Introduction to Brain and Behaviour
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Lecture 04
Neuronal Communication

Hello and welcome to the course introduction to Brain and Behaviour. I am Doctor Ark Verma from the Indian Institute of Technology Kanpur. I am working with the Department of Humanities And Social Sciences and also with the program for cognitive sciences.

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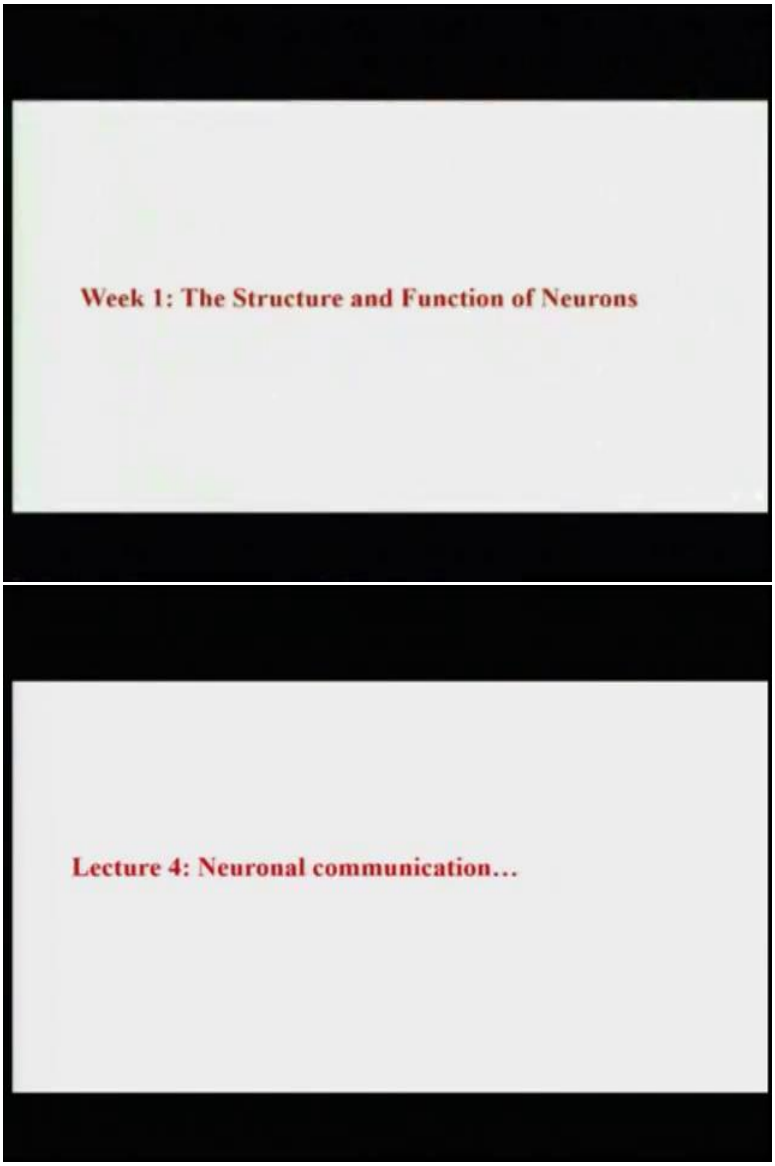

This is the first week and we talking about the structure and the function of neurons. In this particular lecture, we will talk about neuronal communication. If you remember from the last lecture, we talked about, how is the energy generated, how is the potential generated in the neuronal membrane.

And in this lecture, we will basically talk about how does the process how does the process of communication with between two neurons initiate and that is basically through a process called action potential. So, let us talk about this. Now, this process of the generation of action potential actually begins when excitatory postsynaptic potentials at synapses on the neurons dendrites cause ionic currents to flow through the volume of the cell body.

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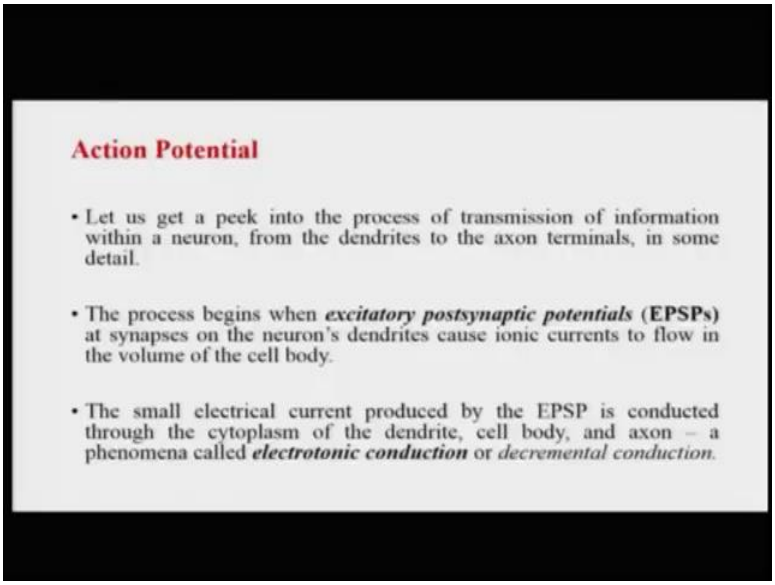

Now, what does this basically mean? is that at a synapse there is a and there is a presynaptic neuron and a postsynaptic neuron, what actually happens is, that based on the signals received, or electrical or chemical communication receive from the presynaptic neuron, the postsynaptic neuron basically experiences low ionic current that flows throughout its body.

Now, this small electrical current that is produced by this that is referred to as the excitatory postsynaptic potential is basically conducted throughout the cytoplasm of the (den) of the cell from the dendrite to the cell body and the axon. This phenomena is referred to as electro-tonic conduction or decremental conduction, because once the current is received at the dendrites of the cell neuron it sort of gradually reduces by the time it reaches the axon terminal of this particular neuron.

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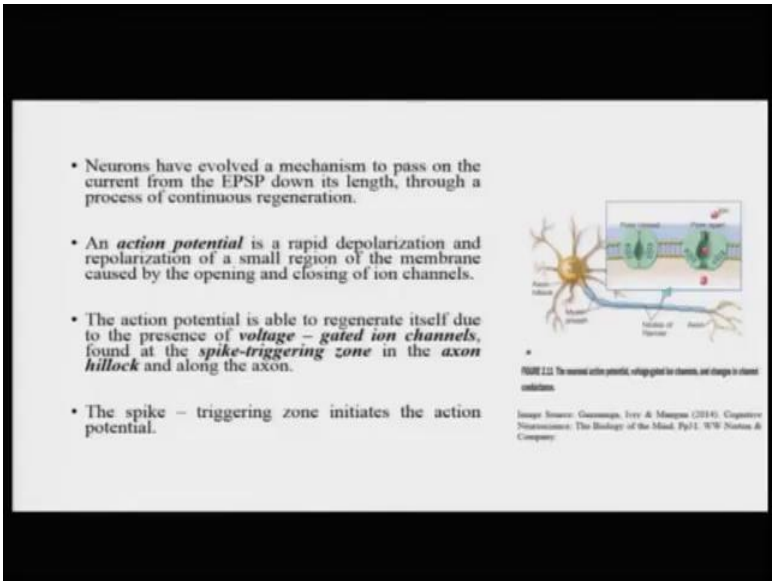

Now, neurons basically in order to propagate this current further, in order to propagate this potential further to other neurons have evolved a mechanism that allows them to pass on this current from the EPSP down to its length and through a process of continuous regeneration. So, as I just mentioned, that because of the process of electro-tonic conduction, this current is kind of is a decremental in nature it will keep reducing over the overtime and over the length of the neuron.

So, in order to be able to successfully transmit this potential to other neurons, what the neuron what this particular neuron or any neuron for that matter will do is it will follow a particular process of regeneration of this current again and again so that this current does not decrease in its potential rather it in some sense could be amplified even.

So, this action potential, the process that I am talking about is basically two part process it is basically rapid polarization, depolarization and repolarization of a small region of the membrane. If you remember, you can see here on the right, the axon is covered with this covering called the myelin sheath and there are gaps in this covering which are referred to as the nodes of Ranvier. Now basically, this is part of the neuronal membrane that sort of would get depolarize and repolarize in the process of generation of this action potential.

Now, this action potential is basically able to regenerate itself due to the presence of these voltage gated ion channels, which we have talked about, basically, which are found towards the spike triggering zone in the axon hillock of the axon hillock along the axon. So, if you can see in the cell body, there is a there is a part where all of these axons are sort of converging

and that is referred to the axon hillock, this creates the spike triggering zone that initiates the action potential in this particular neuron.

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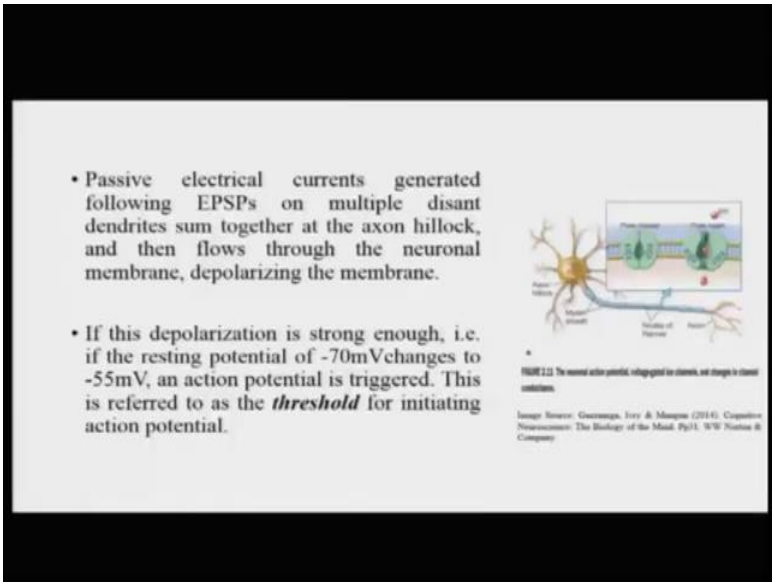

Now, passive electrical currents that are generated following the excitatory postsynaptic potential that is received on multiple distant dendrite basically some together at this axon hillock and then this start flowing towards the neuronal membrane in the process depolarizing the membrane itself.

If this depolarization is strong enough that is, if the resting potential of minus 70 millivolts, as we saw in the last lecture has been changed to around minus 55 millivolt made a little bit more positive, this is where the action potential is supposed to be triggered. And this minus 55 millivolts is supposed to be the threshold voltage for initiating this action potentials. So, this is something that you need to remember.

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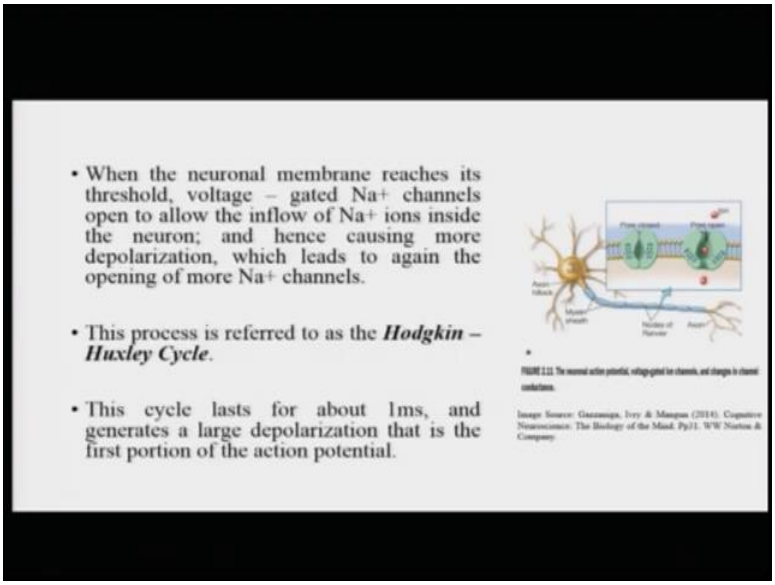

Now, in the neuronal membrane reaches this threshold of minus 55 millivolts, voltage gated sodium channels allow the inflow of the sodium ions in towards the inside of the neuron and hence they causing it to be more depolarized, which then again leads to the opening of more sodium channels which will sort of create a bit of a cycle that the neuronal membrane will keep getting depolarized and it will keep opening sodium channels to bring in more sodium ions.

Now, this process is not a very long process a very short process lasts around not more than 1 millisecond is referred to as the Hodgkin Huxley cycle of regeneration of the action potential. This cycle generates a large depolarization that is the first part or the first portion of what we are referring to as the action potential.

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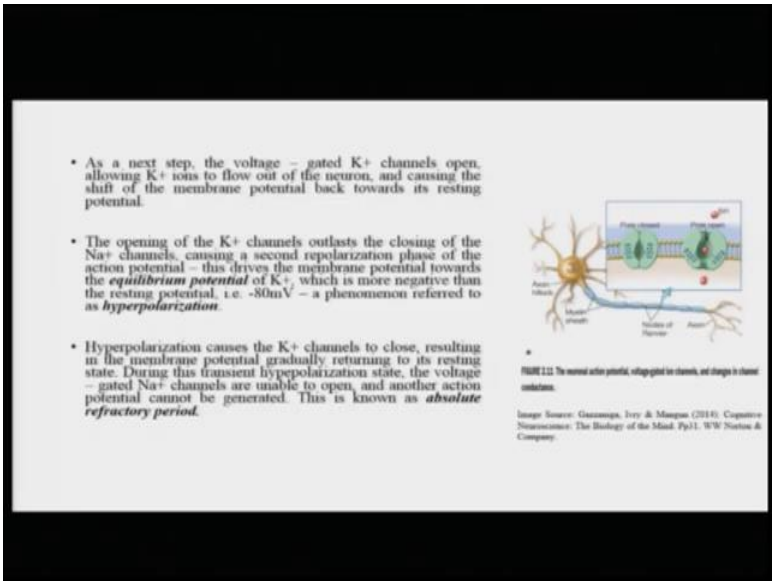

Now, as a next step, once this depolarization has happened, the voltage gated potassium channels will open, allowing the potassium ions to flow out of the neuron from the inside to the outside, causing the shift of the membrane potential back towards its resting potential from minus 55 millivolts to around 70 millivolts.

Now, in this process what happens is that this opening of the potassium channels outlast the closing of the sodium channel, so it kind of goes on for a little bit longer time and that is why what it does is, it drives the membrane potential towards an equilibrium potential of potassium ions.

So, that the inside is sort of more negative than the outside and this resting potential reaches a voltage of about minus 18 millivolts, this process is referred to as the hyperpolarization. When the neuron will reach this process, it will not be able to generate any more action potential and this is basically therefore referred to as a absolute refractory period.

So, hyperpolarization causes these potassium channels to close, resulting in the membrane potential gradually returning to its resting state. Now, during this transient hyperpolarization state the voltage gated sodium channels are not able to open therefore, no further depolarization can happen and another action potential cannot be generated, I am just sort of repeating myself a bit, this is known as the absolute refractory period.

So, some of these things I understand will be slightly more difficult to visualize. So, I basically thinking that, along with when was these slides are uploaded when we are uploading the references, etc. what we will do is? We will add some links to videos where

these processes have been visualized very nicely for you and those are free videos available on the YouTube which you can go and see, but we will just provide the links of them together so that you can understand by seeing the process yourself when it is better visualized in an animated format.

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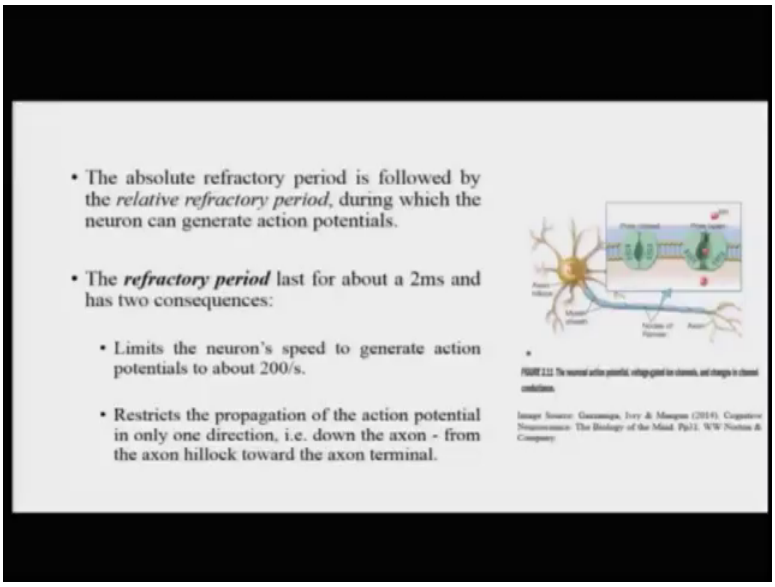

Now, this absolute refractory period is followed by the relative refractory period, during which the neuron cannot generate action potentials to some extent. This refractory period both of them can last for up to about 2 milliseconds and have two important consequences. What are the consequences? This limits the neurons speed to generate action potentials to about not more than 200 action potentials per second one.

Second is that it restricts the propagation of the action potential in only one direction. So, action potential can only happen from the axon hillock downwards towards the axon terminals that is one of the consequences of the happening of this refractory period or let us say the happening of this hyperpolarization which follows the depolarization of the cell.

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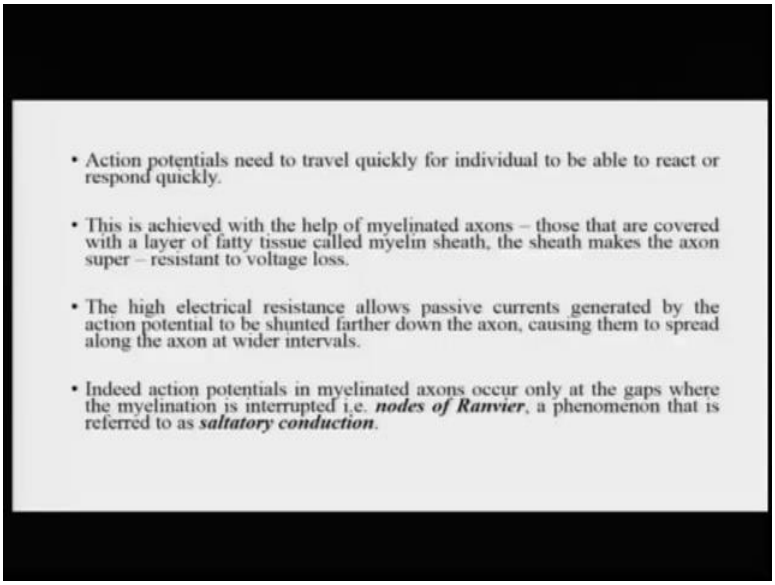

Now, these action potentials need to travel quickly for an individual to be able to react or respond quickly. Say for example, if there are some you know some dangerous stimuli to be received, suppose you are touching something a very hot substance or a prickly substance, you see that the reaction is very quick, you will immediately take your hand off.

So, and this is this applies not only to humans, but all other mammalian all other mammalian species which have this kind of system. So, the action potentials need to travel rather quickly for an individual to be able to react or respond in situations that need quick response. Now, this fast speed of the action potential is basically achieved, because the axon is actually myelinated.

Because of myelinated axons, these axons, as you might have seen in this particular figure, are covered with this bi-layer of lipid tissue or a fatty tissue, which is referred to as the myelin sheath. And what this sheets does? Is that it makes the axon super resistant to Voltage laws.

So, one of the very important functions of this myelin sheet is that it makes the neuron resistant to voltage loss. So, what happens is that the current when it is flowing downwards from the axon hillock to the axon terminal, there is not a lot of current that is lost during this particular process. Now, also this high electrical resistance basically allows passive current generated by the action potential to be shunted further down the axon causing them to spread along the axon at wider intervals.

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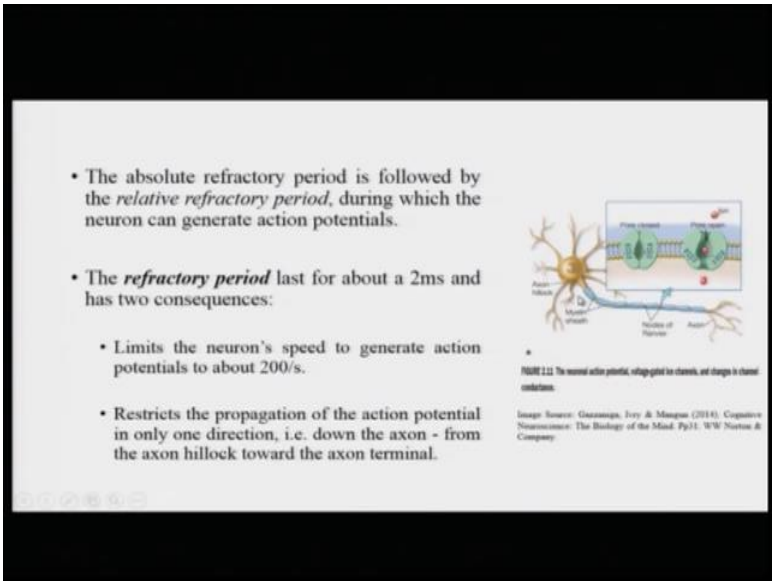

So, this high electrical resistant that this myelin sheath provides, basically what it does, is that once the current is sort of generated at a point, let us say here, it is it is generated for a larger distance when it reaches this kind of point and then this another break and then this another break. So, basically what it does, is that the potential is regenerated at only at these gaps in the myelin sheath, which are actually referred to as the nodes of Ranvier as I was saying.

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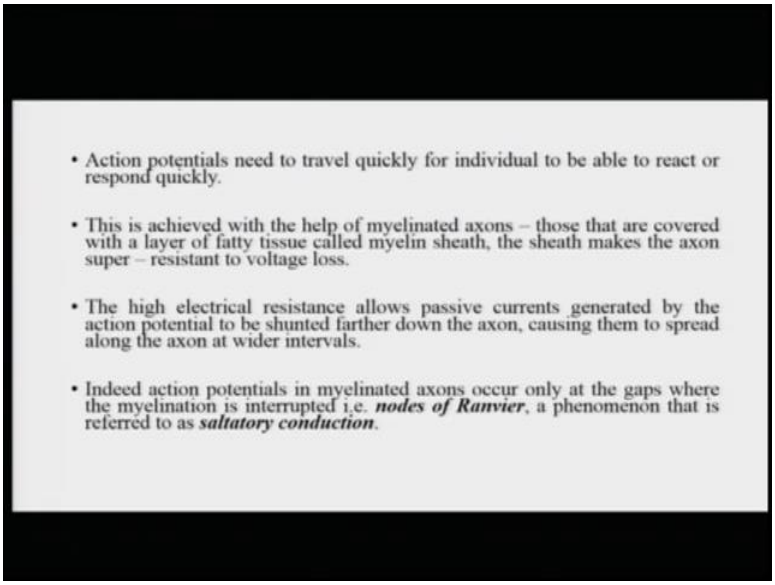

So, indeed action potentials in myelinated axons occur only at these gaps where the myelination is interrupted, which is basically call the nodes of Ranvier. And this phenomena of regeneration of action potentials along the length of the axon at these gaps or nodes of Ranvier is referred to as saltatory conduction. I hope I am making myself clear.

Now, a little bit more about action potentials is that these action potentials are more like an all or none kind of phenomena, because one action potential it is initiated it sort of has the same amplitude at any other action potential, so it basically is not affected by the strength of simulations as the strength of the stimulation that is received.

Say for example, if you are, you know, hearing small sound verses a very loud sound or if you are touching you know surface which has you know 100 degrees Celsius of temperature versus 200 degrees Celsius of temperature, in that sense maybe the number or the firing rates of neurons will vary, but not the strength of the action potential in each particular neuron.

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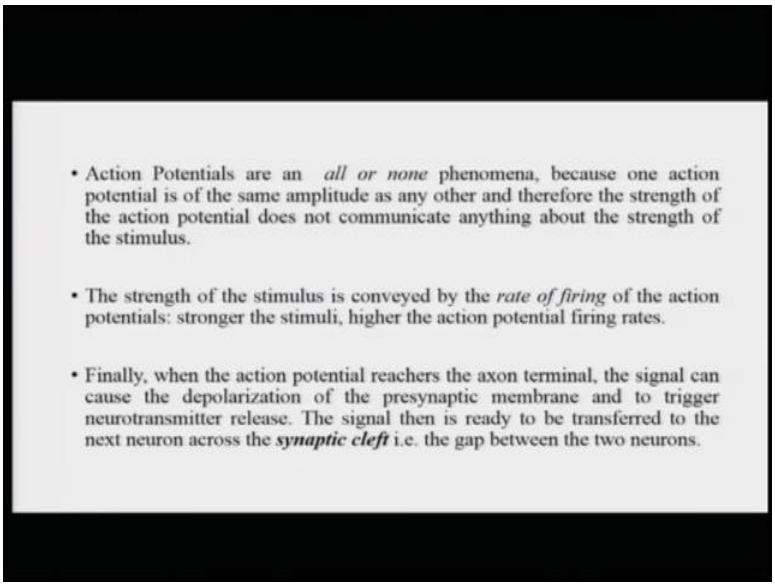

So, the strength of the stimulus is not really conveyed by the amplitude or the strength of the action potential, but rather by the higher action potential firing rates that sort of kind of you know are generated at that point. Now, finally something also important to note is that the action potential once the action potential reaches the axon terminal, the signal will cause further depolarization of this presynaptic membrane near the axon terminals.

And basically, it will lead to triggering off the release of neurotransmitters, this is one of the very important parts in this neuronal communication. The signal in that sense chemical or electrical is now ready to be transferred to the next neuron across the synaptic cleft. If you remember synaptic cleft is the gap between two neurons who are in contact with each other, very closely aligned with each other, for communication to actually happen.

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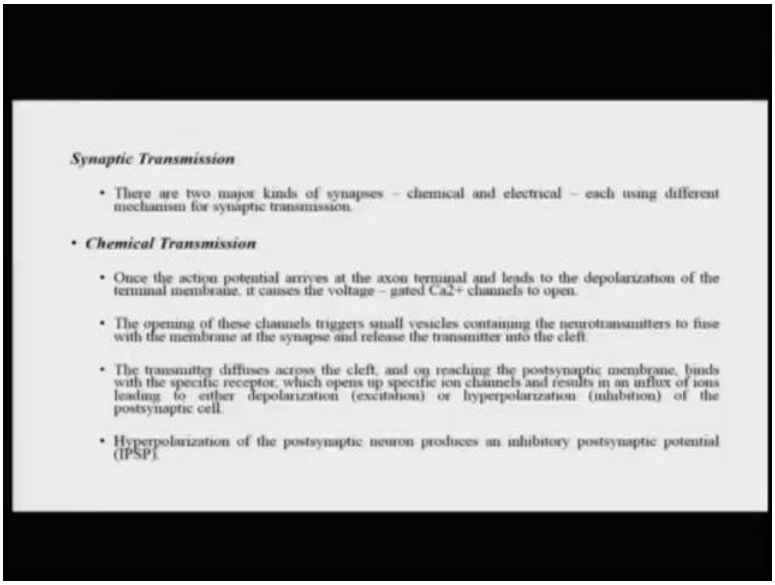

So, now we are say for example, at a point where we can now talk about this communication between neurons and this communication can be referred to as synaptic transmission. And synaptic transmissions, majorly are of two kinds, there are chemical synapses and there are electrical synapses, I will first talk to us talk to you about the chemical synapses.

Now, once the action potential has arrived at the axon terminal, it leads to the depolarization of the terminal membrane basically, of the axon terminals and what it does is, it causes the voltage gated calcium channels that are available at those at those areas to open up.

Now, what happens is once these calcium channels open up, they trigger these small vessels which contain neurotransmitters to fuse with the membrane at the presynaptic neuron, and then they it causes the vesicles to release the neurotransmitters into this synaptic cleft. Once these neurotransmitters are released in the synaptic cleft, they are basically they reached the postsynaptic membrane and they bind with the specific neurotransmitter receptors, which are available on the postsynaptic neurons, or the membrane of the postsynaptic neuron.

What, there are two things that can happen, is that on reaching the postsynaptic neuron they these neurotransmitters can cause either depolarization, which means that they will excite the next neuron and will cause the action potential to propagate further, or it could cause the electric neuron to hyperpolarize or any bit, which means that the action potential will stop there and it will not lead to further communication of this signal.

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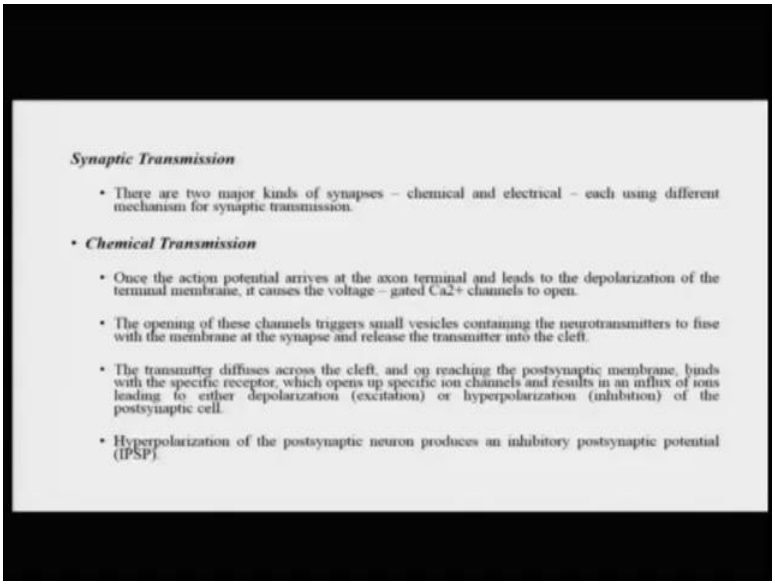

So, hyperpolarization of this postsynaptic neuron is said to produce something, which is called an inhibitory postsynaptic potential. As we were talking about the excitatory postsynaptic potential which causes the generation of the action potential and so on, we can also talk about the inhibitory postsynaptic potential, which basically causes the cessation of this communication.

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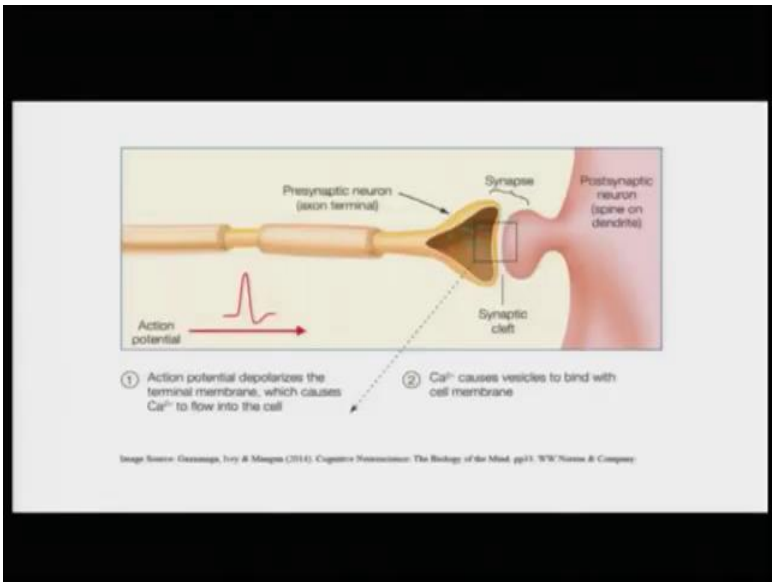

Here we can see, through figures that, once the action potential is generated, the neurons are also closer to each other. There you can see the small vesical which are containing the neurotransmitters, you can see that that is the zone of synapse that gap between the two neurons are that can be referred to as the synaptic cleft.

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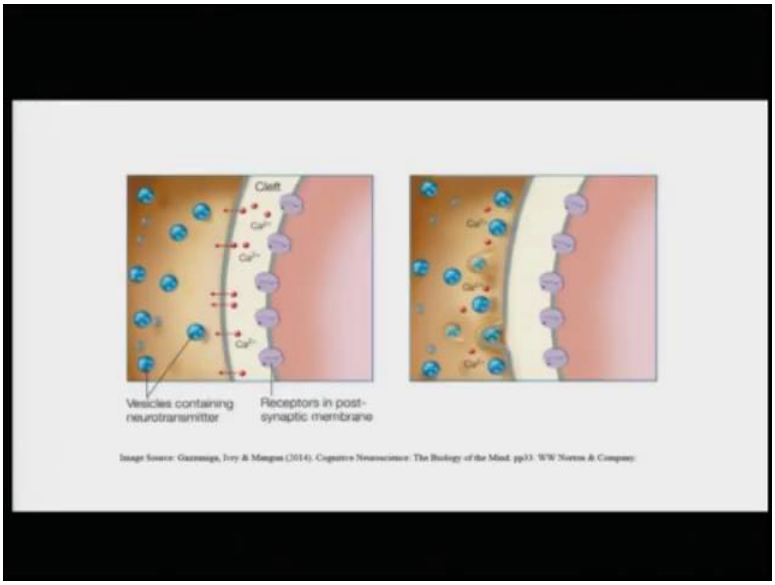

Here you can see that the vesicles basically are causing the calcium channels have opened and the vesicles are moving towards the presynaptic neurons membrane, you can see in the next figure on the right that they then fuse with this membrane.

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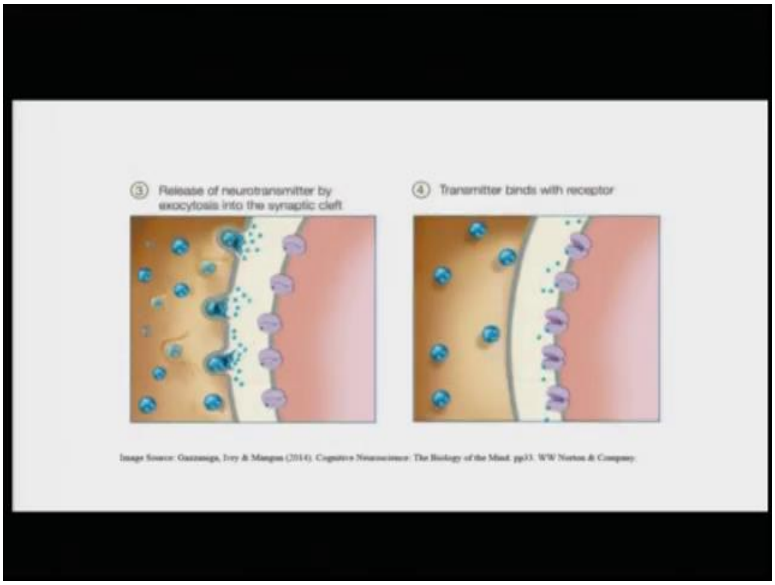

And eventually what they do, is that they will open up releasing the neurotransmitters in the synaptic cleft. These neurotransmitters, we can see on the figure on the right can then bind with the receptor, is received with the receptor.

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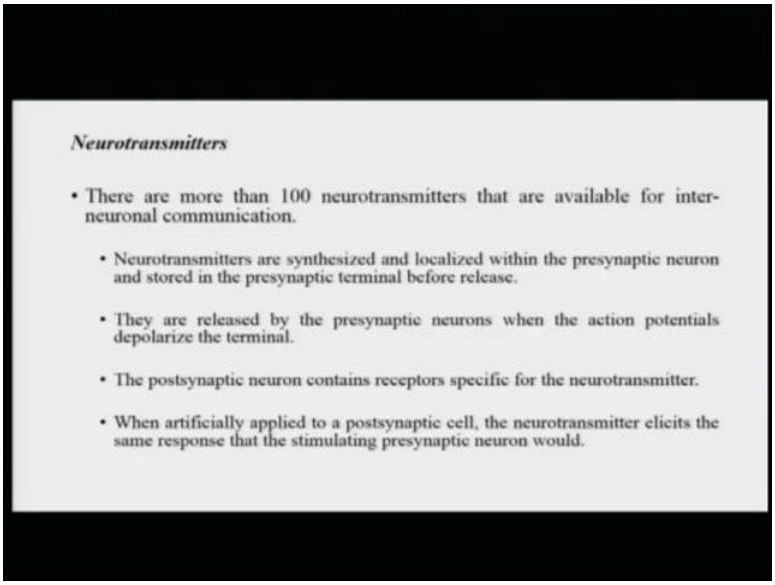

So, we have been talking about neurotransmitters, and neurotransmitters are a very, very important aspect of the chemical synapse that we have been talking about. So, let us look a little bit about what these neurotransmitters are and what do they do. Now, there are more than 100 neurotransmitters known that are available for inter neuronal communication.

These neurotransmitters are synthesized and localized mainly within the presynaptic neuron and they are stored in the presynaptic terminal before their release. So, these neurotransmitters are basically in some sense in some way signals stored in the axon terminals of this presynaptic neuron and can be released on receiving of this excitatory postsynaptic potential or action potential generation, as we have been talking about.

Now, these neurotransmitters are released by this presynaptic neurons when the action potentials depolarized this presynaptic membrane. The postsynaptic neuron typically contains receptors which are specific for each kind of neurotransmitter. So, it is not like that any receptor on the postsynaptic neuron can receive any neurotransmitter being released by the presynaptic neuron, there are specific kinds of receptors for specific kinds of these neurotransmitters.

Now, this is interesting, in the sense that when they were artificially applied to the postsynaptic cell what these neurotransmitters can do, is they can elicits the same response that the stimulating presynaptic neuron would have. Now, this is something which is very much utilized in the field of drugs, in the field of pharmaceuticals, is that some of the drugs you will be aware of and you can kind of go on and search on Google and find out, is that

some of these drugs that people take recreational drugs and also pharmaceuticals, sometimes what they do, what they would do, is that they would mimic the role of a particular neurotransmitter.

Say for example, taking particular kinds of drugs like let us say LSD or Marianna, might basically release in the bloodstream chemical substances, which would mimic the action of dopamine, which is a neurotransmitter and basically what these chemical substance do, is that they would go on and they would bind themselves with the dopamine receptor sites on these specific postsynaptic neurons and what it will I do for you, is that it will give you the same feeling of pleasure as if, you know, dopamine is released by your nervous system.

But in the long run what would happen, is that because these are, these are overstimulation and they are artificial stimulation, sometimes what could happen is, if you are taking the drug again and again for getting the same kind of pleasure or getting the same kind of high, it would basically lead to a degree of tolerance being built up because the neuro because the receptor sites would get damaged because of the overstimulation.

And what it might lead to? Is, it might lead to development of tolerance and even say, for example, what will happen is that higher dosage than usual of the drug will not lead you to the same kind of high. So, that kind of creates a bit of a thing and has been sort of studied well in processes of addiction and drug use and so on, so it is that just a bit of a trivia that I wanted to share with you.

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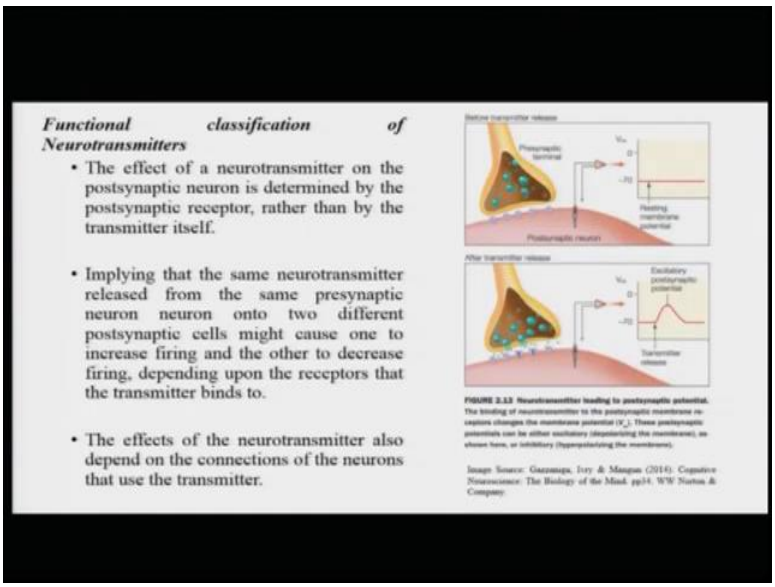

Now, these neurotransmitters can be functionally classified into a couple of types. One of the things is that the neurotransmitters, one of the effects that the neurotransmitters can have on the postsynaptic neuron, is basically decided by the postsynaptic receptor rather than by the neurotransmitter itself. So, this is something very important that you should remember, is that the receptor is basically something that governs what is going to happen further not the neurotransmitter, so that is interesting.

Again another thing is that, the same neurotransmitter release from the same presynaptic neuron onto two different kind of postsynaptic cells might cause one to increase the firing and other to decrease the firing. So, the effects of the neurotransmitters on two different cells of the same neurotransmitters can actually be slightly different. So, this is also something that one should note of note.

Now, the effect of the neurotransmitter also depend on the effects of the neurotransmitter also depend on the kind of connections basically, of the neurons that use this particular neurotransmitter. So, it is not really a very determined, it is not a very deterministic definitive of setup, it basically depends on a few factors on the receptor on the postsynaptic neurons on the kind of connection that these two neurons have and so on. So, this is something which is very, very important and one should be remembering this, about these neurotransmitters.

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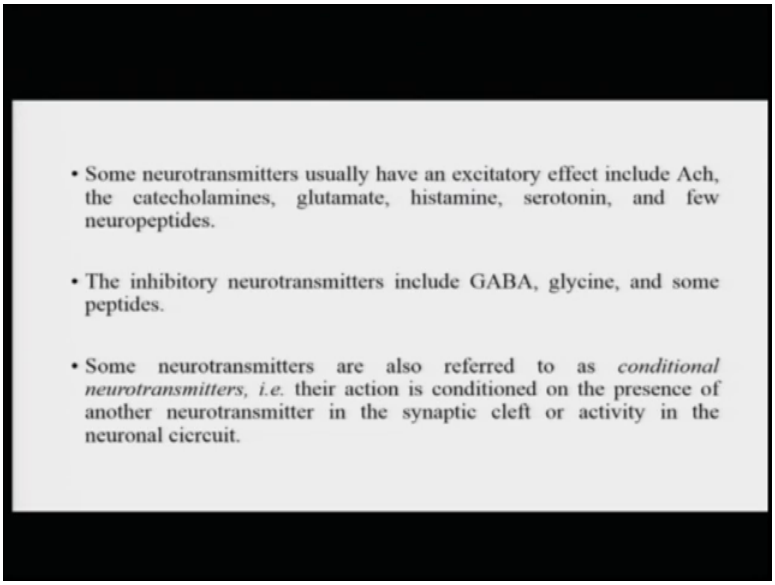

Now also, one would like to sort of remind, that some neurotransmitters actually will have an excitatory effect that will cause the postsynaptic neuron to generate the action potential, which includes acetylcholine, catecholamine and glutamate, histamine, serotonin and few

other kind of neuro peptides. Some of the neurotransmitters will cause inhibitory outcomes, which will include GABA, glycine, and some other kinds of peptides and these are just a names that you would want to sort of remember they will come again when we are talking about specific functions.

Now, some neurotransmitters basically fall somewhere in the middle and they can be referred to as conditional neurotransmitters basically saying that their action is conditioned on the presence of another neurotransmitter. So, say for example, if neurotransmitter A is released, the outcome of the release of neurotransmitters A would depend on the presence of some other neurotransmitter like B or some other, you know, factor in the neuronal circuit.

So, basically this will this should fire but will fire only if neurotransmitter B is also present, that kind of thing. So, these kind of neurotransmitters are referred to as conditional neurotransmitters. So, I hope this section on neurotransmitter clear for you. Now, this was more about chemical transmission. We can also talk a little bit about electrical transmission or electrical synapses.

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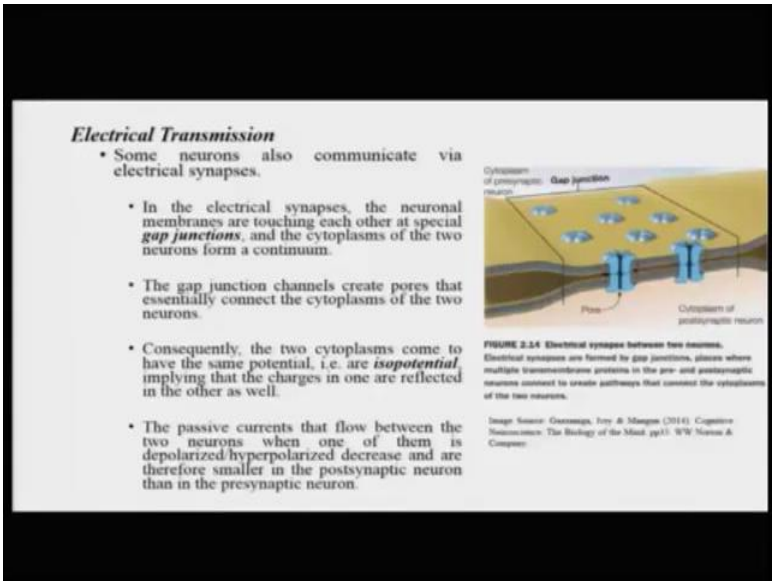

Now, electrical synapses is basically works slightly differently wherein the two neurons actually come in contact with each other, they touch each other via what are called the gap junctions you can see on the in the finger on the right and these gap junctions are specialized areas, specialized structures through which basically what happens, is that the cytoplasm of the two neurons sort of become joined with each other, sort of become connected with each other and almost form a kind of a continuous, you know system.

Now, these gap junction channels basically what they do? Is that they create these pores that essentially will connect the cytoplasm of the two neurons and consequently what will happen, is that the both the cytoplasm of both the neurons will become or will come at the same potential. So, they can be called to have become like isopotential, which is basically which basically means that the potential in one neuron is reflected in the other neuron as also if this one gets polarized, this one will also automatically get depolarized and so.

Now, these passive currents that flow between two neurons, suppose one of them has received the EPSP or the excited reports an epic new potential, what happens is that the strength of that passive current would decrease further because now, this is spread out not only in the cytoplasm of one neuron but on to the cytoplasm of two neurons.

So, the passive current that basically flows between the two neurons when one of them is depolarize or hyperpolarize for that matter decrease and are therefore, much smaller in the postsynaptic neuron than in the presynaptic neuron. So, this can happen when couple of neurons are in the state of electrical transmission.

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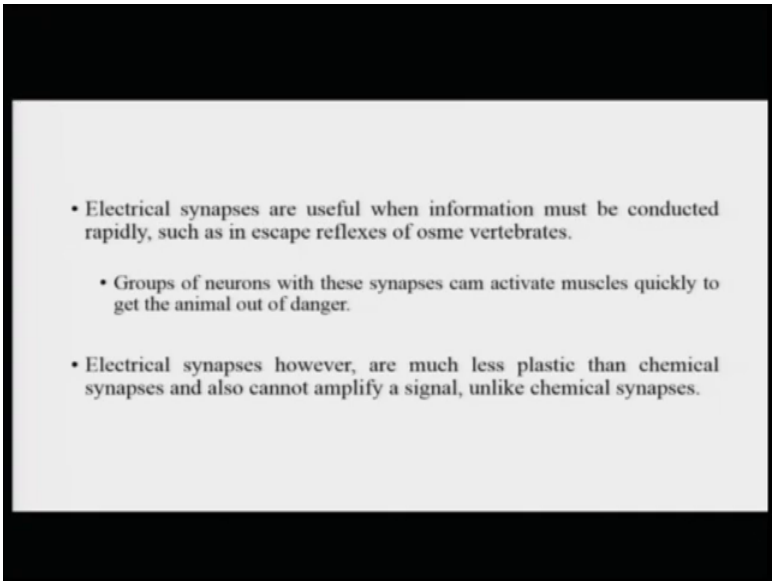

Now, electrical synapses are useful when information must be conducted rapidly, such as in escape reflexes of some kind of vertebrates, so it kind of is slightly faster than the chemical synapses. And what happens is that groups of neurons with these kind of synapses basically they are able to activate muscles quickly and they are able they enable the animal to get out of danger rather rapidly.

Now, electrical synapses however, also have a couple of drawbacks. Electrical synapses are couple of them say for example, these electrical synapses are much less plastic, they are in in a slightly more rigid structure, so they cannot really you know, change other flexibly, then chemical synapses and also they are not able to amplify their signal because of the process of regeneration that we are seeing throughout chemical transmission process.

Electrical synapses do not really cause a lot of, you know, amplification of the received excitatory postsynaptic potential. So, I think this is all about chemical and electrical transmission during synapse. We can move to a different topic. We have so far talked mainly about neurons and neuronal cells in the brain, but there is almost equivalent amount of Glial cells in the brain, which account for almost more than half of the brains volume from the entire nervous system.

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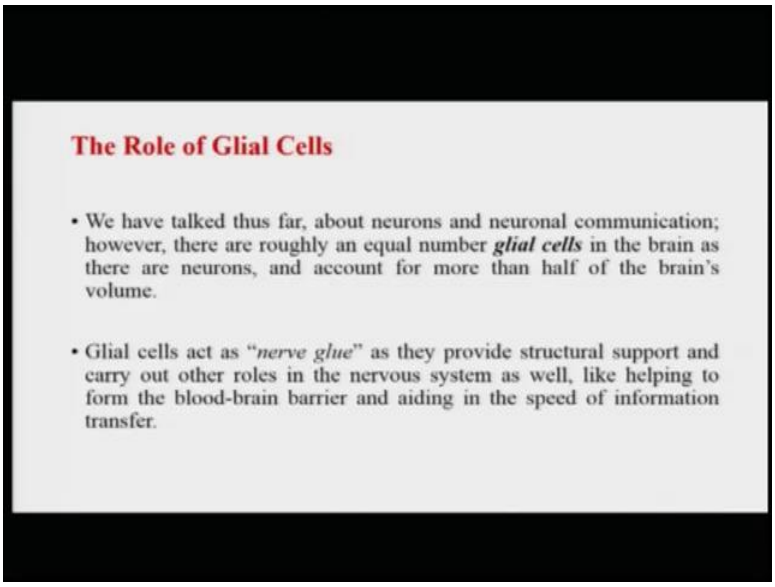

Now, Glial cells they are also referred to as nerve glue originated from the term glia, which is basically means nerve glue in Greek. So, glial cells have important function, say for example, they provide structural support and they also carry out other kinds of functions in the nervous system.

Say, for example, they form this blood brain barrier, which sort of protects the brain from a lot of these pathogens in the bloodstream, we will talk about this in bit of detail in slightly later. And what it also does is it sort of aids in the speed of information transfer between neurons. So, these are two very important functions of these glial cells.

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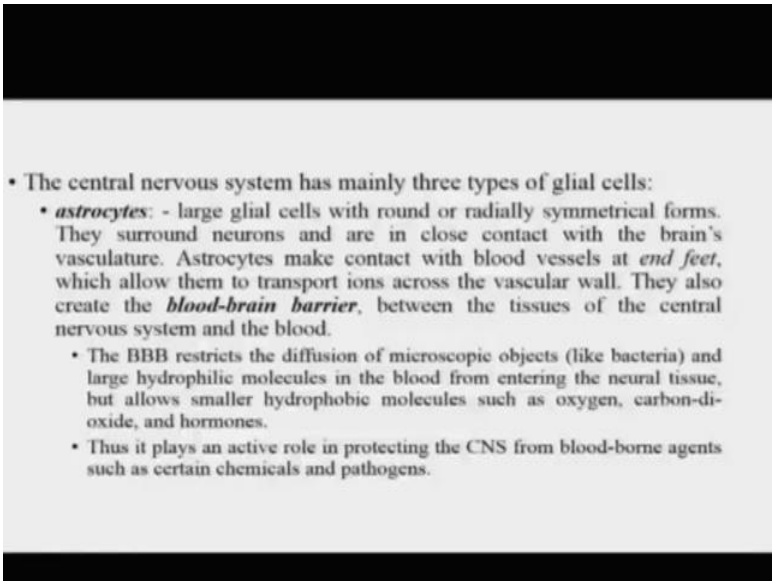

And there are mainly three types of Glial cells we we can talk about. First is these astrocytes, these are large Glial cells with the round or readily symmetrical form. They typically what they do is they surround the neurons, and basically and are in close contact with the brains vasculature.

Now, what these astrocytes do is that they can make contact with the blood vessels at the end feet at these endings, special structures, which basically do a very important function allow them to transport ions across these across the vascular wall. And on top of that, these astrocytes also form or they create what is called the blood brain barrier.

Now, this blood brain barrier basically, is sort of a barrier which exists between the tissues of this nervous central nervous system and the blood. So, it basically blood that is flowing in the body is slightly, you know it is it is restricted from flowing into and through the brain, for example, or through the spinal cord for example.

Now, this blood brain barrier that we are talking about has very important function. One is that it restricts the diffusion of microscopic objects like bacteria and large hydrophilic molecules, like you know, in the blood from entering the neural tissue from entering the brain area, but it also allows small hydrophobic molecules such as oxygen, carbon dioxide, and other kinds of hormones from entering.

It is kind of acts as a selective barrier, allowing the good things to enter which are needed, and basically keeping out the things that can be potentially harmful. So, it basically plays a

very active role in protecting the central nervous system from these blood-borne agents such as chemicals and other kinds of pathogens.

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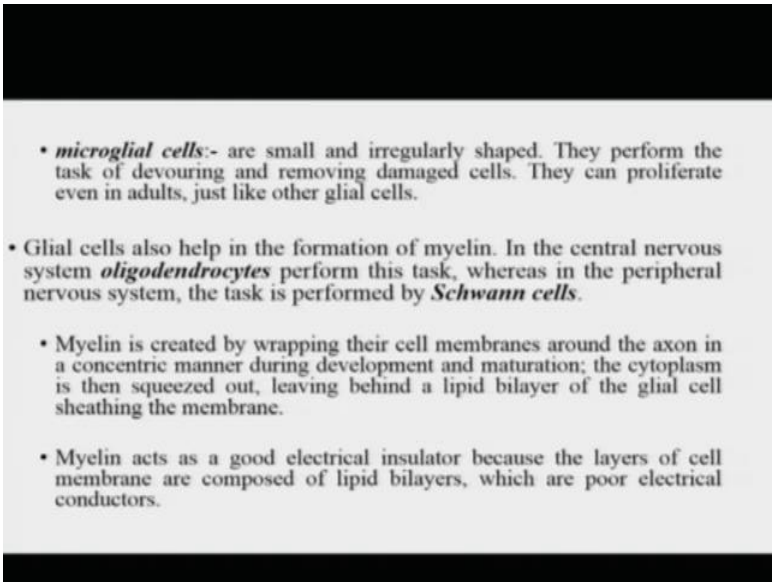

Other kinds of Glial cells are these micro-glial cells, these are small and they are irregularly shaped, and they perform the task of scavenging the act like phagocytes, and they basically perform this task of devouring and removing damaged cells from the nervous system. So, if any cells get damaged the microbial cells basically perform this task of scavenging and cleaning up the damaged tissue.

Now, a very important function of the Glial cells that we are talking about is in is their help in the formation of myelin. We have seen that myelin is very, very important because it aids neuronal communication by making it faster and resistant to voltage loss. So, how these glial cells basically help in the creation of myelin? Is that there are two kind of Glial cells oligodendrocytes, which perform this task in the central nervous system, the brain and the spinal cord. And then there are the Schwann cells, which perform this task in the peripheral nervous system.

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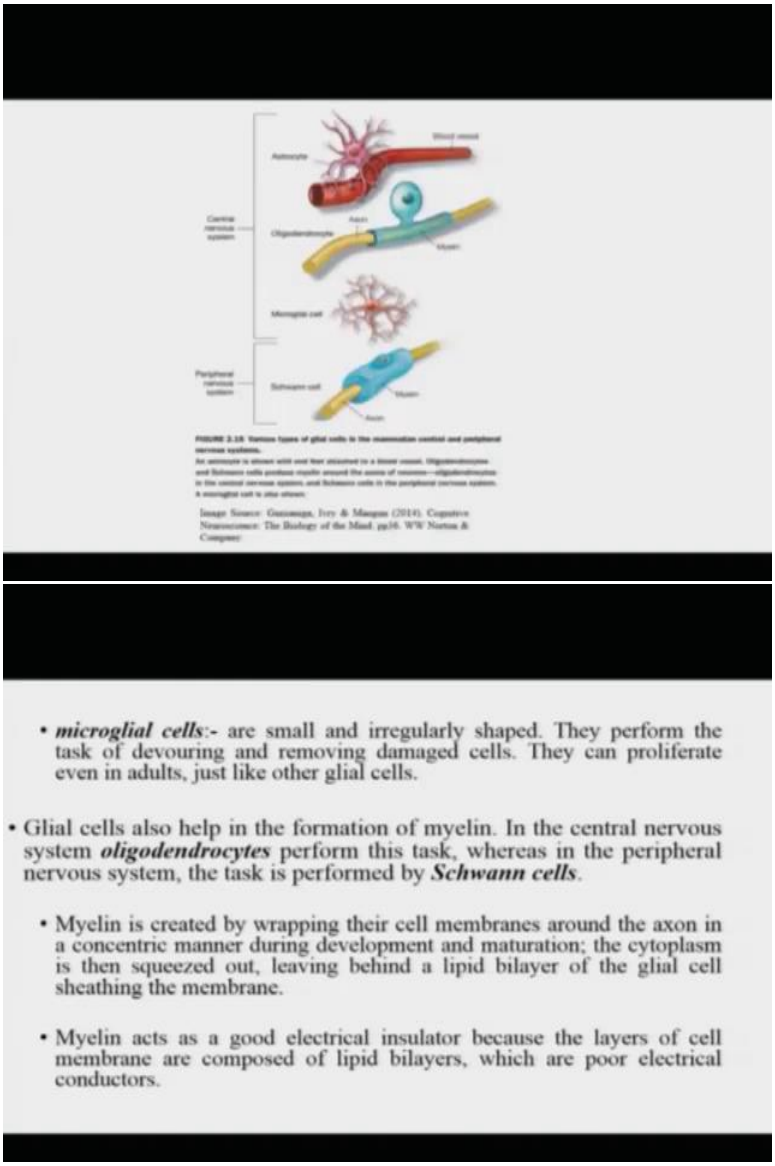

We will see, we will have a quick look at these kind of cells, you can see the astrocyte, astrocyte here, and you can see the myelin sheath basically being formed, this is the micro-glial cell and you can see that basically what happens? Is that these astrocytes basically or these sorry oligodendrocyte what it does?

It wraps itself around the axon and it creates this protective covering. Here you can see the Schwann cell doing the same for the peripheral nervous system, which is just forming this layer, protective layer of fatty tissue on the membrane of the axon.

What it does is? It sort of wraps itself around the axon, and then it gradually squeezes out the entire cytoplasm of the cell. So, that what just remaining is just this bi-layer of fatty tissue, which being very good, you know, very poor conductor of electricity, acts as a very nice

insulator and reduces voltage loss from the entire length of the axon. So, these are these are fairly very, very important.

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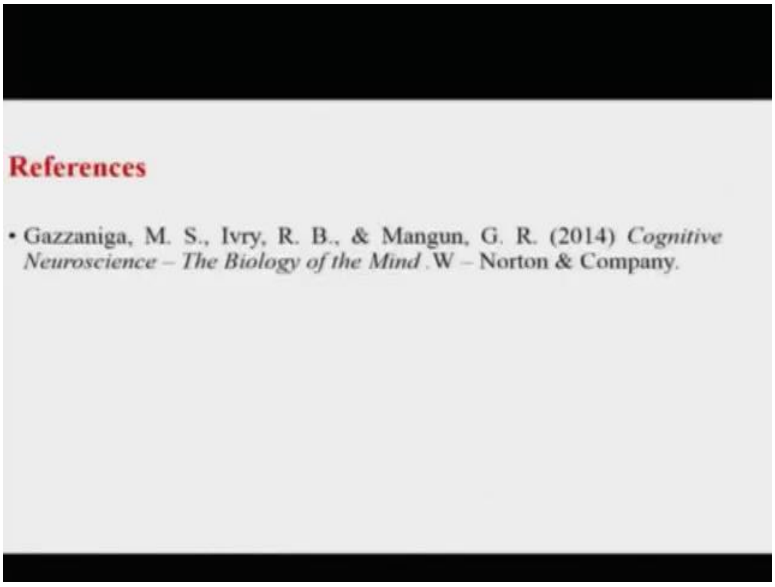

So this is all that I wanted to say in this lecture. We have talked a little bit about, how the action potential is generated. We have talked a little bit about how the action potential is communicated to from the presynaptic neuron to presynaptic neuron by a couple of processes, chemical synapses and electrical synapses. We also talked a little bit about how Glial cells are important to this whole system of the nervous system. Thank you.