Memory

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Lecture - 32
Memory Disorders- Amnesia-III

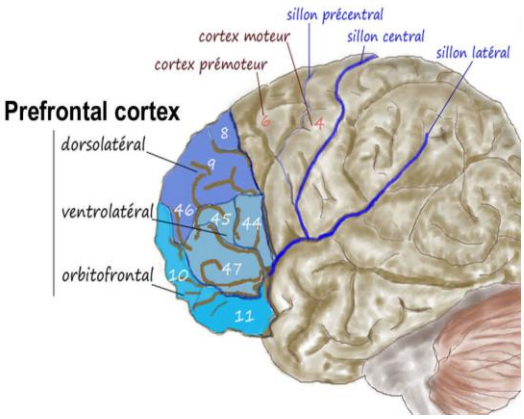
Hello, I welcome you all to the lecture series on memory. As we have been discussing in the previous lecture on memory disorders, we tried to investigate and understand the root causes of memory disorders. There were two case studies which we discussed in detail: the Henry Molaison case and the Clive case. And we tried to understand what the root causes were. These two case studies gave us a lot of insight into memory formation, encoding, storage, and retrieval problems.

Now, what we understand is that there are different types of amnesia, and sometimes amnesia can be caused by the artificial administration of drugs, such as benzodiazepines. While going through all those case studies, we also understood that there should be other forms of interventions which may lead to amnesia, and one such intervention is ECT, electroconvulsive shock therapy. Electroconvulsive shock therapy is very effective for depression. However, it leads to amnesia. The high amplitude of current

short-circuits the entire brain. As a result, amnesia is a problem. Though it is a highly used and very successful intervention for depression, it eventually results in amnesia. So, we need a method where the loss of information should not occur, but the treatment can still be implemented. Now, taking this further down the line, we also discussed different parts of the brain which are responsible for memory disorders. And one such part related to memory disorders is the frontal syndrome. Now, when we talk about the syndrome, multiple symptoms are present which result from the frontal lobe and indicate memory loss. And this we have seen in several cases. If you all remember the case of Phineas Gage, the classic case where a railroad worker had a portion of his frontal cortex removed.

His personality changed. His other forms of cognitive abilities also changed after the accident, after the trauma. So, frontal syndrome, if we talk about it, is characterized by damage to the frontal lobe. Now, we know that the entire brain is divided into four different lobes: frontal, occipital, temporal, and parietal.

It is very important for us to understand that in this case, the frontal lobe is a large region in the brain, and its projections, connections, and dense networking with other lobes play a crucial role in memory formation, memory retrieval, and trace consolidation. Now, when we talk about damage to the frontal lobes, behavioral symptoms include retrograde amnesia, amnesia, and confabulation, where people try to create memories to fill the gaps due to the loss of information, and all these things have been observed. Confabulation is seen very commonly in frontal lobe damage and in cases of frontal syndrome as well. Confabulation has also been seen in many cases like Korsakoff syndrome. Last time when we were having a discussion, we also discussed how confabulation is a common problem in these individuals.



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Because of chronic alcoholism, these individuals do not remember information for a while. And the information they do not remember, they try to fill those gaps with some information. The source of amnesia has been an important concern here, and what we have understood from empirical evidence is that the frontal lobe has been a major contributor to memory, particularly retrograde amnesia. In retrograde amnesia, the frontal lobe plays a major role. So when this happens, people forget the information.

Prior to the incident or the trauma. But the frontal lobe alone is not responsible in that regard either. One has to understand that the frontal lobe, whenever we are discussing the frontal lobe, we have to consider three different types of brain regions: the dorsolateral prefrontal cortex, the medial prefrontal cortex, and the ventromedial prefrontal cortex. The orbital frontal prefrontal cortex and the ventromedial prefrontal cortex. And all three of these brain regions are present on the left and right.

Now, we know that it acquires new information. The frontal cortex is important in the consolidation of new information. Why? Because this is the primary site of information. And it has dense projections and networking with the hippocampus.

So, the frontal cortex and hippocampus, when they are removed or damaged, then the initial loss of information and the initial formation of information are also compromised. And also, we have to understand that that we also have to understand that at the same time, they assist in the formation of new information, and whatever we have heard or recalled earlier is also being compromised. Which means that, as we were discussing, retrograde amnesia and anterograde amnesia are both dependent on the frontal cortex. Some aspects of specific types of memory, such as semantic memory and autobiographical memory, are also affected.

Because the sequence of events, which we are talking about and which is crucial in autobiographical memory, what should be the sequence of information? That seems to be compromised as well. The frontal cortex, as we discussed earlier, is a central executive system. And when the central executive system is present, it involves the phonological loop and the visual-spatial sketchpad all together. And it ensures the binding of information.

So, when we are talking about Frenzel syndrome, these are the symptoms that we have seen. As a result, this Frenzel syndrome arises. Now, what we also... know clearly is that the initial formation of new information is sometimes compromised and sometimes not compromised. So, both anterograde and retrograde amnesia are dependent on the frontal cortex.

Now, here, difficulty in organizing the events in the correct order also. So, let us take an example. The easy example could be the Prime Minister of India, the current Prime Minister of India. And the previous Prime Minister of India and the Prime Minister of India at the time of independence. So, if these three Prime Ministers of India are there, then we have to organize these events accordingly, in the correct order.

For example, we can also say, arrange the ages of the family members in ascending order. So, in that case, we require the information about the ages from all the family members, and then, whosoever is, you know, the youngest, we start from there and then we go to the oldest individual. This deficit also includes, as we have been discussing, not only the arrangement but deficits also include the semantic and autobiographical. One has to

understand clearly here that the deficiency... Or the deficit in the frontal lobe results mostly in the episodic memory.

And semantic memory is being compromised because of the other association networking. And either one of these will be damaged or collectively both will be damaged as well. So, the memory functions, the associated structures, dorsolateral, ventrolateral, ventrolateral prefrontal cortex, dorsolateral prefrontal cortex, but the reward system is also involved. Anterior cingulate cortex. This anterior cingulate cortex ensures the exchange of information.

The reward system. Punishment and reward. Now, earlier we have been discussing that there are certain drug administrations which lead towards amnesia and give a scope to the researchers to investigate. How exactly the memory formation is happening, how the loss of information is impacting the individual's day-to-day activity. And this amnesia is transient in nature, which means it is there for a very short duration. As the drug effect fades away, the amnesia is gone, but as long as the drug is there in the system and the administration of that from the point of administration.

The individual faces the loss of information. It is very difficult for the individual to store the information. Now, what we see here is that it is a rare form of amnesia where the amnesic effects are short-lived, usually on the order of hours. As the drug administration is there, the effect of the drug is there. As it fades away, the amnesia fades off. So, if this is amnesia. And this is the drug effect.

So, as time passes by, the drug effect will reduce, and this will be there. Just one second. So, this has to be the drug effect, and this will be amnesia. So, it also reduces. As time passes by, so amnesia is there, but it is less.

Now, when we are talking about transient global amnesia, it is characterized as anterograde amnesia, which means that after the surgery or after the trauma, after the incident, people fail to form new information. And that has been seen. After the drug administration has been performed, it seems that the effect is there. So, as the drug level decreases in the bloodstream, the amnesia decreases. This is what we have seen now. Even what we also know is that from the point of trauma, this is anterograde, and this is retrograde. What we are seeing is that it is a dense anterograde amnesia observed till here because the drug effect is still high. The amnesia is very dense in nature.

But as we move forward, the effect of retrograde amnesia decreases. So there is no more loss of memory which has been seen. TGA patients may also suffer from retrograde amnesia from a few hours to several years into the past, as we were discussing. Previously, we were also discussing that there will be a loss of information up to one year. Though the retrograde amnesia has occurred after the trauma, the retrograde amnesia is preserved.

But still, some amount of information loss is there. This some amount of information loss can vary from a few hours to several years. And that is what we have also seen in the previous lectures, the bodyguard of the incident which happened with Princess Diana. He was able to recollect some information about the event, but a few hours before the incident, he was completely blank. He was not able to recollect the information.

There is no loss of personal identity, as we have seen in this case. The person remembers the information about their identity, where they live, what they do, where they belong to; all that information is there. But some aspects of cognition appear to be both unimpaired and impaired. So the cognition that is unimpaired is that if you ask the individual about an arithmetic problem, or a grammatical problem, then the individual may respond to it because such amnesia doesn't affect their retrieval of long-term information, but it seems to affect short-term memory information.

The short-term memory information is at that present moment, so the previously learned information seems to be unimpaired. But the short-term memory seems to be impaired. It has also been seen that transient amnesia tends to occur in people from middle age to old age, 50s to 60s, and then decreases among older adults. Now, there could be several reasons for this. Some people believe that it is due to lifestyle conditions.

Some people believe it could be due to hypertension. Some people believe it could be due to an aneurysm. So, there are several examples and underlying causes for it. And that is why it is not so clear what the correct etiology of TGA is. What is the clear underlying cause of TGA?

So transient global amnesia, if you try to understand why it could be there, then some people have suggested that it could be a disruption of the blood flow within the brain. So from the brain to the heart, any form of blockage may result in this transient global amnesia. And once this blockage is cleared from the brain to the heart, then the amnesia fades away. So, if there is a temporary disruption in the flow from the vein to the heart, it results in ischemia.

And as a result of this, there is amnesia. Sometimes compression of veins is there, preventing the proper flow of blood from the brain back to the heart, and this has also been suggested as a potential cause. So this simple blockage, if it is in the brain and the brain stem, then the blockage, the flow, the blood flow seems to be the underlying cause of it. Any pressure, any exertion on these blood veins, because they are very thin, may result in amnesia.

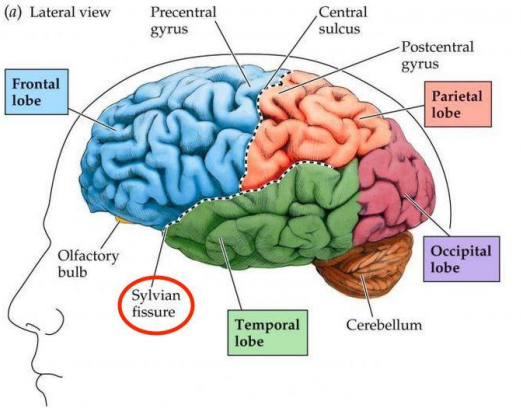
As the pressure gets released, the amnesia fades away. Now, however, it is still not so clear that this could be the underlying root cause. What we really understand about amnesia is that long-term memory in one case gets affected, and in another case, the long-term memory is not affected, but the short-term memory is affected. Now, whenever short-term memory amnesia is present, we say that the person is incapable of forming new memories.

And this made researchers understand what the underlying cause of such amnesia is. Amnesias are of different types. Though we started the classification with retrograde and anterograde, since then we have seen that retrograde and anterograde exist, but along with retrograde and anterograde, there is a frontal syndrome. TGA is there, transient global amnesia is there, and artificially you can create amnesia as a result of drug administration, which is highly common. And that is why many times individuals recommend that high-potency drugs should be avoided.

Because the side effects could be amnesia or any other form of cognitive disability. Now, when we talk about short-term memory amnesia, we are saying that long-term memory is affected. But in anterograde, the short-term memory seems to be impaired. Long-term memory is intact. So, Shallice and Warrington in 1970.

They started their investigation and tried to determine what could be the brain region responsible for amnesia. Of course, by this time, we know that the frontal cortex is an important region, the hippocampus is an important region, the amygdala is an important region, and the brain stem is an important region. However, it is still unclear. What are

the specific brain regions? So there is a case study which Shallice and Warrington studied, KF, a young individual who met with a motorcycle accident.



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This KF, who suffered from a motorcycle incident, showed a selective impairment of short-term memory. His long-term memory was intact. Long-term memory was intact. But what got affected? It was the short-term memory.

It got impaired. What we saw is that the individual was able to perform other types of cognitive tasks. Retrieval of information, episodic information, and semantic information were correct. Language problems were fine. Comprehension and reading tasks were fine.

However, he had difficulty with the digit span task. The digit span task, the same digit span task where we studied that the individual has to remember the digits, which increase in order, and the individual has to recall them. He just had to recall the series of numbers. As the number increases, the individual has to recall these digits. A normal healthy individual can recall 7 plus or minus 2.

This we have discussed earlier also. The magical number. So in this digital span task, he was unable to maintain 2 digits. More than 2 digits, it was very difficult for him to manage. So there are two forms of memory which the frontal cortex is dealing with, which we discussed earlier.

Phonological loop, visual spatial sketchpad. The phonological loop deals with auditory memory, and the visual spatial sketchpad deals with spatial memory or visual memory. And in this digital span task, when tested in auditory modality and when tested with visual presentation, the visual representation was intact. We didn't have any problem with

that. But in his digital span task, where the auditory modality was involved, it seems that he was impaired in that.

So his auditory memory phonological loop seems to be affected after his incident, while his visual memory was intact. So this was a unique case, if you all recall the working memory model proposed by Baddeley and Hitch. We were discussing the visual memory, auditory memory, the buffer, and the central executive system. So here the input which is coming from the auditory memory seems to be impaired.

Extensive damage to the frontal lobe. Of the left hemisphere. So the right hemisphere did not have as much damage, but the left hemisphere, this side of the brain, got damaged. The localization of the lesion was the Sylvian fissure, which connects the frontal and temporal areas, separating the frontal lobe and temporal lobes. So this Sylvian fissure, which we are talking about, is something that has been impaired. Now, this is an area of the brain closely associated with language formation and processing.

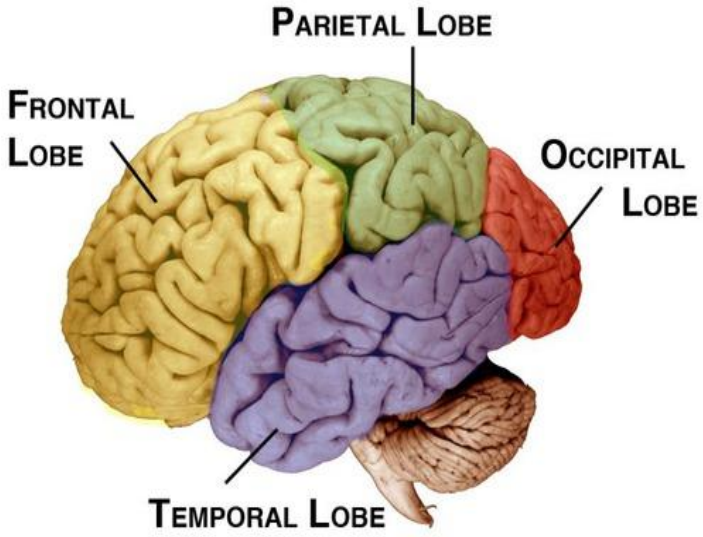
And now, in the case of dyslexics, in the case of learning disability, the Sylvian fissure seems to be an important brain region. Which is trying to separate these brain regions. So what we understand from the case of KF is that his short-term memory amnesia was present, but in the short-term memory amnesia, his phonological loop or auditory memory seems to have been affected the most. As a result, he was not able to perform the two-digit task. The deficit might have been limited to the phonological loop, with less damage to visual working memory.

The visual-spatial sketchpad was fine, but the phonological loop was having a problem. Now, another form of amnesia that came to light was re-duplicative paramnesia. What is re-duplicative paramnesia? In these cases of amnesia, people seem to have places or locations that have been duplicated.

For them, the two locations exist simultaneously. What has been observed in this case is that the patient agreed that the hospital room looked just like the one found in the hospital in their local city. When in fact, they were in their local city, though the doctor said, 'This is your city.' The doctor said, 'This is your city hospital, and the vision which you are having outside is of your own city,' but they denied this. They had this denial that, 'No, this is not my hospital, this is not my local city, this is somewhere else; I am here for the treatment.'

Though the patient agreed that the view out of the window revealed a view of the town just like the small city near his home, the patient insisted that the hospital was a different one and the city as well. So, this patient recognized the surroundings; the patient could recognize the surroundings but lacked the feeling of familiarity. So here, the concept of familiarity came into existence. So, when the person is familiar with the location, the person is familiar with the place but is unable to implement that information at the present moment, it seems to have a problem with familiarity, and as a result, this form of amnesia, we are calling it reduplicative paramnesia because there is duplication of the two places.

So, here is traumatic brain damage. So, Hinkebein in 2001 tried to investigate these two neurological deficits that may create reduplicative paramnesia. The first thing is damage to areas in the right parietal lobe. Now, the parietal lobe where? Whenever we have to look for an object, where it is being placed, then this pathway we are referring to is the 'where' pathway, and this pathway we are referring to as the 'what' pathway.



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So, where and what pathway plays a crucial role, and they are moving from the occipital lobe to the parietal lobe. So, what we see here is that it creates impaired visual perception and visual memory. Now, the visual-spatial sketchpad is giving an input to the central executive system, which is the frontal lobe, about the visual information. In the previous case, in the KF case study, we were talking about the auditory memory problem.

So, the central executive system is not receiving an input from the phonological loop. Leaving the VSSP intact, but here, what we are seeing is that the VSSP is impaired,

leaving the PL intact. So, it creates a feeling that well-known places are not familiar. The familiarity effect seems to be a problem. It induces the experience that familiar places are something peculiar or different in nature.

What is that peculiarity? What is that unique characteristic or feature which these places are having that is playing a crucial role here? And the second part is the bilateral damage to the prefrontal lobe. So, one part is talking about the parietal lobe, and then another part is talking about the frontal damage. But in the parietal, we were talking about the right parietal.

But in the frontal, we are talking about both sides, bilateral. Both sides of the pharyngeal damage are there. So when both sides of the pharyngeal lobe damage are there, it may not be sending projections to the parietal area, temporal area, and occipital area. And because it is damaged, the projection is not there. Projections to the parietal, occipital, and temporal areas are all damaged.

So neither the Inflow is there, nor the outflow is there. Both got impaired. Both got impaired. So this is actually resulting in the deficit and creating an illusion.

Another important part prevents the individual from experiencing familiarity because of this. The circuit is unfamiliar or dismissing its lack as an illusion. So there is no illusion. Researchers also talked about the impairment of visual familiarity. So visual familiarity is there because the projection from the frontal cortex to the occipital seems to be damaged.

What we see in this reduplicative paramnesia is that the region, frontal cortex or parietal cortex, plays a crucial role. And in this case, basically, the visual memory is being affected rather than the auditory memory. Now another form of disorder is the Capgras syndrome. Now the Capgras syndrome is unique in its nature. When we were talking about the familiarity with places or locations, here we are talking about the familiarity with people.

And in this, patients have a belief that people have been duplicated. People have a clone. People are imposters. It occurs when a patient believes that his family or her family members have been duplicated and replaced by imposters or robots. And this is quite seen as a form of memory disorder.

Capgras syndrome is associated with psychiatric problems and particularly with schizophrenia. And here, people become aggressive and also hurtful. It often occurs in conjunction with face recognition, prosopagnosia, where they face problems. In seeing

the faces, and they even many times come up with the conclusion that the person whom they are sharing the house with, whom they are sharing life with, is an imposter. And after medication and treatment, still, this problem emerges.

So there is no clear medication for Capgras syndrome or reduplicative paramnesia. After the treatment, it seems that these individuals still have similar memory problems after the effect of the medication fades away or the medication stops. Cap-Carr syndrome arises from a breakdown in the connection between the temporal lobe areas and the temporal lobe area when we were discussing it. And the limbic system involved in emotion. Now the idea here one has to also understand is that this syndrome is not only limited to people, but some patients seem to have extended it from people to animals also.

So the individual was also having a problem with his cat. Now a 73-year-old man was suffering from retinitis pigmentosa, which left him nearly blind, Parkinson's disease, and Capgras amnesia. He was suffering from visual hallucinations at the age of 71. Strange people approached his wife, met his wife, and might have replaced his wife.

And he even believed that his wife was being replaced with men and women who dressed to impersonate her, who tried to take the identity of his wife, and he is living and sharing his life with the person who is not his wife. He believed that he could always tell the difference between his wife and the imposters. When the effect of the drug and the treatment was okay, but after the effect faded away, it seems that they were again having similar problems. Various changes to the medication were made, but there was no success. To date, there is no effective treatment for Capgras amnesia or reduplicative amnesia.

Then a scan revealed minor scattered damage in the frontal lobes. But nothing is measured. But then nothing is measured. Now, the question which arises is that this frontal lobe, which is there, seems to have been divided into three regions. DLPFC, OFC, and VMPFC.

All three seem to be major contributors, sending projections to different parts of the brain and, at the same time, receiving projections from different parts of the brain. So, what have we studied here? We tried to understand these symptoms, several symptoms resulting in the Frenkel syndrome. And in the Frenkel syndrome, we understood that the phonological loop, visual-spatial sketchpad, and retrograde amnesia seem to be a problem, along with semantic memory problems, autobiographical memory problems, and the challenge of arranging events. All of these things result in Frenkel syndrome.

We also understood that new memory formation may not be affected that much, but the retrieval of old information may be affected. However, in some cases, we have seen that the formation of new information is also affected, leaving the retrieval of old information intact. We discussed transient global amnesia, that its effect is transient, And the etiology of transient global amnesia is still not clear to us. It could be because of ischemia, or it could be because of an aneurysm, or any blockage or disruption which is happening in the blood flow from the brain to the heart or vice versa.

Short-term memory amnesia, we understood that anterograde amnesia is basically the problem after the trauma has arisen, after the trauma has occurred. The anterograde amnesia and retrograde amnesia, and what we are talking about is the formation of new information being affected, leaving the long-term memory intact. And even when we are talking about short-term memory amnesia, we are also talking about the case study of KF, where he was able to seem to have affected his phonological loop auditory memory, leaving the visual-spatial memory or visual memory intact. And then we talked about reduplicative amnesia, where people are having familiarity problems; the places and locations seem to be duplicative in nature and coexist.

Then we discussed that this reduplicative amnesia, which is talking about the places and location duplication, People similarly have extended this and have a problem with people amnesia. So, a group of patients sometimes also have a syndrome where they seem to duplicate the people. Which we discussed, the 73-year-old man who believes that he is living not with his wife but with an imposter. And this is a major challenge.

And this is also a problem that has been seen in schizophrenia. Now, after having discussed these memory disorders, in the next class, we are going to discuss a little bit more about memory disorders, and then we will end this session on memory disorders in the lecture series on memory. So, see you in the next class. Thank you for your attention.