Bioelectricity
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Module No. #01
Lecture No. #22

Welcome back to the lecture, series in bioelectricity. So, we are into lecture number 27 the previous lecture, we talked about we summarized all the different sense organs like vision hearing taste olfaction touch pressure sensors all these things. So, at the higher sensors of the brain all these information's are coded electrical signals are being coded at different regions, and we remember all these information's they get consolidated over a period of time, and this consolidation of these information's, which we recollect whenever need it is called memory which is one of the central problem in science how biological systems remember things. Whenever we think about memory we think about you know in electronics we talk about binary codes 01001001 likewise, and these binary codes code the information's on c d disc or in mobile drives and. So, many other information storage systems.

But how does biology do, So, who these electrical impulses remain embedded within our system in what form how they retain throughout their life how we recollect things what happens when we sleep why is it being said that suppose you have a exam tomorrow morning. So, you should sleep tonight you should not do a night out, because your mind will go blank most of these questions are the questions of the or the questions, which will take us to the next frontier. These are pretty much the final frontier who are we, and what is that thing which ensures our very core of our existence.

Because this questions even become more profoundly challenging, when we see alzhiemers disease patient a patient who losses his own identity they forget who they are they forget the whole surrounding, because there are certain group of neurons within the brain which starts to die for totally unknown reason of course. It is known that they form certain aggregated moieties a beta ion, and bunch of it, but why it is triggered is not known similarly there are patients of parkinson they lose all their mortal functions there're specific areas within the brain, which starts to die out yet there're patients whose situation, then motor neuron in the spinal cord started to die out they suffer from amyotrophic lateral sclerosis. So, all of them have one common feature wherever these

neurons are dying out the assigned functionality of the neurons of that area is lost, and if it is lost in the hippocampal region, and the surrounding cortical region it is termed as alzhiemers disease where the person completely suffers from a permanent dementia they forget everything in, and around, and they die without knowing who really.

They are. So, it is very sad but that is the harsh reality of life that who are we is also fairly we do not know. So, this subject for centuries have inspired the psychologist scientist, and in the modern world the neuro scientist people who are unconscious memory. So, and. So, forth. Today we will just study a kind of scratch on the iceberg, because there is a huge amount of literature on all these things. They are pretty much subject in themselves what we will be doing will be talking about the anatomical features of the brain, and the electrical signals which are believed to code for memory acquisitions, and memory storage machine a very little of it, and of course, we will be talking little bit about sleep, and different rhythms of the brain, and the tools used to understand most of the brain waves electro, and cephalogram, and the different kinds of waves which are found in the brain.

So, before we embark into this brief journey to the very deep process of our brain which distinguishes, who we are we need to assemblate all the censor information's what we have gathered by assigning the part of the brain. Where they are stored. So, first thing we will do we will draw the brain a overall anatomy of the brain, and we will locate the different spots in the cortex or in the cortical region. Where the different information's are stored.

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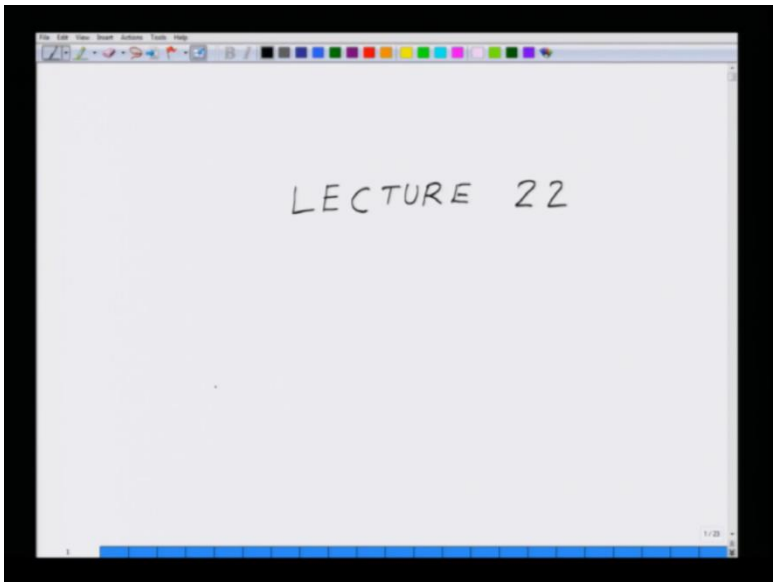

So, start off with. So, this is lecture 22.

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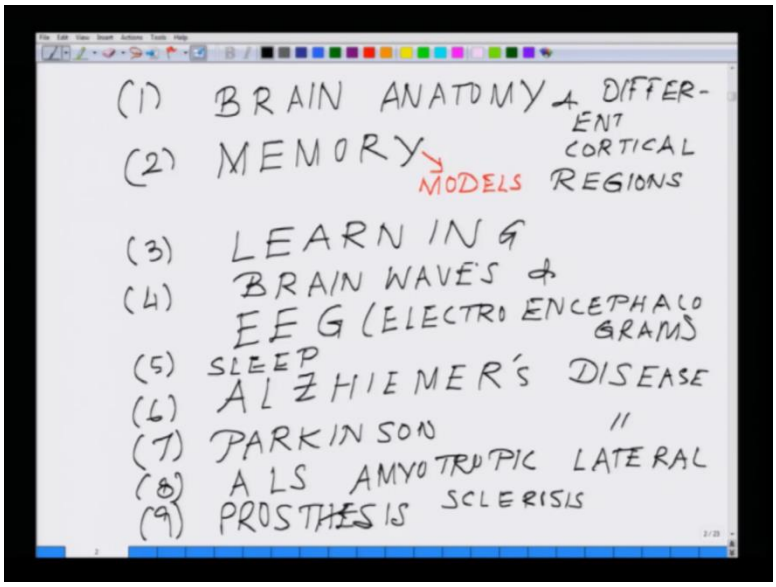

So, here we will be talking about memory learning electroencephalogram, and am I may not fall (( )) of the same sequence, but I am just kind of you know enumerating alzhiemer's disease parkinson disease, and of course, we will be talking about the brain anatomy very very briefly though brain anatomy, and different cortical regions one memory, and the different models of memory existing models learning sleep [fl] brain waves sorry brain waves, and e e g which is also called electro encephalogram.

So, we're talking about sleep alzhiemer's disease parkinson disease, little bit of we will be talking about a l s or amyotropic lateral sclerosis, and and some of the prosthesis. To start off with. So, we may need to you know move on to the next class to cover some these topics, but to start off with. Let us talk about the different regions of the brain which store different kind of information's

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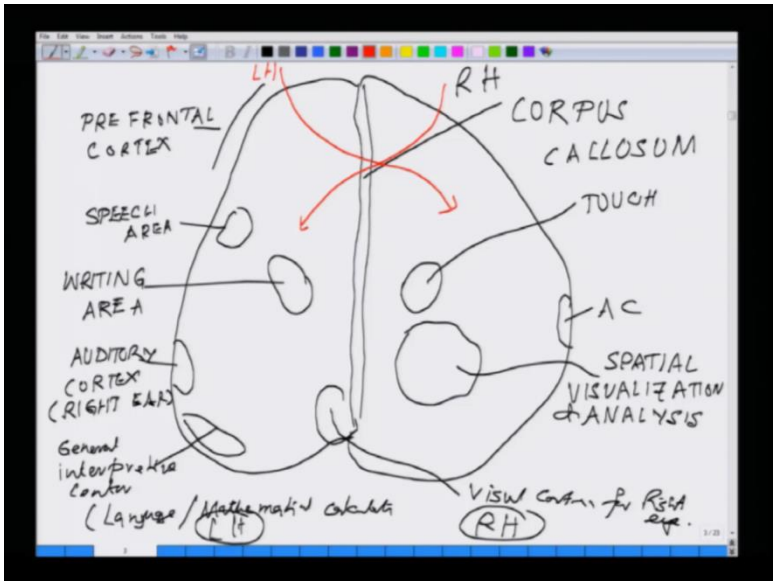

So, in the next slide, let me draw the overall architecture of the brain. So, if you look from the top the brain pretty much looks like this . So, there're two halves of the brain, and there're connected by a tissue which is called corpus callusum, then you have here the pre frontal cortex pre frontal cortex, then you have speech are, then you have writing area, then you have auditory cortex of right ear auditory cortex of right ear, then you have somewhere out here, which is called general interpretive center or mostly language, and mathematical calculations, and out here you have the visual cortex for a right eye.

Now, on the other side, if you look at it you have the sense of touch where of course, if the audital cortex the counterpart on this is a c, then you have special visualization, and the analysis which is sitting somewhere here people. There're certain people who could you know imagine three dimensional structures far better than other especially visualization, and analysis, and likewise in (( )) this is the right hemisphere, and this is the left hemisphere.

And it has been observed that this is the right hand, and as I have already mentioned right eye the information is processed on this side, and the left eye the information is processed the opposite side. So, this is the left hand likewise. So, if you look at it what is very interesting to note is these different areas. So, this is you're seeing from the top.

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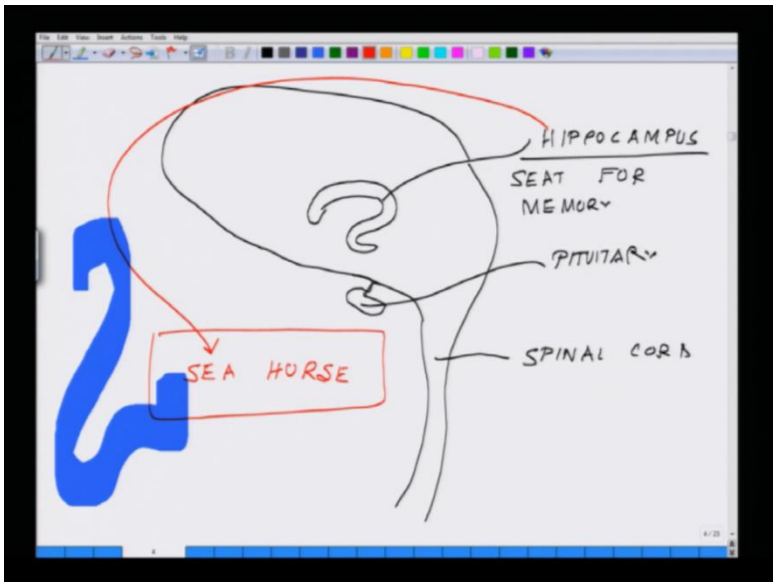

So, if you look underneath, if I just go little bit more on the anatomy, and if I have a side view of the brain it will be something like this out. Here you have organ called pituitary which is your endocrine organ, and here you have the spinal cord see, and out here deep inside there is an organ called hippocampus the seat for memory the name, it got the name, because this organ in Greek, it is called sea horse, and since it almost look like a kind of you know, get a get a three dimensional view it almost looks like looks like a sea horse, and that is why it got a name the sea a sea horse.

So, the first question is how it was discovered that, it is this organ which is the seat of memory. So, all this started somewhere middle of last century around nineteen thirties, and forties before that this whole area of memory acquisition, and memory was fairly dominated by the psychologist the modern year science has its beginning from the time of roman y cajal in the very early 19 century 1901 1910 cajal made his (( )) contribution. Where he they did silver staining with golgi you know silver staining, and all those things, and the whole anatomy was fairly clear, and out of the same time when sherrington, and all other people slowly electrical responses for the neurons were being

start people had started calling all these things. So, that was the kind of beginning of what you see today's modern bioelectrical phenomenon of the nervous system, somewhere around that time. So, 19 forties there was a very unusual event, and simultaneously there was another group thinkers who where developing learning models.

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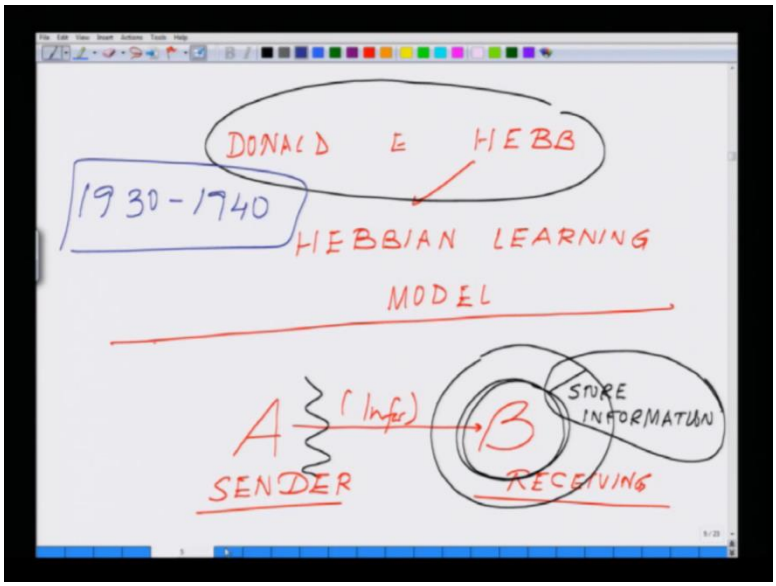

So, one of the learning model which was developed during that time was developed by a guy called Donald e hebb which is also popularly called hebbian, and this is he was psychologist hebbian learning model a very interesting model, this is among the very in the (( )) context is the most primitive or the most or the first one first of the learning models which essentially said very interesting thing it says say for example, there is a signal generated by a, and there is a receiver of signal b a is the sender of signal, and b is receiving the signal.

And both of them are active at the same point of time, then this information is getting. So, I am just putting info info is transmitted from a to b it may happen after a point even when a stops sending signal to b. So, in other word there is no more signal going from a b will still keep on receiving signal from a it sounds to a very paradoxical situation, because a is not sending any signal after a point, but b is still active is as if it is still receiving signal, and it is being said it is that at this stage this is the stage of b when it stores information, but this model of learning, and this is where the permanent changes

takes place in the network, and the network properties lead to the acquisition of information at this stage.

But this Hebbian learning or Donald E Hebb's learning model was not proved in biology till 1970 or half way through 1960s, but in between something else happened. So, if you talk about the genesis of Hebb's model it was around say you know nineteen or say nineteen thirties, and 1940s this learning model, was proposed this is a theoretical model nineteen forties late forties accounted for very interesting piece of event which took place in one of the hospitals in Canada what happened exactly, there was a mine worker this mine worker had a pathological problem. He was he was suffering from chronic epilepsy. So, very frequently he had to you know he used to get this elliptic bouts. So, what essentially happens in epilepsy is something like this

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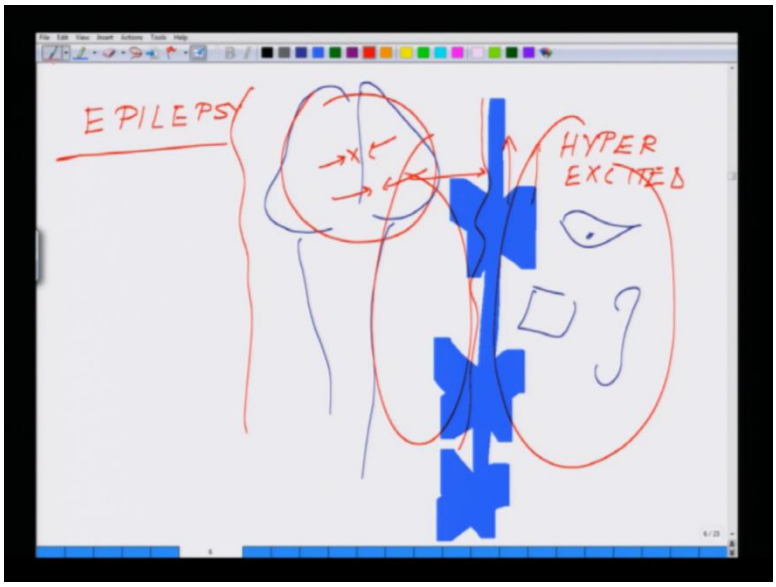

So, for example, this is your brain, and this is the spinal cord, and you know these are the different organs like you know eye is ear, and likewise. So, the epilepsy bout all of a sudden in this whole brain this whole thing becomes hyper excitable hyper excited, This is once again epilepsy, and when you become hyper excited. So, there is pretty much cleation of information's, and this person lose pretty much coordination with rest of its system like all this peripheral system, and this system they kind of dis-aligned from each other there is hardly any control left, and this person faints.

And these kind of elliptic bouts could be very dangerous suppose you're driving or something like you know, lose complete contact with your peripheral system. So, this mine worker was suffering from chronic epilepsy problem, and the every now and then he had to take a leave, and go through the medication, and everything on one occasions the neurosurgeons did something very interesting, what they did was they could figure out the zone of the brain from where this you know hyper excitabilities are originated.

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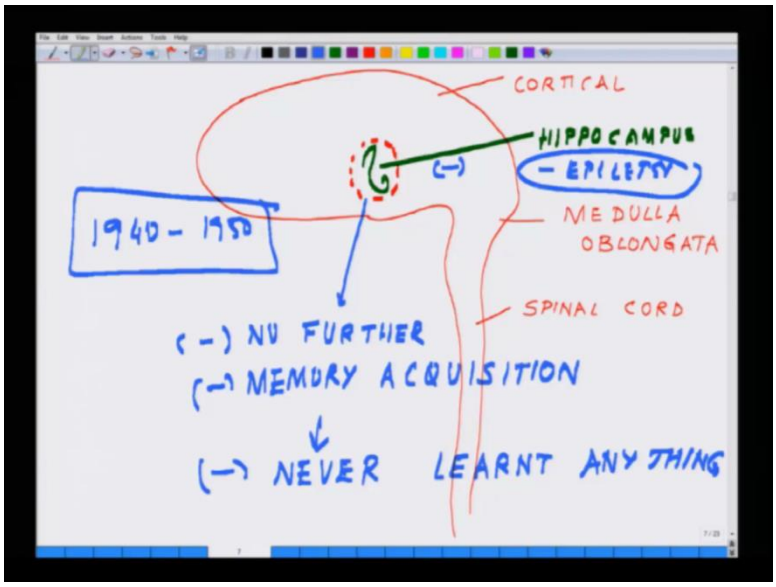

It is observed that within the brain again, I am just going by the side view of the brain. If you look at the brain like this, and if this is a side view this is the spinal cord medulla oblongata, and these are the different cortical regions, what I have showed in the first slide. So, out here I was highlighting about the area of hippocampus hippocampus. So, what the doctors did was they surgically removed, this part when they removed the hippocampus from the brain of this mine worker this individual got rid of his epilepsy though did not suffer from epilepsy, but from that day he never acquired any further memory. So, he lived like you know whatsoever was stored he never from that day he never remembered anything.

Isn't that very strange, but that is what it is that is what happened, they removed it. So, the result. So, you're removing it. I am just showing minus sign no further memory acquisition. So, this person lived all on his previous pieces of information never learned anything after that never learnt anything. This experiment or this is a real life situation

where you can call it experiment or this surgery by the doctors during 1940, and 1950 open up a question of learning, and memory again in a much more bigger way, because now, the doctors realize or the neuro scientist realize the major organ or one of the major one of the rate limiting organ in memory acquisition or memory storage or information processing apart from the cortical regions is hippocampus.

So, if you look at it historically this was absolute accident, because doctors wanted to help this patient, and you know, they just removed that part of the brain which there're suspecting that somewhere most of the epileptic bouts where originating, and result defiantly minus epilepsy, but minus no further memory acquisition, but now, the question arises is this area could also show what has been proposed by donald hebb fairly on the same time does this area showed any form of hebbian learning model, and if. So, how is it.

So, next thing came. So, I am just highlighting. Those wonderful work which had been done by a bunch of people during the last century which set the tone for our modern day. research in understanding the memory acquisition phenomenon And the next thing came was done by three gentlemen bliss lomo, and collingridge.

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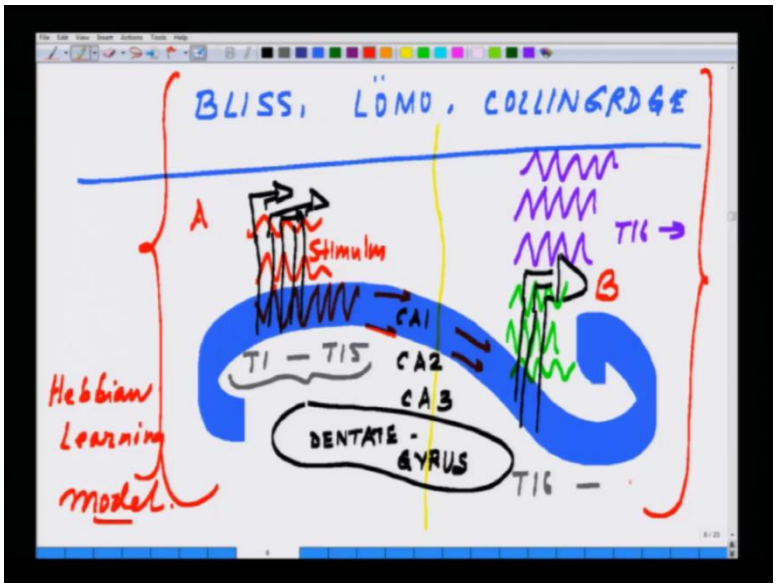

Bliss t v p bliss lomo, and collingridge, and the venue this time was europe. So, this happened in canada, and now, back in england what was being done that is in a guinea pig they removed the one second let me try it they removed the hippocampal region. So,

now, you have a wonderful hippocampal tissue out there, and in that hippocampal tissue they implanted electrodes like, this I am just putting electrodes. Now, and by the way this hippocampal structure is very well organized it has different regions which are named as c a one c a two c a three, and underneath there is a region called dentate gyrus.

So, the implanted electrodes are there vertebral electrodes, and the implanted electrodes in another anatomically another distinct region of the brain within the hippocampus. So, lets name these electrodes as region a, and region b, then what they did they started giving stimulus from here high trend stimulus going. So, automatically through this pathway. So, this is a connecting pathway the signal will be reaching here. So, on these electrodes you could now, receive the signal. So, there're receiving signal.

Now, say for example, I have to time say t 1 to say t 15 I am speaking arbitrary numbers time 1 to time 15 they get down getting a stimulus, and you know automatically as they were giving a stimulus electrode b was receiving the signal, and what they did at t sixteen on they stop giving signal, and what they observed was b is continuously receiving signal in spite of the fact t 16 on for a while for fairly good amount of time in spite of the fact there is no more signal coming from t from the a region this was a stunning discovery.

So, it means now, if you go back to the hebbian learning model, a is sending signal b is receiving signal, but after a point a stop giving signal stop no more signal is still b is receiving signal for the first time this was a wonderful journal of physiology paper for the first time hebbian learning model was proved at indeed. There're regions in the brain which follows a different set of computation, and this process of memory acquisition was termed as long-term potentiation which is one of the most expected models of memory acquisition which in short is also called l t p.

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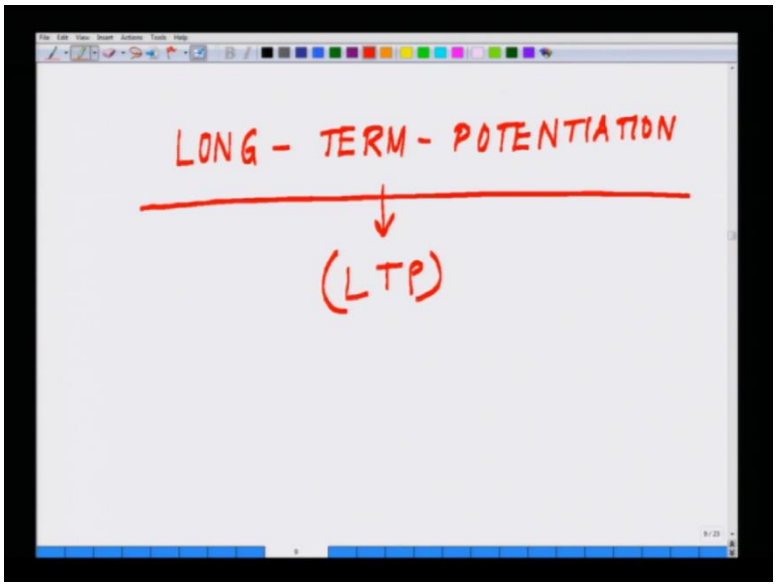

Now, the question arises should, this electrical phenomenon does exist, but does this does phenomenon exist in the intact brain or not, because this is a slice of tissue which you have taken out, this answer in last fifty years we haven't been able to answer this still we have a long way to go, but this question has just opened up the whole field of neuron competition in electrical signal processing that what is exactly happening.

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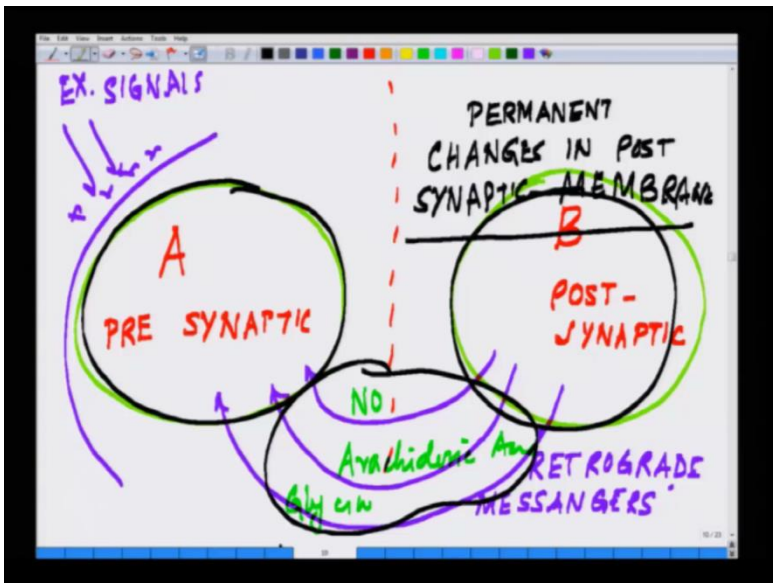

So, now, the first question which comes in mind is that between a, and b is it if I consider these as pre synaptic, and the one which are sending signal, and these are post synaptic

neurons, then is this a pre synaptic phenomenon or is it a post synaptic phenomenon, and if it is whatsoever the phenomenon is are there retrograde transport available are there messengers which are sent even, after the signal is over are there signals which are being sent to a telling that you keep on sending more, and more signal in spite of the fact the external signals have all stopped there is no more external signal being given if these are considered as external signals.

Then if is it. So, which is suspected it is. So, then what are those retrograde messengers who are those retrograde messengers there are indication towards very simple molecules like you know nitric oxide n o arachidonic acid glycin likewise, but again none of them have been proved beyond doubt that yes, these are the retrograde messengers still there is lot of controversy over the fact that is it a pre synaptic event or is it a post synaptic event.

What you know for sure is this phenomenon indeed happened hebbian learning model indeed worked, but this happens when there is very strong train of signal sent by a strong train of signals now, the question arises. What are the permanent changes taking place in the post synaptic membrane permanent changes in post synaptic membrane these are the questions which people are trying to address, but it is another thing which comes in mind.

So, this is a situation when say for example, you're trying to remember a new poem or a new piece of information in terms of it two two za four or something like that, but say for example, you just observer something for a fraction of a moment or you see a snake or you see a blast you still remember it. So, in this situation the signal is not very like you know not a huge train coming for a while is one stuck moment is thus this follows as follows as our long term potentiation model or say for example, you do by cycling your biking your walking how these are coordinated you never do a memory recall that all these processes. There must be other coding information, and that takes us to the next set of information coding which falls under another model which is called long term depression model we will come to that, but before I come to the long term depression model what essentially is happening.

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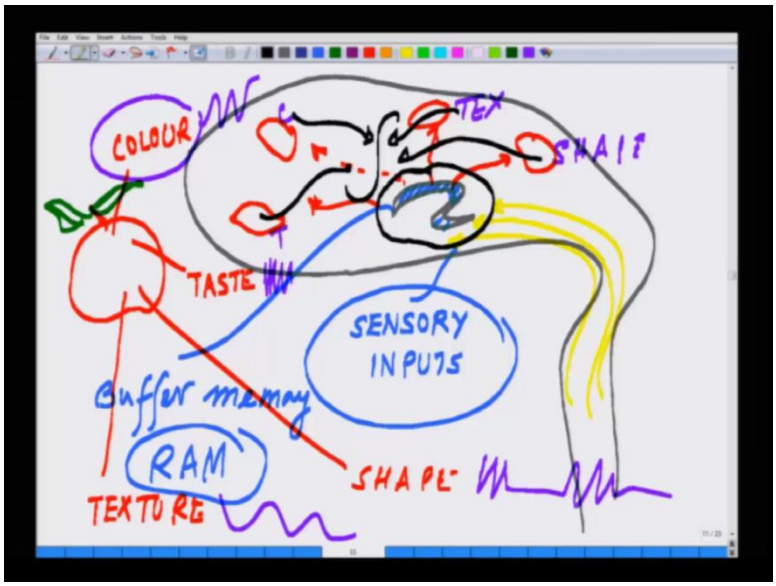

So, when if we believe now, the way the current theory says if I am giving a side view of the brain now, out here if this is the area which is involved in the hippocampal region. So, it is believed what is happening is this first of all the sensory inputs are reaching here through the spinal cord the yellow arc showing the sensory inputs all these different sensory inputs.

These sensory inputs are initially stored just like a buffer memory out here just what you see in a computer as a ram random access memory buffer memory or you can call it random access memory ram chip the bigger the ram you have better of you are, then from here there is a consolidation phenomenon takes place where basically your information's are sorted out, if you remember when I was telling you that if you go back to the first slide second sorry yeah this slide. So, there're different region the speech component there is a writing component. There is a auditory component there is a special visualization component there is a touch component there is a visual component visual cortex out here likewise now, coming back to this it is exactly the same thing now, each one of these different components are going to their different regions say for example, we observe something we observe, and say now, an apple.

So, it has see look at the component it has a colour it has a taste it has a texture it has a shape hmm maybe, it has some emotional value with your something you know, So, all these information. So, for our nervous system these are electrical signal these different

electrical signals are stored at say colour region taste region texture region shape region likewise. So, the same thing is coded at defendant places, and whenever we have to recollect this all has to be further integrated to realize that this is an apple.

So, essentially what is happening is your hippocampus is acting as the zone of buffer memory, and from the hippocampus slowly, and gradually like information's are being transmitted to the different areas of the brain where they are permanently stored. So, when this zone this area starts to die off we do not acquire any further memory, and many a times we lose our own identity, and what exactly happens in the hippocampal region of course, we will come in depth of the cellular architecture of this, and the information.

So, before I even go to the long term depression, and all other models this is very interesting to understand that you know, how this area actually. So, as of now, this is the most accepted model of memory acquisition that something happens or some in the memory is acquired in the hippocampal region, and it is being slowly you know transmitted back to the different regions of the brain where different pieces of information's are getting stored into bits, and pieces.

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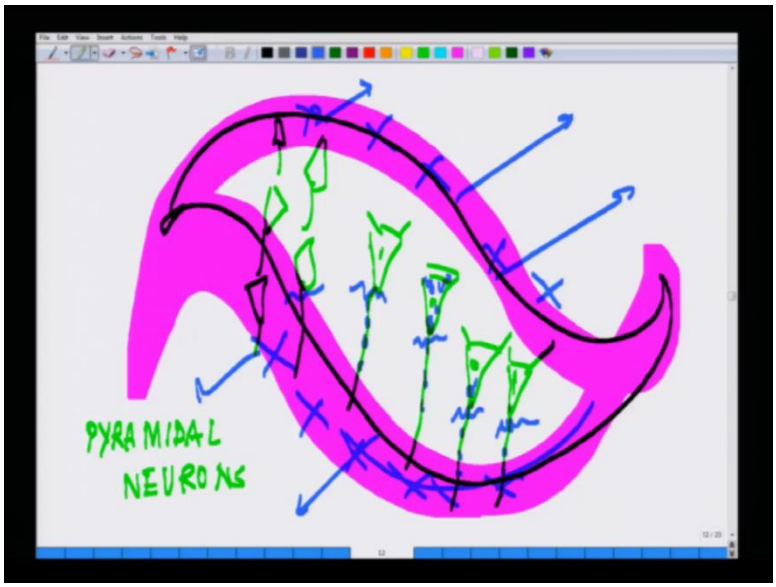

So, what essentially happens in alzhiemer's I am I am not entering into the any long term depression at this stage I will be getting there very soon. So, what happens in the alzhiemer's now, first of all we have to look into this structure. So, let us investigate this

structure this structure is as I told you is a structure like this. So, this structure is a multilayered it is a very three dimensional structure this is not something the way I am drawing it is much more complex structure like this.

So, now, here the neurons are at different level neurons are kind of you know is a layers of neurons which are present out here, and most of these neurons have a very characteristic shape. It is a body more like a pyramid, and these are called pyramidal neurons pyramidal neurons, and do you know alzhiemer's are out. Here there is a accumulation of there is an aggregation of certain specific proteins which are called a beta proteins, and which the current. if the current theory has to be believed leads to a blockage in the electron electic ionic electricity transport pathway, and eventually what happen they are cell bodies there processes started to die out what I am showing now, is the process started to die out, and these cells fail to communicate with the rest of the system, because there processes are now, being chopped off by this a beta peptide which is getting accumulated, and the electrical signal does not pass through, and eventually this whole part of the brain.

If you go back, and this is the side view of the brain, and this whole part is kind of you know gone all the perennial neurons dies out here, and that is what leads to alzhiemer patient to lose his own identity. Same way there are regions within the brains like if I go back here out here there are within the cortex there are motor cortex areas of the brain, where the motor activities which are involved the area of motor cortex within the motor cortex there are very specific areas, which are called substantia Niagara.

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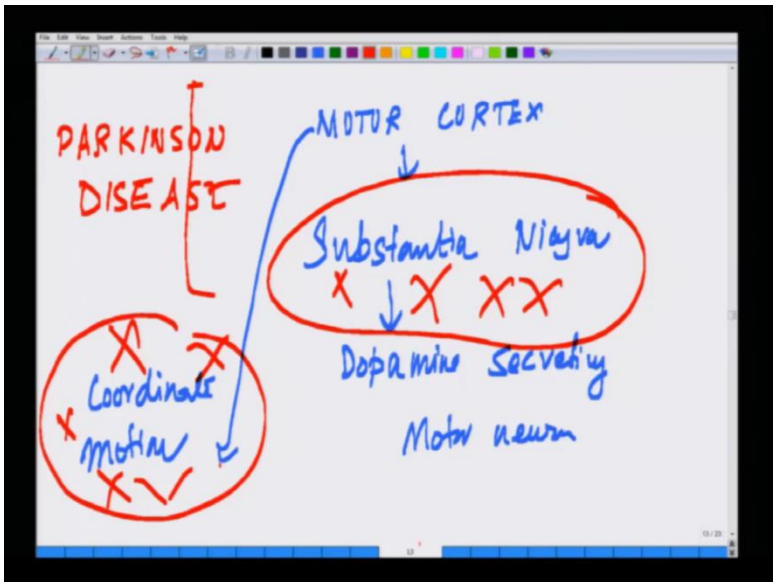

I'll just highlight it in the motor cortex here is this area called substantia niagara these this area there are motor neurons which secretes dopamine, and this dopamine secreting motor neuron, and this is this area which coordinate motion. Now, for some absolutely unknown reason this substantia niagara motor neurons started to die out, and what happens is this motion coordination is all lost, and this is the disease which is called Parkinson disease. So, if you look at if you compare the previous slide where we talk about Alzhiemer's disease, and in this slide. If you're comparing it with the Parkinson disease both of them are neuro degenerated orders, where the neurons are getting degenerated, but is a fundamental difference between the two the fundamental difference is that in case of Parkinson disease, you are losing your motor coordination, and in the case of Alzhiemer's you're losing your very basic who you are you cannot acquire any further memory the Parkinson patient does not suffer from dementia.

So, though this process of neuron death is rarely, similar in this case alpha sonically there is a protein is getting aggregated out there in parkinson. So, they all if one has to get they all fall under a protein aggregation problem, somewhere out there the machinery through the electrical impulses are being transmitted are getting choked up or blocked or as if there is a traffic jam out there. So, if you really look at these neurons the way they there their like you know. If I had to show you in three dimensional picture. So, there will be something like this what what you kind of you experiences.

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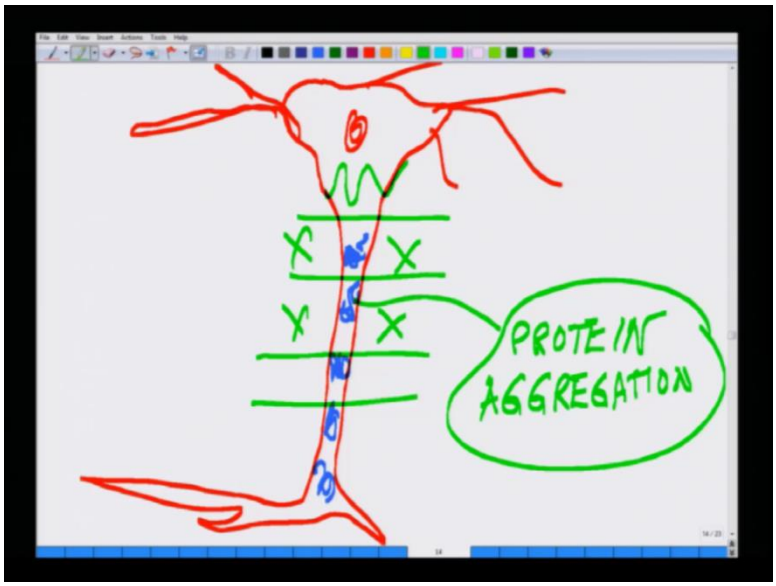

These are the process all over the neurons you know, and body. So, what you see there is lot of aggregation of proteins out here, and, because of this aggregation electrical impulses which are originated out here fails to travel through this, and eventually they die out. So, this is the protein, and peptide aggregation. So, both alzhiemers, and parkinson, and even amyotrophic lateral sclerosis are failed in similar problem there is a neuro degeneration, but there are different proteins involved, and it is very interesting that one area does not influence as the other area as of now, there is no knowledge, but end of the day that destroying that part. So, now, if you see this image what I was trying to show any of these are any of these area kind of you know is affected by any specific disease, then we will be losing that motility or say for example, there's problem with the visual cortex you lose the vision.

If there's a auditory cortex problem you'll lose the auditory ability, if there's a problem in the writing cortex you'll lose the writing ability if there is a problem in the touch cortex touch cortical region involved in touch. We will lose the sensation of touch if this region is kind of getting affected the special in visualization analysis, then we will be we will not be able to coordinate, and if there is a damage in the corpus callusum, then the connectivity, and the cross talk between the left, and the right side of the brain will be hampered.

So, this is what I wanted to highlight in terms, of the long term potentiation, and the memory acquisition process. So, what we will be doing I will close this class here, and in next class we will be talking about long term depression, and we will talk about all the brain waves which are involved in it, and little bit about the sleep, and, then we will talk about some of the neural competition, and neurotransmitter. Thanks a lot.