Retrograde Amnesia

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Retrograde amnesia (RA) is a loss of access to events and information of the past after the onset of disease or injury. RA is often temporally graded, consistent with Ribot's Law: more recent memories closer to the traumatic incident are more likely to be forgotten than more remote memories.

Brain Structures

The most commonly affected areas are associated with episodic and declarative memory such as the hippocampus, the diencephalon, and the temporal lobes.

The hippocampus deals largely with memory consolidation, in particular episodic memory. Its main responsibility is making information go from short-term memories into long-term stores. Amnesic patients with damage to the hippocampus are able to demonstrate some degree of unimpaired semantic memory despite loss of episodic memory due to spared parahippocampal cortex.

The diencephalon and the surrounding areas' role in memory is not well understood. However, this structure appears to be involved in episodic memory recall.

The temporal lobes are essential for semantic and factual memory processing. Aside from helping to consolidate memory with the hippocampus, the temporal lobes are extremely important for semantic memory. Damage to this region of the brain can result in impaired organization and categorization of verbal material, disturbance of language comprehension, and impaired long-term memory. Right side lesions in particular, result in impaired recall of non-verbal material, such as music and drawings. Difficulties in studying this region of the brain extend to its duties in comprehension, naming objects, verbal memory, and other language functions.

Plasticity of the brain is when rewiring occurs because of brain damage, finding different neural pathways and not relying on the damaged structures. Thus, the brain can learn to be independent of the impaired hippocampus, but only to a certain extent. For example, older memories are consolidated over time and in various structures of the brain, including Wernicke's area and the neocortex, making retrieval through alternate pathways possible.

Types of Retrograde Amnesia

As previously mentioned, RA commonly results from damage to the brain regions most closely associated with episodic and declarative memory, including autobiographical information. In extreme cases, the individual may completely forget who he or she is. Generally, this is a more severe type of amnesia known as global or generalized amnesia. However, memory loss can also be selective or categorical, manifested by a person's inability to remember events related to a specific incident or topic.

Temporally Graded Retrograde Amnesia

Recovery often takes place after the onset of RA, which the Standard model accounts for. It suggests that the hippocampal formation is only used in systematic consolidation for a temporary and short period of time, until long-term consolidation takes place by other brain structures. The fact that damage to the hippocampal formation no longer causes RA, suggests that other brain structures are able to function more independently. RA can also progress, as in the case of Korsakoff syndrome and Alzheimer's disease, due to the ongoing nature of the damage. It is very rare to find stable RA, since improvement generally takes place among patients.

Focal, Isolated, and Pure Retrograde Amnesia

These terms are used to describe a pure form of RA, with an absence of anterograde amnesia (AA). In addition, Focal RA in particular, has also been used to describe an RA situation in which there is a lack of observable physical deficit as well. This could be described as a psychogenic form of amnesia with mild anterograde and retrograde loss. A case study of DH revealed that the patient was unable to provide personal or public information, however there was no parahippocampal or entorhinal damage found. Individuals with focal brain damage have minimal RA.

Isolated RA is associated with a visible thalamic lesion. Consistent with other forms of RA, the isolated form is marked by a profound inability to recalled past information.

A pure form of RA is rare as most cases of RA co-occur with AA. A famous example is that of patient ML. The patient's MRI revealed damage to the right ventral frontal cortex and underlying white matter, including the uncinate fasciculus, a band of fibres previously thought to mediate retrieval of specific events from one's personal past.

Causes

The causal explanation of RA is still under investigation, however, it is commonly associated with head injuries or traumas, Korsakoff syndrome, and individuals suffering from AA.

The Three main models used to explain RA assume that the hippocampus is one of the main areas of the brain used in memory consolidation. During consolidation, the hippocampus acts as an intermediate tool that quickly stores new information until it is transferred to the neocortex for the long-term. The temporal lobe, which holds the hippocampus, entorhinal, perirhinal and parahippocampal cortices, has a reciprocal connection with the neocortex. The temporal lobe is temporarily needed when consolidating new information, as the learning becomes stronger; the neocortex becomes more independent of the temporal lobe.

Studies on specific cases demonstrate how particular, impaired areas of the hippocampus are associated with the severity of RA. Damage can be limited to the CA1 field of the hippocampus, causing very limited RA for a duration of about 1 to 2 years. More extensive damage limited to the hippocampus causes temporally graded amnesia for a duration of 15 to 25 years. Another study also suggested that large medial temporal lobe lesions, that extends laterally to include other regions produces more extensive RA, covering 40 to 50 years. These findings suggest that density of RA becomes more severe and long-term as the damage extends beyond the hippocampus to surrounding structures.

It is important to note that the common studied causes of RA do not always lead to the onset of RA.

Traumatic Brain Injury (TBI), also known as Post-Traumatic Amnesia

TBI occurs from an external force that causes structural damage to the brain, such as a sharp blow to the head, a diffuse axonal injury, or childhood brain damage (e.g., shaken baby syndrome). In cases of sudden rapid acceleration or deceleration, the brain continues moving around in the skull, harming brain tissue as it hits internal protrusions.

TBI varies according to impact of external forces, location of structural damage, and severity of damage ranging from mild to severe. RA can be one of the many consequences of brain injury but it is important to note that it is not always the outcome of TBI. An example of a subgroup of people who are often exposed to TBI are individuals who are involved in high contact sports. Research on football players takes a closer look at some of the implications to their high contact activities. Enduring consistent head injuries can have a negative impact on the neural consolidation of memory. In a study examining football players, it was found that players who just sustain head trauma on the field were better able to answer questions about plays made and players involved when they were asked immediately after the event (vs. a delayed period of time).

Specific cases, such as that of patient ML, support the evidence that severe blows to the head can cause the onset of RA. In this specific case there was an onset of isolated RA following a severe head injury. The brain damage did not affect the person's ability to form new memories. Therefore, the idea that specific sections of retrograde memory are independent of anterograde is supported. Normally, there is a very gradual recovery, however, a dense period of amnesia immediately preceding the trauma usually persists.

Traumatic Events

RA can occur without any anatomical damage to the brain, lacking an observable neurobiological basis. Primarily referred to as psychogenic amnesia or psychogenic fugue, it often occurs due to a

traumatic situation that individuals wish to consciously or unconsciously avoid. The onset of psychogenic amnesia can be either global (i.e., individual forgets all history) or situation specific (i.e., individual is unable to retrieve memories of specific situations).

People experiencing psychogenic amnesia have impaired episodic memory, instances of wandering and traveling, and acceptance of a new identity as a result of inaccessible memories pertaining to their previous identity.

Recent research has begun to investigate the effects of stress and fear-inducing situations with the onset of RA. Long-term potentiation (LTP) is the process by which there is a signal transmission between neutrons after the activation of a neuron, which has been known to play a strong role in the hippocampus in terms of learning and memory. Common changes in the hippocampus have been found to be related to stress and induced LTP. The commonalities support the idea that different variations of stress can play a role in producing new memories as well as the onset of RA for other memories. Also, the amygdala plays a crucial role in memory and can be affected by emotional stimuli, evoking RA.

Studies of specific cases, such as AMN, support evidence of traumatic experiences as a plausible cause of RA. AMN escaped a small fire in his house, did not inhale any smoke and had no brain damage. Surprisingly, the next day, he was unable to recall autobiographical based knowledge. This case shows that RA can occur in the absence of structural brain damage.

Nutritional Deficiency

RA has been found among alcohol-dependent patients who suffer from Korsakoff syndrome. Korsakoff syndrome patients suffer from RA due to a thiamine deficiency (lack of vitamin B1). Also, chronic alcohol use disorders are associated with a decrease in volume of the left and right hippocampus.

These patients' regular diet consists mostly of hard alcohol intake, which lacks the necessary nutrients for healthy development and maintenance. Therefore, after a prolonged period of time consuming primarily alcohol, these people will undergo memory difficulties and ultimately suffer from RA. However, some of the drawback of using Korsakoff patients to study RA is the progressive nature of the illness and the unknown time of onset.

Infections

Infections that pass the blood-brain barrier can cause brain damage (encephalitis), sometimes resulting in the onset of RA. In the case of patient SS, the infection led to focal or isolated retrograde amnesia where there was an absence of or limited AA. Brain scans show abnormalities

in the bilateral medial temporal lobes, including two thirds of the hippocampal formation and the posterior part of the amygdala.

Surgery

After a bicycle accident in his childhood, HM suffered from epilepsy which progressed and worsened by his late twenties. The severity of his condition caused HM to undergo surgery in an effort to prevent his seizures. Unfortunately, however, HM's surgeons removed his bilateral medial temporal lobe, causing profound AA and RA. The removed brain structures include the hippocampus, the amygdala, and the parahippocampal gyrus, now called the medial temporal lobe memory system.

Other patients who suffered RA due to surgery are PB and FC who had unilateral removal of the medial areas in the left temporal lobe.

Controlled induction

Clinically induced RA has been achieved using different forms of electrical induction.

Electroconvulsive therapy (ECT)

ECT, used as a depression therapy, can cause impairments in memory. Tests show that information of days and weeks prior to the ECT can be permanently lost. The results of this study also show that severity of RA is more extreme in cases of bilateral ECT rather than unilateral ECT. Damage can also be more intense if ECT is administered repetitively (sine wave simulation) as oppose to a single pulse (brief-pulse stimulation).

Electroconvulsive shock (ECS)

The research in this field has been advanced by using animals as subjects. Researchers induce RA in rats, for example, by giving daily ECS treatments. This is done to further understand RA.

Diagnosis and Treatment

Testing for Retrograde Amnesia

As previously mentioned, RA can affect people's memories in different degrees, but testing is required to help determine if someone is experiencing RA. Several tests exist, for example, testing for factual knowledge such as known public events. A down fall of this form of testing is that people generally differ in their knowledge of such subjects. Other ways to test someone is via

autobiographical knowledge using the Autobiographical Memory Interview (AMI), comprising names of relatives, personal information, and job history. This information could help determine if someone is experiencing RA and the degree of memory affected. However, due to the nature of the information being tested, it is often difficult to verify the accuracy of the memoirs being recalled, especially if they are from a distant past.

Brain abnormalities can be measured using magnetic resonance imaging (MRI), computed tomography scan (CT) and electroencephalograhy (EEG) which can give detailed information about specific brain structures. In many cases an autopsy will help to identify the exact brain region affected and the extent of the damage that caused RA once the patient has died.

There are some aspects essential to the patient that remain unaffected by RA. In many patients, their personality remains the same. Also, semantic memory, that is general knowledge about the world, is usually unaffected. However, episodic memory, which refers to one's life experiences, is impaired.

The legal system had started to push for a standardized test for amnesia. It is common for people who have committed a crime to report having RA for that specific event because they want to avoid their punishment. A standardized test would be able to identify individuals who are lying versus those who are truly suffering from RA.

Spontaneous Recovery

When someone is suffering from RA, their memory cannot be recovered from simply being told personal experiences and their identity. This is called reminder effect or reminder treatment. The reminder effect consists of re-exposing the patient to past personal information, which cannot reverse RA. Thus, reminding the patient details of their life has no scientific bearings on recovering memory. Fortunately, memory can be and usually is recovered due to spontaneous recovery.

Case Studies

Since researchers are interested in examining the effects of disrupted brain areas and conducting experiments for further understanding of an unaffected, normal brain, many individuals with brain damage have volunteered to undergo countless tests to advance our scientific knowledge of the human brain. For example, HM is someone with significant brain damage and participates in a lot of neurological research. Furthermore, he is also the most tested person in neuropsychology. All people who participate are referred to in literature using only their initials in order to protect the privacy of these individuals.

Patients with RA have exhibited an inability to describe future plans, whether in the near future

(e.g., this afternoon) or in the distant future (e.g., next summer) because of their inability to consolidate memories. Also, researchers have found these patients can identify themselves and loved ones in photographs, but cannot determine the time or place the photo was taken. It has also been found that patients with RA greatly differ from the general population in remembering past events.

A few case examples are:

After a head injury, AB had to relearn personal information. Many of AB's habits had also changed.
Patient CD, reported disorientation of place and time following his injuries as well as relearning previously learned information and activities (e.g., using a razor).
EF was examined and found to be very confused about social norms (e.g., appropriate attire outside his home). EF exhibited memory loss of his personal experiences (e.g., childhood), and the impaired ability to recognize his wife and parents.
JG is the first recorded patient suffering from isolated RA.
GH, a mother and a wife, had surgery in August 2002. When GH woke up after the surgery, she believed it was May 1989. Due to her amnesia, GH experienced great difficulty in her social environment, being overwhelmed by relationships to others.

Although it may seem that people living with brain damage have great difficulty continuing the usual day-to-day aspects, they still can accomplish many feats. People with RA are able to lead a normal life. For instance, KC is a man who has many functional aspects intact; normal intelligence, unaffected perceptual and linguistic skills, short-term memory, social skills, and reasoning abilities. All of these things are necessary in everyday life and contribute to normal living. KC also is fully capable of scripted activities (e.g., making reservations or changing a flat tire). In addition, patient HC successfully graduated high school and continued into post-secondary studies, an obvious accomplishment despite her condition.

Other Forms of Amnesia

Other forms of amnesia exist and may be confused with RA. For instance, AA is the inability to learn new information. This describes a problem encoding, storing, or retrieving information that can be used in the future It is important to note that these two conditions can, and often do both occur in the same patient simultaneously, but are otherwise separate forms of amnesia.

RA can also be an inherent aspect of other forms of amnesia, namely transient global amnesia (TGA). TGA is the sudden onset of AA and RA caused by a traumatic event, however it is short lived, typically lasting only 4 to 8 hours. TGA is very difficult to study because of the patients quick recovery. This form of amnesia, like AA, remains distinct from RA.