Indian Institute of Technology Kanpur

National Programme on Technology Enhanced Learning (NPTEL)
Course Title

Basic Cognitive Processes
Lecture-38

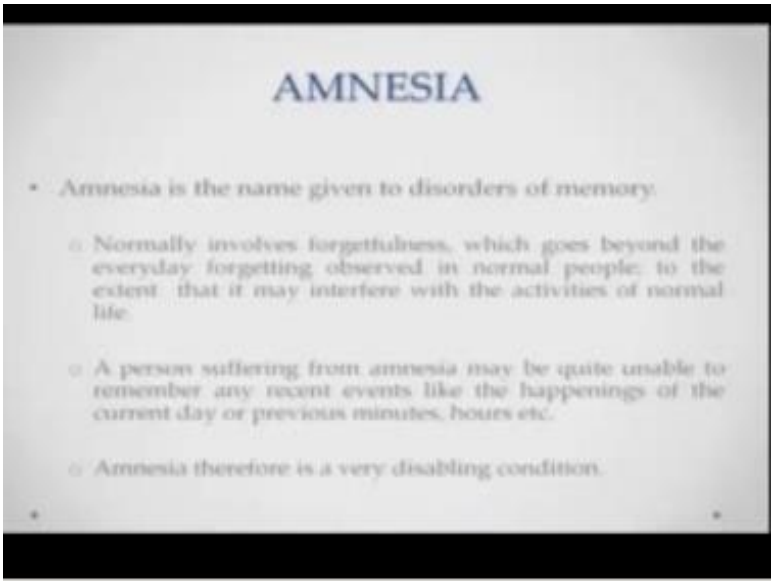
Disorders of Memory

By
Prof.Ark Verma
Department of Humanities and Social Science
Indian Institute of Technology Kanpur

Hello everyone welcome to the core series on basic cognitive processes I am Dr. Ark Verma from IIT Kanpur we have been in the last lecture talking about disorders we first visited disorders of attention and perception today we will be talking about disorders related to memory you have seen we spent a considerable amount of time talking about the organization of memory talking about how memory is an important cognitive function.

If you remember one of the earliest lectures Jerry Fodor Engel also say for example thought of memory as a horizontal function that kind of applies to most mental operations that people would do so disorders of memory and reading about disorders of memory is that important and it is important in that respect so let us begin talking about disorders of memory now the general name given to disorders of memory.

(Refer Slide Time: 01:07)



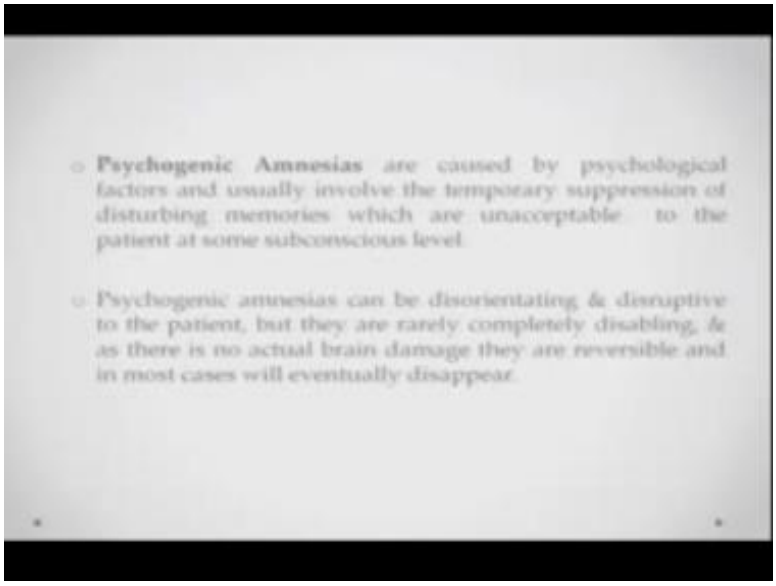
Is the name amnesia, amnesia typically involves forgetfulness which goes beyond the daily everyday forgetting you know you might keep your keys somewhere and forget it you might keep your book somewhere and forget it you might forget the name of a particular person that you meeting or the face of this person or a particular you know concept that you learned in the class but all of these kinds of forgetfulness are rather normal and happened to almost everybody when I am talking of amnesia I am talking of forgetfulness that goes beyond this general level of forgetting so forgetting in normal people is you know observed in everybody.

But we are talking about forgetfulness that is going beyond this part going to the extent that it starts interfering with daily activities of people when you started from your home to you know go to your office or your workplace or your school and during the way you kind of forgot where the office is now this kind of extensive forgetfulness might be you know quite disabling a person suffering from amnesia might quite be unable to you know remember any recent events like the happenings of the current day like happenings of the previous day also say for example in some cases they might not remember who they are whatever they have been experiencing for the past decade or.

So maybe two decades three decades something like that amnesia therefore can be a very disabling condition it can probably you know make the person dependent on a care giver or a caretaker for the rest of their lives so amnesia is therefore a rather serious disorder now I am amnesia this class of disorders of memory can arise out of a number of course there might be a lot of reasons various reasons that could lead to amnesia the first class or the more severe reason of amnesia could be organic emissions now organic amnesia is basically caused by a number of physical damage to the brain.

And those physical damages could be because of infections meningitis encephalitis those kind of things strokes head injuries or degenerative disorders such as the Alzheimer's disease organic amnesia generally tend to be rather severe and disabling and they are also irreversible in nature because any damage to the parts of the brain is irreversible generally these injuries would not heal the in fact the effects of the infection on the brain will not go away and in that sense the amnesia will be rather permanent in nature.

(Refer Slide Time: 03:42)

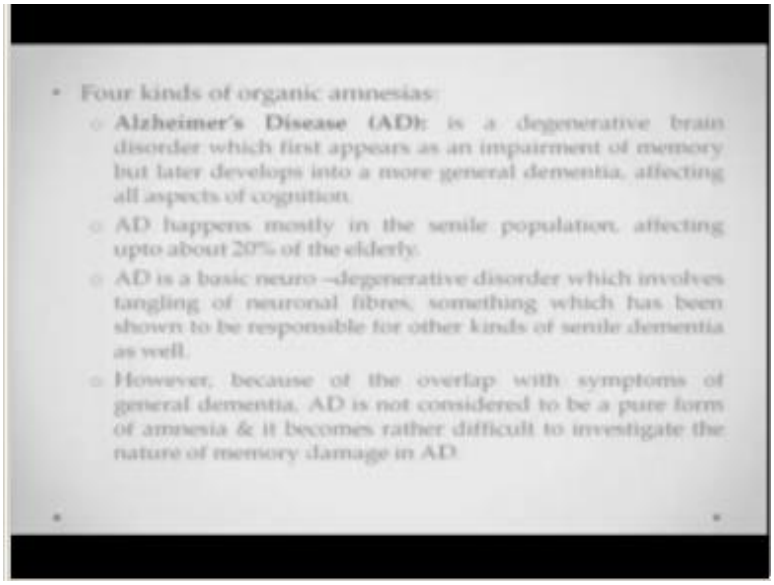


Now there could be psychogenic amnesia as well psychogenic images are caused by psychological factors such as depression increased tension etc these will involve the temporary

suppression of you know disturbing memories somebody might not have gone through a particular brain injury but might have gone through a very traumatic episode in their lives and because of the shock and because of the highly emotionally charged you know nature of that particular event the person chooses unconsciously or subconsciously to suppress that event and that kind of the memory of that entire event goes away this will be an example of the psychogenic knishes.

Now psychogenic images can also be rather disruptive and disorienting to the patient but they are rarely completely disabling they are not really pervasive as the organic amnesia we have been talking about now the psychogenic amnesia is because there is no brain damage involved are also generally reversible after adequate amount of medication or counseling or some kind of rehabilitative therapy and they would eventually with course of time disappear completely as well so we talked about organic amnesia as we talked about psychogenic initials they could also be in the middle we could just talk about four kinds of organic initials that may be.

(Refer Slide Time: 05:02)



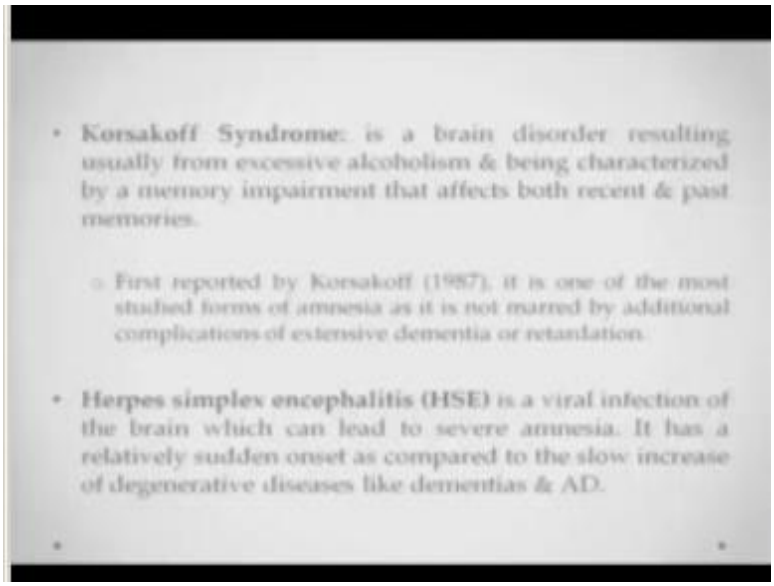
Now the first kind of organic amnesia is basically Alzheimer's disease as immune disease is a de generative brain disorder which first appears as an impairment of memory generally increased

forgetfulness but it later develops into a more general dementia so patients suffering from the Alzheimer's disease might not only have disorders or problems with memory but they might have problems with decision-making orienting themselves and other you know related disorders which are basically linked with the front elopes of the brain as Alzheimer's disease.

Happens mostly in the Sinai population affecting up to about 20% of the elderly from age groups of above 50 55 to higher ages assignments disease is Alzheimer's degenerative disorder which basically involves the tangling of neuronal fibers something which has been shown to be responsible for other kinds of senile dementia as well so this is something which is brought on by Alzheimer's it is a degeneration of areas of the brain tangling of the neuronal fibers and has been linked to other kinds of dementia where in you know basic cognition aspects are lost as well.

Now because of the overlap with symptoms of general dementia Alzheimer's disease cannot be considered to be a pure form of amnesia it is not really a disorder only in memory it's a disorder of memory plus some kind of decision-making process plus some kind of attention and difficulties and so on and so forth so that is why it is you know it is that much more difficult to investigate the nature of memory loss in Alzheimer's disease.

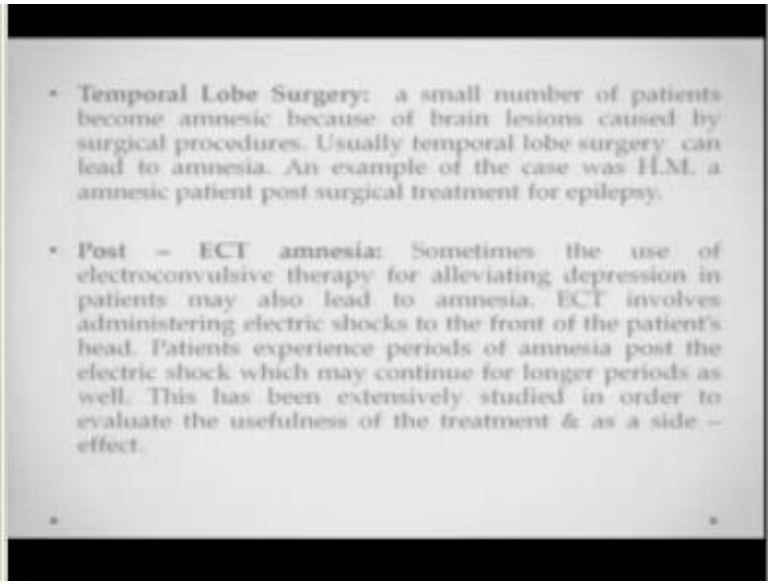
(Refer Slide Time: 06:39)



Another kind of an organic amnesia is basically caused or named as the Korsakoff syndrome now Korsakoff syndrome is basically a brain disorder which results from excessive alcoholism and is characterized by a memory impairment that affects both the recent and the past memories it was first reported by Korsakoff in 1987 and is one of the most studied forms of amnesia till now it is not really marred by additional complications of any extensive dementia or mental retardation or anything.

So it is a pure disorder of memory it is caused by alcoholism some damage to the parts of the brain which we will see later but it is purely a disorder of memory that is why a purely disorder that can be classified as a pure amnesia now there is also another course of organic amnesia which is known as the public's a simplex encephalitis it is a disease it is a viral infection of the brain that can result in severe amnesia it is basically characterized by a relatively sudden onset as compared to the slow onset of amnesia as for example in the case of DJ Alzheimer's disease.

(Refer Slide Time: 07:44)

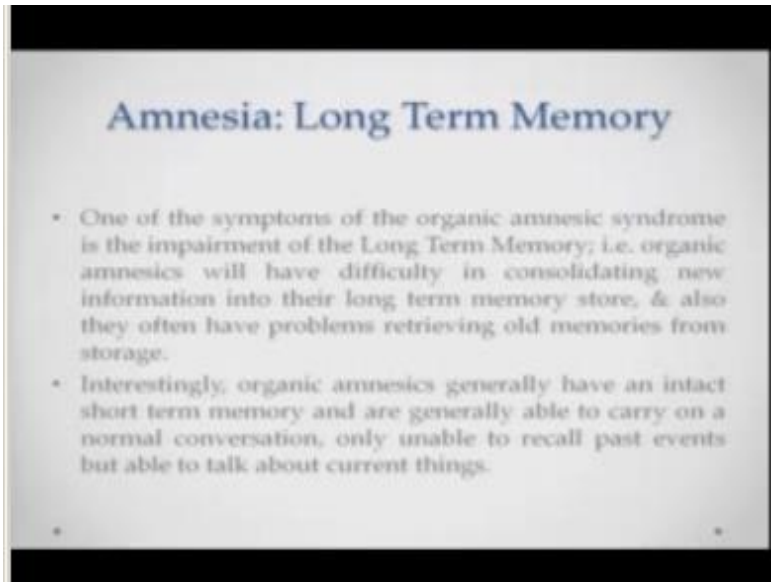


Also organic missions can be brought upon by surgical procedures typically surgery of the temporal lobes might lead to some kind of amnesia in a lot of cases and atypical example of this was the patient called Henry Malloy so and we have talked about him in the past as well he is

basically bilateral hippocampus were removed during a surgical procedure to relieve him from epilepsy but it resulted in a you know vast amnesia of you know a particular kind that we will discuss in some shortly another cause of amnesia could be post electroconvulsive therapy amnesia.

Now what happens is that in case of patients are suffering from mental disorders or psychological disorders a lot of times you will see that people you know resort to the use of electro convulsive therapy or shock treatment for alleviating the depression in these patients or Ziff which this might also lead to amnesia so ECT basically involves administering an electrical shock to the front of the patient's head and sometimes you know patients have reported having periods of amnesia post this electric shock now this also has been extensively studied in order to evaluate the usefulness of ECT for treatment of these depression page.

(Refer Slide Time: 08:59)

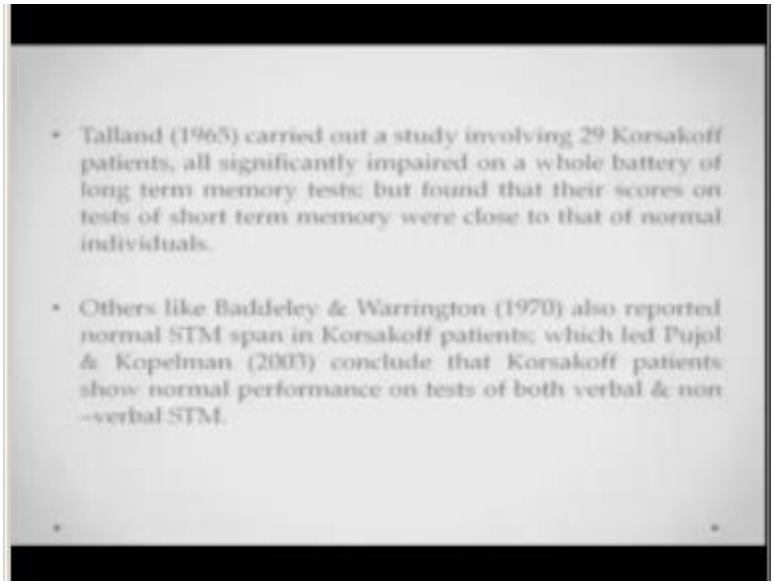


Now let us come to discussing about these amnesia in a slight more detail and how these initials might affect one of the aspects of memory called the long-term memory in Vanilla recent lectures we have talked about how long-term memory autobiographical episodic and semantic is not the kinds of memories that we most talk about and that we are most concerned with and

when we are talking about memory now one of the symptoms of the organic MSX syndrome is the impairment of long-term memory that is organic chemist six will have difficulty in consolidating new information into their long-term memory.

And you will also have problem in retrieving old or past long-term memories from their storage interestingly organic generally have an intact short-term memory and are generally able to carry out normal conversations you might not even notice that is there and that there is a problem with these individuals unless you start talking about things which have passed long ago hat say for example you might discover that these people are not really remembering even things that happened minutes ago talent in 1965 carried out a study involving 29 caustic of patients all of them significantly impaired on a whole battery of long-term memory tests.

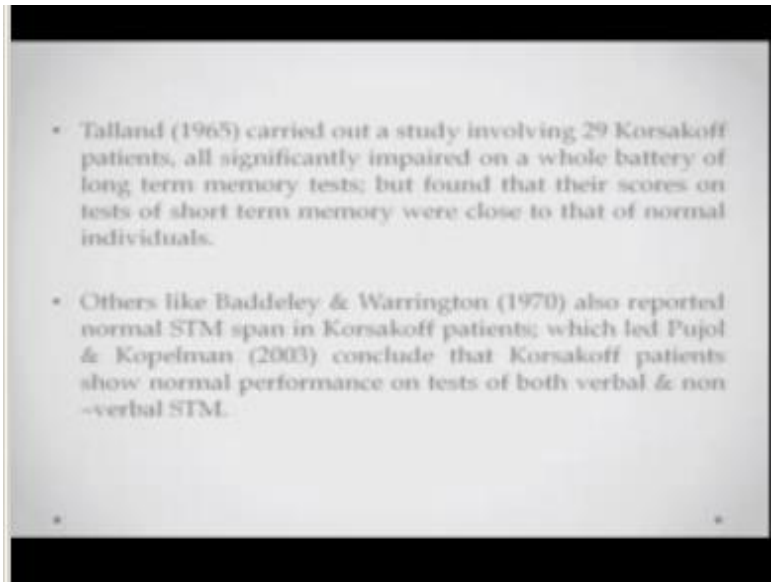
(Refer Slide Time: 10:11)



But found that their scores on a short-term memory test or tests of the short-term memory was almost as close to that of normal patients others liked badly in Warrington in 1970 have also reported normal's and short-term memory span in korsakoff of patients all of these kinds of studies led to hole and Kopelman in 2003 to conclude that korsakoff patients show normal performance on tests of both verbal and non verbal short-term memory so it tells you that

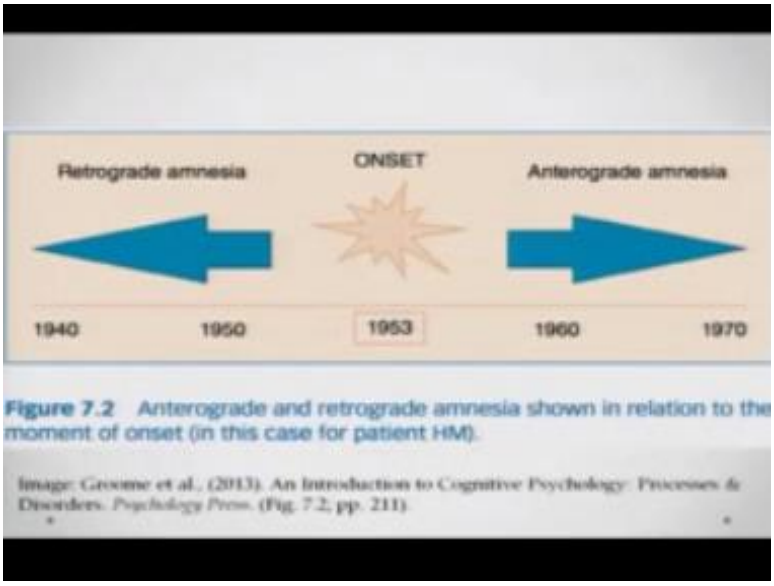
amnesia is can selectively affect and disability the long-term memory of the participants by leaving intact the short-term memory and working memory concepts.

(Refer Slide Time: 10:47)



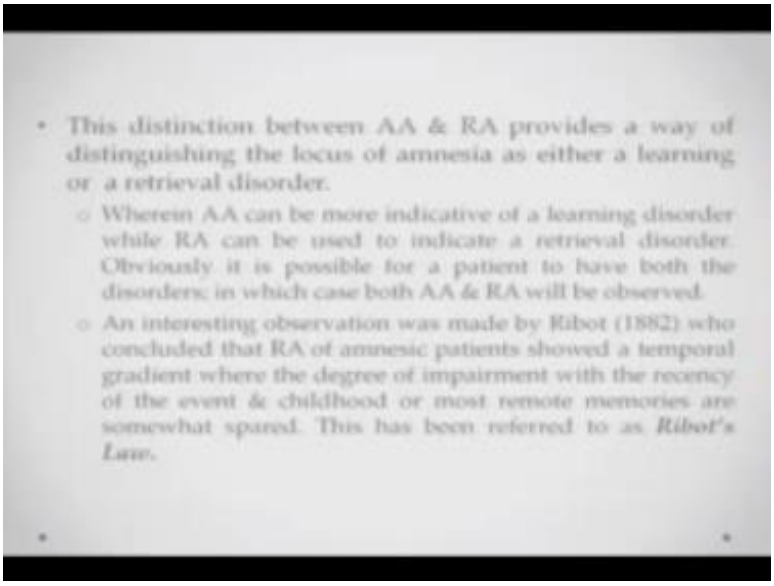
Let us talk about two kinds of finishes that one can talk about anterograde amnesia and retrograde amnesia are the two kinds so anterograde amnesia is basically disability or impairment of memory for events that are happening posts the onset of amnesia so once let us say there is a point in time where somebody has the onset of amnesia everything after that will be very hard to retrieve in retrograde amnesia everything before the onset of amnesia will be very hard to retrieve so these are the two different and two major kinds of emissions that one can actually talk about.

(Refer Slide Time: 11:24)



So hearing you can see so if there is an onset everything after the onset comes under anterograde amnesia everything before the onset comes under retrograde amnesia.

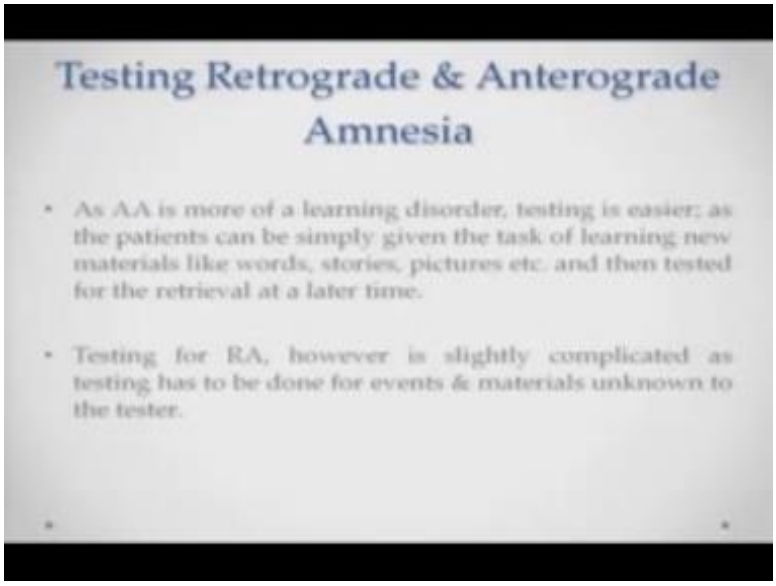
(Refer Slide Time: 11:33)



Now this distinction between anterograde and retrograde amnesia can provide a way of distinguishing the locus of amnesia as either a learning disorder or are trivial disorder now anterograde amnesia can be more indicate obviously a learning disorder well as retrograde amnesia can be used as an indication for retrieval disorder obviously it is possible for a patient to have booths and delayed and retrograde amnesia and in which cases you will say that both AA and RA are present in the body location an interesting observation that was made by a robot in 1882 was that he concluded that the retrograde amnesia of these amnesic patients shows a typical temple.

And gradient wherein the degree of impairment kind of is inversely proportional to the regency of the event so they weren't just very recent the degree of impairment will be too high if they went is too long ago in the past the degree of impairment will be slightly lesser this relationship given by Rabat is also referred to as the robots law.

(Refer Slide Time: 12:33)



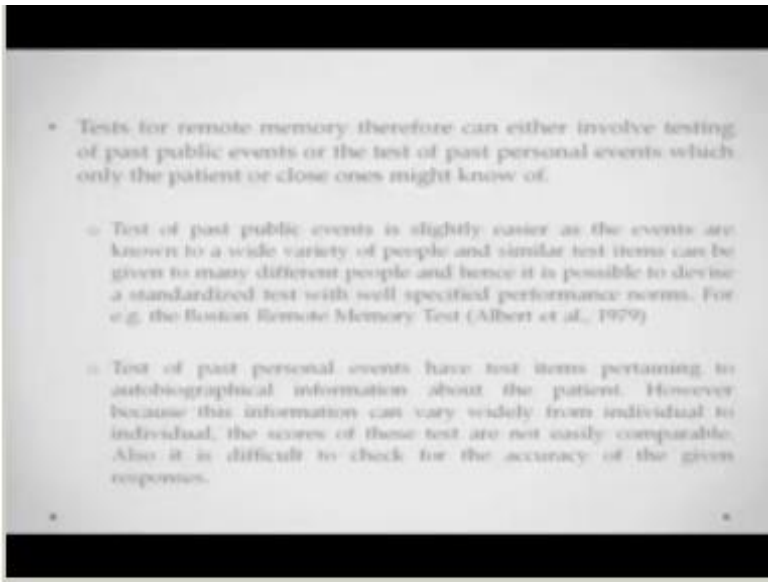
How do we test for these amnesia so if there is a cognitive disorder you would like to be able to test the patient test the presence of this cognitive disorder and there is the extent of this disorder and consequently find out ways to help these patients so if you are trying to test anterograde amnesia it is slightly easier because it is more of a learning disorder so you basically have to test whether the person can learn this new information or not.

And so you can give tests you know you can give new materials to the participant to learn over a period of time words pictures stories names faces whatever you might want to give then test their retrieval after you know a time gap of days or years or you know some time now testing for retrograde amnesia becomes slightly more complicated as the testing has to be done for events that are events details that are not known to the tester himself.

So you cannot really test for a particular patient that what happened when you were six years old because the patient is not there you know because the tested it also does not know and cannot verify whether this person is telling the correct thing or not usually in one of the last lectures you remember.

I have been talking about the constructive nature of memory so the constructive nature of memory might make it very difficult for an individual to be tested for retrograde amnesia because even though he might be remembering maybe in a few strands of information from that particular time that you are asking him about but a lot of information might just be made up and that is very difficult to verify.

(Refer Slide Time: 14:08)

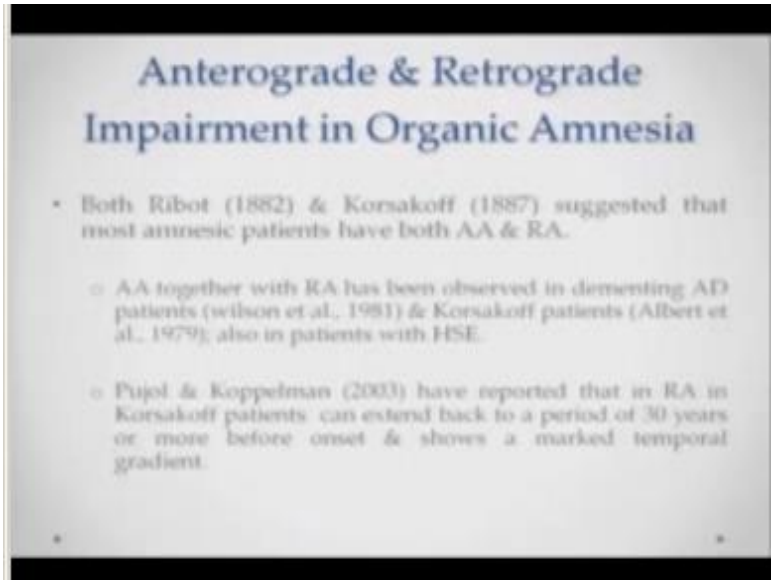


So what happens is tests for remote memory do either involve testing of past public events or the tests of past personal events which only the patient or the close people of the patients is a child and you ask the child about things that happen 10 15 20 years ago you can kind of verify those events with the help of the parents or the close relatives that would have been around that time so tests of past public events by the way is much easier because the events are known to a wide variety of people and similar test items can be given to many different people.

And their performance on these tests can be measured if there were 20 people around me and to each of them I basically asked them to relate what happened when India won the Cricket World Cup 2011 I can kind of compare there the amount of information that you would each recall about this particular event and I can cross verify and I can check whether they remember the

details or not tests of past personal events is difficult for the exact same thing people have cannot be tested for this auto biographical information unless there is somebody to verify it so however this inform and also this information that we are talking about the past personal events may vary widely from person to person it becomes difficult therefore to test anybody for this information also it becomes as I mentioned already very difficult to verify the accuracy of the responses that the batsman's are giving.

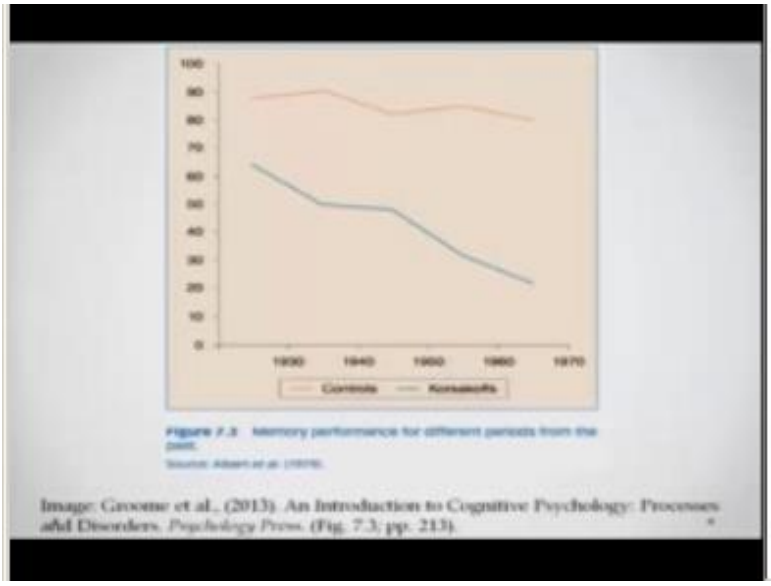
(Refer Slide Time: 15:32)



Now let us talk about more specifically and deliberate and retro amnesia and earlier in retrograde impairments so both Robert and Korsakov suggested that most patients will manifest both interrogate and retrograde amnesia if there is if they are experiencing organic causes anterograde amnesia together with retrograde amnesia has been observed in demanding LC meds patients.

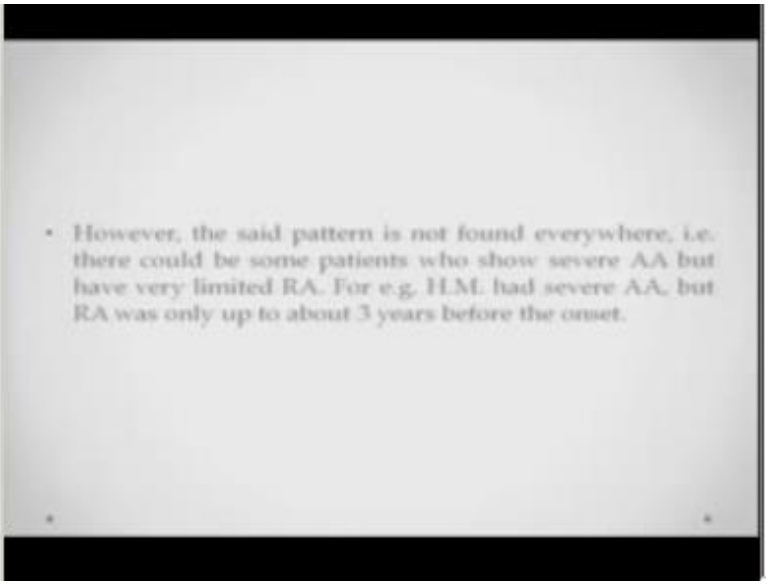
And caustic of patients also patients are herpes simplex encephalitis purulent Korsakoff in 2003 have reported that in retrograde amnesia in caustic of patients can extend back up to 30 years or more before the onset of because I am before the onset of amnesia and it will also show a March temple a gradient of the kind a try both was mentioning under Roberts law.

(Refer Slide Time: 16:30)



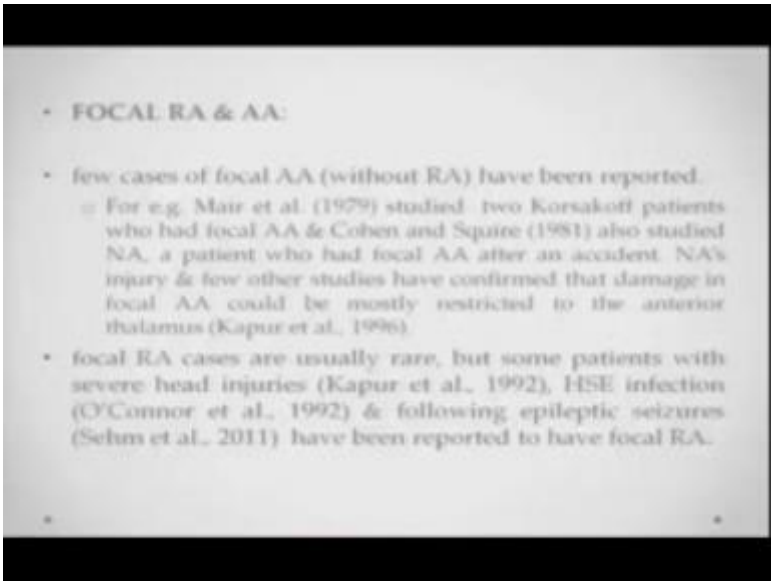
Here you can see this thing it is a source from Albert and colleagues the images from groom's book on cognitive psychology you can see and this blue line that represents caustic of patients the impairment is much more for more recent events and the impairments are much higher for and preservation is much higher for events that were much older so you see for events up to 40 years of old 40 years of age they had the preservation of information instead but events that are as recent as five or ten years old there is a marked impairment.

(Refer Slide Time: 17:02)



Now her the said pattern is not found everywhere so this is again one of the patterns that is reported but this pattern could not be found everywhere there could also be some patients who have severe anterograde amnesia but have very limited data to a dementia HM had a very severe anterograde amnesia he would not form any long-term memories after the surgery but his retrograde amnesia was only for up to three years but in a prior to the onset.

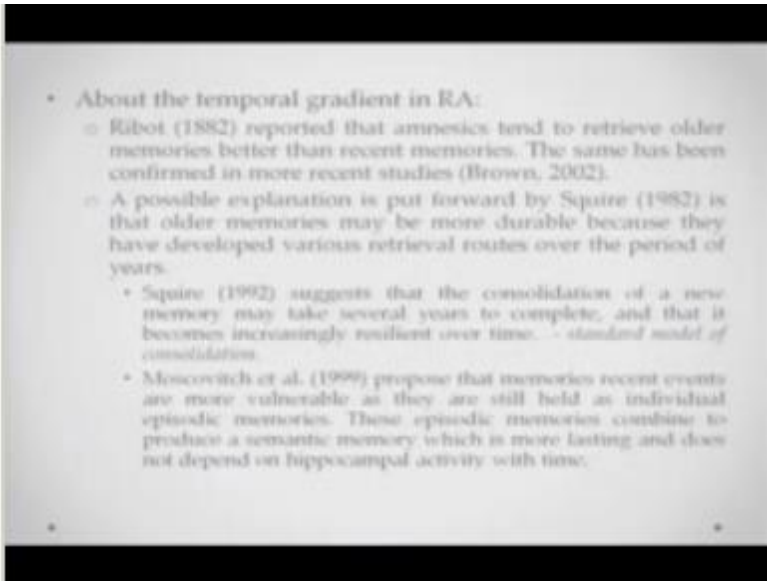
(Refer Slide Time: 17:28)



Now they have also been cases of a focal retrograde and anti debris diminution few cases of focal and early dementia without re have been reported say for example Myron colleagues in 1979 they studied two caustic of patients who had focal and early dementia and then Khan square in 1981 also studied any who was a patient who had focal anterograde amnesia particular accident any injuries and few other studies have confirmed that the damage in the focal anterograde amnesia could be mostly restricted to the anterior thalamus.

So that might be the region which is involved in people learning new events and if that region is damaged selective or focal anterograde amnesia is possible to be observed focal retrograde amnesia cases are rather rare but some of the patients have also with severe head injuries and following epileptic seizures and AJC have been reported to have focal retrograde image as well now focal retrograde amnesia has also been associated with lesions to various brain areas but most commonly it has been linked to lesions in the temporal cortex so that is one of the regions which probably can be linked more to the long-term memory than to the learning part of information.

(Refer Slide Time: 18:45)

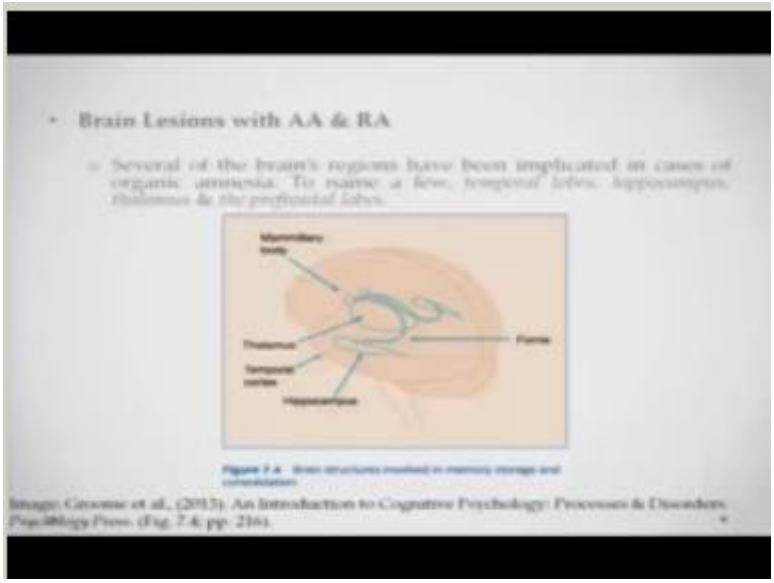


Now again coming and talking about this temporal gradient in retrograde amnesia RIE bought in 1882reported that an asset tended to retrieve older memories much better than more recent memories the same was also been observed in more recent studies like that of Brown in 2002 a possible explanation put forward to explain this temporal gradient put forward by square in 1982 is that older memories might be more durable because they have developed a variety of retrieval routes over periods of years if you remember what I have been talking about in the last part the narrative rehearsal hypothesis because older events have been gone over.

And over again you have retrieved a particular information over and over again over a longer period of time you have given various kinds of cues you have attached that information to too many sources of knowledge so that if one source of knowledge is absent if it is damaged you can still retrieve that information from other B or C air sources of knowledge so Square suggests that the consolidation of a new memory might take several years to complete and that it becomes increasingly resilient over time so once something has become increasingly resilient over a period of 20 30 40 years it might be that that information is preserved even cases of brain damage like organic mission.

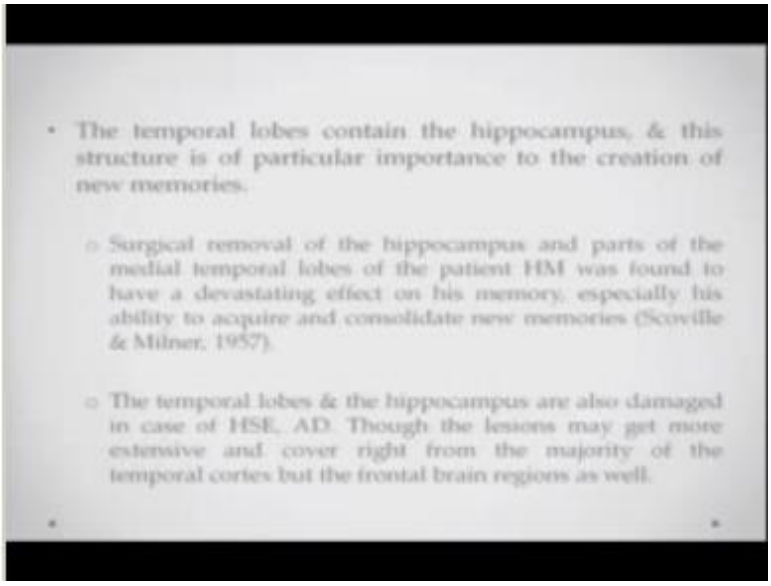
So this model which square was talking about is called the standard model of consolidation we talked about this in more detail as we go ahead Moscow vision colleagues they propose that memories of recent events are more vulnerable and are still held as individual episodic memories that is why they are difficult to retrieve in cases like amnesia these episodic memories combined with you know combine to produce certain kinds of semantic memories which are more lasting and does not really depend upon the hippocampus activity so even in case of damage it might be still possible to retrieve these kind of families now let us talk about the neuroscience part of emmechelle.

(Refer Slide Time: 20:53)



Let us talk about the neural underpinnings of amnesia typically the regions of the brain that are implicated in cases of organic amnesia are these regions the temporal lobes by literally the hippocampus violet ray the thalamus and the prefrontal lobes these are the typical regions that are associated with memory disorders such as amnesia.

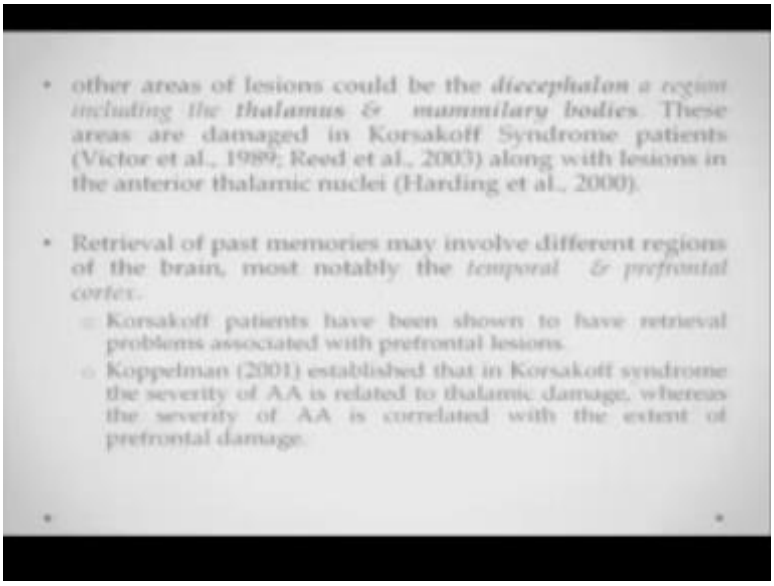
(Refer Slide Time: 21:14)



Is going a bit more detail the temporal lobes contain the hippocampus and this structure is of particular importance to the creation of new memories surgical removal of hippocampus in parts of the medial temporal lobes of the patient known as HM was found to have a devastating effects on his memory and especially his ability to acquire and consolidate new memories if you remember we have mentioned this earlier as well the bilateral hippocampus of HM were removed which led.

Now to the disability of forming new memories you know for him for the period completely after the onset of the answer to this amnesia the temporal lobes and hippocampus are also damaged in cases of herpes simplex encephalitis and Alzheimer's disease though the lesions may get more extensive and cover right from the majority of the temporal cortex with the frontal brain lesion sister so this damage might be more distributed and much more extensive in case of Alzheimer's disease or herpes simplex encephalitis other areas of lesion could be the diencephalon that is a region which includes the thalamus and the mammilla bodies of the brain.

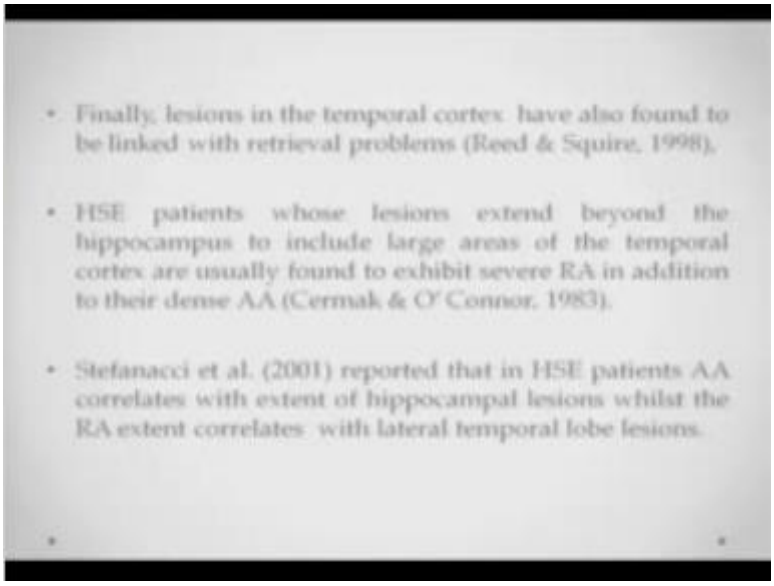
(Refer Slide Time: 22:24)



The areas which are damaged in the caustic of syndrome or patients are along these regions and also regions which include the anterior thalamic nuclei both there trivial of past memories involve different regions of the brain most notably the temporal and prefrontal cortex so if you are talking about retrograde amnesia if you are talking about retrieving information that is long past you might be talking more about the temporal cortex and the prefrontal cortices now caustic of patients have been shown to have a retrieval problems associated with prefrontal lesions Cooperman in 2001in his paper established that in Korsakoff syndrome.

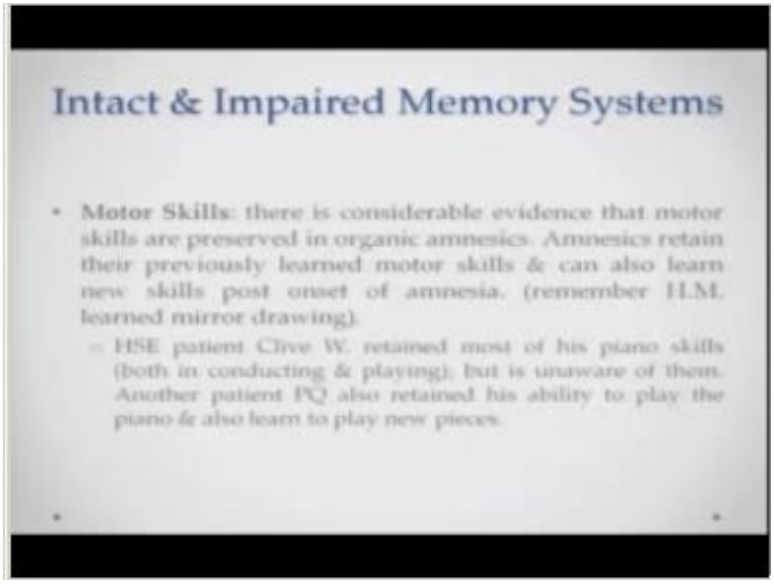
The severity of anterograde amnesia is related to thalamic damage whereas the severity of retrograde amnesia is a colligative with the extent of prefrontal damage so there is a type of here the stability of anterior dementia is a link to a thalamic damage whereas a severity of retrograde which is linked to prefrontal damage.

(Refer Slide Time: 23:29)



Finally lesions in the temporal cortex have also been found to be linked with retrieval problems HSE patients whose lesions extend beyond the hippocampus have been found to include large areas of the temporal cortex are also found to exhibit retrograde amnesia in addition to their dense anterograde amnesia so HSE patients we have damage in hippocampus and large areas of temporal cortex will have both AA and RA Stephan Archie and colleagues in 2001reported that in ACC patients anterograde amnesia correlates with the extent of hippocampus lesions well as retrograde amnesia correlates with the extent of lateral temporal lobe lesions.

(Refer Slide Time: 24:05)

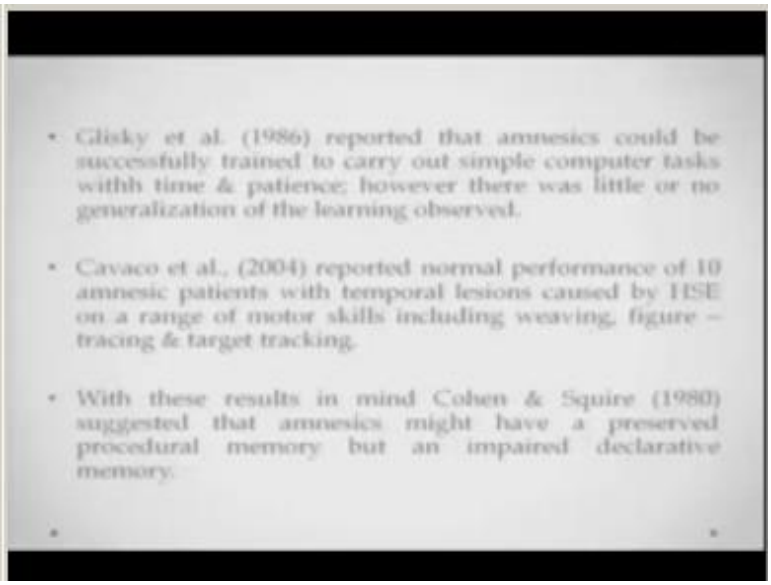


Let us now talk what we have talked about the extent of damage in organic Amnesia's the regions of brain that are involved in these organic and vicious let us now talk about the intact and impaired memory systems what are the memory systems that are left intact even in cases of organic amnesia one of the first areas are the motor skills there is considerable evidence that motor skills are preserved even in organic ms and a-six retained a previously learned motor skills.

And can also learn new motor skills post the onset of amnesia if you remember HM was able to learn mirror drying when he was taught even though he did not have an explicit memory of learning mirror drying but he could he got better at drawing those figures while looking in the mirror HSE patient Clive earring.

We have also talked about him in the past lectures retained most of his piano skills both in conducting the piano session and in playing the piano but it is not really aware of them if you give him the piano he might still be able to play but he is not aware of the fact that he is a piano player he was a piano player also another patient PQ or retained his ability to play the piano and to even you know the ability to learn to play new pieces.

(Refer Slide Time: 25:17)



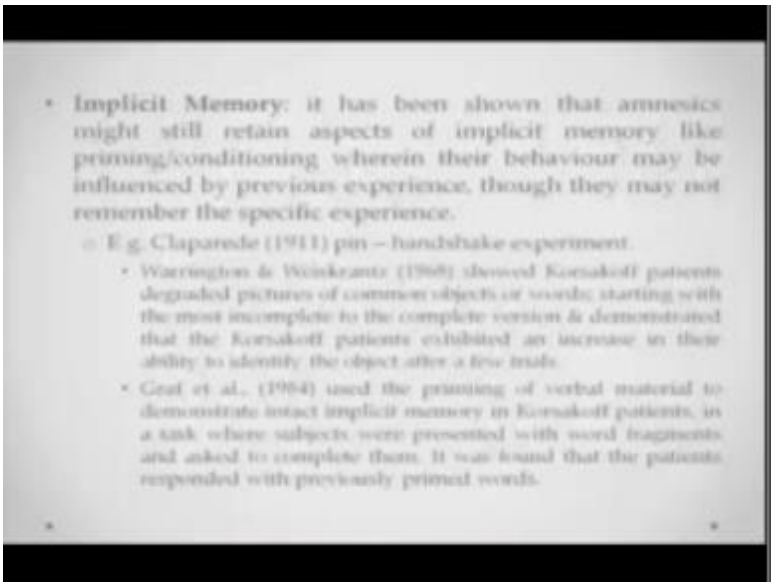
And there have been a lot of experiments in this area so Glee scheme colleagues in 1986 they reported that Alma six could be successfully trained to carry out simple computer tasks with time and patience obviously you will probably need a lot of patience to teach these people you know these particular skills because they will probably forget the episodes you will have to be very simple in your instructions.

And you have to make sure that these people are getting these letters also they observe there will be little to no generalization of the learning of say for example if you taught a person a particular skill in a given setting it is rather difficult for that skill or for that for the learning of that skill to generalize in a different setting.

So it might be that it happens in the same you know it learning happens in one setting it stays to that setting if the person changes the setting if the person goes to let us say different room or a different you know equipment the setting kind of changes and the learning is not generalize cava co increase 2004 reported normal performance of 10 amnesic patients with temporal regions caused by HSC on arrange of motor skills including weaving figure tracing.

And target tracking so you are seeing all of these motor skills that people are doing the parts of memory which are referred to as procedural memory is basically found to be intact even in cases of organic amnesia now with the in the mind coherence choir suggested that the MSX might have a preserved procedural memory but an impaired declarative memory which includes both the explicit memory.

(Refer Slide Time: 26:59)



Let us now talk about implicit memory implicit memory again is that memory which you cannot explicitly k about you cannot really describe those events the episodes and the facts completely but it has been shown that amnesties might still be able to retain aspects of implicit memory effects of priming or conditioning might still be observed now there is a very interesting experiment that was done by a clapper rate in 1999 11.

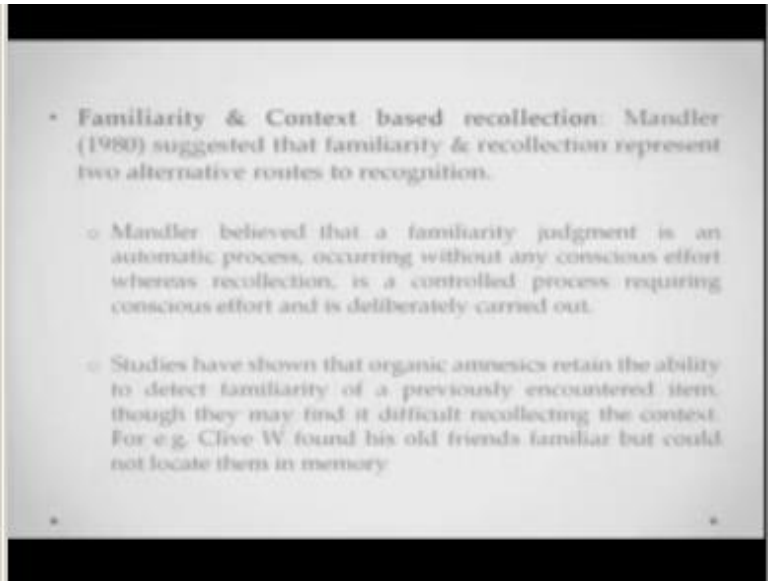
So he did this that whenever he used to shake hands with the amok patients he would have he would hide a pin in his hand and he would actually shake hands with these amnesic patients whenever these MC patients would shake hands the pin will actually you know a pain will actually stick through their skin they will experience some pain and they will take their hands off eventually but they'll not have a memory of this episode after a few minutes.

So clapper 8 will come back again and do the same thing eventually what they discovered Warrington and what they discovered was these patients started you know stopped to shake hands with them so it kind of told cooperate that they are if they are not even explicitly remembering that the pin incident but they kind of have something tells them that you know you should not shake hands with this person there is a pin in his hand.

Warrington in Y strands in 1968 showed that caustic of patients degraded when they were viewing degraded pictures of common objects words starting with the most incomplete and they were kind of trying to rate this starting with the most incomplete version to the complete version they demonstrated that caustic of patients exhibit an increase in the ability to identify the object after a few trials again they will not have the episodic memory of seeing those pictures earlier.

But if you repeat this adequate number of times they will get better at recognizing these pictures graph in clicks in 1984 they use the priming of verbal material could demonstrate the intact implicit memory in causes of patients and what they did was they actually used a task when in subjects were presented with word fragments and they were asked to complete them it was found that patients responded with previously primed words so whatever they were responding with actually was already primed even though these people might not have a good memory of those things.

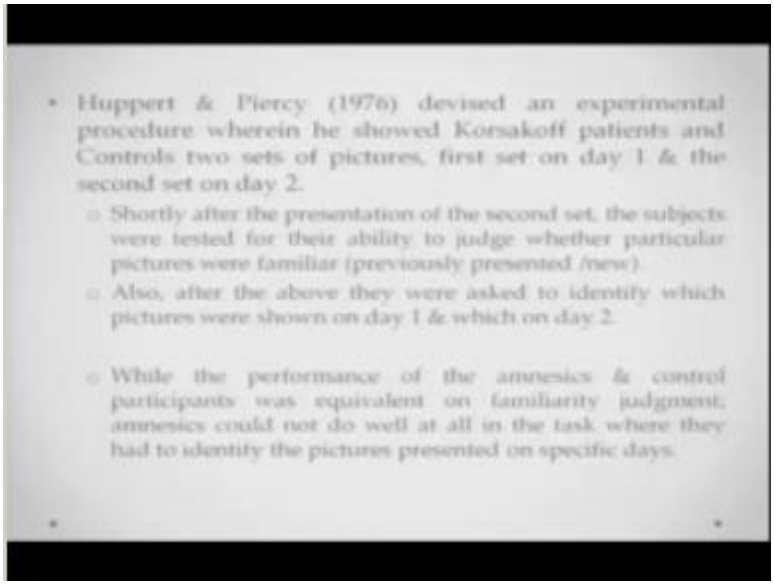
(Refer Slide Time: 29:09)



Also their familiarity and context based recollection might be as pared in patients of amnesia so amender in 1980 suggested that familiarity and recollection represent two alternative routes to recognition he believed that a familiarity judgment is an automatic process occurring without any conscious effort and whereas recollection is a controlled process requiring conscious effort and is deliberately carried out so this is the typical difference between a familiarity judgment and recognition now there have been studies which have shown that organic amnesties retain.

The ability to detect familiarity to an end previously encountered item you remember clapparates experiment something similar live earring found his old friends familiar but could not locate them in memory could not locate seeing them so one of the interesting incidents is recalled John Kennedy who was suffering from or one of the US presidents were suffering from amnesia and he would at some point in time even not recognize the stuff but he would tell his nurse sometimes that you know I know this person he seems to be a rather famous man but I do not recall where I have met him or what do I you know what do I have to do with him.

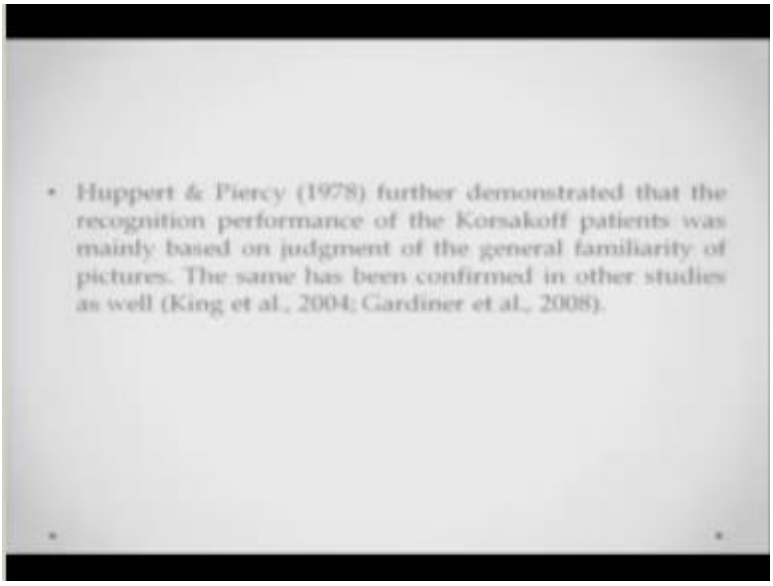
(Refer Slide Time: 30:30)



So similarly you put and Piercy in 1976they devised an experimental procedure wherein they showed their how caustic of patients and controls they were basically showing Kosovo patients and controls two sets of pictures the first set of pictures on day one in the second set of pictures on day two shortly after the presentation the second test the subjects were tested for their ability to judge whether particular pictures were familiar and that is they had previously seen them or they were new.

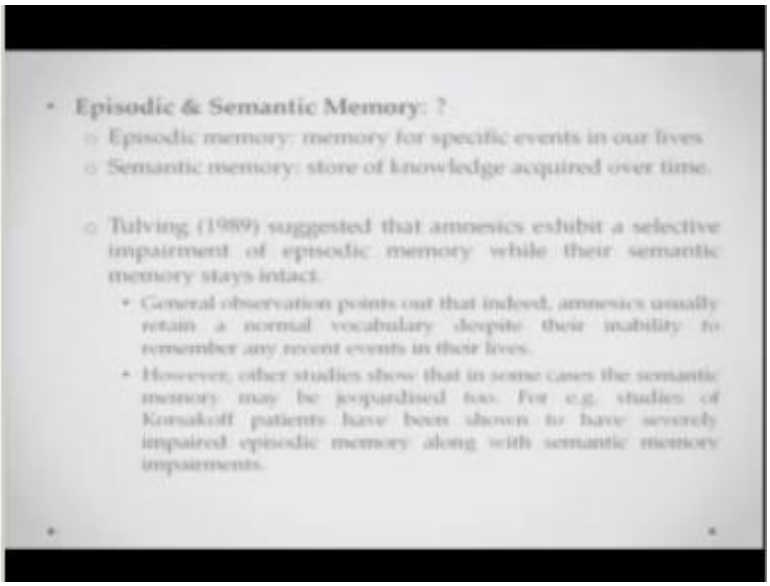
So there they mix this test set with the familiar pictures and some new pictures also after this above test they were asked to identify which of the pictures were shown on day one and which of the pictures were shown on day two so they not only have to judge whether these pictures are familiar or not familiar they also have to judge vary in time they have seen these pictures now while the performance of the amnesties in control participants was equivalent on familiarity judgment and mystics could not do well at all in a locate in the task wherein they had to identify the present the days on which these pictures were presented.

(Refer Slide Time: 31:32)



So you put in piracy they further demonstrated that the recognition performance of caustic of patience was mainly based on the judgment of familiarity it was basically so they kind of had a sense of that I have seen this picture earlier but they did not really have a sense of when had I have I seen this picture so you might see the results I don't really have the figure but this kind of shows that these people performed very well on tasks of familiarity judgment but they could not Locate the source of that information.

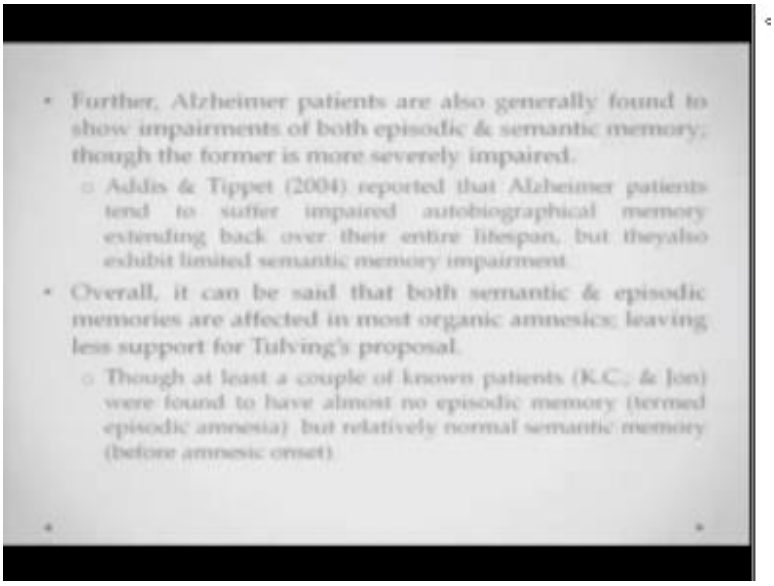
(Refer Slide Time: 32:02)



Now let us talk a little bit about episodic and semantic memory because we have been talking about long-term memory here episodic memory is memory for specific events in lies semantic memory is a set of knowledge or facts that you have acquired over time now Tullfing in 1989 suggested that amnesties exhibit a selective impairment of episodic memory while the semantic memories might still be intact general observation points out that indeed analytics usually remember they are they are fine with the normal vocabulary despite of their inability to remember any recent events in their life.

So they can talk and they can remember words what means those kind of things that you still have but they do not really remember the events in the life's the episodes that have passed however other studies have shown that in some cases the semantic memory might also be jeopardized studies of Corsica patients have been shown to have severely impaired episodic memory along with semantic memory impairments so both kind of memory disorders are possible even though it might be said that this episodic memory is probably more impaired than maybe the semantic memory.

(Refer Slide Time: 33:08)



Alzheimer patients have also found to be shown impairments of both episodic and semantic memory though the former is a more severely impaired so episodic memory is most severely impaired add is on tippet in 2004 reported that Alzheimer patients tend to suffer impaired autobiographical memory or a large period of time or the entire lifespan but they also exhibit sometimes limited semantic memory impairment not really completely extensive as the out of every memory impairment is but also some kind of semantic memory impairment is also generally seen overall if you in of combine this it can be said that most semantic.

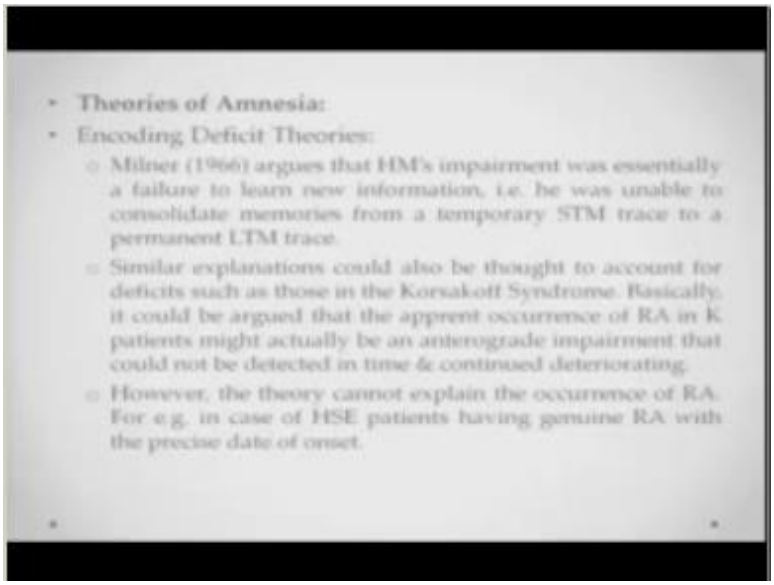
And episodic memory might be impaired in most organic Emily shakes leaving less support for you know at Alvin's proposal that only episodic memory is damaged her at least a couple of known patients say for example case y and john were found to have almost no episodic memory but a relatively normal semantic memory.

So there are obviously some cases where in instances of intact episodic memory or intact semantic memory will be found and completely gone episodic memory will be so again some of the things one of the things I would like to tell you about disorders is that no two patients have

exactly the same characteristics because no two patients might have the exact same extent of the lesion that is when you are talking about disorders.

And typically disorders like organic amnesia you might already come across with a huge variety of manifestations in patients huge variety of lesion sites in patients and that is why it these studies or these theories typically built upon case studies and there is less that you can completely generalize across all patient's obviously you have to make generalizations and come up with theories but those generalizations will tend to be much less accurate in case of neuropsychological patients than in some other areas.

(Refer Slide Time: 34:57)



Now there have been theories of amnesia including deficit theories miller in 1966 argues that a chimps impairment was essentially a failure to learn new information that is because he was unable to consolidate memories to form a temporary short-term and you know from a temporary short-term memory trace to a more formidable long-term memory trace.

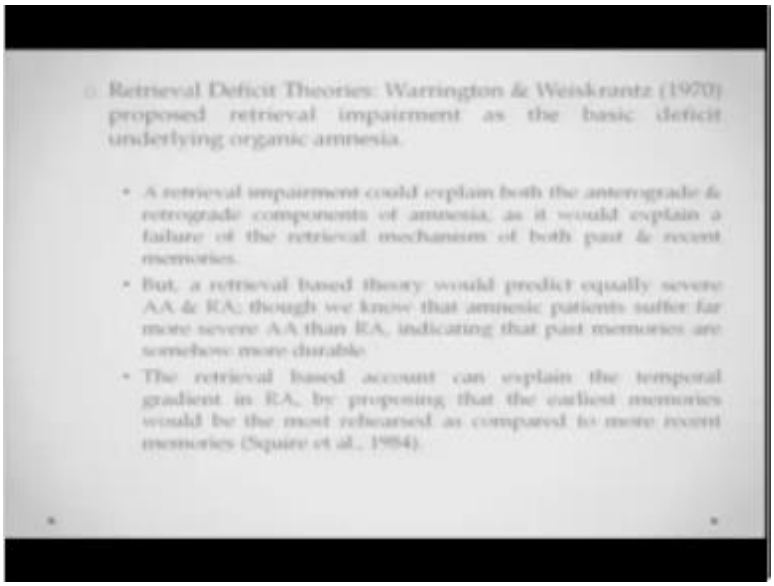
Similar explanations could also be thought to account for deficits such as those happen those which happen in Korsakoff syndrome basically it could be argued that the apparent occurrence of

a retrograde amnesia encaustic of patients might actually be a form of an anterograde impairment that could not be detected in time and has been ensuing since quite a long time and it had been continued to deteriorate so you are kind of saying that whatever they have lost it basically was you know the onset is much earlier.

And the antelope great amnesia had set and so these people have not really consolidated those memories that is when now when they look bad they cannot retrieve those memories back also but if even if you take this into account the theory cannot completely explain the occurrence of retrograde amnesia in case of HSE patients who have a genuine retrograde amnesia with the precise date of onset.

So there if you remember what I was talking about HSE the onset is rather sudden so there you cannot really argue that you know a retrograde amnesia is basically undetected until every diminished that had been lingering on in the patient for quite some time and that is why he is not being able to recall all the previous information so it is not really a really compelling explanation.

(Refer Slide Time: 36:31)

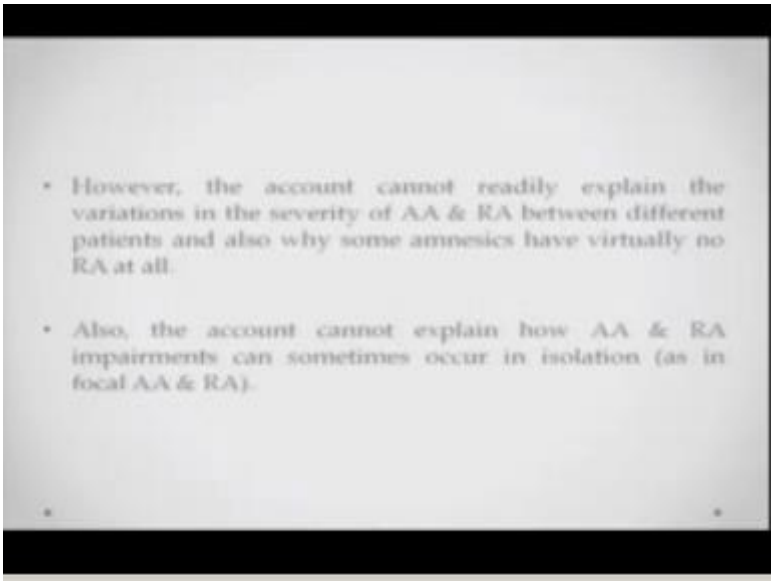


So you turn to a different class of expedition called the retrieval deficit explanations now Warrington NY strands in 1970 proposed a retrieval impairment as the basic deficit underlying organic amnesia so you see all kinds of organic Gnash might basically be because of a retrieval deficit now a retrieval deficit is such that it could explain both and deliberate and retrograde components of amnesia.

Because this would explain a failure of the retrieval mechanism neutral mechanism could be failing for past events and recent events as well so if it is failing for recent events it is basically a manifesting in anti-liberal amnesia if it is a manifesting in past events it is manifesting in retrograde amnesia a retrieval based theory also would predict equally severe anterograde in retrograde amnesia though we know that there are a Mesick patients who suffer from far more severe anterograde amnesia than retrograde amnesia.

So again this theory also does not really do a very good job of explaining the incidence of both anti degrade and retrograde missions the retrieval waste account however can explain the temporal gradient in retrograde amnesia by Proposing that the earliest memories would be the most rehearse and hence they will be a compared to the most recent memories and then hence they will be recalled much better.

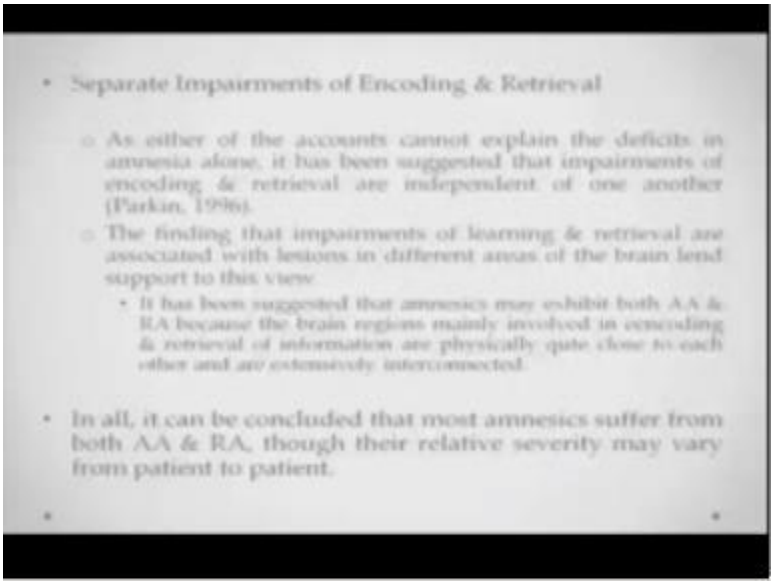
(Refer Slide Time: 37:42)



However the retrieval based account Cannot readily explain the variations in the severity for a and re between different patients and also why some patients can have virtually no retrograde amnesia but a lot of ant degradation say for example the case of HM also this account cannot explain how when they in some patients they could be focal entirely dementia or focal retrograde amnesia because if there is one explanation that fits both the amnesia.

And both the kinds of finishes if there is that impairment if one has to occur the other should definitely occur which does not really happen in case of focal a or RA patients now because both of these theories did not do a great job we could actually combine them and try and see if this can be explained so as either of the counts cannot explain the deficits in amnesia alone it has been suggested that.

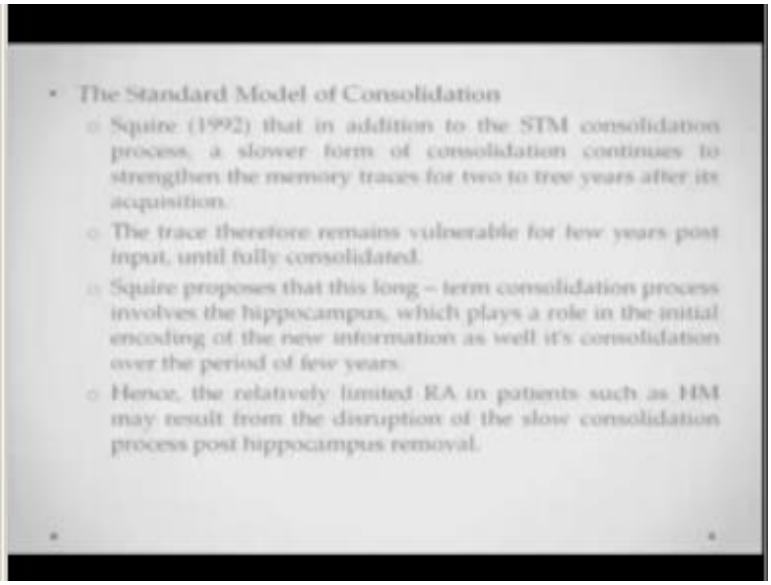
(Refer Slide Time: 38:34)



The impairments of encoding and retrieval are independent of each other and they lead to in different disorders the finding that impairments of learning and retrieval are associated with lesions indifferent areas of brain have also lent support to this view so it has been so they said that amnesiac may exhibit both and deliberate and retrograde amnesia.

Because the brain regions mainly involved in encoding and retrieval are physically quite close to each other all and they are extensively interconnected now in all it can be concluded that most MSX would suffer from both a and RA though their relative severity will vary from patient to patient and depending on the extent of injury one of these models about retrieval as we were mentioning earlier.

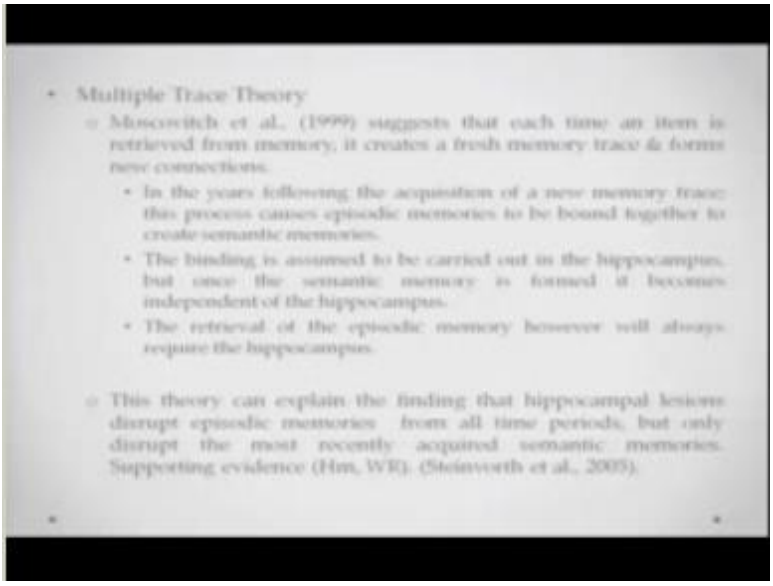
(Refer Slide Time: 39:19)



Is the standard model of consolidation so square basically in 1992 says that in addition to the short-term memory consolidation process a slower form of consolidation continues to strengthen the memory traces for two to three years after its acquisition the trace therefore remains vulnerable for a period of up to two to three years or for a few a few years post the input until it is completely consolidated square proposes that this long-term consolidation process involves the hippocampus which plays a role in the initial encoding of the new information.

As well as its consolidation into a long-term memory hence the relatively limited retrograde amnesia patients such as HM may result from the disruption in the slow consolidation process because their hippocampus has been removed.

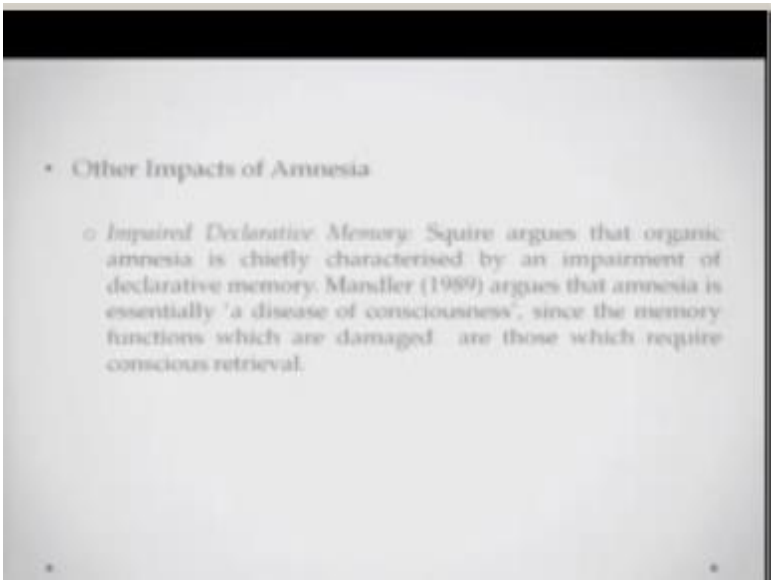
(Refer Slide Time: 40:03)



There is the multiple theory so mascara chin clicks in 1999suggested that each time an item is retrieved from the memory it creates afresh memory stress and forms new connections so in is following the acquisition of a new memory trace this causes the episodic memories to be bound together to create semantic memories the binding is assumed to be carried by the hippocampus but once the semantic memory is formed it becomes independent of the hippocampus.

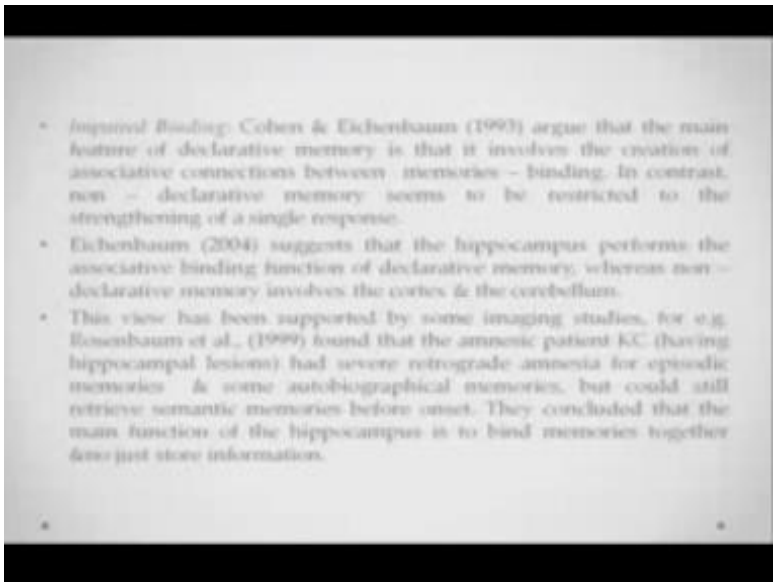
So the binding function of memory is being done by the hippocampus but after the binding is complete the hippocampus does not have a lot to do with this information the retrieval of the episodic memory however always requires the hippocampus so retrieval based deficits could and will generally involve some kind of hippocampus damage on this theory the multiple trace theory can explain the finding that hippocampus lesions disrupt episodic memories from all the time periods but only the most recently acquired you know semantic memories will be deficient because the hippocampus is just recently removed there have been supporting evidence from cases like HM and WR.

(Refer Slide Time: 41:11)



There could be other impacts of amnesia as well let us talk a little bit about those so impaired declarative memory square or use that organic initial chip chiefly characterized by an impairment of what is called declarative memory you can talk about manlier argues that amnesia is essentially as disease of consciousness so you cannot consciously retrieve information and that is and even that is basically what is leading to the amnesia.

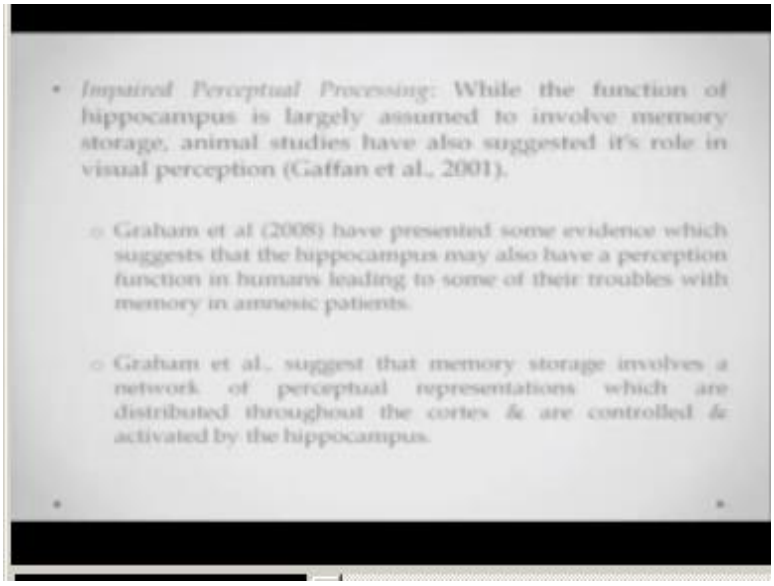
(Refer Slide Time: 41:38)



There could be impaired binding as well so coherently Shan boom 1993 argued that the main feature of declarative memory is that it involves the creation of associative connections between different memories so this binding is the main thing in contrast non-declarative memory seems to be restricted to the strengthening of a single savors I am learning of motor responses I confirm 2004 suggests said that the hippocampus performs the binding function of the memory.

And that and whereas non-declarative memory involves the cortex and cerebrum the areas again involved in motor processing this view has been supported by a lot of imaging studies say for example rosenbaum and colleagues in 1999 found that the patient Casey had severe retrograde amnesia for episodic memories and some autobiographical memories but could still retrieve semantic memories before the onset they concluded that the main function of the hippocampus is to bind these memories together and organize the stored information.

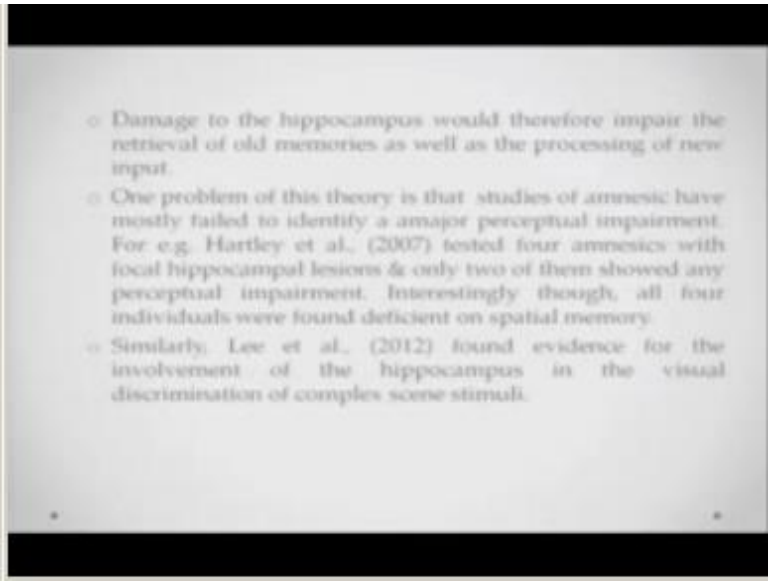
(Refer Slide Time: 42:40)



Impaired perceptual processing impaired perceptual processing while the function of hippocampus is largely assumed to involve a memory storage animal studies has suggested that it also plays a role in visual perception so grime encolleagues in 2008 presented some evidence which suggests that the hippocampus may also have a perceptual function in humans leading to some of their troubles you know some of the troubles of memory in the alma situations.

So they suggested that memory storage involves a network of perceptual representations a sort of a distributed you know representation which are distributed throughout the cortex and controlled and activated by the hippocampus.

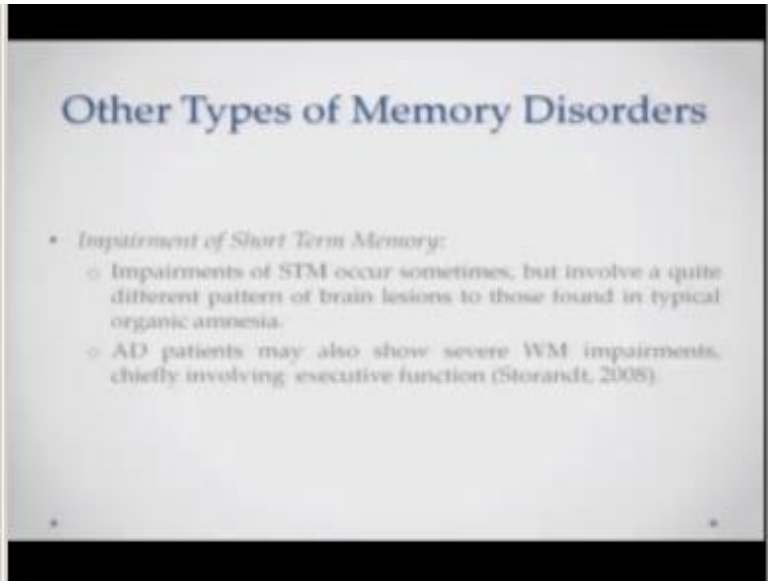
(Refer Slide Time: 43:15)



So what is happening is damage to the hippocampus is basically going to significantly impair the retrieval of these old memories as well as the processing of new input so if hippocampus is damaged you will not be able to store incoming information in that distributed fashion also you will not be able to invoke that information that has been distributed list stored in various regions of the cortex one of the problems for this theory however is that the studies of amnesic patients have mostly failed to identify any major perceptual impairment.

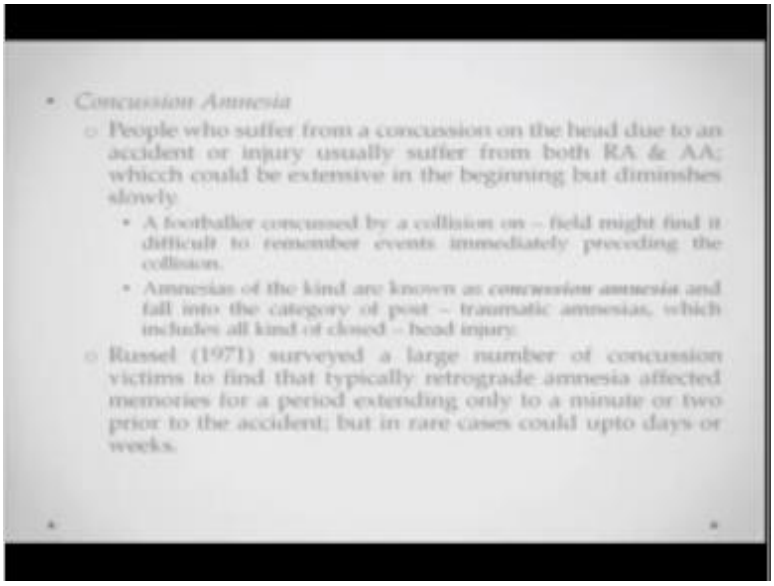
So for example Hartley in colleagues in 2007 it has said for ms6 with for with focal hippocampus lesions and only two of them showed any a trace of perceptual impairment interestingly though however all four of the individuals were found to be impaired on spatial memory similarly Libyan colleagues in 2012 found evidence for the involvement of the hippocampus in the visual discrimination of complex scene stimuli.

(Refer Slide Time: 44:10)



Now we can talk about some other types of memory disorders as well so impairments of short-term memory impairments of short-term memory generally occur sometimes that involve a quite different pattern of blarney brain lesions than what you saw in the case of amnesia or long-term memory impairments as Emus disease patients may sometimes show severe working memory impairment chiefly involving the executive functions alerting orienting alerting orienting and inhibiting.

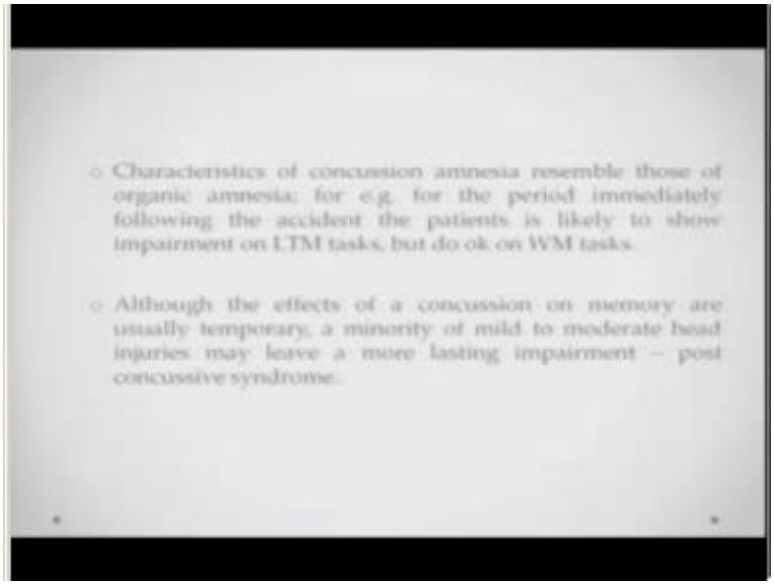
(Refer Slide Time: 44:35)



Concussion amnesia people who suffer from concussion on the head due to a you know accidental injury on the top of the head basically will suffer from both retrograde and anterograde amnesia which could be extensive in the beginning just after you know say for example have banged your head or somebody's hit you on the head but kind of diminishes with time a footballer conquers by a collision on the field might find it difficult to remember events just leading to the concussion.

But will eventually regain those memories back after a particular period of time these kind of Amnesia's are known as concussion amnesia as which fall into the category of posts from traumatic amnesia and includes almost all kinds of brain injuries possible head injuries basically Russell in 1971surveyed a large number of concussion victims and to find that typically retrograde amnesia effects the memories for a period extending or leading up to a minute or two till this concussion or accident happened but in rare cases up to days or weeks could also be from.

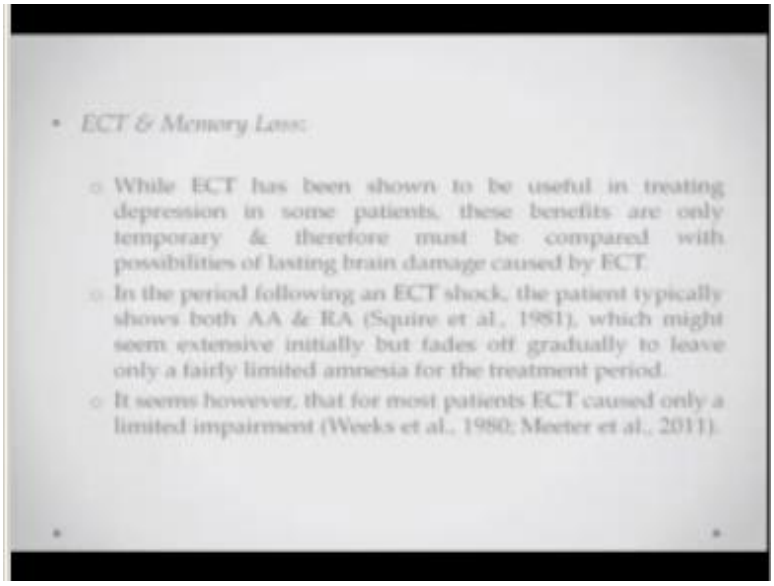
(Refer Slide Time: 45:36)



A characteristics of common concussion amnesia resemble quite a bit of organic amnesia as a period immediately following the accident the patients are likely to show impairments in long term memory tasks but they do slightly ok on working memory tasks although the effects of concussion amnesia are on memory are usually temporary a minority of mild to moderate head injuries may leave a more lasting impairment we have talked about ECT.

And memory loss so ECT has been shown to be useful in treating depression in some patients these benefits have been found to be only temporary and therefore must be compared with the possibilities of a lasting brain damage so if a person has received extensive ECT treatment it might actually lead to you know a lasting brain damage in a period following.

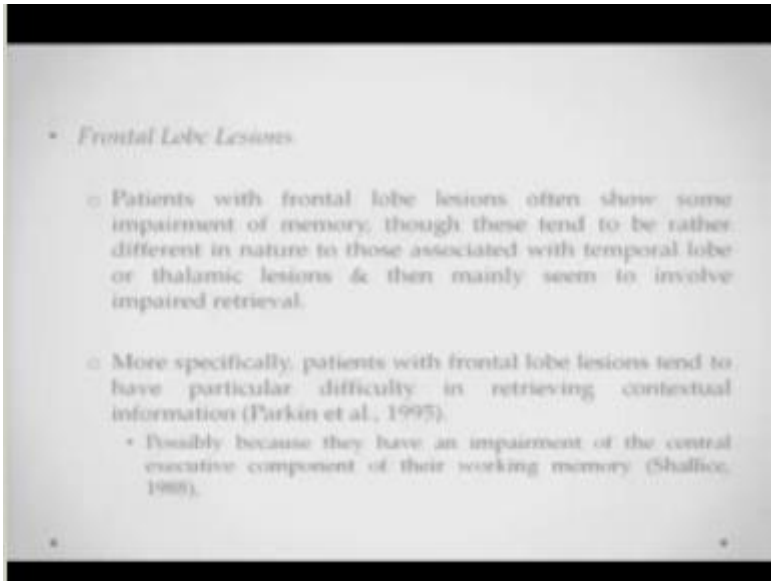
(Refer Slide Time: 46:33)



The ECT shock the patient typically shows both AAA and RA which might seem extensive initially but generally fair fates of you know with the course of time however it seems that for most patients ECT only causes a limited impairment a recent review of previous ECT study is conducted by Reed and menthol in 2010 they concluded that ECT treatment produces no lasting benefits but it does cause significant memory loss in some patients.

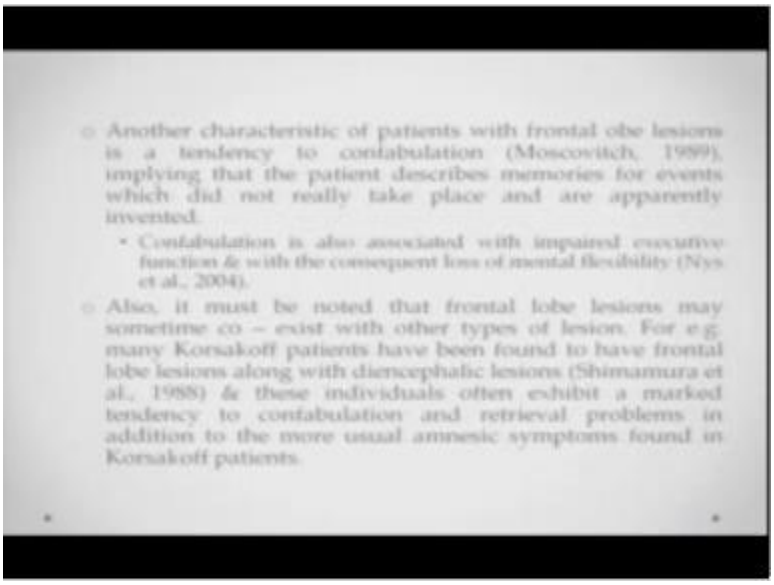
So in that sense you can actually say that you know you have to evaluate whether ECT really benefits the patient so much or it kind of leads to you know a typical impairments which will anyways fade off after a period of time so in that sense it could not be justified to use a CT to treat a large number.

(Refer Slide Time: 47:04)



Patients with front aloe lesions also often show some kind of impairment of memory though the set end to be rather different in the nature associated to them you know different in two different in comparison to lesions of the temporal lobe so these lesions basically say for example the temporal lobe lesions could be thalamic lesions or otherwise more specifically patients with frontal lobe lesions tend to have particular difficulty in retrieving contextual information placed in a particular context possibly because they have an impairment of the central executive component of their working memory so that's again something which was shared and said by Challis in 1988.

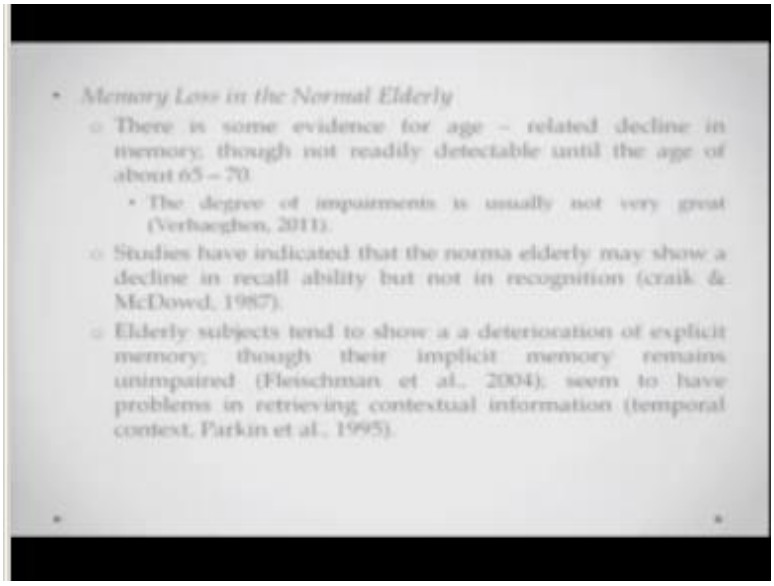
(Refer Slide Time: 47:43)



Another characteristic of patients with frontal lobe lesions is a tendency of confabulation so implying that the patient describes memories for events which did not really take place and are apparently invented so if you talk about patients with frontal lobe lesions they might come up with descriptions of events that have not really occurred or they might be mixing everything there might be a strand of you know event that is correct but everything else is made up so come confabulation this concept is termed is confabulation.

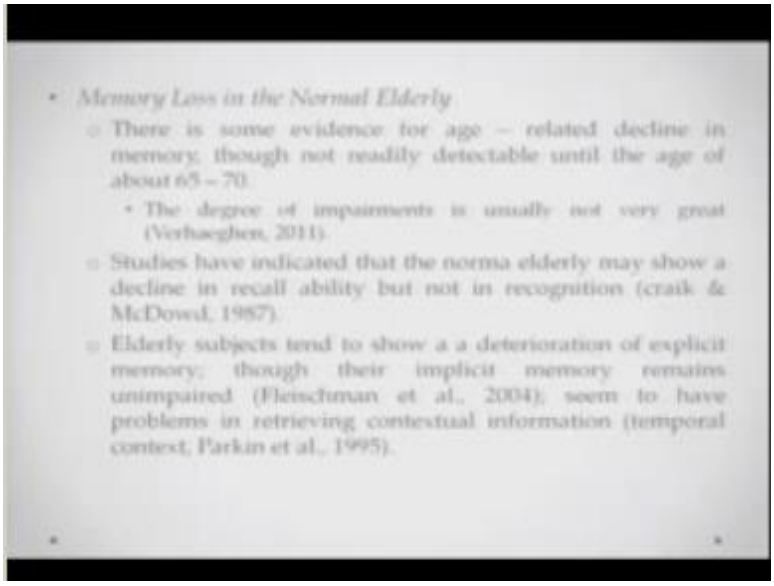
And confabulation has been found to be associated with impaired executive functioning and will also a consequent loss of mental flexibility also it must be noted the frontal lobe lesions may that is may somehow coexist with other kinds of lesions as well for example many caustic of patients have been found to have frontal lobe lesions along with diencephalon radiations and these individuals often exhibit a marked tendency of confabulation and then retrieval problems are also there in addition to their usual amnesic symptoms we can we have been talking about amnesia persons.

(Refer Slide Time: 48:43)



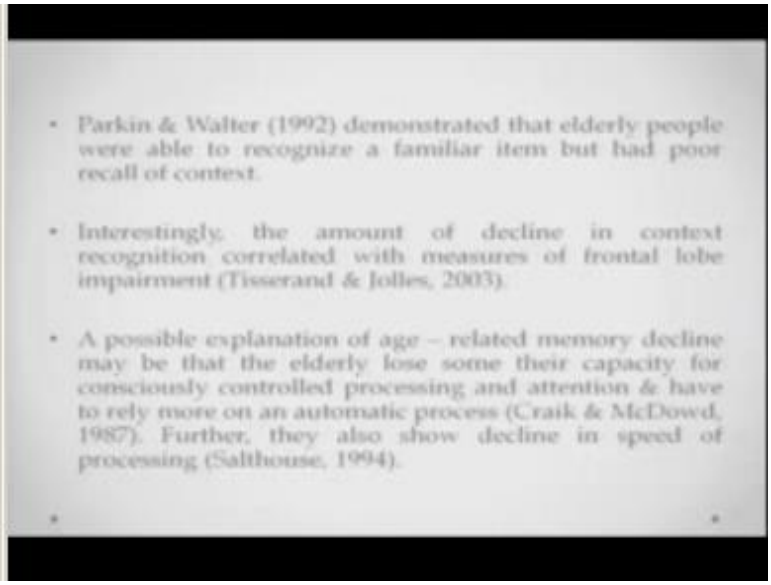
But we can also talk a little bit about the memory loss in normal elderly people there is some evidence for age-related decline in memory though not really readily deductible until till the age of 65 or70 the degree of impairments is usually also not very great so they might be general forgetting and it might be slightly more than you know people experience in younger ages but it's not as much as you know as severe as say for example in patients of amnesia studies were indicated that normal elderly people may show a small you know a relative decline in their recall ability but not really in their recognition so if you kind of have a you know queue in which they do not really have to recall information they might still be able to deal with this information easily elderly subjects.

(Refer Slide Time: 49:15)



I mean elderly subjects have been shown you know and have been ten have tended to show a deterioration of explicit memory so if you ask them explicit facts those kind of things might be deficient but they their implicit memory remains a rather unimpaired they seem to have problems in retrieving contextual information or information from you know a particular temporal context so this happened at that point in time I was doing this that kind of flow of information is generally impaired.

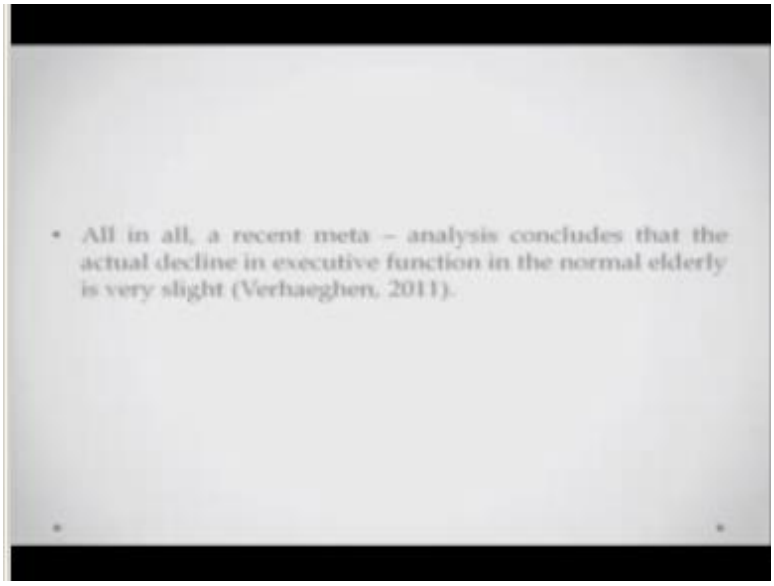
(Refer Slide Time: 50:02)



Parkin in Walter in 1992demonstrated that elderly people were able to recognize a familiar item but they would have a poor recall of context where did I meet this person I know this person but I do not know where I met this guy interestingly the amount of decline in the context recognition has been found to be correlated with the measures of frontal lobe functions a frontal lobe impairment if you take a test of rental Oh functionality with these people and you know amount of decline that you are experiencing in context of cognition.

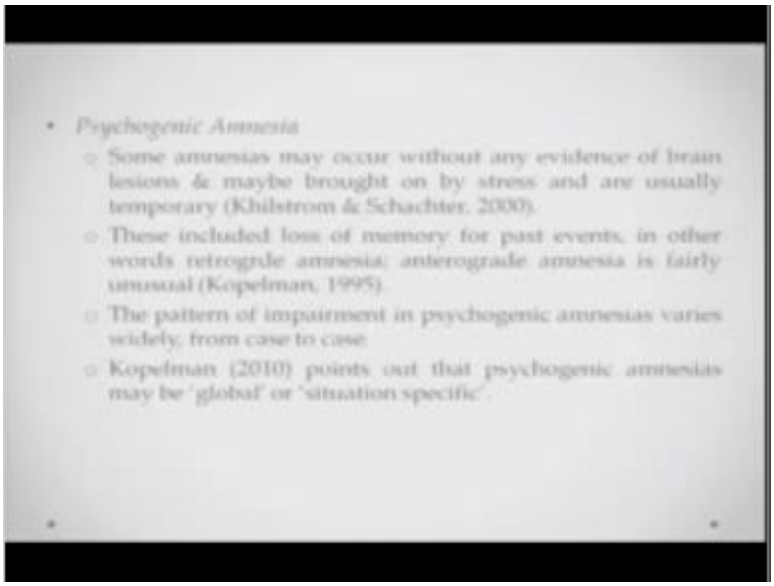
They have been found to be correlated with each other a possible explanation of these kind of age-related memory declines may be that the elderly people lose some of their capacity to control of consciously controlled crossing so they do not have this total a grasp on where to pick up a particular information from and so that they are trying to relying more and more on an automatic or a rather automatic process so that is some information that comes automatically is easy and easily retrievable is there but if the if you ask them to consciously recall some information that might be slightly deficient.

(Refer Slide Time: 51:10)



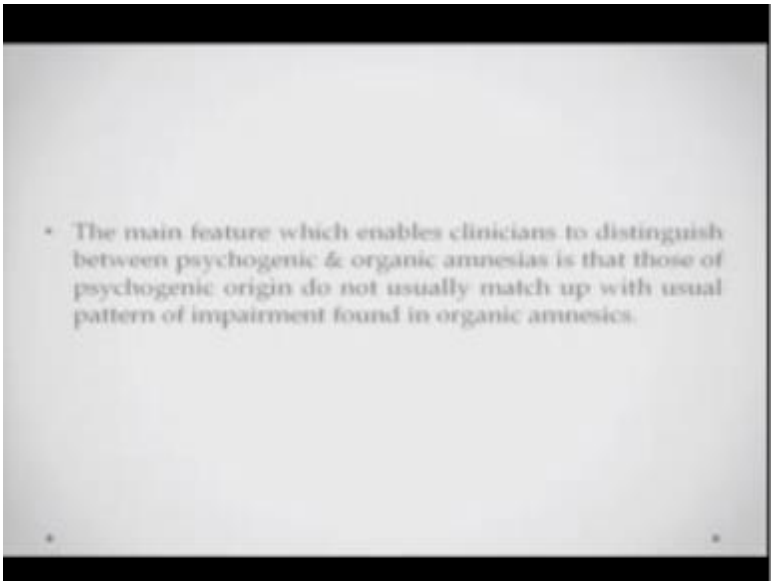
All in all recent meta-analysis concludes that the actual decline in executive function in the normal elderly is rather slight it is not too much.

(Refer Slide Time: 51:15)



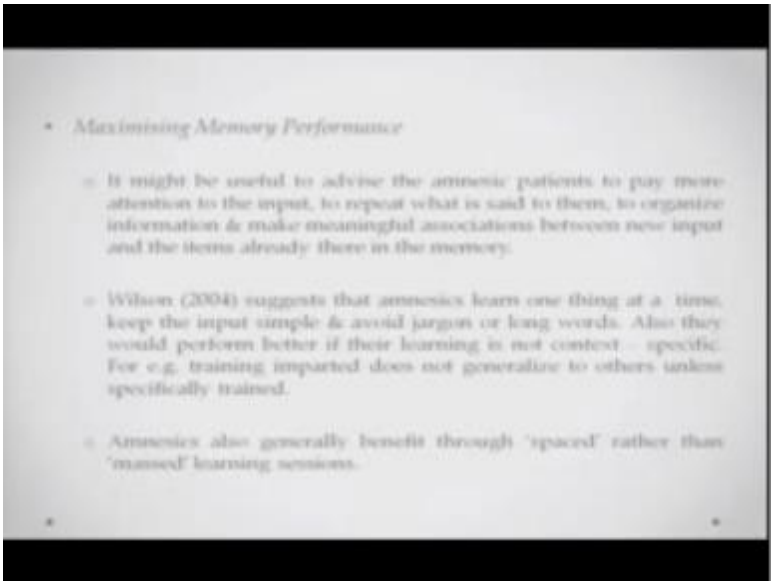
So we can talk about psychogenic missions Israel some emissions might occur without any evidence of brain lesions and may be brought on by stress tension and you know are usually temporary these include loss of memory for past events and in other words retrograde amnesia interrogator Nish is rather unusual it is not does not really happen with the psychogenic cases the pattern of impairment psychogenic amnesia varies widely from case to case and Cooperman 2010 points out that the psychogenic emissions may be global or situation specific it could be either of the cases.

(Refer Slide Time: 51:49)



The main feature which enables clinicians to distinguish between psychogenic and organic amnesia is that those of psychogenic addition do not usually match up with the kind of impairments that are experienced by organic mi-6 we can talk a little bit about rehabilitation why we are winding up so there are a large number of ways in which you would like to you know help these people help the organic so that they can lead a slightly more meaningful life you can come up with ways to help them cope with this loss of information and all of these come up come under the umbrella term of rehabilitation.

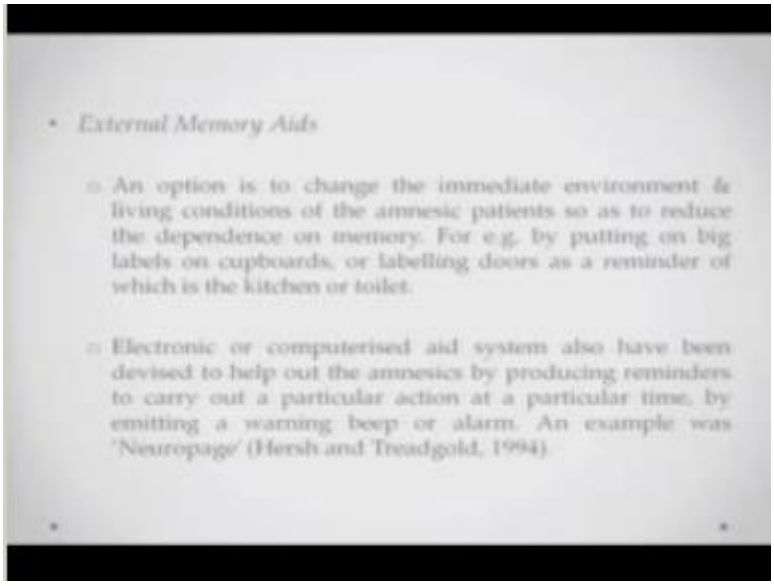
(Refer Slide Time: 52:21)



So for example other things could be maximizing memory informants so it might be maximizing memory performance so it might be useful to advise the amnesic patients or even the elderly to pay more attention to the input to process it more attentively to repeat what is said to them to organize the information you know very in an in a neat way and to make meaningful associations between new input and the items that have already stored in their memory Wilson in 2004 suggests that learn one thing at a time and one has to keep the input very simple avoid jargon.

And use of long words also they would perform better if the learning is not context specific if you are not you know treating them training them in a particular setting because this is not going to generalize to other settings as we have already mentioned and mystics also generally benefit through a space rather than mass learning sessions so if you if you make them learn small information in spaced out sessions they might be able to retain that information much better as compared if you give them a you jump of information at one time.

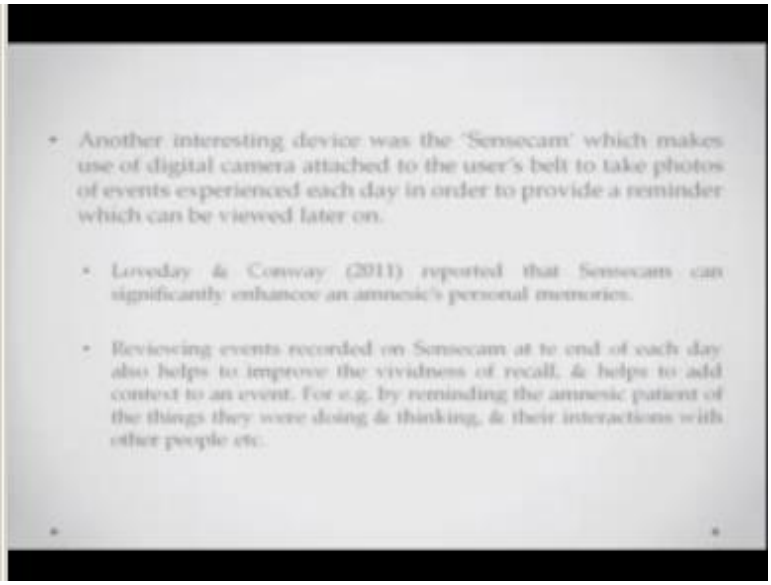
(Refer Slide Time: 53:27)



External memory aids have been found to be useful in the same Mystics say for example an option is to change the immediate environment and the living conditions of the amnesic patients so as to reduce the dependence on memory so you do not have to you know recall everything and then work say for example if you might have seen this movie called memento or the Indian version of this movie called Grainy wherein.

This person had all of those cues written all over his body so that he could remember that information which was relevant to recall so put putting big labels on the cupboards labeling doors as you know kitchen toilet etc might be helpful nowadays we also have the luxury of electronic or computerized eight systems which have been devised to help out the amnesties by producing reminders or carrying a for carrying out particular actions or a say for example reminding them of their medicines etc well simpler example was neuron page which was a fire you know worked upon by her shunt red gold and was found to be very useful.

(Refer Slide Time: 54:23)



Another interest interesting device that has come out recently was the same scam which makes the use of a digital camera attached to the user belt to photograph all the happenings of the day loved a and Conway have found2011 that same scam can significantly improve their music person's personal memories.

So whatever is happening and the end of the day they are missing person is sitting in his room and going through all the photographs of the day and attaching some kind of temporal context to them some kind of information to them that will be relevant to recall those events let us say on a later day so this is all about amnesia we have been talking about amnesia organic psychogenic and other causes that won't lead to memory disorders thank you.

<u>Acknowledgement</u>

Ministry of Human Resources & Development

Prof. Satyaki Roy

Co – ordinator, NPTEL IIT Kanpur

NPTEL Team

Sanjay Pal

Ashish Singh

Badal Pradhan

Tapobrata Das

Ram Chandra

Dilip Tripathi

Manoj Shrivastava

Padam Shukla

Sanjay Mishra

Shubham Rawat

Shikha Gupta

K.K Mishra

Aradhana Singh

Sweta

Ashutosh Gairola

Dilip Katiyar

Sharwan

Hari Ram

Bhadra Rao

Puneet Kumar Bajpai

Lalty Dutta

Ajay Kanaujia

Shivendra Kumar Tiwari

an IIT Kanpur Production