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

Lecture - 29
Monosynaptic Reflexes Part 3

So, welcome to this class on monosynaptic and other reflexes. So, most of this is going to be some Monosynaptic Reflexes.

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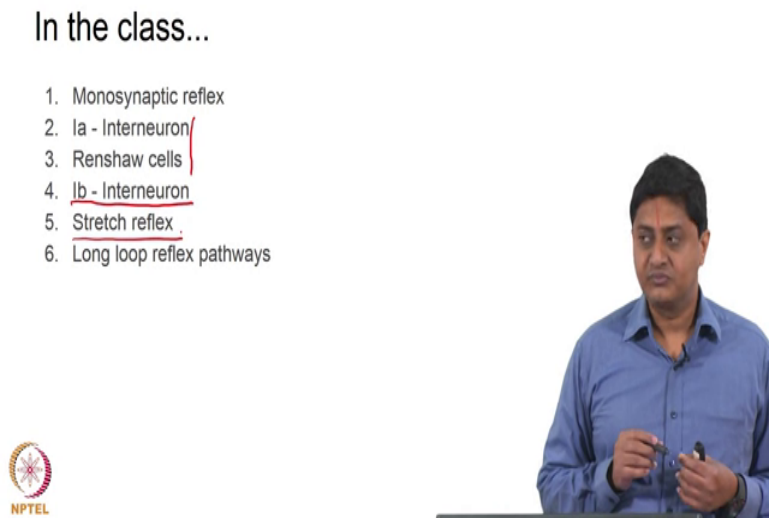

So, we have been discussing in the last two classes the case of monosynaptic reflexes; in today's class we will discuss two other things. We have introduced these notions earlier the concept of a 1 a interneuron and the special inhibitory interneuron in the motor circuit, this is the Renshaw cell. And in today's class we will talk about 1 b interneuron, we will talk about stretch reflex alpha gamma co activation, how these reflexes can also be modulated by descending influence are those that involve brain circuits or what are also called as long loop reflexes.

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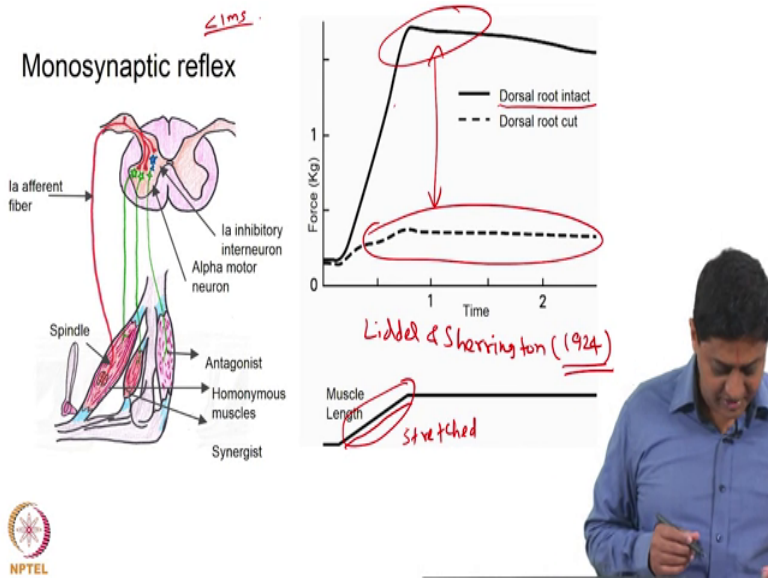

Let us remind ourselves of this case of the monosynaptic reflex, what does this do? This resists the lengthening of a given muscle. So, if a muscle is stretched or lengthened, the muscle spindles one a afferents fire and cause contraction or they excite the alpha motor neurons of the same muscle and cause contraction or prevent stretching in some sense. For a long time for centuries it was thought, that this is actually a property of the muscle, this is like a passive property this is not related to control for a long time this was believed.

Only just about 100 years ago, it was found that if you transect the dorsal roots right, if you transect the dorsal roots this response disappears. So, if you cut the dorsal root what happens is, you there is a big difference between the dorsal root intact. When the dorsal root is intact this is what is found right and when the dorsal root is cut this is what is found, big difference. So, this means that the dorsal root is absolutely necessary for this function, this was first and as the muscle length is changing. So, the muscle length is increasing. So, the muscle length is or the muscle is stretched.

As the muscle is stretched in the presence of an intact dorsal root, you are going to have relatively strong response such as that, if the dorsal root is cut even though there is stretching even though there is a stretching, the response is going to be relatively weak. This means that dorsal root is essential at least forms an essential part of this from Liddel

and Sherrington you have to see the year just to give you a context is 1924. So, this is data from 1924.

From Liddle and Sherrington for the first time showing that you know the information coming from the dorsal root plays an important, crucial role in modulating the reflex responses. So, this means that so, that is how we realize the importance of 1 a of and so, there was even a question, as to how do you know that this is how it works. Many of these things are not seen the neurons, are not seeing the these are just pictorial representations.

So, this picture for example, this is a pictorial representation of our impression of what is going on. What is actually going on is not exactly seen, but understood through latencies. This is classic neurophysiology, understood through latencies. The if you excite only a afferent at the dorsal root, the response at the ventral root of corresponding alpha motor neurons is found at approximately a slightly less than 1 millisecond.

And since, we know that the synaptic transmission it about 0.5 milliseconds to 0.8 or 0.9 milliseconds; we know this must essentially involved a monosynaptic pathway. So, many of these things are found through accurate measurements of latencies, by computing these latencies we are able to come up with how many synapses are involved in this process. So, this is how it is done. So, what is the use of all these reflexes? So, slight deviation from the topic, what is the use of all these reflexes. You see reflexes provide in some sense a crude response to a stimulus, right it need not be you know perfectly acceptable to the context, but it provides a response. In many cases it is performing some protective function.

In other words it is for example if there is a painful stimulus; let us assume that this is a you know nail and I am placing my finger on it and this is a hot object and I am placing my finger on it right. If it is only slightly hot I am going to place and you know withdraw, note the point at which the sensory information is collected is at this point which is the finger. It is not the finger alone that is withdrawing you see the response is found here, is it not?

The response is found at proximal more proximal size, leading to a relatively quick withdrawal from the painful stimulus. If it was very hard let us assume that it was very hard, actually many more muscles are activated leading to a complete withdrawal like for

example; the response could be like that. Of course this a this particular example that I am giving in obviously, involves polysynaptic pathways, it is not monosynaptic. In other words this response is activating circuitry that activates multiple muscles is it not? Because the activation involves you know withdrawal at the elbow at the shoulder at the wrist etcetera multiple joints; that means, multiple muscles must be activated.

Obviously it involves polysynaptic pathways not monosynaptic that is, but still we should remember that this is also considered a reflux or this is also called as the flexion withdrawal response right. So that means, it also involves coordination of multiple muscles, it does two things. First is it activates the flexors or it and inhibits the extensors. So, there is simultaneous activation of one group of muscles and inhibition of the other group of muscles not just that there is more. Suppose this painful stimulus is on my leg on my right leg say I am walking and there is a nail and I am let us say that I am not wearing a footwear I mean in my home or whatever, I am not wearing a footwear I am walking that is a painful stimulus right?

What happens is that you know immediately I withdraw the leg by flexing the knee flexing at the knee, but; that means, the extensors are inhibited extensors are inhibited flexors are activated. What happens that is at the right leg is it not? Because the painful stimulus is felt at the right leg what happens if the left leg is the opposite? At the left leg what happens is the extensors are activated and the flexors are inhibited why? Because I have only two legs if I remove the right leg the entire load of my body the entire weight of my body is going to be rested with just one leg. So, I should ensure that that is going to get the maximum support that is possible by ensuring that the extensors are activated. So, let s remember what is going as it is a bit more complicated than what is shown here.

So, what is happening is simultaneous activation of flexors and inhibition of extensors of the limb that is experiencing the painful stimulus, and this is called as flexors withdrawal response a polysynaptic response. The other case is the crossed extension reflex in which case you know the other limb the limb on the other side of the body experiences you know extension more details on this just wanted to give why these things are even being studied just wanted to talk about that a little bit.

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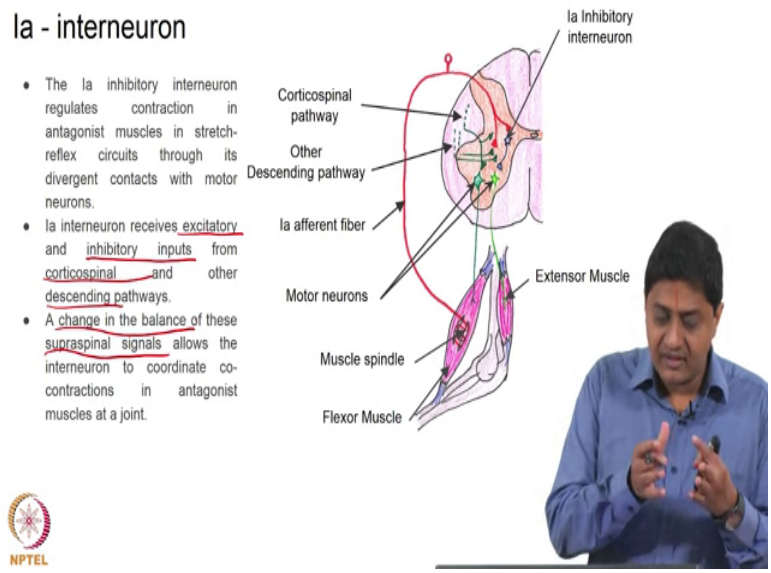

And let us remind ourselves of what the one a inhibitory interneuron does. So, the one a inhibitory interneuron is activated is excited by the one a afferent, and is inhibited by the Renshaw cell ok. In what it does is it regulates contraction in the antagonist muscle. So, if the muscle of interest is biceps it one a inhibitory interneuron that receives inputs from the biceps, regulates contraction of the triceps of the antagonist muscle.

So, let us also remember there are. So, it receives multiple inputs not just from I have only mentioned two sources right not just from the Renshaw cell, and not just from one a afferent it also receives inputs from the descending system. And that can both be excitatory and inhibitory from corticospinal and other descending pathways. So, there are multiple inputs. So, but there is only a single one a inter neuron right are there are groups of this one a interneuron.

So, these one a interneurons there must be a balance of excitation and inhibition. Depending on in which direction this balance is steep it could be tipped in favour of excitation or it could be tipped in favour of inhibition. Depending on that excitation or inhibition of you know of the muscles innervated by this of the neurons that are affected by this will happen. So, this balance must change.

So, a change in the balance allows the interneuron to coordinate muscle control. So, this is like you know coordination happens through multiple balance of multiple pathways. So, from one a often and from Renshaw cells from corticospinal pathways, and let us

also remember we discussed some time ago monomenergic inputs from the brainstem etcetera etcetera. All these things are received by the way monomenerigic inputs go to the motor neuronal pool, but the motor neuronal pool itself is inhibited by one a inhibitory interneuron.

So, a lots of things all these things; so, there is there is a just a balance that is reached depending on what the balance is depending on where the equilibrium is settled at a given point in time, the response will be determined based on that.

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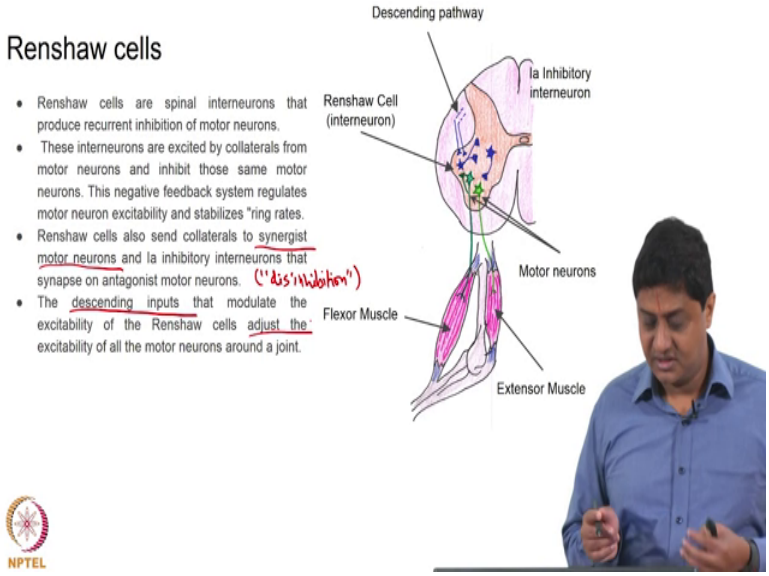

And we discuss the, what are these Renshaw cells. Renshaw cells are inter neurons that produce inhibition of the neuron that neuron that excites it, and also the motor neuronal pool of the synergies right. So, if biceps is contracting its Renshaw cell, we seen will inhibit biceps braqualis and braque radials all its a say just and their gamma motor neurons ok. These interneorons are excitable so, that means they are excited by collaterals from several motor neurons right.

So, by the way let us also remember when braqualism braque radials are contracting, it will inhibit everybody it will inhibit braqualis braque radialis and biceps. So, it will inhibit all the synergistic muscles. They also so, they send you know collaterals, also synergies motor neurons and they also inhibit one a inhibitory interneurons of the antagonists.

So, let us remember one a inhibitory interneurons inhibit the function. So, causing what we discussed this earlier we call this as dis inhibition. Or in the way if they excite or they increase the probability that the antigonish muscle is going to get excited another form of negative feedback. We saw this earlier they also receive the inputs from the descending systems, they also Renshaw cells also receive this inputs from the descending system. So, their excitability are modulated. So, can. So, it is possible to adjust the excitable the probability that the Renshaw cell will be excited.

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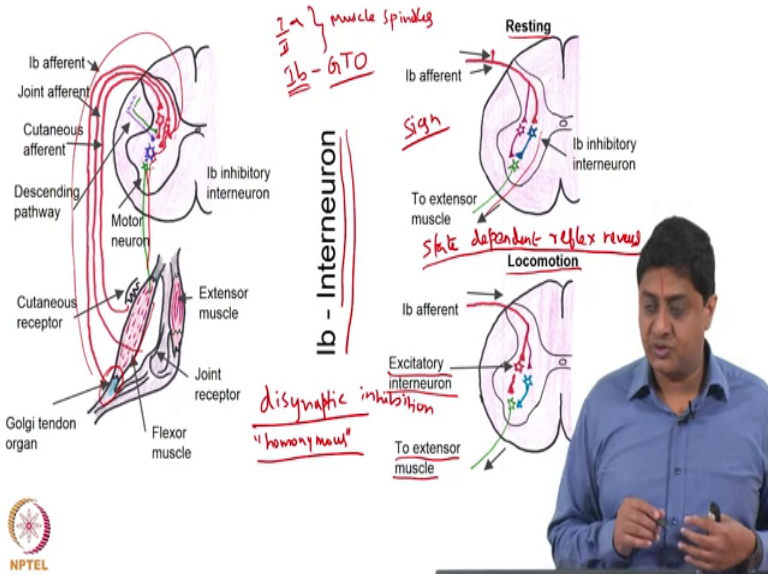

And what is not discussed is the case of 1 b interneuron 1 b inter neuron let us remind ourselves what are these cases 1 a and 2 are muscle spindles, and 1 b is coming from Golgi tendon organs when will this be activated. Whenever, muscle is contracting; if a muscle is contracting the tension in the tendon is going to increase, when the tension in the tendon is going to increase the Golgi tendon argon is going to get activated and send proportional inputs. In through its afferent and its afferent is called as 1 b neuron is it not are called as 1 b afferent this one be afferent.

So; that means, what that muscle is contracting. Then its negative feedback must be to relax that muscle is it not; that means, I should inhibit the alpha motor neuron that excites that muscle let us consider a single muscle let us considers a the biceps right. If biceps is contracting its 1 b afferent we will find a way to must find a way to inhibit the

alpha motor neurons that contract the biceps, but let us remember that all proprioceptors by definition are excitatory they always excite.

So that means, I cannot directly inhibit the alpha motor neurons. So, what this does is that there is an inter neuron there is an interneuron which is called as the 1 b inter neuron. So, the 1 b afferent excites a 1 b interneuron, which inhibits the alpha motor neuron of the same muscle. So, unlike the 1 a case in the 1 a case stretching activates the 1 a afferent and it exits the alpha motor neuron of the same muscle causing it to contract that is a monosynaptic response.

In this case contraction activates 1 b afferent and we have to inhibit the alpha motor neuron for that to happen it cannot do it directly it activates 1 b interneuron, which inhibits an alpha motor neuron. So, this is a die synoptic pathway and so; that means, this die synaptic pathway is inhibiting die synaptic inhibition of which muscle.

Not the opposite muscle inhibition of the not the inhibition of the opposite muscle is performed by the one a system inhibition of the same muscle are homonymous same muscle is happening, same muscle is inhibited ok. And, but in, but the one being inter neuron is receiving inputs not just from 1 b afferent, but also from other sources. There is a great amount of convergence of a information that comes from various other sources, leading to flexibility of responses please do read about this. So, here is the example. So, there is a the Golgi tendon organ here that is detecting the contraction of this muscle. So, you want to you know relax this muscle.

So, that is achieved through. So, that is the. So, through an inter neuron that is inhibiting this and you know the alpha motor neuron is inhibited ok. Now, what also happens is the special case of a locomotion versus resting. I said that this system 1 b system receives inputs not just from the 1 b afferent, but also from multiple sources. It turns out that depending on the state the response can vary.

What actually happens during rest between rest and locomotion of a of a healthy animal healthy mammal say a cat is walking say for example. At rest the 1 b afferent causes inhibition of the homonymous muscle as one would as expect right. But during locomotion what happens is that, there are others who come into the picture there are other excitatory interneurons that come into the picture, that cause the same 1 b system to or the 1 b afferent to cause an excitation in the extensor.

So, this is called as this phenomenon has been widely studied and lots of data available on that, this is called as the state dependent reflex reversal. So, the sign of the reflex can be changed depending on the context. If I am lying down or if I am sitting in a chair right the reflex response is going to be something. Where as if I am walking for the same stimulus the response is going to be of the opposite sign as in s i g n sign is going to be in the opposite direction, this is called as the state dependent reflex reversal especially is shown in the 1 b system.

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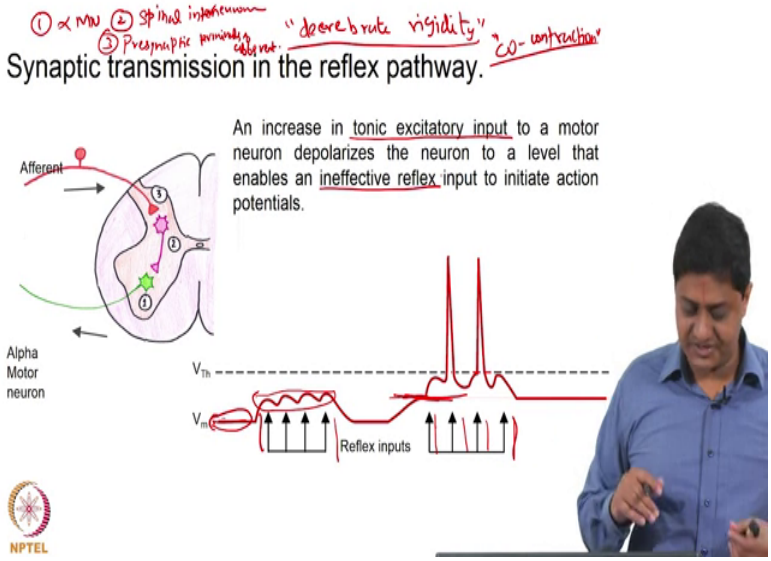

Please do read about this discuss how are these things even. Studied how to study this Sherrington invented a method to study these things he called this we will discuss the various preparations of animals that are studied in a future class. But the however, I will just mentioned that Sherrington invented what was concern at that time a novel method to study the stretch reflexes.

It turns out that if you transect the brainstem between the superior colliculus and the inferior colliculus. So, at the at the mid at the level of the midbrain between the superior and the inferior colliculus if you cut the system, then what happens is the reflex responses or the reflex input inputs provided by the brainstem are usually heightened. So, causing an amount of great amount of extensor tone, or an amount of rigidity called as decrebrute rigidity. Even with the intact animal you do not have so, much extensor muscle tone, but when the current is made at that particular level between superior

colliculus and the inferior colliculus, you will see that there is a great amount of extensor muscle tone that is that is sufficient for the animal to support its body weight. Remember this animals in this animal has been cut from its brain from its cortex from its motor cortex.

So, you would expect that it will go limp that is not what happens? Its body weight can be supported by this muscle tone that is produced from the brain top this rigidity Sherrington called greatly as the famously as the decrebrute, not is decerebrute with a e. Google this I mean definitely Google there is a nice any beautiful paper in which he discusses this is also wonderful to read his language of a, how he describes the situations. So, in that case what happens the reflex responses are heightened. So, then you can study you can modulate, you can you can give specific stimuli and study the responses, the responses are going to be of a higher amplitude than what would find in an intact anyone. This was the invention made by Sherrington way back about 100 years about for this.

So, again and one again an experimental approach more more details and other such preparations in future class in one particular future class ok. Another thing that is of interest for us is not just this that is more; you know it is not only inhibition of the opposite muscle that is always desirable. Let us say that you know I am making a particular movement, I want to excite the you know muscle that is responsible and I want to inhibit the muscle that performs the opposite mechanical action, this is what you would usually consider.

Yes that is an efficient approach, yet there are times when you want to increase the stability or stiffness. In such cases what happens is that you are interested in contracting simultaneously both the agonist and the antagonist, thus increasing the apparent stiffness at that particular joint right. If I do that then what happens well this is something that you could experience. So, you know I want to ensure that suppose I am keeping my arm like that and I want to ensure that nobody can you know push me away then what do I do? I activate a whole number of muscles both the agonist and the antagonist on both sides. When I do that if there is a disturbance I m going to come back to that you know the original state.

So, or we want to reduce the probability that we are going to avoid a perturbation, something like suppose I am carrying this cup let us suppose this is just a glass, and I do

not want to spill the hot liquid in that, but because it could it is it is embarrassing and it is also dangerous if it is hot enough it could cause injury for me is it not. So, I want to do that. If I am walking without the cup if I am just walking without the cup art with the pin say for example, I am not going to have a high tend coconut level or I am going to have relatively more activation of the muscles that is responsible for that moment, whereas, in this case that is going to be a greater amount of co contraction he; that means, we should suspend the reflex responses and activate the other muscles.

So, this is again possible so; that means, co contraction right. This is again possible through descending inputs let us remember. So, whenever I say descending inputs where are these things coming from? These are coming from actually what are the various sites in which you could modulate the you know the reflex responses? There are only three places in which you could modulate the reflex responses with not.

First is first is the alpha motor neuron, second is the spinal inter neurons actually there are too many of them and the third importantly is the presynaptic terminal of the afferent neurons. Now, let us remind ourselves of what this presynaptic businesses is. You know if I would like to have an inhibition right, it is possible for me to cause this inhibition postsynaptically or presynaptically with not. Postsynaptic inhibition is regular postsynaptic inhibition that is caused by inhibitory neurotransmitters etcetera etcetera.

And we saw also saw case of you know inhibition that is caused through presynaptic method we discussed that earlier. This is like having two knobs right gross or we as we see in some of the instruments that we operate; gross adjustment of gain and then fine adjustment of gain the. So, inhibition can be performed at a gross level for multiple sites using a postsynaptic method or selectively for specific neurons using are fine adjustments using the presynaptic approach all these three are possible.

Now, I could change the response to reflex by modifying the tonic excitatory state. If the tonic membrane potential tonic excitatory state is at that level for a given reflex amplitude it is going to take the membrane to that level of this, and that is going to be no response or response depending on what the threshold is.

Now, if I change the descending input to take the membrane potential to that state alright and I supply the same amplitude reflex or the same amplitude stimulus, that causes the reflex that could take the system to action potential and cause a response that is you

know measured. So, depending on the depending on the where the resting state is depending on whether the resting state is here or here, the response will vary and the stimulus let us remember the stimulus remains a constant the stimulus is the same.

For the same stimulus depending on the excitatory inputs coming from the top coming from elsewhere the response can vary. So, with that this is something that the you know the higher centers control can control possibly control. The reflex response is ineffective, then we can make it effective by changing the excitatory tone that is are that is already present in the system right so, that is one.

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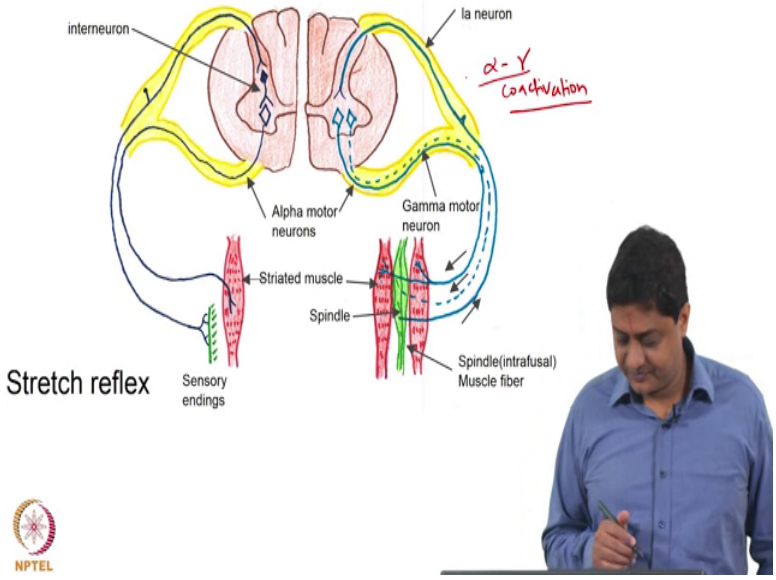

And let us also remember one more thing that whenever the alpha motor neuron is activated, the gamma motor neurons are also simultaneously activated. We said that and we call this as the alpha gamma co activation. The earlier co activation that I spoke just a few minutes ago is the co contraction of two different muscles this is alpha gamma co activation, where fibers of the same muscle extrafusal fibers and the intrafusal fibers of the same muscle are simultaneously activated. This is this we called as the alpha gamma co activation.

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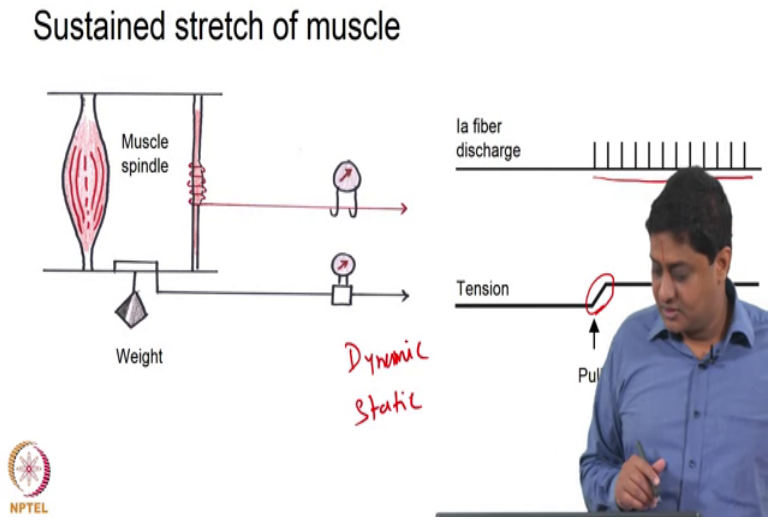

Now, let us remember what happens, we discussed this earlier the function of the muscle spindle is it not. When the fiber is stimulated right when the one year afferent or muscle spindle is stimulated and there is a stretch, that is are it could either be mechanically stimulated or could be electrically stimulated or when a stretch is cause like that there's going to be sustained response. And we are let us also remember that there is both dynamic sensitivity that is adjusted by changing the input to the dynamic gamma motor neuron and static sensitivity.

That is a so, depending on how I change the inputs to the dynamic gamma motor neuron and the static gamma motor neuron, sensitivity of the 1 year system the 1 year system can either function as a velocity sensor or a length sensor is it not.

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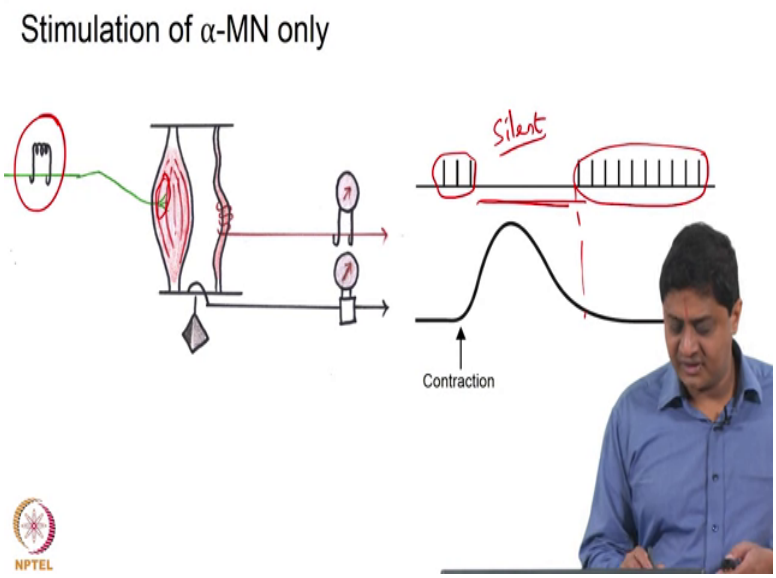

So, that is the response now, suppose I am contracting the muscle I am contacting the muscle. Now, what will happen in the absence of gamma motor input right I am causing a contraction of the muscle through electrical stimulation, and I am activating only the. So; that means, only the alpha motor neuron is activated only the if the alpha motor neuron is activated what you will see is, some length being measured and the length being measured after the contraction is over.

During the contraction this is silent why is this silent? Because the static sensitivity has not been adjusted the gamma motor neurons responsible for changing the static sensitivity, have not been activated. Because of this reason this remains silent if I want to still measure length what I should do?

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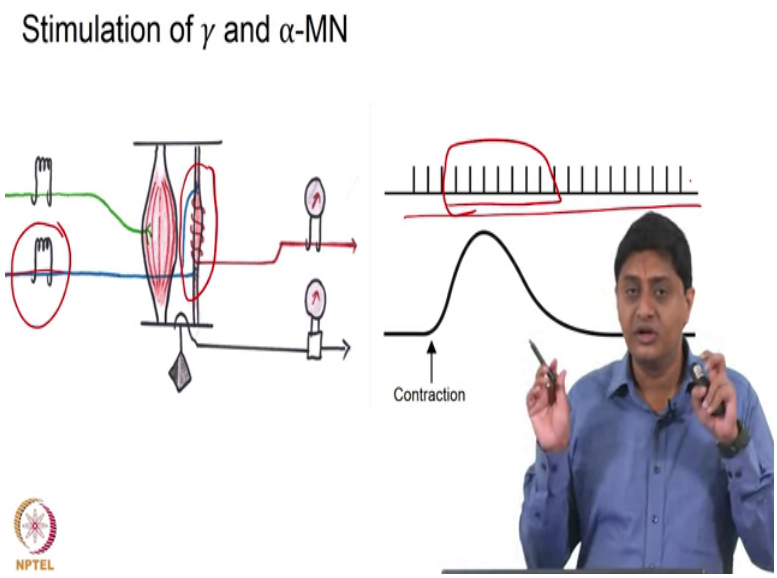

I could activate both alpha motor neuron and the gamma motor neuron. Earlier you see only the alpha motor neuron, that is activating the extra fissile fibers. Here simultaneous activation or simultaneous stimulation of both the alpha motor neuron and the gamma motor neuron both the extrafusal fibers and the intrafusal fibers. When both are done what you see as a difference from the previous picture here it is silent whereas, here it is still measuring length. So, experimentally we have confirmed the important role of the gamma motor neurons and something to remember ok.

I could adjust the sensitivity by so, but if I a simultaneous active activate some alpha and gamma motor neurons, I am going to get a response like this which is considered normal response, this happens only in experimental situations is it not.

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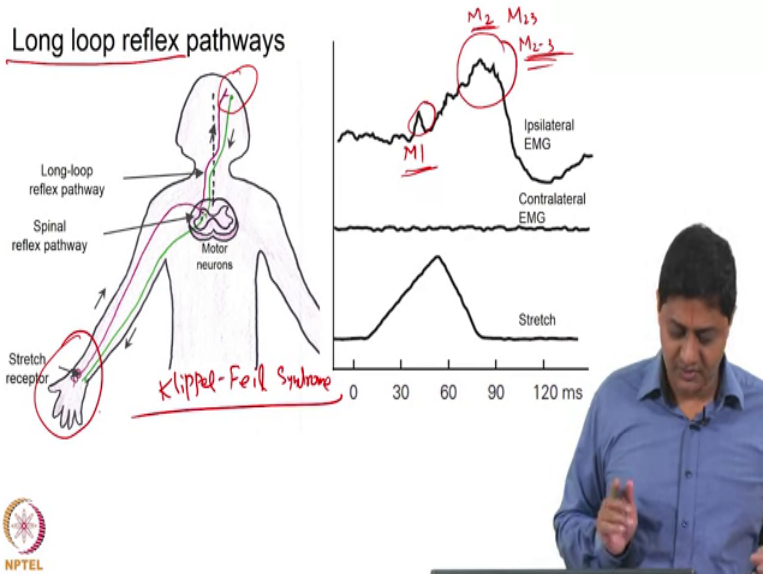

Now, another case is the case of long loop reflexes the case of long loop reflexes. Now what is this? This must somehow involve the higher brain centers we will discuss what this higher brain centers are later. But for now let us just say that there is response that is measured at relatively short latencies, which we are going to call as a for want of better terminology as M 1 now what is this M 1? If the muscle is stretched there is going to be an immediate monosynaptic response to contact that muscle through mediated by the one a afferent system.

So, one a afferent activates the alpha motor neuron and that response is formed say for example, this is what we call as H reflex. But if it also continues right there is going to be an improved response a more contextual response, that is modulated from by inputs from the higher centers which is called as M 2, or M 223 or M 2 hyphen 3 etcetera etcetera lots of names. So, let us not confuse this with the earlier terminology, earlier we called the earlier n first response monosynaptic response as h reflex if it is electrically stimulated right.

And M response is the response that is directly coming through the motor neurons m response stands for M for motor neuron; direct activation of motor neuron causes m response. Here when there are multiple muscle responses the first response is called as M 1 monosynaptic response M 23 or M 2 is a polysynaptic are a long loop response ok. The

say for example, the right hand of this individual is controlled by the left the contralateral see the other side the left motor cortex etcetera so, the input is coming from.

The example that is given is what would happen in people with specific disorders where this function this integrity is compromised; please do check this not I am not going to explain this please do check this syndrome, cripple failed syndrome. Please Google this and see what will happen if you activate one if you stretch one particular one particular hand say the right hand muscle is stretched, M 1 will be present in the response of in the response of the right hand M 23 will be present in the response of both hands; why this happens please do check about that.

By this so, again once again by carefully stimulating individual systems or individual neurons or individual or groups of neurons and measuring simultaneously which of course, synced; responses at multiple sizes we are able to come up with hypotheses and where the pathology is. This is classical neurophysiology this is how it is done. So, please do read about this please do Google this and read about this yourself.

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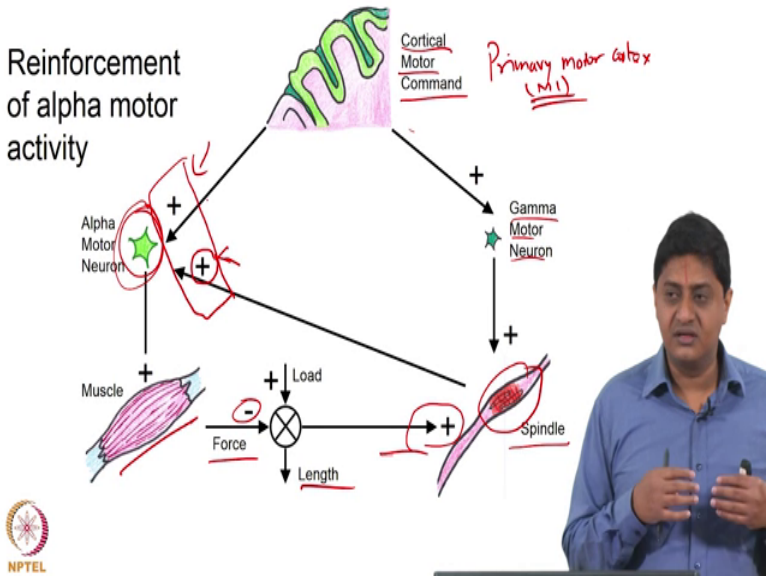

So, one more thing so, let us also remember remind ourselves of the total of the, of what is going on. So, alpha motor neuronal activity is if this is the alpha motor neuronal activity, it is going to get excitement from the cortical system or unfortunately this also has the same name primary motor cortex, which in future classes I will call as M 1. It also controls gamma motor neurons which change as sensitivity of the spindle is it not.

So, the external load if it is stretching right force produced overcomes that, and the length information is measured by the spindle which activates this. So, this activation of the alpha motor neuron is not just through this, or just through this its actually through a combination of both these pluses is, it not so, let us see what happens is it not?

In the case of reflexes it is only this place that is acting. In the case of real everyday movements what is actually going on in real everyday movements is a combination of this. But you are not observing, which one of these is contributing how much. So, we are only observing the net output you are only observing the activity here, you are only observing the activity of the length, you are only observing the force that is produced. But it is produced by a combination of inputs how much of this is due to reflex? How much of this is due to what component is due to what cause is an important question, that is continues to be researched at multiple levels.

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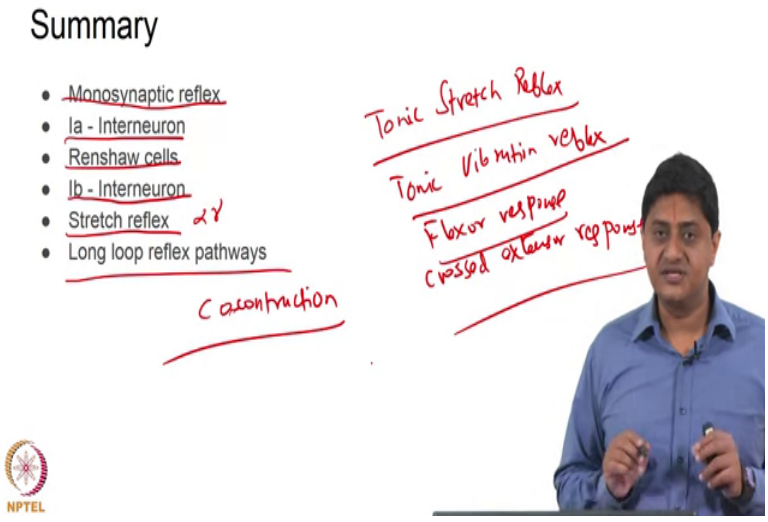

So, in summary we have seen monosynaptic reflex, we have reviewed that we have seen the case of 1 a interneuron we have reviewed the case of Renshaw cells inhibitory interneuron and the 1 b interneuron, that causes a inhibition of the homonymous muscle.

And, also receives input from multiple sides the case of stretch reflex and alpha gamma co activation, and long loop reflex pathways. We have also introduced the topic of flexor withdrawal reflex and crossed extinction reflex and we have discussed the concept of co contraction ok. We have still not done co hyphen construction ok. We have still not done

the case of a oligosynaptic and polysynaptic reflexes the cases that are called as tonic or tonic stretch reflex.

And what is it how what is the difference between the regular stretch reflex and that tonic, stretch reflex what is tonic about the tonic stretch reflex and tonic vibration reflex. We had to discuss once again the case of flexor response and crossed extensor response. These we will do in future classes.

So, with this we come to the end of this class.

Thank you very much.