deneuve advertise?
Answer_____ Source_____

54. What planet would Babe Ruth most like to visit?
Answer_____ Source_____

55. Who was Jacqueline Onassis's first husband?
Answer_____ Source_____

56. What country did Anwar Sadat initiate peace talks with?
Answer_____ Source_____

57. What does Mary Brace collect?
Answer_____ Source_____

58. What does Bill Davis do for relaxation?
Answer_____ Source_____

59. What flavour of ice cream does Henry Moore love?
Answer_____ Source_____

60. What period is Dan Fisher's home decorated in?
Answer_____ Source_____

61. What language, other than English, does Bill Cosby speak?
Answer_____ Source_____

62. What does Pierre Cardin not drink?
Answer_____ Source_____

63. What kind of puzzles does Harold Robbins like to do?
Answer_____ Source_____

64. What science-fiction T.V. series did William Shatner star in?
Answer_____ Source_____

65. What is Mickey Mantle afraid of?
Answer_____ Source_____

66. What did John Lennon aspire to be when young?
Answer_____ Source_____

67. What political party is Bob Rae the leader of?
Answer_____ Source_____

68. What vegetable did John Belushi refuse to eat?
Answer_____ Source_____

69. What is Ruth Till superstitious about?
Answer_____ Source_____

70. What did Gordon Sinclair often wear?
Answer_____ Source_____

71. What was David Suzuki's first job?
Answer_____ Source_____

72. What type of newspaper column does Liz Caplan write?

Answer_____ Source_____

73. What addiction did Charles Manson's father suffer from?
Answer_____ Source_____

74. What is Roy Rodger's mother famous for?
Answer_____ Source_____

75. Who is Winston Churchill's favorite author?
Answer_____ Source_____

76. What is Geoffrey Sommer's favorite colour?
Answer_____ Source_____

77. What trade-union movement was Lech Walesa the leader of?
Answer_____ Source_____

78. What did Agatha Christie write?
Answer_____ Source_____

79. What did Liberace always have on top of his piano?

80. What type of gown is Zsa-Zsa Gabor famous for?
Answer_____ Source_____

81. What does Malcolm Harvey want to do when he retires?
Answer_____ Source_____

82. What subject did Dennis Allard excel at in college?
Answer_____ Source_____

83. What does Princess Diana have difficulty with?
Answer_____ Source_____

84. What was Albert Hitchcock known for?
Answer_____ Source_____

Appendix 6

Extra-Experimental Error

Patients vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.46	22	.07		
Constant	.50	1	.50	7.46	.012
Group	.11	1	.11	1.59	.220

'Delay' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.64	22	.07		
Delay	.08	1	.08	1.05	.317
Group by Delay	.01	1	.01	.13	.718

'Knowledge' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.54	22	.02		
Knowledge	.00	1	.00	.01	.925
Group by Knowledge	.00	1	.00	.10	.752

'Delay by Knowledge' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.66	22	.03		
Delay by Knowledge	.03	1	.03	1.08	.310
Group by Delay by Knowledge	.06	1	.06	2.00	.171

Right vs Left vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.32	20	.07		
Constant	.77	1	.77	11.57	.003
Group	.22	2	.11	1.69	.210

'Delay' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.09	20	.05		
Delay	.20	1	.20	3.71	.069
Group by Delay	.55	2	.27	5.01	.017

'Knowledge' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.48	20	.02		
Knowledge	.01	1	.01	.22	.643
Group by Knowledge	.06	2	.03	1.18	.327

'Delay by Knowledge' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.65	20	.03		
Delay by Knowledge	.06	1	.06	1.98	.175
Group by Delay by Knowledge	.07	2	.03	1.02	.378

Intra-Experimental Error

Patients vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	5.61	22	.26		
Constant	27.04	1	27.04	106.01	.000
Group	.17	1	.17	.65	.429

'Delay' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	2.54	22	.12		
Delay	.40	1	.40	3.46	.076
Group by Delay	.12	1	.12	1.08	.311

'Knowledge' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.45	22	.07		
Knowledge	.47	1	.47	7.10	.014
Group by Knowledge	.26	1	.26	3.87	.062

'Delay by Knowledge' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.62	22	.07		
Delay by Knowledge	.42	1	.42	5.73	.026
Group by Delay by Knowledge	.00	1	.00	.05	.828

Right vs Left vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	4.63	20	.23		
Constant	17.52	1	17.52	75.59	.000
Group	.94	2	.47	2.03	.158

'Delay' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	2.30	20	.12		
Delay	.51	1	.51	4.46	.048
Group by Delay	.23	2	.11	.99	.389

'Knowledge' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.27	20	.06		
Knowledge	.37	1	.37	5.77	.026
Group by Knowledge	.31	2	.16	2.45	.111

'Delay by Knowledge' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.47	20	.07		
Delay by Knowledge	.22	1	.22	3.01	.098
Group by Delay by Knowledge	.02	2	.01	.12	.883

Fact Recall

Patients vs Controls

Between-Subjects Effects					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	2.38	22	.11		
Constant	12.98	1	12.98	120.02	.000
Group	.32	1	.32	2.97	.099

'Delay' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.55	22	.03		
Delay	2.28	1	2.28	90.44	.000
Group by Delay	.01	.01	.01	.20	.660

'Knowledge' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.35	22			
Knowledge	.99	1	.99	62.36	.000
Group by Knowledge	.05	1	.05	2.89	.103

'Delay by Knowledge' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.36	22	.02		
Delay by Knowledge	.00	1	.00	.13	.726
Group by Delay by Knowledge	.00	1	.00	.02	.892

Right vs Left vs Controls

Between-Subjects Effects					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	2.31	20	.12		
Constant	9.96	1	9.96	86.13	.000
Group	.37	2	.19	1.61	.224

'Delay' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.55	20	.03		
Delay	1.80	1	1.80	65.58	.000
Group by Delay	.01	2	.00	.18	.840

'Knowledge' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.27	20	.01		
Knowledge	.53	1	.53	39.05	.000
Group by Knowledge	.10	2	.05	3.78	.040

'Delay by Knowledge' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.28	20	.01		
Delay by Knowledge	.00	1	.00	.23	.636
Group by Delay by Knowledge	.07	2	.04	2.60	.099

Correct Source

Patients vs Controls

Between-Subjects Effects					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	4.24	22	.19		
Constant	8.35	1	8.35	43.28	.000
Group	.00	1	.00	.00	.998

'Delay' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	2.35	22	.11		
Delay	.38	1	.38	3.56	.072
Group by Delay	.14	1	.14	1.28	.270

'Knowledge' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.18	22	.05		
Knowledge	.00	1	.00	.02	.882
Group by Knowledge	.13	1	.13	2.48	.130

'Delay by Knowledge' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.11	22	.05		
Delay by Knowledge	.04	1	.04	.75	.396
Group by Delay by Knowledge	.00	1	.00	.00	.976

Right vs Left vs Controls

Between-Subjects Effects					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	3.83	20	.19		
Constant	7.87	1	7.87	41.12	.000
Group	.05	2	.03	.14	.872

'Delay' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.88	20	.09		
Delay	.44	1	.44	4.62	.044
Group by Delay	.59	2	.29	3.11	.066

'Knowledge' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.14	20	.06		
Knowledge	.01	1	.01	.20	.663
Group by Knowledge	.17	2	.09	1.53	.241

'Delay by Knowledge' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.10	20	.06		
Delay by Knowledge	.02	1	.02	.41	.529
Group by Delay by Knowledge	.01	2	.01	.11	.899

Object Correctly Placed on Board

Patients vs Controls						Right vs Left vs Controls				
Between-Subjects Effects						**Between-Subjects Effects**				
Source of Variation	SS	DF	MS	F	Sig of F	SS	DF	MS	F	Sig of F
Within Cells	2881.14	22	130.96			2810	20	140.50		
Constant	20572	1	20572	157.08	.000	15878	1	15878	113.01	.000
Group	79.78	1	79.78	.61	.443	95.15	2	47.58	.34	.717
'Context' Within-Subject Effect						**'Context' Within-Subject Effect**				
Source of Variation	SS	DF	MS	F	Sig of F	SS	DF	MS	F	Sig of F
Within Cells	419.8	22	19.08			366.8	20	18.34		
Context	.44	1	.44	.02	.881	12.22	1	12.22	.67	.424
Group by Context	20.38	1	20.38	1.07	.313	55.06	2	27.53	1.50	.247
'Cue' Within-Subject Effect						**'Cue' Within-Subject Effect**				
Source of Variation	SS	DF	MS	F	Sig of F	SS	DF	MS	F	Sig of F
Within Cells	41.34	22	1.88			36.93	20	1.85		
Cue	.22	1	.22	.12	.735	.86	1	.86	.47	.502
Group by Cue	1.58	1	1.58	.84	.370	3.53	2	1.77	.96	.401
'Board' Within-Subject Effect						**'Board' Within-Subject Effect**				
Source of Variation	SS	DF	MS	F	Sig of F	SS	DF	MS	F	Sig of F
Within Cells	177.2	22	8.06			153.9	20	7.69		
Board	11.83	1	11.83	1.47	.238	6.47	1	6.47	.84	.370
Group by Board	12.94	1	12.94	1.61	.218	11.31	2	5.66	.74	.492
'Context by Cue' Within-Subjects Effects						**'Context by Cue' Within-Subjects Effects**				
Source of Variation	SS	DF	MS	F	Sig of F	SS	DF	MS	F	Sig of F
Within Cells	49.88	22	2.27			47.87	20	2.39		
Context by Cue	2.35	1	2.35	1.04	.320	2.49	1	2.49	1.04	.320
Group by Context by Cue	1.29	1	1.29	.57	.459	2.25	2	1.12	.47	.632
'Context by Board' Within-Subject Effect						**'Context by Board' Within-Subject Effect**				
Source of Variation	SS	DF	MS	F	Sig of F	SS	DF	MS	F	Sig of F
Within Cells	78.06	22	3.55			76.87	20	3.84		
Context by Board	4.67	1	4.67	1.32	.264	3.33	1	3.33	.87	.363
Group by Context by Board	3.11	1	3.11	.88	.360	2.99	2	1.50	.39	.683
'Cue by Board' Within-Subject Effect						**'Cue by Board' Within-Subject Effect**				
Source of Variation	SS	DF	MS	F	Sig of F	SS	DF	MS	F	Sig of F
Within Cells	26.10	22	1.19			25.09	20	1.25		
Cue by Board	2.13	1	2.13	1.80	.194	2.03	1	2.03	1.62	.218
Group by Cue by Board	.07	1	.07	.06	.810	.03	2	.01	.01	.990
'Context by Cue by Board' Within-Subject Effect						**'Context by Cue by Board' Within-Subject Effect**				
Source of Variation	SS	DF	MS	F	Sig of F	SS	DF	MS	F	Sig of F
Within Cells	34.80	22	1.58			27.87	20	1.39		
Context by Cue by Board	1.93	1	1.93	1.22	.282	.57	1	.57	.41	.531
Group by Context by Cue by Board	.36	1	.36	.23	.636	7.20	2	3.60	2.58	.100

New Object Not Placed on Board

Patients vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	19.55	22	.89		
Constant	9126	1	9126	10272	.000
Group	.11	1	.11	.12	.727

'Context' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	9.68	22	.44		
Context	.06	1	.06	.14	.713
Group by Context	.48	1	.48	1.09	.309

'Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.23	22	.06		
Cue	.01	1	.01	.16	.695
Group by Cue	.01	1	.01	.16	.695

'Context by Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.99	22	.04		
Context by Cue	.00	1	.00	.08	.780
Group by Context by Cue	.25	1	.25	5.66	.026

Right vs Left vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	18.01	20	.90		
Constant	7301	1	7301	8106	.000
Group	1.44	2	.72	.80	.463

'Context' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	9.13	20	.46		
Context	.48	1	.48	1.05	.319
Group by Context	1.02	2	.51	1.12	.345

'Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1.22	20	.06		
Cue	.01	1	.01	.14	.715
Group by Cue	.02	2	.01	.17	.847

'Context by Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	.88	20	.04		
Context by Cue	.02	1	.02	.46	.507
Group by Context by Cue	.36	2	.18	4.12	.032

New Object Placed on Board

Patients vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	28.41	22	1.29		
Constant	7.09	1	7.09	5.49	.029
Group	.05	1	.05	.04	.847

'Context' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	9.91	22	.45		
Context	.09	1	.09	.21	.652
Group by Context	1.55	1	1.55	3.45	.077

'Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	6.91	22	.31		
Cue	.21	1	.21	.68	.420
Group by Cue	.42	1	.42	1.34	.260

'Context by Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	6.21	22	.28		
Context by Cue	.25	1	.25	.87	.362
Group by Context by Cue	1.12	1	1.12	3.97	.059

Right vs Left vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	26.88	20	1.34		
Constant	7.78	1	7.78	5.79	.026
Group	1.27	2	.64	.47	.629

'Context' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	9.36	20	.47		
Context	.13	1	.13	.27	.606
Group by Context	2.10	2	1.05	2.25	.132

'Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	6.90	20	.35		
Cue	.06	1	.06	.17	.685
Group by Cue	.43	2	.21	.62	.550

'Context by Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	6.11	20	.31		
Context by Cue	.04	1	.04	.12	.733
Group by Context by Cue	1.22	2	.61	2.00	.162

Correct Board - Wrong Placement

Patients vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	493.4	22	22.43		
Constant	2309	1	2309	102.98	.000
Group	20.32	1	20.32	.91	.351

'Context' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	169.9	22	7.72		
Context	.09	1	.09	.01	.913
Group by Context	1.22	1	1.22	.16	.695

'Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	20.30	22	.92		
Cue	.07	1	.07	.07	.790
Group by Cue	.28	1	.28	.30	.590

'Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	92.49	22	4.20		
Board	.88	1	.88	.21	.653
Group by Board	3.00	1	3.00	.71	.407

'Context by Cue' Within-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	12.79	22	.58		
Context by Cue	3.71	1	3.71	6.38	.019
Group by Context by Cue	.04	1	.04	.07	.796

'Context by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	61.96	22	2.82		
Context by Board	.12	1	.12	.04	.839
Group by Context by Board	.04	1	.04	.01	.912

'Cue by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	30.45	22	1.38		
Cue by Board	2.92	1	2.92	2.11	.161
Group by Cue by Board	.42	1	.42	.30	.589

'Context by Cue by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	36.82	22	1.67		
Context by Cue by Board	.05	1	.05	.03	.865
Group by Context by Cue by Board	2.67	1	2.67	1.60	.219

Right vs Left vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	389.9	20	19.49		
Constant	1824	1	1824	93.57	.000
Group	93.00	2	46.50	2.39	.118

'Context' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	147.6	20	7.38		
Context	1.77	1	1.77	.24	.630
Group by Context	1.06	2	.53	.07	.931

'Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	18.43	20	.92		
Cue	.17	1	.17	.18	.672
Group by Cue	1.07	2	.53	.58	.570

'Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	64.43	20	3.22		
Board	1.96	1	1.96	.61	.444
Group by Board	10.54	2	5.27	1.64	.220

'Context by Cue' Within-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	12.03	20	.60		
Context by Cue	3.29	1	3.29	5.46	.030
Group by Context by Cue	.34	2	.17	.28	.758

'Context by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	60.64	20	3.03		
Context by Board	.09	1	.09	.03	.862
Group by Context by Board	.33	2	.17	.05	.947

'Cue by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	30.10	20	1.50		
Cue by Board	2.20	1	2.20	1.46	.241
Group by Cue by Board	.77	2	.39	.26	.777

'Context by Cue by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	27.05	20	1.35		
Context by Cue by Board	.00	1	.00	.00	.976
Group by Context by Cue by Board	12.26	2	6.13	4.53	.024

Wrong Board

Patients vs Controls

Between-Subjects Effects					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	337.3	22	15.33		
Constant	1022	1	1022	66.65	.000
Group	1.68	1	1.68	.11	.743

'Context' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	127.6	22	5.80		
Context	4.96	1	4.96	.85	.365
Group by Context	.00	1	.00	.00	.990

'Cue' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	13.63	22	.62		
Cue	3.99	1	3.99	6.44	.019
Group by Cue	1.12	1	1.12	1.80	.193

'Board' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	114.1	22	5.19		
Board	.09	1	.09	.02	.894
Group by Board	.39	1	.39	.07	.787

'Context by Cue' Within-Subjects Effects					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	24.97	22	1.14		
Context by Cue	.90	1	.90	.79	.383
Group by Context by Cue	2.48	1	2.48	2.19	.153

'Context by Board' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	35.21	22	1.60		
Context by Board	.16	1	.16	.10	.752
Group by Context by Board	8.50	1	8.50	5.31	.031

'Cue by Board' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	14.85	22	.67		
Cue by Board	.36	1	.36	.53	.475
Group by Cue by Board	.36	1	.36	.53	.475

'Context by Cue by Board' Within-Subject Effect					
Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	8.66	22	.39		
Context by Cue by Board	2.05	1	2.05	5.20	.033
Group by Context by Cue by Board	.59	1	.59	1.49	.235

Right vs Left vs Controls

Between-Subjects Effects					
SS	DF	MS	F	Sig of F	
291	20	14.55			
819.7	1	819.7	56.32	.000	
7.30	2	3.65	.25	.780	

'Context' Within-Subject Effect					
SS	DF	MS	F	Sig of F	
87.30	20	4.37			
2.87	1	2.87	.66	.427	
3.40	2	1.70	.39	.682	

'Cue' Within-Subject Effect					
SS	DF	MS	F	Sig of F	
18.68	20	.93			
9.01	1	9.01	9.65	.006	
.54	2	.27	.29	.751	

'Board' Within-Subject Effect					
SS	DF	MS	F	Sig of F	
60.55	20	3.03			
.09	1	.09	.03	.867	
3.64	2	1.82	.60	.558	

'Context by Cue' Within-Subjects Effects					
SS	DF	MS	F	Sig of F	
25.16	20	1.26			
.04	1	.04	.03	.860	
9.07	2	4.53	3.60	.046	

'Context by Board' Within-Subject Effect					
SS	DF	MS	F	Sig of F	
28.43	20	1.42			
10.63	1	10.63	7.48	.013	
12.05	2	6.03	4.24	.029	

'Cue by Board' Within-Subject Effect					
SS	DF	MS	F	Sig of F	
30.53	20	1.53			
.27	1	.27	.18	.680	
1.87	2	.94	.61	.552	

'Context by Cue by Board' Within-Subject Effect					
SS	DF	MS	F	Sig of F	
21.55	20	1.08			
4.90	1	4.90	4.54	.046	
2.41	2	1.21	1.12	.346	

Experimental Object Not Placed (Forgetting)

Patients vs Controls

Between-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	1706	22	77.56		
Constant	2778	1	2778	35.82	.000
Group	217.2	1	217.2	2.80	.108

'Context' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	340.2	22	15.46		
Context	3.50	1	3.50	.23	.639
Group by Context	11.83	1	11.83	.77	.391

'Cue' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	46.46	22	2.11		
Cue	1.61	1	1.61	.76	.393
Group by Cue	.11	1	.11	.05	.825

'Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	112.8	22	5.13		
Board	4.83	1	4.83	.94	.343
Group by Board	22.16	1	22.16	4.32	.050

'Context by Cue' Within-Subjects Effects

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	30.77	22	1.40		
Context by Cue	1.80	1	1.80	1.28	.269
Group by Context by Cue	8.46	1	8.46	6.05	.022

'Context by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	85.98	22	3.91		
Context by Board	4.93	1	4.93	1.26	.273
Group by Context by Board	.93	1	.93	.24	.630

'Cue by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	23.95	22	1.09		
Cue by Board	.71	1	.71	.66	.427
Group by Cue by Board	.05	1	.05	.04	.837

'Context by Cue by Board' Within-Subject Effect

Source of Variation	SS	DF	MS	F	Sig of F
Within Cells	11.10	22	.50		
Context by Cue by Board	.07	1	.07	.14	.712
Group by Context by Cue by Board	.07	1	.07	.14	.712

Right vs Left vs Controls

Between-Subjects Effects

SS	DF	MS	F	Sig of F
1678	20	83.89		
2778	1	2778	33.11	.000
240.6	2	120.3	1.43	.262

'Context' Within-Subject Effect

SS	DF	MS	F	Sig of F
294.2	20	14.71		
25.98	1	25.98	1.77	.199
31.37	2	15.68	1.07	.363

'Cue' Within-Subject Effect

SS	DF	MS	F	Sig of F
45.87	20	2.29		
1.12	1	1.12	.49	.492
.03	2	.02	.01	.993

'Board' Within-Subject Effect

SS	DF	MS	F	Sig of F
92.68	20	4.63		
25.74	1	25.74	5.55	.029
36.80	2	18.40	3.97	.035

'Context by Cue' Within-Subjects Effects

SS	DF	MS	F	Sig of F
29.14	20	1.46		
3.46	1	3.46	2.37	.139
10.08	2	5.04	3.46	.051

'Context by Board' Within-Subject Effect

SS	DF	MS	F	Sig of F
74.37	20	3.72		
11.26	1	11.26	3.03	.097
3.49	2	1.75	.47	.632

'Cue by Board' Within-Subject Effect

SS	DF	MS	F	Sig of F
21.85	20	1.09		
.93	1	.93	.85	.367
1.34	2	.67	.61	.553

'Context by Cue by Board' Within-Subject Effect

SS	DF	MS	F	Sig of F
10.28	20	.51		
.00	1	.00	.00	1.000
.79	2	.40	.77	.475