Animal Physiology
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Lecture – 26
Spatial & Temporal Summation of Signal in Mesh of Neurons

Welcome back to the lecture series in Animal Physiology. So, we concluded the last lecture with a very brief introduction about special and temporal summation in the neural networks. So, we are into the sixth week and this is the first class of the sixth week. So, here we will elaborate this further what we exactly kind of physiological significance of special and temporal summation. And from there we will move onto the different pathophysiological situations arising due to irregularities or due to certain diseases state of this network. So, it is start off with if you recollect the last slide start here.

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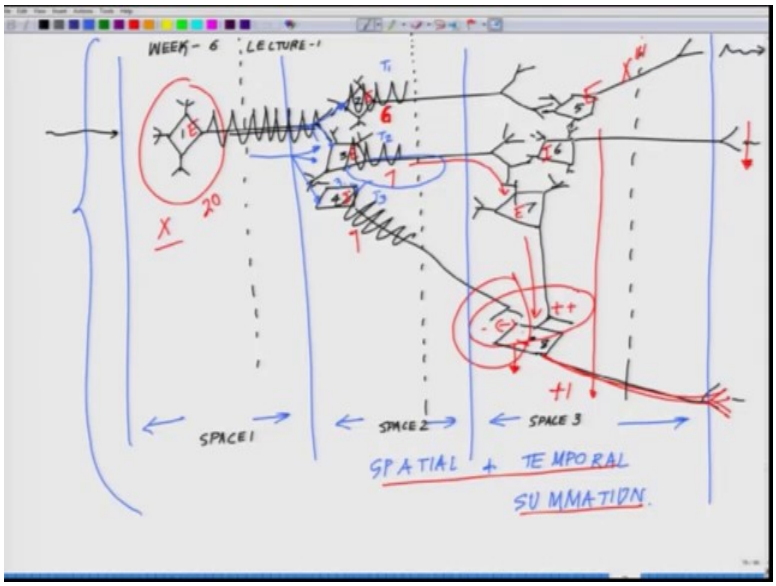

So, this is what we dealt spatial and temporal summation. So, I told you, so there are neurons which are sitting at different places in the network. So, if I call this as so just before that putted line. So, this is continued on week sixth lecture-1. So, if this side considered as space 1, space 2, space 3 likewise, now you can keep on increasing it. So, they are sitting at different places on this space, yet they are connected. So, the communication channel is happening like this. Say for example, this x or if you go by

and solve rather this E, the way we have if this is excitatory. So, this one is sending an signal sending a signal to this space 2, all these different three different neurons.

So, now few things could happen one is at what time who is receiving the signal. So, for example, this signal comes here. So, if this is the first one to receive a signal, then if this is the second one, and if this signal pipe suppose there is a connection something like this or something like this or something like this, and if this is the third one. So, you see you can put certain time into it. So, for example, this one receives a signal at time T 1, this one receives a signal time T 2, this is receiving a signal time T 3. So, let us read write that will make more sense.

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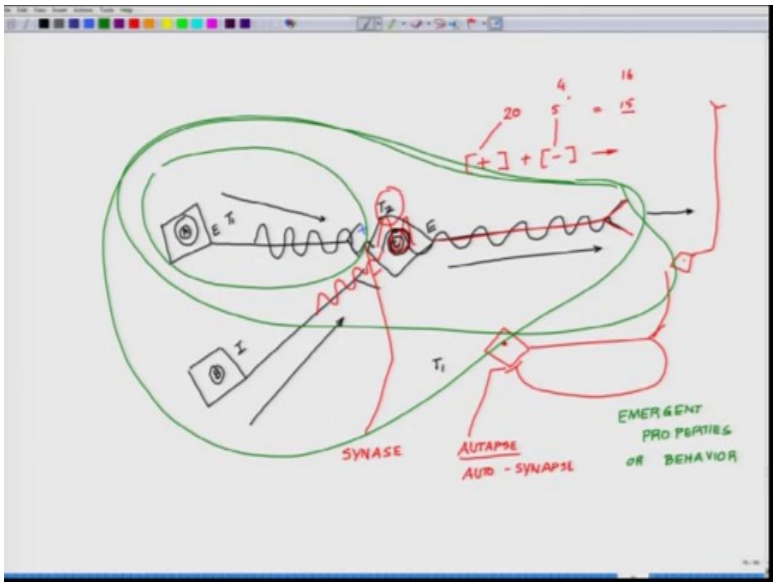

Suppose this is one neuron space, this is another neuron another space. Now, it is sending signal to this. So, this is neuron A, this is neuron B, this is neuron C in the network and the signal transfer is always according to the direction of the arrow. Now, suppose and let us assume this one is excitatory this one is inhibitory this one is excitatory. Now, suppose at time T 1, this A sends a signal. So, this is excitatory, this made this one to generate a signal and this gets transmitted further down. But just imagine at time T 1, this generated a signal; at time T 2 this receives a signal, this the neuron C. But exactly at time T 2, imagine another signal which is a inhibitory signal which arise here, exactly at time T 2, this time T 2 then what will happen.

Then the signal which is supposed, I considered this as in blue as the positive signal or excitatory signal; in red I considered this as negative signal. So, what will happen there will be an addition of the quantity of the positive and the negative signal. So, I have been given it a quantity, I have just given it a sign. And the resultant will decide whether this neuron which is the C neuron which is an excitatory neuron whether this will transmit the signal further or not or what will be the amount of signal which will be transmitted.

So, for example, I see this positive signal is 20 unit some 20, and the negative signal is say 5, then the total signal which will be transmitted will be 15. Or while this one is getting excited with a slight delay this inhibitory signal comes even before it generates an action potential part of it. So, there may be slight reduction in five this may be like you know becomes 4. So, your resultant will become 16 likewise. So, this is the most simplistic system where I am showing you this, but just imagine if this becomes a very complex network where you have inhibitory signal excitatory signal self-signal. There is something so for example, a neuron out here if sends part of the signal and the part if falls back on itself.

So, if you have talked out synapse or the synaptic zones, synapse there is something called autapse or you can break it as auto synapse. So, in other word, a part of the signal will be fallen back, similarly it could have part of the signal going to the next one. So, there are millions of possibilities which could happen in a network. And what we considered as behaviour consciousness, these are emergent properties as they call it of the network. It is the summation of the network what makes us who we are, and it is a very very complex network and if you looked biology with respect to time, so you will observe that the last century is the century of cellular and molecular biology, there are lot of molecular events, cellular events which have been defined.

Earlier to that, it was more of a naturalist way. So, you see the grass structure and its behaviour and all the kind of stuff. So, what you used to see. So, evolution of biology is like that initially we were more we never had the tools, we were more concerned about the behaviour the overall behaviour of the network. But last century saw where we try to understand the individual function or molecular events happening at the individual nodes of the network, which includes say for example, what is happening in a single neuron. What is happening in a small network like this or what is happening in a complex network like this likewise. But now the next generation of physiologist,

neurophysiologist, neural engineers the idea will be much more to understand the holistic viewpoint.

Now, we have a sufficiently good understanding about single neuron or you know couple of neurons are there, but then how these things really leads to the word what I use the emergent behaviour or emergent properties that is something emergent properties or emergent behaviour that is something which will boggle the scientist in the centuries to come. So, it will be a long journey where we will be kind of you know trying to understand this emergent phenomena. But at this point having seen you this I had few other interesting details about this network. So, we have already talked about that at the synapses, if this is the synaptic cleft where neurons are transmitting there signal through the neurotransmitters. So, these neurotransmitters can be either excitatory and inhibitory we have talked about it.

But what is important is that these synapses where the neurotransmitters are release say for example, if this is the presynaptic terminal and this is the postsynaptic terminal my this hand the one which I am waving now is the postsynaptic terminal and this is the presynaptic terminal. And they are like you know close opposition on very close they are not in contact, but very close to each other. So, this one, the one which I am waving now. If this one secretes neurotransmitters and this one receives the neurotransmitter one now which I am waving which is the post one.

But this neurotransmitter has a finite residual time in that small cleft if that residual time exceeds by some x, y, z reason, need not worry about what is the reason at this time then that will create a lot of trouble. Why it will create a lot of trouble just think about it just visualize what will happen. So, this one secretes at time T 1, this one receives a time T 2, and immediately this one gets excited and the signal gets transmitted along this neuron. But if this prolongs what will happen if this is an excitatory one which is getting secreted right.

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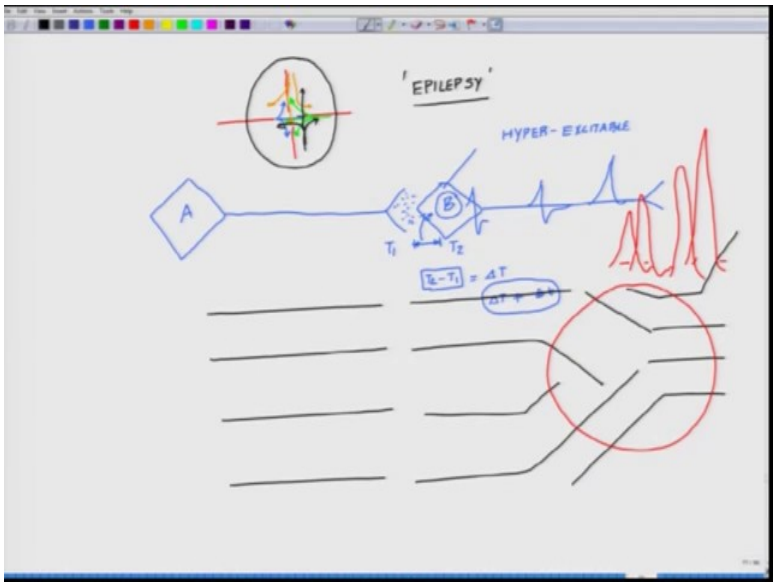

So, for example, just let me put it on the slides that will make more sense. So, this is the one which is sending signal and this is the one which is receiving signal. So, this is A and this is B. Now, this one generates an action potential at say T 1 and this one is supposed to receive at T 2. And this is where the neurotransmitter are release and they bind here, and they open up the sodium channels, and they generate the necessary action potential to travel. So, there is a small T 2 minus T 1 a small time window when this event is happening.

Now, this time window is very critical. If this time window prolongs say for example, this comes as you know delta T, if you have any delta T plus some other delta added to that some other small time then what will happen if this one if A is secrete neuron excitatory neurotransmitter then this B will become hyper excitable. Because it will have much more positively charged ions or sodium ions which will be getting through the voltage gated channels.

Such situation could lead to a different level of activity of the network at times. And at times such situation could be pathological how. I do not know how many of you have seen this. If you have seen a epilepsy patient, the symptoms are very interesting. Whenever a epilepsy patient gets a bout epileptic bouts, what happen you know I mean the person start all of a sudden like you know loose contact of the system and kind of start shivering and fall down. And once they regain consciousness if you ask that person

do you remember anything what happen, they will say no they become dizzy and kind of you know. And there is enormous movement of all the hands legs and everything like you know something like this.

What happen the exactly is from the neurophysiological perspective in the vast networks say for example, if I consider something like this, if this is the network, I am not drawing the individual neurons now. If this is the network, I am just keeping a gap just to tell synaptic at certain zones of the network; there is a higher excitability all of a sudden for a transient period of time. So, much so that the individual kind of faints out because it cannot compensate for that high metabolic activity because whenever these cells are synthesizing such a such vast amount of neurotransmitters and secreting. So, obviously, for that process to happen in a fraction of time that requires enormous amount of energy to be produced by the cells. And this individual who suffers from such thing faints out. It is almost like there is another analogy by which you can understand this it is something like imagine a four way crossing like this. So, four way crossing has a row right.

So, if these ones when this one go there will go straight or they go like this or they go like this fine. Similarly, when the other particles from this side we either will go like this they will go like this they will go like this. Similarly, if you are coming from the top, so something like this something like this and all will go straight and exactly where we see token if you are coming from this side you will go like this or you will go like this.

Now, synapses as to imagine something like a four way crossing, multiple synapses at one point and one is talking to the second one, second one is talking to the third one, and there is a lot of cross talk happening without obstructing each other. It is a very interesting zone, where these cross talks are happening. And one has to understand that if in a four way crossing, if the traffic light goes failure, if the traffic lights are not working, there will be nothing but collision like this like this. And just you can use this that analogy to understand what will happen in epileptic bouts in the case of epilepsy. There is an hyper excitability of the system and the system goes totally heaver, but where this happens, which part of the brain is involved in it.

So, as of now I have not talk to about the demarcation line about epilepsy or the demarcation line between the central nervous system problems, peripheral nervous

system problems and other accessory problems. So, let us do something. Let us draw a map out here for us to proceed further which all aspects are we going to deal here.

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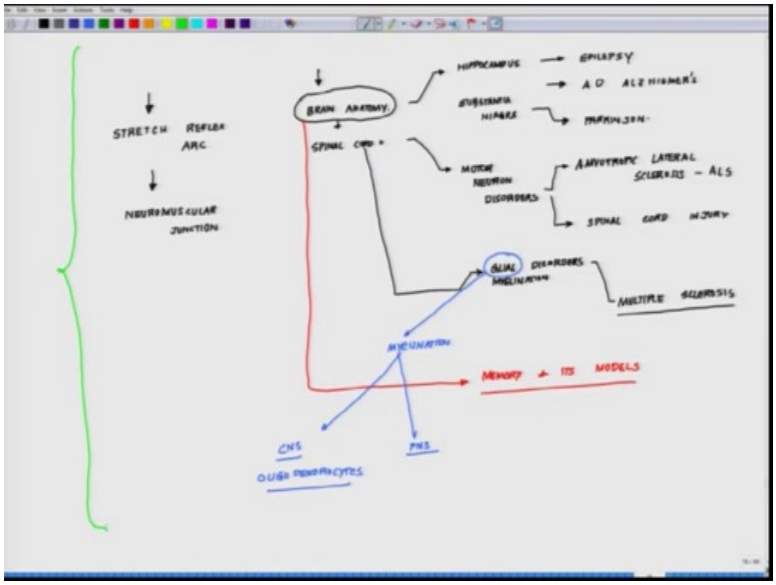

So, what we will do we will talk about briefly we will revisit in the light of this briefly we will revisit a stretch reflects arc which we have already started but we have not added all the necessary components of the action potentials and everything. Second, out here as an extensional, we will talk about neuromuscular junction; in short sometime they call it NMJ. Then we will talk about brain anatomy and spinal cord anatomy which part we have already talked. Here we will talk little bit about hippocampus and related disorders which one we have already started.

We will conclude once I show you the anatomical location epilepsy, AD or alzheimers disease alzheimers. Then we will talk about substantia nigra and Parkinson. In this spinal cord, we will talk about motor neuron disorder which will include amyotrophic lateral sclerosis or in short they call it ALS. Then we will talk about spinal cord injury. And within spinal cord, we will talk about glial disorders. Remember I have introduced about the glial cells, glial myelination disorders, but there will be we will talk about very briefly about multiple sclerosis. And in this whole process talking about the brain anatomy. The most emergent property or evolving property is memory and its modules.

In the light of whatever we have understood about the process itself and while talking about this multiple sclerosis, we will devote little bit time about myelination in CNS and

PNS which is peripheral nervous system. And here we will talk about oligodendrocyte. So, this will be our map what will be following for next few classes to conclude this part of the course. So, I am closing here. In the next class, we will continue following this map.

Thank you.