Lecture 43: Type of Plasticity

Welcome. This is now we are discussing the the synaptic plasticity where we have discussed how the different kinds of modifications can take place. And now we will be discussing about the types of plasticity. So in this in this course for us the main point would be about plasticity that has to do with existing synapses. And that is what we will mean by that is synaptic plasticity and that is that there is already a synapse between two neurons and how the synaptic efficacy the transmission of the synapse efficacy of transmission or the strength of the synapse which is the current that is the postsynaptic sides measure of the synaptic strength that is changed with activity. So we have discussed that it is really the input and output activity that ultimately modifies the synapses because at the synapse if you remember if we have the synapse we will draw this diagram where the axon of the presynaptic terminal and this is spine on the postsynaptic side.

This is the dendrite of the postsynaptic neuron and this is the neuron cell body. This is the postsynaptic neuron cell body and this is our synapse. This is dendrite. So if you remember we are talking of how this existing synapses strength is changing.

However there are other types of plasticity where there are new synapses that are forming new synapses between two neurons or even the same neurons and similarly if certain inputs to neurons are not there then the usually which neurons do not connect to those neurons now they may be ending up creating projections onto those neurons. All these kind of things can happen and similarly here also there can be new spines forming and collaterals may be forming which effectively can increase the strength of these synapses that is due to the spike in the presynaptic side there will be now more current in the dendrite of the postsynaptic neuron and will effectively enhance the synaptic strength. So what are the types of plasticity then in that sense first of all we have what is called the structural plasticity. In structural plasticity we essentially mean that there were no synapses originally and new synapses are forming between two neurons. Also in terms of structural plasticity we can mention that initially there are lots of projections made by a set of axons from a neuron from a structure going to making projections onto another structure and with time with activity not no activity in some of the synapses or many of the synapses they get pruned away and so that is also a kind of a form of structural plasticity in the sense that the synapses that were made there that are not existing anymore.

So some of the examples of these kind of structural plasticity that have been studied is for example when a region of the cortex is devoid of any inputs is I mean the inputs to that region inputs are removed due to let us say surgery or some kind

of lesion some that axons projecting to that region of the cortex let us say there are a bundle of axons that are going into that region and I mean it is necessarily not cortex but for example for the examples we will be talking about it will be cortex and there are neurons here the large number of them and this input is somehow gone. So it is deafferented so afferents or the fibers projecting into that region are gone and so what happens in such cases in their multiple examples is that the surrounding regions actually starts sending collaterals of axons and finally that region is taken over by an other region by including those neurons as their functional counterparts and so this whole region effectively starts to do the same sort of function. So this kind of plasticity is structural and is also sometimes called as a expansion of maps. Expansion of maps kind of plasticity so what these there are multiple examples of these that have been studied and for example the primary example that we can give is that of representation of the digits in the somatosensory system. So if you recall when we discussed the sensory systems briefly we said that in the somatosensory cortex there is a map in the sense that neurons at specific locations in the cortical region in the somatosensory cortex are sensitive to stimulation on particular regions of the skin or the body and neighboring regions were mapped into neighboring regions in the cortex like in the visual system it is called retinotopy in the auditory system it is called tonotopy in the somatosensory system it is somatotopy and so the hand and the palm and the different digits d1, d2, d3, d4, d5 they are represented nearby each other.

So in the cortical region if we say that this the neurons in this region are sensitive to d1 the first digit or finger d2, d3 and so on. Now the experiment that was done is that two of the fingers were bound together as if there was no function of any of a particular of them separately. So whatever those fingers did we are doing it simultaneously. So in other words the two fingers outputs that the information that is being carried by the afferents finally into the somatosensory cortex of d1 and d2 or let us say d2 and d3 are now combined they are turning out to be as if there is only one particular finger and then the d2 and d3 this whole region became developed properties of stimulation on this entire fused digit. So now the representation of d2 and d3 were not separate anymore that is the previous d2 region could now respond to regions of d3 and previous d3 regions could now respond to regions of d2 and so they got combined.

Similarly when a finger is amputated in a case where it is a requirement let us say in that case the representation of the amputated digit so if you have the d2 and let us say d3 this is our d2 and this is our d3 region then if the d3 is region is cut off then neurons from d2 and the neighboring d4 then start to project into the neurons of d3 and gradually take over parts of it. So now since the actual inputs of d3 are

totally gone now the neighboring regions take up the neurons to function as a d2 or a d4 kind of representation. So this kind of plasticity is occurring throughout development and also in such scenarios where in fact it is called the phantom limb effect where a particular limb is cut off then there is a feeling of presence of that limb because the neurons that used to be sensitive to that particular limb or on that limb regions on that limb they are now being used by other regions of the body and hence there is a sensation of that limb still being present and that is only because of that region being taken over the neurons that were representing that limb being taken over by the neighboring kind of positions on the body. So this similar kind of work that has been done in the auditory system is the same as the sensory system is basically just it is an expansion of a particular frequency and this happens in a particular period of development called critical period and that is a window of opportunity to modify the developing cortex permanently and so what is done in experiments let us say if it is a mouse in which the experiment is done or ferret they have specific window during the developmental period after birth and if that animal is exposed to so this is birth and this is the critical period and then normal development in adulthood. So manipulations done to the system in this period causes profound changes that are reflected in the adult.

So in the auditory system is this map expansion has been shown to be like this that if we present a particular frequency tone let us say 5 kilo hertz or 7 kilo hertz tone that is presented during the critical period continuously like tone and then a gap tone like that it is presented continuously. Then what happens is that in the adult when you look at the tonotopic map in the primary auditory cortex if you remember there is low frequency side on one side and the high frequency representation on side and the medium frequencies in between. So if we have regions of 2 kilo hertz neurons that are sensitive to 2 kilo hertz then and gradually shifts to let us say 16 kilo hertz or so in this region that is neurons here are tuned to low frequencies up to high frequencies on the other end. So 7 kilo hertz being somewhere in between this is the actual usual area of representation of 7 kilo hertz in the sense that you get neurons with best frequency of 7 kilo hertz in that region a majority of them. Now when this 7 kilo hertz is presented during the critical period then this 7 kilo hertz region gets an expanded representation in the cortex that is the regions that were for neighboring frequencies like the 6 kilo hertz or the 8 or 10 kilo hertz those regions are now devoted to the same 7 kilo hertz frequency that is in terms of the tonotopic the representation of other frequencies neighboring to 7 kilo hertz get reduced while the 7 kilo hertz get expands at the cost of that reduction and further away there are no changes.

So this again is a form of structural plasticity that is happening that is caused

by an activity during the critical period. So similarly in the visual system there are cases where if you do I if you stop representation I mean if you stop stimulation of one particular eye then the ocular dominance that we have talked about in our systems discussion systems neuroscience discussions or sensory system discussions we have seen that there could be there are neurons in the visual cortex that get input from one eye more strongly than the other eye and there are alternate bands of regions in the visual cortex in the columns which are dominated by inputs from one particular eye. So that in the visual cortex we find the ocular dominance columns in the sense that in the cortical region we have this is the left eye dominated this is the left eye dominated this region is right eye dominated and you have these alternate bands in the cortical region throughout the column. So this is the surface and this is the right eye dominated and this is the white matter at the bottom and the cortical column is along this axis that is and the layers are along this parallel to these lines throughout layer 1 layer 2 to layer 6. So in the normal case we get these ocular dominance columns where the neurons are majorly representing the one of the eye inputs and they alternate if during that critical period if we do an eye switcher if we do an eye switcher we have a structure that is one particular eye is divide of any stimulation then these ocular dominance columns do not form in the adult.

So it is only during the critical period that if you do the deprivation to one eye or stimulus deprivation to one eye then the OD columns do not form ocular dominance columns do not form. So this is again another case of structural plasticity due to lack of activity of inputs from one particular eye and hence that is translated across from the retina to the LGN which is the calamus of the visual system and into the visual cortex where these ocular dominance columns normally form. So these are gone from the visual deprivation of one eye in the critical period. So there are many many more examples I took only a few two of the three systems to show you what this kind of plasticity is like. For us it is going to be now a discussion mainly that we will follow which has to do with the plasticity of synapses that we will call synaptic plasticity and we will be discussing this for the synapses that are already present and they are being modified in terms of their strength or efficacy of transmission.

So and this synaptic plasticity that we will discuss occurs almost at all synapses in the eye. Some way or the other present in all synapses its I mean its presence may not be as we fall clearly as we will be discussing but some form it exists almost in all of the synapses that we know. And this kind of changes in synaptic strength is ongoing and totally input output activity driven and we usually classify this synaptic plasticity in really two forms that is a short term plasticity and long

term. So what we mean by short term plasticity is that let us say we measure the synaptic strength for particular synapse by producing a spike in the presynaptic neuron and we do averaging and in the post synaptic side we measure the membrane potential and the EPSP. So let us say this is 1 milli volt EPSP that we see on average due to the spike in the presynaptic side.

Now with this with some kind of manipulation again later on we can see that with the same measurement that with the same spike in the presynaptic side we may get either a decrease in synaptic strength or we can get maybe an increase in synaptic strength increase over the previous size where the previous size is some let us say was like this. So in the green what we are showing this is both this is facilitation or potentiation facilitation and in this case in the case of the red one where it is reduced it is called depression. Now the difference between the two is that now the effect that we are seeing if the system is left undisturbed like you are not changing making any changes in the system for a while and do the same measurement again. If we get back our original synaptic strength that we had started with. So if this post synaptic potential EPSP is of amplitude A at the end when we measure the synaptic strength again we get back our A.

So this period of time where it recovers or it is left undisturbed is of the order of seconds to minutes and so the effect that we are seeing of depression or facilitation either of those that is not long lasting that is a temporary effect and we get regain our synapse back to the same strength. So this is the effect that we are seeing of the same strength. So this temporary nature of synaptic plasticity is called short term plasticity and particularly the two cases where we have short term facilitation with or short term potentiation these are the two which is our green case and in the other case we have short term depression which is in the red. So this temporary phenomena of change in synaptic strength that is what we are saying because the EPSP is changing its amplitude. This is short term plasticity on the other hand what we mean by long term plasticity which is long term is that we let us say again we are measuring the synaptic strength by producing a spike pre-synaptically and we measure the average strength of the EPSP again let us say it is a millivolt height or height of A.

So this is the post synaptic side now again we do some sort of manipulation to the activity of input and output or just input it depends and then again we do the measurement that we have a pre-synaptic spike and postsynaptically we measure the postsynaptic potential let us say it increases to A'. We leave it undisturbed for a while and measure it again and we see that it is still A' or at least larger than A it can decay a little and it keeps going you measure again it is still A' and for measurable periods of time this remains at A' or value larger than A and saturates

to that and saturates at that value that is it keeps on its synaptic strength. So this can be a period of hours 1 to 2 hours over which this kind of observations have been made and this is what we will term long term plasticity which has an early phase and a late phase and we will be modeling bit of the both short term plasticity and long term plasticity at least the kinds that are used in general in models in the literature and similarly with that same other some other manipulation in this period we can get a depression of synaptic strength. So this is let us say A'' so A'' is less than A and it continues to be so throughout that period of hour to 2 hours and that is long term depression. So what we have here is long term potentiation in green and we have long term depression here in red.

So these are more permanent more longer term changes in synaptic strength and these essentially it is not just the duration that is important it is the temporary nature versus the longer term nature and more specifically in the long term environment of protein synthesis that shows that it is long term plasticity. Once we discuss more in detail about the long term plasticity it will be clear when we will be modeling the processes of long term plasticity then it will become more clearer as to what manipulation we are doing here or what manipulation we will be doing here. In order to observe long term facilitation and long term depression or short term facilitation and short term depression. So next we will go on to more detail discussions of short term plasticity and long term plasticity and will be modeling each of them in order to implement them in networks which will be adaptive in nature which will be learning based on activity. So next lectures will be on short term plasticity. Thank you.