Foundations of Cognitive Robotics
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Lecture - 08

Good morning students. Welcome to the class of introduction to Cognitive Robotics. In this lecture we will focus on the dynamics of a single neuron itself. So far, I have given you the morphological description of a neuron that, what does a neuron consist of and what are the different parts and what are the different types of neuron.

But now, the time has come for us to focus on the neuronal dynamics. Because only if we understand the neuronal dynamics, we will be able to understand how a group of neurons will work together. And then we will be able to understand that how these manifestations of networking of neuron would actually affect the cognitive aspects of let us say, homo sapiens or any such other you know living beings with group of neurons associated in related in relation to their brain action.

So, it is extremely important for us, A. to understand the neuronal dynamic, so that we can understand finally, its manifestation in terms of different cognitive aspects. It is also important for us when we will be developing a cognitive robot in the other side in terms of the development of the robot, we have to understand that similar to these neuronal networks we have to develop some artificial neuronal network of kind of similar complexity.

So, hence it is absolutely essential for us to understand the neuronal dynamics which is what we will be trying in today's lecture.

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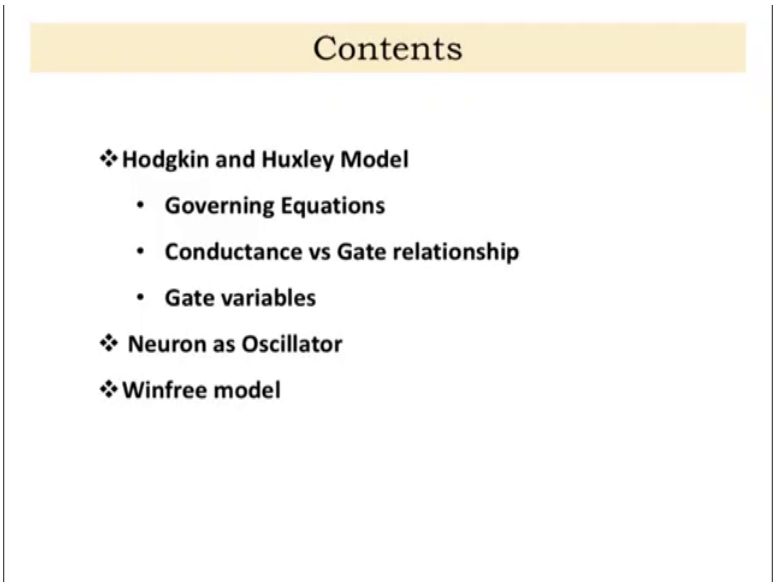

In this lecture, I will mostly focus on Hodgkin and Huxley model which is a classical model of a dynamic model of a neuron. And based on the 1962 Nobel Prize of Hodgkin and Huxley so, that we will focus the governing equations, the conductance versus gate relationship, the gate variables. Then we will talk a little bit about neuron as oscillator and the Winfree model, so that will be the flow of this lecture.

Now, before we talk about the H-H model itself. Let us first see that how the action potential gets generated inside a neuron, because ultimately the dynamics of the neuron will be governed by the dynamics of the action potential. How the action potential moves with respect to different opening of the gates, that is what we will be ultimately matter. So, let us look into that.

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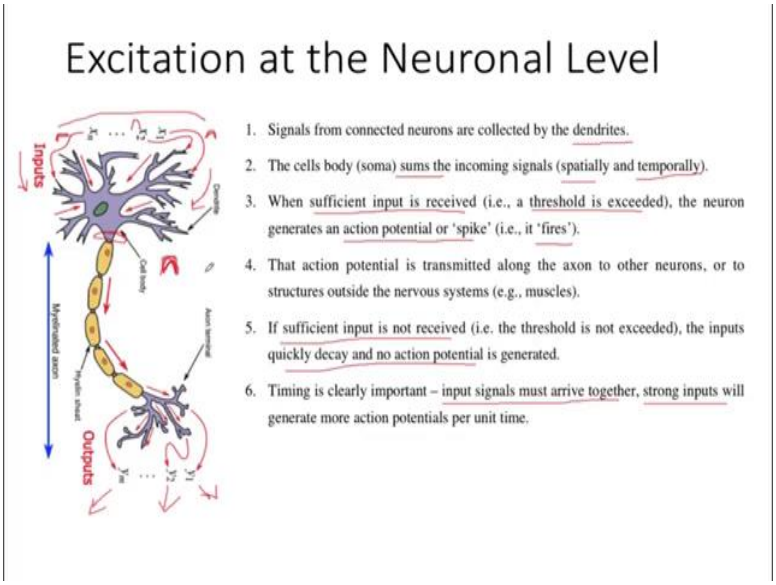

If you look at the excitation at the neuronal level and then what you would see is that. Let us say, there are many many inputs that a neuron will be gathering from its dendrites. Now, there are two types of dendrites there are some called basal dendrite and some called apical dendrite.

Basal dendrites are directly related to the soma and apical dendrites basically come from far away and through a channel that actually comes and sends the signal to the soma. But right now, let us consider that there are n number of inputs that are coming to this dendrites into the soma which is the cell body.

So, if we look at these procedure. So, you have these inputs are coming from all over ok. So, signals are connected by neurons from the dendrites. That is the first part. And then the soma basically it will work like an integrator. So, the cell body soma sums the incoming signals both specially as well as temporally.

So, let us say that if there is one signal coming from this part, another signal coming from this part then, inside the soma this will become a you know something which will be an integration of these two signals.

So, it will be somewhat more than what was there. Of course, the signals will be little bit of decaying as it will be coming from these sides towards this distance ok.

However, if the two signals are actually they are at the same time then there is a good chance that they will be integrated these two dendrite signals they will be integrated here and you will be getting.

So, in terms of special integration like that if there are n number of channels which are contributing say one more channel is also giving. So, you will be getting an even higher signal ok. So, that is how the signal integrations will happen at the soma now, when sufficient input is received. In fact, a threshold of course, there is. Nowadays, people have discovered that there is not a fixed threshold, but there is a threshold band.

But whenever these threshold around that region it is exceeded, then only the neuron will generate an action potential or spike. This is also called firing of the neuron. So, this will happen around this area ok. This is where the firing of the neuron will start and then that action potential will start to travel. So, the action potential then is transmitted along the axon that is how it is going and finally, it will go to other neuron.

So, this x 1's will generate the action potential. It will come here and then from this synaptic junctions it will go to all other neurals y 1 y 2's are different outputs, that are going to go to you know different n number of neurons. And if however, sufficient input is not received, then the inputs will quickly decay and no action potential will be generated. So, the timing is very important. The more input signals will arrive together, the higher the chance that there will be a strong input which will come.

And the more number of the inputs will actually be integrating, there also is a higher chance that this input is going to exceed the threshold level and hence the action firing is going to take place.

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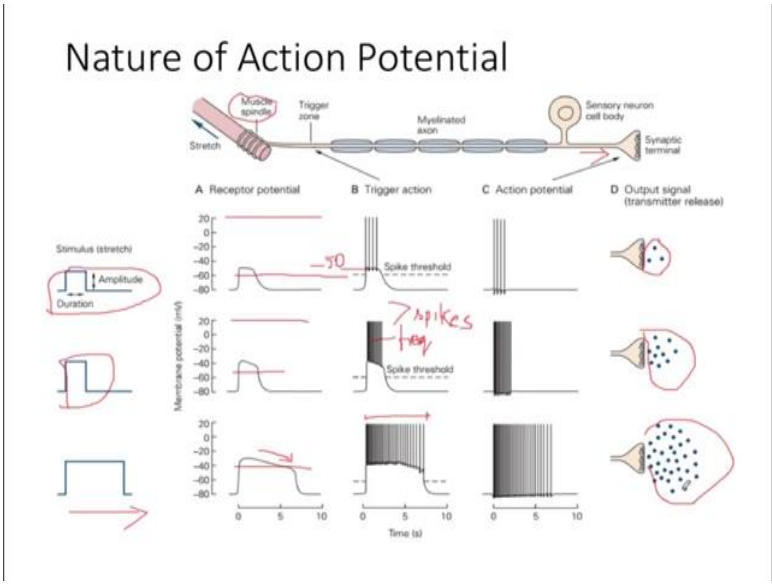

Let us look into it with respect to a practical case. So, we have talked about if you remember the reflex action. So, let us look into it in terms of the reflex action. If you remember that in case of a reflex action, the nerves or the afferent nerves were actually linked with the muscle spindle.

So, this is where is the muscle spindle. So, whenever we were actually giving a little bit of heating, there is this stretching that is happening and this tweaking of the muscle spindle is actually getting the you know the triggering in terms of the neuron and that triggering is coming through the myelinated action to the cell body and from there further it is going to the synaptic terminals.

Now, if you look at it that the initial stimulus of the signal the duration of heat is important ok. So, if this is the initial signal, there will be a little bit of degradation of the signal and finally, that initial signal will create if it crosses the threshold. So, because this is above the threshold level the usual resting potential is about -60 milli volt and about -50 milli volt you will get a threshold level. So, this will be around -50, -50 or so.

So, this varies, but roughly you know it is about -50. If it is above this then you will see that there is a spike that is forming. And as many number of times this is happening, that many number of spikes will be forming.

So, these spikes are formed then the spikes are traveling and at the output level this is actually creating the neuro-transmitters. Now, if the signal continues with a higher value and for a you know some kind of a high intensity values.

Then what can happen is that, again once again it will check that whether it is above the threshold level or not. So, if it is above the threshold level then this spike intensity may actually increase. So, the frequency of the spike not the intensity the frequency of the spike generally the spikes actually the amplitude will not change ok. You can see here also it is about 20 here also it is about 20. So, this is not going to change.

But between this signal and this signal, because this signal has input signal has a higher intensity, what you may see that it will get reflected in terms of more number of action potential spike. So, there will be more spikes there will be greater spikes in this case ok. So, the spikes will be higher in this case and that would mean that the neuronal firing will be actually higher in this case. So, that is what we have to greater spikes greater spikes will be there in this case.

So, that is what we can see. In fact, the other case. Suppose, if the duration is longer in this case the duration is longer. So, it is above the threshold level for a longer period of time. And in that case, what will happen is that, now the number of spikes, the density of the number of spikes will be something like you know that it will be not affected, but it will be more in terms of the duration of the spike that total duration of the spike initially was smaller here, it will be bigger.

And another thing also you can see that as the graded potential is degrading, you can see that here also the density of the spike that is actually getting lighter and lighter. And accordingly, you would see that the generation of you know the neurotransmission in different cases will be different. So, finally, that will be the output of the system. So, this is how typically a neuronal dynamic will actually happen. In this case it is for a reflex action.

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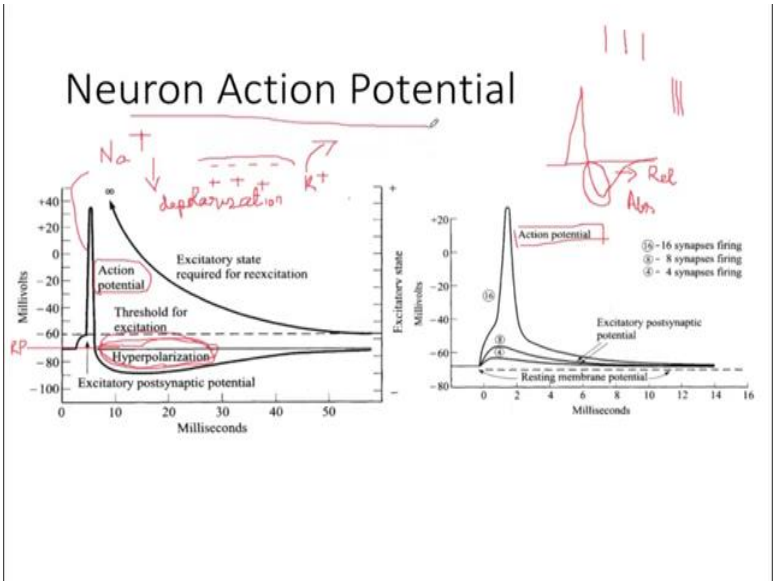

Now, there is one point that we have to always keep in your mind that in the beginning, particular neuron will be always at its resting potential level. So, it will be at its resting potential ok. So, wherever is this resting potential it will be at that level in the beginning. In this case it is showing that this is the resting potential level usually it is up to -60 millivolt. So, that is where it will be. So, that is the resting potential.

Now, once the you know charges or the inputs are accumulating then what you will be having is called the graded potential. So, basically this happens when lot of sodium's are actually you know rushing inside. So, there is a movement of sodium that happen. So, this part in this part there is lot of sodium which will be actually sodium+ which will be actually going inside the neuron ok.

So, basically what happens is that in a neuronal cell the inside part of the neuronal always maintains itself with a negative potential. So, as the sodium will be going inside then, you see that from the resting potential it will be actually going up. So, that is what it is happening it is going towards the positive side.

Now, this is something that we actually call there is a technical term for it is called depolarization. So, that is what is going to happen to the system.

So, as more and more you know sodium is going to go inside the cell there will be more and more depolarization of the cells ok. So, that is what is the first result. That is why it is

crossing the threshold level and it is going to a high level. And as soon as this happens, immediately the action potential will be released; that means, immediately there will be a spike that will come up.

Now, once there is sufficient sodium then the sodium's which are + right. So, they will start to repel each other and hence they will spread inside the system and they will also further reduce the inflow of the sodium. So, that will come down that is why you can see that the potential is coming down. Simultaneously, there is a potassium movement that will start to happen.

So, this k+ is going to leave the system this happens slowly ok. So, this will create the hyperpolarization and that will create actually the further negative part of the signal. So, depolarization will actually inspire the action potential generation whereas, hyper potential will actually create you know negative of action potential.

It will not allow the generation of action potential we have to keep this in mind. That is why in any common signal it will be the firing action potential will be something like a spike followed by a slow negative side until it comes back to the resting potential.

So, that is the very common pattern of a signal that you will find in any neuronal signal. Now, the other point we have to keep in your mind is that the more the receiving signals are like 4 synapses 8 synapses 16 synapses you are going to see that these graded potential is going to increase and that will create finally, the generation of the action potential. However, as I told you earlier also that the magnitude of the action potential by and large remains constant that does not change.

Only if the intensity is very high it may result on many high density action potentials ok. And if the intensity is low it may result on few action potentials which are separated with respect to time in a much lighter density ok. So, these are the common things that we have to keep in our mind.

The other point is that during the hyperpolarization phase the cell generally would not, the neural cell generally would not, initiate one more action spiking. Particularly there is one region up to this region which is also called absolute you know part of the hyperpolarization. So, this is the absolute part ok. So, it is absolute hyperpolarization. There you know it will not allow any firing of the action potential at all.

But it is the later part this part which is known as the relative part of the hyperpolarization this is where it will allow a firing if at all required it will allow the action potential to fire. So, these are the points we have to keep in our mind in terms of the neuron action potential. Now, that you know about that how an action potential gets generated in a neuron, let us look into that what you know how we can mathematically define this action potential generation in the system.

So, and to do that we will take ourselves to the Hodgkin's-Huxley theorem which is the classical you know theorem of explaining this neural dynamics. Later on, we will see that this theory further gets modified, but let us start with the very popular fundamental theorem which governs this.

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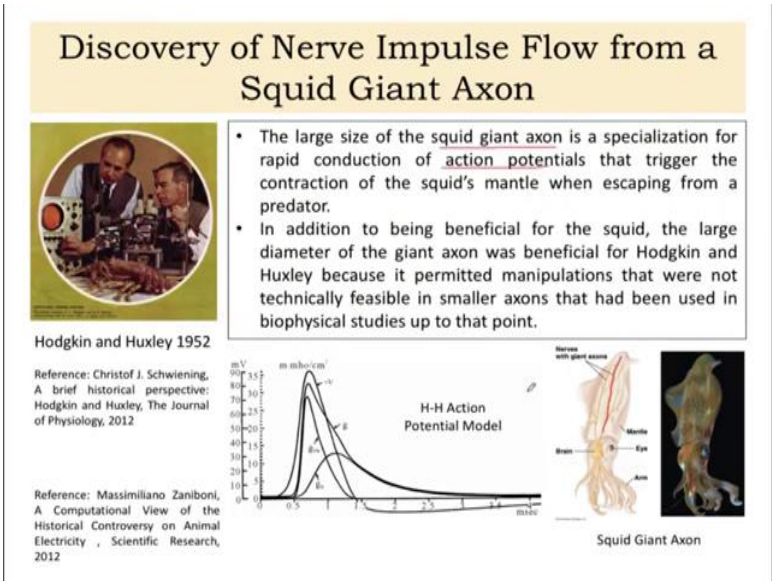

So, we will now talk about the Discovery of the Nerve Impulse Flow from a Squid Giant Axon that was done by the Hodgkin and Huxley in 1952. And as you can see that you know they both Hodgkin and Huxley working on a giant squid.

So, these become like a legendary photograph that was the first time that the record of a typical action potential of a neuron that has been done actually. Now, the reason why they had chosen the giant squid action the action of a giant squid is basically, because this is quite thick actually.

So, it was easier in those days to carry out the experiments and to see the action potential ok. Now so, you can see here that typically how this action potential signal is. But you have to of course, cross the threshold level in order to see this firing that is happening into the system.

And you can see that there is a you know squid here and you can see that the nerves which are associated, the nerves you can see here which are associated, with the giant you know squid giant actions they call it. Now, this is used by the squids by the by for their escapes for all emergency purpose.

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So, let us look into it. Let us look into that, how generally the squids actually propane? As you can see that this is how squid is generally propelling and at that time it does not use much that giant action you know that is there for the squid.

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So, this is how they are tracking. But you will see whenever emergency comes how fast this squid is actually going to work.

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So, if you continuously follow that, how is happening? And you can see that you know this is the squid and suddenly you will see that this is the point where it has actually given this kind of a signal and then it is happening at a very fast rate.

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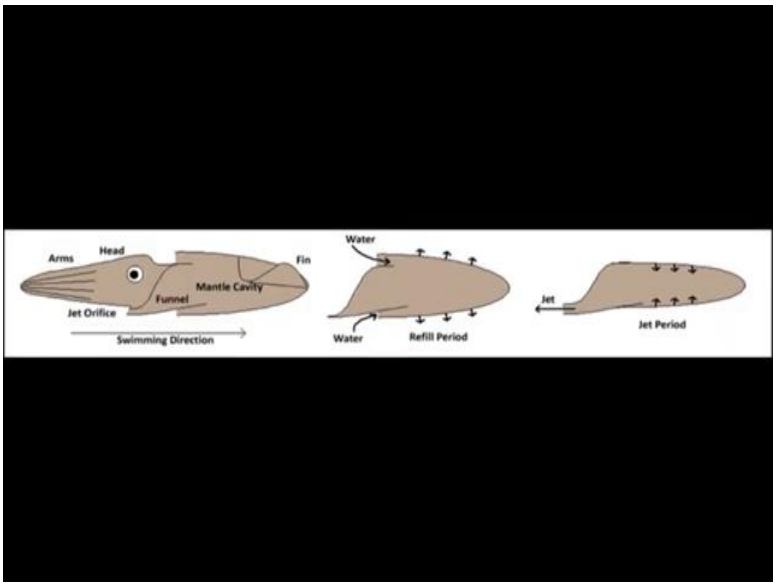

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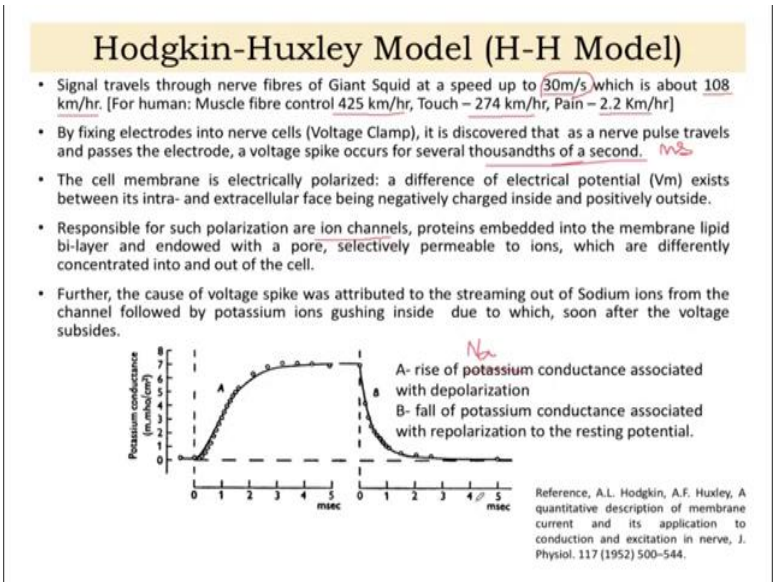

Now, let us look into the Hodgkin-Huxley model that has been developed based on the experiment on the Giant Squid action. Now, what is the nerve speed that was there in this particular case? Well this is about 30 meter per second. So, that is the kind of a nerve speed.

And that means, it will be about 108 kilometer per hour. So, something like a very high speed car or a very high speed train that you see that will be having this kind of a nerve speed. Now, for your information, for human: muscle fiber control speed is something like

425 kilometers per hour almost like the air-crafts taking off speed. And the touch is about 274 kilometer per hour almost like a bullet train speed.

However, the pain signal that goes at a very slow speed something like 2.2 kilometer per hour. So, this is just a reference that where we are in terms of the giant squid speed which is not very high, not very slow.

Now, by fixing electrodes into the nerve cells they call it voltage clamp then, they have actually you know they have been able to excite and they can see the spike which occurs generally in the millisecond level thousandths of a second in the millisecond level this reaction happens. You know it is corresponding to 30 meter per second.

So, the cell membranes are electrically polarized and difference of electric potential exists as I told you in general and that is in the negative side. And that happens and the this change also. So, these are all these all happens because of the presence of the ion channels. So, there are three different types of ion channels: the sodium ion channel, potassium ion channel and leakage channel and also there is a calcium channel.

So, these all about four, but in our model we consider three. Sodium, potassium and leakage channel. The calcium we will not consider in our model now. So, what happens as I told you that there is these sodium's which will be rushing inside ok, and then it will be followed by the potassium which will going out of the nerve cell. So, that would create basically this kind of a positive and then followed by a negative signal.

So, there will be you know part A which is the rise of the potassium conductance, it should be actually not potassium sorry it should be sodium conductance. So, that is one part. And followed by B in which there will be the potassium conductance part of it ok.

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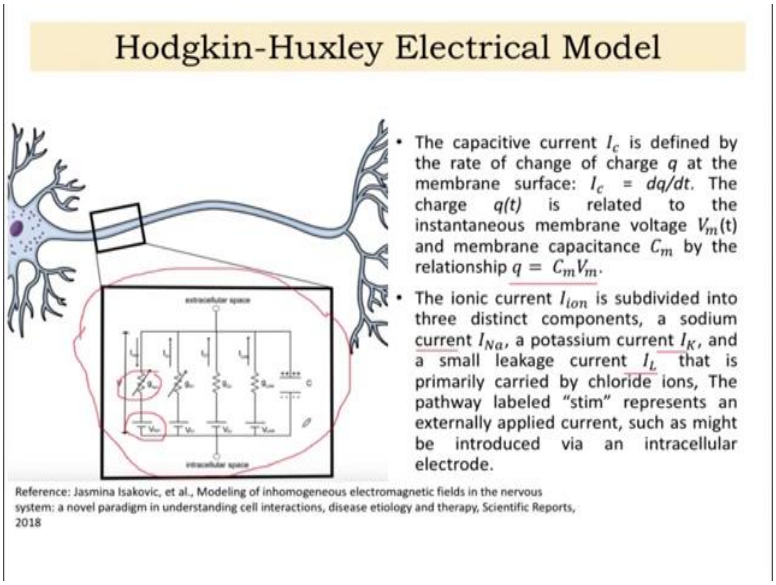

So, let us look into the Hodgkin's-Huxley model now. So, as we will be actually applying the capacitive current I_c which is defined by the rate of change of charge q at the membrane surface.

So, I_c is the rate of change that is dq/dt. Now, the charge q(t) is related to the instantaneous membrane voltage V_m(t) and membrane capacitance C_m. So, you know that there is this relationship that exists which is $q = C_m V_m$.

Now, the ionic current I_{ion}, if I try to actually represent what is happening inside an action potential in terms of a simple electrical circuit, then you can consider it to be something which is a combination of you can say resistors of variable resistance. So, these are the resistors and that is basically shown in terms of conductance. And also these are the capacitors.

So, a combination of them. And as you can see that there are actually three of them that is used in this model, that is the sodium, the potassium and the leakage. Generally, these three that is used for this case.

Now, the ionic current I_{ion} is subdivided into three distinct components, that is the sodium current part corresponding to the sodium part of it. And then a potassium current part I_K and a small leakage current I_L and which is primarily carried by the chloride ions. So, these

are the three currents that are actually streaming into the system. So, and that is the full system along with the capacitance corresponding to each one them.

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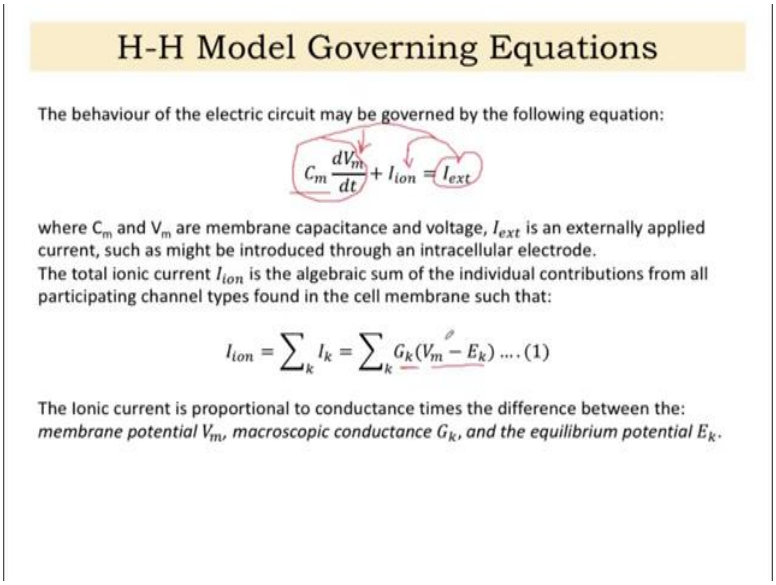

So, you can write a mathematical equation. And which is like the C_m. C_m is the membrane capacitance. So, C_m is the membrane capacitance ok, and V_m is the voltage that you are actually either you are applying the voltage or you are measuring the voltage membrane voltage. And I_{ion} that is the current that is actually passing through the ion circuit. So, that is the I_{ion} part of it. And then $I_{external}$, that is the externally applied current.

$$C_m \frac{dV_m}{dt} + I_{ion} = I_{ext}$$

So, in this model we are applying the current from outside. As a result of it, this will actually generate I_{ion} and also it will generate these $C_m \cdot dV_m/d\,t$ and that is related to the capacitance ok. So, these are the two things that this I_{ext} is going to develop. Now, I_{ion} itself are actually can consist of three or four part of it. So, it depends on the conductance of each one.

$$I_{ion} = \sum_k I_k = \sum_k G_k(V_m - E_k)$$

As I told you sodium, potassium, leakage like that. And then the difference so that conductance time the difference between the voltage V_m of the membrane potential - the

equilibrium potential E_k. So, that will give us the ionic current each ionic current. So, that is what we will be actually this equation will tell us.

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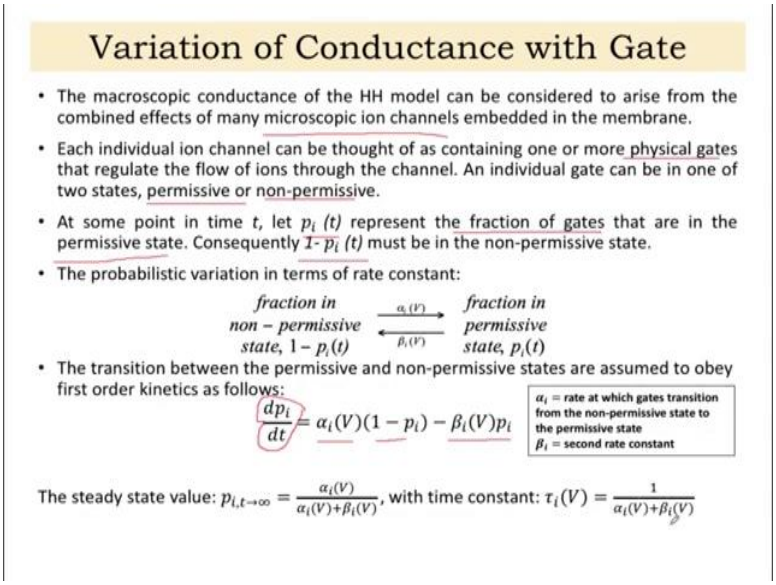

Now, in the macroscopic conductance of the H-H model, this will be actually a combined effect of many many you know microscopic ion channels ok.

So, it is not just one single ion channel. There are many ion channels which are coming into it. Each individual ion channel can be thought of as containing one or more physical gates that regulate the flow of ions through the channel. An individual gate can be in one of the two steps, that is permissive or non-permissive. So, this is either it will allow the thing the you know ion to move inside the neuron or it will not allow.

So, at some point in time, let us say $p_i(t)$ is the fraction of gates that are in the permissive state; that means, it will allow the let us say the sodium to come inside. Then $1 - p_i$ must be in the non-permissive state. So, the probabilistic variation in terms of rate constant.

$$\frac{dp_i}{dt} = \alpha_i(V)(1-p_i) - \beta_i(V)p_i$$

Let us say $\alpha_i(V)$ is fraction which will go from non-permissive to permissive state. And $\beta_i(V)$ is corresponding to that which will take it from non permissive to non-permissive state. So, this is the dynamics that will continuously happen.

So, at any point of time; that means, the fraction of gates that are actually in the permissive state: d p_i/d t. It will depend on two things, one is that this weightage of $\alpha_i(V)$ how many are actually contributing to it? And in a 1-p_i stage ok. So, $\alpha_i(V)(1 - p_i)$. And then how many are in the other way transmission, permissive to non-permissive. So, that is $\beta_i(V)p_i$.

So, this ratio of α and β is going to actually give me the d p_i/d t, which is going to tell me that what is the fraction of gates that are in the permissive state? So, a steady state value of this equation will be something like p_i(t) tends to infinity and that will be coming up if I actually put this as 0, you would see that it will be $\frac{\alpha_i(V)}{\alpha_i(V)+\beta_i(V)}$.

$$p_{i,t\to\infty} = \frac{\alpha_i(V)}{\alpha_i(V) + \beta_i(V)}$$
$$\tau_i(V) = \frac{1}{\alpha_i(V) + \beta_i(V)}$$

So, if you solve this you will see that p_i would become $\frac{\alpha_i(V)}{\alpha_i(V)+\beta_i(V)}$. And the corresponding time constant is $\frac{1}{\alpha_i(V)+\beta_i(V)}$.

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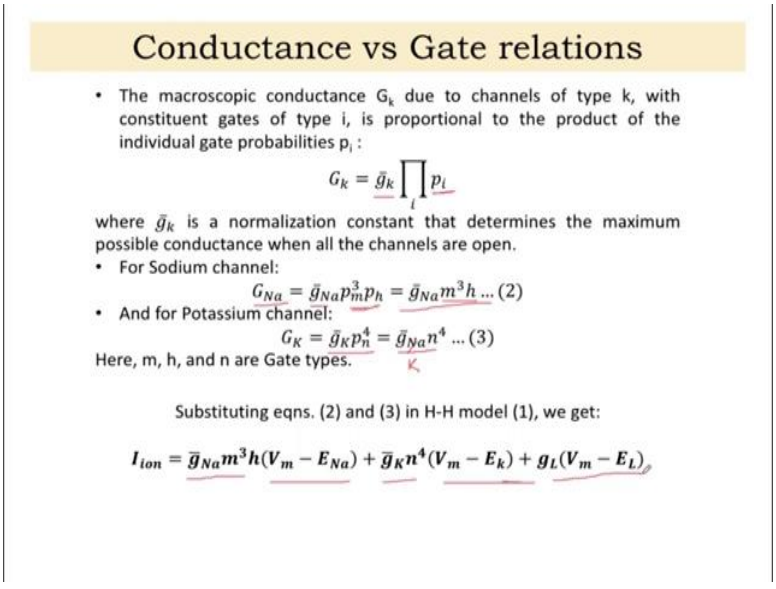

Now, let us say the conductance of the gates. Let us call each one of the conductance of the gates in terms of G_k. So, the conductance of the gates as G_k. So, it will be each individual conductance and that product of this probability of the opening of the gates.

$$G_k = \bar{g}_k \prod_i p_i$$

So, it is g_k times product of that p_is ok. So, let us say we are talking about the sodium channel then there is a gain there, G_{Na} times p_m^3 times p_h ok. We will call it as m^3h ok. And similarly, for the potassium channel this is g_K p_n^4. We will call it g a potassium G_K it should be g_K n^4 and these are different type.

$$G_{Na} = \bar{g}_{Na} p_m^3 p_h = \bar{g}_{Na} m^3 h$$
$$G_K = \bar{g}_K p_n^4 = \bar{g}_K n^4$$

So, now I putting these conductance into the ionic equation, I can write that the sodium part is g_{Na} m^3h ok. So, g_{Na} m^3h times V_m - E_{Na} then, I can write the potassium part as g_K n^4 (V_m – E_K) and then, the leakage part that is g_L x (V_m - E_L). So, that is how the conductance are related to the gate relations.

$$\boldsymbol{I_{ion} = \bar{g}_{Na} m^3 h (V_m - E_{Na}) + \bar{g}_K n^4 (V_m - E_k) + g_L (V_m - E_L)}$$

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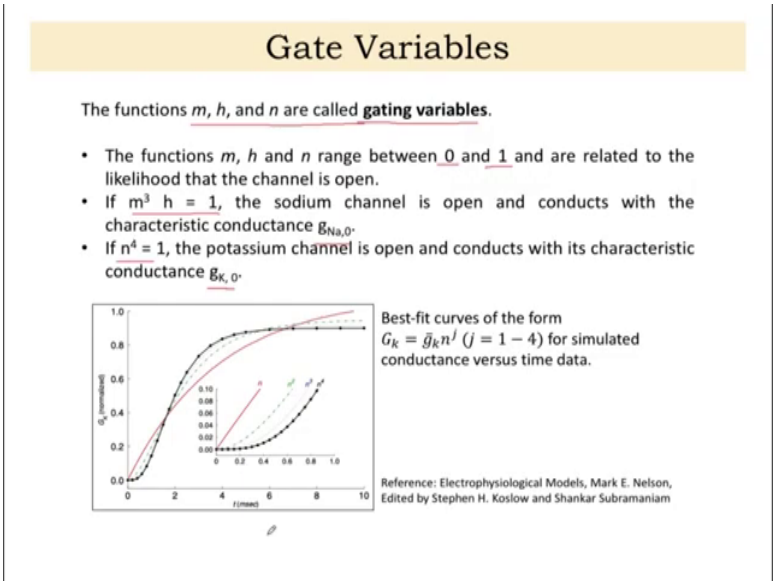

Now, this m, n, h these are all gating variables. Let us look into it how this gating variables itself changes, because they are the things that actually control the ionic current flow in the neuron.

If you look into the gating variables, you would see that there are three gating variables that we have considered in this model m, h and n and their values will be always between 0 and 1.

So, if m cube h is equal to 1, then the sodium channel is open and that conducts with the characteristic conductance g_{Na}. And if n^4 equals to 1 then, the potassium channel is open and that conducts with a characteristic conductance of g_K. So, each one of them it is will happen only when m^3h is 1 then, the sodium channel will be opening or n^4 is 1.

Then the potassium channel is going to open. And as you can see with respect to time. You can see that for each of the cases like, this is the n^4 and you can see that how this n^4 with respect to time how this will be changing and how this g_K will be changing. So, you can. In fact, draw a best-fit curve for each one of these conductance versus the time data.

$$G_k = \bar{g}_k n^j \ (j = 1 - 4)$$

So, that you would know how with respect to time the conductance will be changing or the gate variables will be changing. This you can do for all the gate variables.

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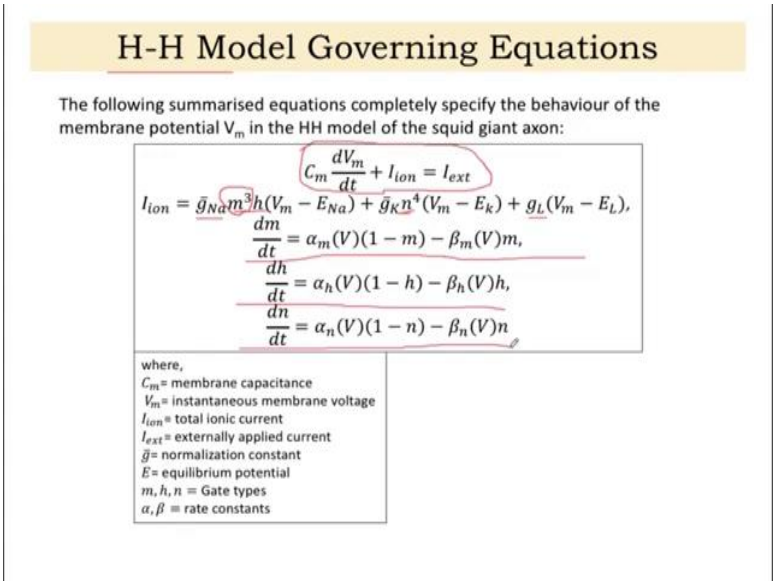

Now, based on all these things together you can actually write the final governing equation which is corresponding to the H-H model. So, the core of it is this first order differential

equation ok, that is the capacitance times the d V_m/d t + I_{ion} equals to the external current that is actually used for excitation of the neuron.

$$C_m \frac{dV_m}{dt} + I_{ion} = I_{ext}$$
$$I_{ion} = \bar{g}_{Na} m^3 h (V_m - E_{Na}) + \bar{g}_K n^4 (V_m - E_k) + g_L (V_m - E_L)$$
$$\frac{dm}{dt} = \alpha_m(V)(1-m) - \beta_m(V)m$$
$$\frac{dh}{dt} = \alpha_h(V)(1-h) - \beta_h(V)h$$
$$\frac{dn}{dt} = \alpha_n(V)(1-n) - \beta_n(V)n$$

Now, the I_{ion} itself as I told you it will be having three parts in it. One part that the sodium part which is governed by the m^3h, which if it is equals to 1, there is a current the sodium current, then there is a potassium current which is governed by n^4, and then there is a leakage current g_L.

And each one of these how many part of fraction of actually the sodium gates will be participating that is governed by this simple first order relationship that, d m/d t is $\alpha_m(V)(1-m) - \beta_m(V)m$.

Similarly, $\frac{dh}{dt} = \alpha_h(V)(1-h) - \beta_h(V)h$ and $\frac{dn}{dt} = \alpha_n(V)(1-n) - \beta_n(V)n$. So, a series of first order equations are to be solved along with the ionic current equation which is a non-linear equation. In order to obtain that what will be let us say, the voltage of the membrane how that will be changing with respect to time.

So far, I have talked of neuron as a dynamic model and how the voltage spikes the origin of the voltage spike. How the voltage will be changing with respect to time as you are applying an excitation current into the system and how you can explain it in terms of different gating variables?

Now, we will see that instead of a time domain representation of a single firing, if a neuron starts to fire continuously can I model that in terms of what you call an oscillator? Now, it is known from the non-linear dynamics representation of a system that, whenever a system actually starts from a quiescent level it is completely placid the neuron before the firing of the action potential to kind of up you know oscillator level where the signals are oscillating.

So, initially it is under a quiescent state then it starts to oscillate. This kind of a behavior is called a bifurcation behavior. And there are different bifurcations model that can actually explain.

So, let us look into the neuron as it fires action potentials with respect to time, how we can model the neuron as an oscillator? Let us look into that. Then we will see how we can feed different bifurcation models into it.

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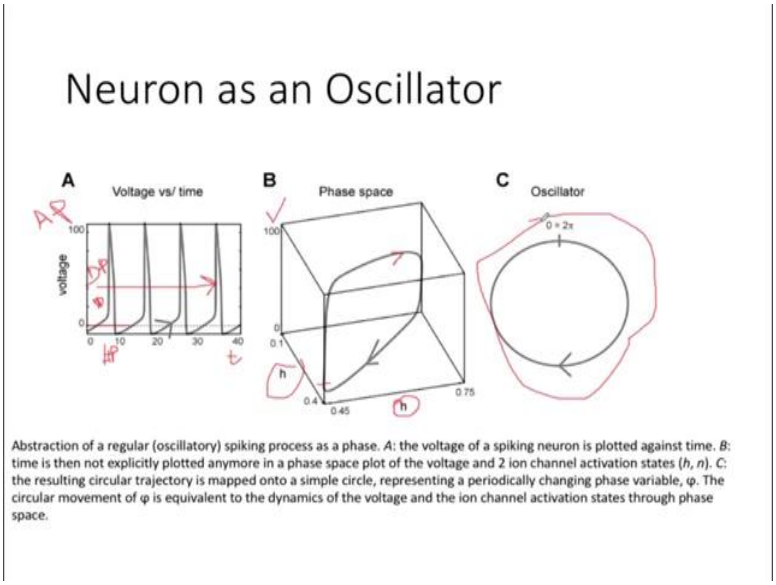

Abstraction of a regular (oscillatory) spiking process as a phase. *A*: the voltage of a spiking neuron is plotted against time. *B*: time is then not explicitly plotted anymore in a phase space plot of the voltage and 2 ion channel activation states (*h*, *n*). *C*: the resulting circular trajectory is mapped onto a simple circle, representing a periodically changing phase variable, φ. The circular movement of φ is equivalent to the dynamics of the voltage and the ion channel activation states through phase space.

Now, here we are talking about a neuron as an oscillator. And as you can see here that this particular signal this is actually the action potential the firing of the action or the action spikes that is happening. So, that is the action potential with respect to time and you can clearly see that is the depolarization phase. You can see and how the sodium currents actually come down.

So, like you can write it as a depolarization phase ok. So, write it as depolarization slightly for a part this is the depolarization. And then you can see that this is what is the hyperpolarization phase, this part below the threshold. So, that is what is the hyperpolarization and you can see how the spiking is happening.

Now, let us say that I would not look into it in terms of this is of course, with respect to time. I am not looking to it with respect to time, but I look into it in terms of this m, h or n

which actually represents the different ionic currents. So, if you look at it that for example, you know how the ionic currents are varying as the spiking is happening.

So, let us look into it from this point of view that here we have three axes, we have h, n and we have the voltage. So, this is the voltage axis end. So, to begin with let us say, that we have a low voltage and then suddenly the voltage is increasing once this threshold is crossing. And as the threshold is crossing and then we will see that there is you know initially there is a value of h that we are having ok.

And as the threshold has crossed, then we can see that the value of h is actually decreasing and it has decreased to a low level. And you can see that the n has started to increase. Now, if you remember it is the n which is related to the potassium part of it right. So, that is you know once again increasing. So, this is what it is continuously happening in the system that h decreasing, n increasing further h changing.

So, that is what continuously happen and this actually can be modeled in terms of an oscillator that the states. If the if I consider the h and n to be two states then, the states themselves are actually forming a circle in a phase diagram which can be depicted in terms of an oscillator.

That is the basis of a neuronal oscillator model just for two channel two activation states. Now, if you consider m, h and n then it will become a three dimensional space.

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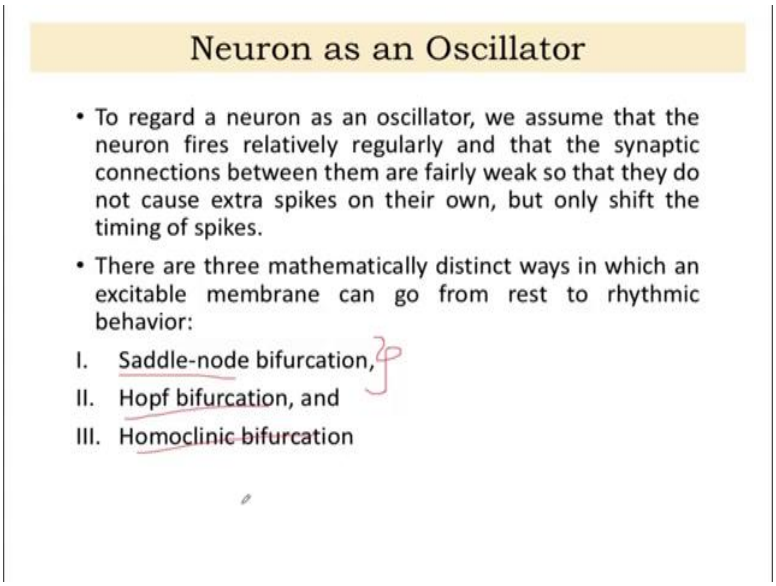

So, as I told you that this oscillator this site of oscillators can be actually modeled mathematically in terms of three different types of bifurcations: A Saddle-node, Hopf bifurcation and Homoclinic bifurcation. But out of them, mostly our discussions will be for the first two cases that is the saddle-node and the Hopf bifurcation case.

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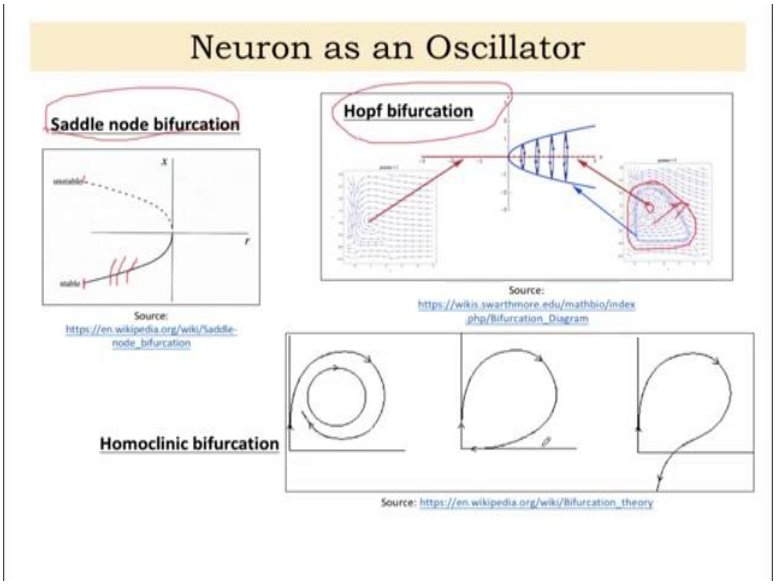

Now, these bifurcations how do they look like the bifurcation diagram if you look at it. Then you would be able to see that for the saddle node there is one node which is stable and one which is unstable.

So, corresponding to the stable node. So, whenever there is this kind of you know the quiescent mode is actually getting you know countered by the generation of the action potential then suddenly, this frequency will start and in the stable node this will create a spiking of the actional signals.

Now, there is also a possibility of two neurons joining together. In such cases what can happen is that signal of one affecting the other may actually create a kind of a bifurcation, in which you know you can see that it is initially at a stage and then it actually goes out and in create a new limit cycle.

So, it is a much bigger limit cycle. So, this is where the oscillation will start. So, this is a smaller limit cycle it is a small you know kind of oscillation, this is the bigger one as the neurons are actually getting synchronized.

So, that is the Hopf bifurcation case. So, these are the two the saddle node and the Hopf bifurcation which we will mostly see the for the neurons as an oscillator. The third one which is a homoclinic bifurcation as you can see the bifurcation diagram here, that is something that is not very natural for the neuronal oscillation system.

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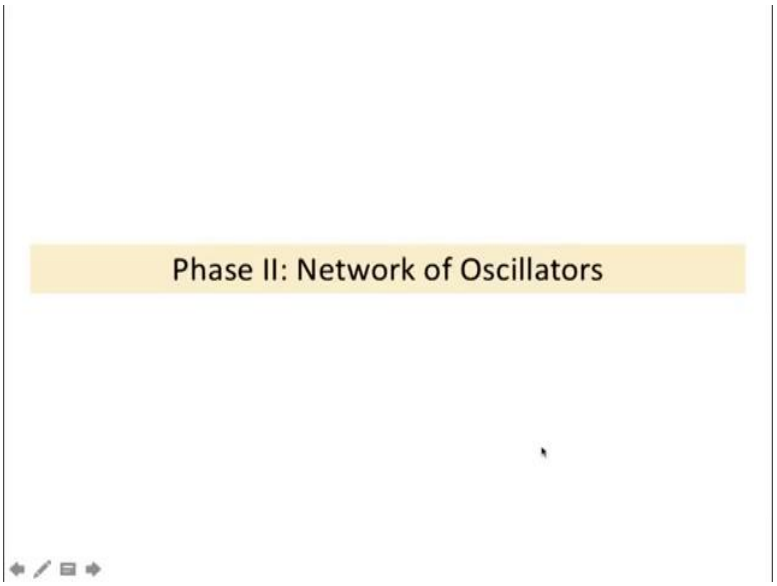

Let us now look into a very simple model of two neurons joining their hands and how a neural oscillation pattern can form out of it.

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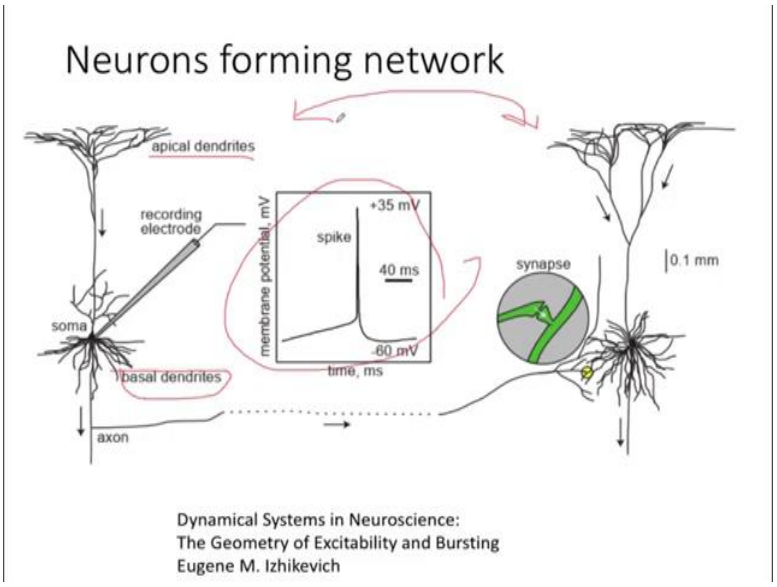

Let us look into the network of the oscillators. Now, to understand that you have to consider two neurons like the two neurons here. They can take inputs from the apical dendrites as I told you that is far away from the soma or they can also take input directly from the dendrites which are directly on the soma, these are called basal dendrites. And based on the integrations they let us say the action will be fired and then you will be getting a spike like this.

Now, there is a synaptic junction here, where the spike is coming and the neurotransmitters are getting released. And that let us say it is taking by the basal dendrites of the second neuron and also some other signals are coming from the apical dendrites based on that the firing happens. So, then the two neurons are actually these are the two pyramidal neurons actually find in found brain.

So, they start to correlate with each other; that means, as one is firing the other also starts to fire.

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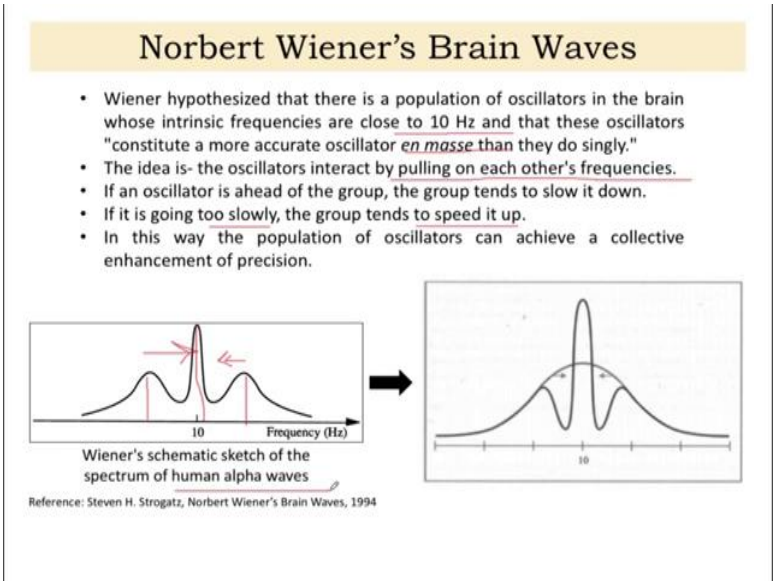

Now, what happen is that. Initially these different neurons can have different frequencies. But Norbert Wiener actually hypothesized that even if initially there are different neurons which are in a cluster and that are operating at different frequency levels, but with respect to time you will see that there is a pulling effect that is happening. You can see that say two neurons here you know one is at a higher one is at a lower level and one neuron which is around this level.

And you can see that these two are also pulled together and somewhere close to 10 Hertz they are going to merge together. Now, whenever this en masse is happening; that means, these oscillators are pulled together to come into a single frequency. This is what we will say that the neurons are getting too are getting synchronized fully synchronized. Now, if a neuron is actually having a frequency of firing too slow, it will try to speed it up or if it is too fast, it will try to slow it down.

Such that, you know it will finally hit into a common platform of a particular frequency. In this case Wiener have shown this with respect to the human alpha waves which is of this kind of a low order between 8 to 10 Hertz.

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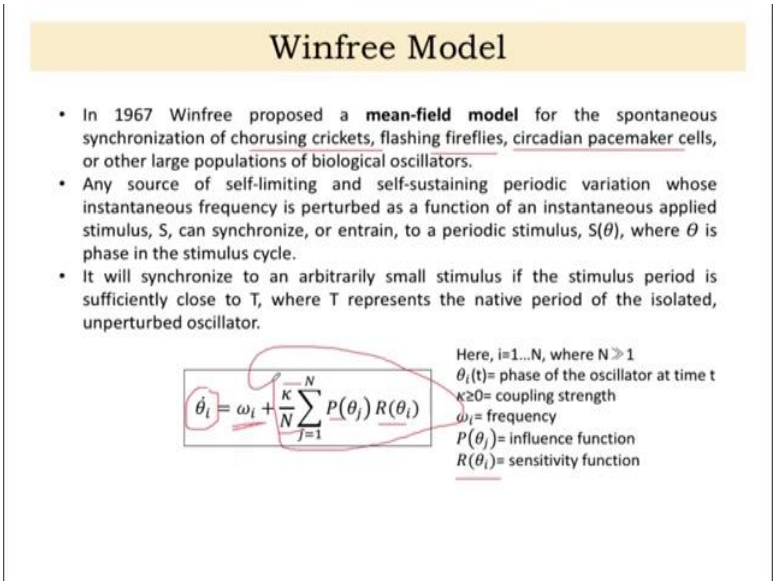

Now, Winfree had proposed a probabilistic model based on this. And he said that this is not only for the neurons, but also it is similar phenomena is found for lots of things like chorusing crickets, flashing of fireflies and of course, the circadian pacemaker cells.

$$\dot{\theta}_i = \omega_i + \frac{\kappa}{N}\sum_{j=1}^{N} P(\theta_j)\, R(\theta_i)$$

So, these similar events happen. And to explain these, one can actually do it with respect to the phases of each one of the individual oscillator. So, the first order relationship of the phase, the rate of change of phase will depend on one frequency, which is the kind of the

frequency of that system along with you can see that there are some constants that influence functions.

For example, P and the total number of oscillators, the coupling strength between the two of these neurons and also the sensitivity function. So, using all these things there is a coupling part that is going to dominate ok. So, and these two things, omega i and this part together, they are going to define the rate of change of phase in a system. So, that is the Winfree's mean field model.

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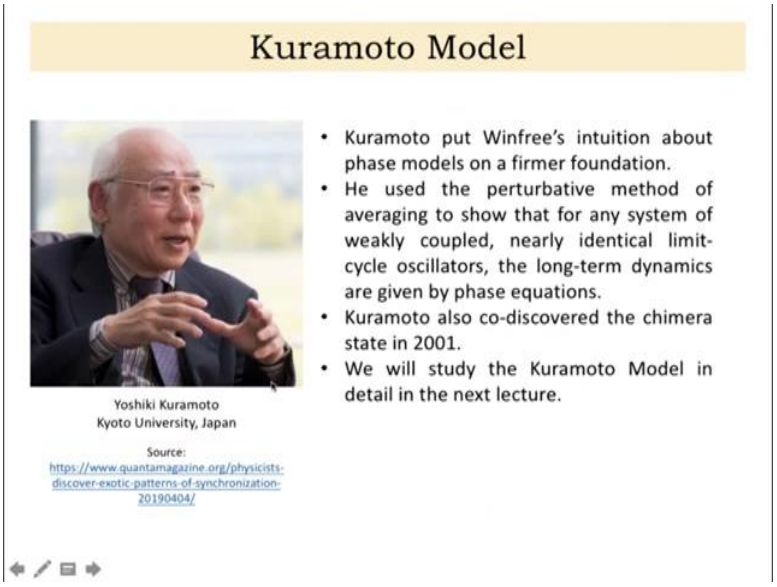

Now, Kuramoto the famous scientist from Kyoto University of Japan has actually expanded this. So, Kuramoto put this Winfree's intuition about phase models on a much firmer foundation.

And he used the perturbative method of averaging to show that for any system of weakly coupled, nearly identical limit cycle oscillators, there is long term dynamics which can be given by the phase equation itself. And that is something along with the Chimera state which you know actually make this pretty famous discovery.