FACETS OF MEMORY DISTORTION: EVIDENCE FROM
AMNESIA, CONFABULATION AND FRONTAL-LOBE DYSFUNCTION

by

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False recall and false recognition of nonstudied words that are preceded by a list of strong associates was examined in amnesic patients with medial temporal lobe or ventromedial frontal-lobe damage and in nonamnesic patients with frontal-lobe damage. We hypothesized that susceptibility to false recall and false recognition is determined by several factors, namely having some memory for the studied words, the ability to extract the semantic and associative information about the studied list, and a deficit in strategic monitoring. Nonamnesic frontal-lobe patients consistently produced high levels of false recall and false recognition. Since these patients had relatively good memory they extracted the semantic information about the list. However, due to their strategic monitoring deficit they failed to reject nonstudied related words. The false recollection pattern of amnesic patients was more variable. Those who extracted the semantic/associative nature of the list and remembered it, produced high levels of false recall.
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Day to day life would be impossible if the human brain was not equipped with mechanisms which allowed memories to be formed and retrieved. Indeed, the survival of the human species is dependent on its ability to access accurate information about the past. Despite its obvious efficiency, our memory system is not perfect and memories themselves do not preserve a literal representation of the world. Consequently, memories are not always accurate, and under some conditions, may be extremely distorted. Beginning with the pioneering studies of Sir Frederic Bartlett (1932), psychologists have been aware that memory is a fundamentally constructive process that is sometimes prone to errors, distortions, and illusions. Clearly, understanding memory distortion has many important implications for everyday life. Recently, cognitive psychologists and neuropsychologists have been especially concerned with constructive aspects of memory, in part as a result of real-world controversies concerning the suggestibility of children’s memory and the accuracy of memories recovered in psychotherapy. These debates raise issues that are relevant to cognitive neuroscience. For example, how accurate is memory and under what conditions is it subject to distortion? In recent years, investigators have begun to explore the phenomena that illuminate constructive aspects of remembering, such as source amnesia, false recognition, false recall, and confabulation.

**HISTORICAL BACKGROUND:**

The first controlled studies that provided evidence of memory distortion were carried out by European investigators who were interested in the reliability and suggestibility of eyewitness testimony, particularly children’s testimony. Binet (1900), for instance, exposed children to various objects and then tested their memories in the presence or absence of misleading questions. Binet found that misleading questions produced systematic distortions
in children’s recollections. Stern (1910) described experiments in which the investigator staged an event in front of a group or a class, and later asked them questions about what happened and who did it. Like Binet, Stern reported that misleading questions could induce distorted memories, and he also observed that young children were apt to confuse real and imagined events. Evidence concerning the unreliability of eyewitness memory was summarized in a well-known monograph by psychologist Hugo Munsterburg (1908).

In addition to these empirical demonstrations of distorted memory, the late 19th- and early 20th- centuries witnessed a number of relevant theoretical developments. In experimental psychology, the predominant account of memory distortion held that stored memories fade, change, and alter spontaneously over time, although it was never specified exactly how such alterations took place (e.g. Kennedy, 1898). An alternative view was advanced by the German biologist Richard Semon (Semon, 1904/1921; 1909/1923).

According to Semon, every act of encoding and storing new information (engraphy) involves some retrieval of thoughts, images, and memories that are activated by the current situation (ecphory). Thus, a newly created memory trace or engram is not a literal replica of reality, but is always an interpretation that includes retrieved information. If the input to the memory system is not an accurate reflection of reality, then the output will necessarily be distorted. Semon also argued that memory distortion arises because every act of ecphory constitutes an act of engraphy. That is, when we remember a past experience, it is encoded anew into the memory system. However, we may focus on or think about only certain aspects of the retrieval experience, thereby changing its subsequent memory representation. Semon demonstrated great foresight in proposing these ideas, though they were generally ignored in his day.
Freud developed a rather different and influential approach to memory distortion. Freud’s best known ideas about distortion are related to his still controversial notion of repression, which holds that painful memories are defensively excluded from consciousness. Although Freud defined the concept of repression differently at different times, he consistently argued that recollection of past experience could be distorted by dynamic and defensive forces. Freud’s discussions of the effects of early sexual trauma on adult psychopathology are also related to issues of memory distortion. In his early writings, Freud (1896) argued that repressed memories of sexual abuse and trauma cause persisting pathological symptoms, and contended that the task of psychoanalysis is to allow patients to retrieve lost traumatic memories.

As is now well known, Freud soon abandoned this “seduction theory” of early trauma in favor of the view that the “memories” that his patients retrieved were in fact fantasies or confabulations. Whatever the reasons for his change in view, it is clear that many aspects of Freud’s later psychoanalytic thought were built on the notion that early memories are highly likely to be distortions that are produced by fantasy-based confabulations. This theme was evident in Freud’s (1899) idea of “screen memories”, which holds that recollections of early childhood experiences are often distortions that protect people from a more unpleasant reality that is hidden behind the superficial “screen” image that is presented to consciousness.

Clinical observations of memory distortion in brain-damaged patients were also made during the turn-of-the-century period. Shortly after Korsakoff’s (1889) initial description of the amnesic syndrome that now bears his name, Bonhoeffer (1904) pointed out that such patients often produce extensive and sometimes bizarre confabulations concerning events that
never occurred. These memory distortions contrasted sharply with the patients’ inability to remember accurately even the simplest episodes from their day-to-day lives.

During the 1920s and 1930s, a number of experiments were conducted in psychological laboratories that examined the hypothesis that memories change autonomously over time in a manner dictated by Gestalt laws of organization. That is, memories change spontaneously toward becoming “good Gestalts”. For example, Wulf (1922) reported that memories of visual forms become more symmetrical with increasing time after initial learning. Koffka (1935) cited these autonomous transformation effects as support for his Gestalt theory of memory. Others, however, argued that such effects are largely experimental artifacts (e.g. Zangwill, 1937). Modern researchers have tended to agree with this latter view, and have thus dismissed the Gestalt account of memory distortion (e.g. Baddeley, 1976).

The single most important development concerning memory distortion during this period was the publication of Bartlett’s (1932) classic monograph *Remembering*. Bartlett exposed his subjects to an engaging story, an old Indian legend entitled *The War of the Ghosts*. After hearing the story subjects were asked to retell it on several occasions. Bartlett found that people rarely recalled all of the events in the story accurately; they often remembered occurrences that made general sense, or fit their expectations of what should have happened, even though they were not in fact part of the original story. Bartlett also observed that the recollections of his participants changed, sometimes substantially, across multiple retellings of the story. Curiously, Bartlett’s original results have not been replicated by others and may have been attributable to the specific test instructions that he gave to his subjects.
Nonetheless, on the basis of his observations Bartlett concluded that memories are imaginative reconstructions of past events that are heavily influenced by the rememberer’s preexisting knowledge structures or schemas. Bartlett argued that schemas influence what is extracted from an experience and determine how it is reconstructed. He argued further that remembering is a fundamentally social activity that is inevitably distorted by the attitudes and needs of the rememberer. Bartlett’s (1932) contribution was an enduring one because he distinguished between reproductive and reconstructive memory. Reproductive memory refers to accurate rote production of material from memory, whereas reconstructive memory emphasizes the active process of filling in missing elements while remembering, with errors frequently occurring.

In the tradition of Bartlett’s early ideas, it has generally been assumed that the act of remembering materials rich in meaning gives rise to reconstructive processes (and therefore errors), whereas the act of remembering simplified materials (e.g. nonsense syllables, word lists) gives rise to reproductive (and thus accurate) memory. For this reason, the investigators of false memories have mostly followed Bartlett’s (1932) lead. Most evidence has been collected in paradigms that use sentences (Bransford & Franks, 1971; Brewer, 1977), prose passages (Sulin & Dooling, 1974), slide sequences (Loftus Miller, & Burns, 1978) or videotapes (Loftus & Palmer, 1974). In all these paradigms, evidence of false memories has been obtained, although the magnitude of the effect depends on the method of testing (McCloskey & Zaragoza, 1985; Payne, Toglia, & Anastasi, 1994). The predominance of materials that tell a story (or can be represented by a script or schema) can probably be attributed to the belief that only such materials will cause false memories to occur.
There were two very significant deviations from this general trend in research on false memories. Both used a list-learning paradigm. Deese (1959), for instance, had subjects study a list of words (e.g. bed, pillows, rest, sheets, etc.) that are strong associates of a nonstudied critical item (e.g. sleep), and then asked for recall of the list words. He reported that subjects often produced the nonstudied critical items on the recall test. That is, they exhibited false memories for the nonstudied but strongly associated items. Recent work indicates that Deese’s procedure is indeed a powerful method for inducing subjectively compelling false recollections (McDermott, 1996; Roediger & McDermott, 1995).

Underwood (1965) reported a similar phenomenon in the domain of recognition memory. He gave subjects a continuous recognition task in which they decided if each presented word had been given previously in the list. Words appearing later in the list bore various relations to previously studied words. Underwood showed that words associatively related to previously presented words were falsely recognized. Underwood argued that these false recognitions arise from “implicit associative responses” that occur during initial study of the list items.

In the neurological and neuropsychological literatures, scattered clinical observations of confabulation in Korsakoff’s patients continued to appear, together with a variety of new theoretical accounts of the phenomena (e.g. Talland, 1961). For instance, Talland (1961) distinguished between two major forms of memory distortion in Korsakoff amnesics: confabulation, where patients misremember the time and other contextual aspects of actual events; and fabrication, where patients concoct improbable and bizarre scenarios that could not have in fact occurred. In addition, Whitty and Lewin (1957) reported that patients who had undergone surgical removal of the anterior cingulate for relief of intractable obsessional
neurosis exhibited spontaneous confabulations even though they did not exhibit amnesia, thereby suggesting that the presence of severe memory disorder is not a necessary condition for the occurrence of confabulation. Other non-Korsakoff amnesic patients were described, such as the famous case of H.M. (Scoville & Milner, 1957), who displayed a profound loss of memory for recent events after bilateral resection of the medial temporal lobes. H.M. did not exhibit extensive confabulation, thus indicating that severe memory disorder is also not a sufficient condition for confabulation (McGlynn & Schacter, 1989; Moscovitch, 1995). These kinds of observations suggest that confabulation is based on brain structures that are different from the structures that are damaged in cases of severe amnesia.

The 1970s witnessed a surge of interest in “cognitive constructivism”. This was stimulated in part by Neisser’s (1967) constructive account of memory which rekindled interest in Bartlett’s ideas about schemas and reconstructive memory. By his view, all memories are constructions because they include general knowledge that was not part of a specific event, but is necessary to reconstruct it. It is this inherently reconstructive nature of memory in turn that makes it susceptible to various kinds of distortions and inaccuracies.

As a result of this rediscovery of Bartlett’s ideas, cognitive psychologists focused intensively on investigating various kinds of memory distortions. For example, Sulin & Dooling (1974) examined errors attributable to schema-based inferences. They gave subjects brief passages to read about a wild and unruly girl; some were told that the passage was about Helen Keller, and others were told that the passage concerned Carol Harris. After a week’s delay, the former group of subjects were much more likely than the latter to claim that they had read the sentence “She was deaf, dumb and blind”, even though the sentence had not been presented in the initial passage.
At around the same time, Loftus and her colleagues published a series of studies that demonstrated that leading questions can systematically alter memory reports (Loftus & Palmer, 1974), and that post-event misinformation can alter memory for an original event (e.g. Loftus, Miller & Burns, 1978). In neuropsychology, new evidence pointed toward a link between frontal-lobe dysfunction and confabulation (Stuss, Alexander, Lieberman, & Levine, 1978), and the theoretical implications of confabulation for theories of memory began to be appreciated (Mercer, Wapner, Gardner, & Benson, 1977).

In the 1980s and 1990s, significant inroads were made in the study of source memory, retrieval environment, and implicit memory, with important implications for reconstructive memory. For example, source memory, also referred to as source monitoring (Johnson, Hashtroudi, & Lindsay, 1993) refers to processes that allow people to remember when, where and how a memory was acquired. Interest in this issue was sparked by several developments. Johnson and colleagues (1981) reported an important series of experiments and ideas concerning the processes involved in distinguishing between memories of actual events and memories of prior imaginings and fantasies (“reality monitoring”). Subsequent studies delineated conditions under which recollections of external events and internal imaginings can be confused, thereby producing distorted memories (Johnson & Suengas, 1989). This line of research also began to yield evidence that young children have difficulties distinguishing between memories of real and imagined events (Johnson & Foley, 1984), a finding that has been confirmed and extended in various recent studies (Ceci, 1995).

Neuropsychological research revealed that brain-damaged patients sometimes exhibit a dramatic phenomenon known as source amnesia. Schacter, Harbluk, and McLachlan (1984) reported that amnesic patients could occasionally learn new fictitious facts such as
“Bob Hope’s father was a fireman”. However, even when they could recall a fact, the patients were often unable to remember that the experimenter had just told it to them minutes earlier. Instead, they frequently invented a plausible source, claiming that they had read the fact in a newspaper or heard about it on the radio. Importantly, amnesics who showed signs of frontal-lobe damage were more vulnerable to source amnesia than amnesics who did not exhibit frontal signs (Schacter et al., 1984; Shimamura & Squire, 1987). Subsequent research indicated that source amnesia could be observed in patients with restricted frontal-lobe lesions (Janowsky, Shimamura, & Squire, 1989). Source memory deficits were also documented in elderly adults, particularly those with neuropsychological signs of frontal impairment (Craik, Morris, Morris, & Loewen, 1990; McIntyre & Craik, 1987; Schacter, Kaszniaik, Kihlstrom, & valdiserri, 1991), and in young children (Lindsay, Johnson, & Kwon, 1991; Schacter, Kagan, & Leichtman, 1995).

Source amnesia became directly linked to memory distortion in several different ways. Neuropsychological studies suggested that failures of source memory and reality monitoring are implicated in the confabulations of brain-damaged patients, and also provided increasing evidence that frontal-lobe pathology is often associated with confabulation (DeLuca & Cicerone, 1991; Johnson, 1991; Moscovitch, 1995; Moscovitch & Melo, 1997). Cognitive research revealed that failures of source memory play a key role in the kind of post-event misinformation effects that were initially studied by Loftus and colleagues (Belli, Lindsay, Gales, & McCarthy, 1994; Lindsay, 1990; Zaragoza & Lane, 1994). These experiments indicated that when people witness a particular event, and are later given misleading information about it, they often fail to remember whether the critical information was part of the original event or was only suggested to them later. In light of other
experiments showing that post-event information does not necessarily eliminate the original memory (McCloskey & Zaragoza, 1985), it is now clear that failures of source memory are a major contributor to memory distortions that are produced by post-event misinformation. Ceci (1995) describes recent developmental evidence indicating that distorting influences of post-event suggestion in young children are closely linked with deficient source memory.

Another important line of research revealed that when people forget the source of their knowledge, they become susceptible to various other kinds of memory distortions and illusions. For instance, Jacoby, Kelley, Brown & Jasechko (1989) found that people who had been exposed to a nonfamous name would later call that name “famous” if they were tested after a long delay and no longer recollected that they had seen the name in a previous experimental session. The name may have seemed like a familiar one when it was exposed a second time, but because people failed to recollect the source of their knowledge they mistakenly attributed the name’s familiarity to the “fame” of the nonfamous person. This result highlights that people often make inferences and attributions concerning the source of retrieved knowledge, and that these source attributions are quite prone to error (Kelley & Jacoby, 1990). This observation provides a link to the various phenomena of implicit memory that were investigated extensively during the 1980s, such as priming and skill learning (Roediger & McDermott, 1993; Schacter, 1987; Schacter, Chiu, & Ochsner, 1993). Implicit memory phenomena, by definition, involve loss of conscious access to source information. Isolated images and feelings that may manifest as priming effects or related phenomena could provide a basis for inaccurate reconstruction of past events when people attempt to make attributions about the source of unexplained images and feelings (Kelley & Jacoby, 1990; Squire, 1995; Schacter, 1996).
There has also been a growing body of laboratory research on memory for emotionally-arousing events, which indicates that emotional arousal typically enhances the accuracy of memory for the central aspects of an event and impairs memory for more peripheral details (Heuer & Risberg, 1992). Some emotionally compelling “flashbulb memories” can even be subject to outright distortion (Neisser & Harsch, 1992). In addition, research on mood and memory during the 1980s began to reveal that a person’s mood can exert biasing effects on memory, such that information that is incongruent with a current mood tends to be well remembered, and information that is incongruent with a current mood tends to be more poorly remembered (Bower, 1992). These effects have been observed in college students and also in patients suffering from depression and other affective disorders (Mineka & Nugent, 1995).

Finally, research conducted during the 1980s and 1990s has supported the idea that memory consists of a variety of dissociable systems and subsystems (Cohen & Eichenbaum, 1993; Eichenbaum, 1997; Gabrielli, 1995, Moscovitch, 1994; Schacter & Tulving, 1994; Squire, 1992; Vargha-Khadem, 1997). This perspective has encouraged the view that memories are constructed on the basis of stored fragments of experience that are distributed throughout a variety of cortical systems that work cooperatively with cortical storage areas during both encoding and retrieval (Damasio, 1989; McClelland, McNaughton, & O’Reilly, 1995; Schacter, 1996; Squire, 1992). From this perspective, memory distortions are a natural by-product of constructive activities that draw on multiple components and subsystems (McClelland, 1995; Squire, 1995).

In many areas of memory research, neuropsychological study of amnesic patients has had a large impact on theorizing about normal memory. Nevertheless, despite a century’s
worth of psychological research concerning memory distortions and illusions (Schacter, 1995a) and scattered observations of confabulations and related false memories in brain-damaged patients (Johnson, 1991; Johnson, O’Connor, & Cantor, 1997; Moscovitch, 1995; Moscovitch & Melo, 1997) there has been little progress in our theoretical understanding of memory illusions. Widening the scope of research on memory illusions to include extensive neuropsychological study of various patient populations would provide useful information about the neural and cognitive processes underlying illusory memories.

One of the few attempts at examining memory distortion in amnesic patients is found in a study of false recognition reported by Cermak, Butters, and Gerrein (1973). Cermak et al. used the false recognition paradigm developed by Underwood (1965). They found that when Korsakoff amnesics and controls encountered the four different types of lures: unrelated, associates, synonyms, and homophones, amnesic patients produced significantly more false alarms to associates and homophones than did control subjects, and they also showed a slight, nonsignificant trend for false alarms to synonyms and unrelated words. These data suggest that amnesic patients are sometimes more prone to false alarms than are nonamnesic controls. Other studies of recognition memory have shown that amnesics sometimes exhibit a higher false alarm rate than control subjects even to nonstudied words that have no particular relation to studied words (Knowlton & Squire, 1995; Verfaellie & Treadwell, 1993). Together with their reduced hit rates, the elevated false alarm rates of amnesic patients in the latter studies may reflect an inability to discriminate between studied and nonstudied items resulting in haphazard guessing.

The available evidence suggesting that amnesic patients sometimes make more false positive responses than control subjects raises the possibility that they might be unusually
susceptible to memory illusions that are expressed by false alarms to nonstudied items.

Reinitz, Verfaellie, and Milberg (1996) report that amnesic patients are more prone than controls to false alarms based on illusory memory conjunctions, where subjects claim to have seen a new stimulus when in fact they have only seen its component features (Reinitz, Lammers, & Cochran, 1992). Kroll, Knight, Metcalfe, Wolf, and Tulving (1996) report similar findings in patients with left, right, or bilateral hippocampal damage. These effects may reflect impaired consolidation processes in amnesic patients, processes that ordinarily serve to bind together distinct attributes of an event into a unified engram.

Yet, the apparent susceptibility of amnesic patients to produce more false memories cannot be applied to all experimental situations. For example, Schacter, Verfaellie & Pradere found that amnesic subjects were far less susceptible to false recognition of critical lures than were controls, whereas they made more false alarms to unrelated lures than control subjects did. They used a version of the Deese (1959) paradigm described earlier, recently revived and modified by Roediger & McDermott (1995). Roediger & McDermott (1995) replicated the false recall effect initially reported by Deese, and extended it to recognition memory. After studying associates of nonpresented target words such as sweet, participants frequently claimed with high confidence that sweet had appeared previously on the study list. Moreover, when asked to make remember/know judgments (Tulving, 1985) indicating whether they maintained a specific recollection of having studied a word during list presentation ("remember"), or thought that a word was on the list because it seemed familiar ("know"), people claimed to "remember" nonpresented associates nearly as often as they claimed to "remember" words that had actually appeared on the list (Roediger & McDermott, 1995).
Using this paradigm, Schacter, Verfaellie, and Pradere (1996) reported that both Korsakoff and non-Korsakoff (i.e. medial temporal lobe) amnesic patients showed reduced levels of veridical recognition memory compared to a matched control group. Amnesics attained fewer hits than controls to previously presented words and made more false alarms than controls to nonpresented words that bore no associative relationship to previously presented words. More importantly, amnesic patients made fewer false alarms than controls to nonpresented associates such as *sweet*.

This latter finding suggests that false recognition of nonpresented associates depends on retention of associative or semantic information that also supports veridical recognition of presented words - information that Schacter and colleagues suggest is not available to amnesic patients. More specifically, they contend that control subjects bind together studied items and generated associates, thereby forming and retaining a well-organized, focused representation of the semantic gist of the study list. Related test distractors that match this semantic gist representation, such as *sweet*, are likely to be falsely recognized; unrelated distractors that do not match it are likely to be correctly rejected. They suggest further that amnesic patients do not form and/or retain a focused semantic representation of gist. On the one hand, this idea accounts for the finding that amnesic patients showed reduced false recognition of related distractors: there is little opportunity for a related test item to match a semantic gist representation. On the other hand, this suggestion can also account for amnesics’ inflated false recognition of unrelated distractors: whereas control subjects can use their semantic gist representation to reject unrelated distractors that do not match it, amnesic patients are less able to do so. Encoding and/or retrieval of semantic gist information presumably depends on medial temporal lobe structures that are damaged in amnesia.
Schacter and colleagues (1996) suggest that when numerous associates of a false target are presented, as in the Roediger and McDermott/Deese paradigm, nonamnesic controls establish a well-organized gist representation that enhances the sense of familiarity or recollection associated with a matching false target, and thus increases the magnitude of the false recognition effect. Amnesic patients, by contrast, encode or retain less gist information and hence show reduced levels of false recognition.

However, when only a single related item precedes a false target, as in the Underwood paradigm, nonamnesic controls establish a less robust gist representation and can use their intact explicit memory abilities to counteract or oppose (Jacoby, 1991) the sense of familiarity or recollection they may experience when encountering a false target. Thus, for example, a nonamnesic individual who encounters the false target table, and can recollect having previously studied the associate chair, can use this information to avoid making a false recognition response (Brainerd et al., 1995). Amnesics, however, are less able to use recollection to oppose the sense of familiarity engendered by a false target, and thus exhibit increased levels of false recognition compared to nonamnesic controls.

The studies of false memory in the neuropsychological patients just described have focused on those with damage to medial temporal lobe structures including the hippocampus. The difference in production of false memories in the two types of paradigms described would suggest that these structures are critical for the processes underlying false memory in the Deese/Roediger and McDermott (1959/1995) task, namely remembering semantic or associative information.

In a PET study with normal control subject, Schacter et al. (1996) made an important observation. They found that the medial temporal lobe was activated as much during
recognition of false targets as true targets, suggesting that “faulty” output from the medial temporal lobe is a contributing factor to false memory. This suggests that because output from the medial temporal lobe is not perfect even in normal people, memory distortions or false memories are necessarily a common feature of normal memory. However, it appears that other false memory phenomena may not be dependent on these particular structures.

A number of neuropsychologists have noted that damage to the ventromedial aspect of the frontal-lobes and basal forebrain are often associated with the most flagrant form of memory distortion, namely confabulation (Dalla Barba, 1993; Johnson, 1991, Johnson et al., 1997; Moscovitch, 1995, Moscovitch & Melo, 1997). Moscovitch and his colleagues have proposed that the prefrontal cortex is necessary for strategic search, monitoring, verification and organization of the automatic output from medial temporal lobe structures. Confabulation, therefore, arises as a result of impairment to strategic retrieval and monitoring processes that depend on prefrontal regions (Moscovitch, 1995; Moscovitch & Melo, 1997).

Recently, the frontal-lobes have been implicated in other false memory phenomena such as false recognition. Schacter and colleagues (1996) have recently studied a 65-year-old man, BG, who suffered an infarction restricted to the right frontal-lobe (Schacter, Curran, Galluocco, Milberg, & Bates, 1996). From their description of this case study, it was clear that BG does not spontaneously generate extensive confabulations, shows no signs of amnesia, and is generally alert, attentive, and cooperative. He does, however, exhibit a striking pattern of false recognitions that provides useful clues concerning the role of prefrontal regions in illusory memories. Parkin and colleagues (1996) have reported a patient with left frontal-lobe damage who makes excessive numbers of false alarms and resembles patient BG in many respects. Schacter et al. (1997) also found that normal old people, whose
medial temporal lobe and prefrontal structures tend to be compromised, exhibited an exaggerated false memory effect. Taken together, these findings support the idea that memory distortion is exaggerated when output from the medial temporal lobes is faulty and monitoring by the prefrontal cortex is impaired, as is likely in the case of the elderly. When few, if any, of the associates (true targets) are remembered, as in the case of amnesia, there effectively is no medial temporal lobe output that can be prone to distortion.

It appears then that a variety of distinct neural and psychological processes underlie different kinds of memory illusions and these are not yet fully understood. The broader question that emerges from these findings concerns the monitoring function of the prefrontal cortex in producing memory distortions. Because the Deese/McDermott and Roediger (1959/1995) paradigm induces faulty output from the medial temporal lobe even in normal people, it might be the case that poor monitoring has no role in producing false memories in this laboratory procedure. On the other hand, if poor monitoring contributes to false memories, prefrontal cortex patients should show increased levels of false memories in this paradigm. This prediction is appropriate because an exaggerated production of false recall and false recognition in the Deese/Roediger and McDermott (1959/1994) paradigm appears to be the product of three cognitive processes: 1) having some memory for the study lists, 2) being able to extract the semantic/associative gist of the lists, and 3) having a deficit in monitoring that makes decisions about specific items in a given list prone to error. The first two processes are subserved by medial temporal lobe structures and the third is dependent on the prefrontal cortex. These criteria are met by patients with damage restricted to the frontal-lobes so they should show elevated levels of false memory. The purpose of the present study is to elucidate the relationship between prefrontal cortex function and memory distortion.
To address this question we administered the false memory paradigm, as described by Schacter, Verfaellie & Pradere (1996) to three groups of patients, namely non-confabulating amnesics with damage to the medial temporal lobe or diencephalic structures, confabulating amnesics who had medial temporal lobe damage but also had additional damage to the ventromedial prefrontal regions, and non-confabulating, non-amnesic patients with damage restricted to the prefrontal cortex which did not include the ventromedial prefrontal regions. It was predicted that if the above three criteria are necessary for increased production of false recall and false recognition than it should be possible to replicate Schacter et al.’s (1996) finding that amnesic patients, with damage restricted to medial temporal lobe structures, should produce significantly less false recall and recognition than normal controls and that non-amnesic frontal-lobe patients should produce more false recall and false recognition than normal controls. We have included this other, very interesting subgroup of amnesics, those who have additional damage to the ventromedial aspect of the prefrontal cortex because on the one hand, they have a deficit in monitoring which probably leads to their propensity to confabulate, but on the other hand they have very severe memory deficits which will impair their ability to remember the lists and extract the semantic gist. Thus, we would predict that, in the Deese/Roediger & McDermott paradigm, any propensity they may have towards memory distortion (i.e. false recall and false recognition) will be counteracted by their severe amnesia.

METHOD

Participants.
The participants in this study were 12 people with brain damage (5 females, 7 males). Seven of these people were amnesic patients with various etiologies. Three amnesics were classified as confabulators and 4 as non-confabulators, depending on their behaviour in real life and on their performance on a test that elicited confabulations (see Moscovitch & Melo, 1997). Lesion localization was not as precise as one would have liked for this group of patients. Not all patients had focal lesions, in some cases the lesion was diffuse. The damage in all 3 of the patients who were classified as confabulators was caused by some form of cerebral vascular accident. All 3 of these patients had confirmed lesions in the ventromedial aspect of the prefrontal cortex. This damage is consistent with that reported by other investigators to be associated with confabulation (De Luca & Diamond, 1995; Fisher, Alexander, D’Esposito, & Otto, 1995; Moscovitch, 1995; Moscovitch & Melo, 1997). These patients also had evidence of additional damage to diencephalic or medial temporal-lobe structures. Because the non-confabulating amnesics all had damage to diencephalic or medial temporal-lobe structures (but not to the ventromedial frontal cortex), it is unlikely that damage to these structures leads to confabulation, though it does produce severe memory loss which may be a prerequisite for confabulation (Moscovitch & Melo, 1997). The average age of the confabulating amnesics was 60, and their mean education level was 11.7 years. The nonconfabulating amnesics were slightly younger and more educated than the confabulating amnesics; they had a mean age of 50.5, and an average of 16.5 years of education. The intellectual functioning of the entire group of amnesics was in the average range, as indicated by a mean Full Scale IQ of 92.3 on the Wechsler Adult Intelligence Scale - Revised. In contrast, they consistently exhibited severe deficits on a variety of explicit memory tasks.
from the Wechsler Memory Scale - Revised. They obtained a mean General Memory Index of 69 and a mean Delayed Memory Index of 53.

A second group of participants consisted of 5 people who had focal lesions restricted to the frontal-lobes but which did not include the ventromedial region. Their memory was not severely impaired and they were not classified as confabulators. The average age of these patients was 57.4, and their education level was on average 15.2 years. The overall intellectual functioning of these patients was in the average range. They did not exhibit severe memory impairment.

Table 1 shows, for each subject, the etiology, probable or confirmed site of brain damage as determined by MRI, CAT or SPECT scans, and demographic data.

Control subjects consisted of eight individuals (4 males, 4 females) with an average age of 54.5 and a mean education level of 13 years.
Table 1: Demographic data on Patients

<table>
<thead>
<tr>
<th>I.D.</th>
<th>Sex</th>
<th>Age</th>
<th>Edu.</th>
<th>Etiology</th>
<th>Lesion Location</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>Non-confabulating Amnesics</strong></td>
<td></td>
</tr>
<tr>
<td>DA</td>
<td>male</td>
<td>45</td>
<td>17</td>
<td>Herpes encephalitis</td>
<td>bilat. MTL</td>
</tr>
<tr>
<td>KC</td>
<td>male</td>
<td>45</td>
<td>15</td>
<td>Traumatic brain injury</td>
<td>bilat. MTL, R-fronto-par, occ</td>
</tr>
<tr>
<td>CC</td>
<td>male</td>
<td>60</td>
<td>22</td>
<td>Astrocytoma and hydroceph.</td>
<td>3rd vent., diencephalon</td>
</tr>
<tr>
<td>PT</td>
<td>female</td>
<td>52</td>
<td>14</td>
<td>R-Temporal artery malformation</td>
<td>R-TL</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>Confabulating Amnesics</strong></td>
<td></td>
</tr>
<tr>
<td>FE</td>
<td>female</td>
<td>60</td>
<td>15</td>
<td>ACommA aneurysm clipped</td>
<td>L-orbitofrontal and dorsolateral frontal</td>
</tr>
<tr>
<td>RC</td>
<td>male</td>
<td>61</td>
<td>10</td>
<td>CVA</td>
<td>bilat. orbitofrontal incl. VMF region</td>
</tr>
<tr>
<td>GE</td>
<td>male</td>
<td>59</td>
<td>10</td>
<td>CVA</td>
<td>bilat. frontal including bilat VMF region</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td><strong>Frontal Patients</strong></td>
<td></td>
</tr>
<tr>
<td>CB</td>
<td>female</td>
<td>29</td>
<td>12</td>
<td>Epilepsy/Resection</td>
<td>Right dorsolateral frontal</td>
</tr>
<tr>
<td>DW</td>
<td>male</td>
<td>59</td>
<td>13</td>
<td>Brain Tumour</td>
<td>Right frontal</td>
</tr>
<tr>
<td>PK</td>
<td>female</td>
<td>35</td>
<td>16</td>
<td>Traumatic brain injury</td>
<td>Right frontal</td>
</tr>
<tr>
<td>ED</td>
<td>female</td>
<td>65</td>
<td>7</td>
<td>ACommA aneurysm clipped</td>
<td>Bilateral frontal</td>
</tr>
<tr>
<td>DS</td>
<td>male</td>
<td>60</td>
<td>16</td>
<td>ACommA aneurysm clipped</td>
<td>R-Inferior medial frontal</td>
</tr>
</tbody>
</table>
Materials

The materials consisted of 24 lists of 16 words, identical to those used by Roediger and McDermott (1995). Each list contains 15 words to be presented for study and a target word (critical lure) that is not presented for study. The study words are all highly associated to the critical lure and are ordered such that the strongest associates occur first in the list. In order to counterbalance the list order, the 24 lists were subdivided into three sets of 8 lists. The lists were presented auditorily via a desktop computer equipped with Soundblaster software.

Design and procedure

Because one purpose of this study was to replicate Schacter, Verfaellie & Pradere’s (1996) findings with amnesic patients, all participants were tested in the two conditions used in that experiment (i.e. Study + Recall condition and a Study + Arithmetic condition, administered in two sessions separated by at least one week). Half of the subjects received the Study + Recall condition during the first session and the Study + Arithmetic condition during the second session. For the other half of the participants, this order was reversed.

In each condition a set of eight lists was used. The remaining eight lists were not studied. Four of these appeared on the recognition test that accompanied the Study + Recall condition, whereas the other four appeared on the recognition test that accompaned the Study + Arithmetic condition. Lists were counterbalanced so that they were used equally often in the Study + Recall condition, in the Study + Arithmetic condition, and as nonstudied lists. Nonstudied lists were counterbalanced across the two recognition tests. Before presentation of each study list, the subjects were told that they would hear several lists of words via speakers attached to a PC and that they should try and remember the words. The words were
recorded in a female voice and were presented at a rate of one word every 1.5 seconds.

Immediately following presentation of a study list, participants were asked to say out loud as many of the words as they could remember (Study + Recall condition) or to perform simple addition and multiplication problems (Study + Arithmetic condition). Approximately 1 minute was given for the completion of either of these tasks, after which the next study list was presented. Eight study lists were presented during a single session. Presentation of all eight study lists took approximately 20 minutes.

The recognition test was administered approximately 2 minutes after completion of the recall or arithmetic task that followed the final study list. The recognition test contained 48 words, 24 studied words and 24 nonstudied words. The studied words were obtained by selecting for each of the eight study lists the items in serial positions 1, 8, and 10. The nonstudied words consisted of the critical lures corresponding to each of the eight studied lists, the critical lures corresponding to four nonstudied lists, and the items in serial positions 1, 8, and 10 of these nonstudied lists. The words were presented all at once in columns on a computer screen.

Subjects were asked to indicate for each word whether they had heard it earlier in any of the lists. They were instructed to move down the columns using the cursor key and to select (by pressing a different key- the space bar) only those words they had heard previously. For each of the words they selected, they were then asked to indicate whether they remembered or knew the word by typing an “R” (remember) or a “K” (know) beside the word. Instructions for the remember/know judgment were similar to those used by Roediger and McDermott (1995) and Schacter, Verfaellie & Pradere (1996). Participants were told that a remember judgment should be made if they can specifically recollect hearing a word
via the speakers- that is, they can actually travel back in their minds to the precise moment of hearing and studying the word and remember something about that event. It was explained that such recollection might include remembering something about the speaker’s voice or about the thoughts they had when they heard the word. They were told that a know judgment should be made if they feel or know that a word was presented earlier on the tape recorder, but if they cannot recollect anything specific about the word or its occurrence.

RESULTS:

**Free Recall.** The mean proportion of study list words and critical lures produced by the three patient groups and control subjects on the free recall test averaged across the eight target lists is presented in Table 2. Due to the relatively small number of subjects in each of the patient groups, nonparametric analyses were conducted. As expected both groups of amnesic patients (nonconfabulators and confabulators) recalled on average a much smaller proportion of study list words (.29 and .21 respectively) than did controls (.49), $Z = 2.318$, $p<.05$ (nonconfabulators); $Z = 2.311$, $p<.05$ (confabulators). The difference between the proportion of study list words recalled by nonconfabulating amnesics was not significantly different from that recalled by confabulating amnesics, $Z = 1.427$, $p<.5$. Although the proportion of study list words recalled by control subjects was higher than the proportion of study list words recalled by frontal patients (.38) this difference was not significant, $Z = 1.8$, $p<1$. There was also no significant difference between the proportion of study list words recalled by frontal patients and that recalled by nonconfabulating amnesics, $Z = 1.246$, $p<.5$. The difference between the proportion of study list words recalled by frontal patients and that recalled by confabulating amnesic patients approached significance, $Z = 1.938$, $p<.06$. 
Contrary to our expectations, nonconfabulating amnesic patients intruded a larger proportion of critical lures (.50) than did controls (.38), although this difference did not attain significance, \( Z = .631, p < 1 \). In Schacter et al.’s study, amnesics produced about the same proportion of study list targets and critical lures (.27 vs .29), whereas control subjects produced significantly more study list targets than critical lures (.52 vs .33). Although the control subjects produced significantly more study list targets than critical lures (.49 vs .38), the proportion of study list words recalled by the nonconfabulating amnesic patients was considerably, though nonsignificantly, lower than the proportion of critical lures intruded (.29 vs .50). In contrast, confabulating amnesics intruded a nonsignificantly smaller proportion of critical lures (.21) than did controls (.39), \( Z = 1.713, p < .5 \). Also the proportion of critical lures intruded by confabulating amnesics was not significantly different from that produced by nonconfabulating amnesics. Interestingly, the pattern of performance for the confabulating amnesics was similar to the performance of the amnesic patients in Schacter et al.’s (1996) study. That is, the confabulating amnesics produced the same proportion of study list targets and critical lures (.21 vs .21). The frontal patients showed a similar pattern of performance as the nonconfabulating amnesics. They produced a larger proportion of critical lures (.45) than did controls (.39), although this difference was nonsignificant, \( Z = .279, p < 1 \). They also produced a smaller proportion of study list words than critical lures (.38 vs .45). The proportion of critical lures intruded by frontal patients was not significantly different from nonconfabulating amnesics. However, it differed significantly from confabulating amnesics, \( Z = 2.037, p < .05 \). Although they are inconclusive the preceding nonparametric analyses provide some evidence for a difference in the relation between critical lure intrusions and recall of target items in the patients and controls.
However, this difference, at least with respect to the nonconfabulating amnesic patients is not in the predicted direction based on Schacter et al.'s (1996) findings. Nonconfabulating amnesic patients in this study did not produce a significantly smaller proportion of critical lures than either control subjects or frontal patients. Figure 1 depicts the relation of critical lure intrusions and recall of target items for each group.

Following Schacter et al. (1996), the intrusions of critical lures were compared to the mean proportion of words recalled from the nonrecency and nonprimacy regions of the serial position curve (i.e. positions 4-11). These data are presented in Table 3. For control subjects, the only group in the previous analyses whose overall recall rate was higher than the proportion of critical lures intruded, the proportion of studied words recalled from these positions (.37) was now about equal to the proportion of lures intruded (.38). A similar pattern, though more pronounced, was found in all three patient groups. For nonconfabulating and confabulating amnesics, the proportion of words recalled from the middle positions of the serial position curve (.18 and .10 respectively) was nonsignificantly smaller than the proportion of critical lures intruded (.50, .21 respectively), $Z = 1.461$, $p=.14$ (nonconfabulating amnesics); $Z = 1.069$, $p=.29$ (confabulating amnesics). These findings do not support the notion that the relation between recall of studied words and intrusion of critical lures differs in amnesic patients and control subjects. The proportion of study list words recalled from serial position 4-11 (.25) was significantly lower than the proportion of critical lures intruded (.45) by frontal patients, $Z = 2.023$, $p<.05$. This suggests that frontal patients are more likely than control subjects to produce false recall of critical lures.
Table 2: Mean proportion of study list words and critical lures produced by patients and control subjects.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Proportion of Study List Words Produced</th>
<th>Mean Proportion of Critical Lures Intruded</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonconfabulating Amnesics</td>
<td>0.29</td>
<td>0.50</td>
</tr>
<tr>
<td>Confabulating Amnesics</td>
<td>0.21</td>
<td>0.21</td>
</tr>
<tr>
<td>Frontal Patients</td>
<td>0.38</td>
<td>0.45</td>
</tr>
<tr>
<td>Control Subjects</td>
<td>0.49</td>
<td>0.38</td>
</tr>
</tbody>
</table>
Figure 1: Mean proportion of study list words compared to critical lures intruded by patients and controls
Table 3: Mean proportion of study list words recalled from the nonrecency and nonprimacy regions of the serial position curve (positions 4-11) and critical lures produced by patients and control subjects.

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean proportion of studied words from serial positions 4-11</th>
<th>Mean proportion of critical lures intruded</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonconfabulating Amnesics</td>
<td>0.18</td>
<td>0.50</td>
</tr>
<tr>
<td>Confabulating Amnesics</td>
<td>0.10</td>
<td>0.21</td>
</tr>
<tr>
<td>Frontal Patients</td>
<td>0.25</td>
<td>0.45</td>
</tr>
<tr>
<td>Control Subjects</td>
<td>0.37</td>
<td>0.38</td>
</tr>
</tbody>
</table>
The findings by Schacter et al. (1996) suggested that amnesics make on average many more noncritical lure intrusions than control subjects. Furthermore, the nature of these intrusions is somewhat different for the two groups. Specifically, while the two groups were equally likely to produce unrelated intrusions, amnesic patients were less likely than controls to produce intrusions related to the just studied list, and correspondingly more likely to produce perseverations to previously studied lists. This pattern of intrusions did not hold for the patients in the present study. Aside from one nonconfabulating amnesic patient who produced a tremendous number of noncritical lure intrusions (38 in total), amnesic patients in general produced very few noncritical lure intrusions. In fact when the one nonconfabulating amnesic patient who produced such a large number of intrusions was removed, the nonconfabulating amnesics produced an average of .292 noncritical intrusions per list and confabulators produced an average of .125 noncritical intrusions per list. This is in comparison to control subjects who produced .472 noncritical intrusions per list.

Furthermore, with respect to the nature of these intrusions, there were no apparent differences between controls and amnesics in terms of the type of intrusions made. Frontal patients also made relatively few noncritical intrusions (.275/list) and the nature of these intrusions did not differ from the other groups.

If critical lure intrusions arise from associative processes operating during recall itself than one might expect more critical lure intrusions when subjects recall many targets from a list than when they recall few targets. Schacter et al. (1996) addressed this issue which was originally raised by Roediger & McDermott (1995). They found that amnesic patients recalled more target items from lists for which they produced the critical lure than from lists for which they did not produce the critical lure. Control subjects in their experiment showed
comparable levels of target recall whether they produced the critical lure or not. In the present experiment control subjects, showed comparable levels of target recall whether they produced the critical lure (mean = 7.1) or not (mean = 7.8). However, it was not the case that either nonconfabulating amnesics or confabulating amnesics produced more target items from lists for which they produced the critical lure (mean = 4.0 for nonconfabulators, mean = 3.0 for confabulators) than from lists for which they did not produce the critical lure (mean = 4.0 for nonconfabulators, mean = 3.2 for confabulators). Rather, like controls subjects, amnesic patients, regardless of whether they were confabulators or not produced comparable levels of target recall regardless of whether they produced the critical lure or not. This pattern was also true of the frontal patients whose mean recall was 5.2 when they produced the critical lure and 5.7 when they did not. This analysis, although not conclusive due to the small number of patients in each group, does not appear to support the suggestion that associative processes during the recall test play a more prominent role in the critical lure intrusions of amnesic patients than control subjects or frontal patients.

To examine this issue further, we followed Schacter et al. (1996) once again and examined the output position of the critical lure. It was reasoned that a relatively late output position for the critical lure would tend to indicate a role for associatively related items produced previously during the recall test. For nonconfabulating amnesics, the average output position for critical lures was 4.46 (of 4.02 words produced for lists in which there was a critical lure intrusion). For nonconfabulating amnesics, the average output position for critical lures was 3.5 (of 3 words produced for lists in which there was a critical lure intrusion). For frontal patients, the mean output position was 5 and the average number of items produced for these lists was 5.2. For controls, the average output position for critical
lures was 5.45, but the average number of target items produced for these lists was 6.81. Thus, in relative terms, and in support of the finding by Schacter et al., all three patient groups produced the critical lure later than did the control subjects. This finding appears to support the idea that in control subjects the intrusion of critical lures is less dependent on associative processes during recall than it is in amnesic and frontal patients.

**Recognition.** Table 4 presents the proportion of old responses to studied words, critical lures, and their corresponding distractors for the three patient groups and control subjects. As already mentioned, subjects were tested on two separate occasions. On one occasion they were asked to engage in free recall prior to the recognition test and on the other they were asked to do simple arithmetic questions prior to the recognition test. As in Schacter et al.’s (1996) study it was found that type of task (free recall vs. arithmetic) did not yield any main effects or interactions, so all analyses are collapsed across the free recall and arithmetic conditions.

Analyses of studied words and corresponding distractors revealed a significantly higher hit rate in control subjects (.70) than in nonconfabulating amnesic patients (.47), $Z = 2.224$, $p < .05$. Frontal patients also produced a significantly higher hit rate (.75) than nonconfabulating amnesics, $Z = 2.183$, $p < .05$. Although control subjects and frontal patients produced higher hit rates than confabulating amnesics (.66), these differences did not attain significance. There was a significantly higher false alarm rate in the nonconfabulating amnesics (.54) than in control subjects (.13), $Z = 3.154$, $p = .01$. Similarly, confabulating amnesics showed significantly higher levels of false alarms (.43) than the control subjects (.13), $Z = 3.059$, $p < .01$. Frontal patients produced significantly lower levels of false alarms (.18) than either nonconfabulating amnesics ($Z = 2.612$, $p < .01$) or confabulating amnesics ($Z$
but there was no significant difference between the false alarm rate of the frontal patients and the control subjects.

In correcting for standard high-threshold, the false alarm rate was subtracted from the hit rate. This analysis revealed that recognition accuracy in nonconfabulating amnesic patients (.20) was significantly lower than in control subjects (.65), (Z = 3.719, p<.0001) or frontal patients (.67), (Z = 3.339, p<.005). Once again control subjects and frontal patients had proportionally higher recognition accuracy than confabulating amnesics (.45), however, these differences were not significant.

False alarms rates to critical lures (nonpresented words preceded by a list of high associates) were compared to false alarm rates for their corresponding distractors (words drawn from the same pool of critical lures that were not preceded by a list of high associates). Overall, nonconfabulating amnesic patients made fewer false alarms to critical lures (.66) than did control subjects (.83) but this difference did not achieve significance. However, nonconfabulating patients made significantly fewer false alarms to critical lures than did frontal patients (.93), Z=2.099, p<.01. The false alarm rate of confabulating amnesics to critical lures (.65) was proportionally lower than those of control subjects and frontal patients, however this difference was not significant. Nonconfabulating amnesics made more false alarms to the distractors for critical lures (.50) than did control subjects (.13), Z = 2.797, p<0.01; and frontal patients (.25) although the difference between these amnesics and frontal patients only approached significance (Z = 1.729, p<.09). Similarly, confabulating amnesics made more false alarms to the distractors for critical lures (.70) than did control subjects, Z =
Table 4: Recognition data (mean proportions) for studied and target words and studied and nonstudied critical lures in patients and controls

<table>
<thead>
<tr>
<th>Group</th>
<th>Studied items</th>
<th>Critical lures</th>
<th>Distractors for studied items</th>
<th>Distractors for critical lures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonconfab. Amnesics</td>
<td>0.47</td>
<td>0.66</td>
<td>0.54</td>
<td>0.50</td>
</tr>
<tr>
<td>Confabulating Amnesics</td>
<td>0.66</td>
<td>0.65</td>
<td>0.43</td>
<td>0.70</td>
</tr>
<tr>
<td>Frontal Patients</td>
<td>0.75</td>
<td>0.93</td>
<td>0.18</td>
<td>0.25</td>
</tr>
<tr>
<td>Control Subjects</td>
<td>0.70</td>
<td>0.83</td>
<td>0.12</td>
<td>0.13</td>
</tr>
</tbody>
</table>
3.551, p<.0001; and frontal patients Z = 2.874, p<.01. The difference in proportions of false alarms to the distractors for critical lures for control subjects and frontal patients was not significant.

These analyses provide evidence for the fact that amnesic patients and control subjects respond in a qualitatively different manner to the critical lures and their distractors. It also shows that amnesic patients and frontal patients differ in their response to critical lures and their distractors.

Although both groups of amnesic patients made more false alarms to distractors than did control subjects or frontal patients, they made fewer false alarms to critical lures than did control subjects or frontal patients. When the proportion of old responses to distractors for critical lures was subtracted from the proportion of old responses to critical lures, the corrected proportion was significantly smaller in both nonconfabulating (.40) and confabulating amnesics (.29) than in either control subjects (.75), Z = 2.573, p=.01 (nonconfabulators), Z = 2.561, p=.01 (confabulators); or frontal patients (.87), Z = 2.368, p<.01 (nonconfabulators), Z = 2.367, p<.01 (confabulators). Schacter et al. (1996) proposed that if subjects make significantly more old responses to critical lures than to distractors, this may suggest that the presentation of associatively related items reliably influenced their recognition performance. In the present study it was certainly the case that control subjects produced significantly more old responses to critical lures than to distractors, Z = 2.120, p<.0001; as did frontal patients, Z = 2.820, p<.005. Nonconfabulating amnesics also showed this effect, Z = 2.120, p<.05; whereas confabulating amnesics did not produce significantly more old responses to critical lures than to distractors.
A comparison of studied items and critical lures revealed that nonconfabulating amnesics, frontal patients, and control subjects made more old responses to critical lures than to studied items (.66 vs .47 for nonconfabulating amnesics, .93 vs .75 for frontal patients and .83 vs .70 for control subjects). This was a finding also made by Roediger & McDermott, 1995 and Schacter et al. (1996).

**Remember vs know responses.** We subdivided the proportion of old responses made by amnesic patients and controls into remember and know responses, respectively. Roediger and McDermott (1995) found that subjects claimed to remember critical lures as often as they claimed to remember studied words, and our data show a similar pattern for both control subjects and patients. Overall, control subjects provided remember responses to .60 of the studied words and .65 of the critical lures. Similarly, frontal patients provided remember responses for .65 of the studied words and .66 of the critical lures. Nonconfabulating amnesic patients provided remember responses to .47 of the studied words and .40 of the critical lures; the corresponding proportions for confabulating amnesics was .55 and .58. These findings are in line with Roediger & McDermott’s suggestions. However, as Schacter et al. (1996) found in their study, these affects are difficult to interpret in light of the fact that none of the groups used “know” responses more often for studied words than for their corresponding distractors. Similarly, none of the groups used know responses significantly more often for critical lures than for their corresponding distractors. These analyses imply that all subject groups tended to use the know response when they were just guessing that a word had appeared on the list, and used the remember response whenever they felt a degree of certainty that a word had been studied previously.
DISCUSSION:

The primary results of this experiment can be summarized as follows: At recall, when their overall intrusion rate was taken into account, confabulating amnesic patients showed a lower proportion of critical lure intrusions than did control subjects. However, nonconfabulating amnesics and frontal-lobe patients demonstrated a higher proportion of critical lure intrusions than control subjects. Furthermore, control subjects recalled more targets than critical lures, whereas nonconfabulating amnesic patients and frontal-lobe patients "recalled" more critical lures than targets. At recognition, both nonconfabulating and confabulating amnesics recognized a smaller proportion of studied words and intruded a smaller proportion of critical lures than did either controls or frontal lobe patients. The qualitative differences between these four groups are intriguing. At the outset of the study, it was hypothesized that individuals who met three specific criteria, would be most likely to show the highest levels of false recognition and recall would be high. Those conditions included having some memory for the word lists, being able to extract the semantic/associative gist of the lists and having a deficit in strategic monitoring processes. We, therefore, proposed that frontal-lobe patients would show the highest levels of false recall and false recognition because they fulfill all three criteria. While the data from this experiment support this prediction, they also provide evidence to suggest that high levels of false recall can occur if even one or two of these criteria are met.

The main finding in a study by Schacter, Verfaellie & Pradere (1996) was that amnesic patients, with primarily medial temporal lobe damage, were less likely than normal control subjects to produce false recall or false recognition (i.e. they were less likely to produce the unpresented critical lures for studied words). It was thus surprising to find that
nonconfabulating amnesic patients in our study actually produced a higher proportion of critical lures than controls at recall. It is even more surprising that amnesics produced at least as much false recall as frontal-lobe patients. In fact, it was predicted that if any of the amnesics were going to produce high levels of false memories, it would be the confabulators since they have frontal-lobe damage and they already demonstrate a propensity for memory distortion in their everyday life. Why is it then that nonconfabulating amnesics perform so much like frontal-lobe patients? Although one would not want to imply that exactly the same cognitive processes are occurring in both frontal-lobe patients and nonconfabulating amnesics, it appears that the end result is similar; namely these two groups of patients produced the highest levels of false recall. Nonconfabulating amnesics presumably have faulty medial temporal lobe output. Unlike frontal-lobe patients, nonconfabulating amnesics do not have a deficit in monitoring. Having suggested that the production of false memories in this paradigm is dependent on memory for the general theme of the study list and the individual words in it, both processes necessitating intact medial temporal lobe functioning, how can the high levels of false recall shown by these amnesic patients be explained?

Underwood’s (1965) classic “implicit associative response” account, can still account for this effect. According to this perspective, at the time of study, target words activate associates that are later confused with the target itself. One version of this idea holds that associates are truly activated implicitly, in the sense that the subject does not become consciously aware of the associate during study; it is generated by spreading activation through an associative network. Roediger and McDermott (1995) note a problem with this view. They propose that people claim to remember the critical lures, whereas responses based on implicit spreading activation would not be expected to generate such recollective
experiences. The data in Schacter et al.'s (1996) study, and our own findings speak to this issue - if responses to critical lures were based solely on nonconscious activation processes, we might expect amnesic patients to be influenced to the same degree as control subjects even at recognition since explicit memory processes would not be involved.

Another version of Underwood's account implies that subjects consciously think of the word at the time of study and are later subject to source memory confusion (Johnson, Hashtroudi & Lindsay, 1993), consequently, they can no longer remember whether they actually heard it or only thought of it themselves. This hypothesis is important to understanding false recognition of critical lures because it highlights the point at which distortion of memory occurs (i.e. when subjects mistake their prior thoughts for prior perceptions) (Schacter, Verfaellie, & Pradere, 1996). It has been found that frontal patients and amnesics tend to have more difficulty remembering source information than do controls (Schacter, Harbluk, & McLachlan, 1984; Shimamura & Squire, 1987). However, problems in source monitoring alone cannot be responsible for false recognition of critical lures in this experiment. That is, if amnesic patients (whether they are confabulators or nonconfabulators) are as likely as control subjects to generate the critical lure at the time of study (there is no empirical basis for suggesting that they can not, see Schacter, Verfaellie & Pradere, 1996), one would expect them all to make more false alarms to critical lures at recall and recognition.

Brainerd and Reyna’s “fuzzy trace” (Brainerd, Reyna & Kneer, 1995; Brainerd, Reyna & Brandes, 1995, Reyna & Brainerd, 1995) theory provides a useful framework for interpreting the data from this experiment. They account for the phenomena of false recall and recognition by proposing that memory can be based either on a “gist trace” that
preserves the general meanings and interpretations engendered by studied items or an a "verbatim trace" that preserves specific information about the exact identity of each item. It appears that although nonconfabulating amnesics are clearly impaired at recalling specific studied words (verbatim trace), they are nevertheless able to extract and retain the semantic gist of the list and so they produce the critical lure at recall. Certainly, this is a useful and generally effective strategy for amnesic patients to employ in everyday life. Since they are so impaired at retaining the specific details about a situation, they do well to focus their efforts on extracting the main theme. Indeed, for many situations in everyday life, we must all employ this strategy so as not to be overwhelmed by the sheer amount of information we are bombarded with.

The performance of these patients at recognition also lends support to Brainerd et al's theoretical stance. Because they are severely memory impaired, even the "gist trace" becomes degraded over time so that at recognition they no longer produce higher levels of false memory than controls or frontal patients; in fact they perform in much the same fashion as the confabulating amnesics. In comparison to frontal lobe patients and control subjects, both groups of amnesics produce significantly lower levels of false recognition.

Apparently though, not all amnesics use this strategy—as was evident from the performance of confabulating amnesics who produced low levels of false recall in our study. Why is there such a difference in the performance of confabulating and nonconfabulating amnesic patients at recall? Both groups have medial temporal lobe damage which causes their severe amnesia. However, the confabulating amnesics also have frontal-lobe damage. This additional damage to critical frontal regions may affect their ability to employ cognitive strategies effectively. Indeed, as Moscovitch and colleagues have proposed (Moscovitch,
1995; Moscovitch & Melo, 1997) confabulating amnesics have impaired strategic retrieval processes resulting from damage to the region of the ventromedial prefrontal cortex. These strategic retrieval processes not only help monitor output from the medial temporal lobe system but they also help initiate and guide search in episodic and semantic memory and they help organize the output from those systems as well. (Moscovitch & Melo, 1997). The fact that the confabulating patients do not use the strategy of extracting the gist of the study list may implicate the ventromedial prefrontal region in processes necessary for flexibility in implementing such a strategy. It is interesting that the very same pattern of damage leads to flagrant memory distortion (i.e. confabulation) on the one hand, but does not contribute to false recall and false recognition in the Deese/Roediger & McDermott paradigm on the other. Thus, while confabulating amnesics seem neurologically like good candidates for false recall and false recognition, due to their history of confabulation and their poor monitoring; as was mentioned earlier, a deficit in monitoring does not matter much when the output from the medial temporal lobe is almost nonexistent. Furthermore, their may be other deficits arising from their ventromedial frontal-lobe damage that do not allow them to employ strategies at encoding (such as extracting the semantic/associative gist of the lists).

The nonamnesic frontal-lobe patients are similar to the control subjects in several ways. They have a reasonably good memory of studied words and they are also able to remember the main theme of the lists. However, they differ from controls in that they also have a deficit in their ability to monitor their memories effectively. If poor monitoring contributes to the production of false recall and recognition in this paradigm, the frontal-lobe patients should produce more false recall and recognition than control subjects. Although the results of this study are consistent with this hypothesis, the statistical significance of our
findings is limited by the small number of subjects in each group. At both recall and recognition, the mean proportion of critical lures intruded was higher for frontal patients.

It is noteworthy that all of the frontal patients we tested had right frontal-lobe involvement. Schacter and colleagues (1996) have recently documented a case of a man who suffered an infarction restricted to the right frontal lobe. He exhibits a striking pattern of false recognitions that provides useful clues concerning the role of the prefrontal regions in illusory memories. The investigators suggest that BG relies excessively on information about the general correspondence between a test item and previously studied words when making a recognition decision. Control subjects in this study typically claimed to “remember” that a word or picture had appeared on a study list only when they retrieved specific information about a particular word or picture. BG by contrast, relied inappropriately on a match between a test item and general characteristics of the study episode when making his recognition decisions. In addition, Schacter et al. (1996) analyzed what he claims to recall when he makes a “remember” false alarm. They found that he tends to provide associations to other words or to events in his life, specific information from an inappropriate context. Given previous evidence implicating the frontal lobes in memory for source or contextual information (Schacter, Harbluck & McLachlan, 1984), these authors suggest that it seems likely that a source memory deficit contributes to the character of BG’s false recollection. According to this view, the right prefrontal cortex may be involved in setting up contextual representations that guide retrieval by allowing one to focus in on a target episode and filter out irrelevant material. Norman and Schacter (1996) have argued that BG’s deficits are related to problems generating a specific, focused representation of a study episode and filtering out nonessential information. These observations would also hold for the frontal-
lobe patients who participated in this experiment and are very closely related to the issue of defective monitoring processes in frontal-lobe patients. Just like BG, the frontal-lobe patients in our study may be capable of generating only a vague or unfocused representation of characteristic features of the target words against which test items are matched. Items, in this case the critical lures, that are generally similar to previously studied ones are accepted as old because they match the general contextual representation and are not filtered out.

The findings from this study illustrate the fact that the relationship between various kinds of memory distortions are complicated. Memory distortions are in no way a unitary process. By comparing and contrasting different kinds of memory illusions, it should be possible to delineate the component processes involved in each of them.
References


Curran, T., Schacter, D. L., Norman, K. A., & Gallucio, L. (?). False recognition after a right frontal-lobe infarction: Memory for general and specific information. *Neuropsychologia*


Schacter, D.L., Reiman, E., Curran, T., Sheng Yun, L., Bandy, D., McDermott, K. B., &


