Proceedings of ICSPHS 2027 Symposium: Psychology, Physical Activity, and Student Wellbeing
DOI: 10.54254/2753-7048/2027.KL38313

Memory System, Processing Stages and Neural Mechanisms: A Review of Normal Function and Disorder Patterns

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Abstract. Human memory is a collection of interacting systems that differ in the type of information they store, the timescale over which they operate, and the brain structures that support them. Despite decades of research, questions remain about how cleanly memory systems and processing stages map onto specific neural substrates, and about what memory disorders can reveal about the healthy architecture of memory. This review synthesizes literature on memory classification (sensory, short-term, and long-term memory), on the sequential processes of encoding, consolidation, and storage/retrieval, and on the neural mechanisms that underline these systems, including the medial temporal lobe, prefrontal cortex, basal ganglia, cerebellum, and amygdala. It then examines four common patterns of memory disorder—anterograde and retrograde amnesia, Alzheimer's disease, working-memory impairment as seen in attention-deficit/hyperactivity disorder (ADHD), and emotional-memory disturbance as seen in post-traumatic stress disorder (PTSD)—as case studies of how damage to specific circuits produces selective, rather than global, memory impairment. The review is organized as a narrative synthesis of experimental, clinical, and neuroimaging findings rather than a new empirical study. Its central question is whether distinct memory systems and processing stages correspond to dissociable brain regions, and whether disorders of memory are best understood as exaggerations or breakdowns of these normal mechanisms.

Keywords: memory systems, hippocampus, working memory, memory disorders

1. Introduction

Memory research has moved, over the past half-century, from treating memory as a unitary store to recognizing it as a family of dissociable systems supported by different brain circuits [1]. Work with amnesic patients and later, functional neuroimaging established that the capacity to consciously recollect facts and events (declarative memory) can be selectively damaged while the capacity to acquire skills and habits (non-declarative memory) remains intact, and that immediate retention and long-term storage rely on partially separable mechanisms [1]. Despite this progress, gaps remain. First, most systems-level accounts of memory were developed from a combination of animal lesion studies and rare human cases, so the mapping between specific processing stages—encoding, consolidation, storage, and retrieval—and specific brain regions is still debated. Second, disorders that disrupt memory, such as amnesia, dementia, ADHD and PTSD, are often studied as separate

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clinical literatures, which can obscure what they jointly reveal about the organization of normal memory? This review addresses two related questions: (1) how are the major memory systems and processing stages currently understood to be organized, and (2) how do characteristic patterns of memory disorder map onto, and thereby clarify, the neural mechanisms of normal memory. The review uses a narrative synthesis of foundational and contemporary literature drawn from cognitive psychology, neuropsychology, and clinical neuroscience. Clarifying these relationships is intended to provide a consolidated reference for students and clinicians, to highlight where the evidence is strong versus provisional, and to suggest directions, such as more integrative, transdiagnostic models of memory dysfunction, for future research.

2. Memory system

2.1. Sensory memory

Sensory memory is the initial, very brief stage in which large amounts of raw sensory information are held in a modality-specific format immediately after stimulation ends, commonly divided into iconic (visual) and echoic (auditory) memory. Its capacity is comparatively large, but its duration is on the order of a few hundred milliseconds to a few seconds, and unattended information decays quickly without being encoded further [1]. Sensory memory therefore functions as a buffer that gives the perceptual system enough time to select which incoming information is relevant and pass it on for further processing, it is generally considered pre-attentive and outside conscious control.

2.2. Short-term memory

Short-term memory holds a small amount of information—classically estimated at about seven items—for a period of seconds to roughly a minute without rehearsal. Contemporary accounts have largely reframed short-term storage as "working memory," an active system that not only holds information but manipulates it in the service of ongoing tasks. Baddeley's multicomponent model describes a central executive that coordinates attention alongside two subsidiary stores—the phonological loop for verbal material and the visuospatial sketchpad for visual and spatial material—together with an episodic buffer that integrates information from these subsystems with long-term memory into a unified, temporary representation [2]. This model helps explain why verbal and visuospatial tasks can be performed simultaneously with little interference, yet compete when both draw on the same subsystem.

2.3. Long-term memory

Long-term memory refers to the relatively permanent storage of information after it has left short-term or working memory, with a capacity that is, for practical purposes, unlimited. It is conventionally divided into declarative (explicit) memory—knowledge that can be consciously recollected and described, including episodic memory for personally experienced events and semantic memory for general facts—and non-declarative (implicit) memory, an umbrella term for procedural skills, priming, and simple conditioned responses that are expressed through performance rather than conscious recall [1]. This declarative/non-declarative distinction, first motivated by dissociations observed in amnesic patients, remains the most widely used framework for classifying long-term memory and provides the organizing logic for the neural mechanisms discussed in Section 4.

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3. Memory processes

3.1. Encoding

Encoding is the process by which incoming information is transformed into a representation that can be stored. The depth and elaborateness of encoding—for example, relating new material to existing knowledge or forming multiple associative links—strongly predicts how well that material will later be retrieved. Encoding is not a passive registration of stimuli but an active, selective process shaped by attention, prior knowledge, and the intended use of the information.

3.2. Consolidation

Consolidation is the process by which an initially labile memory trace is stabilized into a more permanent form. McGaugh's synthesis of a century of consolidation research describes this as a time-dependent process: for a period after learning, memories remain vulnerable to disruption, for example, by interference, brain injury, or pharmacological intervention, and hormonal systems—particularly the stress hormones released during emotionally arousing events—can modulate how strongly a memory is consolidated [3]. Consolidation is now understood to operate at two levels: rapid synaptic consolidation occurring within the hippocampus over minutes to hours, and slower systems-level consolidation occurring over days to years, during which memories are thought to become gradually less dependent on the hippocampus and more reliant on distributed neocortical networks.

3.3. Storage and retrieval

Storage refers to the maintenance of consolidated information over time, while retrieval is the process of accessing and reconstructing that information when it is needed. Retrieval is not a simple readout of a fixed trace, it is reconstructive and can itself return a memory to a labile state, making it once again susceptible to modification, a phenomenon studied under the heading of reconsolidation. The efficiency of retrieval depends heavily on the match between the cues present at retrieval and the context present at encoding, which is part of why forgetting is often better characterized as a failure of access rather than a true loss of the stored trace.

4. Neural mechanisms of memory

4.1. The medial temporal lobe and hippocampus in declarative memory

The clearest evidence for a discrete declarative memory system comes from patient H.M., who underwent bilateral removal of the hippocampus and surrounding medial temporal lobe structures to control severe epilepsy and subsequently developed a profound and persistent inability to form new declarative memories despite intact intelligence, perception, and short-term memory [4]. Building on such cases, Squire and Zola-Morgan characterized the medial temporal lobe memory system as comprising the hippocampus together with the adjacent entorhinal, perirhinal, and parahippocampal cortices, which work together to bind the distributed cortical representations of an experience into a single, retrievable episodic memory [5]. Damage confined to this system produces anterograde amnesia and a temporally graded retrograde amnesia while sparing non-declarative learning, illustrating that the medial temporal lobe is necessary for the initial formation of declarative memory rather than for its permanent storage.

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4.2. The prefrontal cortex in working memory and retrieval control

The prefrontal cortex, particularly its dorsolateral region, is central to the active maintenance and manipulation of information in working memory. Goldman-Rakic's studies of primates showed that neurons in the dorsolateral prefrontal cortex sustain elevated, stimulus-specific firing across a delay period, providing a plausible cellular mechanism for holding information "in mind" in the absence of external stimulation [6]. Beyond maintenance, the prefrontal cortex also contributes to strategic retrieval from long-term memory by organizing search strategies, monitoring the accuracy of retrieved information, and suppressing irrelevant or competing memories, so that damage to this region tends to produce difficulties in the effortful, self-directed use of memory rather than a loss of stored content itself.

4.3. The basal ganglia and cerebellum in procedural memory

Procedural memory, the gradual, trial-and-error acquisition of skills and habits, depends on circuits largely distinct from the medial temporal lobe system. Packard and Knowlton's review of the basal ganglia describes how the dorsal striatum supports the incremental learning of stimulus-response associations, producing behavior that becomes rigid, automatic, and relatively insensitive to changes in the value of the outcome, in contrast to the flexible, context-sensitive nature of declarative memory [7]. Patients with basal ganglia dysfunction, such as those with Parkinson's disease, show impaired habit learning while their declarative memory can remain comparatively intact, and the reverse dissociation is seen in amnesic patients with hippocampal damage, who can acquire new motor and perceptual skills normally despite being unable to recall the training episodes themselves. The cerebellum complements this system by supporting the precise timing and refinement of learned movements.

4.4. The amygdala and emotional memory

The amygdala does not store memories itself so much as modulate how strongly other systems, particularly the hippocampus, encode and consolidate emotionally significant experiences. LaBar and Cabeza's review describes how the amygdala interacts with the hippocampus and prefrontal cortex at multiple stages of processing—encoding, consolidation, and later retrieval—to produce the well-documented advantage in long-term memory for emotionally arousing material relative to neutral material [8]. This modulatory role depends substantially on stress-hormone systems, consistent with McGaugh's consolidation framework [3], and helps explain both the vividness of emotional memories in healthy individuals and, as discussed below, the intrusive, poorly regulated emotional memories characteristic of PTSD.

5. Memory disorder patterns

5.1. Anterograde and retrograde amnesia

Anterograde amnesia is the inability to form new long-term memories after the onset of brain damage, while retrograde amnesia is the loss of memories formed before the damage occurred, the two frequently co-occur following medial temporal lobe injury, as in the case of H.M. [4]. Retrograde amnesia is typically temporally graded, with older memories better preserved than more recent ones, a pattern consistent with the view that memories become gradually independent of the hippocampus as systems-level consolidation transfers them to neocortical storage [3, 5]. This

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dissociation between old and recent memories, and between declarative and non-declarative learning, has been one of the principal sources of evidence for the existence of multiple memory systems.

5.2. Alzheimer's disease

Alzheimer's disease typically begins with a selective impairment of episodic memory that is disproportionate to other cognitive changes, reflecting the early and pronounced pathology in the hippocampus and entorhinal cortex. Di Paola and colleagues used voxel-based morphometry to show that the severity of episodic memory impairment in Alzheimer's disease patients correlates specifically with the degree of entorhinal cortex atrophy, consistent with the entorhinal cortex's role as the principal gateway of neocortical input into the hippocampal memory system [9]. As the disease progresses and pathology spreads beyond the medial temporal lobe, semantic memory and eventually non-declarative abilities also become affected, producing a pattern that broadly mirrors, in slow motion, the systems-level organization described in Section 4.

5.3. Working memory disorder

Attention-deficit/hyperactivity disorder is not classically defined as a memory disorder, but working-memory impairment is one of its most consistent cognitive correlates. Barkley's influential model proposes that ADHD reflects a core deficit in behavioral inhibition that secondarily disrupts several executive functions dependent on inhibition, including working memory, and that these deficits are linked to atypical prefrontal cortex function [10]. Because working memory depends on sustained, top-down prefrontal control over the maintenance of information, disruption of inhibitory control can manifest as difficulty holding goal-relevant information in mind while ignoring distraction, illustrating how a deficit that is not primarily "about memory" can nonetheless produce a memory-like impairment through its effects on prefrontal function.

5.4. Emotional memory disorder

Post-traumatic stress disorder can be understood as a disorder of emotional memory regulation rather than of memory storage capacity per se. Neuroimaging models reviewed by Rauch, Shin, and Phelps describe PTSD in terms of an amygdala that shows exaggerated reactivity to trauma-related and fear-related cues, paired with insufficient regulatory input from the medial prefrontal cortex and reduced hippocampal engagement during extinction learning, which together impair the ability to update or extinguish a traumatic memory once it has been formed [11]. This pattern is, in effect, an exaggeration of the normal amygdala-mediated enhancement of emotional memory described in Section 4.4 [8]: the same mechanism that makes emotionally significant events more memorable in general appears to become dysregulated in PTSD, producing intrusive, vivid, and poorly contextualized traumatic memories.

6. Conclusion

This review has organized human memory along two complementary dimensions: the systems that store information (sensory, short-term/working, and long-term declarative and non-declarative memory) and the processes that act on that information over time (encoding, consolidation, storage, and retrieval). Bringing these together with the neuroscience literature suggests a reasonably consistent, though not perfectly modular, mapping between memory systems and brain structures:

DOI: 10.54254/2753-7048/2027.KL38313

the medial temporal lobe and hippocampus for the initial formation of declarative memory, the prefrontal cortex for the active maintenance and strategic retrieval of information, the basal ganglia and cerebellum for procedural and habit learning, and the amygdala for the emotional modulation of memory strength. The four disorder patterns examined here—anterograde/retrograde amnesia, Alzheimer's disease, ADHD-related working-memory impairment, and PTSD-related emotional-memory dysregulation—support this framework by showing that damage or dysfunction confined to one circuit tends to produce selective rather than global memory impairment, which is itself strong evidence that memory is organized as multiple, partially independent systems rather than a single unitary faculty.

At the same time, this review has several limitations. It relies substantially on classic, foundational studies and on a small number of representative disorders, so it does not capture the full diversity of memory-related conditions, such as transient global amnesia, dissociative amnesia, or the memory changes associated with normal aging, nor the more recent molecular and computational literature on synaptic plasticity and memory engrams. The reliance on narrative synthesis rather than systematic or meta-analytic methods also means that the selection of sources, while illustrative, is not exhaustive. Future research would benefit from more integrative, transdiagnostic models that examine how the systems described here interact, for instance, how prefrontal and amygdala dysfunction jointly shape memory in trauma-related disorders, and from longitudinal and computational work that traces how memory representations change as consolidation proceeds from the hippocampus to distributed cortical networks over months and years. Such work would help move the field from a set of largely separate systems-level and disorder-specific literatures toward a more unified account of how memory is built, maintained, and, in illness, selectively broken.

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