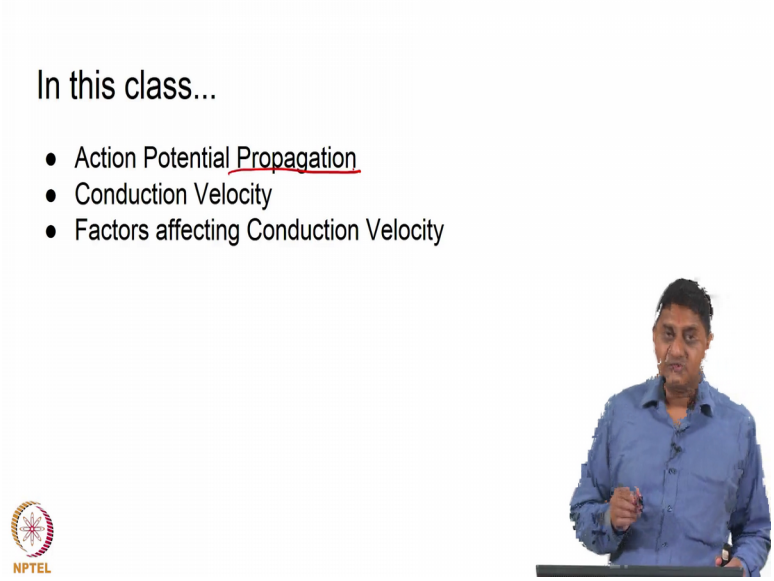
Neuroscience of Human Movement
Department of Multidisciplinary
Indian Institute of Technology, Madras

Lecture – 09
Action Potential - Part 4

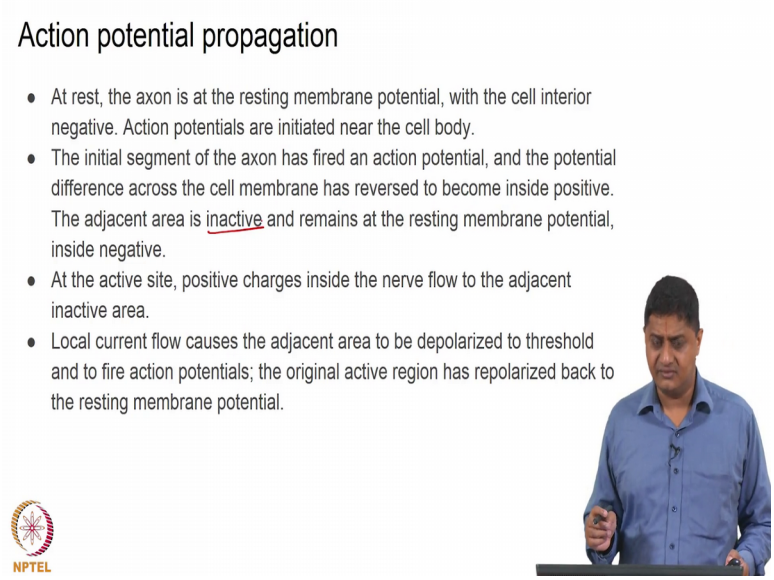
Welcome to this class on Neuroscience of Human Movement. In this class, we will be talking about Action Potential, so this is part 4 of our discussion on action potentials. So, in this class we will be talking about an important property of action potential which is propagation.

(Refer Slide Time: 00:28)



So, whenever something moves from one point to another, you will call it, you call that, it has been display as it has moved, so this is propagation of information; that is one. So, whenever you say that something is getting displaced, that second question that arises how fast has it been displaced that is related to conduction velocity in this case, and what are the factors that affect conduction velocity. So these are three topics we will discuss in today's class propagation, velocity and factors affecting conduction velocity right.

(Refer Slide Time: 01:05)



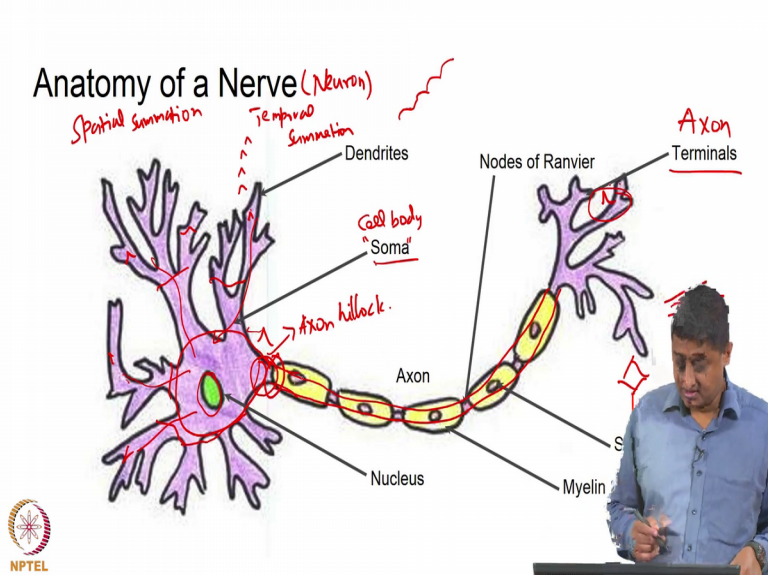
So, at rest the action or the neuronal cell membrane is at resting membrane potential right. So that means, the inside of the cell is more negative when compared with the outside, and at some points either due to a stimulus or due to the opening of voltage gated sodium channels for a brief amount of time, the membrane will become more positive, this situation is called as action potential.

An action potentials are usually initiated near the cell body or soma, will discuss that in the next slide. So, whenever the initial segment of the axon fires an action potential then this potential have the tendency, have the ability to cause action potential in neighbouring regions, because it turns out that the amount of sodium that enters through the voltage gated sodium channel propagates a basically diffuses through the intracellular fluid.

So, causing the nearby areas to become depolarized and this depolarization, when it crosses the threshold will cost the neighbouring voltage gated sodium channels to open. So, this is the mechanism which we will also detail in the next slide, so the neighbouring area becomes positive and then the nearby area, the previous area remains inactive ok. So, at the active site positive charges, basically sodium ions flow to the adjacent inactive area. This local current flow diffusion causes the adjacent side to be depolarized to threshold and fire action potentials in that region. So, at each point on the membrane a new action potential is generated.

The, but the original region will repolarized back to resting membrane potential. How does this happens? Let us discuss this in bit more detail right.

(Refer Slide Time: 03:23)



So, here is neuron and its input and output structures with some detail, right. So, here are dendrites which are major input structures to the neurone and here is the axon. This is the axon which is long cable like output structure of the neuron and this terminates in the axonal terminals or axon terminals, so these are called axon terminals. They communicate with say other neurons or maybe muscle fibres etcetera. Now the neuron has a cell body which is this and a nucleus right.

The neuronal cell body is called as soma ok. The point where the soma attaches to the axon, the neighbourhood is called as axon hillock ok. This is the region that there is a relatively high probability of action potentials getting generated ok. Now this dendrites receive inputs from multiple sources, they may be the same neurones or they may receive input from multiple neurons for the, for all purpose. We will discuss how the input enters and causes an action potential in this neuron without go into details, how the previous neuron causes an action potential in this neuron. For now let us suppose this dendrite is receiving enough information or stimulation that causes a relatively small depolarization current or a relatively small depolarization say for example, this

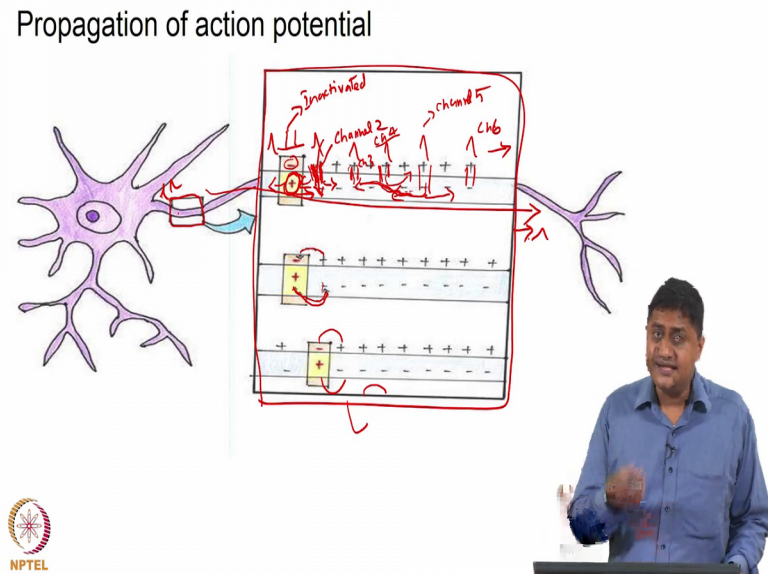
This small depolarization is probably not enough to cause and action potential in this neuron. Suppose it's possible that the same dendrite receives multiple stimulations one

after the other closely followed index. So, before the previous stimulation or before the effect of the previous stimulation dies down, the next stimulation comes in this leads to a situation, where if the response is like this, then the next input will cause a response like that and the next input will cause a response like that and the next input will cause a response like that. This case the output, the response is summed overtime this situation is called as temporal summation ok, will also discuss this in some details in future slides, what is also possible is the dendrites from multiple sources, say from multiple regions can receive inputs at the same time.

So, at a given point in time multiple inputs arrive from various different dendrites that are especially distributed. They all arrive at the cell body at approximately the same time, this will lead to a situation that all these responses from this multiple inputs can sum at a given point in time, this kind of a situation is called as spatial summation ok, without referring to how an action potential is generated, whether by spatial summation or temporal summation. We will look at how action potential gets propagated; that is of interest for us in this class right.

Let us assume that an action potential is generated either by spatial summation or by temporal summation at this point. Now it has to be communicated it has to reach say that point ok, how does it travel from that point to that point, how does it travel that is the question. Now let us take a deeper look.

(Refer Slide Time: 07:30)



Let us assume that an action potential is generated at that point right, so essentially before the action potential came in, before the action potential arrived or before the action potential was generated, this part of the axon was at resting membrane potential. Now what is the resting membrane potential? it is about minus 65 millivolts; say for example. So, or in other words the inside of the axon is negative when compared with the outside. The inside of the axon has more negativity when compare with the outsides, so that is the usual case right.

And making it less negative is what is called as depolarization right. This we discuss in previous class, an action potential causes transient brief depolarization sufficient to take the inside of the membrane more positive right. This is what we saw in the previous class, an action potential essentially makes a very small region of the membrane like that region. So, when we zoom out is that, so that is this region alone zoomed out in that region, a relatively smaller area becomes positive here.

See for example, like this ok, when compared with the outside, the inside is more positive. Let us remember this is a transient event, this is going to go back to inside negative, very soon why? Because of the restorative mechanism that we have discussed earlier and also because the sodium conductance does not remain high for a long period. It remains high for a very brief period and then becomes zero or very very small value.

And then the potassium conductance increases and remains high for a relatively long period. This is what we saw, because of this reason. This is a transient phenomenon for a brief amount of time, the inside of the cell becomes more positive than the outside, but that amount of time is sufficient to let in a lot of sodium inside. This is the behaviour of the voltage gated sodium channel that we saw. A lot of sodium enters inside taking the inside of the cell to a more positive value for a brief amount of time and that is sodium, when it enters actually diffuses in both directions and if the amount of sodium that enters, is sufficient to take this voltage gated sodium channel to threshold, which it does.

Usually, so this voltage gated sodium channel goes to threshold and once it crosses the threshold what happens? Basically the activation gate of this voltage gated sodium channel opens and the inactivation gate is trying to close. In the meanwhile a lot of sodium will enter through this gate not through this gate at that time, at around the same time this gate will be its inactivation gate, will be closed, or it will be inactivated or it

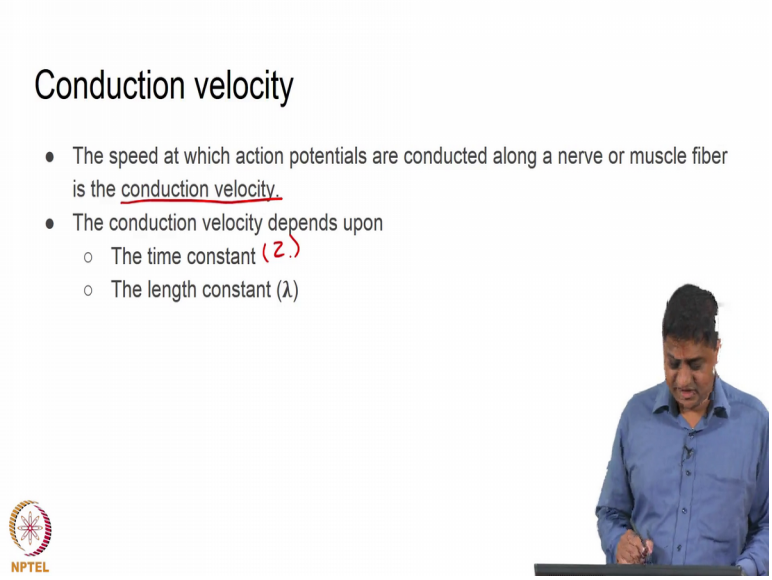
will be in its refractory period which we saw in the previous class. So, a lot of sodium enters inside and in this in this channel a lot of or I am going to called this as channel 2. In channel 2 a lot of sodium enters inside and the sodium diffuses in both direction depolarization from. This voltage gated sodium channel takes the neighbouring voltage gated sodium channel to threshold.

And the depolarization of that takes the neighbouring voltage gated sodium channel to threshold depolarization of that will take the neighbouring voltage gated sodium channel to threshold. So, essentially you will have action potential first here and then another action potential here and then another action potential there, another action potential there, another action potential there, another action potential there.

Note at each point new action potential is generated right, because of the voltage gated sodium channel at that point. Now the question is, why sodium entering from this region is not sufficient to cause an action potential in, say this region. Let us assume the location of these channels are such that are the distribution of these channels along the membrane are such that the sodium entering through channel 5, when by the time diffuses the strength or the amount of sodium that reaches channel 3 and channel 2 are not sufficient to take channel 3 and channel 2 to threshold

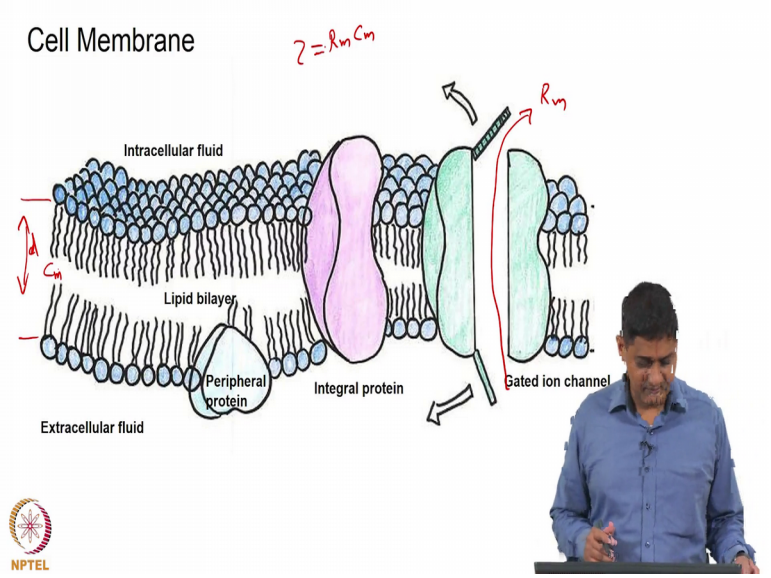
But channel 4 can be taken to threshold, but it is in its refractive period, then the only channel that will get depolarized due to the sodium entering is the channel in that direction, just basically channel 6 that will go to depolarization and so on and so forth, which is why action potential is usually unidirectional. It usually travels from that point to that point in that direction right. So, this is a action potential travels from one point to another. Essentially it is not action potential that is travelling, it is sodium that is travelling from one point to another at each voltage gated sodium channel, you have a new action potential; that is getting generated. Then the next point is about how fast can it can this travel?

(Refer Slide Time: 12:56)



The speed at which action potentials are conducted is called as conduction velocity and there are two factors that govern the conduction velocity; one is the time constant of the, other is a length constant lambda

(Refer Slide Time: 13:12)

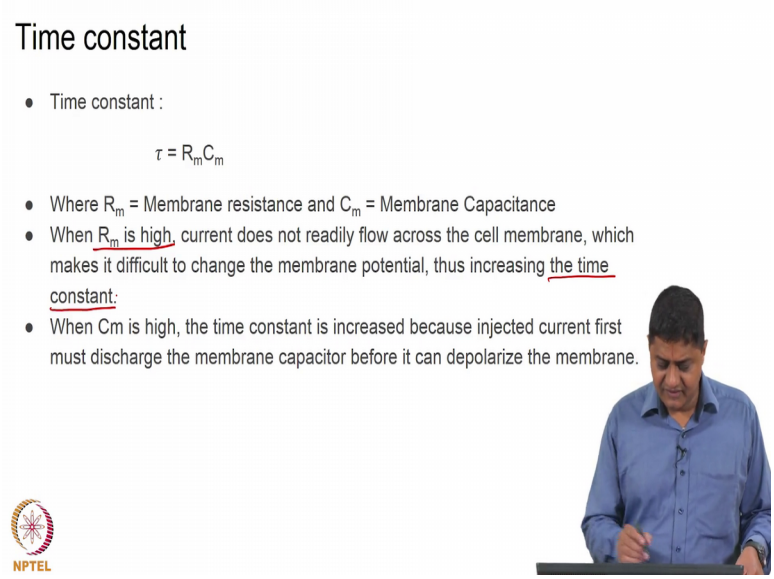


Now, let us remember that the cell membrane can be considered to be a parallel plate capacitor ok, so there is some distance d and this parallel capacitor has say some membrane capacitance cm and also remember the movement of ions through the channels, are also governed by the channels opening or closer. If the channels are closed,

then the membrane has high resistance to the moment of that ion or if the channel is open then the membrane has relatively low resistance. We saw these things earlier, we said that there is quantity conductance which varies as a function of time.

And depending on the conductance value the contribution of each ion to the membrane potential will vary something that we saw earlier, is it not. So; that means, the membrane also has some resistance Rm, so it turns out that the time constant is Rm cm similar to what we would find in basic electrical engineering right. The time constant is Rc here we find that the time constant is basically the product of the membrane resistance and the membrane capacitance ok.

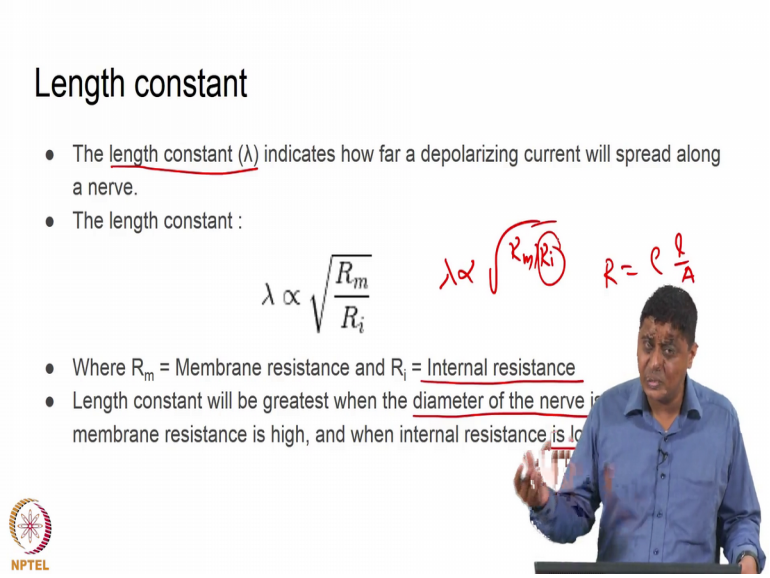
(Refer Slide Time: 14:41)



So, what will take the time constant to a high or low value is something of interest for us right. So when the membrane resistance is high, then basically current does not readily flow through the membrane right which makes it difficult to change the membrane potential, thus increasing the time constant. So when Rms is high basically time constant is high.

When cm is high what happens is that, the injected current must essentially first discharge the membrane capacitor before it can depolarized the membrane, so essentially when cm is high then tau is high, so both cm and Rm when they increased they increase tau due to this physical reasons right, what about the length constant?

(Refer Slide Time: 15:28)



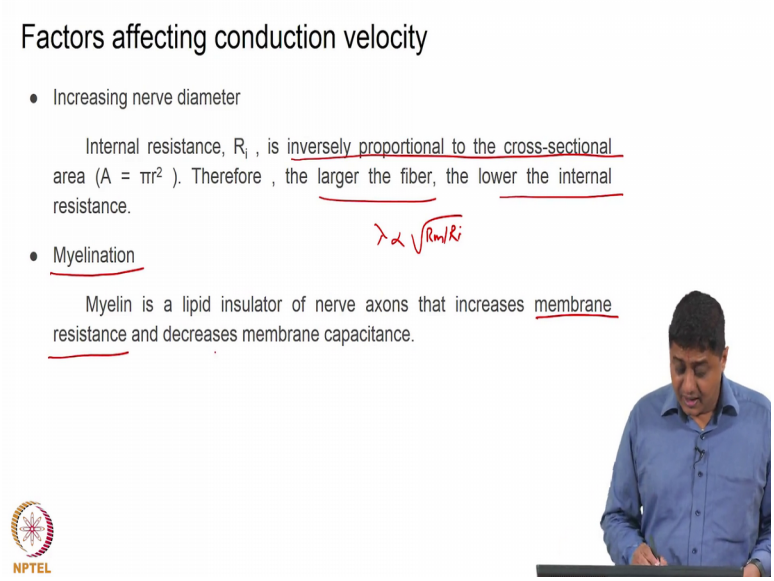
Essentially length constant is proportional, so length constant is proportional to the square root of Rm and Ri to what extent the depolarizing current will go along neuron before it loses the strength is the question right. This will, if I were to design the system, this will give me an idea where do I place the next voltage gated sodium channel, you do not want them to be placed too close or too far away.

If the voltage gated sodium channel is spread, for example, too far away, it is possible that the sodium current will not be a sufficient to cast the action potential in that one so to what extent, can you travel that is the question, that is given by the length constant lambda right and I said lambda is basically square root of Rm Rm divided by Ri ok, so where Ri is the internal resistance and we know something from metallic conductors right, and their resistance is basically R is rho l by A. Basically as the length increases, the resistance increases and as the area increases the resistance decreases. A similar situation is true also in the biological membrane the internal resistance is low when the diameter of the neuron is large right.

So, one way for me to have a relatively large length constant to one way for me to ensure that the depolarizing current will travel the far distance is to ensure that the nerve diameter is relatively large, the larger the diameter the lower is the resistance ri and the larger is the length constant to increase the length constant. I can increase the diameter

and thereby reducing the internal resistance, but I cannot keep on doing this is a, keep on doing this at some point body size related constraint will come into the picture.

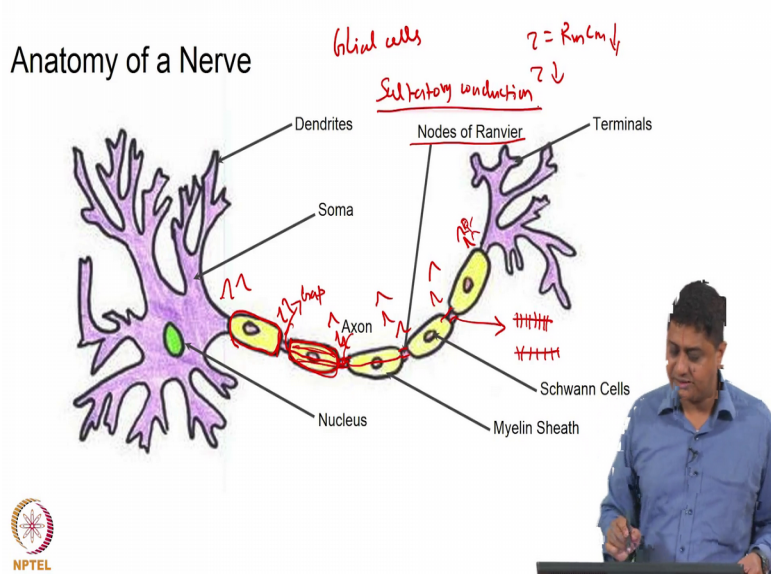
(Refer Slide Time: 17:49)



As I said the internal resistance is inversely proportional to the cross sectional area; therefore, larger the fibre basically the internal resistance will be lower thus increasing lambda.

The other way is to use an efficient method which is myelination. Myelination is basically an insulator of the neuron, are the neuronal axons that increases the membrane resistance. So; that means, what lambda, if it is if it is proportional to Rm by Ri. If I insulate the neuronal axon with insulator is to increase a nerve diameter the other way of increasing lambda is to increase Rm, simply that is possible by using an insulation around the neuronal axons right. So basically myelination increases membrane resistance and thus increases lambda, but what myelination also does is that, it decreases membrane capacitance.

(Refer Slide Time: 19:04)



So, here what you have is myelination. So this is myelin, these are basically neurones are Glial cells. These are Glial cells that insulate other neurones basically there that insulate and this insulation happens in multiple layers right between two patches of this insulation, there is a gap between two of these insulations, there is a gap and that is called as this gap is called as a node of Ranvier. What happens in this node of Ranvier? Suppose I have this node of Ranvier and I am zooming out, this what is actually happening is that, there is a great increase in the density of voltage gated sodium channels and voltage gated potassium channels.

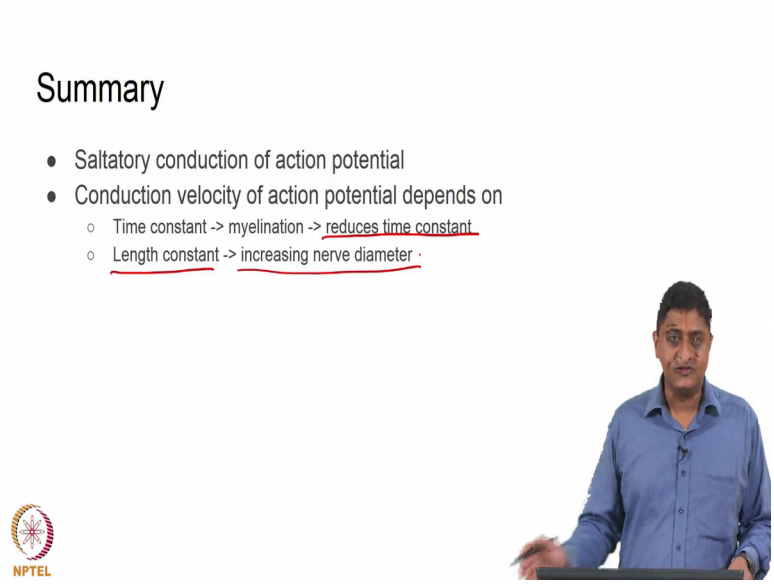
So, the density of voltage gated sodium and potassium channels is very high when compared with the rest of the axon, which is passing inside the insulation. So this leads to a situation where this leads to a situation, where the action potential can be quickly generated here, why because membrane capacitance is reduced essentially.

Let us remember tau is Rm Cm when cm is reduced greatly right, this will lead to a situation where tau is reduced to a large extent. This means by myelination, I can increase the length constant and I can decrease the time constant; multiple factors come into the picture, some of this are not discussed. It is sufficient to know that the distribution of the voltage gated sodium channels in the voltage gated potassium channel, channels is high at the node of Ranvier.

So, that action potentials are quickly caused in the node of Ranvier when compare with other regions. So and sodium cannot escape through the membrane, because there is insulation, so once sodium enters here, it diffuses and causes action potential at this point. And once sodium enters here, it diffuses and causes an action potential this point. So, when we are looking say there is a person here, there is a person here.

Let us assume there is a person, this person is watching an action potential is generated at that point and after a very small gap in time, an action potential is generated at that point and then an action potential are at that point, an action potential at that point, an action potential are at that point and then an action potential is generated at that point, it seems as if a wave of action potential is arriving toward this person who is observing from this point ok. for this person a wave of action potential seems to be arriving and its seems as if the action potential is jumping from this point to that point, to that point to that point, to that point to that point right. It seems as if the action potential is jumping, actually this not the action potential that is jumping right. So, this situation where an appearance of conduction as if it is jumping is called as Saltatory conduction ok, it is called as Saltatory conduction salt, or Saltatory has its our roots in dance as in dance jumping conduction.

(Refer Slide Time: 23:03)



So, in summary what we have seen is action potential is conducted through saltatory conduction in myelinated axons. And the conduction velocity basically depends on time constant and length constant and the time constant can be reduced by myelination by

essentially myelination reduces time constant, length constant is increased by increasing nerve diameter, but there are limitations as to how much you can increase the nerve diameter using this ok. So, with this we come to the end of this lecture, we will continue this discussion in future class.

Thank you.