Amnesia

Amnesia	
Other names	Amnesic syndrome
Specialty	Psychiatry, neurology

Amnesia is a deficit in memory caused by brain damage or disease,[1] but it can also be caused temporarily by the use of various sedatives and hypnotic drugs. The memory can be either wholly or partially lost due to the extent of damage that was caused.[2] There are two main types of amnesia: retrograde amnesia and anterograde amnesia. Retrograde amnesia is the inability to retrieve information that was acquired before a particular date, usually the date of an accident or operation.[3] In some cases the memory loss can extend back decades, while in others the person may lose only a few months of memory. Anterograde amnesia is the inability to transfer new information from the short-term store into the long-term store. People with anterograde amnesia cannot remember things for long periods of time. These two types are not mutually exclusive; both can occur simultaneously.

Case studies also show that amnesia is typically associated with damage to the medial temporal lobe. In addition, specific areas of the hippocampus (the CA1 region) are involved with memory. Research has also shown that when areas of the diencephalon are damaged, amnesia can occur. Recent studies have shown a correlation between deficiency of RbAp48 protein and memory loss. Scientists were able to find that mice with damaged memory have a lower level of RbAp48 protein compared to normal, healthy mice. In people suffering with amnesia, the ability to recall *immediate information* is still retained,[4] and they may still be able to form new memories. However, a severe reduction in the ability to learn new material and retrieve old information can be observed. Patients can learn new procedural knowledge. In addition, priming (both perceptual and conceptual) can assist amnesiacs in the learning of fresh non-declarative knowledge.[1] Amnesic patients also retain substantial intellectual, linguistic, and social skill despite profound impairments in the ability to recall specific information encountered in prior learning episodes.[5][6][7]

The term is from Ancient Greek, meaning 'forgetfulness'; from ἀ- *(a-)*, meaning 'without', and μνήσις *(mnesis)*, meaning 'memory'.

Contents

Signs and symptoms

Individuals with amnesia can learn new information, particularly if the information is non-declarative knowledge. However, in some situations, people with dense anterograde amnesia do not remember the episodes during which they previously learned or observed the information. Some people who suffer from amnesia show abnormal amounts of memory loss, confusion, and difficulty recalling other people or places. People who recover often do not remember having amnesia. [8]

Declarative information

Some patients with anterograde amnesia can still acquire some semantic information, even though it might be more difficult and might remain rather unrelated to more general knowledge. H.M. could accurately draw a floor plan of the home in which he lived after surgery, even though he had not lived there in years. The reason patients could not form new episodic memories is likely because the CA1 region of the hippocampus was a lesion, and thus the hippocampus could not make connections to the cortex. After an ischemic episode following surgery, an MRI of patient R.B. showed his hippocampus to be intact except for a specific lesion restricted to the CA1 pyramidal cells.[1]

Non-declarative information

Some retrograde and anterograde amnesics are capable of non-declarative memory, including implicit learning and procedural learning. For example, some patients show improvement on the pseudorandom sequences experiment as healthy people do. Therefore, procedural learning can proceed independently of the brain system required for declarative memory. According to fMRI studies, the acquisition of procedural memories activates the basal ganglia, the premotor cortex and the supplementary motor area, regions which are not normally associated with the formation of declarative memories. This type of dissociation between declarative and procedural memory can also be found in patients with diencephalic amnesia such as Korsakoff's syndrome. Another example demonstrated by some patients, such as K.C. and H.M, who have medial temporal damage and anterograde amnesia, still have perceptual priming. Those patients did well in the word fragment completion test.[1]

Causes

There are three generalized categories in which amnesia could be acquired by a person. The three categories are head trauma (example: head injuries), traumatic events (example: seeing something devastating to the mind), or physical deficiencies (example: atrophy of the hippocampus). The majority of amnesia and related memory issues derive from the first two categories as these are more common and the third could be considered a subcategory of the first.

- Head trauma is a very broad range as it deals with any kind of injury or active action toward the brain which might cause amnesia. Retrograde and anterograde amnesia is more often seen from events like this, an exact example of a cause of the two would be electroconvulsive therapy, which would cause both briefly for the receiving patient.
- Traumatic events are more subjective. What is traumatic is dependent on what the person finds to be traumatic. Regardless, a traumatic event is an event where something so distressing occurs that the mind chooses to forget rather than deal with the stress. A common example of amnesia that is caused by traumatic events is dissociative amnesia, which occurs when the person forgets an event that has deeply disturbed them.[9] An example would be a person forgetting a fatal and graphic car accident involving their loved ones.
- Physical deficiencies are different from head trauma because physical deficiencies lean more toward passive physical issues.

Among specific causes of amnesia are the following:

- Electroconvulsive therapy in which seizures are electrically induced in patients for therapeutic effect can have acute effects including both retrograde and anterograde amnesia.[10]
- Alcohol can both cause blackouts[11] and have deleterious effects on memory formation.[12]

Diagnosis

Types

- **Anterograde amnesia** is the inability to create new memories due to brain damage, while long-term memories from before the event remain intact. The brain damage can be caused by the effects of long-term alcoholism, severe malnutrition, stroke, head trauma, encephalitis, surgery, Wernicke–Korsakoff syndrome, cerebrovascular events, anoxia or other trauma.[13] The two brain regions related with this condition are medial temporal lobe and medial diencephalon. Anterograde amnesia cannot be treated with pharmacological methods due to neuronal loss.[14] However, treatment exists in educating patients to define their daily routines and after several steps they begin to benefit from their procedural memory. Likewise, social and emotional support is critical to improving quality of life for anterograde amnesia sufferers.[14] Fentanyl use by opioid users has been identified as a potential cause in a cluster of cases that occurred in Boston, MA.[15]
- **Retrograde amnesia** is inability to recall memories before onset of amnesia. One may be able to encode new memories after the incident. Retrograde is usually caused by head trauma or brain damage to parts of the brain besides the hippocampus. The hippocampus is responsible for encoding new memory. Episodic memory is more likely to be affected than semantic memory. The damage is usually caused by head trauma, cerebrovascular accident, stroke, tumor, hypoxia, encephalitis, or chronic alcoholism. People suffering from retrograde amnesia are more likely to remember general knowledge rather than specifics. Recent memories are less likely to be recovered, but older memories will be easier to recall due to strengthening over time.[16] Retrograde amnesia is usually temporary and can be treated by exposing them to memories from the loss.[17] Another type of consolidation (process by which memories become stable in the brain) occurs over much longer periods of time/days, weeks, months and years and likely involves transfer of information from the hippocampus to more permanent storage site in the cortex. The operation of this longer-term consolidation process is seen in the retrograde amnesia of patients with hippocampal damage who can recall memories from childhood relatively normally, but are impaired when

recalling experiences that occurred just a few years prior to the time they became amnesic. (Kirwan et al.,2008)

- **Post-traumatic amnesia** is generally due to a head injury (example: a fall, a knock on the head). Traumatic amnesia is often transient, but may be permanent or either anterograde, retrograde, or mixed type. The extent of the period covered by the amnesia is related to the degree of injury and may give an indication of the prognosis for recovery of other functions. Mild trauma, such as a car accident that results in no more than mild whiplash, might cause the occupant of a car to have no memory of the moments just before the accident due to a brief interruption in the short/long-term memory transfer mechanism. The sufferer may also lose knowledge of who people are. Having longer periods of amnesia or consciousness after an injury may be an indication that recovery from remaining concussion symptoms will take much longer.[18]
- **Dissociative amnesia** results from a psychological cause as opposed to direct damage to the brain caused by head injury, physical trauma or disease, which is known as organic amnesia. Individuals with organic amnesia have difficulty with emotion expression as well as undermining the seriousness of their condition. The damage to the memory is permanent.[19] Dissociative amnesia can include:
  - **Repressed memory** is the inability to recall information, usually about stressful or traumatic events in persons' lives, such as a violent attack or disaster. The memory is stored in long-term memory, but access to it is impaired because of psychological defense mechanisms. Persons retain the capacity to learn new information and there may be some later partial or complete recovery of memory. Formerly known as "Psychogenic Amnesia".
  - **Dissociative fugue** (*formerly* psychogenic fugue) is also known as fugue state. It is caused by psychological trauma and is usually temporary and unresolved, and therefore may return. An individual with dissociative fugue disorder is unaware or confused about his or her identity and will travel in journeys away from familiar surroundings to discover or create new identities.[20] The Merck Manual defines it as "one or more episodes of amnesia in which patients cannot recall some or all of their past and either lose their identity or form a new identity. The episodes, called fugues, result from trauma or stress. Dissociative fugue often manifests as sudden, unexpected, purposeful travel away from home."[21] While popular in fiction, it is extremely rare.
  - **Posthypnotic amnesia** occurs when events during hypnosis are forgotten, or where past memories are unable to be recalled. The failure to remember those events is induced by suggestions made during the hypnosis.[22] Some characteristics of posthypnotic amnesia include inability to remember specific events while under hypnotic influence, reversibility, and having no relation between the implicit and explicit memory. Research has shown that there could be selectivity with amnesia when posthypnotic amnesia occurs. [23]
- **Lacunar amnesia** is the loss of memory about one specific event.
- **Childhood amnesia** (also known as infantile amnesia) is the common inability to remember events from one's own childhood. Sigmund Freud notoriously attributed this to sexual repression, while modern scientific approaches generally attribute it to aspects of brain development or developmental psychology, including language development, which may be why people do not easily remember pre-language events. Some research states that most adults cannot remember memories as early as two or three years old. Research suggests there are cultural influences that affect memories that are recalled.[24] Researchers have found that implicit memories cannot be recalled or described. Remembering how to play the piano is a common example of implicit memory, as are walking, speaking, and other everyday activities that would be difficult to focus on if they had to be relearned every time one got up in the morning. Explicit memories, on the other hand, can be recalled and

described in words. Remembering the first time meeting a teacher is an example of an explicit memory.[25]

- **Transient global amnesia** is a well-described medical and clinical phenomenon. This form of amnesia is distinct in that abnormalities in the hippocampus can sometimes be visualized using a special form of magnetic resonance imaging of the brain known as diffusion-weighted imaging (DWI). Symptoms typically last for less than a day and there is often no clear precipitating factor or any other neurological deficits. The cause of this syndrome is not clear. The hypothesis of the syndrome includes transient reduced blood flow, possible seizure or an atypical type of a migraine. Patients are typically amnestic of events more than a few minutes in the past, though immediate recall is usually preserved.
- **Source amnesia** is the inability to remember where, when or how previously learned information has been acquired, while retaining the factual knowledge.[26] When individuals are unable to remember, false memories can occur and cause great confusion. [27]
- **Korsakoff's syndrome** can result from long-term alcoholism or malnutrition. It is caused by brain damage due to a vitamin B_1 deficiency and will be progressive if alcohol intake and nutrition pattern are not modified. Other neurological problems are likely to be present in combination with this type of Amnesia, such as problems with the medial temporal lobe and frontal lobe dysfunction.[28] Korsakoff's syndrome is also known to be connected with confabulation. The person's short-term memory may appear to be normal, but the person may have a difficult time attempting to recall a past story, or with unrelated words, as well as complicated patterns.[29] Korsakoff's syndrome is unique because it involves both anterograde and retrograde amnesia. [28]
- **Drug-induced amnesia** is intentionally caused by injection of an amnestic drug to help a patient forget surgery or medical procedures, particularly those not performed under full anesthesia, or likely to be particularly traumatic. Such drugs are also referred to as "premedicants". Most commonly, a 2-halogenated benzodiazepine such as midazolam or flunitrazepam is the drug of choice, although other strongly amnestic drugs such as propofol or scopolamine may also be used for this application. Memories of the short time-frame in which the procedure was performed are permanently lost or at least substantially reduced, but once the drug wears off, memory is no longer affected.
- **Situation-specific amnesia** can arise in a variety of circumstances (for example, committing an offence, child sexual abuse) resulting in PTSD. It has been claimed that it involves a narrowing of consciousness with attention focused on central perceptual details and/or that the emotional or traumatic events are processed differently from ordinary memories.
- **Transient epileptic amnesia** is a rare and unrecognized form of temporal lobe epilepsy, which is typically an episodic isolated memory loss. It has been recognized as a treatment-responsive syndrome congenial to anti-epileptic drugs.[30]
- **Semantic amnesia** affects semantic memory and primarily expresses itself in the form of problems with language use and acquisition.[31] Semantic amnesia can lead to dementia. [32]

Treatment

Many forms of amnesia fix themselves without being treated.[33] However, there are a few ways to cope with memory loss if treatment is needed. Since there are a variety of causes that form different amnesia, it is important to note that there are different methods that response better with the certain type of Amnesia. Emotional support and love as well as medication and psychological therapy have been proven effective. [34]

One technique for Amnesia treatment is cognitive or occupational therapy. In therapy, amnesiacs will develop the memory skills they have and try to regain some they have lost by finding which techniques help retrieve memories or create new retrieval paths.[35] This may also include strategies for organizing information to remember it more easily and for improving understanding of lengthy conversation.[36]

Another coping mechanism is taking advantage of technological assistance, such as a personal digital device to keep track of day-to-day tasks. Reminders can be set up for appointments when to take medications, birthdays and other important events. Many pictures can also be stored to help amnesiacs remember names of friends, family, and co-workers.[35] Notebooks, wall calendars, pill reminders and photographs of people and places are low-tech memory aids that can help as well.[36]

While there are no medications available to treat amnesia, underlying medical conditions can be treated to improve memory. Such conditions include but are not limited to low thyroid function, liver or kidney disease, stroke, depression, bipolar disorder and blood clots in the brain.[37] Wernicke–Korsakoff syndrome involves a lack of thiamin and replacing this vitamin by consuming thiamin-rich foods such as whole-grain cereals, legumes (beans and lentils), nuts, lean pork, and yeast.[33] Treating alcoholism and preventing alcohol and illicit drug use can prevent further damage, but in most cases will not recover lost memory.[36]

Although improvements occur when patients receive certain treatments, there is still no actual cure remedy for amnesia so far. To what extent the patient recovers and how long the amnesia will continue depends on the type and severity of the lesion.[38]

History

French psychologist Theodule-Armand Ribot was among the first scientists to study amnesia. He proposed Ribot's Law which states that there is a time gradient in retrograde amnesia. The law follows a logical progression of memory loss due to disease. First, a patient loses the recent memories, then personal memories, and finally intellectual memories. He implied that the most recent memories were lost first.[39]

Case studies have played a large role in the discovery of amnesia and the parts of the brain that were affected. The studies gave important insight into how amnesia affects the brain. The studies also gave scientists the resources into improving their knowledge about amnesia and insight into a cure or prevention. There are several extremely important case studies: Henry Molaison, R.B, and G.D.

Henry Molaison

Henry Molaison, formerly known as H.M., changed the way people thought of memory. The case was first reported in a paper by William Beecher Scoville and Brenda Milner in 1957.[40] He was a patient who suffered from severe epilepsy attributed to a bicycle accident at the age of nine. Physicians were unable to control his seizures with drugs, so the neurosurgeon Scoville tried a new approach involving brain surgery. He removed his medial temporal lobe bilaterally by doing a temporal lobectomy. His epilepsy did improve, but Molaison lost the ability to form new long-term memories (anterograde amnesia). He exhibited normal short-term memory ability. If he was given a list of words, he would forget them in about a minute's time. In fact, he would forget that he was even given a list in the first place.[41] Once Molaison stopped thinking about the lists he was unable to recall them again from long term memory. This gave researchers evidence that short-term and long-term memory are in fact two

different processes.[42] Even though he forgot about the lists, he was still able to learn things through his implicit memory. The psychologists would ask him to draw something on a piece of paper, but to look at the paper using a mirror. Though he could never remember ever doing that task, he would improve after doing it over and over again. This showed the psychologists that he was learning and remembering things unconsciously.[43]

Studies were completed consistently throughout Molaison's lifetime to discover more about amnesia.[1] Researchers did a 14-year follow-up study on Molaison. They studied him for a period of two weeks to learn more about his amnesia. After 14 years, Molaison still could not recall things that had happened since his surgery. However, he could still remember things that had happened prior to the operation. Researchers also found that, when asked, Molaison could answer questions about national or international events, but he could not remember his own personal memories.[41] After his death Molaison donated his brain to science, where they were able to discover the areas of the brain that had the lesions which caused his amnesia.[42] This case study provided important insight to the areas of the brain that are affected in anterograde amnesia, as well as how amnesia works.

Patient R.B.

Patient R.B. was a normally functioning man until the age of 52. At age 50, he had been diagnosed with angina and had surgery for heart problems on two occasions. After an ischemic episode (reduction of blood to the brain) that was caused from a heart bypass surgery, R.B. demonstrated a loss of anterograde memory, but almost no loss of retrograde memory, with the exception of a couple of years before his surgery, and presented no sign of any other cognitive impairment. It wasn't until after his death that researchers had the chance to examine his brain, when they found his lesions were restricted to the CA1 portion of the hippocampus. This case study led to important research involving the role of the hippocampus and the function of memory.[44]

Patient G.D.

Patient G.D. was a white male born in 1940 who served in the Navy. He was diagnosed with chronic renal failure and received hemodialysis treatment for the rest of his life. In 1983, he went to the hospital for elective parathyroidectomy. He also had a left thyroid lobectomy because of the severe loss of blood in his left lobe. He began having cardiac problems as a result of the surgery and became very agitated. Even five days after being released from the hospital he was unable to remember what had happened to him. Aside from memory impairment, none of his other cognitive processes seemed to be affected. He did not want to be involved in much research, but through memory tests he took with doctors, they were able to ascertain that his memory problems were present for the next 9.5 years until his death. After he died, his brain was donated to science, photographed, and preserved for future study.[45]

In fiction

Global amnesia is a common motif in fiction despite being extraordinarily rare in reality. In the introduction to his anthology *The Vintage Book of Amnesia*, Jonathan Lethem writes:

> Real, diagnosable amnesia – people getting knocked on the head and forgetting their names – is mostly just a rumor in the world. It's a rare condition, and usually a brief one. In books and movie, though, versions of amnesia lurk everywhere, from episodes of *Mission*

> *Impossible* to metafictional and absurdist masterpieces, with dozens of stops in between. Amnesiacs might not much exist, but amnesiac characters stumble everywhere through comic books, movies, and our dreams. We've all met them and been them.[46]

Lethem traces the roots of literary amnesia to Franz Kafka and Samuel Beckett, among others, fueled in large part by the seeping into popular culture of the work of Sigmund Freud, which also strongly influenced genre films such as *film noir*. Amnesia is so often used as a plot device in films, that a widely recognized stereotypical dialogue has even developed around it, with the victim melodramatically asking "Where am I? Who am I? What am I?", or sometimes inquiring of his own name, "Bill? Who's Bill?"[46]

In movies and television, particularly sitcoms and soap operas, it is often depicted that a second blow to the head, similar to the first one which caused the amnesia, will then cure it. In reality, however, repeat concussions may cause cumulative deficits including cognitive problems, and in extremely rare cases may even cause deadly swelling of the brain associated with second-impact syndrome.

See also

- Aphasia
- Betrayal
- Emotion and memory
- False memory
- Gollin figure test
- List of films featuring mental illness
- Memory erasure
- Nostalgia
- Repressed memories
- Transient epileptic amnesia

Amnesiacs

- Benjaman Kyle
- Clive Wearing
- Doug Bruce
- KC (patient)
- Scott Bolzan
- Sywald Skeid

References

1. Gazzaniga, M., Ivry, R., & Mangun, G. (2009) Cognitive Neuroscience: The biology of the mind. New York: W.W. Norton & Company.
2. "Amnesia." The Gale Encyclopedia of Science. Ed. K. Lee Lerner and Brenda Wilmoth Lerner. 4th ed. Vol. 1. Detroit: Gale, 2008. 182–184. Gale Virtual Reference Library.
3. Schacter, Daniel. L "Psychology"
4. Benson, D. Frank (1978). "Amnesia". *Southern Medical Journal*. **71** (10): 1221–1227. doi:10.1097/00007611-197810000-00011 (https://doi.org/10.1097%2F00007611-197810000-00011). PMID 360401 (https://pubmed.ncbi.nlm.nih.gov/360401).

5. LS., Cermak (1984). *The episodic-semantic distinction in amnesia*. New York: Guilford Press. p. 55.
6. M, Kinsbourne (1975). *Short-term memory processes and the amnesic syndrome*. New York: Academic. pp. 258–91.
7. H, Weingartner (1983). *Forms of cognitive failure*. Sc alzheimerience. pp. 221:380–2.
8. Services, Department of Health & Human. "Amnesia" (https://www.betterhealth.vic.gov.au:443/health/conditionsandtreatments/amnesia). *www.betterhealth.vic.gov.au*. Retrieved 3 December 2019.
9. Myers, David G. Psychology. fifth ed. New York: Worth Publishers, 1998. N. pag. Print
10. Benbow, SM (2004) "Adverse effects of ECT". In AIF Scott (ed.) *The ECT Handbook, second edition.* (http://www.rcpsych.ac.uk/publications/collegereports/cr/cr128.aspx) London: The Royal College of Psychiatrists, pp. 170–174.
11. Goodwin DW; Crane JB; Guze SB (August 1969). "Alcoholic "blackouts": a review and clinical study of 100 alcoholics". *Am J Psychiatry*. **126** (2): 191–8. doi:10.1176/ajp.126.2.191 (https://doi.org/10.1176%2Fajp.126.2.191). PMID 5804804 (https://pubmed.ncbi.nlm.nih.gov/5804804).
12. Parker ES; Birnbaum IM; Noble EP (December 1976). "Alcohol and memory: Storage and state dependency". *Journal of Verbal Learning and Verbal Behavior*. **15** (6): 691–702. doi:10.1016/0022-5371(76)90061-X (https://doi.org/10.1016%2F0022-5371%2876%2990061-X).
13. Carlson, N. R. (19992000). Memory. Psychology: the science of behaviour (Canandian ed., p. 250). Scarborough, Ont.: Allyn and Bacon Canada.
14. Erdogan, Serap (2010). "Anterograde Amnesia" (http://www.cappsy.org/archives/vol2/no2/cap_02_10.pdf) (PDF). *Psikiyatride Guncel Yaklasimlar-Current Approaches in Psychiatry*. **2** (2): 174–189. Retrieved 27 November 2011.
15. Barash, Jed A.; Ganetsky, Michael; Boyle, Katherine; Raman, Vinod; Toce, Michael S.; Kaplan, Scott; Lev, Michael H.; Worth, Jonathan L.; DeMaria, Alfred (2018). "Acute Amnestic Syndrome Associated with Fentanyl Overdose". *New England Journal of Medicine*. **378** (12): 1157–1158. doi:10.1056/NEJMc1716355 (https://doi.org/10.1056%2FNEJMc1716355). ISSN 1533-4406 (https://www.worldcat.org/issn/1533-4406). PMID 29562161 (https://pubmed.ncbi.nlm.nih.gov/29562161).
16. Mastin, L. (2010). The human memory: Retrograde amnesia . Retrieved from http://www.human-memory.net/disorders_retrograde.html
17. "memory abnormality." Encyclopædia Britannica. Encyclopædia Britannica Online Academic Edition. Encyclopædia Britannica Inc., 2012. Web. 21 April 2012.
18. Masferrer, Roberto; Masferrer, Mauricio; Prendergast, Virginia; Harrington, Timothy R (2000). "Grading Scale for Cerebral Concussions" (https://www.barrowneuro.org/education/grand-rounds-publications-and-media/barrow-quarterly/volume-16-no-1-2000/grading-scale-for-cerebral-concussions/). *BNI Quarterly*. **16** (1). ISSN 0894-5799 (https://www.worldcat.org/issn/0894-5799). Retrieved 5 February 2017.
19. Bourget, Dominique, and Laurie Whitehurst. "Amnesia and crime." *Journal of the American Academy of Psychiatry and the Law Online* 35.4 (2007): 469–480.
20. "Dissociative Fugue. Retrieved 7 August 2012" (https://web.archive.org/web/20121104104007/http://my.clevelandclinic.org/disorders/dissociative_disorders/hic_dissociative_fugue.aspx). My.clevelandclinic.org. Archived from the original (http://my.clevelandclinic.org/disorders/dissociative_disorders/hic_dissociative_fugue.aspx) on 4 November 2012. Retrieved 22 December 2012.
21. "The Merck Manuals Online" (http://www.merckmanuals.com/professional/psychiatric_disorders/dissociative_disorders/dissociative_fugue.html?qt=dissociative%20fugue&alt=sh). Merckmanuals.com. Retrieved 22 December 2012.

22. Carlson, Neil (2007). *Psychology the Science of Behaviour*. Toronto: Pearson. p. 283. ISBN 978-0-205-64524-4. OCLC 441151384 (https://www.worldcat.org/oclc/441151384).
23. Enea, Violeta, and Ion Dafinoiu. "Posthypnotic amnesia and autobiographical memory in adolescents." *Journal of Evidence-Based Psychotherapies* 8.2 (2008): 201.
24. Wang, Qi (January 2003). "Infantile amnesia reconsidered: A cross-cultural analysis". *Memory*. **11** (1): 65–80. doi:10.1080/741938173 (https://doi.org/10.1080%2F741938173). ISSN 0965-8211 (https://www.worldcat.org/issn/0965-8211). PMID 12653489 (https://pubmed.ncbi.nlm.nih.gov/12653489).
25. Hayne, Harlene; Jack, Fiona (2011). "Childhood amnesia". *Wiley Interdisciplinary Reviews: Cognitive Science*. **2** (2): 136–145. doi:10.1002/wcs.107 (https://doi.org/10.1002%2Fwcs.107). PMID 26302005 (https://pubmed.ncbi.nlm.nih.gov/26302005).
26. Schacter, D.L.; Harbluk, J.L.; McLachlen, D.R. (1984). "Retrieval without recollection: an experimental analysis of source amnesia" (https://www.academia.edu/10546602). *Journal of Verbal Learning and Verbal Behavior*. **23** (5): 593–611. doi:10.1016/s0022-5371(84)90373-6 (https://doi.org/10.1016%2Fs0022-5371%2884%2990373-6).
27. "Psychology: Memory" (https://www.evl.uic.edu/sugimoto/memSrc.html). *www.evl.uic.edu*. Retrieved 3 December 2019.
28. Spiegel, David R (June 2011). "A Case of Probable Korsakoff's Syndrome: A Syndrome of Frontal Lobe and Diencephalic Structural Pathogenesis and a Comparison with Medial Temporal Lobe Dementias" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3140893). *Innovations in Clinical Neuroscience*. **8** (6): 15–19. PMC 3140893 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3140893). PMID 21779537 (https://pubmed.ncbi.nlm.nih.gov/21779537).
29. "Types of Amnesia" (https://web.archive.org/web/20120420132739/http://ahsmail.uwaterloo.ca/kin356/amnesia/amnesia2.html). uwaterloo. Archived from the original (http://ahsmail.uwaterloo.ca/kin356/amnesia/amnesia2.html) on 20 April 2012. Retrieved 9 April 2012.
30. Walsh RD, Jr; Wharen RE, IV; Tatum WO (2011). "Complex transient epileptic amnesia" (http://journals2.scholarsportal.info/show_html.xqy?uri=/15255050/v20i0002/410_ctea.xml&school=toronto). *Epilepsy & Behavior*. **20** (2): 410–413. doi:10.1016/j.yebeh.2010.12.026 (https://doi.org/10.1016%2Fj.yebeh.2010.12.026).
31. Murray, B. D.; Kensinger, E. A. (2012). "Semantic Memory in Profound Amnesia". *Encyclopedia of the Sciences of Learning*. Boston, MA: Springer. pp. 3022–3025.
32. "Semantic Variant Primary Progressive Aphasia" (https://memory.ucsf.edu/dementia/primary-progressive-aphasia/semantic-variant-primary-progressive-aphasia). *Memory and Aging Center*. Retrieved 3 December 2019.
33. Nordqvist, Christian (21 January 2017) [2004]. "Amnesia: Causes, Symptoms, and Treatments" (http://www.medicalnewstoday.com/articles/9673.php). *Medical News Today*. Retrieved 5 February 2017.
34. Services, Department of Health & Human. "Amnesia" (https://www.betterhealth.vic.gov.au:443/health/conditionsandtreatments/amnesia). *www.betterhealth.vic.gov.au*. Retrieved 3 December 2019.
35. "Treating Amnesia". *Neurology Now*. **4** (4): 37. 2008. doi:10.1097/01.NNN.0000333846.54546.f8 (https://doi.org/10.1097%2F01.NNN.0000333846.54546.f8).
36. Mayo Clinic Staff (2011) Amnesia: Treatments and Drugs. Mayo Clinic. Retrieved from: http://www.mayoclinic.com/health/amnesia/DS01041/DSECTION=treatments-and-drugs.
37. Mandal, A. (n.d) Treatment of Amnesia. News Medical. Retrieved From: http://www.news-medical.net/health/Treatment-of-amnesia.aspx

38. Benson DF (October 1978). "Amnesia". *Southern Medical Journal*. **71** (10): 1221–1227. doi:10.1097/00007611-197810000-00011 (https://doi.org/10.1097%2F00007611-197810000-00011). ISSN 0038-4348 (https://www.worldcat.org/issn/0038-4348). PMID 360401 (https://pubmed.ncbi.nlm.nih.gov/360401).
39. Ribot, T. (1882). *Diseases of Memory: An essay in the positive psychology* (https://archive.org/details/diseasesmemorya01ribogoog). London: D. Appleton and company.
40. Scoville, W.B.; Milner, B. (1957). "Loss of recent memory after bilateral hippocampal lesions" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC497229). *Journal of Neurology, Neurosurgery, and Psychiatry*. **20** (1): 11–21. doi:10.1136/jnnp.20.1.11 (https://doi.org/10.1136%2Fjnnp.20.1.11). PMC 497229 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC497229). PMID 13406589 (https://pubmed.ncbi.nlm.nih.gov/13406589).
41. Corkin, S.; Milner, B.; Teuber, H. (1968). "Further Analysis of the Hippocampal Amnesic Syndrome: 14-Year Follow-up Study on Patient H.M." (https://is.muni.cz/el/1423/podzim2011/PSY221_P11/um/27733942/Milner__Corkin___Teuber__1968_.pdf) (PDF). *Neuropsychologia*. **6** (3): 215–234. doi:10.1016/0028-3932(68)90021-3 (https://doi.org/10.1016%2F0028-3932%2868%2990021-3).
42. Draaisma, D. (2013). "Neuroscience: Losing the past". *Nature*. **497** (7449): 313–314. Bibcode:2013Natur.497..313D (https://ui.adsabs.harvard.edu/abs/2013Natur.497..313D). doi:10.1038/497313a (https://doi.org/10.1038%2F497313a).
43. Rosenbaum, R. S.; Murphy, K. J.; Rich, J. B. (2012). "The amnesias". *Wiley Interdisciplinary Reviews: Cognitive Science*. **3** (1): 47–63. doi:10.1002/wcs.155 (https://doi.org/10.1002%2Fwcs.155). PMID 26302472 (https://pubmed.ncbi.nlm.nih.gov/26302472).
44. Zola-Morgan, S; Squire, LR; Amaral, DG (1986). "Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6568782). *The Journal of Neuroscience*. **6** (10): 2950–2967. doi:10.1523/JNEUROSCI.06-10-02950.1986 (https://doi.org/10.1523%2FJNEUROSCI.06-10-02950.1986). PMC 6568782 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6568782). PMID 3760943 (https://pubmed.ncbi.nlm.nih.gov/3760943).
45. Rempel-Clower, NL; Zola, SM; Squire, LR; Amaral, DG (1996). "Three cases of enduring memory impairment after bilateral damage limited to the hippocampal formation" (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6579309). *The Journal of Neuroscience*. **16** (16): 5233–5255. doi:10.1523/JNEUROSCI.16-16-05233.1996 (https://doi.org/10.1523%2FJNEUROSCI.16-16-05233.1996). PMC 6579309 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6579309). PMID 8756452 (https://pubmed.ncbi.nlm.nih.gov/8756452).
46. Lethem, Jonathan (ed.) *The Vintage Book of Amnesia* New York: Vintage, 2000 ISBN 0-375-70661-5

External links

Classification	**ICD-10**: F04, R41.3 (http://apps.who.int/classifications/icd10/browse/2016/en#/R41.3) · **ICD-9-CM**: 294.0 (http://www.icd9data.com/getICD9Code.ashx?icd9=294.0), 780.9 (http://www.icd9data.com/getICD

D

	9Code.ashx?icd9=780.9), 780.93 (http://www.icd9data.com/getICD9Code.ashx?icd9=780.93) · **MeSH**: D000647 (https://www.nlm.nih.gov/cgi/mesh/2015/MB_cgi?field=uid&term=D000647)
External resources	**MedlinePlus**: 003257 (https://www.nlm.nih.gov/medlineplus/ency/article/003257.htm)

Retrieved from "https://en.wikipedia.org/w/index.php?title=Amnesia&oldid=935482420"