Memory
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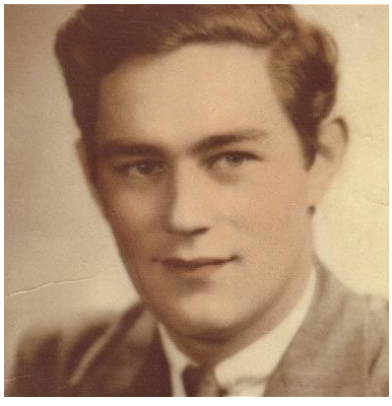
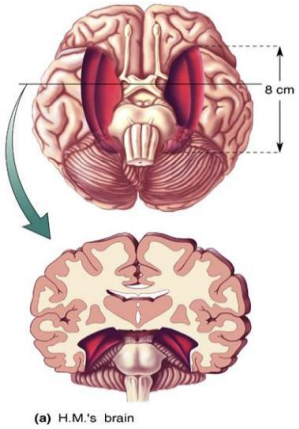
Lecture - 31
Memory Disorders- Amnesia-II

Hello, I welcome you all to the lecture series on memory. As we were discussing memory disorders in the previous lecture, we talked about amnesia. The problem of amnesia, how it starts, and the types of amnesia. In today's lecture, we are going to address different case studies related to amnesia and understand in more detail what is really happening with these individuals. What is really happening with memory?

What is really happening when memory formation occurs? It gets disrupted after brain trauma. In the previous lecture, we also discussed brain structures such as the diencephalon, medial temporal lobe, adjacent areas of the medial temporal lobe, and their contribution to memory formation. Now, the classical case study of Henry Molaison has provided a lot of insight into understanding the nature of memory, particularly the formation of new memory.

Henry Molaison, a young individual, 27 years old, was suffering from epilepsy. He was having difficulty performing day-to-day activities. Due to his high number of epileptic seizures, it was very difficult for him to live a normal life. So he went to the doctors, and they recommended that one solution to this problem was a lobotomy.





(a) H.M.'s brain

Lobotomy at that time was very popular, so they just recommended the only solution to reduce the number of seizures was lobotomy, and what could we do about it? We will just remove a small piece of brain from his brain, and this will reduce the seizures. He

will not have any problem with the seizures. The doctors were correct; they performed the surgery and removed a piece of the brain, the medial temporal lobe, including the hippocampus and the amygdala regions. Now, after the removal of an 8-centimeter brain region, the individual was fine, with fewer epileptic seizures. He was able to execute tasks, but as he recovered from the surgery, a new problem started to emerge. This new problem was that he became amnesic, severely amnesic of events. He was not able to form new information. Lots of information was there. Anything he was encountering or experiencing in reality, he was not able to form.

Unlike the previous information, he had the preserved information, old information prior to the injury, but the new information got impaired. The old information was intact, but the new information got impaired. He was not able to consolidate or store this information for future reference.

His inability to learn and retain any new information about facts and events gave us a lot of insight into the importance and relevance of these brain structures. The brain structures which were removed from H.M.'s brain. The role of the medial temporal lobe, the role of the hippocampus, the role of the amygdala. The connection and projection of these brain regions and these neural correlates give us insight into how Henry, in the Henry Molaison case,

or Henry Molaison, was unable to form new memories. H.M.'s personality, perception, and intelligence, however, didn't change. They remained intact. So one more conclusion came from this: the structures which were surgically removed were only responsible for storing information, particularly storing new information. So the type of amnesia which he was suffering from was anterograde amnesia. Now, in retrograde amnesia, what we realized is that the information before the surgery was preserved.

And the area which was removed had nothing to do with personality, nothing to do with intelligence, and nothing to do with perception. He did not have problems even with short-term memory or learning new motor skills. But the information related to episodic memory was more impaired or severely impaired than semantic memory. His episodic memory was more impaired, leaving the semantic memory intact. He was unable to recall the current date, where he was living, what he had for breakfast, or whom he may have met a few minutes earlier.

His retention capacity—how long he can remember or store information—is limited. 30 seconds. Beyond 30 seconds, it becomes challenging for him to retain information. So, he

was suffering from endocrine amnesia. Now, to study endocrine amnesia, certain drugs like benzodiazepines are used.

It has been shown that they can create artificial or temporary amnesia resembling the amnesic syndrome. After taking the drug, the individual is unable to form new memories. The individual can have a conversation. The individual can recollect information. Retrieval is not intact.

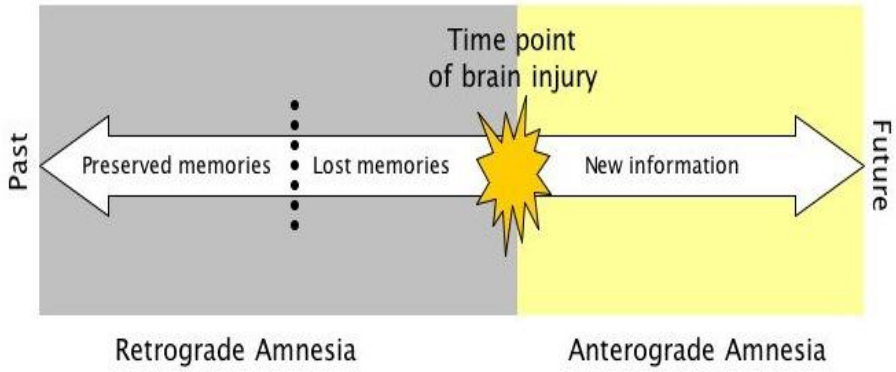
But forming new information is a challenge for these individuals. In many instances, unlawful acts have been performed using this drug, benzodiazepines. That is where people have understood that the drug resembles the amnesic syndrome. In some experiments, people have used these drugs to understand the concept of amnesia. In some cases, to relieve patients from anxiety or stress-related disorders, benzodiazepines seem to have a beneficial role.

However, transient amnesia is very common in these individuals. On many occasions, people feel that they cannot recollect information. There is a loss of new memory formation. But this is very transient, lasting for a very short period of time. Under the influence of this medication, they can converse normally and retrieve well-known information.

There is no such problem for these individuals to recollect information. They cannot encode new information, particularly into episodic memory. Patients who develop transient amnesia following the administration of benzodiazepines are unaware of their episodic memory deficit. They do not know that there is a certain deficiency in their memory formation. After the administration of this drug, the individual is not capable or able to understand and be aware that there is a memory deficit which has occurred during that period.

And that is why, as I have mentioned, many unlawful acts have been performed using similar kinds of drugs. This kind of transient amnesic episode or episodic memory deficiency is known as anosognosia. The failure to become aware of a cognitive deficit, where the individual is not aware that they may be having a memory deficit under the influence of this drug administration. So, as we have been discussing in the previous lecture about one form of amnesia, that was anterograde amnesia, today we will be talking about retrograde amnesia. Now, we know that anterograde amnesia, when we were talking about it, we discussed the Henry Molaison case, where he was incapable of forming new information.

But what is happening with the information that is stored? Is there a condition, is there a case where the previously stored information becomes impaired, leaving the new memory formation intact? Which means that you can easily form new information, but when the retrieval of previous information is required, we are failing. So as you can see in this cartoon, when the trauma point arises, the new information problem leads to the intricate amnesia, but when the retrieval of information is required, Preserved memory, recollection.



Source: https://fnfdoc.com/amnesia-types-symptoms-causes/

Recollection of preserved memory is there. Failure of recollection is known as retrograde amnesia. More than that, what we have even seen in retrograde amnesia, or even in the retrograde amnesia, a partial loss of memory is there just before the trauma. Just before the trauma, there is a loss of information. Why?

Because of the loss of trace consolidation. The trace consolidation gets affected because of the trauma. Any traumatic event, any traumatic event, it affects the trace consolidation. So when we are talking about the preserved memories, we are talking about the old memories. And this stress consolidation, which is getting affected or influenced by the trauma, could go up to one year.

So, an individual may not be able to recollect information dating back to one year. Even those individuals who are having anterograde amnesia. So, this duration can go up to one year, approximately. So, they cannot recollect the information about the search. Now, but when we are talking about retrograde amnesia, we are talking about preserved memory which is way older than one year.

So, it extends back to a particular point in the past. People with retrograde amnesia will have a period from their lives from which they cannot recall any specific events. They cannot recollect the information at all. When given an autobiographical quiz, all of their reported memories will come back from a different period of their life. Then the ones which are the retrograde amnesia.

Now, it is very important for us to understand that the preserved memories which we are talking about become independent of these structures. So, it doesn't depend on the bone structures. So, in the case of Henry Molaison, preserved memories were not dependent on the middle temporal lobe, hippocampus, or amygdala, which were being removed. So, it was dependent on the other associated structures like the frontal cortex, motor area, visual area, and many more areas, parietal areas. So, once the memory structures were being removed, still Henry Molaison was able to recollect information, the information which had become independent of his structure.

There is a very curious case of retrograde amnesia about Clive Wearing. Now, Clive Wearing was a singer, piano player, conductor, and composer. He also gained fame because he played the music a British musician, who played music at Princess Diana and Prince Charles' wedding. So he gained fame from their wedding, and after that, he was also known as a well-known pianist.

But the problem, what else is he known for? He is also well-known for the interesting case of memory loss, amnesia. He had a severe disease or infection, which was viral encephalitis. Because of this viral encephalitis, he was affected by the virus, which affected his brain in such a way that he developed retrograde amnesia. And he was also suffering from retrograde amnesia.

So it was very difficult. That is why it was a very curious case. Because in Henry Molaison, we understood that he was suffering from anterograde amnesia. But Wearing was suffering from both types of amnesia. So neither his past was okay, nor his future was okay.

He was living only in the present. And even while living in the present, though he was maintaining a diary, his storage of information was less than 6 seconds. And he lived with this disease, with this disorder, for 30 years. So in 1985, when he contracted the viral encephalitis disease, massive swelling of the brain occurred, which damaged his medial temporal lobe and prefrontal lobes. Now we know, as we have discussed in

previous lectures, that the hippocampus has dense projections to the amygdala and to the frontal cortex.

And if damage to these areas occurs, then the projections will still be there, but they will not be receiving any input from these regions. So, if this is the prefrontal cortex, and this is the hippocampus, and this is the amygdala, then though the projections will be there, if these areas are very unique. If these networks get broken or damaged, then the failure of memory loss or amnesia may emerge, and that is exactly what is happening in these individuals. So here, the damage occurred in his medial temporal lobe and prefrontal lobes. We don't have severe anterogradegate amnesia.

He had not learned a single new thing about the world in 30 years. Where Henry Mollison was preparing a diary, a new rehabilitation technique, a tool to preserve the information, to preserve the information by documenting it. But with viewing, it was a little challenging. He also had severe retrograde amnesia. So he was not able to remember a single event from his past.

So he was not able to recollect that he played music at the Princess Diana and Prince Charles wedding. He lived in a perpetual present. Only the present he was living in. His working memory maintained information for about 10 seconds. As I said, 6 seconds.

Roughly around 6 to 10 seconds, he was able to maintain the information, and after that, everything was lost. It's like you have just opened the tap, and your tank is empty again. You are filling, and the tap is open. You are filling the water in the tank, but the tap is open, so there is no storage of water. Exactly, this is what was happening with the cleaves also.

This process, where new memories are more affected by retrograde amnesia than older memories, is known as Ribbitt's law. Now, as we were talking about the cleave and as we discussed earlier about this aspect where the trauma has happened and this loss of memory is happening up to one year. This is known as Ribbitt's law. What is this Ribbitt's law? It's very simple.

Trauma has occurred here, T. So this side is anterograde amnesia, and this side is retrograde. What people observed is that when there is trauma, the recent memory The recent memory close to the trauma seems to be affected by the trauma itself. So this trace, this trace of memory, is affected. It didn't undergo consolidation.

This has undergone consolidation from here onwards. But this has not undergone consolidation. New memories are more affected by retrograde amnesia than older memories. So these memories, new memories, are more affected by retrograde amnesia. Memory consolidation is disrupted in retrograde amnesia.

So retrograde amnesia interfered with or disrupted the consolidation process of the memory. So the traces of memory which are undergoing consolidation got interfered with or damaged by retrograde amnesia. As a result, the newer memories got unconsolidated. They didn't get stored at all. And in this case, a well-known example is Trevor Rees-Jones.

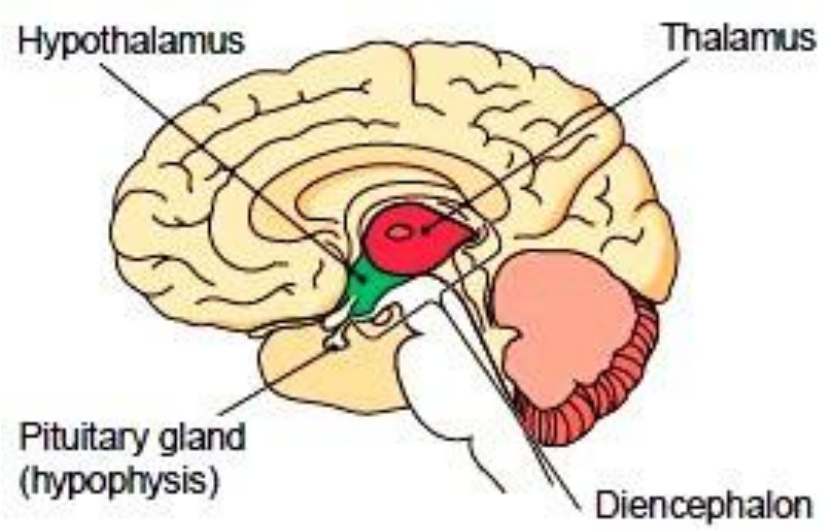
Trevor Rees-Jones was Princess Diana's bodyguard. He was the only survivor in that severe car crash. After the trauma, when he was repeatedly asked what happened and how it happened, he was unable to recollect the details of the incident and the time before the incident. Trevor survived the car crash but demonstrated a unique form of amnesia. This unique form of amnesia meant he was unable to recollect the details of the incident and the period when the incident occurred, whether it was a few minutes or a few hours ago. However, he was able to recollect information about that event. What happened?

After that event, how they collectively got into the car and were then chased by reporters. But then what happened? Then he was unable to recollect. In some cases, this retrograde period is somewhat longer. It could be some days, even months, or it could extend up to approximately one year.

As a person recovers from trauma, provided there is no permanent brain damage, the retrograde period shrinks. Older memories return first, followed by more recent memories. This is because older memories are more preserved and have become independent of memory structures. Since these older memories are independent of memory structures, their projection slowly comes to light. However, the more recent ones, along with their associated regions, were affected.

When we talk about the brain regions in retrograde amnesia, the brain regions that are important are the diencephalon and the brain stem, which sends information from the brain to the peripheral nervous system. Frontal lobes, which are important for the central executive system, decision-making, etc., and the medial temporal lobes. Now, what we understand is that damage to the hippocampus may still preserve memory, and people may be able to retrieve information. Because in retrograde amnesia, the previous

information is lost, but if the hippocampus is intact, people may still not be able to recollect information.



Source: https://www.toppr.com/ask/question/describe-briefly-the-diencephalon-of-human/

Because the hippocampus is required for the initial formation of memory. So, for the new formation of memory or for new memories, the hippocampus is essential. But for old information, the hippocampus is not necessarily required. But in that case, researchers still believe that the medial temporal lobe has a significant role here.

Now, it is very important for us to understand how amnesia can also come into existence. First, we were talking about benzodiazepines and the administration of this drug. The other part is electroconvulsive shock therapy. which seems to be an effective treatment for some neurological disorders, such as depression. In this case, a person is given a high amplitude of current; a strong electric shock is administered to the head of the patient to short-circuit the firing of the neurons. In the case of schizophrenia and depression, it seems to be an effective treatment. What happens after repeated short-circuiting of your nervous system is that you are creating greater amnesia.

The person is not able to recollect information. The person loses the information. A high number of ECT sessions results in a high amount of information loss. The more ECT is given to an individual, the more information loss is seen, particularly retrograde amnesia. The formation of new memory information is not impaired at all.

People seem to overcome depression, but along with that, they lose information about the episodes. Here, semantic information also seems to be less impaired with ECT. It creates a period of reticulation, and in most cases, it wears off once the sessions are complete. But usually, it remains intact for the ECT procedure itself. Somehow, the speculated memory loss itself creates the improved mood.

So when ECT is given, and when information loss happens, this actually elevates the individual. This elevates the mood of the individual because there is no information left to bother them, to confuse them, or to put them under stress. And the patient can no longer recall what is depressing them so much. So ECT seems to be a very effective tool. However, the side effects include memory loss.

Now, as we were discussing ECT, electroconvulsive shock therapy, there is another procedure where there is no loss of memory, but it improves memory, and that may be interesting for some of you to look into: transcranial electrical stimulation. It is quite the reverse of ECT. Here, when we are giving a high amplitude of current, here we give a low amplitude of current. And it seems that it has a beneficial effect on depression and many other neurological disorders. Without resulting in amnesia.

Okay. So, let me conclude what we studied in today's lecture. We discussed the Henry Molaison case, which gave us a lot of insight into anterograde amnesia, why it happens, the structures, the associated structures, how HM's personality, perception, and intelligence remained intact after the lobotomy. We even discussed how Artificially, we can induce amnesia, that is, benzodiazepines.

Administration of this drug leads to transient anterograde amnesia for a period of time where retrieval of information is not impaired, but the formation of information is impaired. Retrograde amnesia, where we discussed that the retrieval of previous information is impaired because of trauma. And we discussed the curious case of Clive Wearing, where he was suffering from retrograde and anterograde amnesia at the same time, severely impaired, memory loss is there, living in the present. We even discussed that after trauma, retrograde amnesia targets recent memory and affects the trace consolidation of recent memory. That is its law.

And then, brain regions which are responsible for retrograde amnesia, the cerebral cortex, and the medial temporal lobe. And finally, we discussed how electroconvulsive shock therapy, which is an effective treatment for depression, etc., seems to result in amnesia.

With this, I will end today's lecture. And in the next lecture, we will continue our discussion on memory disorders. Thank you.