Anterograde Amnesia

Authored by

mohammad looti

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Anterograde amnesia is a loss of the ability to create new memories after the event that caused the amnesia, leading to a partial or complete inability to recall the recent past, while long-term memories from before the event remain intact. Anterograde amnesia and retrograde amnesia, where memories created prior to the event are lost, can occur together in the same patient. To a large degree, anterograde amnesia remains a mysterious ailment because the precise mechanism of storing memories is not yet well understood, although it is known that the regions involved are certain sites in the temporal cortex, especially in the hippocampus and nearby subcortical regions.

Causes

This disorder is usually acquired in one of few ways: One type of cause is from agents like benzodiazepines, such as midazolam, flunitrazepam,lorazepam, temazepam, triazolam, and nimetazepam, which are known to have powerful amnesic effects. Another type of cause is a traumatic brain injury in which there is usually damage to the hippocampus or surrounding cortices. It can also be caused by shock or an emotional disorder. Illness, though much rarer, can also cause anterograde amnesia if it causes encephalitis, which is the inflammation of brain tissue. For example, herpes simplex virus type I, when left untreated for over ninety-six hours, may lead to permanent damage in hippocampal regions and lead to a permanent reduced or eliminated ability to encode new explicit memory (also known as declarative memory), which consists of two main subdivisions: episodic memory and semantic memory. If the damage due to encephalitis is over a certain threshold, encoding new episodic and/or semantic memory becomes impossible for the patient, leading to anterograde amnesia. However, it must be stressed that patients suffering from anterograde amnesia may have either episodic, semantic, or both types of explicit memory impaired for events after the trauma that caused the amnesia. The reasoning behind this is that memory consolidation for different types of memory takes place in different regions of the brain. Despite this, current knowledge on human memory is still decades away from the ability to "map out" the wiring of a human brain in order to discover which parts of which lobe are responsible for the various episodic and semantic knowledge within a person's memory.

Amnesia is seen in patients who, for the reason of preventing another more serious disorder, have parts of their brain that are known to be involved in memory circuits removed, the most notable of which is known as the medial temporal lobe (MTL) memory system, described below. Patients with seizures that originate in the MTL may have either side or both structures removed (there is one structure per hemisphere). In addition, patients with tumors who undergo surgery will often sustain damage to these structures, as is described in a case below. Damage to any part of this system, including the hippocampus and surrounding cortices, results in amnesic syndromes. This is why people who suffer from strokes have a chance of developing cognitive deficits that result in anterograde amnesia, since strokes can involve the temporal lobe and the temporal cortex, and the temporal cortex houses the hippocampus. As mentioned above, damage to the hippocampus and surrounding subcortical regions directly lead to anterograde amnesia.

Alcohol intoxication

Anterograde amnesia can also be caused by alcohol intoxication, a phenomenon commonly known as a blackout. Studies show that rapid rises in blood alcohol concentration over a short period of time severely impairs or in some cases completely blocks the brain's ability to transfer short-term memories created during the period of intoxication to long-term memory for storage and later retrieval. Such rapid rises in blood alcohol concentration are caused by drinking large amounts of alcohol in short periods of time, especially on an empty stomach, as the digestion of food slows the absorption of alcohol. Alcohol-related anterograde amnesia is directly related to the rate of consumption of alcohol (and is often associated with binge drinking), and not just the total amount of alcohol consumed in a drinking episode. Test subjects have been found not to experience amnesia when drinking slowly, despite being heavily intoxicated by the end of the experiment. When alcohol is consumed at a rapid rate, the point at which most healthy people's long-term memory creation starts to fail usually occurs at approximately 0.2% BAC, but can be reached as low as 0.14% BAC for inexperienced drinkers. The exact duration of these blackout periods is hard to determine, because most people fall asleep before they end. Upon reaching sobriety, usually after waking, long-term memory creation is completely restored. Another common cause of anterograde amnesia related to consumption of alcohol is the effects of Korsakoff's syndrome, a neurological disorder caused by the lack of thiamine (vitamin B1) in the brain. Chronic alcoholism causes this malnutrition, resulting in the syndrome, and among the symptoms of Korsakoff's syndrome are apathy, confabulation (delusions that result in invented memories), and anterograde amnesia.

Symptoms

Patients who suffer from anterograde amnesic syndromes may present with widely varying degrees of forgetfulness. Some patients with severe cases have a combined form of anterograde and retrograde amnesia, sometimes called global amnesia.

In the case that the amnesia is drug-induced, it may be short-lived and patients can recover from it. In the other case, which has been studied extensively since the early 1970s, patients often have damage that is permanent, although some recovery is possible, depending on the nature of the pathophysiology. Usually, there remains some capacity for learning although it may be very elementary. In cases of pure anterograde amnesia, patients have recollections of events prior to the injury but cannot recall day-to-day information or new facts that were presented to them after the injury occurred.

In most cases of anterograde amnesia, patients lose declarative memory, or the recollection of facts, but they retain non-declarative memory, often called procedural memory. For instance, they are able to remember and in some cases learn how to do things such as talking on the phone or

riding a bicycle, but they may not remember what they had eaten earlier that day for lunch. One extensively studied anterograde amnesiac patient, codenamed H.M., demonstrated that despite his amnesia preventing him from learning new declarative information, procedural memory consolidation was still possible, albeit severely reduced in power. He, along with other patients with anterograde amnesia, were given the same maze to complete day after day. Despite having no memory of having completed the maze the day before, unconscious practice of completing the same maze over and over reduced the amount of time needed to complete it in subsequent trials. From these results, Corkin et al. concluded that despite having no declarative memory (i.e. no conscious memory of completing the maze exists), the patients still had a working procedural memory (learning done unconsciously through practice). This supports the notion that declarative and procedural memory are consolidated in different areas of the brain. In addition, patients have a diminished ability to remember the temporal context in which objects were presented. Certain authors claim that the deficit in temporal context memory is more significant than the deficit in semantic learning ability (described below).

Pathophysiology

The pathophysiology of anterograde amnesic syndromes varies with the extent of damage and the regions of the brains that were damaged. The most well-described regions indicated in this disorder are the medial temporal lobe (MTL), basal forebrain, and fornix. Beyond the details described below, the precise process of how we remember -- on a micro scale -- remains a mystery. Neuropsychologists and scientists are still not in total agreement over whether forgetting is due to faulty encoding, accelerated forgetting, or faulty retrieval, although a great deal of data seem to point to the encoding hypothesis. In addition, neuroscientists are also in disagreement about the length of time involved in memory consolidation. Though most researchers including Hasselmo et al., have found that the consolidation process is spread out over several hours before transitioning from a fragile to a more permanent state, others, including Brown et al., posit that memory consolidation can take months or even years in a drawn-out process of consolidation and reinforcement. Further research into the length of time of memory consolidation will shed more light on why anterograde amnesia sometimes affects some memories gained after the event(s) that caused the amnesia but does not affect other such memories.

Medial temporal lobe

The MTL memory system includes the hippocampal formation (CA fields, dentate gyrus, subicular complex), perirhinal, entorhinal, and parahippocampal cortices. It is known to be important for the storage and processing of declarative memory, which allows for factual recall. It is also known to communicate with the neocortex in the establishment and maintenance of long-term memories, although its known functions are independent of long-term memory. Non-declarative memory, on

the other hand, which allows for the performance of different skills and habits, is not part of the MTL memory system. Most data point to a "division of labor" among the parts of this system, although this is still being debated and is described in detail below.

In animal models, researchers have shown that monkeys with damage to both the hippocampus and its adjacent cortical regions were more severely impaired in terms of anterograde amnesia than monkeys with damage localized to hippocampal structures. However, conflicting data in another primate study point to the observation that the amount of tissue damaged does not necessarily correlate with the severity of the memory loss. Furthermore, the data does not explain the dichotomy that exists in the MTL memory system between episodic memory and semantic memory (described below).

An important finding in amnesic patients with MTL damage is the impairment of memory in all sensory modalities - sound, touch, smell, taste, sight. This reflects the fact that the MTL is a processor for all of the sensory modalities, and helps store these kind of thoughts into memory. In addition, subjects can often remember how to perform relatively simple tasks immediately (on the order of 10 seconds), but when the task becomes more difficult, even on the same time scale, subjects tend to forget. This demonstrates the difficulty of separating procedural memory tasks from declarative memory; some elements of declarative memory may be used in learning procedural tasks.

MTL amnesic patients with localized damage to the hippocampus retain other perceptual abilities, such as the ability to intelligently function in society, to make conversation, to make one's bed, etc. Additionally, anterograde amnesics without combined retrograde disorders (localized damage to the MTL system) have memories prior to the traumatic event. For this reason, the MTL is not the storage place of all memories; other regions in the brain also store memories. The key is that the MTL is responsible for the learning of new materials.

Other memory systems

A limited number of cases have been described in which patients with damage to other parts of the brain acquired anterograde amnesia. Easton and Parker observed that damage to either the hippocampus or the surrounding cortices does not seem to result in severe amnesia in primate models. They suggested that damage to the hippocampus and surrounding structures alone does not explain the amnesia that they saw in patients, or the fact that increasing damage does not correlate with the degree of impairment. Furthermore, the data do not explain the dichotomy that exists in the MTL memory system between episodic and semantic memory (described below). To demonstrate their hypothesis, they used a primate model with damage to the basal forebrain. They proposed that the disruption of neurons that project from the basal forebrain to the MTL are responsible for some of the impairment in anterograde amnesia. Easton and Parker also report

that MRI scans of patients with severe anterograde amnesia show damage beyond to cortical areas around the hippocampus and amygdala (a region of brain involved in emotions) and to surrounding white matter (white matter in the brain consists of axons, long projections of neuronal cell bodies).

Another case described the onset of anterograde amnesia as a result of cell death in the fornix, another structure that carries information from the hippocampus to the structures of the limbic system and the diencephalon. The patient in this case did not show any disconnection syndrome, which is unexpected since the structures involved divide the brain hemispheres (both sides of her brain were able to communicate). Instead, she showed signs of amnesia. The final diagnosis was made by MRI. This particular amnesic syndrome is difficult to diagnose and often gets misdiagnosed by physicians as an acute psychiatric disorder.

Reorganization of memory

When there is damage to just one side of the MTL, there is opportunity for normal functioning or near-normal function for memories. Neuroplasticity describes the ability of the cortex to remap when necessary. Remapping can occur in cases like the one above, and, with time, the patient can recover and become more skilled at remembering. A case report describing a patient who had two lobectomies - in the first, doctors removed part of her right MTL first because of seizures originating from the region, and later her left because she developed a tumor -- demonstrates this. This case is unique because it is the only one in which both sides of the MTL were removed at different times. The authors observed that the patient was able to recover some ability to learn when she had only one MTL, but observed the deterioration of function when both sides of the MTL were afflicted. The reorganization of brain function for epileptic patients has not been investigated much, but imaging results show that it is likely.

Famous cases

The most famous case that has been reported is that of patient Henry Molaison, known as H.M., in March 1953. H.M.'s chief complaint was the persistence of severe seizures after he had a bilateral lobectomy (both of his MTLs were removed). As a result, H.M. had bilateral damage to both the hippocampal formation and the perirhinal cortex. H.M. had normal intelligence, perceptual ability, and a decent vocabulary, but he could not remember any new words or learn new tasks. He was the first well-documented case of severe anterograde amnesia, and was still being studied up until his death in 2008.

A similarly notable case was Clive Wearing, an accomplished musicologist who contracted a cold virus that attacked his brain, causing Herpes simplex encephalitis. As a result, Wearing developed anterograde amnesia as well as retrograde amnesia, so he has little memory of what happened

before the virus struck him in 1985 and cannot learn new declarative knowledge after the virus struck him as well. As a result of anterograde amnesia, Wearing repeatedly "wakes up" every day in thirty second intervals until his wife stops him because his episodic memory is non-functional (so he does not consciously recall having woken up thirty seconds prior). Despite this, however, Wearing maintained his ability to play the piano and conduct choirs. This is significant because it is a case study that demonstrates that declarative and procedural memory are separate. Therefore, despite that anterograde amnesia prevented Wearing from learning new bits of information that can be explained in words (declarative memory) and also prevented him from storing new memories of events or episodes (also part of declarative memory), he has little trouble in retaining his musical abilities (procedural memory) even though he has no conscious memory of having learned music.

Another remarkable case in the literature is E.P., a severely amnesic patient who was able to learn 3-word sentences. He performed better on consecutive tests over a 12-week period (24 study sessions). However, when asked how confident he was about the answers, it did not appear that his confidence increased. Bayley and Squire proposed that his learning was similar to the process required by procedural memory tasks; E.P. could not get the answers right when one word in the 3-word sentence was changed or the order of words was changed, and his ability to answer correctly, thus, became more of a "habit." Bayley and Squire claim that the learning may have happened in the neocortex, and that it happened without the conscious knowledge of E.P. They hypothesized that information may be acquired directly by the neocortex (which the hippocampus projects to) when there is repetition. This case illustrates the difficulty in separating procedural from declarative tasks; this adds another dimension to the complexity of anterograde amnesia.

Controversies

Episodic versus semantic memory

As described above, patients with anterograde amnesia have a wide range of forgetfulness. Declarative memory can be further subdivided into episodic and semantic memory. Episodic memory is described as the recollection of autobiographical information with a temporal and/or spatial context, whereas semantic memory involves recall of factual information with no such association (language, history, geography, etc.) In a case study of a girl who developed anterograde amnesia during childhood, it was determined that the patient C.L. retained semantic memory while suffering an extreme impairment of episodic memory.

One patient known by the codename Gene was involved in a motorcycle accident that damaged significant portions of his frontal and temporal lobes, including his left hippocampus. As a result, he cannot remember any specific episode in his life, such as a train derailment near his house. However, his semantic memory is intact; he remembers that he owns a car and two motorcycles, and he can even remember the names of his classmates in a school photograph.

In stark contrast, a woman whose temporal lobes were damaged in the front due to encephalitis lost her semantic memory; she lost her memory of many simple words, historical events, and other trivial information categorized under semantic memory. However, her episodic memory was left intact; she can recall episodes such as her wedding and her father's death with great detail.

Vicari et al. describe that it remains unclear whether or not neural circuits involved in semantic and episodic memory overlap partially or completely, and this case seems to suggest that the two systems are independent. Both of the patient's hippocampal and diencephalic structures on the right and left sides were disconnected. When she came to Vicari et al.'s office, the patient C.L.'s chief complaint was forgetfulness involving both semantic and episodic memory. After administering a battery of neuropsychological tests, Vicari determined that C.L. performed well in tests of visual naming and sentence comprehension, visual-spatial ability, and "general semantic knowledge about the world." They also noted an improved vocabulary and general knowledge base after 18 months. C.L.'s episodic memory, on the other hand, was far below expectations: She could not retain daily events, where she had gone on vacation, the names of places she had been, and other such information. However, this study and others like it are susceptible to subjectivity, since it is not always clear to distinguish between episodic and semantic memory. For this reason, the topic remains controversial and debated.

Familiarity and the fractionation of memory

It is clear that the right hippocampus is necessary for familiarity in spatial tasks, whereas the left hippocampus is necessary for familiarity-based recollection in verbal tasks. Some researchers claim that the hippocampus is important for the retrieval of memories, whereas adjacent cortical regions can support familiarity-based memories. These are memory decisions that are made based on matching already-existing memories (before the onset of pathology) to the current situation. According to Gilboa et al., patients with localized hippocampal damage can score well on a test if it is based on familiarity.

Poreh et al. describe a case study of patient A.D., whose damage to the fornix rendered the hippocampus useless but spared adjacent cortical areas -- a fairly rare injury. When the patient was given a test with something he had some familiarity with, the patient was able to score well. In general, however, A.D. had severely impaired episodic memory but had some ability to learn semantic knowledge. Other studies show that animals with similar injuries can recognize objects with which they are familiar, but, when the objects are presented in a context that is unexpected, they do not score well on recognition tests.

Islands of memory

Patients with anterograde amnesia have trouble recalling new information and new

autobiographical events, but the data are less consistent in regards to the latter. Medveds and Hirst recorded the presence of islands of memory -- detailed accounts -- that were described by such patients. The island memories were a combination of semantic and episodic memories. The researchers recorded patients giving long narratives with a fair amount of detail that resembled memories that the patients had prior to the trauma. The appearance of islands of memory could have something to do with the functioning of adjacent cortical areas and the neocortex. In addition, the researchers suspect that the amygdala played a role in the narratives.

Anterograde amnesia in popular culture

In both film and literature, anterograde amnesia is used for its (often puzzling) dramaturgical possibilities, and both its humorous and tragical implications, often as a plot device; more often than not, the story relies on the condition. Examples for all four are the films Memento (dramaturgy), 50 First Dates (humor), and the Star Trek: Enterprise episode "Twilight" (plot device/tragic - a tragedy averted when the cure turns out to be retroactive). Dana Carvey's character in the movie Clean Slate also had anterograde amnesia as a central plot point. The Indian action film Ghajini and its Hindi remake also uses anterograde amnesia as a central plot point, with the amnesiac protagonist played by Surya Sivakumar in the Tamil version and by Aamir Khan in the Hindi version.

Another fictional character who suffers the illness is the Professor in the novel The Professor and the Housekeeper by Yoko Ogawa. It is also suffered by Dory in the film Finding Nemo, by Liz Lemon's brother Mitch Lemon on 30 Rock, and by Chihiro Shindo from Ef: A Fairy Tale of the Two. Gene Wolfe's trilogy beginning with Soldier of the Mist is written as the diary of a soldier in the Greco-Persian Wars who suffers from the condition. The main character in Meg Gardiner's novel The Memory Collector suffers brain damage brought on by contamination with a material containing carbon nanotubes.