

Non-Print Items

Abstract:

Amnesia is a neurobehavioral syndrome characterized by a selective impairment in memory in the context of preserved intelligence and other cognitive abilities. Permanent amnesia can be caused by a range of etiologies that damage neural structures in the limbic system or the basal forebrain. Amnesic patients are impaired at acquiring new information (anterograde amnesia) and can also be impaired at retrieving information acquired prior to the onset of amnesia (retrograde amnesia). The clinical features of anterograde and retrograde amnesia are an important source of information about the cognitive and neural architecture of memory systems.

Keywords: Anterograde amnesia; Declarative memory; Episodic memory; Hippocampus; Medial temporal lobe; Retrograde amnesia; Semantic memory

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Amnesia and the Brain

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[Au4]	Glossary	Episodic memory Memory for personally experienced events or 'episodes' that are specific in time and place.	dt0035
dt0010	Amnesia Pathological memory loss resulting from brain injury, illness, or psychological disturbance.	Limbic system System of brain structures associated with memory function, including the medial temporal lobes, diencephalon, mammillary bodies, and fornix.	dt0040
dt0015	Anterograde amnesia Impaired memory for facts or events encountered after the onset of amnesia (postmorbid memory loss).	Medial temporal lobes Medial region of the temporal lobes associated with memory function. Subregions include the hippocampus, parahippocampal gyrus, entorhinal cortex, and perirhinal cortex.	dt0045
dt0020	Confabulation Falsification of memory without the intent to mislead.	Nondeclarative memory Memory expressed through behavioral performance without conscious memory content. Includes classical conditioning, skill learning, and repetition priming.	dt0050
dt0025	Consolidation Process by which information is assimilated and stabilized into memory. Consolidation includes both fast processes operating at the cellular level and slow processes operating at the systems level.	Retrograde amnesia Impaired memory for information acquired prior to the onset of amnesia (premorbid memory loss).	dt0055
dt0030	Declarative memory A form of memory characterized by the ability to consciously bring to mind facts and events. Declarative memory is dependent on the integrity of the limbic system. Episodic memory and semantic memory are both forms of declarative memory.	Semantic memory Memory for general knowledge (facts or concepts) that is impersonal and context-free.	dt0060

s0010 The Amnesic Syndrome

p0010 Amnesia can be defined as a special case of memory loss that is distinct from ordinary forgetting. The amnesic syndrome is characterized by a profound disorder of memory in the context of preserved general intelligence and other cognitive abilities such as language, perception, and attention. Permanent amnesia can be differentiated from more common forms of memory loss, such as memory loss associated with aging or depression. In particular, amnesia is characterized by anterograde memory loss, in which patients cannot remember events that occur after the onset of amnesia. Retrograde amnesia, in which patients cannot remember experiences or information acquired before the onset of the illness, is also commonly observed, though the extent of retrograde amnesia is more variable. Occasionally patients present with focal retrograde amnesia, but this presentation has been argued to be psychogenic in nature.

p0015 The most widely studied clinical case of amnesia is patient H.M. In 1953, H.M. underwent surgery in which his medial temporal lobes (MTLs) were bilaterally resected to relieve medically intractable epileptic seizures. Though the surgery succeeded in alleviating his epileptic symptoms, H.M. immediately showed profound anterograde amnesia that persisted until his death in 2008. In the 55 years following his surgery, H.M. demonstrated a remarkable inability to learn and intentionally retrieve new information. His memory impairment was evident in many types of memory tests (e.g., cued recall, free recall, and recognition) across multiple domains of information (including verbal, visual, auditory, and somatosensory). In everyday

life, H.M. was unable to recall conversations that he had with a visitor 30 min ago, or the fact that he had a visitor at all. He could not acquire new information about salient life events occurring after his surgery, such as the death of his father, and he also had difficulty recalling premorbid memories, with most of his personal memories deriving from the age of 16 or earlier. Interestingly, despite these severe memory problems, H.M.'s IQ was above average (118 on the Wechsler Adult Intelligence Scale) and he performed normally on standard tests of perception, short-term memory, and language comprehension. The specificity of H.M.'s impairment to long-term memory provided strong evidence that the MTL is critical for normal human memory function.

p0020 While amnesia can also have a psychogenic origin, the focus of this article is on organic amnesia caused by nonprogressive brain damage to the MTL and related limbic system structures. The laterality of damage to the limbic system results in modality-specific memory impairments, with damage to left-lateralized limbic structures producing primarily impaired verbal memory (such as story recall and list learning) while damage to right-lateralized limbic structures produces impaired visual memory (such as memory for figures or designs) and sometimes, spatial memory. While left-sided lesions seem to produce more functionally significant memory problems in humans, likely due to the importance of verbal memory and language to human behavior, bilateral lesions are required for permanent global amnesia. A range of vascular, infectious, and traumatic processes can cause damage to the limbic system. The following section describes the most common etiologies of permanent amnesia.

s0015 **Etiologies of Amnesia**

s0020 **Herpes Simplex Encephalitis**

p0025 Herpes simplex encephalitis (HSE) is a viral infection that causes hemorrhagic lesions in the brain. Neural damage due to HSE typically occurs in MTL regions, including the hippocampus, entorhinal cortex, perirhinal cortex and parahippocampal cortex. Damage can also extend into the insular cortex, basal forebrain, and anterolateral and inferior temporal lobe regions. Anterograde amnesia is a prominent feature of HSE and can be accompanied by aphasia and agnosia when neural damage includes the anterolateral and inferior temporal lobes. In addition, lesions extending into lateral temporal regions can result in extensive retrograde amnesia.

p0030 Patient S.S. provides a striking demonstration of the memory impairments following HSE. Patient S.S. became densely amnesic in 1971 after developing HSE that caused bilateral lesions in anterolateral and medial portions of his temporal lobes, as well as lesions in his insula and putamen. S.S. has not been able to form any new declarative memories since the time of his illness. He cannot retain information regarding significant family matters or recent public events, and he cannot acquire new general knowledge such as vocabulary introduced into the English language since the onset of his illness. S.S. also has extensive retrograde amnesia for autobiographical and personal semantic information that includes most of his adult life. Despite these severe memory impairments, S.S. is of superior intelligence and performs well on tasks of short-term memory, language, executive function, and deductive reasoning skills.

s0025 **Anoxia**

p0035 Anoxic injury results from a loss of oxygen to the brain and can be caused by a variety of conditions such as cardiac arrest, respiratory distress, or carbon monoxide poisoning. Certain brain regions, such as the hippocampus, are particularly vulnerable to anoxic injury due to their physical location and biochemical makeup. Selective memory impairment following anoxia can vary from mild to severe, depending on the extent of MTL damage, and can include both anterograde and retrograde amnesia. Anoxic brain damage that extends outside the MTL to regions such as the basal ganglia, thalamus, white matter, or other neocortical regions can affect additional cognitive and perceptual abilities. In addition, anoxic injuries occurring shortly after birth can cause bilateral hippocampal atrophy leading to developmental amnesia that presents as impaired learning and retention in the face of preserved attention, reasoning, and visuospatial skills.

s0030 **Stroke**

p0040 Stroke can produce amnesia by blocking blood supply to various components of the limbic system, including limbic system white matter. Stroke involving the bilateral posterior cerebral artery (PCA) can cause lesions in the posterior hippocampus, parahippocampal gyrus, and occipitotemporal gyrus and can result in a dense amnesic syndrome. Anterograde amnesia following bilateral PCA stroke can include impairment in verbal recall and recognition as well as in visuospatial memory.

Patients can also present with retrograde amnesia. Memory problems can also occur following left-lateralized PCA lesions, and can affect verbal and visual information either transiently or permanently.

Stroke can also cause amnesia by damaging tissue in the thalamus. Strokes affecting blood supply to the anterior or medial regions of the thalamus most commonly occur due to small vessel occlusive disease or hypertensive hemorrhages and can damage the anterior thalamic nuclei as well as thalamo-frontal connections. Executive dysfunction frequently accompanies memory impairments, and patients often display worse performance on recall than on recognition tests. The extent and persistence of retrograde amnesia is variable.

Several features of stroke make it an important source of information about the nature of memory impairments in amnesia. First, there are usually no preillness neurological issues to complicate interpretation of the effects of stroke. Second, the abrupt onset of stroke avoids concerns about adaptive changes in the brain before initial assessment. Finally, stroke generates relatively discrete lesion boundaries without co-occurring diffuse injury, enabling regional effects to be mapped with greater specificity.

Wernicke–Korsakoff Syndrome

Wernicke–Korsakoff syndrome (WKS) is caused by thiamine deficiency and is most commonly observed as an effect of alcohol abuse in populations over 40 years of age. Because thiamine helps to produce energy needed for proper neuronal function, insufficient thiamine can cause neuronal damage or death. The thalamus and mammillary bodies are particularly sensitive to damage due to thiamine deficiency.

Diagnosis of WKS is given when an individual with a history of chronic, heavy drinking or malnutrition presents with anterograde amnesia. This diagnosis can be supported by neuroimaging or autopsy findings showing degeneration of the thalamus and mammillary bodies and loss of brain volume around the fourth ventricle. In the acute phase, patients exhibit a confused state, eye movement disturbances, and ataxia. In the chronic phase, patients exhibit dense anterograde amnesia that is attributed to neural damage in the anterior and dorsomedial nuclei of the thalamus and mammillothalamic tract. Confabulation and source memory errors can also occur in WKS and are thought to reflect additional frontal dysfunction. In addition to anterograde amnesia, many WKS patients also have profound retrograde amnesia that is characterized by a temporal gradient for autobiographical information (i.e., greater impairment for recent compared to remote personal memories). However, interpretation of retrograde amnesia is complicated by pre-morbid lifestyle characteristics and frontal dysfunction that may interfere with memory encoding and retrieval.

Anterior Communicating Artery Aneurysm

Rupture of an anterior communicating artery (ACoA) aneurysm can cause lesions in the basal forebrain, which are the origin of the cholinergic pathways to the hippocampus, the striatum, and the frontal lobes. Amnesia with ACoA aneurysm is likely due to disruption of hippocampal function, but controversy remains about the specific role of other lesioned

regions. Lesions in the basal forebrain are usually bilateral and if damage includes the basal forebrain, striatum, and frontal lobes, the resultant amnesia is severe and persistent.

p0070 Approximately 18% of ACoA aneurysm survivors will have some form of cognitive deficit, of which severe amnesia is the most common. Anterograde amnesia is characterized by delayed recall deficits for verbal and nonverbal material and memory recall is worse than recognition. Severe retrograde amnesia is also common, but is not universally observed. The clinical presentation of patients suffering from ACoA aneurysm can be complicated by significant executive deficits and prominent confabulation. Of all the etiologies of amnesia, patients with ACoA aneurysm are the most likely to be unaware of their memory deficits, which can cause safety and behavioral problems.

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s0045 **Information-Processing Descriptions of Amnesia**

p0075 Though functional heterogeneity exists amongst the different etiologies of amnesia, comparison across cases with different lesion locations and associated deficits has enabled identification of the core information-processing deficits in amnesia. The following sections describe the core information-processing characteristics of anterograde and retrograde amnesia.

s0050 **Anterograde Amnesia**

p0080 Anterograde amnesia includes impaired memory for personally experienced events (episodic memory) as well as impersonal facts and concepts (semantic memory). Together, these forms of memory are categorized as declarative (explicit) memory and can be distinguished from nondeclarative (implicit) forms of memory that are largely preserved in amnesia. To demonstrate the distinction between impaired declarative and preserved nondeclarative memory in amnesia, consider the example of learning to ride a bicycle. While amnesic patients may not be able to recall specific instances when they have ridden a bicycle in the past (impaired declarative memory), they can nonetheless demonstrate the motoric skill of riding a bicycle (preserved nondeclarative memory). This example reflects one form of nondeclarative memory, procedural memory, that is intact in amnesia: amnesic patients are able to retrieve premorbidly learned motor skills and are able to acquire new ones, even if they are unable to retain declarative knowledge of having learned these skills. Other forms of nondeclarative memory that are preserved in amnesia include conditioning (provided that the conditioned stimulus and the unconditioned stimulus are overlapping) and perceptual and conceptual repetition priming. Repetition priming is the behavioral facilitation that occurs when processing a repeated compared to a novel stimulus, even without explicit awareness of the repetition. Perceptual repetition priming refers to facilitated processing of visual information, such as stimulus form, when visual information is repeated after an initial occurrence. Conceptual repetition priming refers to facilitated processing of stimulus meaning, independent from stimulus form, when analysis of stimulus meaning is repeated. The preservation of nondeclarative memory functions in amnesia is thought to reflect intact neural function in regions outside the limbic

system, such as the basal ganglia and premotor/motor cortices (motor learning), cerebellum (conditioning), and inferior and lateral temporal cortex (perceptual and conceptual priming). In the case of perceptual and conceptual priming, neocortical regions in inferior and lateral temporal cortex that store perceptual and conceptual representations are thought to be tuned with repetition such that previously accessed information can be more effectively activated and retrieved.

In contrast to largely preserved nondeclarative memory function, amnesic patients demonstrate profound declarative memory impairments on explicit memory tests requiring conscious retrieval of recent experiences. Explicit memory tests can take the form of recall or recognition. Although both recall and recognition tests require conscious retrieval of mnemonic information, they differ with respect to their processing demands and can be differentially impaired in amnesia.

According to dual-process models, recognition memory relies on two distinct processes: recollection and familiarity. Recollection refers to the effortful and intentional remembering of a prior encounter with its contextual source, whereas familiarity refers to the subjective feeling of knowing that an item was previously encountered without any knowledge of the context in which the item was acquired. Recollection and familiarity can be differentially impaired in amnesia depending on the locus of MTL damage. Amnesic patients with damage limited to the hippocampus have been described as having impaired recollection but preserved familiarity. Consistent with this theory, these patients perform poorly on free recall tasks but demonstrate intact performance on item recognition tasks. Recall deficits with hippocampal damage are thought to reflect impaired binding of contextual information into the memory trace, a process that is normally mediated by the hippocampus and makes recollection possible.

In contrast, when lesions extend into extrahippocampal regions of MTL, such as perirhinal cortex, patients demonstrate more severe declarative memory impairments that include both recollection and familiarity. These impairments are evident in both recall and recognition tests, though impairments in recall may be more severe than impairments in recognition. Extrahippocampal regions of MTL are thought to support memory for individual items without their accompanying episodic context.

While neuroimaging and neurophysiological studies provide convergent evidence for a division of labor in the MTL according to regions that support familiarity and those that support recollection, functional dissociations along these lines have not been consistently observed. An alternative proposal is that the hippocampus supports both recollection and familiarity (supporting both item recognition and recall) and that MTL subregions functionally dissociate according to whether they support stronger memories (hippocampus) or weaker memories (extrahippocampal regions of MTL). While the precise contributions of different MTL lesions to functional impairments in amnesia remain to be specified, episodic memory deficits can be considered to be a core characteristic of amnesia.

In addition to pervasive deficits in acquiring episodic information, new semantic learning can also be impaired in amnesia when MTL lesions are extensive. In such cases, patients demonstrate minimal ability to acquire new facts or concepts, particularly when this new information is difficult to

incorporate into existing knowledge structures. For example, patient H.M. was unable to acquire the meaning of new words that had entered into the vocabulary since the onset of his amnesia in 1953 (e.g., words such as 'granola' or 'jacuzzi').

p0110 The magnitude of semantic learning deficits in amnesia has been found to correspond to the amount of MTL damage. Patients with lesions limited to the hippocampus can acquire some degree of new semantic knowledge, including vocabulary words, indicating that new semantic learning can be supported by regions outside the hippocampus proper (e.g., perirhinal, parahippocampal, or lateral temporal cortex). Patients with larger MTL lesions extending into lateral temporal lobe have more severe impairments in semantic learning. Though these patients can acquire small amounts of new semantic knowledge with very extensive repetition, likely reflecting the ability of intact lateral temporal regions to gradually acquire new semantic knowledge after a great number of encoding episodes, such learning is typically very inflexible.

p0115 While the extent and location of lesions within the MTL greatly impacts the nature of anterograde memory impairments in amnesia, even more striking are the cognitive symptoms that occur when frontal deficits are superimposed on the core amnesia. Frontal lesions impair executive processes that contribute to memory, such as the ability to mentally manipulate or organize information to be encoded into memory and the ability to initiate and evaluate memory search. Executive deficits can lead to impairments in temporal or contextual aspects of memory and can lead to high levels of intrusions in recall tests or false endorsements in recognition tests. Rather than exhibiting a qualitatively different form of amnesia, patients with additional frontal lesions display a pattern of deficits consistent with a combination of a classic amnesic syndrome and additional problems associated with a frontal dysexecutive syndrome.

s0055 **Retrograde Amnesia**

p0120 Though many premorbid memories are preserved in amnesia, some degree of retrograde memory loss is often observed. Retrograde amnesia for events immediately preceding the onset of the brain lesion is particularly pronounced. According to Ribot's law, retrograde amnesia is characterized by a time gradient in which the memory impairment is inversely related to the recency of the to-be-recalled event. By this view, memory is worse for more recent compared to more remote memories. While patients with MTL damage reliably demonstrate such a temporal gradient of retrograde memory loss for semantic information, with relative sparing of remote memory compared to recent memory, MTL damage does not spare remote memories in all cases, and patterns of retrograde amnesia appear to depend on the type of memory under investigation. In particular, when personal episodic (autobiographical) memories from different past time periods are probed (e.g., memory for events occurring during childhood, during early adulthood, and within the past 5 years), memory impairments can extend for decades and in some cases can include a patient's entire lifespan.

p0125 Temporally graded retrograde amnesia has been interpreted in terms of standard models of consolidation. In these models, memories are thought to depend on the MTL, and particularly

the hippocampus, until they can be transferred to more stable cortical storage systems. The hippocampus plays a time-limited role in memory and is crucial only for the initial consolidation of declarative memories. Thus, while MTL mechanisms are critical for the retrieval of recent memories that have yet to be reconsolidated, the MTL is no longer needed to retain and recover memories once consolidation is complete.

Observations of extensive temporal gradients of autobio- p0130
graphical memory loss are difficult to incorporate into consolidation theory, as the loss of such memories extends well beyond the time frame during which biological consolidation is likely to operate. An alternative theory, multiple trace theory, suggests that the MTL is always necessary for the retrieval of episodic memories, including both recent and remote autobiographical memories. According to multiple trace theory, older memories become more resistant to disruption because each time a memory is retrieved, a new memory trace is established that is linked to the older memory traces. Thus, older memories are represented by more and stronger traces and are more resistant to partial lesions of the MTL whereas newer memories are represented by fewer and weaker traces and are more vulnerable to damage. By this view, the extent of retrograde amnesia is determined by the amount of MTL damage as well as the number of traces by which a memory is represented. As such, very extensive MTL damage can affect autobiographical memories for all time periods.

Yet another different pattern of remote memory loss is seen p0135
in patients whose lesions extend beyond the MTL into the anterolateral temporal lobes. In these cases, retrograde amnesia is characterized not only by extensive autobiographical memory loss, but also by severe semantic memory loss. The latter is thought to reflect the damage to the neocortical sites Au3 where semantic information is stored. Impairments in semantic memory are often more prominent after lesions to the left temporal lobes, likely reflecting the role of the left hemisphere in verbal lexical-semantic processing.

Patients with frontal lobe damage can also present with p0140
extensive retrograde amnesia that may encompass both semantic and episodic memory. These retrograde memory impairments are particularly evident under high demands of self-initiated retrieval and likely reflect deficits in frontal executive processing. Confabulatory responses may also be prominent with frontal lobe damage, reflecting impaired monitoring of information retrieved from memory.

Theoretical and Clinical Implications

While memory deficits are the hallmark of amnesia, this article p0145
has described the important point that not all forms of memory are similarly impaired in amnesia. Memory impairments can include both retrograde and anterograde components but differ according to episodic and semantic demands and depend on lesion location and extent. Though structurally and functionally heterogeneous, amnesia provides an important window onto the multiple forms of memory supported by the human brain. The patterns of impaired and preserved function in amnesia clearly demonstrate that rather than being a unitary system, long-term memory comprises functionally and structurally distinct memory subsystems.

p0150 In addition to expanding our knowledge about long-term memory systems, studies of patients with amnesia have also influenced our understanding of cognitive functions outside the memory domain. Emerging evidence indicates that amnesic patients have difficulty envisioning the future and are impaired at generating imaginary scenes. These results suggest that the brain regions involved in remembering the past overlap with those involved in thinking about novel future scenarios. It has been proposed that richly envisioning future scenarios requires flexible retrieval and recombination of details from the past, drawing on the same remote memory processes that are impaired in amnesia. The integrity of long-term memory mechanisms may also impact other related functions in amnesia, such as planning and problem solving.

p0155 While the theoretical advances provided by the study of amnesic patients are significant, caution must be taken when inferring strict one-to-one mappings of neural structure to cognitive function. In particular, the behavioral measurements indexed by standard neuropsychological tests may reflect contributions from multiple underlying cognitive processes or neural systems. Indeed, recent evidence raises questions about the independence between memory systems that has been inferred from the performance of amnesic patients on standard neuropsychological tests (such as implicit memory vs. explicit memory and short-term memory vs. long-term memory). For example, while standard neuropsychological measures suggest that memory impairments in amnesia are selective to long-term memory, a growing body of evidence suggests that short-term memory may also be impaired. In particular, recent behavioral studies of amnesic patients with damage to the MTL have demonstrated impairments in retaining relational information over delays as brief as one second. Together with recent functional neuroimaging data in humans that demonstrates delay-period activity in the MTL during short-term memory tasks, these results challenge traditional neuroanatomical dissociations between short-term memory and long-term memory and suggest that the mnemonic role of the MTL may extend beyond the long-term memory domain.

p0160 Though the nature of memory impairments in amnesia must be interpreted carefully, research into this topic will continue to enable greater clarification of how memory representations are formed in the human brain and how they contribute to behavior. In turn, such insights may serve as a foundation for improved diagnosis and more targeted treatment of memory disorders. In addition, improved understanding of how the brain supports memory can inform new

rehabilitation techniques. Because pharmacological treatments for organic amnesia are not widely tested or available, behavioral techniques currently offer the most promising method for helping amnesic patients manage and remediate their memory problems. Rehabilitative techniques that take advantage of learning mechanisms that are preserved in amnesia, such as repetitive training and implicit learning, can be effective in teaching amnesic patients new information. With such techniques, patients can be trained to use external memory aids such as personal digital assistants (PDAs) or paging devices. Such memory aids can provide useful reminders about important events, such as the need to take medications, providing functional support in everyday life.

See also: Amnesia (00021); Episodic Memory (00152); Memory (00229); Semantic Memory (00315).

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