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THE STRUCTURE AND ORGANIZATION OF MEMORY

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INTRODUCTION

A major goal of psychology is to understand the underlying organization of cognition—that is, to develop formal accounts of cognitive processes, information flow, and representations. Ultimately, one wants to understand cognition not just as an abstraction, or in terms that are simply plausible or internally consistent. Rather, one wants to know as specifically and concretely as possible how the job is actually done. It is often said, working from logical considerations alone, that in describing the function of a complex device one can separate consideration of its formal operations (the software) from consideration of the mechanisms used to implement the operations (the hardware). In the history of cognitive psychology it has been traditional to separate psychological theory from neurobiological detail. Until recently, this approach could be justified by the fact that relevant neurobiological information was simply not available. Yet it is increasingly true that the domains of psychology and neuroscience are reinforcing each other and working hand in hand (Kandel & Squire 1992). Neuroscience has become relevant and useful for elucidating the structure and organization of cognition.

Here we consider recent work on learning and memory from a combined psychology-neuroscience point of view. We focus on the characteristics of various forms of memory, their relationship to each other, and how they are organized in the brain. Although work with normal human subjects has been vital to this line of inquiry, our discussion draws especially on neuropsychological studies of memory-impaired patients and related studies with experimental animals. For recent reviews that emphasize work with normal subjects, see Hintzman (1990), Richardson-Klavehn & Bjork (1988), Schacter et al (1993), and Tulving (1991).

SHORT-TERM MEMORY

One of the oldest and most widely accepted ideas about memory is that short-term memory (STM) can be usefully distinguished from long-term memory (LTM) (James 1890; Waugh & Norman 1965; Glanzer & Cunitz 1966; Atkinson & Shiffrin 1968). That this distinction is prominently reflected in the organization of brain systems is demonstrated by the fact that amnesic patients have intact STM despite severely impaired LTM (Baddeley & Warrington 1970; Drachman & Arbit 1966; Milner 1971). A recent study of amnesic patients has made this proposition even more secure (Cave & Squire 1992a). Verbal STM was assessed with seven separate administrations of the standard digit span test in order to obtain a more precise measure of STM than has previously been available. In addition, nonverbal STM was assessed with four tests, including a test of STM for spatial information. Amnesic patients with hippocampal formation damage had the same average digit span as normal subjects (6.8 digits) and performed entirely normally on the other tests. Thus,

STM is independent of LTM and independent of the structures and connections damaged in amnesia.

An important development has been the separation of STM and LTM in experimental animals, which raises the possibility of investigating the biological basis of STM. In one compelling study (Wright et al 1985), the same recognition memory test consisting of four sequentially presented colored slides was given to pigeons, monkeys, and humans. (The humans viewed patterns from a kaleidoscope, and the pigeons and monkeys viewed pictures.) After a variable delay interval, a probe item was presented that on half the trials matched one of the four list items. Subjects made one response if the probe matched a list item, another if it did not. All three species exhibited primacy and recency effects as indicated by U-shaped serial position functions in which the first and fourth items in the list were remembered better than the second and third items. (The primacy effect refers to the superiority of the first item, and the recency effect refers to the superiority of the last item.) Pigeons exhibited a U-shaped serial position function at delay intervals of 1 and 2 sec, monkeys at delays of 1–10 sec, and humans at delays of 10–60 sec. At shorter delays than these, the primacy effect was absent. At longer delays, the recency effect was absent. The results for all three species require two distinct memory processes—e.g. a transient STM to account for the short-lived recency effect and, to account for the primacy effect, a longer-lasting LTM that emerges as retroactive interference decays during the delay interval.

A strong parallel between humans and experimental animals is also indicated by the finding that hippocampal lesions in rats eliminate the primacy portion of the serial position curve but not the recency portion (Kesner & Novak 1982), just as occurs in amnesic patients (Baddeley & Warrington 1970; Milner 1978). Finally, monkeys with large medial temporal lobe lesions were entirely normal at relearning postoperatively the trial-unique delayed nonmatching to sample task (Mishkin & Delacour 1975) with a delay interval of 1 sec between the sample and the choice (Overman 1990). In contrast, a severe impairment in performance was observed at longer delays. This finding is noteworthy because the usefulness of the delayed nonmatching to sample task for studying memory in monkeys was questioned recently, precisely because this task has not always distinguished STM and LTM (Ringo 1991). This issue was subsequently considered more fully (Alvarez-Royo et al, in press). Monkeys with medial temporal lobe lesions exhibited impaired memory at long retention delays and normal memory at short retention delays, when the delay intervals were presented in mixed order. Moreover, the normal monkeys and the monkeys with lesions exhibited a statistically significant group X retention delay interaction, which could be demonstrated whether the data were analyzed using a percentage correct measure, a d′ (discriminability) measure, or an arcsine transform. Thus, the work with monkeys is fully consistent with the facts of human amnesia and provides an additional illustration of the separation between STM and LTM.

The traditional view of the distinction between STM and LTM has been that the systems operate serially (Atkinson & Shiffrin 1968; Glanzer & Cunitz 1966; Waugh & Norman 1965). Information initially enters STM and subsequently becomes incorporated into a more stable LTM. This view was challenged some years ago (Shallice & Warrington 1970; Warrington & Shallice 1969) based on findings from a carefully studied patient (K.F.). Following a left parietal injury from a motorcycle accident, K.F. had a severely deficient verbal STM, as reflected by a digit span of one item, but nevertheless exhibited normal verbal LTM as measured, for example, by paired-associate learning of words and word-list learning. This pattern of findings led to the proposal that information may not need to enter STM before reaching LTM because the inputs to these two systems are arranged in parallel (Shallice & Warrington 1970; Weiskrantz 1990).

As the result of newer work, the findings from patient K.F. can now be understood fully without postulating parallel STM and LTM stores. STM has come to be viewed as a diverse collection of temporary capacities that are distributed across multiple, separate processing modules (Baddeley & Hitch 1974; Goldman-Rakic 1987; Monsell 1984; Squire 1987). In this view, auditory-verbal STM is a temporary storage system only for phonologically coded information. If one supposes that STM and LTM are serially organized, then one would expect a deficit in auditory-verbal STM to result in a corresponding deficit in LTM, but only to the extent that tests of LTM also depend critically on phonological analysis of verbal material. Findings from recent studies support this perspective.

Baddeley and his colleagues studied a patient (P.V.) who had suffered a cerebrovascular accident involving the left perisylvian region (Baddeley et al 1988). The patient appeared to have a deficit in STM similar to that of patient K.F. Thus, her auditory digit span was two items, but prose recall and free recall of word lists were intact. Yet, when tests of LTM were specially devised that required P.V. to depend on phonological analysis at the time of learning (e.g. visual or auditory presentation of foreign-language word pairs that would be difficult to learn by forming semantic associations), performance was distinctly impaired.

Related evidence on this point came from studies of 4–6-year-old normal children who were selected according to their repetition ability for single nonwords. Children who had low repetition scores for nonwords also had difficulty in a LTM task involving the learning and retention of arbitrary, unfamiliar names for toys (Gathercole & Baddeley 1990). Finally, articulatory suppression (whereby subvocal rehearsal is discouraged by requiring subjects to perform an interfering task) impaired the long-term learning of Russian vocabulary in normal adult subjects but not the learning of native-language paired associates (Papagno et al 1991). These findings all suggest that a deficit in short-term phonological memory leads to a deficit in LTM when the long-term learning also depends on phonological information.

Accordingly, the findings from patients K.F., P.V., and other similar patients with impaired verbal STM can be understood as a selective deficit in one component of STM, and a correspondingly selective deficit in LTM for information that is ordinarily processed by the defective STM component. Such a deficit leaves other components of STM available for the establishment of LTM. This perspective thus holds to the traditional view that STM grades into LTM and is essential for its formation.

LONG-TERM MEMORY

Declarative Memory

One important insight to emerge recently is that LTM is not a single entity but is composed of several different components, which are mediated by separate brain systems. Precursors to this idea can be found in many earlier writings (for reviews, see Schacter 1987; Squire 1987), but it became the subject of wide interest beginning only in the early 1980s as the result of experimental findings with normal adult subjects, amnesic patients, and experimental animals (see, e.g., Cohen & Squire 1980; Graf et al 1984; Jacoby & Witherspoon 1982; Malamut et al 1984; Tulving et al 1982; Warrington & Weiskrantz 1982; for two important earlier proposals, see Hirsh 1974; O'Keefe & Nadel 1978). The major distinction is between conscious memory for facts and events and various forms of nonconscious memory, including skill and habit learning, simple classical conditioning, the phenomenon of priming, and other instances where memory is expressed through performance rather than by recollection (see the section below on Nondeclarative Memory).

Studies of amnesic patients have provided particularly strong evidence for this distinction. These patients fail conventional memory tasks that involve, for example, recall or recognition but nevertheless perform entirely normally on a wide variety of other tasks. Although various terms have been used to describe these kinds of memory, the terms have remarkably similar meanings. Declarative memory (explicit memory, relational memory) is a brain-systems construct, referring to memory that is dependent on the integrity of the hippocampus and anatomically related structures in the medial temporal lobe and diencephalon (Squire & Zola-Morgan 1991; Zola-Morgan & Squire 1993). Nondeclarative (implicit) memory is a heterogeneous collection of separate abilities that can be additionally dissociated from each other (Butters et al 1990; Heindel et al 1989, 1991). These memory abilities depend on brain systems outside of the medial temporal lobe and diencephalon.

A number of important questions have been raised about whether distinctions between kinds of memory can be defined and established outside of the experimental contexts in which they were first developed. For example, the distinction between declarative and nondeclarative (or explicit and implicit) emphasizes the notion of conscious recollection, which is not useful when

considering learning and memory in nonhuman animals. It has therefore been important to ask whether terms like declarative and nondeclarative have meaning independent of the concept of conscious recollection and independent of an empirically determined list of what amnesic patients can and cannot do. Recent work has helped to free these terms from such potential circularity by showing that different kinds of memory have different characteristics (for additional discussion see Sherry & Schacter 1987; Squire 1992a).

Declarative memory is fast, it is not always reliable (i.e. forgetting and retrieval failure can occur), and it is flexible in the sense that it is accessible to multiple response systems. Nondeclarative memory is slow (priming is an exception), reliable, and inflexible—that is, the information is not readily expressed by response systems that were not involved in the original learning. Two important experiments have illustrated that declarative and nondeclarative memory differ in flexibility. In the first experiment (Eichenbaum et al 1989), rats with damage to the hippocampal system were trained concurrently on two separate odor discrimination tasks (A+B−, C+D−) that they could eventually perform about as well as normal rats. Thus, both normal rats and rats with lesions came to choose odor A when it was presented in the odor pair AB and odor C when it was presented in the odor pair CD. However, a transfer task showed that something different had been learned by the two groups. Specifically, when rats were presented with recombined odor pairs (AD or CB), the normal rats tended to choose odor A, performing about as well as on the regular learning trials. That is, they were not disrupted by the new combination of stimuli; they were able to use relational information about the odors in a flexible way. In contrast, the rats with lesions behaved as if they were confronting a new problem and performed near chance. In their case, it appeared that the kind of knowledge that had been acquired was inaccessible when the original learning event was not precisely repeated.

A similar result was obtained with monkeys who had lesions of the hippocampus or related structures (Saunders & Weiskrantz 1989). Monkeys first learned which pairs of four objects were rewarded (e.g. AB+ and CD+) and which were not (e.g. AC− and BD−). Specifically, normal monkeys and monkeys with lesions were given a series of two-choice discrimination tasks in which a positive object pair was always presented together with a negative pair (e.g. AB+ and AC−). In this way, monkeys were required to respond on the basis of both objects in each pair (e.g. object A was correct when it appeared with object B but not when it appeared with object C). In a subsequent transfer task, monkeys were tested to determine what they had learned about the associations. One element of a previously rewarded pair was first presented (e.g. object A), and monkeys were immediately given a choice between two other objects (in this example, objects B and C). The normal monkeys selected object B, which had been part of the two-element, rewarded pair (AB), on 70% of the trials; but the monkeys with lesions performed at chance. Thus, what the

operated monkeys had learned about the object-reward associations was bound to the original learning situation.

This issue has been addressed to some extent in studies of human learning and memory. In one study, amnesic patients who had gradually (and abnormally slowly) learned computer programming commands had difficulty applying their knowledge to new situations and difficulty answering open-ended questions about what they had learned (Glisky et al 1986a). In another study, in which much less training was given, amnesic patients acquired a limited ability to complete sentences in response to cue words (cued recall) (Shimamura & Squire 1988). In this case, it was shown that the knowledge acquired by the patients was as flexible, as accessible to indirect cues, and as available to awareness as the knowledge acquired by normal subjects. A likely possibility is that these patients relied on residual declarative memory to learn the sentences, while the patients who learned computer commands acquired the information as procedural memory for programming skills. Hyperspecificity appears to be a property of nondeclarative memory (also see Tulving & Schacter 1990), not a property of whatever information amnesic patients are able to acquire. When tasks are amenable to declarative memory strategies, amnesic patients will attempt to learn with their impaired declarative memory system, and whatever they succeed in remembering will be flexible and accessible to awareness.

Episodic and Semantic Memory

Episodic and semantic memory are two types of declarative memory (Tulving 1983, 1991). Episodic memory refers to autobiographical memory for events that occupy a particular spatial and temporal context, and semantic memory refers to general knowledge about the world. Both types of memory are declarative, in the sense that retrieval of information is carried out explicitly and subjects are aware that stored information is being accessed. While it is agreed that episodic memory is severely impaired in amnesia and dependent on the integrity of the brain system damaged in amnesia, the relationship between semantic memory and this brain system has not been so clear. Amnesic patients do have great difficulty acquiring semantic knowledge (Glisky et al 1986a,b; Kovner et al 1983), but they can typically succeed to some extent after much repetition. In one report (Tulving et al 1991), a severely amnesic patient (K.C.) eventually learned to complete arbitrary three-word sentences during a large number of training trials distributed over many months. This occurred despite the apparent absence of any memory at all for specific episodes.

An issue that remains to be addressed is whether episodic memory can truly be absent altogether, in the presence of gradually successful semantic learning, or whether semantic learning succeeds by building on residual episodic memory. Even a small amount of residual episodic memory might, in the fullness of time and after sufficient repetition, develop into serviceable semantic knowl-

edge. When memory is impaired, the ability to acquire new semantic knowledge through repetition will always exceed the ability to acquire episodic memory, because episodic memory is by definition unique to time and place and cannot be repeated (see Ostergaard & Squire 1990).

One proposal is that episodic and semantic memory are dissociated in amnesia (Cermak 1984; Kinsbourne & Wood 1975; Parkin 1982). For example, it has been proposed that amnesia selectively affects episodic memory, that semantic learning is fully intact in amnesia, and that the advantage of normal subjects over amnesic patients in tests of semantic learning is due to the fact that normal subjects can perform these tests by drawing on episodic memory (Tulving 1991). By this view, repeated exposure to factual material can lead gradually and directly to long-term memory storage without requiring the participation of the brain system damaged in amnesia. A problem with this view is that amnesic patients have difficulty with factual information even when the contribution of episodic retrieval is unlikely. Thus, they fail remote memory questions about past public events that occurred more than a decade before the onset of amnesia (Squire et al 1989). This deficit would appear to reflect a failure of semantic memory because it is unlikely that normal subjects gain their advantage over amnesic patients on such remote memory tests by using episodic memory to answer the questions. Can episodic memory materially contribute to one's ability to identify Sara Jane Moore (the woman who attempted the assassination of President Ford) or to recall what dance the Peppermint Lounge was famous for (the Twist)?

A second difficulty turns on the question of how memory systems in humans relate to memory systems in nonhuman animals. If semantic memory is independent of the brain system damaged in amnesia, then experimental animals should be affected by damage to this brain system only to the extent that they use episodic memory to perform tasks. The difficulty is that rats, monkeys, and other animals are severely impaired on a wide variety of memory tasks following damage to the hippocampus and related structures (for reviews, see Sutherland & Rudy 1989; Squire 1992a), and the tasks that are affected involve much more than is usually intended by the term episodic memory (e.g. maze tasks and object recognition tasks). Indeed, episodic memory is usually considered to be either unavailable to nonhuman animals altogether or analogous to particular forms of trial-dependent memory (Olton 1985; Tulving 1985). Thus, one must suppose either that animals use episodic memory extensively, or that in animals some kinds of memory other than episodic memory depend on the hippocampus and related structures.

If the distinction between episodic and semantic memory is not relevant to the function of the brain system damaged in amnesia, the distinction is no less interesting or important. One possibility is that both episodic and semantic memory depend on the brain system damaged in amnesia (i.e. the hippocampus and related structures) and that episodic memory additionally depends on the integrity of the frontal lobes. Patients with frontal lobe damage, who are

not amnesic, exhibit a phenomenon termed source amnesia (Janowsky et al 1989b). Source amnesia refers to loss of information about when and where a remembered item was acquired (Evans & Thorn 1966; Schacter et al 1984; Shimamura & Squire 1987). Thus, source amnesia amounts to a loss of autobiographical involvement with recollected material—i.e. a disturbance of episodic memory. It is important to note that amnesic patients who commit source errors can subsequently demonstrate by multiple-choice testing that they have as much knowledge about the learning event as amnesic patients who do not commit source errors (Shimamura & Squire 1991). Thus, source amnesia appears to reflect a loss of episodic memory, related to frontal lobe dysfunction, which reflects a disconnection between facts and their contexts.

If episodic memory were understood in this way, a number of points would be clarified. First, the biological validity of the distinction between episodic memory and semantic memory is based on the greater contribution that frontal lobe function makes to episodic memory, compared to semantic memory. Second, episodic memory is available to nonhuman animals in a limited way, in the sense that animals do not acquire or express information about past events in the same way that people do—i.e. as recollections of past personal happenings. According to this view, the difference between human episodic memory and that of animals is attributable to the greater size and complexity of the human frontal lobe.

Third, episodic memory can be virtually absent in some severely amnesic patients who can still accomplish some semantic learning (e.g. patient K. C.). Such a condition depends in part on source amnesia, pursuant to frontal lobe pathology, which is superimposed on a severe difficulty in acquiring information about both facts and events. This view suggests two possibilities: 1. patients more amnesic than K. C., but without frontal damage, might be unable to accomplish semantic learning as well as patient K. C.; 2. severely amnesic patients might be able to accomplish some semantic learning as well as a corresponding degree of learning about single past events, albeit at an impaired level, provided they were tested with a method that does not require source memory and is not sensitive to frontal lobe pathology. For example, patients could be tested with multiple-choice methods that asked about what occurred in a specific event without requiring that the patients be able to place themselves autobiographically within the episode.

THE BRAIN SYSTEM SUPPORTING DECLARATIVE MEMORY

During the past decade, an animal model of human amnesia was established in the monkey (Mishkin 1982; Squire & Zola-Morgan 1983; Mahut & Moss 1984). Cumulative work with monkeys based on this animal model, together with findings from rats and new information from memory-impaired patients, has identified in broad outline the structures and connections important for

declarative memory. Damage within the medial temporal lobe or the medial thalamus is sufficient to cause severe memory impairment. Within the medial temporal lobe, the important structures are the hippocampus and adjacent, anatomically related cortex (i.e. entorhinal, perirhinal, and parahippocampal cortices) (Squire & Zola-Morgan 1991). Within the diencephalon, the most important structures are in the medial thalamus: the anterior thalamic nucleus, the mediodorsal nucleus, and connections to and from the medial thalamus that lie within the internal medullary lamina. The medial thalamus receives well-described projections from several anatomical components of the medial temporal lobe. It is not clear whether or not the mammillary nuclei (MN) make an important separate contribution to memory functions, although damage to MN has sometimes been reported to produce a small degree of memory impairment (for reviews, see Markowitsch 1988; Victor et al 1989; Zola-Morgan & Squire in press).

Both the medial temporal lobe and the medial thalamus project to the frontal lobe, thereby providing a route by which recollections can be translated into action. Damage to the frontal lobe does not itself cause amnesia (Janowsky et al 1989); but frontal lobe pathology markedly affects cognition (Levin et al 1991), and it substantially alters the nature of the memory impairment when it occurs together with damage to the medial temporal lobe or medial thalamus (Shimamura et al 1991).

Transient amnesic conditions leave patients permanently unable to remember the events that occurred while they were amnesic (Kritchevsky et al 1988). This shows that the medial temporal/diencephalic system is required at the time of learning if an enduring and retrievable long-term (declarative) memory is to be established. How long after learning this brain system remains essential can in principle be determined by examining the phenomenon of retrograde amnesia. In particular, one wants to know which periods are lost from the period before amnesia began. In practice, it has been difficult to settle this matter with memory-impaired patients. First, the moment when amnesia begins is often difficult to establish. Second, there is usually considerable uncertainty about the precise locus and extent of damage in the particular patients being studied. Third, studies of remote memory in patients necessarily rely on retrospective methods and imperfect tests. Despite these difficulties, something useful has been learned about retrograde amnesia through quantitative studies of memory-impaired patients. More recently, the matter has been clarified by prospective studies of retrograde amnesia in mice, rats, and monkeys.

TIME-LIMITED FUNCTION OF THE BRAIN SYSTEM SUPPORTING DECLARATIVE MEMORY

The characteristics of retrograde amnesia vary enormously across different patients and patient groups. For one patient (R.B.), in whom the damage was restricted to the CA1 region of the hippocampus, retrograde amnesia was

limited to perhaps one or two years prior to the onset of amnesia (Zola-Morgan et al 1986). Other patients exhibit temporally graded memory loss covering one to two decades (Squire et al 1989). Still other patients, usually ones with severe impairment, exhibit retrograde amnesia that appears extensive and ungraded, covering most of adult life (for reviews, see Butters & Stuss 1989; Squire 1992a). One possibility is that the extent of retrograde memory loss is related simply to the severity of amnesia and to the extent of damage within the medial temporal/diencephalic system. By this view, extended, ungraded retrograde memory loss represents an extreme condition on a continuum of severity. This alternative seems unlikely, because the severity of anterograde amnesia and the severity of remote memory impairment are not always correlated (Barr et al 1990; Shimamura & Squire 1986; Kopelman 1989) and because the severely amnesic patient H.M. is capable of recalling well-formed episodic memories from his early life (Sagar et al 1985). Many of the patients who have been reported to have extended, ungraded remote memory impairment have damage outside of the medial temporal lobe and medial thalamus. Thus, another possibility is that temporally graded retrograde amnesia occurs when damage is limited to the medial temporal lobe or medial thalamus and that extended, ungraded loss occurs only when there is damage outside this system.

Retrograde amnesia exhibits quite similar characteristics in medial temporal lobe amnesia and diencephalic amnesia (Squire et al 1989). The amnesia reflects a loss of usable knowledge, not a loss of accessibility that can be compensated for by providing repeated retrieval opportunities. Moreover, there are no compelling demonstrations that retrograde amnesia can be remediated by simple changes in the test procedures [e.g. asking patients to complete a famous name from a few letters instead of matching the name to a photograph (see Squire et al 1990)].

Retrograde amnesia can in one sense be described as a retrieval deficit. This description fits the observation that most lost memories return following transient amnesia (Benson & Geschwind 1967; Squire et al 1975). Yet, in another sense this description does not capture the nature of the impairment. First, memories acquired just prior to the amnesic episode cannot be recovered. Second, it is not clear that lost memories would return so fully if the system remained dysfunctional for a long time. Third, recent treatments of the medial temporal/diencephalic brain system favor a role for the system in establishing long-term memory that does not fit easily either a storage or a retrieval interpretation (Eichenbaum et al 1992; Halgren 1984; McNaughton & Nadel 1990; Milner 1989; Rolls 1990; Teyler & Discenna 1986; Squire 1992a). For example, the system has been proposed as the storage site for a summary sketch, a conjunction, or an index; and it has been proposed that one critical event is the induction of long-term potentiation (LTP) in the hippocampus at the time of learning.

Prospective studies with experimental animals have addressed long-standing questions concerning the precise shape of retrograde amnesia gradients (see Squire et al 1984). In one study, monkeys learned 100 object pairs prior to removal of the hippocampal formation (Zola-Morgan & Squire 1990). Twenty object pairs were learned at each of 5 preoperative periods (16, 12, 8, 4, and 2 weeks). After surgery, memory was tested by presenting all 100 objects in a mixed order for a single trial. Normal monkeys remembered objects learned recently better than objects learned 12–16 weeks earlier. Operated monkeys exhibited the opposite pattern, remembering objects learned remote from surgery significantly better than objects learned recently. Moreover, memory for remotely learned object pairs was entirely normal. Similar temporal gradients of retrograde amnesia have recently been demonstrated for rats acquiring a context-dependent fear response at different times prior to hippocampal damage (Kim & Fanselow 1992), for rats acquiring a food preference prior to hippocampal or diencephalic damage (Winocur 1990), and for mice acquiring maze habits at different times prior to damage of entorhinal cortex (Cho et al 1991).

These results show that the medial temporal/diencephalic memory system is not a repository of long-term memory. Indeed, in each of the animal experiments it was possible to identify a time after learning when damage to this system had no effect on memory for what had been learned. Thus, information that initially depends on the medial temporal/diencephalic system can eventually become independent of it. Initially, this system participates in the storage and retrieval of declarative memory. As time passes after learning, a process of consolidation and reorganization occurs such that a more permanent memory is established that is independent of the system. Permanent storage is likely to occur in neocortex where information is first processed and held in short-term memory.

A more specific version of this idea states that the medial temporal/diencephalic memory system initially binds together the distributed sites in neocortex that together represent the memory of a whole event (Zola-Morgan & Squire 1990). This low-capacity, fast system permits the acquisition and storage of representations involving arbitrarily different elements, and for a period it provides a basis for retrieving the full representation, even when a partial cue is presented. As time passes, the burden of long-term memory storage is assumed fully by neocortex. The time course of consolidation will vary depending on the species, the strength of initial learning, and the rate of forgetting. The changes can be expected to continue during a significant portion of the lifetime of a memory.

THE DEVELOPMENT OF DECLARATIVE MEMORY

When the notion of multiple memory systems was first developed, it provided a new way to think about the phenomenon of infantile amnesia—i.e. the

relative unavailability of memories for events that occurred before the third year of life. The traditional view, as influenced by psychoanalytic theory (Freud 1962), has been that memories are acquired in infancy but are later repressed or become otherwise inaccessible (Neisser 1962; White & Pillemer 1979). Another possibility, based on notions about multiple memory systems, is that the memory system that supports declarative memory develops late and that declarative (conscious) memories are simply not formed early in life (Bachevalier & Mishkin 1984; Douglas 1975; Nadel & Zola-Morgan 1984; Overman 1990; Schacter & Moscovitch 1984).

This newer idea initially found support in the fact that the delayed nonmatching to sample task, which in adult humans and nonhuman primates depends on the integrity of medial temporal/diencephalic memory structures, is performed poorly by infant monkeys (Bachevalier & Mishkin 1984) and by human infants (Diamond 1990; Overman 1990). By contrast, habit learning, which does not depend on this same brain system, is possible in monkeys as early as 3 months of age (Bachevalier 1990). Moreover, many of the tasks that can support learning and memory in infants younger than one year can be construed as implicit memory tasks, i.e. as tasks of habituation, conditioning, and skill learning (see Schacter & Moscovitch 1984).

However, recent data have cast doubts on this view (for discussion, see Diamond 1990). One focus of interest has been the visual paired-comparison task (Fantz 1964; Fagan 1970), in which two identical items are presented together followed later by presentation of a familiar item and a novel item. Infants as young as 5 months of age tend to look more at the new item than the old item, thus providing a spontaneous measure of their memory for the previously encountered item. What kind of memory is exhibited here? Does visual paired-comparison depend on implicit (nondeclarative) memory or does it reflect early-developing declarative memory?

There are two relevant findings. First, performance on the visual paired-comparison task is severely impaired in infant monkeys with large bilateral medial temporal lobe lesions (Bachevalier 1990). Second, performance on this task is also severely impaired in human amnesic patients (McKee & Squire 1993). Thus, performance on this task is dependent on the medial temporal/diencephalic structures that are essential for declarative memory. It therefore seems reasonable to suppose that successful performance on the visual paired-comparison task reflects an early capacity for declarative memory. If so, the medial temporal/diencephalic memory system must be functional early in life, and its absence or slow development cannot account for infantile amnesia. The view that declarative memory is available early in life is also consistent with recent demonstrations of long-term-recall-like memory abilities in human infants (Baillargeon & DeVos 1991; Bauer & Mandler 1989; Mandler 1990; Meltzoff 1985). For example, infants younger than one year will reproduce actions involving toys, even one day after viewing a single demonstration of the actions by the experimenter.

If some degree of declarative memory is available to infants, what accounts for infantile amnesia? Recent evidence from nonhuman primates suggests that inferotemporal cortex, a higher-order visual association area in neocortex, is functionally immature early in life and less mature than medial temporal lobe structures (Bachevalier 1990; Bachevalier et al 1986). Thus, what limits the formation and persistence of declarative memory may be, not the maturation of the medial temporal/diencephalic structures that are essential for declarative memory, but rather the gradual maturation of the neocortical areas that are served by these structures and that are believed to be the repositories of long-term memory. This perspective provides points of contact between a neural account of infantile amnesia and accounts founded in cognitive psychology that emphasize the gradual maturation of cognition, the emergence of strategies for organizing information, the development of language, and the growth of individual identity (Neisser 1962; White & Pillemer 1979; Nelson 1988).

CAN THE BRAIN SYSTEM SUPPORTING DECLARATIVE MEMORY BE SUBDIVIDED?

Do the anatomical components of the medial temporal/diencephalic memory system make similar or different contributions to memory? Although it is entirely reasonable, and even likely, that specialization exists within this large system, it has been difficult to find compelling evidence for this idea (for two points of view, see Parkin 1984 and Victor et al 1989). For many years there was confusion on this point. Patients with Korsakoff's syndrome, an example of diencephalic amnesia, differ behaviorally in a number of respects from other amnesic patients, including those with medial temporal lobe lesions. However, it is now clear that amnesic patients with Korsakoff's syndrome typically have frontal lobe pathology (Jacobson & Lishman 1987; Shimamura et al 1988). Their frontal lobe pathology produces certain symptoms that are not essential to memory impairment itself and that can also be found in patients with frontal lobe lesions who are not globally amnesic. For example, frontal lobe pathology produces difficulty in making temporal order judgments (Meudell et al 1985; Milner 1971; McAndrews & Milner 1991; Squire 1982), it impairs metamemory (Janowsky et al 1989a; Shimamura & Squire 1986), and it produces source amnesia (Janowsky et al 1989b; Schacter et al 1984). While these findings concerning the frontal lobes are important, they do not speak to possible differences in the contributions of diencephalic or medial temporal lobe structures to memory function.

Forgetting Rates

With respect to diencephalic and medial temporal lobe brain structures, one early suggestion was that both regions are essential for establishing long-term memory but that medial temporal lobe damage is associated with rapid forget-

ting (of whatever information enters long-term memory) and that diencephalic damage is associated with a normal rate of forgetting (Huppert & Piercy 1979; Squire 1981). However, the case for medial temporal lobe damage rested on data from a single patient (H.M.), and subsequent testing of the same patient has not confirmed the original impression (Freed et al 1987). In support of these later results, patients with Alzheimer's disease, who have severe memory impairment and prominent pathology in the medial temporal lobe, also exhibited a normal rate of forgetting within long-term memory (Kopelman 1985).

It has recently been possible to study forgetting rates in amnesic patients with confirmed medial temporal lobe lesions or diencephalic lesions (McKee & Squire 1992), using the same procedure used in the original study by Huppert & Piercy (1979). The two groups of patients saw 120 colored pictures, each for 8 sec, and normal subjects saw the same pictures for 1 sec each. This procedure resulted in all three groups' performing equivalently at a 10-min retention delay. The important finding was that performance was also equivalent at retention delays of 2 hr and 1 day. Thus, the two amnesic groups exhibited equivalent (and apparently normal) rates of forgetting. The available data favor the idea that the medial temporal lobe and diencephalic structures damaged in amnesia are part of a single memory system. While it is likely that the two regions make different contributions to the function of the system, convincing evidence for this idea has yet to be demonstrated.

Spatial Memory

The medial temporal/diencephalic memory system, and more commonly the hippocampus proper, has sometimes been considered particularly important for spatial memory (O'Keefe & Nadel 1978). This idea originated in electrophysiological data from rats showing that cells in the hippocampus respond selectively when the animal is in a particular place (Ranck 1973); and also in hippocampal lesion studies, which demonstrated striking deficits in rats performing spatial memory tasks. However, hippocampal cells respond to many properties of the stimulus environment besides spatial location (Berger & Thompson 1978; Eichenbaum et al 1986; Wible et al 1986), and hippocampal lesions impair memory on a variety of nonspatial tasks, including odor discrimination learning in rats (Eichenbaum et al 1988), configural learning in rats (Sutherland & Rudy 1989), object discrimination and delayed nonmatching to sample in monkeys (Zola-Morgan et al 1989), and numerous human memory tests that assess retention of recently learned facts and events (Mayes 1988; Squire 1987). These considerations show clearly that the function of the hippocampus is not exclusively spatial, but the question remains whether the hippocampus and related limbic/diencephalic structures are more important for spatial memory than for other kinds of memory.

One approach has been to assess the status of spatial memory in human amnesia. In one study (Shoqeirat & Mayes 1991) subjects were presented with 16 nameable shapes arranged in a 7×7 grid. The scores of amnesic patients and control subjects were matched on a recognition task for the shapes by increasing the number of presentations given to the patients and by decreasing the delay between study and test. Under these conditions, the amnesic patients performed worse than the control subjects both on tests of free recall for the shapes and on tests of incidental recall for their spatial locations. In a second study (Mayes et al 1991), subjects were shown words in one of the four corners of a computer screen and instructed to remember the words and their locations. Amnesic patients and control subjects were matched on a recognition task for the words by requiring the amnesic patients to retain a shorter list of words, by providing them longer exposure to each word, and by using a shorter retention delay between study and test. Under these conditions, the amnesic patients performed worse than the control subjects in recollecting the locations of the words they had seen.

A complicating feature of these two experiments is that spatial and nonspatial memory were confounded with recall and recognition memory. Also, whenever a match is forced between amnesic patients and control subjects based on just one data point and using just one measure of memory, it is possible that amnesic patients would perform poorly on many other measures. Further, some spatial tasks might be failed because they approximate tests of source memory—e.g. tests in which spatial location provides important context for what is to be remembered. Finally, a recent study of human amnesia found only proportionate impairments of spatial memory relative to recall and recognition memory, using variations of the same tasks just described (MacAndrew & Jones 1993). An additional complication is that many of the amnesic patients in these studies had frontal lobe pathology, which can especially affect recall performance and can cause source amnesia (Janowsky et al 1989; Janowsky et al 1989b; Jetter et al 1986).

In another study, object name recall, object name recognition, and object location memory were tested in patients with confirmed damage to the diencephalon or the hippocampal formation (Cave & Squire 1991). Amnesic patients and normal subjects were matched on both recall and recognition by testing amnesic patients after a 5-min delay and different groups of control subjects after delays from 5 min to 5 weeks. The main finding was that, when the recall and recognition performance of amnesic patients was matched to the recall and recognition performance of control subjects, spatial memory performance was equivalent in the two groups. Taken together, the available data in humans do not provide strong support for the idea that the hippocampus, or other components of the medial temporal/diencephalic memory system, are especially involved in spatial memory. A reasonable alternative is that spatial memory is simply a good example of a broader class of (declarative, rela-

tional) memory abilities that are dependent on the integrity of this system (also see Eichenbaum et al 1992; Squire & Cave 1991).

This issue was also explored in monkeys with lesions of the posterior medial temporal lobe that included the hippocampus, the parahippocampal gyrus, and the posterior entorhinal cortex (Parkinson et al 1988). The monkeys were severely impaired in forming associations between objects and places. In addition, they were more severely impaired on this object-place association task than on a recognition memory task for objects (delayed nonmatching to sample). A comparison group with lesions of the anterior medial temporal lobe, which included the amygdala and underlying perirhinal cortex, performed about as well on the recognition task as the monkeys with hippocampal formation lesions, but were only mildly impaired on the spatial task.

Although more work is needed, these results can be interpreted in the light of recent information concerning the anatomical projections from neocortex to the anterior and posterior portions of the medial temporal lobe. Parietal cortex, which processes spatial information, projects posteriorly to parahippocampal cortex but not anteriorly to perirhinal cortex. Inferotemporal cortex, which processes visual pattern information, projects more strongly to perirhinal cortex than to parahippocampal cortex (Suzuki et al 1991). The perirhinal and parahippocampal cortices provide nearly two thirds of the input to entorhinal cortex, which in turn originates most of the afferent projections to the hippocampus. Based on these considerations, spatial memory functions may be associated more with parahippocampal cortex than with perirhinal cortex. Accordingly, posterior medial temporal lobe lesions (i.e. lesions that include parahippocampal cortex) would be expected to disrupt spatial memory more than anterior lesions. By this view, although a specialization for spatial memory might exist in the parahippocampal cortex, and a specialization for visual memory in the perirhinal cortex, no such specialization should be found in the entorhinal cortex or in the hippocampus itself, because these structures receive convergent projections from both the perirhinal and parahippocampal cortices.

Recall and Recognition

Another important question about the function of the brain system that supports declarative memory is whether it is equivalently involved in the two fundamental processes of recall and recognition. By one view, recall and recognition are closely linked functions of declarative memory (Tulving 1983; Hayman & Tulving 1989). Alternatively, recall has been proposed to depend on declarative memory, while recognition depends partly on declarative memory and partly on increased perceptual fluency—i.e. priming (Gardiner 1988; Jacoby 1983; Mandler 1980). By this view, subjects can detect the facility with which they process a test item and can attribute this improved fluency to the fact that the item was recently presented.

Evidence relevant to this issue could come from the study of amnesia, because amnesia spares priming while severely impairing declarative memory.

Accordingly, if recognition performance is supported significantly by implicit (nondeclarative) memory, amnesic patients should perform disproportionately better on recognition tests than on recall tests, in comparison to normal subjects. Two early studies that examined this issue (Hirst et al 1986, 1988) reported that amnesic patients exhibited disproportionate sparing of recognition memory. In these studies, recall and recognition performance were compared at a single performance level.

In another study, amnesic patients and control subjects were compared across a range of retention intervals (15 sec to 8 weeks), and performance was assessed independently by recall, recognition, and confidence ratings for the recognition choices (Haist et al 1992). The results were that recall and recognition were proportionately impaired in the patients, and their confidence ratings were commensurate with the level of impaired performance.

It is not entirely clear what accounts for the different findings by Hirst and his colleagues concerning the relative status of recall and recognition in amnesia. When an attempt was made to reproduce the experimental conditions from the second of these studies (Hirst et al 1988), the findings were not replicated (Haist et al 1992). The explanation may lie in differences in the locus of pathology in the patient populations and differences in the pattern of cognitive deficits present in addition to memory impairment. For example, some of the patients in the studies by Hirst et al (1988) became amnesic from a condition that produces signs of frontal lobe dysfunction, and frontal lobe pathology can affect recall performance more than recognition performance (Janowsky et al 1989; Jetter et al 1986).

The available findings provide little support for the view that recognition memory differs from recall in depending importantly on processes like priming that are intact in amnesia. Some behavioral findings with normal subjects have been taken as evidence that recognition memory regularly and typically depends on priming (i.e. increased fluency). However, the results appear to support this idea only indirectly, and recognition is usually not considered in relation to recall [Graf & Mandler 1984; Jacoby & Dallas 1981; Mandler 1980; Gardiner 1988; Gardiner & Java 1990; Johnston et al 1985; also, see Jacoby 1991 for a different method of assessing in normal subjects the separate contributions of intentional and automatic processes (recollection and familiarity) to recognition performance]. While one cannot rule out a possible contribution of priming-like phenomena to recognition performance under some conditions, another possibility is that implicit (nondeclarative) memory does not ordinarily contribute to performance on the typical recognition memory task. That is, when a recently encountered percept is encountered again, perceptual fluency will be operating and detection will be improved, but these effects need not contribute to overt judgments concerning whether the percept is familiar, in the sense of having been presented previously. Johnston et al (1991) have also concluded that the contribution of perceptual fluency to recognition memory may occur under limited conditions, perhaps when ex-

plicit (declarative) memory is weak. More work is needed to understand the dissociations that have been demonstrated in normal recognition memory performance, the implications of these dissociations for conscious and nonconscious forms of memory, and the relationship between recognition performance and free recall.

In summary, the idea developed here is that limbic/diencephalic brain structures are equivalently involved in recall and recognition. Recall and recognition are no doubt different in important ways, and the differential contribution of other brain structures, including the frontal lobe, to recall and recognition will be important in understanding the difference. For example, recognition memory would be expected to depend on processes that can be dissociated from other components of memory processing. Thus, recognition memory should be dissociable from the component of recall that depends on the contribution of the frontal lobe. The experiments reviewed here suggest simply that implicit (nondeclarative) memory probably does not typically support recognition memory performance, at least no more than it also contributes to free recall.

NONDECLARATIVE MEMORY

Whereas declarative memory is a brain-systems construct, a form of memory that is reflected in the operation of an anatomically real neural system and its interaction with neocortex, nondeclarative (or implicit) memory includes several forms of learning and memory abilities and depends on multiple brain systems. Although it is too early to develop a classification scheme for all the nondeclarative forms of memory, one can tentatively distinguish among skills and habits, some forms of conditioning, and the phenomena of priming. Information is emerging about the neural basis of these major types, and this information can be expected to be relevant to the problem of classification.

Skills and Habits

Skills are procedures (motor, perceptual, and cognitive) for operating in the world; habits are dispositions and tendencies that are specific to a set of stimuli and that guide behavior. Under some circumstances, skills and habits can be acquired in the absence of awareness of what has been learned and independently of long-term declarative memory for the specific episodes in which learning occurred. However, many skill-like tasks are also amenable to declarative learning strategies. For example, if a task is sufficiently simple and the information being acquired becomes accessible to awareness, then performance can be enhanced by engaging declarative memory strategies. Examples are available of human learning tasks that result in both declarative and nondeclarative knowledge (Willingham et al 1989), and of tasks that are learned nondeclaratively by monkeys but declaratively by humans (pattern discrimination and the 24-hr concurrent discrimination task; Zola-Morgan & Squire

1984; Malamut et al 1984; Squire et al 1988). Accordingly, identifying the varieties of nondeclarative memory is not straightforward. The most compelling examples have come from dissociations in normal human subjects, findings of fully intact performance in otherwise severely amnesic patients, and findings of fully normal performance in experimental animals with lesions of the hippocampus or related structures.

The earliest evidence that skill learning can proceed independently of long-term declarative memory came from the finding that the severely amnesic patient H.M. was capable of day-to-day improvement on a mirror-drawing task, despite being unable to remember that he had practiced the task (Milner 1962). Later, it was demonstrated that perceptuomotor learning can occur at an entirely normal rate in amnesia (Brooks & Baddeley 1976). During the past decade, it has become clear that motor-skill learning is a small subset of a much broader category of skill-based abilities that also include perceptual and cognitive skills. The perceptual skills that have now been found to be fully intact in human amnesia include mirror reading (Cohen & Squire 1980), speeded reading of normal text (Musen et al 1990), speeded reading of repeated nonwords (Musen & Squire 1991), the ability to resolve random-dot stereograms (Benzing & Squire 1989), and adaptation-level effects based on sampling sets of weights (Benzing & Squire 1989).

One particularly interesting group of experiments has demonstrated implicit learning of a sequence of regularly repeating spatial locations (Cleeremans & McClelland 1991; Lewicki et al 1987; Stadler 1989; Nissen & Bullemer 1987) or words (Hartman et al 1989). The evidence that the learning was implicit is that subjects improved their performance (*a*) in the absence of awareness that a sequence had been presented; or (*b*) in the absence of the ability to generate the sequence at the completion of testing. In this case, the sequence was presented once again, and subjects attempted to predict each successive element in the sequence before it appeared. In one study (Nissen & Bullemer 1987) it was also shown that amnesic patients could acquire the sequence at a normal rate. If the sequence tasks are complex enough, they can be attention demanding in the sense that learning is impeded by requiring subjects to perform a competing task (Nissen & Bullemer 1987). Alternatively, simpler versions of such tasks may be acquired automatically without requiring attention (Cohen et al 1990).

One reason for identifying these tasks as skill-based is that patients with Huntington's disease, who have pathological, degenerative changes in the neostriatum, have been found to be deficient in many of these tasks, including mirror reading (Martone et al 1984), adaptation-level effects (Heindel et al 1991), and sequence learning (Knopman & Nissen 1991). In two of the studies just cited, the patients with Huntington's disease performed better than other memory-impaired patients on conventional tasks of recognition memory.

Some tasks that are neither perceptual nor motor can also be acquired implicitly. For example, cognitive tasks have been studied in which subjects

attempt to achieve and then maintain a specific target value across trials. On each trial, the response needed to achieve the target value is determined algorithmically by current task conditions. When the relationship is sufficiently obscure, and not amenable to easy discovery or memorization, subjects improve their performance despite having little or no understanding of what they have done (Berry & Broadbent 1984). For example, one task asked subjects to achieve a target level of production in a fictitious sugar factory by determining how many workers should be hired on each trial. In this case, subjects learned the mapping function that related the level of sugar production on the previous trial to the target value. Amnesic patients were entirely intact at the early stages of this task, although normal subjects eventually acquired declarative knowledge about the task structure and outperformed the patients (Squire & Frambach 1990). The important finding is that early-stage acquisition of skilled behavior can sometimes proceed independently of verbal mediation and declarative knowledge.

There are other ways in which subjects can apparently learn regularities in their environment implicitly and then reveal what they have learned in their judgments or choices. In one notable study (Lewicki 1986a), normal subjects saw a few photographs of women, some with short hair and some with long hair. Hair length was systematically associated with narratives that described the women as either kind or capable. (These terms did not appear in the narratives.) A few minutes later, subjects decided (yes or no) whether new photographs depicted someone who was \"kind" or, in other cases, \"capable." Reaction times for yes and no decisions were slower when subjects judged photographs of women whose hair length had previously been associated with the corresponding attribute than when they judged photographs that were discordant with the attribute. It was suggested that processing time is increased whenever subjects have information available about the relevant covariation. In addition to these findings for reaction time, subjects more often judged photographs of women as \"kind" or \"capable" when hair length had been associated with the corresponding narrative than when it had been associated with the other narrative. However, these effects were rather small and were not consistently observed across experiments. Nevertheless, the finding that subjects indicated no awareness that hair length was linked to any attributes raises the possibility that whatever was learned about the covariance between physical features and attributes was learned independently of declarative knowledge. However, in these and similar experiments (Lewicki 1986b), it is difficult to rule out a threshold interpretation based on weak declarative memories that are more or less accessible depending on how memory is tested. For example, in the hair-length experiment, the results could mean simply that as declarative memory weakens, the ability to make judgments based on the learned relationships between stimuli will usually remain in evidence after the ability to report the relationships has reached chance levels.

Artificial grammar learning is an extensively studied problem domain in which subjects acquire knowledge through multiple presentations of unique material. Subjects see letter strings (e.g. BFZBZ) in which the letter order is determined by a finite-state rule system. After the letter strings are presented, subjects are told for the first time that the letter strings were in fact all determined by a complex set of rules. Subjects then attempt to classify new items as being either consistent (grammatical) or inconsistent (nongrammatical) with these rules. Reber (1967, 1989), who introduced this paradigm, suggested that the learning is implicit and independent of conscious access to the training items. For example, subjects can usually provide little information about the basis for their judgments, and telling subjects beforehand about the existence of the rules does not improve classification performance (Reber 1976; Dienes et al 1991). Another point of view has been that artificial grammar learning is based on conscious application of declarative knowledge that is weak and imperfect (Dulany et al 1984; Perruchet & Pacteau 1990). In support of this idea, it has been shown that the ability of subjects to recognize grammatically valid fragments of letter strings (bigrams and trigrams) was sufficient to account for their classification performance (Perruchet & Pacteau 1990).

This issue was clarified by the finding that amnesic patients, who were much poorer than normal subjects at recognizing which letter strings had been presented, were nevertheless able to classify letter strings (grammatical vs nongrammatical) as well as normal subjects (Knowlton et al 1992). This finding supports the view that artificial grammar learning is implicit, and it appears to rule out the idea that the learning is based on consciously accessible rules, declarative memory for permissible letter groups, or direct and conscious comparisons with letter strings that are stored in declarative memory.

Although these possibilities can probably be excluded, several other possibilities remain for how implicit learning of artificial grammars could occur: the implicit acquisition of abstract rules (Reber 1989; Mathews et al 1989), analogic comparisons to individual test items based on acquired (but implicit) associations between the test items and the grammatical category (Brooks & Vokey 1991; Vokey & Brooks 1992), or the acquisition of implicit associations between letter groups (chunks) and the grammatical category (Servan-Schreiber & Anderson 1990).

The ability to classify is more commonly based on natural categories, like chairs and birds, where class membership is defined by experience with exemplars rather than by fixed rules (Rosch 1973). In this case, too, a number of possible mechanisms have been proposed by which category-level knowledge is achieved (for reviews, see Estes 1988, 1991; Smith & Medin 1981). One possibility is that category-level knowledge is acquired in the form of knowledge about prototypes (a representative instance) or knowledge of the statistical characteristics of groups of exemplars, and that this knowledge is represented distinctly from knowledge about the exemplars themselves (Fried

& Holyoak 1984; Posner & Keele 1968; Reed 1972). Another possibility is that category-level knowledge has no special status but is derivative from item memory (Brooks 1978; Hintzman 1986; Medin & Schaffer 1978; Nosofsky 1984). By this view, knowledge about prototypes emerges as a property of the way in which items are stored. Specifically, a test item will be recognized as a good representative of a category because it shares many features with items in storage. Exemplar-based models of category learning can account for important aspects of classification performance such as the ability of subjects to identify the prototype more accurately than the items that were actually presented, even when subjects did not see the prototype itself and when the prototype itself is not actually represented.

A third possibility is illustrated by connectionist models in which the elements of the model are neither features nor items but homogeneous units that can vary in the strengths of their connections with each other (Estes 1991; Gluck & Bower 1988; McClelland & Rumelhart 1985; see the section below on Conditioning). In such models, knowledge about prototypes emerges naturally during the learning process as a result of the fact that multiple instances are stored in a distributed fashion within the network. Models that combine elements of these approaches have also been proposed [e.g. exemplar-based connectionist models (Kruschke 1992; Nosofsky et al 1992)].

Preliminary findings with amnesic patients suggest that prototype learning proceeds in parallel with and independently of declarative memory for specific instances (Knowlton & Squire 1992). If so, it cannot be the case that prototype knowledge is derived from or is in any way dependent on long-term declarative memory for individual instances. Whereas limbic/diencephalic brain structures support memory for individual instances, a different brain system may support the development of category-level knowledge.

One possibility is that learning based either on rules (e.g. artificial grammar learning) or natural categories (e.g. prototype learning) is best classified as habit learning. In both cases, category learning can be viewed as the acquisition of implicit associations between items or features and a category. A growing body of evidence from studies with experimental animals, reviewed below, suggests that the neural substrates of habit learning are different from those of declarative memory.

Neural Evidence for Distinguishing Skills and Habits from Declarative Memory

Recent work suggests that the brain structures important for acquiring skills and habits involve the corticostriatal system—i.e. projections from the neocortex to the caudate and putamen. Patients with Parkinson's disease, who have striatal dysfunction as the result of primary pathology in the substantia nigra pars compacta, were impaired on a cognitive skill task but intact at the declarative memory tasks of recall and recognition (Saint-Cyr et al 1988). Recent results for the delayed nonmatching to sample task and the 24-hr

concurrent discrimination task, two memory tasks developed for the monkey, have been especially illuminating. Delayed nonmatching to sample is a test of recognition memory, in which the monkey attempts to select in a two-choice test the object that was *not* presented recently. New pairs of objects are used for each trial. Monkeys initially learn to perform the task across a short delay interval and are then tested at increasing delays that can be 10 min or even longer. In the 24-hr concurrent discrimination task, monkeys are presented with 20 pairs of objects for one trial each day. One of the objects in each pair is always correct. Learning in this task occurs gradually in about 10 days.

In humans, both these tasks are learned declaratively—i.e. subjects memorize the material to be learned—and performance is impaired in amnesic patients (Squire et al 1988). In monkeys, the findings are quite different. Both tasks are impaired by damage to inferotemporal cortex (area TE), a higher-order visual area in neocortex that is essential for processing information about visually presented objects (Mishkin 1982; Phillips et al 1988). However, the two tasks can be differentiated in an important way. Performance on delayed nonmatching to sample is impaired by large medial temporal lobe lesions (Mishkin 1978; Squire & Zola-Morgan 1991), but monkeys with these same lesions learn the 24-hr concurrent discrimination task about as well as normal animals (Malamut et al 1984). In contrast, the 24-hr concurrent task is impaired by damage to the tail of the caudate nucleus, which is a target of cortical projections from area TE, but performance on delayed nonmatching to sample is not affected (Wang et al 1990).

Thus, an interaction between visual area TE and limbic/diencephalic areas is critical for visual recognition memory, but an interaction between TE and the neostriatum is critical for the 24-hr concurrent task. The results are similar for two-choice, visual pattern-discrimination learning, which is unaffected by large medial temporal lobe lesions (Zola-Morgan & Squire 1984) but is impaired by lesions of the caudate nucleus (Divak et al 1967). These differential effects have been interpreted in terms of two qualitatively different memory systems, a system that supports cognitive (or declarative) memory and a second system, involving the caudate and putamen, that supports noncognitive habit memory (Mishkin et al 1984; Phillips et al 1988).

A similar distinction was drawn on the basis of work with rats (Packard et al 1989). A win-shift task, which required animals to remember which arms of a radial maze had been recently visited, was impaired by fornix lesions but not by caudate lesions. Conversely, a win-stay task that required animals to visit arms that were marked by a light was impaired by caudate lesions but not by fornix lesions.

It is tempting to relate these habit-like tasks to habit learning in humans. A complication is that win-stay tasks and the 24-hr concurrent discrimination task are readily learned by humans using their well-developed declarative memory strategies, particularly when the rules governing reward contingencies are simple ones (Squire et al 1988). It is significant that patients with

Huntington's disease are impaired on a number of skill-like tasks involving motor responses, but neuropsychological studies are needed with habit-like tasks that have no motor component. The ability to relate findings from experimental animals and humans should improve as it becomes possible to define tasks in terms of what strategies are being used to learn them rather than in terms of the logical structure of the tasks (see the section, above, on Long-term Memory: Declarative Memory).

Conditioning

Learning of simple conditioned responses of the skeletal musculature or conditioned autonomic responses occurs normally in experimental animals despite complete removal of the hippocampus (Solomon & Moore 1975; Caul et al 1969). Moreover, amnesic patients exhibit progressive learning and 24-hr retention of a conditioned eyeblink response, despite inability to describe the apparatus or what it had been used for (Weiskrantz & Warrington 1979; Daum et al 1989). Thus, although conditioning in humans has been reported to require awareness of the CS-US contingency (Marinkovic et al 1989), the successful conditioning that has been observed in amnesic patients and in decerebrate animals (Norman et al 1977) suggests that awareness is not always necessary for conditioning to occur. However, until control subjects are tested to determine whether the learning in amnesic patients is entirely normal, the possibility remains that an essential part of conditioned performance in humans is due to declarative knowledge about the structure of the task. If so, the limbic/diencephalic structures important for declarative memory could play some role. In any case, other brain structures and connections are known to be critically important (see Thompson 1988 and Lavond et al, in this volume, for eyeblink conditioning; LeDoux 1987 for fear conditioning; Dunn & Everitt 1988 for taste aversion learning).

Limbic/diencephalic structures are not essential when experimental animals acquire a simple conditioned response—i.e. when a single CS and US are used in a standard delay paradigm, CS onset occurs about 250 msec prior to US onset, and CS and US offset occurs together. However, these structures are important for more complex conditioning procedures such as reversal of conditioned discriminations (Berger & Orr 1983), occasion setting (Ross et al 1984), trace conditioning (Moyer et al 1990), or when configural (Sutherland & Rudy 1989) or contextual cues (Winocur et al 1987) are used. An examination of these and other paradigms in human amnesic patients should help to identify fundamental aspects of declarative memory.

Some recent work on classification learning in human subjects has been inspired by theories of animal conditioning. In one paradigm, subjects performed a medical diagnosis task in which each of four different symptoms was probabilistically associated across trials with each of two fictitious diseases (Gluck & Bower 1988; Shanks 1990). On each trial, subjects were presented with a "patient" who exhibited one, two, three, or four symptoms in any

combination and tried to guess which disease the "patient" had. In this case, performance could be modeled by a connectionist network that learned according to the Rescorla-Wagner rule, as derived from studies of associative learning in animals (Rescorla & Wagner 1972). Thus, subjects could be viewed as learning to associate each symptom with one of the diseases in much the same way that a CS gradually becomes associated with a US (for other connectionist models of classification learning, see Kruschke 1992; Nosofsky et al 1992).

Other experiments with human subjects using similar tasks have demonstrated the phenomena of blocking, overshadowing, and conditioned inhibition (Chapman & Robbins 1990; Gluck & Bower 1988; Shanks 1991). These phenomena can be understood as resulting from competition among cues for associative strength. According to theories derived from animal conditioning, very predictive cues will successfully compete for the available associative strength at the expense of less predictive cues. Because the framework developed in animal conditioning accounts for these phenomena, and because simple forms of animal conditioning are known to occur independently of limbic/diencephalic brain structures, it is reasonable to expect that human learning of associations between features and categories will also occur independently of these brain structures (so long as the associative rules cannot easily be discovered and memorized).

Although some examples of human classification learning can be illuminated by theories of classical conditioning, the similarities between classification learning and classical conditioning should not be pushed too far. In terms of neural organization, the cerebellum is essential for classical conditioning of skeletal musculature (Thompson 1988; Lavond et al, in this volume), perhaps because precise timing of responses is needed (Ivry & Baldo 1992). For conditioned emotional responses, the amygdala is important. In contrast, when subjects must learn the predictive value of two or more cues, and the predictive relationship is not easily discovered, such learning is probably better viewed as another example of habit learning, just as has been suggested for artificial grammar learning and prototype learning. If so, the neostriatum may be an important substrate for classification learning.

Priming

Priming refers to an improved facility for detecting or identifying perceptual stimuli based on recent experience with them. Priming is currently the most intensively studied example of nondeclarative memory, and a number of reviews are available that consider this topic in some detail (Richardson-Klavehn & Bjork 1988; Shimamura 1986; Schacter 1990; Schacter et al 1993; Tulving & Schacter 1990). The discussion here identifies the key features of priming and considers the phenomenon in the context of brain systems. In a typical experiment, subjects see lists of words, pictures of objects, or nonverbal materials such as novel objects or line drawings. Subsequently, subjects are tested with both old and new items and asked to name words or objects, to

produce items from fragments, or to make rapid decisions about new and old items. The finding is that performance is better for old than for new items.

Two lines of evidence show that priming is dissociable from and independent of declarative memory. First, manipulations in normal subjects that markedly affect the strength of declarative memory, such as variations in the extent of elaborative processing carried out at the time of encoding, have little or no effect on priming (for review, see Schacter et al 1993). Second, several examples of priming have been shown to be fully intact in amnesic patients, including word priming as measured by word-stem completion, perceptual identification, and lexical decision (Cermak et al 1985; Graf et al 1984; Smith & Oscar-Berman 1990), visual object priming (Cave & Squire 1992b), and priming of novel objects or line patterns (Gabrieli et al 1990; Musen & Squire 1992; Schacter et al 1991). Amnesic patients provide a favorable way to establish the distinction between priming and declarative memory, because amnesic patients are impaired on conventional recall and recognition tests. If declarative memory significantly supports priming, then amnesic patients should be impaired on tests that measure priming. Finally, it has also been pointed out that measures of priming and measures of declarative memory often exhibit statistical independence (Tulving & Schacter 1990), but this criterion for making inferences about the independence of memory systems has been questioned by a number of authors (Hintzman & Hartry 1990; Ostergaard 1992; Shimamura 1985).

An early view, based especially on work with amnesic patients, was that priming involves the activation of pre-existing memory representations (Diamond & Rozin 1984; Cermak et al 1985, 1991). However, a number of studies with amnesic patients have now demonstrated robust and intact priming of nonwords as well as nonverbal material such as novel objects and line drawings that have no pre-existing representations (Haist et al 1991; Musen & Squire 1992; Schacter et al 1991; Squire & McKee 1992; for other recent studies involving normal subjects, see Bentin & Moscovitch 1988; Kersteen-Tucker 1991; Musen & Treisman 1990; Schacter et al 1991). An exception appears to be the priming of nonwords on lexical-decision tasks, which is weak even in normal subjects (Bentin & Moscovitch 1988; Verfaillie et al 1991; for a report that nonword lexical-decision priming occurs in normal subjects but not in amnesic patients, see Smith & Oscar-Berman 1990).

One of the striking features of priming is that it can sometimes be extraordinarly long-lasting. Word-stem completion priming, which was among the first well-studied examples of priming, disappears within 2 hr, at least when multiple completions are available for each word stem (Squire et al 1987). In contrast, in normal subjects priming of object naming is still present 6 weeks after a single exposure to a picture (Mitchell & Brown 1988); and word-fragment completion priming, when only one solution is available for each fragment, has been demonstrated in normal subjects after a delay of 16 months (Sloman et al 1988). The question of how long priming persists is

complicated by the possibility that tests for priming can be contaminated by declarative memory strategies. A contribution from declarative memory has been ruled out in one case by the finding of fully intact object-naming priming in amnesic patients, even 7 days after a single exposure to pictures (Cave & Squire 1992b). Thus, stimuli can result in long-lasting effects on performance that are supported independently of the limbic/diencephalic structures important for declarative memory.

Presentation of stimuli can also influence preferences and judgments about the stimuli, even when the stimuli are exposed so briefly that they cannot later be recognized (Bonnano & Stillings 1986; Kunst-Wilson & Zajonc 1980; Mandler et al 1987). A related phenomenon is that subjects are more likely to judge a proper name as famous if the name has been encountered previously. Dividing attention during the initial presentation of famous and nonfamous names markedly reduced recognition memory scores but had no effect on the fame-judgment effect (Jacoby et al 1989). Moreover, amnesic patients exhibited the fame-judgment effect at full strength (Squire & McKee 1992). These results suggest that priming not only improves the ability to identify stimuli but can also alter judgments about the stimuli.

The kinds of priming discussed so far are perceptual in the sense that the effects are pre-semantic and highly determined by the specific perceptual features of the originally presented item. For example, when pictures of objects are presented and subjects are asked to name them as quickly as possible, the priming effect is greatly attenuated by changing the orientation of the object, adding shading to the object, or changing from one example of an object to another example that has the same name (Bartram 1974; Biederman & Cooper 1991a; Cave & Squire 1992b). Also, in word-priming tasks, priming can be attenuated by changes in sensory modality from study to test and by changes in the voice of the speaker (Graf et al 1985; Jacoby & Dallas 1981; Schacter & Church 1992). Finally, priming effects are sometimes reduced by changes in type case or other surface features of words, although such effects are not always obtained (see Schacter et al 1993).

Although priming effects are highly specific, the representation that supports priming does not retain all the perceptual information in the stimulus. For example, changes in size or left-right mirror reflection of objects did not affect priming, despite the fact that these same changes significantly affected performance on tests of declarative memory (Biederman & Cooper 1991b, 1992; Cooper et al 1992). Because declarative memory was sensitive to these stimulus features, it is difficult to explain priming as depending on the same process or system that supports declarative memory.

Priming effects can also occur on tests that require semantic or conceptual processing, but these effects can be dissociated from and are likely quite different from perceptual priming (Srinivas & Roediger 1990; Tulving & Schacter 1990). For example, conceptually driven priming depends on the extent of elaborative encoding at the time of study (Hamman 1990). Neverthe-

less, this kind of priming is also independent of declarative memory, as demonstrated by the fact that amnesic patients are fully intact at tests of free-association priming (Shimamura & Squire 1984) and priming of category exemplars (Gardner et al 1973; Graf et al 1985; Schacter 1985).

There has also been interest in whether associative priming effects can occur for previously unrelated pairs of items (Graf & Schacter 1985; Moscovitch et al 1986). Recent work suggests that the most commonly studied paradigm (word-stem completion priming using novel associates as cues) does not yield associative priming in severely amnesic patients (Cermak et al 1988; Mayes & Gooding 1989; Schacter & Graf 1986; Shimamura & Squire 1989). Although the phenomenon as a whole can be dissociated from declarative memory in normal subjects (see Schacter et al 1993), the initial formation of novel associations probably places a critical demand on declarative memory (Shimamura & Squire 1989). In addition, the rapid (one-trial) formation of implicit associations between unrelated word pairs (using a paradigm based on reading speed; Moscovitch et al 1986) has proven difficult to demonstrate within implicit memory (Musen & Squire 1993).

Some information has recently become available about the neural basis of perceptual priming. In divided visual-field studies with normal subjects, word-stem completion priming was greater when word stems were presented to the right hemisphere than to the left (Marsolek et al 1992). This effect was obtained if and only if the study and test items were in the same sensory modality and in the same type case. Thus, the right cerebral hemisphere appears to be more effective than the left at supporting form-specific components of perceptual priming. The left hemisphere may support more abstract components of perceptual priming—e.g. the priming that survives type-case changes and modality changes. These results suggest that the two hemispheres contribute to priming in different ways, and that the results of priming studies can be expected to differ depending on which hemisphere is dominant in performing the task.

A recent study using positron emission tomography (PET) has provided direct evidence for the involvement of right posterior cortex in word priming (Squire et al 1992). Study and test items were presented visually and always in uppercase letters. During word-stem completion priming there was a significant reduction of cerebral blood flow in right extrastriate cortex, in the region of the lingual gyrus, in comparison to a baseline condition in which subjects also completed word stems but none of the possible word completions had been presented for study. This finding suggests a simplifying hypothesis for perceptual priming: After a word has been presented for study, less neural activity is subsequently required to process the same stimulus. The right posterior cortical locus identified by PET in this study is precisely the same region that in earlier studies was activated by the visual features of words (Petersen et al 1990). Words, nonwords, letter strings, and letter-like shapes were all effective at activating this locus.

The PET findings count against earlier proposals that a left-hemisphere word-form area is the locus of word priming (Schacter 1990; Tulving & Schacter 1990). More likely, left or right posterior cerebral cortex is important depending on whether priming is based on more abtract or more form-specific mechanisms. Indeed, we suggest that perceptual priming may occur in any of the more than 30 cortical areas known to be involved in visual information processing (Felleman & Van Essen 1991). Which areas are involved in any particular case would depend on the extent of the match between study and test materials and task demands. Indeed, this diversity of cortical areas potentially relevant to priming may help to explain why so many dissociations have been found among different kinds of verbal priming tests (Keane et al 1991; Srinivas & Roediger 1990; Witherspoon & Moscovitch 1989).

Priming is presumably adaptive because animals evolved in a world where stimuli that are encountered once are likely to be encountered again. Perceptual priming improves the speed and fluency by which organisms interact with familiar stimuli. For example, in the case of visual priming, the posterior visual cortex becomes more efficient at processing precisely those stimuli that have been processed recently. This plasticity occurs well before information reaches the limbic/diencephalic structures important for declarative memory.

PERSPECTIVE

This review has considered several kinds of memory as well as the distinct brain systems that support them. It has sometimes been proposed that distinctions between kinds of memory are best understood as reflecting the different processes that can be used to access a common memory trace (Blaxton 1989; Jacoby 1988; Masson 1989; Roediger 1990). When discussion of this issue is limited to priming, the matter can seem difficult to settle (see Schacter 1990). For example, the same single words can be remembered intentionally, or they can be produced in a priming paradigm. However, when discussion of memory is broadened to include the learning of skills and habits, and conditioning phenomena, the data favor a systems perspective over a processing perspective (for discussion of points of contact between these two views, see Roediger 1990; Schacter 1992; Tulving & Schacter 1990). Indeed, it cannot even be assumed that long-term storage of declarative and nondeclarative memories occurs in the same brain region. Declarative memories require the reciprocal anatomical connections that enable the neocortex to interact with the hippocampus and related structures, and the neocortex is thought to be the final repository of declarative memory. Skills and habits depend on corticostriatal projections, and these projections are not reciprocated by return projections to neocortex from the neostriatum. Accordingly, one possibility is that the storage of information underlying skills and habits occurs at the synapses between cortical neurons and neurons in the neostriatum.

The findings from PET also strongly endorse a brain-systems orientation. Word-stem completion priming was supported significantly by right extrastriate visual cortex. Intentional recall of words using word stems as cues engaged the right hippocampal region significantly more than the priming condition did. (The priming condition also engaged the hippocampal region more than the above-mentioned baseline condition did. Because subjects became aware of the link between word stems and study words during the priming task, some explicit visual recognition probably occurred as the word stems were presented, even though the performance measure in the priming task does not itself depend on declarative memory.)

Recent studies of event-related potentials (ERPs) also suggest that different brain regions are involved in word recall and recognition on the one hand, and word priming, on the other (Paller 1990; Paller & Kutas 1992). For example, the ERP associated with intentional recognition had a different scalp distribution and a different latency from the ERP associated with perceptual identification priming (Paller & Kutas 1992).

It has been noted previously that the finding of task dissociations in normal subjects is an insufficient basis on which to postulate two or more memory systems (Roediger 1990; Schacter 1992). Indeed, as several authors have noted (Graf et al 1984; Jacoby 1991; Roediger 1990; Schacter 1990; Squire 1992b), the proper emphasis is on the processes and strategies that subjects use, not the tasks used to measure memory. Moreover, to support hypotheses about multiple memory systems, evidence is needed that is independent of dissociation experiments. This kind of evidence has come from findings in experimental animals and neurological patients where the contributions to performance of anatomically defined brain systems can be evaluated directly. For example, a consideration of this evidence has led us in this review to suggest that superficially different tasks including artificial grammar learning and classification learning in human subjects, the 24-hr concurrent discrimination task in monkeys, and win-stay, lose-shift maze tasks in rodents all depend on similar underlying computations and might usefully be categorized together under the generic heading of habit learning.

One difficulty with the processing view is that it has been stated rather abstractly, so that it is sometimes difficult to appreciate what would count for or against it. A difficulty with the systems view is that the definition of the term \"system" is uncertain, and it is not always clear from studies of normal subjects when behavioral findings justify postulating a separate memory system. The concept of brain systems, while not entirely free of problems itself, provides a more concrete and in the end a more satisfying basis for thinking about memory systems. This is because a long tradition of anatomical and physiological work on the structure and organization of the brain has concerned itself with the identification and study of separable neural systems, sometimes independently of or in advance of any understanding of their functional significance.

This kind of information provides powerful convergent evidence that becomes extremely compelling when a function identified and characterized from psychological data appears to map onto a neural system that has been defined previously by anatomical and physiological criteria. Indeed, this is approximately what has happened in the case of limbic/diencephalic structures (for declarative memory), the neostriatum (for skills and habits), and the cerebellum (for some forms of conditioning). In any case, it should be clear that the issue is not a philosophical or semantic one about whether a processing or systems view provides the best research approach. The issue is about how memory is actually organized and how the brain accomplishes learning and memory.

A fundamental issue that so far has yielded little biological information concerns the nature and locus of long-term declarative representations. However, one can find a few clues and identify some guiding principles. The brain is highly specialized and differentiated, and it is organized such that different regions of neocortex carry out parallel computations on many different dimensions of external stimuli. Memory for an event, even memory for a single object, is stored in component parts and in a distributed fashion across geographically separate parts of the brain (Mishkin 1982; Squire 1987). Although direct evidence is not available, permanent information storage is thought to occur in the same processing areas that are engaged during learning. By this view, long-term memory is stored as outcomes of processing operations and in the same cortical regions that are involved in the perception and analysis of the events and items to be remembered.

Available information about the organization and structure of knowledge systems suggests a surprising degree of specialization in how information is stored. Cortical lesions in humans can produce remarkably selective losses of category-specific knowledge—e.g. loss of the ability to comprehend the names of small \"indoor" objects with relative preservation of the names of large \"outdoor" objects; or loss of knowledge about inanimate, man-made objects with relative preservation of knowledge about foods and living things (Damasio 1990; Farah et al 1991; Hart et al 1985; Warrington & Shallice 1984; Yamadori & Albert 1973). It has been proposed that these specializations can be understood in terms of the nature of the interaction between the perceiver and objects in the world during the time that objects are learned about (Damasio 1990; Farah & McClelland 1991; Warrington & McCarthy 1987). By this view, the sensory modality that is relevant to learning about an item and the nature of the relevant information (physical or functional) will influence the locus of information storage. For example, information based especially on physical features such as shape and color (e.g. gems, animals) will be stored in different loci from information based more on manual interaction and an understanding of function (e.g. tools and furniture).

What is needed is a way to access neurons within the networks that actually represent long-term declarative knowledge, so that the locus and organization

of representations can be studied directly. There are abundant examples, from single-cell recordings of neurons in the temporal lobe of awake monkeys, where neurons change their activity rather quickly in response to behaviorally relevant stimuli (Fuster & Jervey 1981; Miller et al 1991; Riches et al 1991). However, it is difficult to know in these cases what kind(s) of memory the neurons might be involved in. Particularly in experiments that require retention of newly acquired information across delays of less than a minute, neurons that respond either during the delay or when test stimuli are presented at the end of the delay could be related to short-term memory or priming. The question is how would one determine whether or not a neuron being recorded from were part of a network representing information in long-term declarative memory?

One promising approach is suggested by a recent study of paired-associate learning in the awake monkey (Sakai & Miyashita 1991). During extended training, monkeys learned 12 pairs of computer-generated patterns. On each trial, a monkey observed one of the pictures (the cue) and then 4 sec later selected its associate from among two patterns. A reward was delivered if a correct response occurred within 1.2 sec. Neurons were found in the anterior temporal cortex that responded strongly to one of the pictures when that picture served as a cue in the paired-associate test. These same neurons were found to exhibit increased activity during the 4-sec delay on trials when the associate of that picture served as a cue. These neurons were termed \"pair-recall" neurons. Thus, neurons acquired information about the specific pairings of the patterns that were used, and they exhibited activity related to the process of stimulus recall.

These results should make it possible to pursue several interesting experimental questions. Does development of pair-specific neuronal activity require a contribution from the limbic/diencephalic brain system that is essential for declarative memory? What would be the effect of inactivating circuitry within the hippocampus or inactivating efferent projections from entorhinal cortex to neocortex? If the limbic/diencephalic system proved essential, then should pair-recall neurons be viewed as belonging to a network that represents long-term declarative memory of the associations? What is the role of the limbic/diencephalic system in the acquisition of pair-specific activity, its maintenance, and its expression? In other words, how does the limbic/diencephalic system interact with neocortex during learning, consolidation, and retrieval? If it becomes feasible, using this or some other paradigm, to observe directly the development of cortical plasticity related to declarative memory, one can expect the entire discussion of memory systems to be raised to a new level.

In the span of just a few years, the field of memory research has moved from a rather monolithic view of long-term memory to a view that distinguishes several kinds of memory. One system involves limbic/diencephalic structures, which in concert with neocortex provides the basis for conscious

recollections. This system is fast, phylogenetically recent, and specialized for one-trial learning—e.g. for the rapid acquisition of associations, propositions, or items in a context. The system is fallible in the sense that it is sensitive to interference and prone to retrieval failure. It is also precious, giving rise to the capacity for personal autobiography and the possibility of cultural evolution.

Other kinds of memory have also been identified—e.g. those involved in skills and habits, priming, conditioning, and perhaps the ability to acquire category-level generic knowledge. Such memories can be acquired, stored, and retrieved without the participation of the limbic/diencephalic brain system. These forms of memory are phylogenetically early, they are reliable and consistent, and they provide for myriad, nonconsious ways of responding to the world. In no small part, by virtue of the nonconscious status of these forms of memory, they create much of the mystery of human experience. Here arise the dispositions, habits, and preferences that are inaccessible to conscious recollection but that nevertheless are shaped by past events, influence our behavior, and are a part of who we are.

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