The Status of Semantic and Episodic Memory in Amnesia

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Abstract

Since Scoville and Milner's landmark report of the amnesic patient referred to as H.M., it has been known that the medial temporal lobes play a critical role in our ability to form new memories. Importantly, studies of amnesia have highlighted the fact that not all forms of memory are equally affected by damage to the medial temporal lobes. Nondeclarative or implicit memory is largely intact, allowing patients to acquire perceptual and motor skills, conditioned responses, and to demonstrate priming. Declarative or explicit memory, in contrast, is disrupted. While this distinction between nondeclarative and declarative memory in amnesia has been explored in detail over the past five decades, more recent studies have begun to examine possible dissociations within the domain of declarative memory. One issue regards the extent to which new semantic information can be learned following damage to the medial temporal lobe. The present chapter will review evidence suggesting that (a) the medial temporal lobe is not required for normal retrieval of semantic knowledge but is necessary for normal acquisition of factual information and that (b) while semantic learning is not normal following damage to the hippocampal formation, acquisition of some new semantic knowledge can be supported by regions outside of the hippocampus proper (e.g., perirhinal, parahippocampal, or lateral temporal cortices). Another issue concerns whether the cognitive processes contributing to episodic memory performance are uniformly disrupted following damage to the medial temporal lobe. The current chapter will review data suggesting that (a) amnesia disrupts recollection-based mechanisms more so than familiarity-based mechanisms and that (b) when amnesic patients are encouraged to rely on their feelings of familiarity, their performance on recognition tasks can be enhanced.

Key words: semantic memory, episodic memory, amnesia, hippocampus, medial temporal lobe, neuroimaging

Introduction
To function in daily life, we must be able to retrieve facts about the world (semantic knowledge) and to remember the specific spatial and temporal details of prior experiences in our lives (episodic memory). In addition, we must be able to learn new facts and to record new experiences. Insight into the neural processes underlying these essential abilities came in 1953, when the neurosurgeon William Beecher Scoville performed an experimental operation to treat intractable epilepsy. Scoville removed the medial temporal lobes of a man known by the initials H.M. As reported in the landmark paper coauthored with Brenda Milner (Scoville and Milner, 1957), this surgery resulted in a dense anterograde amnesia. Since the time of his operation, H.M. has shown a dramatic deficit in his ability to acquire new, long-term declarative memories (see Corkin, 2002, for review). This impairment in declarative learning extends across all types of information (verbal and nonverbal, semantic and episodic) and exists regardless of the way in which memory is assessed (using recall, recognition, or learning to criterion). Thus, it has been clear since the early studies with H.M. that amnesic patients show decrements in their declarative (episodic and semantic) memory ability, indicating that the medial temporal lobes are critical for the normal acquisition of this information.

**Retrieval of Semantic Knowledge**

A more recent debate has concerned the extent to which medial temporal-lobe structures are required for accessing established semantic memories (e.g., Nadel and Moscovitch, 1997; Squire and Zola, 1998; Tulving and Markowitsch, 1998; Vargha-Khadem et al., 1997). On the one hand, it is plausible that the same medial temporal-lobe structures that are required to learn new facts would also be required to retrieve information that has already been learned. On the other hand, these abilities (the acquisition and retrieval of semantic knowledge) could be supported by distinct processes, with acquisition relying on medial temporal-lobe structures and retrieval depending on structures outside of the medial temporal lobe. One way to adjudicate between these alternatives is to examine the semantic retrieval ability of amnesic patients who have extensive damage to the medial temporal lobe. If these structures are critical for semantic retrieval, than these patients should be impaired on tasks requiring retrieval of world knowledge. Conversely, if semantic retrieval can be supported by regions outside of the medial temporal lobe, than these patients should not show impairments on these tasks.

To distinguish these alternatives, Kensinger, Ullman and Corkin, 2001 examined H.M.’s ability to retrieve lexical knowledge (stored information about words, including their meanings and proper forms; Tulving, 1972; Ullman et al., 1997; Ullman 2001), believed to be one component of semantic memory. H.M. performed a comprehensive series of tasks assessing his lexical memory ability. On a variety of tasks, including picture naming and spelling, H.M. performed within 1 standard deviation of the mean of age- and education- matched control patients. He also performed within the normal range on tasks that required him to generate irregular word forms (e.g., “Every day I dig a hole. Yesterday I _____ a hole.”), an ability believed to rely on the lexical memory system.

Additional evidence that H.M.’s ability to retrieve semantic information was unaffected by his surgery was gleaned from a longitudinal examination of his performance on the Information, Similarities, Comprehension, and Vocabulary subtests of the Wechsler-Bellevue tests and the Wechsler Adult Intelligence Scale (Kensinger, Ullman, and Corkin, 2001). These subtests assess memory for general world knowledge (e.g., from the Comprehension task: “What is the thing to
do if you find an envelope in the street that is sealed, addressed, and has a fresh stamp on it?"; from the Information task: “Whose name is typically associated with the theory of relativity?”) and word meanings (e.g., on the Similarities task, participants are asked what two words, such as eye and ear, have in common; on the Vocabulary task, participants are asked to orally define words). H.M. had been tested on these subtests 20 times between 1953 (preoperatively) and 2000. When his performance across these test sessions was analyzed, there was no main effect of time, suggesting that H.M.’s semantic knowledge had remained consistent over time, and was not negatively affected by his surgery.

These examinations with H.M. suggest that the medial temporal-lobes are not required for normal retrieval of semantic knowledge, but they do not speak to what neural processes do support semantic retrieval. To address this question, researchers have examined the neural processes affected in individuals who show semantic memory deficits. Much of this research has been focused on patients with semantic dementia. Semantic dementia is characterized by a progressive loss of fluent speech and degradation of world knowledge (Hodges et al., 1992; 2000; Lambon Ralph et al., 1998, 2001). Critically, patients with semantic dementia typically have atrophy and dysfunction in the antero-lateral temporal cortex, with relative sparing of the medial temporal lobes (Hodges et al., 1992; Garrard et al., 1997). Moreover, across a series of patients with semantic dementia, the most consistent locus of atrophy appears to be in the left anterolateral temporal lobe (Mummery et al., 2000). Thus, these studies provide convergent evidence that regions of the temporal neocortex likely are critical for normal semantic retrieval.

Further evidence to support this hypothesis has come from patients with focal (non-progressive) lesions of the anterotemporal lobe. Particularly when the lesions are left-lateralized, these patients often show semantic retrieval deficits. For example, Siri, Kensinger, Cappa, Hood, and Corkin (2003) reported a patient with a deficit retrieving knowledge about particular categories of objects (e.g., fruits, vegetables, birds, musical instruments) following damage to the anterior temporal lobe (as a result of herpes encephalitis). This deficit was stable over a number of years, and existed across a variety of tasks. When the patient’s semantic priming performance was assessed, it became apparent that while he showed normal priming for some specific items within these categories, there were other items for which he did not show normal priming (Kensinger, Siri, Cappa, and Corkin, 2003). In other words, for some items, the representation appeared intact, but it could not typically be accessed (those items for which the patient showed priming), whereas other items no longer appeared to have a semantic representation (those items for which the patient did not show priming). Thus, it appeared that the anterior temporal damage caused a destruction of the representations of some items, and a disruption in the ability to access representations of other items.

These data converge on the conclusion that regions of the temporal neocortex support semantic retrieval. Additional evidence to support this hypothesis came from a study of a variety of amnesic patients, some with damage restricted to the hippocampus proper, and others with damage extending to other regions of the medial temporal lobe and to the temporal neocortex (Schmolck, Kensinger, Squire, and Corkin, 2002). This investigation revealed that patients with damage limited to the medial temporal lobe showed no deficits on semantic learning tasks. In contrast, patients with damage to the lateral temporal lobe did show semantic retrieval deficits, and the magnitude of their deficits correlated with the extent of their damage in lateral temporal cortex.

These studies, therefore, provide strong evidence that medial temporal lobe structures are not required for normal retrieval of semantic knowledge. Instead, it seems that the ability to retrieve (or to store) semantic representations relies on the anterior and lateral temporal cortices.
Acquisition of Semantic Knowledge

Although the medial temporal lobes do not appear critical for the retrieval of already-established semantic knowledge, numerous studies have provided evidence that these structures are required for the normal acquisition of new semantic information. Thus, amnesic patients with damage to the medial temporal lobes show profound deficits in acquiring factual information (Milner, 1959), and damage circumscribed to the hippocampus proper seems sufficient to disrupt normal semantic learning (Manns et al., 2003). Moreover, the amount of damage to the medial temporal lobe appears to correspond with the magnitude of the deficit in learning new semantic information (e.g., Verfaellie et al., 2000).

These studies, therefore, highlight the fact that the medial temporal lobes (and, more specifically, the hippocampus proper) are required for normal semantic memory acquisition, and that amnesic patients with damage to those regions show impaired semantic learning.

Nevertheless, it is important to note that, even with extensive medial temporal lobe damage, some semantic learning does appear to occur. For example, patients with dense anterograde amnesia have been able to remember the words used to complete meaningful sentences (Shimamura and Squire, 1988), and to learn new computer-related vocabulary and computer commands (e.g., Glisky and Schacter, 1988; van der Linden and Coyette, 1995). H.M. has also shown the ability to learn new factual information when he can anchor it to premorbidly-acquired knowledge (e.g., learning that the Salk vaccine protects against polio, given that polio was a familiar disease to H.M.; Skotko et al., 2004).

It is plausible that some of this semantic acquisition is supported by nondeclarative memory mechanisms, such as habit learning. For example, Bayley and Squire (2002) found that an amnesic patient, E.P., with extensive medial temporal-lobe damage, as well as damage to temporal neocortex (Stefanacci et al., 2000), was nevertheless capable of acquiring semantic knowledge. However, this knowledge tended to be rigid and inflexible, showing no characteristics of declarative memory.

There have been demonstrations of semantic learning in amnesia, however, in which the semantic learning appears to reflect declarative memory. For example, H.M performs well above chance at selecting famous names among nonfamous foils, even when these individuals became famous following the onset of his amnesia (O’Kane, Kensinger and Corkin, 2004). He also is able to freely recall information about a small number of people who have become well-known after 1953. For example, H.M. indicated that Woody Allen was a “comic, in movie pictures” and that Michael Gorbachev was “famous for making speeches…head of the Russian parliament” (examples from O’Kane et al., 2003). H.M. also is able to draw the floor plan of the house where he lived for many years, despite the fact that he did not move into the house until after his surgery (discussed in Corkin, 2002).

It is important to note that this semantic knowledge differs from that of control participants in a number of ways. Most notably, while control participants typically are better at generating knowledge about individuals who became famous recently as compared to remotely, amnesic patients often show the reverse pattern of performance (O’Kane, Kensinger, and Corkin, 2004; Verfaellie et al., 2000; Westmacott and Moscovitch, 2001). In addition, there seems to be a less consistent access to the semantic representations in patients with extensive medial temporal lobe damage. For example, while H.M. is often able to indicate that Ronald Reagan was President as
well as an actor, that J.F.K. was assassinated, and that Margaret Thatcher was a British politician (Kensinger et al., unpublished observations; cited in O’Kane et al., 2004), he is not always able to generate this information. Moreover, in one demonstration of semantic learning in H.M. (Skotko et al., 2004), the learning was not permanent, but rather had decayed after a relatively short period of time (a few months).

This pattern of results suggests that the semantic learning that proceeds in the absence of a functioning hippocampus requires multiple exposures and extended repetitions, and even with such repetitions it does not reach the same level of detail and stability as is the hallmark of semantic learning in the presence of intact hippocampal function. Thus, it seems likely that while the hippocampus is essential for the one-trial learning that can often underlie the accumulation of rich semantic knowledge. In contrast, regions outside of the hippocampus proper (either posterior parahippocampal gyrus or lateral temporal cortices) may be able to support the gradual accumulation of semantic knowledge over multiple repetitions. Even after these repetitions, the knowledge learned may not be as stable or accessible as information learned via hippocampal mechanisms.

This set of conclusions would be consistent with prior failures to demonstrate semantic learning in H.M. and in other amnesic patients when information is taught over a restricted period of time in a laboratory setting (Gabrieli et al., 1998) or when memory is assessed for infrequently encountered new vocabulary words (Postle and Corkin, 1998). It is plausible that encoding variability, and encoding repetitions, are critical for new semantic learning to occur following extensive medial temporal lobe damage. There may not have been sufficient repetitions, or sufficient encoding variability, to allow for the non-hippocampal mechanisms to build up semantic memories in these other situations.

Clarification of the neural processes supporting this gradual acquisition of semantic knowledge may come from comparisons of the amnesic patient H.M. who, as discussed above, shows evidence of semantic learning with the amnesic patient E.P., who has not shown evidence of such learning. E.P. has more diffuse damage than H.M., in both the caudal parahippocampal gyrus and in the lateral temporal cortex (Stefanacci et al., 2000). Thus, it may be that these regions damaged in E.P., but spared in H.M., are those that allow some gradual learning of declarative, semantic knowledge.

Taken together, the investigations of semantic learning in amnesia suggest a couple of conclusions. First, some new semantic learning can proceed in the absence of a functioning hippocampus. This learning may be supported either by parahippocampal cortex or by the lateral temporal cortex. Second, while these extra-hippocampal mechanisms can support limited semantic knowledge acquisition, they do not appear capable of supporting the rapid and flexible acquisition of semantic knowledge that individuals with intact hippocampi engage.

**Semantic Memory In Amnesia: Conclusions**

In summary, studies of semantic memory in medial temporal-lobe amnesia have confirmed that damage limited to the medial temporal lobes does not disrupt the ability to retrieve already-acquired semantic knowledge. Rather, it appears that damage to the temporal neocortex is required for semantic retrieval deficits to emerge. In contrast, the medial temporal-lobe is necessary for normal semantic learning. Damage to the hippocampal formation impairs semantic learning, and the magnitude of the impairment is correlated with the extent of the damage. However, even with
extensive damage to medial temporal-lobe structures (as with the patient H.M.), some new semantic learning is possible (though the learning is clearly not normal). It may be that the structures of the medial temporal-lobe spared in H.M. (i.e., the caudal portion of the parahippocampal gyrus) are sufficient to support new semantic learning. It also may be the case that lateral temporal regions are capable of the gradual acquisition of declarative semantic knowledge when a sufficient number of encoding episodes occur.

**Episodic Memory in Amnesia**

A pervasive deficit in episodic memory is dramatically exemplified in patients with anterograde amnesia, who are unable to acquire and retrieve any events or episodes from their personal life that have occurred since the onset of their amnesia. Such an impairment is typically revealed on explicit tests of memory, which ask patients to consciously retrieve recent experiences in the form of recall or recognition (Giovanello and Verfaellie, 2001a). Although both recall and recognition tests require conscious retrieval of recent experiences, their processing demands are not identical. Dual-process models of recognition memory postulate two distinct bases of performance: recollection and familiarity (Jacoby and Dallas, 1981; Mandler, 1980). Recollection is thought to be a conscious, attention demanding process in which prior aspects of an experience are retrieved. Familiarity, in contrast, is thought to be an unconscious, automatic process that arises when fluent processing of a stimulus is attributed to prior experience with that stimulus. Whereas recall tasks require conscious recollection of contextually appropriate information, recognition tasks can be supported either by recollection or by familiarity.

Amnesic patients’ impaired performance on tasks of recall and recognition suggests that both recollection and familiarity rely on the integrity of the medial-temporal lobes. Under certain conditions, however, amnesic patients can show surprisingly good recognition memory (Bowers, et al., 1988; Johnson and Kim, 1985). Based on a review of the literature, as well as their own data, Yonelinas and colleagues (1998) concluded that amnesic patients, although impaired in both processes, evidence a disruption in familiarity that is consistently smaller than the disruption in recollection. In contrast to this view, however, others have argued that recollection and familiarity are equally impaired in amnesia (Knowlton and Squire, 1995; Reed and Squire, 1997; Reed and Squire, 1998).

**Evidence for a Disproportionate Deficit in Recollection in Amnesia**

At present it remains controversial as to whether amnesic patients evidence a disproportionate disruption in recollection. One line of research that is relevant to this debate concerns the comparison of amnesic patients’ recall and recognition performance. It has been hypothesized that if recollection and familiarity-based processes are equally impaired in amnesia, then proportionate deficits in recall and recognition should be seen. Alternatively, if familiarity-based recognition is relatively preserved in amnesia, then amnesics’ recall should be more severely impaired than their recognition.

It is not possible, however, to compare directly the severity of impairments across recall and recognition tasks because these tasks use different scales of measurement. To overcome this
problem, studies examining whether amnesic patients show disproportionate deficits in recall typically equate the recognition performance of amnesics and controls, and then assess whether, under these conditions, recall performance is equated as well. Recognition has been equated by delaying the testing for control subjects or by giving the amnesic patients additional study exposure. Perhaps because of the complexities inherent in this approach, the results of such studies have been inconsistent. Some studies have reported that when recognition was equated between amnesic patients and control participants, the patients’ recall remained impaired (Hirst et al., 1986, 1988). Others have found that once recognition memory was matched across groups, recall was similar across groups as well. (Haist, Shimamura, and Squire, 1992; Kopelman and Stanhope, 1998; MacAndrew, Jones, and Mayes, 1994; Shimamura and Squire, 1988).

In a more recent study, Giovanello and Verfaellie (2001b) provided evidence that the relationship between amnesics’ recall and recognition performance depends, at least in part, on the method by which amnesics’ recognition is matched to that of controls. In one experiment, both amnesics and controls were tested after the same delay, but amnesics were provided with additional study exposures. It was hypothesized that even with additional study exposures, the patients’ recognition would be mediated primarily by familiarity, while control participants would able to use both recollection and familiarity as a basis for recognition. Consistent with this hypothesis, amnesic patients recall remained impaired even when recognition was matched to that of controls. In a second experiment, recognition memory was equated by giving the amnesic and control groups equal study exposure, but by testing controls following a 24-hour delay. Because recollection declines more rapidly than does familiarity over a delay (Gardiner and Java, 1991; Tulving, 1985), it was hypothesized that the performance of controls would be mediated mainly by familiarity, as is that of amnesic patients. As hypothesized, recall was equated across groups under these equating conditions. These findings highlight the importance of analyzing the performance of amnesic patients and control participants in term of underlying processes. When such an analysis was conducted, two apparently contradictory patterns of performance could both be explained with reference to the notion that amnesic patients have a disproportionate deficit in recollection.

Another line of research relevant to the debate over whether recollection is disproportionately disrupted in amnesia, concerns the comparison of amnesic patients’ item and associative recognition memory. The study of associative memory in amnesia has been motivated by cognitive studies in young adults that provide empirical evidence for the distinction between item and associative recognition. For example, employing unrelated word pairs in a study with young control participants, Hockley and Consoli (1999) showed that item and associative recognition are differentially associated with familiarity and recollection; associative recognition was accompanied by a greater proportion of "remember" responses (assumed to reflect recollection) than was item recognition, whereas item recognition was accompanied by a greater proportion of "know" responses (assumed to reflect familiarity) than was associative recognition. Based on their findings, Hockely and Consoli (1999) concluded that associative recognition is based on conscious recollection to a greater extent than is item recognition, and conversely, that item memory is based on familiarity to a greater extent than is associative recognition.

These findings suggest that amnesics’ associative recognition performance should differ depending upon whether or not recollection is disproportionately impaired. If amnesia disrupts recollection-based recognition more than familiarity-based recognition, amnesic patients should be disproportionately impaired in associative, relative to item, recognition. Alternatively, if recollection and familiarity-based processes are equally impaired in amnesia, then proportionate deficits in item recognition and associative recognition should be observed.
To determine whether amnesic patients evidence a disproportionate impairment in associative memory, several studies have equated item recognition between amnesic patients and control participants and have then examined whether, under these conditions, associative recognition is matched as well (Giovanello, Verfaellie, and Keane, 2003b; Stark, Bayley, and Squire, 2002; Turriziani, et al., 2004). While item recognition involves discriminating between studied and unstudied stimuli, associative recognition involves distinguishing between studied associations (referred to as “intact pairs”) and novel associations that consist of stimulus components previously seen, though not seen together (referred to as “recombined pairs”). These patient studies have revealed that associative recognition remains impaired in amnesia, even when performance on item recognition is matched (Giovanello, Verfaellie, and Keane, 2003b; Turriziani, et al., 2004) but see (Stark, Bayley and Squire, 2002). Specifically, relative to controls’ performance, amnesic patients’ hit rates (i.e., correct endorsements) for intact pairs were lower, while their false alarm rates (i.e., incorrect endorsements) for recombined pairs were higher. Such a pattern of performance is consistent with a disproportionate deficit in recollection in amnesia.

Taken together, these findings are inconsistent with the view that recollection and familiarity are equally impaired in amnesia (Knowlton and Squire, 1995; Reed and Squire, 1997; Reed and Squire, 1998). Rather, these findings lend support to the notion that amnesia disrupts recollection-based mechanisms to a greater extent than familiarity-based mechanisms (Aggleton and Shaw, 1996; Verfaellie and Treadwell, 1993; Yonelinas et al., 1998).

**Enhancing Familiarity-Based Processing in Amnesia**

If amnesic patients have a relative preservation of familiarity-based recognition, the question arises as to whether conditions can be created that enhance patients reliance on familiarity, thus boosting their recognition performance. Verfaellie, Giovanello, and Keane (2001) explored this possibility by manipulating amnesic patients’ response criterion in a recognition task. Their interest in this manipulation arose from a study that compared the recognition performance of patients with ECT-induced amnesia under a high-criterion condition and a low-criterion condition (Dorfman et al., 1995). In the high-criterion condition, patients were told to endorse a test word only if they were relatively sure that it had appeared on the study list. In the low-criterion condition, patients were encouraged to say yes to a test word if it at all seemed familiar. Patients endorsed more studied, but not unstudied, words in the low- than in the high-criterion condition, indicating a change in discriminability. The patients’ enhanced recognition was attributed to the contribution of familiarity to performance in the low-criterion condition.

Using a similar manipulation in patients with global amnesia, Verfaellie, Giovanello, and Keane (2001) did not find that changes in response criterion affected amnesic patients’ recognition accuracy (see also Reber and Squire, 1999). This direct manipulation on response criterion, however, was generally weak and failed to affect overall performance either in amnesic patients or in control participants. In a second experiment, response criterion was manipulated indirectly by providing information about the alleged base-rate of study items on the recognition test (30% versus 70%). In the 30% base-rate condition, participants were told that 3 out of every 10 words would be from the study list. In the 70% base-rate condition, participants were told that 7 out of every 10 words would be from the study list. The base-rate manipulation was successful in affecting the performance of control participants, as controls endorsed more items (both studied...
and unstudied) in the 70% than in the 30% condition. Most importantly, while this manipulation affected control participants’ response bias, it enhanced amnesic patients’ discriminability. Furthermore, an analysis of patients’ “remember” and “know” responses indicated that that the improved accuracy in amnesia was associated with enhanced familiarity-based recognition. These findings demonstrate that encouraging amnesic patients to rely on familiarity can boost their item recognition performance.

More recently, Giovanello, Keane, and Verfaellie (2003a) examined whether conditions exists in which familiarity can support associative recognition, and if so, whether amnesics’ associative recognition is less impaired under these conditions. In one experiment, using the remember/know paradigm with control participants, they compared the contribution of familiarity to associative recognition for stimuli that were meaningfully integrated pre-experimentally (compound words) with stimuli that were not (unrelated words). It was hypothesized that familiarity may contribute to a greater extent to associative recognition for compound stimuli than to associative recognition of unrelated word pairs. This hypothesis arose from Yonelinas and colleagues’ (1999) finding that, in the nonverbal domain, when stimuli are encoded as a whole, familiarity makes a larger contribution to associative judgments, whereas when stimuli are encoded as a set of features, the contribution of familiarity is greatly reduced. It was demonstrated that familiarity makes a greater contribution to associative recognition of compound stimuli than to associative recognition of unrelated word pairs.

In a further experiment, Giovanello, Keane, and Verfaellie (2003a) examined associative recognition memory in amnesic patients and in control participants for the stimuli employed in their previous experiment. It was hypothesized that if amnesic patients’ deficit in associative memory is due to the fact that associative memory typically depends on recollection, amnesics’ performance should be less impaired under conditions in which associative memory can be supported by familiarity for the association, as is the case for compounds. Alternatively, if the associative memory deficit occurs regardless of the mechanism that supports recognition, than amnesics’ associative recognition performance should be equally impaired for compound stimuli and for unrelated stimuli. The findings were consistent with the former hypothesis: Whereas associative recognition for compounds and unrelated words was nearly identical in control participants, associative recognition was greater for compounds than for unrelated words in amnesic patients. These results demonstrate that amnesics’ associative recognition is enhanced when associative recognition can be supported by familiarity.

Taken together, these findings demonstrate that conditions can be created that enhance patients reliance on familiarity, thus boosting their recognition performance. Furthermore, these results show that familiarity can support item recognition performance, as well as associative recognition performance, in amnesia.

**Episodic Memory in Amnesia: Conclusions**

In summary, recent findings from studies of episodic memory in amnesia have been interpreted in the context of dual process models of recognition memory which postulate two distinct bases of performance: recollection and familiarity. While neither process is normal in amnesia, several findings suggest that the disruption in familiarity is consistently smaller than the disruption in recollection. Consistent with this notion, comparisons of free recall and item recognition, as well as comparisons of item recognition and associative recognition, in amnesia
demonstrate a disproportionate deficit in recollection-based mechanisms. In light of the fact that amnesic patients may have a relative preservation of familiarity-based mechanisms, several studies have examined whether conditions can be created that enhance patients’ reliance on familiarity, thus boosting their recognition performance. The studies show that encouraging patients to rely on their feelings of familiarity can boost their performance.

General Conclusions

While memory deficits are the hallmark of amnesia, the research discussed in this chapter has highlighted the fact that not all aspects of declarative memory are equally affected. This research has opened the door for research into the neural processes that continue to operate in amnesia, allowing patients to gradually acquire at least some fragmentary semantic knowledge, and to gain a sense of familiarity about information they have previously encountered.

References

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Acknowledgements

E.A.K. is supported by NIH grant MH 070199 and K.S.G. is supported by NIH grant AG 023439