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COMMENTARY

Episodic Memory, Semantic Memory, and Amnesia

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ABSTRACT: Episodic memory and semantic memory are two types of declarative memory. There have been two principal views about how this distinction might be reflected in the organization of memory functions in the brain. One view, that episodic memory and semantic memory are both dependent on the integrity of medial temporal lobe and midline diencephalic structures, predicts that amnesic patients with medial temporal lobe/diencephalic damage should be proportionately impaired in both episodic and semantic memory. An alternative view is that the capacity for semantic memory is spared, or partially spared, in amnesia relative to episodic memory ability. This article reviews two kinds of relevant data: 1) case studies where amnesia has occurred early in childhood, before much of an individual's semantic knowledge has been acquired, and 2) experimental studies with amnesic patients of fact and event learning, remembering and knowing, and remote memory. The data provide no compelling support for the view that episodic and semantic memory are affected differently in medial temporal lobe/diencephalic amnesia. However, episodic and semantic memory may be dissociable in those amnesic patients who additionally have severe frontal lobe damage.
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KEY WORDS: human amnesia; declarative memory; fact memory; event memory; hippocampus; perirhinal cortex; entorhinal cortex; frontal lobes

INTRODUCTION

Recently there has been renewed interest in the distinction between episodic memory and semantic memory (Tulving, 1972, 1992). Episodic memory refers to the capacity for recollecting happenings from the past, for remembering events that occurred in particular spatial and temporal contexts. Semantic memory refers to the capacity for recollecting facts and general knowledge about the world. A critical question has been how this distinction might be reflected in the organization of memory functions in the brain.

Episodic memory and semantic memory are two types of declarative memory (Tulving, 1983, 1991; Squire, 1987). One view is that episodic memory and semantic memory are both dependent on the integrity of medial temporal lobe and midline diencephalic structures,[1] and that episodic memory depends additionally on the frontal lobes (Shimamura and Squire, 1987; Squire, 1987; Tulving, 1989; Knowlton and Squire, 1995). According to this view, amnesic patients with medial temporal lobe/diencephalic damage should be deficient in remembering both events and facts (episodic and semantic memory). Amnesic patients who in addition have frontal lobe damage should be especially deficient in episodic memory, because the frontal lobes are involved in an important aspect of episodic remembering, that is, associating the content of an event with its source (when and where the event occurred) in order to construct an autobiographical recollection (Janowsky et al., 1989; Schacter, 1987). Under this view, episodic memory is the gateway to semantic memory. New information is always presented initially as part of some event, but through repetition or rehearsal the new information can be abstracted from its original context and be represented as semantic memory. Thus, when

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[1]Within the diencephalon, the most important structures for declarative memory are in the medial thalamus: the anterior thalamic nucleus, the mediodorsal nucleus, and the connections to and from the medial thalamus that lie within the internal medullary lamina (Zola-Morgan and Squire, 1993). Within the medial temporal lobe, the important structures are the hippocampal formation (the hippocampus proper, the dentate gyrus, the subicular complex, and entorhinal cortex) and adjacent, anatomically related cortex, i.e., perirhinal and parahippocampal cortices (Squire and Zola-Morgan, 1991).

episodic memory is impaired as the result of medial temporal lobe/diencephalic amnesia, semantic memory should be correspondingly impaired. However, when episodic memory is impaired because of frontal lobe dysfunction, then new semantic learning should be possible.

An alternative view is that episodic memory is not critical for the formation of semantic memory (Cermak, 1984; Kinsbourne and Wood, 1975; Parkin, 1982). This view is based in part on the observation that amnesic patients can acquire some semantic knowledge successfully after much repetition (Kovner et al., 1983; Glisky et al., 1986a,b; Shimamura and Squire, 1987; Tulving et al., 1991; Hayman et al., 1993). Drawing on such observations, Tulving (1991) proposed that new information can enter semantic memory through the perceptual systems and independently of the medial temporal lobe/diencephalic brain structures that are damaged in amnesia. This account holds semantic memory to be partially or wholly preserved in amnesia. "The hypothesis that semantic learning ability is preserved in some amnesics implies that these amnesics would perform normally in all semantic learning tasks in which normal subjects could not rely on their intact episodic memory" (Tulving, 1991:24). A more specific version of this idea is that semantic memory is relatively preserved in amnesic patients who have damage limited to the hippocampus (Vargha-Khadem et al., 1997).

The views outlined above are based on two kinds of data. The first are cases where amnesia has occurred early in childhood, before much of an individual's semantic knowledge has been acquired. The question of interest is whether such individuals can acquire semantic knowledge, for example as a result of formal schooling, more successfully than would be expected from their impairment in moment-to-moment episodic memory. The second kind of data comes from experimental studies of amnesic patients where the ability to accomplish fact learning and event learning have been directly compared, studies where retrieval from episodic and semantic memory have been directly compared, and studies of remote memory. This article reviews the available data and considers their implications for the neurologic foundations of episodic and semantic memory.

REPORTS OF AMNESIA OCCURRING EARLY IN CHILDHOOD

There have been three reports of early childhood amnesia. One report concerned an amnesic patient (T.C.) who developed amnesia at the age of 9 years after an episode of herpes simplex encephalitis (Wood et al., 1989). The patient continued to attend school and graduated from high school with her class. Yet her academic progress occurred against a severe memory impairment for moment-to-moment memory which gave the impression of preserved, or partially preserved, semantic memory capacity in the absence of episodic memory. However, a closer analysis of this case reveals that T.C. was capable of some episodic learning and her progress in school was abnormally slow. With respect to episodic memory, on the Rey Auditory Verbal Learning task, she managed to recall eight of 15 words on the fifth trial, and after a delay with a distractor list she was still able to recall three of the words. Thus, her episodic memory was impaired, but it was not altogether absent. With respect to progress in school (semantic memory), two assessments (one at age 14 and another at age 20) indicated that during these 6 years she progressed only two grade levels in reading and spelling ability (from grade six to grade eight). In addition, during the same period, she did not progress at all in mathematics, as measured by the Wide Range Achievement Test.

An early commentary on this case report suggested that T.C.'s ability to learn in school (semantic memory) was not obviously better than would have been expected from her performance on standard tests of anterograde memory ability, i.e., her ability to remember moment-to-moment information (episodic memory) (Ostergaard and Squire, 1990). The real difficulty, of course, is that no formula exists for determining what level of school achievement should in fact be expected, given an impairment in moment-to-moment memory. Accordingly, case reports like this one cannot establish whether semantic memory is spared relative to episodic memory or whether they are affected similarly.

A second report of childhood amnesia concerned a patient (C.C.) who developed amnesia at the age of 10 years as the result of an anoxic episode (Ostergaard, 1987). Formal testing during a 4-year period indicated that C.C. had persistent and severely impaired declarative memory, both episodic and semantic. With respect to episodic memory, for free recall of 10 different 10-word lists, each tested after a 20-s distraction-filled interval, C.C. averaged only 1.1. words correct. Also, after a 45-min delay he could recall nothing of short prose passages and virtually nothing of the Rey-Osterrieth figure. With respect to semantic memory, C.C. did improve in school, but his progress was not normal and as time passed he fell further and further behind his peers. For example, he required 52 months to progress 25 months in reading age. In the laboratory, he was impaired relative to age-matched control subjects on several tests of semantic memory, including tests of verbal fluency, reading, spelling, vocabulary, and semantic classification. On the latter test, which involved classifying the names of living and nonliving things, his performance was poorer for names that are ordinarily learned after the age of 8 than for names that are ordinarily learned earlier. As with T.C., there is no basis for deciding whether the semantic knowledge that C.C. accrued over the years is unusual or simply what would have been expected from estimates of his moment-to-moment (episodic) memory ability.

For these two patients, there was minimal documentation concerning the locus and extent of brain damage. Patient T.C. was assumed to have medial temporal lobe damage, though a computerized tomography (CT) scan taken 2 years after the onset of her amnesia was interpreted as normal (Wood et al., 1989). For patient C.C., a CT scan suggested bilateral medial temporal lobe damage and additional damage in the left occipital lobe, right orbitofrontal cortex, and right neostriatum (Ostergaard, 1987).

The third report described three patients who early in life sustained bilateral injury to the hippocampus (as determined by magnetic resonance imaging) (Vargha-Khadem et al., 1997). The damage was judged to have spared the perirhinal, entorhinal, and parahippocampal cortices. In one of the three cases the damage

occurred at birth (Beth), in another at age 4 years (Jon), and in the third at age 9 years (Kate), "before they had acquired the knowledge base that characterizes semantic memory" (Vargha-Khadem et al., 1997:376). All three patients were reported to have considerable capacity for semantic memory (speech and language competence, literacy, and factual knowledge) despite pronounced amnesia for episodes of everyday life. Accordingly, the authors interpreted their findings as evidence that "early bilateral pathology that is limited largely to the hippocampus produces severe loss of episodic memory but leaves general cognitive development, based mainly on semantic memory functions, relatively intact" (Vargha-Khadem et al., 1997:373). They further suggested that episodic memory ordinarily depends primarily on the integrity of the hippocampal component of the medial temporal lobe memory system, whereas semantic memory ordinarily depends primarily on the adjacent cortical areas of the medial temporal lobe memory system, e.g., the entorhinal and perirhinal cortex. If true, this finding would be important, because of the implication that the hippocampal region is critical for only one aspect of declarative memory.

Evidence for impaired episodic memory in the three patients was documented by their low scores on delayed recall of the stories from the logical memory subtest of the Wechsler Memory Scale (WMS), and on delayed reproduction of the WMS designs. All three patients also obtained close to the lowest possible scores on other standardized tests, including delayed recall of the word list from the Children's Auditory Verbal Learning Test, despite performing within normal limits on immediate memory for the word list. Additionally, questionnaires completed by the patients' parents revealed everyday memory difficulties, including difficulty finding their way in familiar surroundings, difficulty remembering where objects and belongings were placed, impaired orientation for date and time, and difficulty recounting recent telephone conversations, television programs, or the day's activities.

Against this background of impaired episodic memory, all three individuals were reported to have fared well in mainstream education and to have acquired considerable semantic knowledge. For example, their verbal IQ scores (which depend on acquired knowledge) were 82 (Beth), 109 (Jon), and 86 (Kate). On the Wechsler Objective Reading Dimensions (WORD) Test (normal mean scores for the subtests are 100 with a standard deviation of 15; Rust et al., 1993), the three patients obtained competent scores in reading (Beth = 85; Jon = 102; Kate = 102), spelling (Beth = 77; Jon = 84; Kate = 99), and reading comprehension (Beth = 84, Jon = 97; Kate = 88). All of their scores (with the exception of Jon's spelling) were reported as commensurate with their verbal IQ scores. In addition, all three patients scored within the normal range for the population on the Vocabulary, Information, and Comprehension subtests of the Wechsler Intelligence Scale for Children (WISC-III) and the Wechsler Adult Intelligence Scale—Revised (WAIS-R).

Given the fund of knowledge that the children had acquired, Vargha-Khadem et al. (1997) asked how sensory information can enter a semantic memory store in the face of early-onset amnesia and a disabling loss of episodic memory. They suggested that semantic memories were stored partly independently of episodic memory by way of the intact perirhinal and entorhinal cortices. However, it is also possible to propose that each patient had some residual episodic memory and that even a little episodic memory is able, in the fullness of time and after sufficient repetition, to support the acquisition of a good deal of semantic knowledge. If so, then episodic and semantic memory may actually have been affected in these three patients to the same degree.

The neuropsychological data presented by Vargha-Khadem et al. (1997) are not inconsistent with this possibility. Like most amnesic patients, their patients did exhibit some residual moment-to-moment (episodic) memory ability. They scored above zero on most of the recall tests (story recall, WMS design recall, and the Rivermead Behavioural Memory Test). Moreover, they reportedly scored quite well on all but two of the 12 recognition memory tests that were given. Regardless whether recognition memory function is considered to be relatively spared in these patients or impaired in proportion to their recall scores, it is notable that these patients exhibited good recognition performance on memory tests for words, faces, and other material that had been presented only once. Thus, before one interprets the ability of these patients to accrue factual knowledge during their years in school, it is important to keep in mind that the patients have considerable capacity for moment-to-moment memory, which could provide a foundation for the acquisition of factual knowledge.

With respect to acquiring information in school, the level of semantic memory that the patients were able to achieve may not be as high as first appears. First, because one does not know what IQ scores would have been obtained by these patients if they had not sustained early brain damage, it is unclear what it means when the performance of these patients on knowledge tests is sometimes found to be commensurate with their IQ scores. Second, their scores on knowledge tests are not always commensurate with their IQ scores. On the WORD Test, Jon's scores for basic reading, spelling, and reading comprehension are all below what would be predicted from his verbal IQ score, and Beth's spelling score is below what would be predicted by her IQ score. Third, for two of the patients (Beth and Kate), the verbal and performance IQ scores themselves range from one to three standard deviations below the mean scores obtained by the control subjects included in this report.

Thus, just as for T.C. and C.C., it is unclear that Beth, Jon, or Kate have achieved more in school (i.e., acquired more semantic memory) than might be expected from their ability to remember moment-to-moment information (episodic memory). The difficulty is that one simply does not know what can be achieved in school over the years, on the basis of residual episodic memory ability. Accordingly, documentation of progress in school, in the face of impaired episodic memory, cannot on its own provide a critical test of the relationship between episodic and semantic memory.

EXPERIMENTAL STUDIES OF AMNESIC PATIENTS

Another kind of data relevant to the organization of episodic and semantic memory comes from experiments comparing the

performance of amnesic patients and control subjects. One approach has been to assess the ability of amnesic patients to acquire new factual information as well as new information about specific episodes. The question of interest is whether the semantic memory that amnesic patients can acquire is disproportionately better than the event memory that they can acquire. A second approach has been to compare two kinds of retrieval in amnesic patients, remembering and knowing, which are thought to reflect the operation of episodic and semantic memory, respectively. The question of interest is whether amnesia affects remembering more than it affects knowing. A third approach has been to assess remote memory in amnesic patients. The question of interest is whether patients have difficulty remembering factual information from the past. Each of these approaches will now be considered in turn.

Acquiring Factual Knowledge vs. Learning About Specific Events

In a study that compared fact and event learning (Hamann and Squire, 1995), amnesic patients with diencephalic lesions or lesions of the hippocampal formation were taught new factual knowledge (40 three-word sentences such as "MEDICINE cured HICCUP"). Training occurred during four weekly sessions (two training trials/session). For testing, sentence fragments were presented with the instruction to complete each fragment with a word that had been studied (e.g., MEDICINE cured _____). The patients learned at an abnormally slow rate, progressing from 0% correct to 19% correct, as measured 1 week after their fourth training session. Control subjects achieved better than 75% correct performance 1 week after their second session. Event memory was tested in the second session by asking about specific events that had occurred during the first session. The finding was that amnesic patients were impaired on both fact memory and event memory to a similar degree. Indeed, their performance on both the fact and event tests one week after the first training session closely matched the performance of control subjects who had the same training on facts and events but who were tested after a delay of 4 weeks. Thus, unlike the situation where one observes children in school, in formal experiments one can establish whether the level of fact memory ability attained by amnesic patients is or is not what it should have been given their ability to remember specific events. In this study, there was no indication that the capacity for fact learning reflects some spared or partially spared ability, relative to the capacity for event memory.

A different conclusion about fact and event memory in amnesia was reached by Tulving (1991) on the basis of his work with a severely amnesic patient (K.C.). This work introduced a novel learning method for amnesic patients, which was designed to facilitate the acquisition of new semantic knowledge by reducing interference. This method (the study-only procedure) prevents incorrect, potentially interfering responses during learning by testing retention for the first time after several distributed study sessions. The important finding was that across several weeks K.C. was able to acquire considerable semantic knowledge (e.g., the three-word sentences described earlier) using the study-only procedure, despite what was described as a completely dysfunctional episodic memory for specific past events (Tulving et al., 1991). The rate at which K.C. acquired semantic knowledge was far from normal, but what he did learn was striking, given his severe deficit in episodic memory.

Extensive study of K.C.'s fact learning ability led to the proposal that semantic learning is spared, or partially spared, in amnesia (Tulving, 1991). To explain earlier failures to demonstrate good semantic learning in amnesia, Tulving (1991) raised two important points. First, the poor performance of amnesic patients in earlier studies (e.g., patient H.M.; Gabrieli et al., 1988) might be attributable to the conventional learning methods that were used. Second, the performance of amnesic patients on tests of semantic learning may compare poorly to the performance of normal subjects, because normal subjects (but not amnesic patients) are able on such tests to draw on their intact episodic memory.

There are three reasons why the results for patient K.C. cannot be taken as clear evidence for sparing, or partial sparing, of semantic memory in amnesia. First, Hamann and Squire (1995) also used the study-only method to teach amnesic patients three-word sentences. Although the study-only method resulted in better learning than conventional learning methods (32% correct vs. 19% correct after the fourth weekly session), the amount of acquired knowledge was commensurate with the ability of the patients to recollect events that had occurred on the previous day. Second, Hamann and Squire (1995) also tested E.P., a severely amnesic patient with no detectable episodic memory (Squire and Knowlton, 1995). E.P. was given four separate training sessions during a 2-week period using the study-only procedure. He exhibited no learning at all, obtaining a score of zero. Thus, in a patient with no detectable capacity for episodic memory, there was also no detectable capacity for acquiring semantic knowledge.

Third, some questions can be raised about the claim that K.C.'s episodic memory is "completely dysfunctional" (Hayman et al., 1993). On yes-no recognition tests involving 107–116 target items, K.C. failed to endorse a single item as familiar (Tulving et al., 1991). This observation could reflect actual at-chance memory performance or a strong "no" bias. Additional memory tests based on forced-choice recognition could settle the issue by showing whether K.C. can discriminate at all between familiar and novel test items. However, little documentation was provided concerning K.C.'s performance on forced-choice recognition tests. In addition, it was stated: "Most of his [K.C.'s] scores on the WMS-R [Wechsler Memory Scale—Revised] are comparable to the mean scores of amnesic subjects used in experiments in other laboratories" (Tulving et al., 1991:598). Yet, most amnesic study patients do not have completely dysfunctional episodic memory.

An additional difficulty is that the neuropathology in this (closed head injury) patient is complicated. Magnetic resonance imaging studies of K.C. revealed "a predominance of observable abnormal signal in the left hemisphere. . . . Abnormal signal in the right hemisphere is less severe, observable only in a small portion of the medial temporal region and in superior aspects of the medial parietal region" (Tulving et al., 1991:597). Thus, K.C.'s

medial temporal lobe damage, while bilateral, is asymmetric and present in only a small portion of the right medial temporal lobe. Moreover, his brain damage involves other cortical areas in the left hemisphere, including frontal, parietal, retrosplenial, and occipital cortices, and it involves the right parietal cortex.

One way to understand K.C.'s capacity for gradual semantic learning, in the face of his severely impaired episodic memory, is that his episodic memory problems, and especially his apparent inability to have autobiographical recollections, are due especially to his left frontal damage. This scenario is consistent with our finding that patient E.P., who is profoundly amnesic (and has extensive bilateral damage in the medial temporal lobe, but does not have K.C.'s frontal lobe damage), could not accomplish semantic learning at all. It would be interesting to use forced-choice recognition testing to ask whether K.C. could learn about single events (episodic memory), so long as the test did not require that he place himself autobiographically within any past episode. If so, perhaps K.C.'s main difficulty is his inability to personally experience his past through autobiographical remembering. Perhaps he has some capacity to learn about single events, just as he has some capacity to acquire semantic knowledge through repetition. In summary, the findings for patient K.C. are interesting, but it is not clear that his findings illuminate the status of episodic and semantic memory in other amnesic patients or the relative dependence of episodic and semantic memory on the medial temporal lobe or diencephalon.

Remembering and Knowing in Amnesia

Remembering and knowing are thought to reflect the operation of episodic memory and semantic memory, respectively (Tulving, 1989). When a recently presented item evokes a recollection of having specifically encountered that item, an individual is said to "remember." By contrast, when one has simply a sense of familiarity about a previously presented item, without actually recollecting a specific prior encounter with the item, one is said to experience "knowing." In other words, "remember" (R) responses measure the recollection of information about an item that is embedded within or associated with the learning episode, whereas "know" (K) responses measure context-free item familiarity.

With this framework in mind, the pattern of R and K responses provides a method for determining the status of episodic and semantic memory in amnesia. If semantic memory is relatively preserved following damage to medial temporal lobe/diencephalic structures, then the accuracy of K responses in recognition memory tests should be less affected than the accuracy of R responses. In a study that directly tested this idea, 13 amnesic patients were given a yes/no recognition test 10 min after studying 36 words. For each word that was endorsed as a study item, subjects indicated whether they remembered it (R) or whether they simply knew that the word had been presented but had no specific recollection about it (K). Amnesic patients were impaired in the accuracy of both R and K responses, and they performed like control subjects who were tested after a 1-week delay interval. That is, amnesic patients tested 10 min after learning and control subjects tested 1 week after learning exhibited similiar reductions in R and K responses (Knowlton and Squire, 1995). Of the 13 patients in this study, four had bilateral damage to the hippocampal formation as determined by quantitative MRI. These four patients also had similar reductions in R and K accuracy (controls: d' for R = 1.96 ± 0.11; d' for K = 0.93 ± 0.13; four amnesic patients: d' for R = 0.33 ± 0.25; d' for K = 0.30 ± 0.22; controls tested after a 1-week delay: d' for R = 0.64 ± 0.21; d' for K = 0.31 ± 0.25).

A recent reanalysis of the available R and K data from three studies of amnesic patients reached a similar conclusion that both R and K accuracy are impaired in amnesia (Kroll and Yonelinas, 1997). These findings provide strong evidence that the components of memory that support R and K responses (episodic and semantic memory) both depend on the integrity of the medial temporal lobe/diencephalic brain structures damaged in amnesia. Most important, the data for the four patients studied by Knowlton and Squire (1995) suggest that R and K accuracy depends similarly on the hippocampal formation. There is no indication in the data that hippocampal formation lesions spare, or partially spare, K responses (semantic memory) relative to R responses (episodic memory).

Studies of Remote Memory

Performance on remote memory tests provides another test of the idea that episodic and semantic memory can be differentially affected in amnesia. As mentioned earlier, it has been suggested that the reported advantage of normal subjects over amnesic patients in tests of semantic memory is due to the fact that normal subjects can perform these tests by drawing upon episodic memory (Tulving, 1991). Arguing against this point of view, however, is the finding that amnesic patients, including patients with histologically confirmed lesions limited to the hippocampal formation (patients L.M. and W.H., Rempel-Clower et al 1996), can be impaired on factual questions about news events that occurred more than a decade before the onset of their amnesia (Squire et al., 1989; Beatty et al., 1987; Salmon et al., 1988). It is unclear how normal subjects could gain advantage over amnesic subjects by using episodic memory in this kind of test. The point is that amnesic patients have difficulty retrieving factual information even when the contribution of episodic retrieval is quite unlikely (for additional evidence, see Verfaellie et al., 1995; Schmidtke and Vollmer, 1997).

Remote memory performance is also relevant to episodic and semantic memory in the case of patients who have been amnesic for many years. Vargha-Khadem et al. (1997) suggest that semantic learning can proceed rather well despite damage to the hippocampus. Reed and Squire (1998) studied two patients, A.B. and L.J., who have been amnesic for 22 and 9 years, respectively. MRI findings for L.J. suggest that damage is limited to the hippocampal region (see next section for limitations of MRI). A.B.'s lesion is presumed to be hippocampal on the basis of etiology (anoxia and cardiac arrest). Compared to control subjects who were asked about the same past time periods as the patients, these two patients were found to have acquired an abnormally small amount of new factual knowledge about vocabulary, famous

people, and news events during the years since they became amnesic (Reed and Squire, 1998). The finding that A.B. and L.J. possess deficient fact knowledge about the years since the onset of their amnesia shows that factual knowledge does not inevitably accrue to normal levels in the face of hippocampal pathology.

CONCLUSION

The proposal that episodic and semantic memory are affected differently in amnesia (Tulving, 1991) or, more specifically, that they are affected differently by hippocampal damage (Vargha-Khadem et al., 1997), is an interesting idea and in the spirit of current efforts to find specificity within the medial temporal lobe memory system. There appear to be two reasons why it has been difficult to arrange a decisive test of this proposal. First, rather stringent neuropsychological evidence is required to support such a claim. Either double dissociations between episodic and semantic memory are needed, or compelling evidence is required that episodic memory is disproportionately affected relative to semantic memory.

Second, with human material one seldom has available the requisite neuropathological detail. MRI data are essential, but even high-resolution MRI cannot detect cell loss that is easily detected in histological examination. For example, MRI indicated clearly that amnesic patients L.M. and W.H. had damage to the hippocampal region (Squire et al., 1990). Subsequent neurohistological analysis confirmed this finding but also provided additional information: W.H., but not L.M., had damage to the subicular complex, and both patients had cell loss in the entorhinal cortex (Rempel-Clower et al., 1996). Accordingly, it is doubtful that the neuroimaging techniques currently available can reliably identify patients who have damage limited to the hippocampus and no damage to adjacent structures such as entorhinal cortex. Yet this level of resolution is required to evaluate the hypothesis advanced by Vargha-Khadem et al. (1997).

With respect to the two specific proposals under consideration—that semantic and episodic memory are differentially affected following either medial temporal lobe lesions (Tulving, 1991) or more restricted hippocampal lesions (Vargha-Khadem et al., 1997)—there is at this time no compelling support for these proposals and some evidence against them. 1) Reported cases of childhood amnesia have not yet provided the kind of rigorous comparison between episodic and semantic memory that is needed to test the hypothesis. The cases described to date are inconclusive because there is no basis for judging whether the amount of semantic knowledge eventually acquired by amnesic patients is unusual or simply what would be expected after repeated effort over many years. 2) Experiments comparing the ability of amnesic patients to acquire episodic and semantic memory suggest that both kinds of memory are impaired to the same degree. 3) Experiments comparing the ability to retrieve from episodic and semantic memory suggest that episodic and semantic retrieval are impaired similarly. 4) Amnesic patients can have difficulty remembering factual knowledge that occurred more than a decade prior to the onset of their amnesia, direct evidence that semantic memory is impaired. Also, amnesic patients do not inevitably acquire factual knowledge to the degree that normal individuals do, during the years after they become amnesic.

Thus, the data suggest that episodic and semantic memory depend similarly on the medial temporal lobe/diencephalic structures damaged in amnesia. Although episodic and semantic memory do not appear to be dissociable in medial temporal lobe/diencephalic amnesia, the distinction remains useful for understanding the contribution of the frontal lobes to episodic memory. Indeed, the findings from patient K.C. might be viewed in this light—as support for the idea that episodic and semantic memory are dissociable in amnesic patients with severe frontal lobe damage.

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