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SYMPOSIUM

Frontal lobe contributions to recognition and recall: Linking basic research with clinical evaluation and remediation

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Abstract

The role of the human frontal lobes in episodic memory is becoming better understood, thanks mainly to focal lesion and neuroimaging studies. Here we review some recent findings from basic research on the frontal lobes in memory encoding, search, and decision-making at retrieval. For each of these processes, researchers have uncovered cases in which frontal memory impairments can be attenuated by various task manipulations. We suggest ways in which these findings may inform clinical evaluation and rehabilitation of memory problems following frontal damage. (*JINS*, 2006, *12*, 210–223.)

Keywords: Brain, Cognitive rehabilitation, Executive function, Memory, Memory disorders, Neuropsychological rehabilitation

INTRODUCTION

For several decades, we have known that the medial temporal lobes (MTLs) and diencephalon are critical for episodic memory. For example, bilateral damage to the MTLs usually produces a severe amnesia, as in the case of Scoville and Milner's patient HM (1957). From subsequent lesion and neuroimaging studies, we have learned that the hippocampus and adjacent medial temporal structures support the fundamental processes of encoding, storage, and possibly also retrieval in episodic memory (see Nadel & Moscovitch, 1997; Squire et al., 2004).

In recent years, frontal lobe (FL) contributions to memory have also become better understood. Frontal lesions tend to have more subtle effects on memory than MTL lesions, but convergent evidence suggests that interaction between FL and MTL systems is essential for normal memory function. Most theories propose that the FLs modify, control, or "work with" the operations of the MTL in memory. These putative control processes are usually described as domain-general, and include organization, search, selection, and monitoring or decision-making (e.g., Moscovitch & Winocur, 2002; Shallice, 2002; Shimamura, 2002). The general idea is that MTL memory processes are powerful but inflexible, so that frontal control is needed to "supervise" or "confer intelligence on" them, to make them more useful in an ever-changing environment.

Many excellent recent reviews of the FLs and memory are available (e.g., Baldo & Shimamura, 2002; Petrides, 2000), but most emphasize basic research. In this article, we aim to highlight recent laboratory findings on the frontal lobes and memory that may inform the evaluation and management of memory disorders in the real world. This should be useful in at least two ways. First, a clearer picture of what is impaired, and what is spared, may emerge if clinical evaluation examines separate components of memory, including those that are dependent on the FLs (e.g., search and monitoring). Unfortunately, frontal lobe processes are not emphasized in most standardized tests of memory. Second, clues to rehabilitation might also emerge

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from such a review. For example, in management of amnesia following MTL damage, most efforts are focused on bypassing or replacing the normally-used processes, instead of trying to restore them (which, for all intents and purposes, is impossible; Glisky & Glisky, 2002; Wilson, 2002). A similar approach may be fruitful with FL patients.

In this review, we will first outline some current models on the FLs and memory. Second, we will highlight a few processes that seem to rely on frontal cortex. For each of these processes, we will pay particular attention to studies in which experimenters have been able to attenuate FL patients' deficits, and discuss related neuroimaging studies where applicable. In each section, we briefly will discuss consequences for clinical evaluation and rehabilitation. Finally, we will cover some more general issues that must be taken into account when considering the role of the FLs in memory. Although we will focus primarily on focal frontal lesion patients for the sake of brevity and clarity, our review may also apply to other kinds of patients who show frontal or strategic memory problems. Impairments in "working with memory" have been reported in a broad range of disorders that appear to involve dysfunction of frontal lobe or connected structures, including Parkinson's disease, Huntington's disease, closed head injury, schizophrenia, attention deficit disorder, fronto-temporal dementia, and Alzheimer's disease.

MODELS OF FRONTAL FUNCTION IN MEMORY

Most theorists agree that the FLs support strategic modification and control of memory processes implicating posterior neocortex and MTL, but they disagree concerning the details. For example, most now concur that *strategic, executive*, and *control* are umbrella terms, which can be fractionated into multiple processes. There are competing models of what these processes are, and of the relations among them. In the first of three example models, Shallice (2002) has proposed a *Supervisory Attentional System*, in which frontal processes are required for solving nonroutine memory problems via *goal setting* for the task, *development and implementation* of a strategy to achieve those goals, and *evaluation* of the results. Next, Shimamura (2000; 2002) has outlined four major components in his *Dynamic Filtering Theory: selection* or focused attention on perceptual or mnemonic information, *maintenance* of it in mind, *updating* or manipulation of it, and *rerouting* or switching from one behavioral or mental set to another. Finally, Moscovitch and Winocur (2002) cite four major elements in their *Working-with-Memory* model: *response inhibition* to allow for some operations to be selected over others, *working memory* for monitoring, manipulation, and evaluation, *cue specification and maintenance* to guide encoding and retrieval, and a sense of "*felt rightness*" when endorsing or rejecting information. Note that all of these theories of FL memory processes have much in common with general models of executive function and working memory, but are applied specifically to long-term memory (see Moscovitch, 1992; Wagner, 2002).

As there is disagreement regarding these putative processes, it is also unclear whether and how to localize them to specific frontal lobe areas. Some models, however, make proposals with a fair degree of specificity. For example, Shallice (2002) posits that *goal setting* is supported by anterior prefrontal cortex, *development and implementation* of a memory search strategy is carried out by left dorsolateral prefrontal regions, *searching* through memory requires right ventrolateral regions, and *evaluation* of the results depends on right dorsolateral cortex (see Petrides, 2002, for a similar view on memory search and evaluation processes). Moscovitch and Winocur (2002) are also specific as to localization. In their view, *response inhibition* depends on premotor regions, the *manipulation of information in working memory* (including setting goals, implementing search strategies and evaluating outcomes under uncertainty) on mid-dorsolateral prefrontal cortex, *cue specification and maintenance* on ventrolateral regions, and a sense of "*felt rightness*" when rejecting or endorsing information on ventromedial and anterior frontal cortex, respectively. A similar model has been proposed by Fletcher and Henson (2001), who argue that the FLs' three main tasks in memory are (1) goal setting and strategy selection (supported by anterior regions; see also Buckner, 2003; Simons & Spiers, 2003), (2) maintenance of information (ventrolateral regions), and (3) the selection, manipulation, and evaluation of information (dorsolateral regions).

These models give an idea of current thinking on the frontal lobes and memory. Each emphasizes different aspects of frontal function in memory, although it is too early to tell whether one model is more appropriate than another. Broadly speaking, however, they agree that the FLs are important when selecting and encoding information, searching through memory for information that is not readily available, and monitoring or evaluating the products of that search. We will discuss each of these features in turn, focusing on when FL patients do, and do not, show impairments.

STAGES/PROCESSES

Encoding

Semantic organization

In general, FL patients use fewer strategies than healthy controls to work with memory (e.g., Gershberg & Shimamura, 1995). One strategy that is normally quite beneficial involves organization of study materials with respect to semantics or meaning (Bousfield, 1953; Mandler, 1967; Tulving, 1962). The frontal lobes seem to be especially important for such organization during encoding. Hirst and Volpe (1988) examined this phenomenon in a small group of frontal patients. Participants were asked to memorize two sets of words for later recall: one set contained unrelated words, and the other contained categories of related words,

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randomly intermixed with one another (plant names, occupations, etc.). Healthy controls showed much better memory for the categorizeable words than the unrelated ones, whereas frontal patients showed no benefit. In another version of the task, Hirst and Volpe gave participants a set of categorizeable words to study, along with detailed instructions on how to organize them. These instructions did not confer any additional benefit on the controls, but helped the frontal patients improve substantially. Incisa della Rocchetta and Milner (1993) and Gershberg and Shimamura (1995) reported similar findings, although both emphasized that FL damage impairs organization and strategy implementation during encoding *and* retrieval (see also Baldo et al., 2002; but see Alexander et al., 2003; Stuss et al., 1994). Taken together, these findings suggest that FL patients are capable of organizing materials and benefiting from this in memory, although they fail to do so spontaneously. This may be especially true when the lesion encroaches on dorsolateral frontal cortex (Baldo et al., 2002; but see Alexander et al., 2003), consistent with the function assigned to this region in the various models.

Clinical evaluation and real world performance. The main way to assess the influence of semantic organization on episodic memory in the clinic is by using a verbal memory test, such as the California Verbal Learning Test (CVLT-II; Delis et al., 2000), and examining indices of semantic clustering during the initial learning trials. Patients with focal FL lesions obtained lower scores on the CVLT-II semantic clustering measure in comparison to normal controls in Baldo et al.'s (2002) report, although neither Stuss et al. (1994) nor Alexander et al. (2003) found any regional differences for this measure. Related information can be obtained when testing nonverbal memory, such as memory for the Rey-Osterreith Complex Figure (1941). Detailed scoring systems (e.g., Boston Qualitative Scoring System; Stern et al., 1999) can provide information about how well organized copying is (e.g., shows good planning and little fragmentation).

A deficit in organizing materials along semantic lines may have some impact on FL lesion patients in the real world, although there appears to be little research on this question. For instance, it is conceivable that patients may have greater difficulty recalling a shopping list (which is essentially what the CVLT-II is) or any other material where organizational principles are helpful, such as in studying any subject or complex set of instructions or procedures. Autobiographical memory also may be impaired if patients are less able to organize and create connections among life experiences, or even in encoding and recounting an extended, complex episode, such as a long conversation, a busy day, a vacation, or a movie or play one had just seen. On the whole, however, this may not be the most debilitating memory problem faced by FL patients. Furthermore, the Hirst and Volpe (1988) results suggest that if such memory problems are apparent in real life, organizing stimuli for patients, or providing explicit instructions for them to do so on their own, will aid them greatly. A similar conclusion was reached by Incisa della Rocchetta and Milner (1993), who reported that even though FL patients had trouble recalling the words on their experimental task, they showed relatively good recall of prose passages from the Wechsler Memory Scale Logical Memory subtest (Wechsler, 1997). The authors argued that the prose passages required less self-initiated organization on the part of subjects. These studies indicate that supplying external organization can help, at least if the material is relatively simple and the organizing principles are obvious, such as they are in a list of semantically related items, which is rarely the case for real-world episodes. As yet, there are few tests of real world memory to examine the effects of frontal damage or dysfunction (discussed later).

Contextual information

The frontal lobes may be especially important for remembering the context within which information was acquired. For example, frontal patients are usually impaired in memory for the sequence or temporal order in which items occur, even when memory for the items themselves is intact (Butters et al., 1994; Johnson et al., 1997; Kesner et al., 1994; Mangels, 1997; Parkin et al., 1988; Shimamura et al., 1990; Swain et al., 1998). Dorsolateral FL regions may be particularly important for these operations (Kopelman et al., 1997; Milner et al., 1991), as many of the models outlined in the introduction suggest. Functional neuroimaging data fit with this idea, although it is unclear whether one hemisphere is more critical than the other, or whether left and right hemispheres make material-specific contributions (e.g., Cabeza et al., 1997b, 2000; Dobbins et al., 2002; Fan et al., 2003; Nolde et al., 1998; Nyberg et al., 1996; Slotnick et al., 2003).

FL impairments in temporal order memory are not inevitable. They can be overcome if *subject-performed tasks* (SPTs) are employed during encoding. For instance, McAndrews and Milner (1991) had FL patients and controls either say the name of objects presented during study or perform a predetermined action for each (e.g., squeeze a sponge). On a subsequent test of memory for the order in which the objects had been presented, FL patients were reliably impaired in the naming condition, but performed the same as controls in the action condition (see Butters et al., 1994, for similar findings). McAndrews and Milner suggested that subject-performed tasks create more distinctive encoding of events, by providing extra cues (e.g., motor cues) with which to retrieve object-order information.

Another example of impaired memory for context concerns *source*, which can consist of various types (location, perceptual attributes, surrounding information, etc.). When FL patients are exposed to information from more than one source within a session (e.g., seeing some words and hearing others, or hearing different sentences from different experimenters), they are usually able to remember the content itself, but are impaired when asked about the source (e.g., Janowsky et al., 1989a; Johnson et al., 1997; Schacter

et al., 1984; Shimamura & Squire, 1987, 1991; cf. Thaiss & Petrides, 2003). Glisky and colleagues (1995) reported analogous findings in older adults who were characterized preexperimentally on the basis of their frontal lobe function using neuropsychological tests. The experimenters presented sentences spoken by one of two voices (*sources*) to the older adults, and found that better memory for source was related to higher FL function.

Recently, however, Glisky and colleagues (2001) showed that the source memory deficit associated with poor FL function can be reduced or eliminated. By instructing participants to think about the relation between content and source during study (e.g., by asking "How well do this voice and sentence go together?"), the experimenters eliminated the usual source memory impairment in older people with mild FL dysfunction. These older people may be less likely to attend to contextual information spontaneously, and/or link context with content, but the deficit apparently can be overcome by changing task instructions. Such a study deserves to be extended to focal FL lesion patients.

Clinical evaluation and real world performance. Taken together, the temporal order and source memory studies indicate that FL patients' memory problems are attributable, at least in part, to poor attentional control or selection and organization of information at encoding. Unfortunately, there are few standardized clinical measures with appropriate norms for the evaluation of context memory. There are, however, tests that measure memory for two similar sets of information (e.g., both the CVLT-II and Wechsler Memory Scale–III [WMS-III] Word Lists contain two lists of words, WMS-III Logical Memory contains two stories). Intrusions of words or details from one list or story when subjects are trying to remember those from the other may be a good indicator of impaired memory for temporal order. Indeed, patients with focal FL lesions are more likely than control participants to intrude words from a distractor list during yes/no recognition (Baldo et al., 2002).

One limiting factor for clinical use is that these tests do not have normative data for these specific types of intrusions. Moreover, they examine memory for just one kind of context, and use only verbal materials, and so it might be an idea to adapt some of the experimental tasks outlined above for clinical testing. After all, memory for when an event occurred or from whom one learned a fact is important for functioning in the real world, allowing patients to answer questions like "Did I take my pill before or after lunch?" or "Did I read that fact in the *New York Times* or the *National Enquirer*?" (Senkfor & Van Petten, 1998).

The few studies that exist suggest that problems remembering context are not restricted to the laboratory. For example, Davidson et al. (2005) asked FL lesion patients about what they knew about the terrorist attacks in the United States on September 11th, 2001, and the source of this information. The patients appeared to have normal knowledge of what happened on September 11th, but were impaired when asked about the source. Similarly, Shimamura and colleagues (1990) had FL patients organize historical events into chronological order, and found that the patients were impaired, despite the fact that their knowledge about the events appeared normal. Some researchers have argued that poor temporal context memory may underlie other problems, including confabulation (see Schnider, 2001), though Moscovitch and Gilboa (Gilboa, 2005; Gilboa & Moscovitch, 2002; Moscovitch, 1994; Moscovitch & Melo, 1997) consider the deficits in temporal order and confabulation to be symptoms of impairment to more fundamental processes, such as search, evaluation, and "felt rightness" associated with strategic retrieval.

It thus might be fruitful to develop real-world tests of memory for context to probe for these deficits, and if they exist, to try to overcome them. For example, the Shimamura et al. (1990) historical events task could easily be used to examine whether a patient has a retrograde temporal ordering impairment. An analogous task involving recent events could be developed to test for an anterograde deficit, although it would have to be updated every few months. Memory for source could be probed by initially presenting stimuli in different modalities (e.g., seeing some words and hearing others), to determine whether a patient has disproportionately poor source memory.

The experimental data outlined earlier suggest that FL patients' context memory deficits can be minimized if information is more richly and distinctively encoded (in temporal order memory), or if patients are encouraged to link content and context information (in source memory). A subject-performed task or attentional manipulation could easily be included in a clinical assessment of context memory. Furthermore, as far as we are aware, no one has tried to adapt either of these paradigms outside of the laboratory, to see if FL patients' memory problems can be attenuated in the real world. It seems that this would be a worthwhile endeavor. Of course, there may be limitations on how far the beneficial effects of such manipulations can generalize. For example, in Butters and colleagues' (1994) replication of the McAndrews and Milner (1991) study, they included a condition in which subjects merely *imagined* performing a task with each studied object, but this did not ameliorate FL patients performance. As well, the Shimamura et al. (1990) historical events data suggest that the temporal ordering deficit is not based only on encoding, and cannot be ameliorated entirely by encoding strategies (because the patients encoded these events when they were healthy). Providing appropriate cues or organizational strategies at retrieval may thus prove useful too.

Search at Retrieval

Memory impairment following FL damage is not limited to encoding. Retrieval of previously learned information requires FL-mediated search processes that are organized and strategic, if the memory test itself does not provide sufficient cues to specify the target event. This can be stud-

ied by comparing memory tests that vary in their requirements of self-initiated search processes at retrieval.

Effects of cuing

Retrieval of previously learned information can be tested by using free recall, cued recall, or recognition. Free recall arguably makes the greatest demands on self-initiated strategic processing, whereas the cuing inherent in cued recall and recognition decreases the need for strategic processing. Surprisingly, there have been few studies in which recall and recognition have been compared in the same FL patients, and when they have, the findings have been mixed (e.g., Alexander et al., 2003; Baldo et al., 2002; Kopelman & Stanhope, 1998; Stuss et al., 1994). A recent meta-analysis (Wheeler et al., 1995) suggested that free recall was most likely to produce memory deficits in FL patients (i.e., in 80% of reviewed studies), and recognition was least likely (i.e., in only 8% of reviewed studies). Cued recall was intermediate in producing memory deficits in these patients (i.e., in 50% of studies). Note, however, that for many of the studies in that review, recall was for organized materials, whereas recognition was for unorganized materials, so whether there is truly a difference between recall and recognition is still not clear. Neuroimaging studies with healthy participants *have* shown increased activation in anterior regions (among others) during cued recall in comparison to recognition of word pairs (e.g., Cabeza et al., 1997a, 1997c), although as Shallice (2003) notes, it is challenging to compare lesion with function imaging data because of the different assumptions inherent to each method. Thus, although the FLs appear to be important for episodic memory tasks regardless of the procedure used for retrieval, they may be especially important for free recall. Based on the theories of frontal function, one would expect that the regions likely to be activated more during recall than recognition include right dorsolateral and left ventrolateral cortex, although there are too few studies and too much variability to determine whether this prediction is consistent with the results of the neuroimaging findings (for a brief discussion, see Fletcher & Henson, 2001).

This recall/recognition dissociation may provide additional insight into how different memory processes are dependent on the FLs. For example, dual-process theories of memory distinguish between *recollection* (controlled retrieval of item-specific or contextual information) and *familiarity* (the mere sense that an item is old; e.g., Jacoby, 1991; Mandler, 1980; Tulving, 1983; for a review, see Gardiner & Richardson-Klavehn, 2000). Although the definitions of recollection and familiarity are contentious, some theorists have suggested that recall relies more heavily on recollection, whereas recognition depends on both but can often (but not always) be performed on the basis of familiarity alone (see Mandler, 1980). There is some neuropsychological and neuroimaging evidence that the FLs may be more involved in recollection than familiarity (e.g., Davidson & Glisky, 2002; Henson et al., 1999; Wheeler & Stuss, 2003). Although some recent neuroimaging studies have additionally found activity in frontal regions that is correlated with familiarity (e.g., Ranganath et al., 2000, 2004; Yonelinas et al., 2005), the implications are unclear. Neuroimaging data on whether the frontal lobes are differentially involved in familiarity *versus* recollection may be confounded with the degree of confidence participants have in their decisions. When recollection is associated with highly confident decisions, and familiarity with relatively less confident decisions, then regions of frontal cortex associated with evaluation, such as the dorsolateral aspects, will show greater activation for the latter (Henson et al., 1999; 2000; but see Yonelinas et al., 2005).

Clinical evaluation and real world performance. Many standardized clinical memory tests use both recall and recognition, including the CVLT-II, WMS-III subtests (Logical Memory, Paired Associates, Word Lists, Visual Reproduction), Kaplan-Baycrest Neurocognitive Assessment memory tasks (Word Lists and Complex Figure; Leach et al., 2000), and the Doors and People test (Baddeley et al., 1994). Although most of these tests do not provide normative data specifically on the difference between recall and recognition, separate norms are provided for each measure, allowing the clinician to judge whether there are meaningful differences between recall and recognition in a patient.

This relative difficulty with free recall as opposed to recognition has obvious implications for compensating for everyday memory problems of FL patients. These patients may be better able to answer questions that rely on recognition (e.g., "Did you talk to your sister on the telephone yesterday?") rather than on recall (e.g., "Who did you talk to on the telephone yesterday?"). Providing organization at encoding (Gershberg & Shimamura, 1995; Incisa della Rocchetta & Milner, 1993) or giving cues in the form of notes or reminders should also decrease the demands on strategic search at retrieval.

Given the difficulty that FL patients have when presented with insufficient cues, it would be useful to develop a self-cuing retrieval strategy for everyday life. Most mnemonics (such as the method of loci) essentially provide a framework of cuing, but the problem is that one has to use the framework at encoding for it to be effective at retrieval. Can there be some procedure designed to help people cue themselves at retrieval without also engaging in some stilted methods of encoding? At present, there seem to be few studies concerning this question. Yet even if one could devise a good retrieval strategy, it might be difficult to train patients to implement it. In order to use a retrieval strategy in a sophisticated, flexible way, a patient would need (at a minimum) to (1) recognize there is a problem, (2) retrieve the appropriate strategy, (3) initiate and maintain it, (4) evaluate the result, and (5) switch to a new strategy if he or she recognizes that the current one is ineffective. Performing this series of operations requires good monitoring, metamemory, and inhibition—the very processes with which FL patients have difficulty (see Burgess et al., 2000). Never-

theless, research in the rehabilitation of executive function and problem-solving is currently in its infancy (see Turner & Levine, 2004), and so, if in the future, FL patients can be taught to implement other sorts of problem-solving strategies, perhaps they can be taught to do so for memory as well.

Monitoring at Retrieval

There are at least two cases where FL damage seems to impair monitoring of memories at retrieval (i.e., decision-making about whether the products of a memory search are accurate or not). We discuss each, in turn, next.

Liberal response bias

Although FL patients have fewer difficulties with recognition than recall, there are aspects of recognition that appear to be abnormal. For example, on a yes-no recognition memory test, people are shown a mix of previously seen items and new ones, and have to discriminate the old from the new. A person endorsing more of the new items (*false alarms*) relative to the old ones (*hits*) is said to have a more liberal response bias. Persons with MTL damage usually have a reduced hit rate, but show normal (or conservative) response bias. In contrast, FL damage may have little effect on hit rate, but can lead to an elevated false alarm rate, suggesting more liberal bias (e.g., Swick & Knight, 1999; but see Verfaellie et al., 2004). Frontal patients are especially susceptible to producing false alarms when the foils are perceptually or conceptually similar to targets (Baldo et al., 2002; Melo et al., 1999; Schacter et al., 1996). There may be some regional, and hemispheric, specialization within FL with regard to false alarms. For example, when Rapcsak and colleagues (2001) divided FL patients into those with unilateral left *versus* right hemisphere damage, the right frontal group was more liberal on a face memory task than the left frontal and control groups (compare to Alexander et al., 2003, who reported abnormal bias for verbal materials in left frontal patients).

The studies outlined earlier concern large groups of FL patients, who usually show a small but significant shift in bias, but there are single cases that are more extreme. Rapcsak and colleagues have reported several patients with right FL damage who show pathologically high false recognition rates, across a variety of stimuli. Most of these patients also show false recognition of information in the retrograde domain, suggesting a retrieval deficit. In one study, Rapcsak et al. (1999) examined whether changing retrieval instructions could attenuate false recognition. They administered a face recognition task, in which famous and nonfamous faces were presented, and subjects were asked to endorse only the famous ones. The FL patients showed normal memory for the famous faces, but pathologically high false recognition of the nonfamous ones. The investigators then administered a similar task, but this time they changed the instructions during the test, asking participants only to endorse a face if they could state the name, occupation, or other identifying information for it. Giving these extra instructions had little effect on controls but led to a dramatic improvement in the patients, presumably by encouraging them to be more cautious, and/or make an endorsement based on more detailed information (i.e., using "recollection" rather than mere "familiarity" or "gist;" Jacoby, 1991; Mandler, 1980).

Schacter and colleagues (1996) made a similar argument in describing B.G., a right FL lesion patient who also showed pathological false alarming. The authors found that when foils were conceptually similar to targets (e.g., members of the same category), B.G. was very likely to false alarm to the foils, but when they were not similar, B.G.'s false alarm rate fell greatly. Note, however, that Parkin and colleagues (1999) reported a similar patient, but argued that his deficit involved faulty encoding. It could be that some patients are liberal due to poor encoding (e.g., impoverished, general coding of items) and others due to poor retrieval (e.g., abnormal bias and/or confidence), and this may depend on the hemisphere or frontal region damaged. Although many of the models mentioned at the outset describe criterion setting as a function of prefrontal cortex (e.g., Henson et al., 2000), Moscovitch and Winocur's (2002) is the only one which considers these biases explicitly by assigning criterion-setting and automatic monitoring functions to the ventromedial and frontopolar regions, respectively, with damage to them leading to changes in negative and positive biases, respectively. Thus, damage to ventromedial prefrontal cortex can lead to acceptance of false information typical of confabulation, and damage to frontopolar regions can lead to impairment in endorsing true memories or imbuing them with a sense of self-relatedness and recollection (for further discussion see Moscovitch & Winocur, 2002).

Clinical evaluation and real world performance. Some clinical memory measures include yes-no recognition, but the most popular of these uses only verbal stimuli (the CVLT-II), and liberal bias could be a material-specific problem (cf. Alexander et al., 2003; Rapcsak et al., 2001). The WMS-III has a yes-no face recognition subtest, but does not include normative data for false alarms or a criterion measure. The ideal clinical measure would compare yes-no to forced-choice recognition across verbal and nonverbal stimuli, because the latter may be easier for FL patients.

In the real world, it is currently unclear what proportion of FL patients have major problems resulting from a liberal bias in recognition, but there are at least a few cases that do. A good example is described by Ward and colleagues (1999). This patient would approach strangers on the street, asking if they were personal acquaintances or television stars, because they seemed so familiar to him. This spurious sense of familiarity might cause FL patients to be more vulnerable to scams and exploitation in the real world, where they might be more likely to believe what is told to them because it seems familiar (Jacoby, 1999). Harassment or other inappropriate social interactions may also be a concern. Fortu-

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nately, even in more extreme cases, encouraging patients to use a more conservative response criterion, or to search their memory carefully before making a decision, may help reduce false alarm rates greatly (as in Rapcsak et al., 1999; Schacter et al., 1996; but see Parkin et al., 1999).

Confabulation

Confabulation (or "honest lying;" Moscovitch, 1989; for recent reviews, see Gilboa & Moscovitch, 2002; Johnson et al., 2000), describes the phenomenon in which patients retrieve information that is untrue, accompanied by a strong feeling that it *is* true, and are unaware of any discrepancy, and usually of any memory impairment at all. Confabulation is most common in autobiographical memory, although it can be evoked in other domains, including semantics or general knowledge (Moscovitch & Melo, 1997). Many theorists have dichotomized confabulations (albeit, using different nomenclatures and criteria) into those that are severe (e.g., bizarre, spontaneous, persistent, frequent, etc.) *versus* those that are mild (e.g., realistic, provoked, momentary, rare, etc.), although a comprehensive taxonomy of confabulation has yet to be established.

The most parsimonious explanation of confabulation attributes it to a retrieval deficit, because patients often confabulate regarding memories acquired long before brain damage. In separate reviews of the literature, Gilboa and Moscovitch (2002) and Johnson et al. (2000) found that ventromedial FL regions (in either hemisphere) were most often implicated in confabulation, consistent with the function ascribed to this region in various models. Although basal forebrain damage (as in cases of anterior communicating artery aneurysm; AcoA) is common in confabulating patients, it does not always lead to confabulation, and may not be necessary to produce it. When confabulation resolves over time, it does so in a gradual and seemingly spontaneous manner, and does not necessarily improve at the same rate as other memory abilities (see Gilboa & Moscovitch, 2002).

Clinical evaluation and real world performance. Confabulation may be difficult to detect in the clinic, because we often take patients at their word, and do not seek corroboration of their reports unless we suspect a need. Although confabulation is associated with poor memory and executive function, the literature suggests these correlations are not very strong. Spontaneous or fantastic confabulations may be easier to spot than mild ones (simply because they are implausible, inconsistent, or bizarre), but mild ones may also cause problems in daily functioning. Two published methods may aid in documenting confabulation in the clinic. Dalla Barba (1993) has designed a series of questions to which most normal people would answer "I don't know" (e.g., "What did you have for lunch on March 12, 1998?"). As well, Moscovitch and Melo (1997) used a cue-word procedure and found that confabulations could be elicited for both personal and semantic information. Another good way to detect confabulation is by asking both the patient and a relative or friend about a few of the patient's life experiences, and seeing how the two reports square with one another.

Despite its rarity, confabulation can have severe real-life consequences. As stated earlier, it is usually accompanied by unawareness of deficit. If there is no reason for a patient to think he is confabulating, there is no reason for him not to act according to his memory. Few reports of rehabilitation or management of confabulation exist in the literature, possibly because it is so difficult. Asking the patient to use a stricter criterion at retrieval, or to reason through a series of logical steps to see that his confabulation cannot be true, may do little to convince him, especially in the long run (see Moscovitch, 1989). Burgess and McNeil (1999), however, did report a successful case of rehabilitation of an AcoA patient who would try to leave his house each morning for a nonexistent job. The authors had the patient keep a diary of his daily experiences, and trained him to check it at the beginning of each day to convince himself that he had not been working recently, and had nothing scheduled for the day. Of course, this method may not be successful with every patient. Confabulation may have multiple possible causes, for example, faulty memory search, decision-making, or temporal context memory (Burgess & Shallice, 1996; Moscovitch & Melo, 1997; Schnider, 2001; for reviews of models, see Gilboa & Moscovitch, 2002; Johnson et al., 2000). If so, different rehabilitation strategies may work with different patients.

ADAPTING CURRENT MEMORY REHABILITATION METHODS FOR FL DYSFUNCTION

Given the memory impairments resulting from FL damage or dysfunction, a number of rehabilitation or compensatory techniques may be particularly useful in this group. Although few studies exist on the efficacy of traditional memory training techniques in FL patients, many of these techniques address general problems such as decreased self-initiation and interference, and thus may be relevant to this population. We outline a few briefly.

Memory book training is frequently used in patients with impaired memory resulting from MTL lesions (Kapur, 1995). The use of a memory book bypasses impaired memory processes by using the external aid as a memory storage and retrieval device. Procedural/implicit memory can be used to learn the new procedures inherent in the use of a memory book. This may be also fruitful in FL patients, because procedural/implicit memory is by and large intact in them (Robinson, 2001). This approach is being evaluated by our colleagues at Baycrest (Richards et al., 1990; Wu et al., 2004), as well as elsewhere.

Prospective memory involves remembering to do something in the future. The ability to perform future intended tasks is impaired in FL patients (e.g., Brunfaut et al., 2000), likely because of poor initiation of the action or distraction

before the action is completed. The formation of implementation intentions is one way to circumvent these difficulties (Gollwitzer, 1999). This technique simply involves forming and mentally rehearsing plans of action to occur in response to specific situations (e.g., "Each night when I go to bed, I will take my white pill.") Forming these implementation intentions is thought to create situational cues that, when they are encountered later, automatically activate the desired behavior and effectively bypass the need for effortful self-initiation. Although to our knowledge this technique has not been applied to patients with focal FL damage, it can improve prospective memory in healthy older adults (Chasteen et al., 2001), a population with subtle but reliable decline in frontal and executive function (e.g., Raz et al., 1997; West, 1996).

Another potentially useful procedure involves *training recollection*, using a repetition lag procedure (Jennings & Jacoby, 2003). As previously described, FL patients may rely more on automatic influences on memory (i.e., familiarity) than consciously controlled memory processes (i.e., recollection). The repetition lag procedure trains recollection over multiple sessions using a continuous recognition task that requires recollection (as opposed to familiarity) of repeated items over gradually increasing intervals. Training with older adults has shown enhanced recollection of information within the training task (Jennings & Jacoby, 2003) and generalization of training effects to other memory tasks (Jennings et al., 2005).

Finally, *errorless learning* may be particularly important for patients with FL dysfunction (Baddeley & Wilson, 1994). In general, the ability to learn new information is enhanced when errors are prevented (reviewed in Kessels & de Haan, 2003). Frontal patients may be likely to incorporate errors into memory when learning, and, having done so, be unlikely to inhibit or unlearn them (Wilson, 2002). Errorless learning is more effective than an errorful technique in the acquisition of new information by patients with amnesia if they have FL dysfunction (Komatsu et al., 2000), presumably because errorless learning decreases interference from incorrect responses.

NEUROTRANSMITTERS AND PHARMACOTHERAPY

The previous sections discussed cognitive manipulations that may attenuate FL memory deficits, but others kinds of manipulations may also be successful. For example, Kramer and colleagues have suggested that aerobic exercise can improve cerebral function and thereby attenuate age-related declines in memory and executive function (for a review, see Colcombe et al., 2004). It is possible that such an approach might also benefit patients with brain injuries.

As well, there is a small but growing literature on the neuropharmacology of memory. Many neurotransmitters are implicated in memory (for a review, see Arnsten & Robbins, 2002), but we will focus on one, dopamine (DA), to illustrate current knowledge and future potential. One clue regarding dopamine comes from studies of Parkinson's disease (PD), which is associated with decreased levels of DA in the basal ganglia and cerebral cortex.[1] Although motor problems are the most obvious aspect of the disease, Parkinson's patients have subtle memory problems akin to those of frontal patients (especially those with dorsolateral FL lesions; for reviews, see Bondi & Troster, 1997; Saint-Cyr, 2003). These deficits likely occur because low levels of DA cause dysfunction of FL cortex itself, which is rich in dopamine receptors, or interfere with the interaction between FL cortex and the basal ganglia. When PD patients are given DA agonist drugs to improve their motor signs (e.g., l-dopa), they show a modest improvement on many of the memory and executive tasks on which they are normally impaired (e.g., Gotham et al., 1988; Lange et al., 1992). The FLs are probably implicated in this effect, although to date there are few functional neuroimaging data linking drug-related improvements in patients' memory with changes in FL activation (but see Cools et al., 2002 for an initial step).

Could these drugs ameliorate memory problems in people with other kinds of FL damage or dysfunction? McDowell et al. (1998) recently conducted one of the few human studies along these lines. They gave either a DA agonist (bromocriptine) or placebo to TBI patients (who showed impaired executive function, and probably had some FL damage). The drug improved performance on many of the executive measures, such as reducing perseverative errors on the Wisconsin Card Sorting Test, but did not improve performance on nonexecutive tasks, such as letter cancellation. Perhaps results of this kind could generalize to other patients with FL damage or dysfunction. There are, however, at least three reasons to be cautious. First, there may be vast individual differences in who benefits from a drug; for example, some PD patients show little response to DA agonists. Second, drugs may improve some processes but have a minimal, or detrimental, effect on others. For example, Cools et al. (2003) reported that giving DA agonists to PD patients improved their task-switching performance, but made the same patients abnormally liberal in a decision-making task (see also Gotham et al., 1988). They argued that that by increasing levels of DA they restored normal function to the system supporting task-switching, but "overdosed" the system responsible for decision-making. Finally, in cases of focal lesion there may not be enough tissue left to function properly, even if the patient is given the right drugs (Arnsten & Robbins, 2002).

EVALUATING MODELS OF FRONTAL FUNCTION IN MEMORY

A few decades ago, it was widely held that the FLs had little to do with episodic memory. It was only in the 1980s that a

[1]Dopamine has also been implicated in animal studies of memory. For example, the prefrontal cells that fire during delayed objects and location memory tasks are usually dopaminergic (Goldman-Rakic, 1998), although there is some controversy over whether the D1 or D2 receptor is most important, and other neurotransmitters undoubtedly play a role, as well.

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first generation of models began to differentiate clearly frontal from medial temporal contributions to memory (e.g., Moscovitch and Winocur, 1992; Petrides & Milner, 1982). Since then, a second generation of models has emerged, some of which we outlined at the beginning of this article. Each posits different cognitive processes, and (with a few exceptions) maps these onto different frontal lobe regions. Because many of the models are arguably heuristic and still in their early stages, at present it may be more useful to focus on similarities among them than differences.

The first, and possibly most important, point of agreement among current models is that there are multiple frontal processes supporting memory. In the past, this would have been a more controversial statement, but an abundance of dissociations in lesion and neuroimaging studies supports the fractionation of frontal function into multiple subcomponents (even if there is disagreement as to how many there are, and what they each do). Second, there is *some* convergence among current models already. For example, with respect to retrieval, there appears to be a consensus that at least two major stages are required: Cue specification and memory search have been attributed to ventrolateral FL by many authors (e.g., Moscovitch & Winocur, 2002; Petrides, 2002; Shallice, 2002; Simons & Spiers, 2003), whereas post-retrieval evaluation of the output of the memory search, especially when the decision is uncertain, has been localized to dorsolateral FL (Fletcher & Henson, 2001; Moscovitch & Winocur, 2002; Petrides, 2002; Shallice, 2002; Simons & Spiers, 2003). There also appears to be a growing consensus regarding the role of the ventromedial FL in confabulation.

A third generation of model development and testing is clearly needed, one which will allow us to identify more precisely the memory operations supported by the frontal lobes. Many of the present models are still somewhat underspecified, so that any given finding can be explained according to multiple theories, and there seem to be few cases in which one can unambiguously pit one theory of FL function and memory against another (although for initial attempts see Burgess & Shallice, 1996; Shimamura, 2000). Further complicating matters, of course, is the high degree of within- and between-subject variability following frontal damage. Nevertheless, as our understanding of frontal lobe function and memory improves, this will obviously help to guide evaluation and rehabilitation.

EVALUATION OF FRONTAL MEMORY PROBLEMS

As we have discussed, the FLs likely support multiple processes in episodic memory. Consequently, no single test will provide a comprehensive measure of the ability to work with memory. Undoubtedly, new tasks will become available as researchers develop more precise and sophisticated theories of FL memory functions. However, of all the currently available clinical measures of memory, perhaps the CVLT and CVLT-II cover the most ground when it comes to frontal memory impairments. This test allows one to estimate organization (both semantic and subjective), context memory (albeit, only for temporal order memory), memory search (by contrasting recall and recognition), and response bias (in yes/no recognition). It would be useful if there were a similar nonverbal test, because frontal memory deficits can be material-specific; however, clinicians may be able to use some of the nonverbal tasks we have reviewed in a piecemeal fashion.

Two other tests that have made their way from the experimental literature to the clinical domain are Petrides and coworkers' self-ordered pointing (Petrides & Milner, 1982) and conditional-associative learning tasks (Petrides, 1985). In the self-ordered pointing task, participants are shown a set of items (words or pictures) on a sheet, and are required to point to one of them. Then they are shown another sheet with the same items in new locations and they are required to point to a new item, and so on until all the possible items have been sampled. Although patients with MTL damage fail on this test because they cannot remember the items they have seen, patients with FL damage fail because they cannot monitor their responses effectively. The deficit is associated with mid-dorsolateral lesions (Brodmann area 9/46) on the left or right depending on whether the material is verbal or spatial. The conditional-associative learning test is associated with response selection deficits caused by lesions in premotor cortex (Brodmann area 6/8). In this task, participants are required to map different responses to different stimuli, which patients with lesions to those areas have difficulty accomplishing, as do patients with Parkinson's disease (Vriezen & Moscovitch, 1990).

Collecting subjective information (from the patient and a relative or caregiver) may also be useful for evaluation. First, memory problems may be evident in the real world, but not in the clinic. Second, comparing a patient's own ratings to those of a relative or caregiver can give one the sense of whether insight and meta-memory are normal. Unfortunately, although many rating scales of memory, executive ability, and/or behavior exist (e.g., Frontal Systems Behavior Scale, Grace & Malloy, 2001; Memory Assessment Clinics Self-Rating Scale, Crook & Larrabee, 1990; Metamemory in Aging, Dixon et al., 1988), none are specific to the memory problems commonly seen in FL disorders. We would suggest having the patient and a family member answer questions about frontal memory processes by using a Likert-scale rating to describe the frequency and/or severity of FL-related memory problems, or forcing them to choose between "worse than before" and "same as before" to answer. Table 1 shows examples of the kinds of questions we would include in such a questionnaire.

Finally, more general problems may underlie what appear to be "frontal" memory impairments, and must be addressed in diagnosis and rehabilitation. First, apathy, depression, and other mood disorders are common in brain injury and many brain diseases. Left hemisphere lesions may be more likely to produce depression, whereas right hemisphere dam-

Table 1. Examples of possible items for a frontal memory questionnaire

1. Do you organize complex information that you need to remember, for example, by categorizing a grocery list by food groups? (*Semantic organization*)
2. Do you use some kind of strategy to help you remember things (e.g., a to-do list, shopping list, appointment calendar)? (*Organization*)
3. Can you recount the plot of a movie or TV show you have seen, or book you have read? (*Organization, temporal order*)
4. Can you remember the order of events that occurred during the day or on a vacation? (*Temporal order*)
5. Do you have difficulty remembering where or when you read or heard a specific piece of information? (*Contextual information*)
6. Do you have difficulty remembering who told you something? (*Contextual information*)
7. Do you have difficulty remembering whom you have told something? (*Contextual information*)
8. When you want to retrieve something from your memory, do you use some kind of strategy to help you? (*Cuing/retrieval*)
9. Do you forget a name or fact, but recall it later? (*Cuing/retrieval*)
10. Do you forget to do something, but remember it when prompted by someone else? (*Cuing/retrieval; Prospective memory*)
11. Do you have to use some kind of reminder to help you to remember to do something? (*Cuing/retrieval*)
12. When you are uncertain whether you truly remember something, do you lean more towards saying "yes" than "no?" (*Liberal response bias*)
13. Do you tend to say "I don't know" when people ask you about something you're not sure of? (*Liberal response bias*)
14. Do you tend to give the first response that comes to you when somebody asks you to recall something? (*Liberal response bias*)
15. Can you be convinced of something that never happened? (*Liberal bias/confabulation*)
16. Do you make up a story or "invent" details when you have difficulty remembering the actual details of an event? (*Confabulation*)

age may yield indifference or euphoria (for a review, see Mayberg, 2002). Patients with mood disorders may be less likely to want to work with memory, or work with anything else for that matter (Wilson, 2002). Second, FL damage may greatly reduce metamemory and metacognition (e.g., Janowsky et al., 1989b). In more extreme cases, such as confabulation, FL patients are typically unaware of their deficit. If a patient is not aware that he or she has a memory problem, then he or she is less likely to engage the self-initiated strategies that are the hallmarks of FL memory function. The former problem (mood) may be more amenable to therapy, whereas the latter (metacognition and awareness of deficit) may be less so, but both should be considered when evaluating and planning a rehabilitation program for FL memory impairment.

SUMMARY AND CONCLUSIONS

In this review, we have outlined the effects of FL lesions on memory, and highlighted cases where FL impairment can be attenuated or eliminated in the laboratory. Evaluating FL memory processes in the clinic may give the clinician a better idea of what processes are impaired, what kinds of problems patients may face in the real world, and what to do to minimize such problems. Administering a brief memory test may not be enough to detect a memory problem. It seems critical to measure performance on some of the FL memory tasks outlined earlier, or at least to examine the more "frontal" scores on clinical tasks like the CVLT, because patients have to work-with-memory very often in everyday life.

Two kinds of questions, theoretical and practical, must be answered in future research. On the theoretical side, we must learn more about the basic functioning of the FLs in memory, and the connections and interactions among frontal, medial temporal, and other brain regions. Recently it has become easier to do so, using functional neuroimaging. Regional specificity within the FLs means that damage to different subregions or subsystems will produce different kinds of memory problems, and as we learn more about these we will be better able to know exactly what to expect in individuals with different kinds of FL lesions. In rehabilitation theory, unanswered questions include why some functions are more likely to recover than others, and how best to encourage recovery (by teaching alternative cognitive strategies, and/or promoting neural plasticity).

On the practical side, we must develop applied neuropsychological studies of the FLs and memory. We are a long way from applying the detailed experimental knowledge and theoretical models to the clinic, but the broad conclusions derived from the laboratory can guide clinical practice. Indeed, the time is ripe for such an undertaking. Few of the behavioral interventions outlined here seem to have been attempted in the real world. Although it remains unclear whether what we have reviewed can translate from the lab to a patient's home or work, we believe that some procedures are highly likely to succeed. Fortunately, research on the frontal lobes and memory has grown rapidly in recent years, and should continue to do so. When it comes to linking basic research with real world management and rehabilitation of frontal memory impairment, hopefully we will make as much progress in next few decades as we have in the last few.

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