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COGNITIVE NEUROSCIENCE OF HUMAN MEMORY

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ABSTRACT

Current knowledge is summarized about long-term memory systems of the human brain, with memory systems defined as specific neural networks that support specific mnemonic processes. The summary integrates convergent evidence from neuropsychological studies of patients with brain lesions and from functional neuroimaging studies using positron emission tomography (PET) or functional magnetic resonance imaging (fMRI). Evidence is reviewed about the specific roles of hippocampal and parahippocampal regions, the amygdala, the basal ganglia, and various neocortical areas in declarative memory. Evidence is also reviewed about which brain regions mediate specific kinds of procedural memory, including sensorimotor, perceptual, and cognitive skill learning; perceptual and conceptual repetition priming; and several forms of conditioning. Findings are discussed in terms of the functional neural architecture of normal memory, age-related changes in memory performance, and neurological conditions that affect memory such as amnesia, Alzheimer's disease, Parkinson's disease, and Huntington's disease.

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INTRODUCTION

The cognitive neuroscience of human memory aims to understand how we record, retain, and retrieve experience in terms of memory systems—specific neural networks that support specific mnemonic processes. Advances in the study of the cognitive neuroscience of human memory reveal the functional neural architecture of normal human memory and illuminate why focal or degenerative injuries to specific memory systems lead to characteristic patterns of mnemonic failure.

Studies of patients with brain lesions have provided the foundations of our knowledge about the biological organization of human memory. Lesions have produced dramatic and often unexpected mnemonic deficits that provide clues about which brain regions are necessary for which memory processes. The behavior of memory-impaired patients with brain lesions, however, does not delineate what process is subserved by the injured tissue. Rather, the behavior reflects what uninjured brain regions can accomplish after the lesion. Further, naturally occurring lesions often impair multiple brain systems, either by direct insult or by disconnection of interactive brain regions. It is therefore difficult to determine which deficit is the consequence of which part of a lesion.

Although lesion studies continue to provide new evidence, functional neuroimaging studies using positron emission tomography (PET) or functional magnetic resonance imaging (fMRI) now permit the visualization of memory processes in the healthy brain. Functional neuroimaging studies allow for the design of psychological experiments targeted at specific memory processes. They are limited, however, by several factors. PET and fMRI derive their signals not from neural activity but rather from local changes in blood flow or metabolism correlated with neural activity. The local vascular changes affect the distribution of an injected radionuclide (usually O^{15}) in PET or magnetic properties that are blood-oxygen level dependent (BOLD) in fMRI. The indirect

measure of neural activity limits the temporal and spatial fidelity of activations.

There is also a great deal of psychological interpretation involved in understanding the meaning of an activation, i.e. in specifying what mental process is signified by an activation. Most imaging studies report activations arising from the difference between two tasks. Such differences are not only open to a variety of interpretations but also are often confounded with factors such as task difficulty or trial duration. Further, neuroimaging constraints influence task designs, such as the need to block stimuli in homogenous conditions for between-condition comparisons where each condition often lasts for 30 seconds (fMRI) or 2 minutes (PET). Ongoing developments, however, are expanding the range of experimentation that can be performed within the constraints of fMRI measurement, including parametric task designs (Cohen et al 1997), multiple regression analyses (Courtney et al 1997), and single-trial analysis (Buckner et al 1996). Even with these improvements, remarkable progress in brain imaging techniques does not compete with the psychological analysis of behavior but instead places a new premium upon the thoughtfulness and accuracy of such analysis.

The combination of lesion and neuroimaging studies may overcome the limitations of each source of evidence and provide powerful, mutual constraints on ideas about memory systems. For example, activations for some memory tasks occur in brain regions that can be severely injured without affecting performance on that task. Those activations may represent correlated memory processes that are not participating in the form of memory being measured in the neuroimaging study. Without the lesion evidence, it would be difficult, if not impossible, to discriminate between activations signifying processes that are essential or nonessential for the specific form of memory being measured. Thus, convergent evidence from lesion and functional neuroimaging studies should help both in advancing the understanding and in avoiding the misunderstanding of human memory systems.

The present review emphasizes how lesion and functional neuroimaging evidence converge to identify the neural networks and characterize the mnemonic processes of long-term memory systems. Progress in delineating short-term and working memory systems is reviewed by Smith & Jonides (1994, 1997).

DECLARATIVE MEMORY

Declarative memory encompasses the acquisition, retention, and retrieval of knowledge that can be consciously and intentionally recollected (Cohen & Squire 1980). Such knowledge includes memory for events (episodic memory)

or facts (semantic memory) (Tulving 1983). Episodic memories are measured by direct or explicit tests of memory, such as free recall, cued recall, or recognition, that refer to a prior episode (Graf & Schacter 1985). In contrast, nondeclarative or procedural kinds of memory encompass the acquisition, retention, and retrieval of knowledge expressed through experience-induced changes in performance. These kinds of memory are measured by indirect or implicit tests where no reference is made to that experience. Skill learning, repetition priming, and conditioning are classes of implicit tests that often reveal procedural memory processes dissociable from declarative memory.

A source of common confusion and theoretical challenge lies in the distinction between test instructions and memory processes. It is easy to classify a test as explicit when subjects are asked to intentionally retrieve memories from a specified episode, or as implicit when subjects are asked to perform a task and no reference is made to any prior episode. It is not easy to determine, however, what kind of memory processes are involved when performing the test. There are many examples where memory on an implicit test is correlated with memory on a related explicit test. A parsimonious interpretation is that these implicit tests invoke some of the same declarative memory processes typically invoked by explicit memory tests. There are theories (e.g. Cohen & Eichenbaum 1993) and methods (Bowers & Schacter 1990) that address the distinction between implicit tests that measure processes associated with or dissociated from declarative memory. Presently, however, these theories and methods cannot predict in principle whether a specific implicit test will or will not invoke declarative memory processes.

Medial-Temporal and Diencephalic Systems

Lesions to medial-temporal and diencephalic brain regions yield amnesia, a selective deficit in declarative memory with sparing of short-term memory, remote memories, and motor, perceptual, and cognitive capacities (Scoville & Milner 1957, Cohen & Squire 1980). All amnesic patients have an anterograde amnesia—an inability to learn new information after the onset of the amnesia. Amnesic patients vary in their severity and extent of their retrograde amnesia—a loss of information gained before the onset of the amnesia. Retrograde losses of memory in amnesia are usually temporally graded in that they are most severe for time periods closest to amnesia onset. Unilateral left or right lesions produce material-specific declarative memory deficits for verbal or nonverbal information, respectively (Milner 1971). Bilateral lesions produce a global amnesia that extends to verbal and nonverbal information. Global amnesia impairs the ability to acquire both episodic and semantic memories, such as the meaning of words and concepts (Gabrieli et al 1988).

Diencephalic lesions that produce amnesia, as seen in patients with alcoholic Korsakoff's syndrome, involve damage to the medial thalamus and often the mammillary nuclei. Damage to these regions is sufficient to produce severe memory impairments even when medial-temporal regions remain anatomically intact (Press et al 1989). The medial thalamic lesions appear to have a greater effect than the mammillary body lesions upon declarative memory. It is unclear at present, however, what specific aspect of the medial thalamic lesions accounts for amnesia.

Medial temporal lesions may result from resection (as in the case of the noted amnesic patient HM), anoxia, herpes simplex encephalitis, infarction, or sclerosis. The first lesions in most cases of Alzheimer's disease (AD) may occur in the medial temporal lobe (Hyman et al 1984), and this may account for amnesia being the most common initial problem in AD. Unlike patients with pure amnesia, however, AD patients have a dementia defined by the compromise of at least one additional, nonmnemonic function. Further, AD patients also have early damage to cholinergic neurons in the basal forebrain (Arendt et al 1983), and lesions in that area cause declarative memory impairments. Therefore, it is difficult to ascribe the amnesia in AD exclusively to medial-temporal injuries.

The medial temporal-lobe memory system consists of multiple structures, most of which may be classified as belonging to one of two major regions. High-level unimodal and polymodal cortical regions provide convergent inputs to the parahippocampal region, which is comprised of parahippocampal and perirhinal cortices (Suzuki & Amaral 1994). The parahippocampal region provides major inputs to the hippocampal region, which is composed of the subiculum, the CA fields, and the dentate gyrus. Entorhinal cortex is variably classified as belonging to either the hippocampal or parahippocampal region. The amygdala is located in the medial temporal lobe, but it has a limited role in declarative memory that is discussed later.

Postmortem analysis of medial-temporal damage in patients with well-characterized amnesias shows that damage restricted to a small part of the hippocampal region, the CA1 field, is sufficient to produce a clinically significant anterograde amnesia. More extensive damage to additional medial-temporal structures aggravates both the severity of the anterograde amnesia and the temporal extent of the retrograde amnesia. When lesions extend beyond the hippocampal region to entorhinal and perirhinal cortices, retrograde amnesias extend back one or two decades (Corkin et al 1997, Rempel-Clower et al 1996).

Neuroimaging studies provide convergent evidence about the participation of medial-temporal regions in declarative memory. Medial-temporal activations are observed during intentional memory retrieval (Squire et al 1992; Schacter et al 1995b, 1996a,c). These activations are associated with success-

ful memory retrieval: Activations are greater when people make memory judgments for studied than for novel materials and for well-remembered than for poorly remembered words. Medial-temporal activations occur also during the encoding of memories. The encoding activations appear to index stimulus novelty: They are greater for stimuli seen initially rather than repeatedly (Tulving et al 1994, Stern et al 1996).

One study showed that encoding and retrieval activations occurred in different medial-temporal regions (Gabrieli et al 1997a). Retrieving well-learned memories resulted in an anterior activation in the subiculum, a component of the hippocampal region. Encoding novel memories resulted in a posterior activation in parahippocampal cortex, a component of the parahippocampal region. The two locations are in agreement with findings of a posterior locus for encoding (Stern et al 1996) and a positive correlation between the magnitude of anterior hippocampal activation and retrieval accuracy in a recognition memory test (Nyberg et al 1996).

Lesion studies showed that a medial-temporal system is critical for declarative memory, but it has been difficult to glean the specific declarative processes mediated by components of that system because lesions typically traverse multiple medial-temporal structures. Imaging studies are beginning to provide information about the specific contributions of different components of the medial-temporal memory system to declarative memory. The finding that different medial-temporal structures make different contributions to declarative memory may help explain why more extensive lesions, which may compromise multiple declarative memory processes, yield more severe anterograde and retrograde amnesias.

Amygdala

Lesion and functional neuroimaging findings have illuminated the importance of the amygdala in emotional aspects of human memory (reviewed in Phelps & Anderson 1997). Because the amygdala is near the hippocampal formation, amnesic patients, such as HM, often have damage to both structures. It was, therefore, difficult to distinguish between the specific mnemonic roles of these adjacent limbic structures. However, a rare congenital dermatological disorder, Urbach-Weithe syndrome, leads to mineralization of the amygdala that spares the hippocampal formation. The amygdala is also resected for treatment of pharmacologically intractable epilepsy, although the resection usually involves additional medial-temporal structures. Studies with these patients have allowed for a more direct examination of the consequence of amygdala lesions in humans.

There is convergent evidence for a limited role for the amygdala in declarative memory. Normal subjects show superior memory for emotionally disturb-

ing relative to emotionally neutral stimuli. An Urbach-Weithe patient showed normal memory for neutral slides but failed to show the normal additional memory for the emotionally salient slides (Cahill et al 1995). In one PET study, amygdala activation correlated with individual differences in later recall for emotional, but not for neutral, film clips (Cahill et al 1996). In another PET study, amygdala activation was noted during retrieval of autobiographical memories that were likely to have personal emotional salience (Fink et al 1996).

At present, lesion and neuroimaging evidence indicates that the amygdala has a circumscribed role in declarative memory for emotionally disturbing or aversive experiences. The amygdala participates not only in explicit memory for aversive stimuli but also implicit memory for aversive stimuli tested via fear conditioning (reviewed below). Patients with amygdala lesions show selective deficits in the identification of fearful or angry facial expressions (Adolphs et al 1994) or prosody (Scott et al 1997). Amygdala activations occur in PET and fMRI studies during the perception of fearful facial expressions or scenes (Morris et al 1996). Thus, the amygdala appears to have a widespread role in processing negatively salient stimuli.

Neocortical Systems

Declarative memory is generally thought to reflect an interaction between medial-temporal/diencephalic and neocortical brain regions. The fact that medial-temporal or diencephalic lesions spare remote memories has encouraged the view that the neocortex is the ultimate repository of consolidated long-term memory. Neocortical areas are also viewed as critical for encoding (processing and analyzing) current experience. This may occur in a domain-specific fashion, with different cortical regions processing different perceptual (e.g. visual, auditory, tactual) and cognitive (e.g. verbal, spatial) features of an experience. Thus, the neocortex contributes to the encoding, storage, and retrieval of declarative memories.

CORTICAL REPRESENTATION OF KNOWLEDGE Lesions have revealed remarkable specificity in the cortical representation of long-term memories. Some patients with cortical lesions have shown category-specific inabilities to produce the names of objects (anomias). Thus, patients have shown selective deficits for retrieving the names of (*a*) people and other proper nouns (Semenza & Zettin 1989); (*b*) fruits and vegetables (Hart et al 1985); (*c*) living things such as animals (Damasio et al 1996); and (*d*) manufactured things such as tools (Damasio et al 1996). These patients can demonstrate retention of knowledge about objects that they cannot name by, for example, selecting the names of such objects from multiple choices. Other patients appear to have category-

specific losses of knowledge for objects, with disproportionate losses of knowledge for either living (Warrington & Shallice 1984) or manufactured objects (Warrington & McCarthy 1983). Even within the category of living things, a patient has exhibited a dissociation between impaired verbal versus intact pictorial knowledge (McCarthy & Warrington 1988). Yet other patients have shown focal losses of autobiographical knowledge following injury to the right anterior temporal lobe (Kapur et al 1992). These patients differ from amnesic patients in that declarative memory is relatively spared and that the retrograde amnesia is not temporally graded.

Neuroimaging studies motivated by these surprising patient findings have provided corroborating evidence. Thus, separate loci of activations are found in the left-temporal lobe during the naming of people (proper nouns), tools, and animals that correspond to the lesion sites producing selective anomias (Damasio et al 1996). The naming of tools or animals yields both shared and separate activations (Martin et al 1996) as does answering conceptual questions about corresponding pictures and words (Vandenberghe et al 1996). Listening to one's own autobiographical passage, relative to another person's autobiographical passage, results in activation of right frontal- and temporal-lobe regions (Fink et al 1996). These neuroimaging studies indicate that the unexpected dissociations of knowledge in patients are not idiosyncratic phenomena, but rather the consequence of the differential cortical geography of knowledge in the healthy brain.

Two emerging principles may be discerned from these neuroimaging studies. First, knowledge in any domain (e.g. for pictures or words, living or manufactured objects) is distributed over a specific, but extensive, neural network that often extends over several lobes. Injury to any component of that network could affect performance in that domain, with the specific effect reflecting what aspect of that knowledge is represented in that component of the network. Second, some localization appears to be a consequence of how various classes of knowledge interact with different perceptual and motor systems. Thus, we have motor experiences with tools that vary systematically in relation to each tool's function. In contrast, most people have far fewer motor experiences with animals. Perhaps for this reason, naming tools relative to naming animals, or naming an action (writing) relative to naming a color (yellow) associated with an object (pencil), yields activation in left prefrontal regions near motor cortex (Martin et al 1995, 1996).

ENCODING OF MEMORIES Left frontal activations, especially in the anterior portion of the inferior prefrontal gyrus, have been found when subjects perform tasks that enhance memory for the encoded information. There is greater left prefrontal activation when subjects make semantic (deep) versus nonse-

mantic (shallow) decisions about words (Demb et al 1995, Gabrieli et al 1996a, Kapur et al 1994), study words with a mild versus a severe division of attention (Shallice et al 1994), or generate versus read words (Petersen et al 1989). In one of the first lesion studies inspired by imaging findings, it was found that patients with left frontal lesions were impaired at making the same semantic judgments that had yielded left frontal activations in healthy subjects (Swick & Knight 1996). Although the left frontal activation is evident also for nonverbal stimuli such as faces (Haxby et al 1996), it seems likely that this activation is most closely linked to semantic processes associated with language. This kind of activation, which may also be considered one of semantic memory retrieval, occurs in the right prefrontal cortex of patients who are right-hemisphere dominant for language (Desmond et al 1995).

STRATEGIC MEMORY Declarative memory tasks differ in their strategic memory demands, i.e. in how much retrieved memories must be evaluated, manipulated, and transformed. Recognition tests given shortly after study may have minimal strategic demands as subjects quickly decide whether or not a particular stimulus had been included in a study list. Tests of free recall, delayed recognition, temporal order, and source may have much greater strategic demands because subjects have to figure out how they will recall stimuli or what time or place a familiar stimulus was encountered.

Frontal-lobe lesions can compromise performance on strategic memory tasks even when patients perform normally or near normally on recognition tests (this pattern differs from amnesia where performance on both strategic and nonstrategic memory tasks is severely impaired). Patients with frontal-lobe lesions have disproportionate impairments on tests of free recall (Janowsky et al 1989), recency or temporal order judgments (Milner 1971), frequency judgments (Smith & Milner 1988), self-ordered pointing (Petrides & Milner 1982), and recollection of the source of information (Janowsky et al 1989). An imaging study with normal subjects found frontal-lobe activation during the performance of self-ordered pointing (Petrides et al 1993a).

Strategic memory tasks may require subjects to reason about their memories, and there is evidence that the frontal lobes are important in reasoning. Frontal patients perform poorly on problem-solving or reasoning tasks that require the generation, flexible maintenance, and shifting of plans, such as the Wisconsin Card Sorting Test (Milner 1963) and the Tower-of-London Test (Shallice 1982). Neuroimaging studies have found prominent frontal-lobe activations when people reason as they perform problem-solving tasks (Baker et al 1996, Prabhakaran et al 1997). Thus, the frontal-lobe contribution to strategic memory may be one of problem-solving and reasoning in the service of difficult declarative memory demands.

Selective deficits of strategic declarative memory have been found also in degenerative or developmental diseases of the basal ganglia, such as Parkinson's disease (PD), Huntington's disease (HD), and Gilles de la Tourette's syndrome (GTS) (Gabrieli 1996). Striatal diseases also impair reasoning (Lees & Smith 1983). In addition, PD, HD, and GTS patients have significantly reduced working memory capacities, and the reductions are highly correlated with the strategic memory and reasoning deficits. Indeed, difficult tasks that tax working memory capacity routinely yield frontal activations (e.g. Cohen et al 1997, Petrides et al 1993a,b). Thus, it may be hypothesized that fronto-striatal lesions reduce working memory capacity, which limits reasoning ability and, in turn, impairs strategic memory performance. Further, the neurotransmitter dopamine may be critical for working memory. PD patients have severely reduced dopamine functioning, and dopamine treatment can enhance working memory performance in PD patients (Cooper et al 1992).

There are several reasons to hypothesize that age-related decline in fronto-striatal function may account for a great deal of normal age-related decline in memory performance (Gabrieli 1995). Working memory, reasoning, and strategic memory performance decline linearly across the life span. Similarly, dopaminergic function appears to decline linearly across the life span, with a 5–10% decline per decade. The notion that it is specifically fronto-striatal dysfunction that accounts for age-related declines in memory performance is supported by a neuroimaging study that found age-related differences in frontal but not medial-temporal regions during explicit retrieval (Schacter et al 1996c).

Associations between working memory, reasoning, and strategic memory occur in patient studies, normal aging, and functional neuroimaging. All three capacities appear to depend upon dopaminergic fronto-striatal systems. The extent to which these associations reflect shared versus neighboring processes and whether reductions in one capacity are causal or merely correlated with changes in the other capacities remains to be determined.

INTENTIONAL RETRIEVAL A consistent but poorly understood activation occurs in right frontal cortex during intentional declarative or episodic retrieval of memory for words (Schacter et al 1996a, Shallice et al 1994, Squire et al 1992, Tulving et al 1994), faces (Haxby et al 1996), scenes (Tulving et al 1996), or meaningless objects (Schacter et al 1995b). These robust activations were unexpected because they applied to verbal and nonverbal memories, and because right frontal lesions have modest effects on declarative memory. Further, it has been difficult to specify the nature of the retrieval conditions that yield right frontal activations. In some studies, the activations occur during memory judgments for old (studied) stimuli relative to new (unstudied) stim-

uli, and such activations are considered to reflect retrieval success (greater for old than new stimuli) (e.g. Rugg et al 1996, Tulving et al 1994). In other studies, they occur equally for well-remembered old stimuli, poorly remembered old stimuli, and new stimuli; this pattern of results is interpreted as reflecting retrieval attempt or mode that occurs irrespective of the memorial status of the stimulus (e.g. Kapur et al 1995, Nyberg et al 1995). In yet other studies, the activations appear slightly greater for poorly remembered than well-remembered information and are interpreted as indexing retrieval effort (Schacter et al 1996a).

One speculative interpretation is that right-frontal retrieval activations reflect working memory processes that guide or evaluate the products of episodic retrieval. If right-frontal activation were required for intentional retrieval, patients with right-frontal lesions would be globally amnesic because they would be unable to retrieve memories. To the contrary, deficits after right-frontal lesions are limited to more subtle impairments in strategic memory. For example, a right-frontal lesion can result in a propensity for false recognition under some, but not all, circumstances (Schacter et al 1996b). Thus, the degree of right-frontal activation during intentional retrieval may reflect the degree of strategic monitoring of memory retrieval. If the right-frontal activations reflect working with or reasoning about memory judgments, then such activations could vary considerably depending on what strategies are encouraged by particular retrieval conditions. Such an interpretation would posit a special role for right-frontal cortex in the working memory aspects of intentional retrieval.

SKILL LEARNING

In skill-learning tasks, subjects perform a challenging task on repeated trials in one or more sessions. The indirect or implicit measure of learning is the improvement in speed or accuracy achieved by a subject across trials and sessions. Preservation of sensorimotor, perceptual, and cognitive skill learning in amnesia indicates that such learning for some skills is not dependent upon declarative memory. Some of the neural systems underlying such skill learning have been identified in neuropyschological and neuroimaging studies.

Sensorimotor Skills

Intact sensorimotor skill learning in amnesia is well documented for three tasks: mirror tracing, rotary pursuit, and serial reaction time (SRT). In mirror tracing, subjects trace a figure with a stylus only seeing their hand, the stylus, and the figure reflected in a mirror. With practice, subjects trace the figure

more quickly and make fewer errors (departures from the figure). Such skill learning is intact in patients with declarative memory problems due to amnesia (Milner 1962) or AD (Gabrieli et al 1993). In rotary pursuit, subjects attempt to maintain contact between a hand-held stylus and a target metal disk, the size of a nickel, on a revolving turntable. With practice, subjects increase the time per trial that they are able to maintain contact with the disk. Rotary-pursuit skill learning is intact in amnesia (Corkin 1968) and in AD (Eslinger & Damasio 1986, Heindel et al 1989). In the SRT task, subjects see targets appear in one of four horizontal locations on a computer monitor and press one of four keys placed directly below those locations as soon as a target appears in the corresponding location. In the critical trials, unbeknown to subjects, targets appear in a repeating 10- or 12-trial sequence of locations. With practice, subjects perform more quickly, and pattern-specific skill learning is measured by a slowing in performance when the targets are presented in random locations. SRT learning is intact in amnesia (Nissen & Bullemer 1987) and intact in some but not all AD patients (Ferraro et al 1993, Knopman & Nissen 1987). Variability in AD performance may reflect dementia severity and perhaps specific impairment in spatial working memory.

Sensorimotor skill learning is often impaired in patients with basal ganglia diseases. Rotary-pursuit skill learning is impaired in HD patients (Gabrieli et al 1997c, Heindel et al 1989), GTS patients (Stebbins et al 1995), and, more variably, PD patients (Heindel et al 1989). SRT learning is impaired in HD patients (Willingham & Koroshetz 1993) and PD patients (Ferraro et al 1993). Basal ganglia diseases do not, however, have uniform effects on sensorimotor skill learning. In one study, HD patients showed a dissociation between impaired rotary-pursuit and intact mirror-tracing skill learning (Gabrieli et al 1997c). Cerebellar lesions, however, do impair mirror-tracing skill learning (Sanes et al 1990). In another study, HD patients showed a dissociation between impaired SRT skill learning and intact learning when subjects had to press a key one position to the right of the target (there was no repeating sequence) (Willingham & Koroshetz 1993). Further, HD patients showed a normal pattern of skill learning when tracking with a joystick a cursor that moved randomly, but impaired learning when the cursor moved in a repeating pattern (Willingham et al 1996).

Thus, the basal ganglia and cerebellum appear to make different contributions to sensorimotor skill learning. Two related hypotheses have been proposed about what differentiates those contributions. One hypothesis proposes that the learning of repetitive motor sequences depends upon the basal ganglia, whereas the learning of new mappings between visual cues and motor responses depends upon the cerebellum (Willingham et al 1996). Another hypothesis is that closed-loop skill learning, which involves continuous external,

visual feedback about errors in movement, depends upon the cerebellum. In contrast, open-loop skill learning, which involves the planning of movements and delayed feedback about errors, depends upon the basal ganglia (Gabrieli et al 1997c).

Functional neuroimaging studies have not only supported the importance of the basal ganglia and cerebellum in sensorimotor skill learning but have also shed light upon the importance of motor neocortex in such learning. Rotary-pursuit skill learning is associated with increases in activation of the primary and secondary motor cortices (Grafton et al 1992). SRT skill learning, and similar tasks involving the learning of specific manual sequences, are associated with increased activations in primary and secondary motor cortices and in the basal ganglia (e.g. Doyon et al 1996, Hazeltine et al 1997, Karni et al 1995). In some studies, there is a decrease in cerebellar activation associated with the learning of finger-movement sequences (Friston et al 1992). The possibility that cerebellar activity reflects error-correction, which would decrease as skill increases, is supported by a study finding a correlation between cerebellar activity and errors in a perceptual-motor task (Flament et al 1996). Imaging studies typically report complex patterns of increases and decreases in activation that reflect not only learning but also the changes in performance that occur with learning. These studies reveal that skill learning involves a complex, dynamic set of interactive neural networks.

Perceptual Skills

Learning to read mirror-reversed text is a perceptual skill that has been well studied in patients. Amnesic patients gain skill in reading such text at a normal rate, despite poor declarative memory for the particular words read or the episodes in which they gained their skill. In contrast, HD patients have mildly impaired mirror-reading skill learning despite relatively good declarative memory for words read and the reading experiences (Martone et al 1984).

An imaging study examined activation in posterior cortical areas as normal subjects gained skill in mirror reading (Poldrack et al 1996). As skill improved, activation increased in left inferior occipito-temporal cortex and decreased in right parietal cortex. These shifts in activity may represent a change in reliance upon visuospatial decoding of mirror-reversed words in unskilled performance to more direct reading in skilled performance. Such shifts from effortful to automatic neural networks occur also in conceptual task performance (Raichle et al 1994).

Cognitive Skills

Cognitive skills may be acquired normally by amnesic patients, but under relatively narrow circumstances. Amnesic patients have shown normal skill learn-

ing on Tower tasks that require planning and problem-solving under some circumstances (Cohen et al 1985, Saint-Cyr et al 1988), but not other circumstances (Butters et al 1985). Amnesic patients have also shown normal learning in the early but not later stages of probabilistic classification problems (Knowlton et al 1994). Cognitive skill learning, however, is impaired in HD and PD patients for Tower tasks (Saint-Cyr et al 1988) and probabilistic classification problems (Knowlton et al 1996a,b). Thus, at least some aspects of cognitive skill learning depend upon the integrity of the basal ganglia, but not upon the medial-temporal and diencephalic structures that support declarative memory.

The basal ganglia appear to be critical for a variety of motor, perceptual, and cognitive skills. These various skill learning deficits may reflect separable damage to distinct striatal-thalamic-cortical loops (Alexander et al 1986). There is evidence, for example, of a dissociation between motor and perceptual skill learning in PD patients (Harrington et al 1991). Each loop may mediate striatal-thalamic-cortical functions in separate motor or cognitive domains, but the loops may share a common abstract or computational property. For example, each loop may provide working memory modulation of domain-relevant cortices.

REPETITION PRIMING

Repetition priming refers to a change in the processing of a stimulus, usually words or pictures, due to prior exposure to the same or a related stimulus. In a typical experiment, participants process a set of stimuli in a study phase. In a subsequent test phase, participants perform a task with "old" stimuli identical or related to the study-phase stimuli and with "new" stimuli unrelated to the study-phase that provide a baseline measure of performance. The difference in performance with old and new stimuli constitutes the measure of repetition priming (hereafter referred to as priming).

One important distinction is that between perceptual priming, which reflects prior processing of stimulus form, and conceptual priming, which reflects prior processing of stimulus meaning (reviewed in Roediger & McDermott 1993). Perceptual priming occurs in visual, auditory, and tactual modalities. It is maximal when study-phase and test-stimuli are perceptually identical, and reduced when there is a study-test change in modality (e.g. from auditory to visual) or symbolic notation (e.g. from words to pictures). Priming has been characterized as perceptual for tasks such as identification of words presented at threshold, word-stem completion (e.g. complete STA into a word), word-fragment completion (e.g. what letters would make _ T _ M _ into a word), and picture naming. Conceptual priming is maximal when study-phase

processing enhances semantic analysis of stimulus meaning, and reduced when study-phase processing diminishes semantic analysis. Priming has been characterized as conceptual for word-association generation (what word goes with KING?) and category-exemplar generation (name FRUITS). Perceptual priming is often unaffected by the level of semantic analysis at study, whereas conceptual priming is often unaffected by study-test relations in perceptual form. Although many priming tasks are well characterized as predominately perceptual or conceptual in nature, there is a growing literature of priming tasks that are difficult to characterize in terms of the perceptual/conceptual dichotomy (e.g. Vaidya et al 1997a).

Repetition priming has been dissociated from declarative memory because of two convergent sources of evidence. First, amnesic patients exhibit normal magnitudes of priming on many tasks, including word identification (Cermak et al 1985), word-stem completion (Graf et al 1984, Warrington & Weiskrantz 1970), word-fragment-completion (Vaidya et al 1995), picture naming (Cave & Squire 1992, Verfaellie et al 1996), word-association generation (Shimamura & Squire 1984), and category-exemplar generation (Graf et al 1985). Second, parallel dissociations between these forms of priming and declarative memory have been obtained in normal subjects (Roediger & McDermott 1993). Two issues of interest are what constitutes the limits of priming in the absence of declarative memory and what neural networks mediate such priming.

Limits of Priming in Amnesia

PROCESSING/SYSTEM DEBATE Some investigators have hypothesized that amnesia is better characterized by a distinction between impaired conceptual and intact perceptual memory processes (e.g. Blaxton 1992) than one between impaired explicit and intact implicit retrieval modes. This hypothesis constitutes the core of the "processing-systems" debate. According to the "processing" view, amnesic patients are impaired on explicit memory tests not because these tests require intentional memory retrieval but because performance on these tests is conceptually driven. Amnesic patients show intact priming on word-identification, word-stem completion, and word-fragment completion because such priming reflects perceptual processes. Indeed, the normal status of perceptual priming in amnesia has been well documented as amnesic patients have shown normal reductions in cross-modal word-stem completion (Graf et al 1985), in cross-font word-stem completion (Vaidya et al 1997b), and in cross-exemplar picture naming (Cave & Squire 1992).

The processing and systems views make contradictory predictions about the status of conceptual priming and perceptually cued recall in amnesia. The processing view holds that conceptual memory processes are impaired and

perceptual memory processes are intact in amnesia regardless of the explicit or implicit nature of test-phase retrieval. The systems view posits that explicit retrieval is impaired and implicit retrieval is intact in amnesia regardless of the perceptual or conceptual nature of the test-phase retrieval. One study pitted these hypotheses against each other by using identical perceptual (word fragments) and conceptual (word associates) test-phase cues and varying explicit and implicit test-phase retrieval (Vaidya et al 1995). The results were clear: Amnesic patients showed intact perceptual and conceptual priming (implicit retrieval) and impaired perceptual and conceptual cued recall (explicit retrieval). Cermak et al 1995 report similar conclusions. Further, amnesic patients have shown normal insensitivity to modality manipulation (Vaidya et al 1995) and sensitivity to conceptual manipulation (Keane et al 1997) in their intact conceptual priming.

The hypothesis that explicit and implicit conceptual memory performance reflects a unitary process has now been controverted not only by findings in amnesia but also by similar dissociations in normal subjects (Vaidya et al 1997a), normal aging (Monti et al 1996), and schizophrenia (Schwartz et al 1993). The explicit/implicit distinction is superior to the perceptual/conceptual distinction for predicting amnesic performance, but it still cannot explain amnesic impairments on a variety of perceptual and conceptual priming tasks (e.g. Gabrieli et al 1994, Vaidya et al 1996, Schacter 1995a, Verfaellie et al 1996).

PRIMING FOR NOVEL STIMULI A second theoretical concern was spurred by early studies indicating that amnesic patients could show priming for familiar words known before the onset of amnesia but not for novel pseudowords (Cermak et al 1985, Diamond & Rozin 1984). These results were interpreted as indicating that priming in the absence of declarative memory was limited to the activation of premorbidly acquired memory representations. There is now, however, abundant evidence that amnesic patients can show normal priming for novel information, including nonverbal patterns (Gabrieli et al 1990, Knowlton & Squire 1993) and novel pronounceable (Haist et al 1991) or unpronounceable letter strings (Keane et al 1995). In retrospect, the earlier studies appear to have encouraged normal subjects to use explicit retrieval to support their test-phase performance, a source of support unavailable to amnesic patients. Thus, priming for novel verbal and nonverbal stimuli can occur in the absence of declarative memory processes.

PRIMING FOR NOVEL ASSOCIATIONS A related theoretical concern was whether amnesic patients could show priming for novel associations between unrelated stimuli. Priming for new associations may be measured by exposing participants to unrelated word pairs in a study phase (e.g. MARCH—SHAVE,

ABOVE—FLEET, AMAZE—VOTER). In a test phase, participants perform a task with three kinds of word pairs—1. Old pairs seen in the study phase (MARCH—SHAVE); 2. Recombined study-phase pairs (ABOVE—VOTER); and 3. New baseline pairs. Superior performance for Recombined relative to New pairs reflects single-word priming. Superior performance for Old relative to Recombined pairs must reflect new associations made between words by their arbitrary study-phase pairing because all words in Old and Recombined pairs were seen in the study phase.

Despite intact word-stem completion priming for single words, amnesic patients have failed to show normal associative priming for word-stem completion (e.g. being more likely to provide SHAVE when seeing MARCH—SHA____ than ABOVE—SHA____) (Cermak et al 1988, Schacter & Graf 1986). These findings raised the possibility that declarative memory was required for explicit and implicit associative memory processes. Amnesic patients, however, have shown normal associative priming on tasks of word identification (Gabrieli et al 1997b), reading time (Moscovitch et al 1986), and color-word naming (Musen & Squire 1993). It is unclear at present why some but not other forms of associative priming depend on the same brain structures and mental processes that mediate declarative memory. The preservation of some forms of associative priming in amnesia, however, provides a possible mechanism for intact priming for novel stimuli. Priming for novel pseudowords, for example, could reflect novel associations among the letters presented together in the pseudoword.

FLUENCY AND FAMILIARITY The foregoing discussion has emphasized dissociations between memory for stimuli as measured by explicit tests of recall or recognition or by implicit tests of repetition priming. Some processes, however, may be shared by explicit and implicit memory performance. It has been hypothesized that explicit retrieval and perceptual priming may share a common process of fluency and familiarity (Jacoby & Dallas 1981, Mandler 1980). By this view, prior perceptual processing of a stimulus makes more fluent the later reprocessing of that stimulus. Such fluency could mediate priming in implicit tests of word identification or word-stem completion. The same fluency could give rise to a sense of familiarity with a stimulus that contributes to explicit recognition memory performance.

These speculations became testable with the development of two methods aimed at dissociating the roles of conscious recollection and automatic familiarity in explicit recognition performance. The processes dissociation procedure uses inclusion and exclusion tasks that have recollection and familiarity working in concert or in opposition so that separate values for recollection and familiarity can be calculated (Jacoby 1991). This procedure, however, indi-

cates that both recollection and familiarity in explicit recognition are more influenced by conceptual than perceptual factors (Wagner et al 1997). Further, explicit recognition familiarity is intact in a patient with impaired visual priming on word-identification and stem-completion tasks (Wagner et al 1995). Thus, convergent behavioral and neurological evidence dissociates explicit recognition familiarity from perceptual priming.

A second method used to dissociate recollection from familiarity in explicit recognition memory is the remember/know procedure (Gardiner 1988). Subjects are asked to designate which items in a recognition test they "remember" from the study list (have a conscious recollection of the study event) and which items they "know" were on the study list but for which cannot explicitly recollect a study event. Amnesic patients were impaired on both "remember" and "know" responses on a recognition memory test (Knowlton & Squire 1995). Because amnesic patients have intact priming, it does not seem the same processes could underlie priming and "know" recognition responses. The aim of delineating processes that are shared by explicit and implicit retrieval remains an important one, but it does not appear that current methods used to isolate familiarity in explicit recognition are identifying the same processes that mediate perceptual priming.

Brain Systems Mediating Perceptual and Conceptual Priming

The above findings indicate that perceptual and conceptual priming do not depend upon the medial-temporal and diencephalic structures that mediate declarative memory. HD patients show intact priming (Heindel et al 1989), so priming is also not dependent upon basal ganglia structures critical for skill learning. What neural systems mediate priming?

Several lines of evidence indicate that priming is mediated by neocortical areas, with perceptual priming being mediated by modality-specific cortical regions and conceptual priming by amodal language areas. One source of evidence is the performance of AD patients who exhibit severely reduced conceptual priming (Monti et al 1996) but intact perceptual priming on visual tasks (Fleischman et al 1995; Keane et al 1991, 1995). This pattern of impaired conceptual and intact perceptual priming may be interpreted in terms of the characteristic neocortical neuropathology in AD. In vivo metabolic imaging studies (e.g. Frackowiak et al 1981) and postmortem studies of late-stage AD patients (Brun & Englund 1981) find substantial damage to association neocortices in the frontal, parietal, and temporal lobes but relatively little compromise of primary visual, somatosensory, auditory, and motor cortices, the basal ganglia, or the cerebellum. The sparing of the basal ganglia and cerebellum may account for intact rotary-pursuit and mirror-tracing in AD. The sparing of modality-specific cortices and the compromise of association cortices may ac-

count, respectively, for intact perceptual and impaired conceptual priming.

There is more direct evidence that modality-specific neocortex mediates modality-specific perceptual priming. Patients with right occipital lesions have shown an absence of priming on visual word-identification tasks, and of modality and font visual specificity on word-stem completion priming (Fleischman et al 1995, Gabrieli et al 1995b, Keane et al 1995, Vaidya et al 1997b). These patients demonstrate intact performance on explicit tests of recall and recognition and on implicit tests of conceptual priming. Thus, these patients provide two double dissociations: in comparison with amnesia between visual implicit and explicit memory for words, and in comparison with AD between perceptual and conceptual priming.

Neuroimaging studies also indicate that separate cortical areas mediate perceptual and conceptual priming. Priming on visual word-stem completion tasks is associated with reduced activity, relative to baseline word-stem completion, in bilateral occipito-temporal regions (Schacter et al 1996a, Squire et al 1992). Priming on conceptual tasks is associated with reduced activity in left frontal neocortex on tasks involving abstract/concrete decisions about words (Demb et al 1995, Gabrieli et al 1996b), living/nonliving decisions about words and pictures (Wagner et al 1997), generation of verbs to nouns (Raichle et al 1994), and generation of semantically related words (Blaxton et al 1996). Amnesic patients, who show normal priming when making abstract/concrete decisions about words, also show a priming-related reduction in left frontal cortex (Gabrieli et al 1996b).

Thus, lesion and imaging studies provide convergent evidence that different forms of priming reflect process-specific plasticity in separate neocortical regions. It is hypothesized that auditory and tactual priming will be mediated by changes in auditory and somatosensory neocortices. Lexical and semantic priming may reflect changes in association areas of the frontal and temporal lobes. Thus, repetition priming in a given domain appears to reflect experience-induced changes in the same neural networks that subserved initial processing in that domain (Gabrieli et al 1996a, Raichle et al 1994). These changes facilitate or bias the subsequent reprocessing of the stimuli. The enhanced efficiency of reprocessing may diminish computational demands and thus lead to reduced activations relative to baseline conditions.

Earlier, the cortical geography of semantic memory was reviewed. It may be hypothesized that perceptual, lexical, and semantic knowledge systems must be constantly molded by experience to enhance efficiency for identifying objects and words and for using concepts. Repetition priming, psychological domain by psychological domain, and cortical area by cortical area may be revealing how experience constantly tunes the representation of perceptual and conceptual knowledge.

CONDITIONING

The neural circuitry underlying classical and other forms of conditioning has been studied extensively in rabbits and rats. Parallel studies have now been conducted systematically in humans, and a question of interest is whether the same memory systems mediate conditioning across these mammalian species.

Delay Conditioning

The memory system underlying classical delay eyeblink conditioning has been delineated with great precision in the rabbit (Thompson 1990). In the typical delay paradigm, a 250–500 ms tone (conditioned stimulus or CS) is repeatedly followed by an air-puff (unconditioned stimulus or US) delivered to the eye that elicits reflexively a blink, the unconditioned response (UR). The tone and air-puff coterminate. With repeated CS-US pairings, subjects learn to associate the tone with the air-puff and initiate an eyeblink (conditioned response or CR) in response to the CS before the onset of the US. In the rabbit, electrophysiological activity in the cerebellum (McCormick & Thompson 1987) and in the hippocampus (Disterhoft et al 1986) parallels the development of behavioral CRs. The convergence of CS and US projections in eyeblink conditioning occurs in the cerebellum ipsilateral to the eye receiving the air-puff. Lesions of the cerebellar dentate-interpositus nuclei prevent acquisition or abolish retention of the conditioned association. Hippocampal lesions, however, do not impair delay conditioning in the rabbit (Schmaltz & Theios 1972). Presumably, CR-correlated electrophysiological activity in the hippocampus reflects a parallel learning circuit that does not mediate delay conditioning.

Results with human beings provide three striking parallels with animal findings. First, cerebellar lesions in human beings abolish delay eyeblink conditioning (Daum et al 1993). Second, delay eyeblink conditioning is intact in amnesic patients with bilateral medial-temporal (Gabrieli et al 1995a) or bilateral thalamic lesions (Daum & Ackermann 1994). Such conditioning is somewhat impaired in HM (Woodruff-Pak 1993) and greatly impaired in alcoholic Korsakoff's patients (McGlinchey-Berroth et al 1995), but these deficits appear to reflect cerebellar damage due to chronic exposure to anticonvulsant medications or alcohol, respectively. Third, PET studies have reported both cerebellar and medial-temporal activations associated with delay conditioning that parallel the development of behavioral CRs (Blaxton et al 1996, Logan & Grafton 1995).

Delay eyeblink conditioning is not diminished by the basal ganglia lesions in HD (Woodruff-Pak & Papka 1996). It does diminish across the normal adult life span (Woodruff-Pak & Thompson 1988) and is virtually abolished in AD (Woodruff-Pak et al 1990). The brain basis for diminished delay eyeblink con-

ditioning in aging or in AD is unknown. Such conditioning does not depend upon either the medial-temporal structures critical for declarative memory or the striatal structures critical for many forms of skill learning.

Trace and Discrimination Reversal Conditioning

The hippocampal activation evident in human and animal recordings during delay eyeblink conditioning does not appear to be essential for such learning but it may reflect correlated learning that is essential for other forms of conditioning. In animals, medial-temporal lesions impair trace eyeblink conditioning, which differs from delay conditioning in that there is a short time period—a second or less—between the offset of the CS and the onset of the US (Solomon et al 1986). Amnesic patients with medial-temporal lesions who are unimpaired on delay conditioning show impaired trace conditioning with CS-US trace intervals as short as 500 ms (McGlinchey-Berroth et al 1997). In animals, medial-temporal lesions also impair discrimination reversal, in which the two CSs are switched in their association with the US (Berger & Orr 1983). Amnesic patients with medial-temporal lesions also have impaired conditioning for discrimination reversal (Daum et al 1989). These findings suggest that the same medial-temporal lobe structures that are essential for declarative memory also mediate processes required for more complex forms of conditioning in human beings as they do in rabbits.

Fear Conditioning

The critical role of the amygdala in fear conditioning to aversive stimuli such as electric shocks has been well established in rats (Davis et al 1987). Two studies have shown now that amygdala damage impairs fear conditioning in humans. In both studies, participants were exposed to pairings of initially neutral conditioned visual stimuli (CS) preceding aversive unconditioned auditory stimuli (US), white-noise or boat-horn bursts, which elicited an unconditioned response measured as a change in skin conductance response (SCR). Over multiple trials, normal participants showed fear conditioning by making conditioned SCRs to the CS. An Urbach-Weithe patient (Bechara et al 1995) and patients with amygdala resections (LaBar et al 1995) showed little or no fear conditioning. The fear-conditioning deficit was dissociated from declarative memory because the patients had excellent declarative memory for the experimental experience (e.g. for the stimuli). In contrast, amnesic patients without amygdala damage demonstrated intact fear conditioning but impaired declarative memory for their experimental experience (Bechara et al 1995). Thus, the critical role of the amygdala in fear conditioning appears to be conserved in the human brain.

PERSPECTIVE

The emergence of functional neuroimaging techniques offers unprecedented opportunities to discover how the brain learns and remembers. Understanding of the brain organization of memory had heretofore relied on the coincidence of brain injuries, and scientists prepared to understand the significance of the memory failures that followed. This path to knowledge took us a long way. We learned about the critical role of medial-temporal and diencephalic structures in declarative memory, the amygdala in emotional modulation of memory, the basal ganglia in skill learning, the cerebellum in conditioning, and the neocortex in repetition priming. In some cases, studies of human lesions guided parallel lesion research in animals (e.g. medial-temporal and diencephalic lesions), and in other cases animal lesions guided research in patients (e.g. amygdala, cerebellum).

It may be thought that the greater freedom of neuroimaging studies, where systematic experiments can be performed on many normal subjects, will render lesion studies obsolete. This thought ignores how much psychological interpretation is required to comprehend the significance of neuroimaging activations (in addition to a host of neurobiological, image analysis, and statistical issues). With imaging studies alone, it might have been concluded that (*a*) global amnesia would follow left or right frontal lesions because they would prevent the encoding or retrieval of new memories; (*b*) the hippocampus is not important for declarative memory because it often was not active during explicit retrieval (e.g. Shallice et al 1994, Tulving et al 1994); and (*c*) the hippocampus is critical for delay conditioning. Each of these conclusions would have been wrong. Thus, animal lesion, human lesion, and imaging studies will provide powerful sources of mutual constraints for a long time to come.

There is, however, a turning of the wheel in the cognitive neuroscience of human memory. For nearly a quarter of a century, our understanding of the normal brain organization of memory depended upon studies of diseased memory. Now, functional neuroimaging studies of healthy brains can begin to illuminate how and why injuries to specific memory systems result in various diseases of memory.

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