Cognition and its Computation
Prof. Rajlakshmi Guha
Prof. Sharba Bandyopadhyay
Biotechnology and Bioengineering
Indian Institute of Technology, Kharagpur

Lecture - 19
Sensory Circuits: Auditory - I

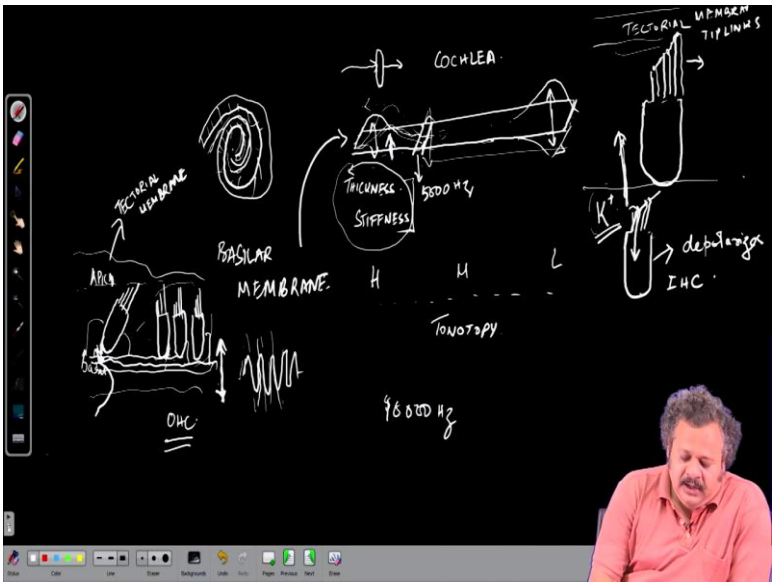
Welcome. So, we have been discussing the Sensory Circuits and we have covered the Visual System next we move on to the Auditory System to similarly get the basic tools needed in terms of understanding auditory object perception. We say auditory object, but in general it is very difficult to define an auditory object. So, we will say more an auditory perception or auditory scene analysis when we go into the cognitive aspects of this.

So, as we saw in the visual system the basic scheme of organization and hierarchical processing is that, at the very periphery we have receptive fields that are very small in size in terms of the visual field. And gradually as we go up it becomes more complicated and becomes larger in size in terms of the visual field. And we will later see that finally, based on these small features that are obtained in the initial stages at the cortex we will build the percept of a full object along the object recognition pathway.

So, similarly in the auditory system also we will start from the periphery where we will see that the receptive fields are small in the sense of the way the auditory system works. That is, they are they have narrow bandwidth instead of the visual fields physical size of a receptive field. In this case in the auditory case, it is more of the amount or the number of frequencies or the range of frequencies in the sound that a neuron is responsive to.

So, the and similarly there will be other features also that get integrated as we go along the auditory pathway. So, in the periphery the first stage where the transduction happens is in the cochlea in our inner ear, which is two and a half turns bony structure which is a spiral like so.

(Refer Slide Time: 03:10)



And this is tubular in nature and basically this is the region that is filled with fluid with three parallel chambers in there running through and that is where the main auditory transduction happens. So, the sound pressure waveform that impinges on to the ear drums that is the first stage of processing. That is transduced or transferred rather in this case through the middle ear bones on to the mechanical movement of in this fluid in the cochlear; in the cochlea.

We will not go too much into the details here. But what we will see here is that the cochlea in the middle has membranal structure. If we pull this out of here this membranal structure and stretched it out. We will see that it is flimsy in one end and very thick in the other end. And so, basically it has a general variation of thickness along the length of the basilar membrane.

So, the basilar membrane in the cochlea provides the basis for the transduction and it is it varies in thickness and stiffness along this basilar membrane. And the physical properties are such that the pressure that is being transferred into the fluid along an on top of the basilar membrane or in the cochlear tube in which the basilar membrane is present.

The coupling is such that the fluid pressure is or the frequency present in the fluid pressure changes causes resonances in the basilar membrane movement in the up and down direction. So, because of this varying thickness and stiffness the properties of the

basilar membrane is such that one end resonates at very high frequencies which is the front end and the other end resonates at the lowest frequencies.

So, in other words when high frequency is present then the basilar membrane fluctuations are high in one end and when low frequencies are present the basilar membranes fluctuations to the movement of the fluid is high on the other end. So, the this fluctuations that I have drawn is exaggerated, but the basic idea is that there is a coupling of the flow of the liquid or fluid in the basilar membrane to the resonance being caused in the vertical movement of the basilar membrane, ok.

And because of the varying mechanical properties of the basilar membrane different frequencies cause fluctuations in different regions along the vascular membrane. So, there is a gradual gradation of high frequency selectivity in the mechanical movement of the basilar membrane to low frequency and medium frequency. So, it is a gradual change in frequency along the basilar membrane.

So, this forms the basis of organization along the auditory pathway just like we have been talking about retinotopy in the visual pathway. This change in frequency selectivity that first starts at a mechanical level in the basilar membrane is called tonotopy. So, if you think about sounds that impinge on our ear drums they are not single frequencies and like this lecture that I am presenting if you record them and look at the fluctuations or the sound pressure waveform as a signal.

And then analyze its frequency content, we will find that very different kinds of frequencies are present at different times along the presentation of the sound or during this lecture or any sound that we hear. So, what the basilar membrane essentially does is that it breaks down the entire speech over different durations of time into the component frequencies in the sound that is impinging by the physical fluctuations at different locations on the basilar membrane.

And so, if let us say this region this particular point in the basilar membrane is selective to 5 the 5000 or 10000 hertz. That is if I present a 10000 hertz tone at a soft intensity. The basilar membrane will fluctuate the most at this particular region with its with it dying off the fluctuations dying off along this axis along the axis of the basilar membrane.

If I increase the amplitude of the sound make it louder. Then the fluctuations would increase and it would broaden also along the basilar membrane. And in that sense the intensity based broadening becomes a part of the properties of the neurons along the basilar membrane along the auditory pathway, sorry. So, now we have only converted sound pressure waveform into the fluctuations of the basilar membrane.

But remember we need an electrical signal to convey that same information into the auditory pathway. And so, this is done by inner ear cells or the particular types of cells that are present sitting on the basilar membrane. So, if we take one particular slice of the basilar membrane, we will see that there are cells of this nature with this kind of shape where this is the basal side and this is the apical side. And they have finger like stereo cilia structures sitting on top of them like this.

And so, this is let us say the basilar membrane. There are other supporting cells that hold them in place. And there is a row of inner outer ear cells that are three rows of outer ear cells that are also similar in shape with similar stereo cilia at the at the top. And this underlying here is the basilar membrane here with other supporting cells and everything present.

And on top of this is something that is called the tectorial membrane that is present throughout on the top region this is what we call the tectorial membrane another membranal structure. So, as we said the basilar membrane is fluctuating in this direction and this fluctuation is different along the length of the basilar membrane. We are considering one small strip here and let us say this region fluctuates the most when 5000 hertz signal is presented or tone is presented rather.

And so, this fluctuation that occurs this what this does is then the finger like structures that touch the tectorial membrane. Because of the pushing up of the basilar membrane shears with the shearing force these stereo cilia bend. So, if I oh draw a bigger picture like this. They are these finger like structures they are connected by narrow protein small proteins called tiplinks. They connect the tip of the stereo cilia from one to the other. And there is an ion channel that is present at the end of the tiplinks on top of the stereo cilia.

And this bending when there is a force pushing this on to the tectorial membrane here, with the basilar membrane here. If when the basilar membrane is fluctuating the stereo

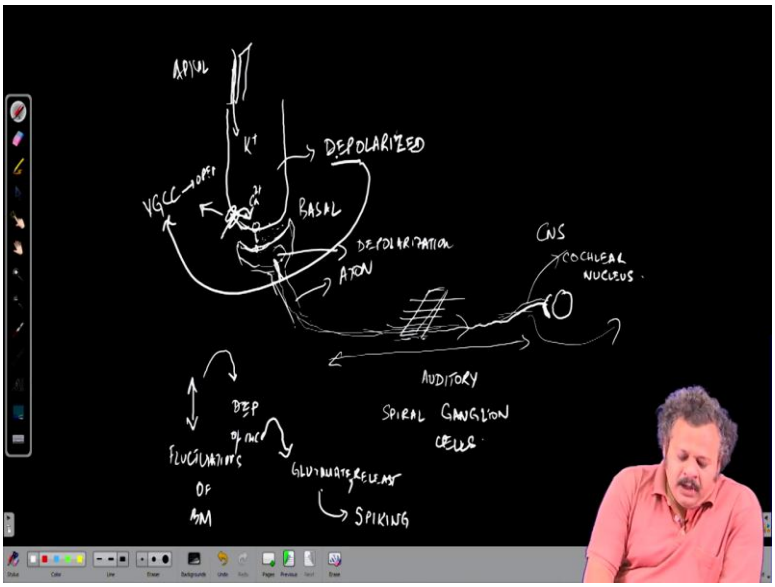
cilia bend over in this way and when it goes down it closes back then again closes back and this goes on. So, what that does is physically the causes pull on the tiplink and opens up an ion channel here.

So, when this is bent that opens up an ion channel physically when there is a bending. Because the tiplinks get pulled and the outside medium here is very high in potassium unlike the extracellular medium that we talked about in the usual neuron and extracellular space intracellular space and extracellular space. Instead, here potassium is very high concentration in the outside of the inner ear cells and outer ear cells.

So, what that causes is this pulling of the tiplinks causes potassium to come in. So, it is a mechanically gated ion channel not voltage gated. And because of the electrochemical gradient of potassium, potassium goes into the inner ear cell or outer ear cell like what whatever it is. But we are talking about the inner ear cell now. And what that does is depolarizes the inner ear cell IHC. What this does is now.

So, basically, we have got a an a physical motion of the basilar membrane correlated to that motion there is an increase or entry of potassium into the inner ear cell. So, we have got from the mechanical movement into an electrical signal which is the depolarization of the inner ear cell.

(Refer Slide Time: 16:40)



And so, what happens next is, if we think of the inner ear cell like this the this is the basal side and this is the apical side. With this entry of potassium this gets depolarized from its resting state. So, this depolarization causes voltage gated calcium channels that are present here. Similar to the pre synaptic terminal voltage gated calcium channels are present here that allows calcium to come in. So, this depolarization is causing the voltage gated calcium channels to open and that causes calcium entry into the inner ear cell in the basal side.

And so, there is a whole pre synaptic neurotransmitter release machinery present in the inner ear cell and the basal side. So, it is not actually a neuron, but it has what it takes to release neurotransmitter when depolarized. Because this voltage gated calcium channels open calcium comes in and through that same sort of mechanism that vesicles release neurotransmitter in the basal end outside from vesicles out into the extra ear region of the basal part of the inner ear cells.

And on the other side that is outside it actually a synapse like structure is found. In fact, it is a synapse, but it is different in some way is that there is machinery here that can cause that causes action potentials here if there is enough depolarization. So, glutamate in this case glutamate is the neurotransmitter that is released this glutamate goes here there are AMPA receptors of glutamate on the post synaptic side or rather the it is going to be the starting point of the auditory electrical auditory pathway.

The starting of the auditory pathway is from this particular synapse and it is not the other side is not really like the post synaptic neurons dendrite or so it is actually an axon here. Interestingly this is an axon. In the sense that, this axon goes and projects on to a neuron on the in the central nervous system in the auditory pathway. This region is the region of the auditory nerve or the 8 nerve which also contains the vestibular nerve.

And the cell bodies of these axons which are the spiral ganglion cells they are present in the middle in along this axons ganglion cells. And they do not participate in the spiking activity, the spike is happening with the glutamate release taken up by AMPA receptors, depolarization occurring in this region in the auditory nerve ear cell synapse and a spike is generated at this location in the in the axon.

Which then travels along the auditory nerve fiber to the synaptic terminal again which is like the synapse that we have known. That is instead of starting at a soma or the axon

hillock initial segment region in the soma or in the beginning of the axon. It is this particular region which is at the end at the one end of the axon where the spike initiation happens. Which is carried forward into the central nervous system the first stage being in the cochlear nucleus.

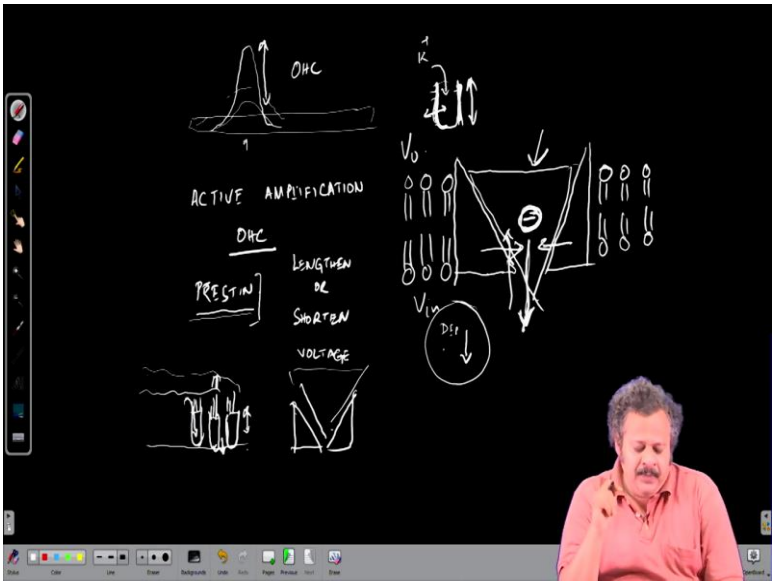
So, what we find now is that the movement or fluctuations of the basilar membrane causes. So, this is fluctuations of basilar membrane causes depolarization of inner ear cells which causes glutamate release through calcium in inflow, which then causes spiking depolarization in the synapse and spiking in the synapse on the auditory nerve ear cell synapse.

And this spike then travels along the axon fiber on to the neuron in the cochlear nucleus wherever it is supposed to project and there are collaterals in the sense that they can project to multiple neurons in the cochlear nucleus. So, just like in the visual system we had a particular point in the visual field that got transduced into spiking activity of the retinal ganglion neurons.

And then through the optic nerve the information or the spiking events carried on into the central nervous system that is in the lateral geniculate nucleus in the thalamus. In this case the movement are caused in the basilar membrane caused by impingement of sound is converted into spiking activity in the auditory nerve that is carried forward into the central nervous system. And in the auditory pathway the first stage is in the brain stem and in the particular structure called the cochlear nucleus.

So, this is the basis of auditory transduction. And in order to understand how auditory information is encoded in the auditory pathway and to know how the receptive fields are how what the properties of receptive fields of neurons in the cochlear nucleus and beyond r in neurons along the auditory pathway we must understand, what information is actually going in and into the central nervous system. And that is through our understanding of response properties of these auditory nerve fibers that are going into the central nervous system.

(Refer Slide Time: 24:54)



So, as we were talking earlier it is in the basilar membrane that we have fluctuations in a particular at a particular frequency which is most in one particular region. And as the intensity increases the fluctuation increases and the breadth of the region of the basilar membrane in that is excited or that starts moving more increases or broadens. These are the properties that are imparted on to the spiking activity of the auditory nerve fibers and that is the information that is being passed on into the auditory pathway.

So, an important aspect here as you might be wondering based on my earlier drawing of the basilar membrane with inner ear cells. So, here so, we have this end has the auditory nerve fiber that is carrying the information out into the central nervous system in the post synaptic side of the outer ear inner ear cell. So, the question naturally arises is what is the what is the role of the outer ear cells?

Auditory nerve fibers that carry information into the auditory pathway they are not similar they are not inner waiting the outer ear cells in the same way. So, the outer ear cells are present for a particular region and that is basically to provide active amplification. That is done by the outer ear cells. And this is a very interesting way in which it provides this amplification and what we know now about this is that there is a particular protein called prestin that was discovered couple of decades ago or maybe around yeah 20 25 years ago.

And now we know more about this protein and what role it plays. And what we know is that the outer ear cells because of this protein prestin have a unique property and that is

being able to having the ability of being able to lengthen or shorten based on the voltage. So, the way in which the inner ear cells got depolarized due to the inflow of potassium on the bending of the stereo cilia.

We similarly have a depolarization of the outer ear cells as well with the flow of potassium in there. And what this protein prestin which is on the membrane along the wall of the outer ear cells this protein itself can become shorter or longer depending on the voltage the that is the extracellular and intracellular potential here. So, it is kind of like this that if you remember the membrane is this lipid bilayer and sort of a working kind of model of this action is that it is we have the prestin protein is almost like this.

That we have something that is merged in and when the inside is depolarized it is in this location that is the let us say this has a large minus charge on a. So, when the inside is depolarized this particular component of the protein is pulled in due to the negative charge here. And that is when at rest that is when the potential here goes down to the resting state this electro electrical force that is pulling this particular segment in the into the neuron into the in intracellular side is actually pushed back out. And in pushing back out it actually pulls in the outer segments.

And so, if you think of it in this way with this pushing pulling back in or shortening it is getting close it is now taking up less space in the membrane. So, effectively it is reducing the space in the membrane and so it is pull along with it is pulling the membrane to be shorter. And so, this length can be manipulated if you have many such proteins along this wall here.

So, they are coming closer and going further these segments is actually going to lengthen and shorten the length of the outer ear cell. And this is sort of how we can explain the working of the protein prestin and how based on the voltage here that is V in minus V out we can have shortening and lengthening of the outer ear cell. So, what this now does is that, if the tectorial membrane is lying above here and we have the outer ear cells that are sitting on the basilar membrane.

When it lengthens or shortens because of the potential what it does is it actively pushes onto the membrane here and here when it extends itself. So, it is actually pushing adding this energy based on or rather adding this fluctuation based on the depolarization of the

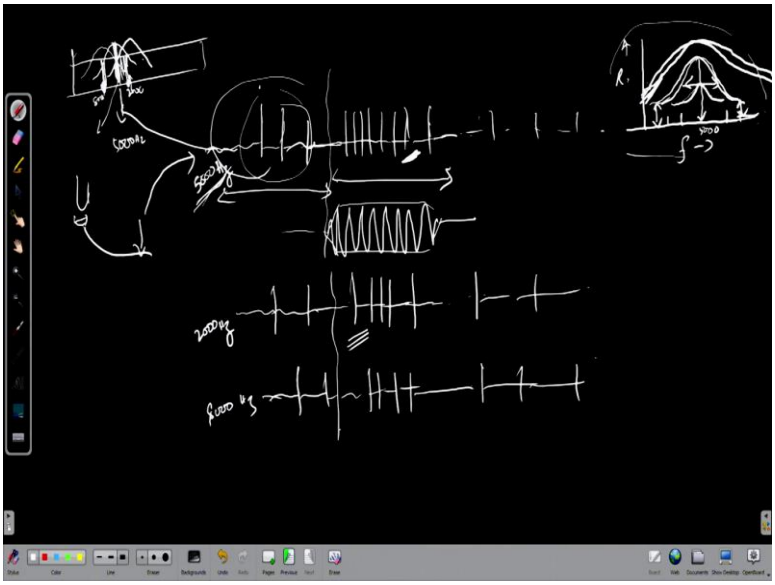
neuron on top of the fluctuation produced by the sound itself or the tone frequency that is causing the fluctuation itself.

So, the basilar membrane starts to move because of the sound, but at the same time or within a very short period of time because of the depolarization of the outer ear cells it actively amplifies this fluctuation because of its lengthening and shortening. So, what that does is it actively increases this kind of fluctuation of the basilar membrane. Of course, this drawing is exaggerated, but when we will look at the response properties of the auditory nerve fibers when we discuss the response how the response changes with intensity and so on.

We will see that the outer ear cell provides a very very critical role or plays a very very critical role in determining how sensitive we are in terms of our hearing. So, what if the outer ear cell is damaged what usually happens is most of this active amplification is gone. And so, as you can imagine if this active amplification is gone that essentially means that you have you are changing the transduction that is going to happen in the inner ear cells.

And so, the spiking events that are going to happen in the axon or in the auditory nerve fibers that project that are starting their path from the basal end of the inner ear cell though a spiking activity is going to change completely. So, if we go on into the response properties of the auditory nerve fibers.

(Refer Slide Time: 34:42)



What we will find is that if we are recording from one particular auditory nerve fiber, we can have spontaneous activity during the baseline. And let us say we play a particular tone stimulus in this region. Let us say the 5000 hertz stimulus is played. If we remember our basilar membrane on this side which means that this is the 5000 hertz region that is making this basilar membrane fluctuate here the most.

And the auditory nerve fiber that is project that is starting at the inner ear cell at this particular location we are recording from that and that is being shown here the spiking activity of that neuron that fiber is shown here. And with the stimulus we see number of spikes happening and then it stops and then it goes back to its spontaneous activity. So, similarly you can think of another ear cell that is inner ear cell that is slightly away or at a lower a higher frequency that is let us say 8000 hertz or let us say 2000 hertz.

They are fluctuating less and so they are going to have. So, this is the auditory nerve fiber that is located at the basilar membrane relocation where it is sensitive most to 5000 hertz. Let us say this is the example of the 2000 hertz regions in our ear cell connected axon and similarly the 8000 hertz in this case. So, all let us say have some spontaneous activity like so. And when this sound is being played this will also have increased firing or number of spikes. But not as much as in the case of the this particular fiber which is at 5 kilohertz.

So, this neuron the auditory nerve fiber starting from here and the auditory nerve fiber starting from here are also firing because there are fluctuations there, but lesser fluctuations compared to what is happening at 5 kilohertz. So, they are going to have lesser number of spikes, but more than spontaneous to say that they are also responding. So, similarly we can think of it in this way that if I play a 8000 hertz stone.

Now, this particular fiber will respond more like this particular neuron. And the 5000 hertz fiber is now going to respond more like the 2000 hertz auditory nerve fiber. That is when we are playing let us say 8 kilohertz that is 8000 hertz.

Now, this is the fluctuation and so the same fiber that is selective to 5 kilohertz will have lesser responses. And similarly, when I play 2 kilohertz or 2000 hertz the fluctuations are like this and so that original fiber that we were talking about that is going to respond less now compared to what it was doing at 5 kilo hertz.

So, if we play different frequencies this is the frequency axis and compute the rate of spiking activity in the auditory nerve fiber in one particular auditory nerve fiber rate then we will get something like a frequency tuning. That is, it responds most in this case to 5000 hertz and its response dies off as frequency changes and further out it is basically at the spontaneous rate that is the rate observed in baseline.

And so, they have a particular bandwidth and as we were saying this intensity of this 5 kilohertz or 8 kilohertz or 2 kilohertz or 1 kilohertz or 12 kilohertz whatever we are playing and recording from auditory nerve fibers that is going to vary and determine basically the response of the auditory nerve fiber and the bandwidth of the fiber. And the lower the intensity again it becomes narrower and as we increase the intensity the responses the it becomes broader.

And then ultimately, they will saturate at some point. So, we will continue this discussion of bandwidths and tuning in the context of the central nervous system more when we discuss how the information that is conveyed by the auditory nerve is carried forward into the auditory pathway. So, with this we will stop at this lecture and then continue further with the auditory nerve and further information about the central nervous system aspects of the auditory processing and the auditory hierarchy.

Thank you.