Basics of Biology
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Module - IX : Human Physiology (Part-II)
Lecture – 42
Muscular System (Part-II)

Hello everyone, this is Dr. Vishal Trivedi from Department of Biosciences and Bioengineering, IIT Guwahati. And what we were discussing, we were discussing about the different properties of the living organisms, and in this context, in this particular module, we are discussing about the locomotion or the in general, we are discussing about the muscular system.

So, in previous module, what we have discussed? We have discussed about the digestions and digestion is required, so that it actually can convert a complex food into a less complex nutrients and that nutrients can be absorbed by the body, so that it actually going to provide the energy.

Once you are going to have the energy from the nutrients, you can actually be able to utilize for running the different types of physiological processes. You can use that energy to run the circulatory system, you can run that energy to for the muscular system, so that you can be able to use that for locomotion, and you can use that for moving from the one place to another place. And apart from that, we also have the different types of physiological processes where you can actually be able to utilize that energy.

So, in the previous lecture, while we were discussing about the muscular system, we have discussed about the, what is the requirement of the muscular system? and how the muscular system is actually going to help the organism to move from the one place to another place? In that context, we have also discussed about the different types of muscles which are responsible for providing the, providing the different types of locomotion. So, we have discussed about the skeleton muscles, we discussed about the voluntary muscles, and we have discussed about the cardiac muscles. So, we have we have discussed about the skeleton muscles, we have discussed about the non-voluntary muscles, and we have discussed about the cardiac muscles.

In that context, we have also discussed about the structures and as well as the composition of the different types of muscles. So, we have discussed that the skeleton muscles is made up of the many types of proteins such as actin, myosin, troponin, tropomyosin's, and all these

proteins are actually having the exclusive functions which are they are going to contribute, so that the system is going to utilize that and that is actually they are going to use for the locomotion's.

In today's lecture, we are going to discuss how these actin, how these different fibres are interacting with each other and how they are actually going to provide the locomotive power into the system.

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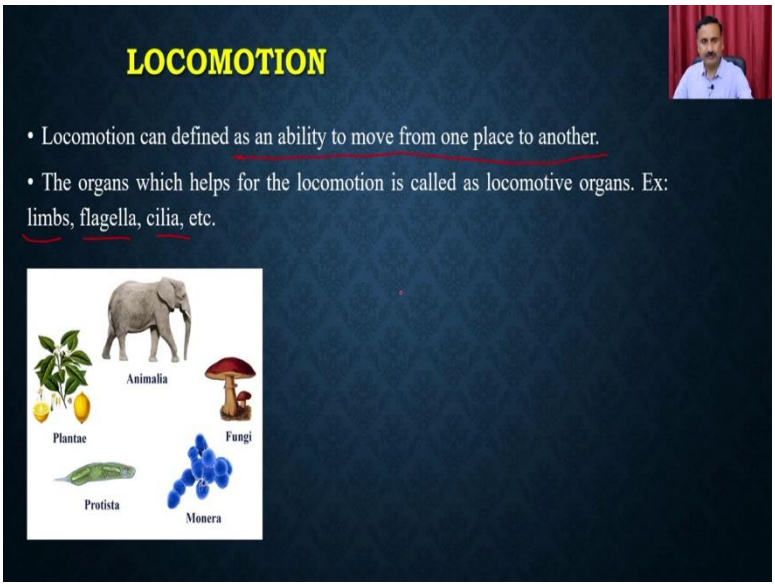

So, if you recall, what we have discussed that the locomotion is a is can be defined as an ability for an individual to move from the one place to another place and the different types of any organisms they are utilizing the different types of machinery for the locomotion's whether it is the limb, flagella, or the cilia. And there is an exclusive requirement of the locomotion that actually is protecting the organism from the prey, it is actually allowing the organism to catch its food and so on.

So, muscular system is actually, since it has the different types of muscles, it actually can be used to perform the different types of muscular actions, okay. So, these actions could be different in the different conditions. So, let us first discuss about the type of muscle contraction, what is found in the human.

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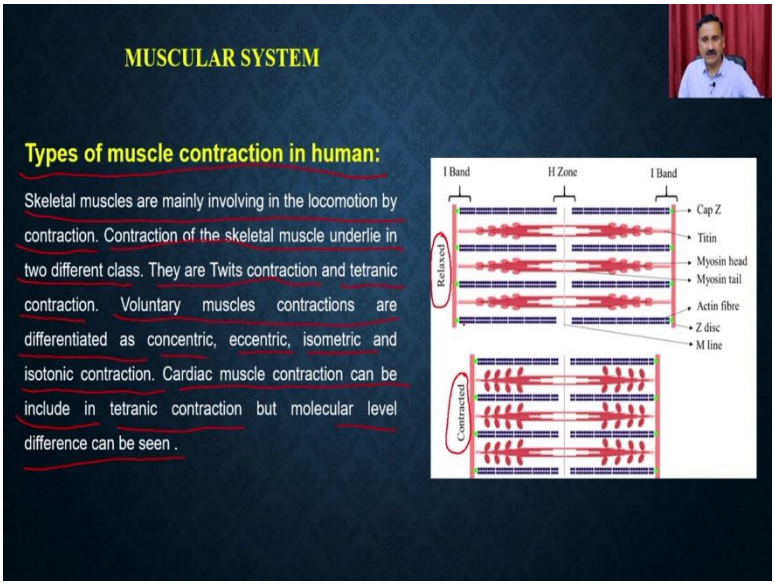

So, the type of muscle contractions are always been done in terms of context of the skeletal muscles. So, the skeletal muscles are mainly involved in the locomotion by the contraction, okay. And the contraction of the skeletal muscles underlie in two different class. They are Twits contraction or the tetranic contractions.

Voluntarily muscles contractions are different as concentric, eccentric, isometric, and the isotonic contractions. Whereas, the cardiac muscle contraction can be included in the tetranic contractions and the molecular level it is actually going to have the differences from the skeletal muscles.

So, this is what you see here is that all the muscle fibres are actually going to be present in the two different modes, one is called as the relaxed mode, where all the muscle fibres are going to be stretched to its maximum length and then they are actually going to be present in the contraction stage, so where the muscle fibre are actually going to be on to the shorter length, okay.

So, here there will be a tension between the fibres. where here the fibres are going to be acquire its maximum length so they are actually going to be very very relaxed. These are the two different conditions which actually can be utilized for the muscular system and that is how they can be have the different types of contractions.

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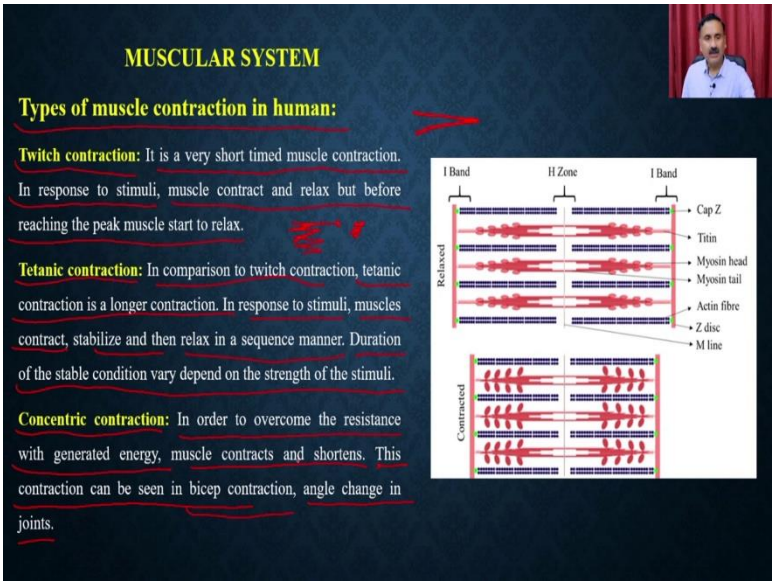

So, we can have the different types of muscle contraction in the human, we can have the twitch contractions, so twitch contraction it is a very short time muscle contractions. In response to a stimuli, the muscle contract and relax but before reaching the peak muscle start to relax. So, this is like a very fast. It goes like this and before it reaches to the maxima, it comes back, okay, so it goes like this, okay. So, this is the contraction, this is the relaxation, this is the contraction, whereas the muscle actually can go up to here but it will not go, it is like a flickering, okay, it is like a twitching contraction, so it will be going to happen very fast and then it will come down to resting stage.

Then, we have the tetranic contractions. So, in comparison to the Twit contraction, the tetranic contraction is a longer contraction, so in response to stimuli, the muscle contracts, then it stabilizes, and then relax in a sequence manner. This means in this case, the muscle is going to contract, okay or it is going to contract and then it is actually slowly going to relax, okay slowly going to release, so that is going to be at tetranic contractions.

During the tetranic contraction, the stable condition may depend on the stress of the stimuli. So, as long as you have the stimuli it is actually going to be keep contracting and then it will come down to relax, so it is actually going to be longer contractions compared to the twitch contractions.

Then, we have the concentric contractions. So in order to overcome the resistance with the generated energy the muscle contract and shorten, okay. So, this contraction can be seen in the biceps, it can be changed in the angle change in the joint, so it is actually going to be

present where you have the maximum energy, and it is going to utilize that for the muscle contraction, and it is actually going to be very very short.

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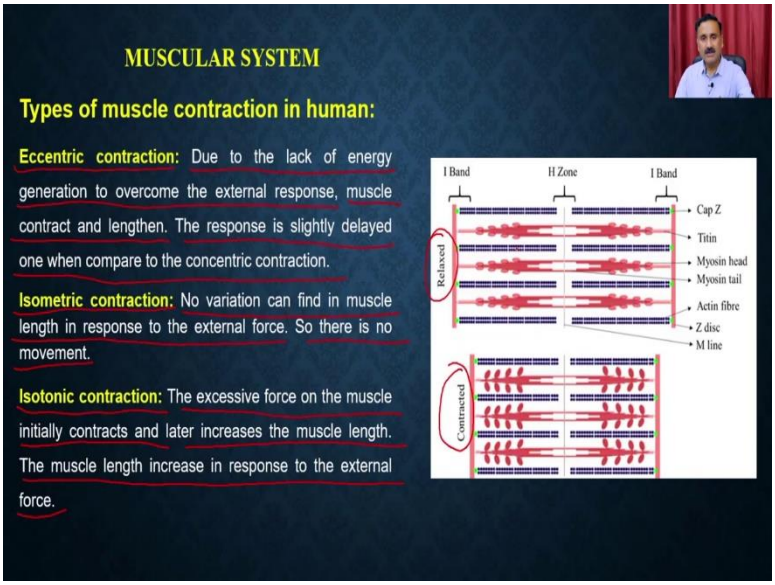

Then we have the eccentric contractions. So, due to the lack of the energy generation to overcome the external response, the muscle contract and lengthen, so this, the response is slightly delayed when compared to the concentric contractions, okay. So, these concentric contractions, eccentric contractions, and isometric contractions are actually going to happen into the smooth muscles.

And then we have the isometric contractions. So, no variation can be found in the muscle length in response to the external force, so there is a no movement, so it is actually going to have the contraction but there will be no physical or there will be no distance or there will be no movement of the muscles from the its original place, okay.

Then we have the isotonic contractions, the excessive force onto the muscles initially contract and the later increases the muscle length, the muscle length increases in response to the external force. So, isotonic contractions and all these contractions what we discussed are actually going to occur mostly in the all different types of muscles, whether it is the skeleton muscles, smooth muscles, or the cardiac muscles. Now, let us discuss how these fibres, which are present in the relaxed mode or to the contracted mode are actually going to do any kind of contraction and relaxation.

So, what actually happens during the contraction and relaxation? So, we have the two different types of mechanism, one, mechanism where we have the general mechanism like

the how it is actually going to happen and then we are actually, once we understand the general mechanism, how these fibres are interacting and stretching and you know relaxing to each other. Then we are actually going to tell you that, how the actually is happening at the molecular level, what are the different changes are happening within the cell at the molecular level.

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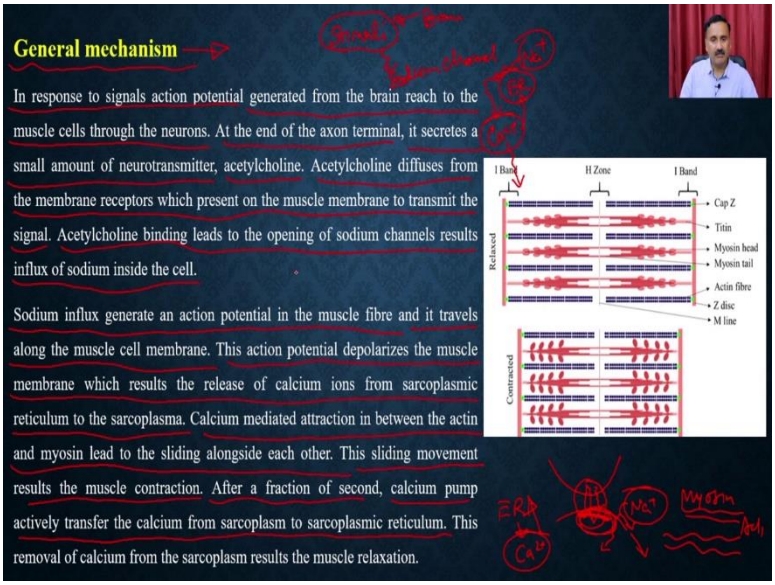

So, in the general mechanism, what happened is that in response to a signal, you know that there will be a synaptic vesicle where synaptic neurons and where the neurons are actually going to interact with the muscles. So, these are the places from where the signal is actually going to convey to the muscles, okay, and once the signal is received, then only the muscles is actually going to contract.

So, in the response to the signal action potential generated from the brain reach to the muscles through the neurons. So, that is the first step, first the neurons are going to be excited and they will get the signal from the brain, so this is what we are writing in context to these skeletal muscles.

At the end of the axon terminal, it secretes a small amount of neurotransmitter like the acetylcholine, so you are going to send the signal. So, you are going to secrete the acetylcholine and the acetylcholine diffuses from the membrane receptor which present on the muscle membrane to transmit the signal, so that acetylcholine is going to receive by the signal, by the muscles, by the receptors.

Then the acetylcholine binding leads to the opening of the sodium challenge resulting into the influx of sodium inside the cell. So, once the acetylcholine is going to bind onto the muscle cell it is actually going to open the sodium channel and that is actually going to have start the influx of the sodium inside the cell, so it is actually going to start receiving the sodium which is present in the outside.

Sodium influx is going to generate the action potential into the muscle fibre and it travels along the muscle cell membrane. So, once this happened, if there will be a change in the polarity, there will be change in the charge. Because you know that the sodium is positively charged, so when it enters into the cell it is actually going to impart a charge onto the membrane and that charge is actually going to relay and that is how it is actually going to activate the muscle cells.

This action potential depolarizes the muscle membrane which results the release of calcium ion from the sarcoplasmic reticulum to the sarcoplasm. So, once this action potential is going to be generated the calcium is going to be released from the ER, so ER is going to be having a storage of calcium, so that calcium is going to be released from the endoplasmic reticulum.

And the calcium mediated attraction in between the actin and myosin lead to the sliding along the each other. So, once the calcium is going to release, you have the two different types of fibres, you have the actin fibre, and you have the myosin fibre and these actin and myosin fibres are actually going to slide on to each other and the sliding movement is requiring additional factors and additional players. So, sliding moment is going to result into the muscle contraction.

So, what happens is that you have the actin fibre and the muscle, the myosin fibres are actually going to slide onto this and then it in this process it is going to utilize the calcium which is going to be released from the endoplasmic reticulum. And once this happens, there will be a disconnection, so there will be a shortage.

So, after a fraction of seconds, when this has started, when this happened, the calcium pump activity actively transfers the calcium ion from the sarcoplasm to the sarcoplasmic reticulum. So, once this start after a few seconds, the calcium is actually going to transfer from the sarcoplasm to the sarcoplasmic reticulum, which means it is actually going to withdraw. And this removal of the calcium from the sarcoplasm results into the muscle contraction, muscle relaxations.

So, if you see the in general scheme, the general scheme is that you are going to first have the stimuli, so stimuli is going to be generated from the brain, so from the brain you are going to have the stimuli and then from the stimuli it is actually going to activate the sodium channels, so it is going to open the sodium channels and that sodium channel is actually going to allow the entry of the sodium, once there will be entry of sodium, then there will be it is actually going to generate the action potential and once the action potential is going to be generated that is going to activate the release of the calcium and once there will be a release of calcium that is actually going to activate the sliding movement between the actin and myosin fibres, and that will be resulted into the contraction of the muscles.

Once that contraction is over, then the calcium which is going to be released is going to be returned back to the ER and once it is going to be returned back to the ER, the all the molecular event downstream to this is going to be reversed and that is how the muscle is going to be relaxed. So, this is just a general mechanism, but if you want to understand more precisely we are going to discuss about the molecular mechanism, how different types of molecular molecules which are actually going to participate into this particular type of mechanisms.

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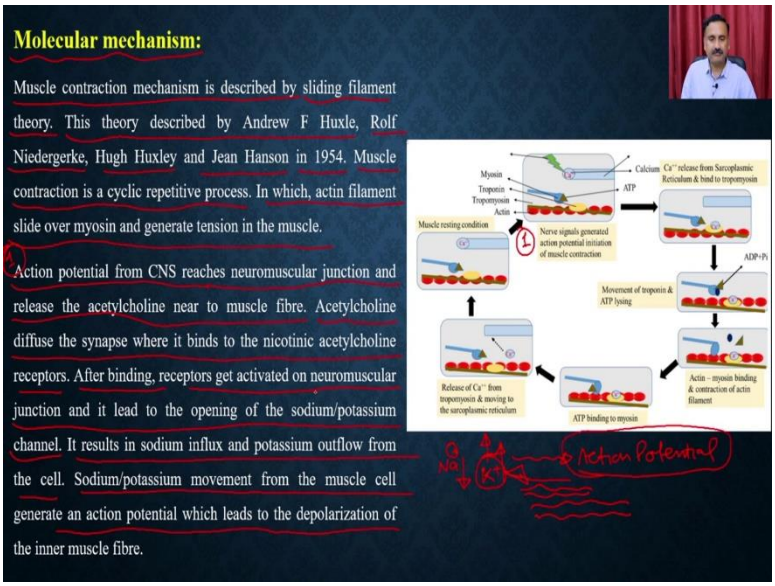

So, what is the molecular mechanism? So, molecular mechanism is to explain the muscle contraction is being explained by a muscle sliding filament theory, okay. So, the muscle contraction mechanism is described by the sliding filament theory and this theory is described by the Andrew F Huxle, and Rolf Neidergerke, Hugh Huxley, and the Jean Hanson in the year of 1954.

Muscle contraction is a slight contraction is a cyclic repetitive process in which the actin filament slides over the myosin and generate tension into the muscles, which means you have the, you have the myosin fibre and then the actin fibres are actually going to slide and that is how they are actually going to generate the tension into the muscle and that is how they are actually going to allow the muscles to contract.

First step is the generation of the signal. So, action potential from the brain or the central nervous system reaches the neurotransmitter junctions and releases the acetylcholine near to the muscle fibre, so this is what is going to happen in the first step. So, first step is the neural signals are going to be generated at the synapses where the neurons are going to interact with the muscles and the acetylcholine diffuses the synapse where it binds to the nicotinic acetylcholine receptor.

After binding the receptor get activated on the neuromuscular junction and it leads to the opening of the sodium or the potassium channel. It results in the sodium influx and the potassium outflow from the cell. Sodium potassium movement from the muscle cell generate an action potential which leads to the depolarization of the inner muscle fibre. So, this is what it is going to happen at this stage.

So, in this stage first of all the signal is going to be generated from the brain and once the signal is reaches to the this particular site, it is actually going to release the acetylcholine and acetylcholine is going to be received by the acetylcholine receptor and that is going to allow or it is going to start the release of the sodium and potassium channel.

So, in that sodium potassium channel if sodium is going to taken up from the outside and the potassium is going to be expelled outside. So, because of this the sodium is positively charged, potassium is also positively charged, it is going to generate action potential and that action potential is going to utilized for the muscle cells for its contraction.

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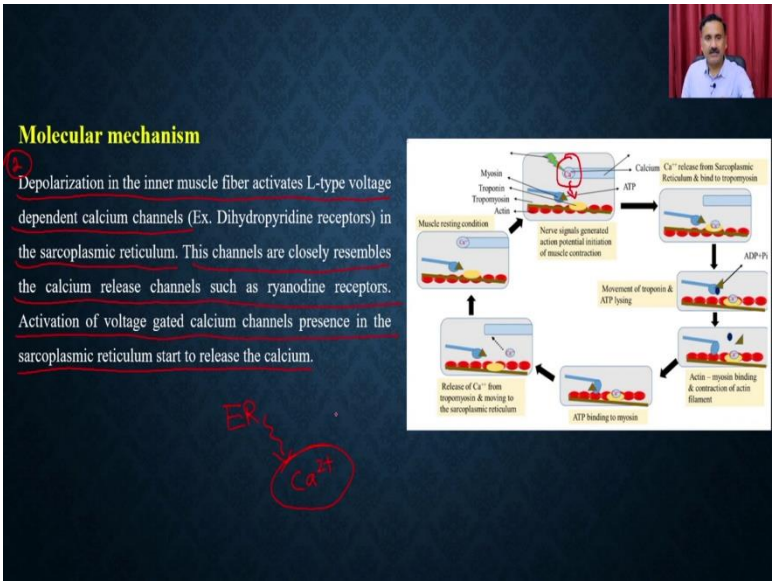

Now, in the step two, what will happen? Once this happened, the depolarization, so in the step two the depolarization in the inner-muscle fibre activates the voltage-dependent calcium channels, okay. So, once this happens then it is actually going to activate the voltage dependent calcium channel. So, voltage dependent calcium channels are the channel which are going to be activated when there is a change in the charge onto the membrane. So, in the cytoplasmic reticulum so it from the ER the voltage gated channel is going to be activated and then it is actually going to allow the release of the calcium.

These channels are closely resemble the calcium release channels such as the ryanodine receptors. The activation of the voltage-gated calcium channel presence in the cytoplasmic trigonometry start to release the calcium. So, in the step two since there is a voltage-gated calcium channel, it is going to be activated, and then the from the ER, there will be a release of calcium, okay. Now once you have the release of the calcium that is actually going to trigger the downstream events.

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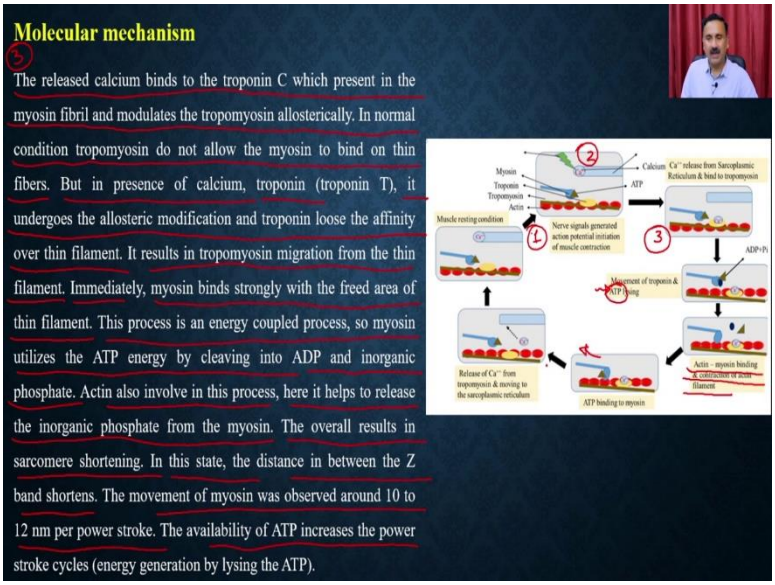

So, in the step three, the released calcium binds to the troponin C which is present onto the myosin fibril and modulates the tropomyosin allosterically. So, once the calcium is going to be released in the, this is the step one, this is the step two, and in the step three, the calcium which is going to be released from the sarcoplasmic reticulum, it is binds to the tropomyosin. The tropomyosin which is binding to the, which is winding to the myosin fibrils.

In normal conditions, tropomyosin do not allow the myosin to bind the thin fibrils but in the presence of calcium, the troponin it undergoes the allosteric modification and the troponin loses the affinity over the thin filament. It results in the tropomyosin migration from the thin filament, immediately, the myosin binds strongly with the free area of the thin filament. This process is an energy dependent process, so myosin utilizes the ATP energy by cleaving it into the ATP and the inorganic phosphate.

So, when the calcium is binding, it is elastically changing the activity of the tropomyosin and as a result the tropomyosin is actually allowing the myosin to interact with the thin fibrils and the myosin is actually utilizing the ATP, so it is actually going to cleave the ATP, and that is how it is going to interact with the thin fibres.

Actin also involved in this process, here it helps to release the inorganic phosphate from the myosin. The overall results in the sarcomere shortening. In this state the distance in between the z band is going to shorten the moment of myosin was observed around 10 to 12 nanometre power stroke.

The availability of the ATP increases the power stroke. So, that is why the energy is very important, because the myosin is going to utilize the ATP and that is why you remember that when you run from one place to another place you actually require the energy and that is why it says, that if you have a very high quantity of the ATP what is present into the muscles, you are actually going to do the locomotion very fast, you are actually going to do the muscle contraction very fast, and that is why you can be having the better stamina, okay.

So, the inherent ability of having a very high quantity of ATP is actually going to determine the stamina of that muscle cells. So, what has happened is that at this stage, the actin is also going to involve. So, actin, myosin is going to interact and the myosin (())(23:37) and that is how the (())(23:39) is actually going to bind to the myosin filament and then it is actually going to have the contraction, so the actin is going to pull this particular fibre, okay, so once it binds it is actually going to pull and that is how there will be a contraction of the muscles.

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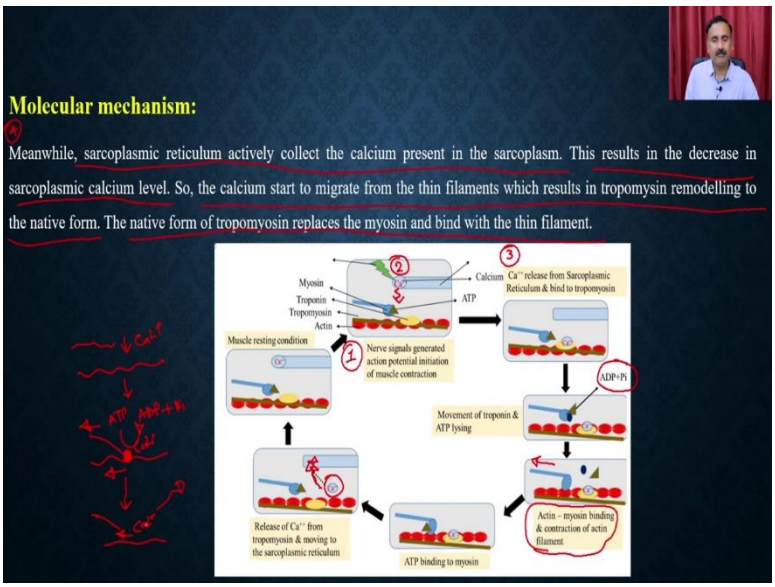

Now, so this is going to be the step number four. Like the sarcoplasmic reticulum actively collect the calcium present in the sarcoplasm, this results in the decrease of the sarcoplasmic calcium level. So, the calcium starts to migrate from the thin filament which results in the tropomyosin remodelling to its native form, the native form of the tropomyosin replaces the myosin bind with the thin filament.

So, after this there will be a release of the calcium from the tropomyosin and it actually will go back to the endoplasmic reticulum, or sarcoplasmic reticulum. Once that happens, that since there is a no calcium and that is actually and you know that the calcium is allosterically modifying the tropomyosin and that is why the myosin could be able to interact with the thin

filaments or the actin filaments, it is actually going to dislodge the actin and that is how the muscles are actually going to enter into the relaxation stage.

So, these are that all the events what you are going to have when there will be muscle contractions. If you summarize all these in the step one, there will be a nerve signal which is going to be generated from the brain, and once the nerve signal is going to be generated, it is actually going to allow the release of the acetylcholine and acetylcholine is going to bind onto the neuromuscular junctions and that is actually going to activate the voltage dependent voltage-gated channels, okay.

So, once that is going to happen, it is actually going to release the sodium and it is actually going to have the exchange of the sodium and potassium and once there is the exchange of the sodium and potassium, it is actually going to generate the actin potential onto the muscle cells.

Once there is a depending or once there is a generation of the actin potential that is going to activate the opening of the voltage-gated calcium channel onto the sarcoplasm reticulum. So, in the step 2 once that happens the sarcoplasmic reticulum is going to release the calcium and once there is a release of the calcium the calcium is going to bind on to the tropomyosin. So, in the step 3 the calcium is going to be released from the sarcoplasmic reticulum and then it is actually going to bind onto the tropomyosin.

Once the calcium is going to bind onto the tropomyosin the tropomyosin is going to be allosterically modified and that is how it is going to induce the myosin fibril to interact with the thin fibres and in that process the myosin is actually going to utilize the ATP in the, and it is going to cleave the ATP to generate the ATP and PI and then there will be a interaction of the actin fibrils with the myosin.

Once that has happened it is actually going to bind and then there will be the pull step, so it is actually going to pull the fibres and the contraction of the actin filament is going to resultant into the contraction of the actin fibres. Once that happens and the contraction is going to occur, then the calcium what is present into the tropomyosin on calcium what is bind to the tropomyosin is going to be taken back by the sarcoplasmic reticulum and that is how when there is a release, when there is a removal of the calcium, the tropomyosin is actually going to acquire in its native form conformations and as long as the tropomyosin is present in the its native conformation, it is going to destroy or it is going to abolish the interaction between the myosin and the actin or the thin fibres.

And once that happens, the cell, the muscles is actually going to undergo into the relaxation stage. So, as soon as you have the signal if the contraction is going to happen, once the signal is dropped then the all these events are going to be reversed, and that is how the, there will be a contraction and relaxation and that is going to resultant into the muscle movement. So, you can imagine that you have myosin fibrils and you have the actin fibrils, okay. So, as soon as there will be a calcium what is going to be released, okay, now this fibre is actually going to go and bind the lower fibre, okay, and then this fibre is actually going to pull so if it is binding and if it is pulling this also going to pull and that is how it is actually going to shorten the lower fibril and that is how there will be a contraction and the calcium is already there, okay. And in this process it there will be a one ATP which is going to be consumed by this system, okay.

But as soon as the signal is dropped, so contraction signal is dropped from the brain then the calcium is going to be released, so there will be no calcium so calcium is going to be released and that is how the interaction between the actin and the myosin fibril is also going to be broken down and that is how there will be a relaxation, so it is going to relax, so it is going to return back to its normal state. So, these are the molecular events what is going to happen when there is a muscle contractions.

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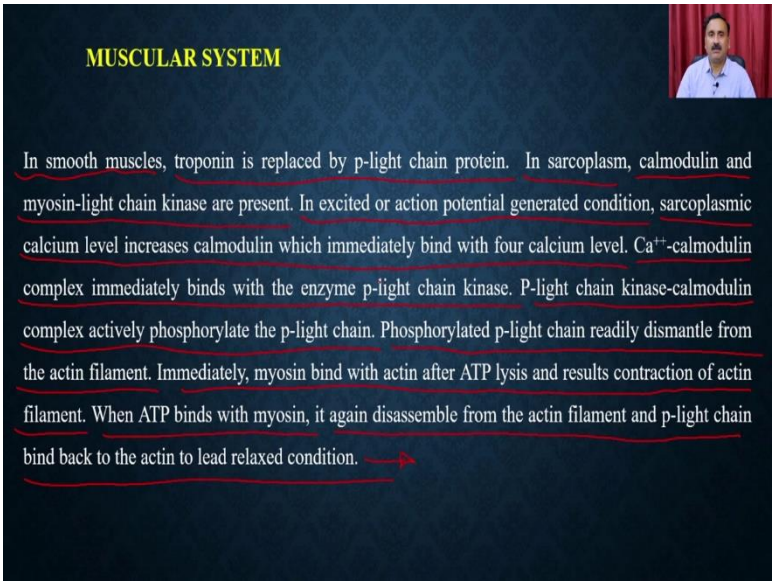

Now, the muscle contraction in the smooth muscles are also going to happen and but the molecular players are also once molecular players are going to be different. So, troponin is going to be replaced by the p-light chain protein. And, in the sarcoplasm, the calmodulin, and the myosin light chain kinase are present.

So, in the excited or the action potential generation conditions, the sarcoplasmic calcium level increases calmodulin which immediately binds with four calcium. So, the calcium calmodulin complex immediately binds with the enzyme p-light chain kinase and the p-light chain kinase calmodulin complex actively phosphorylate the p-light chain.

Phosphorylated p-light readily dismantled from the actin filament and immediately myosin bind with the actin after the ATP lysis and the results in the contraction of the actin filament. When the ATP binds with the myosin, it again dismantle from the actin filament and the p-light chain bind back to the actin to lead to the relaxation condition. So, overall the molecular mechanism remains the same whether it is the smooth muscles or the skeletal muscles or the cardiac muscles, except that the molecular players are little different.

So, this is all about the muscle contractions but since the muscle is a machine, the machine can also get broken down, machine can also get affected when you utilize that without maintaining the machines and so on and that is how you are actually the person can actually be able to develop the different types of muscular disease, okay. So, what are the disease the common diseases what are there in the muscular system.

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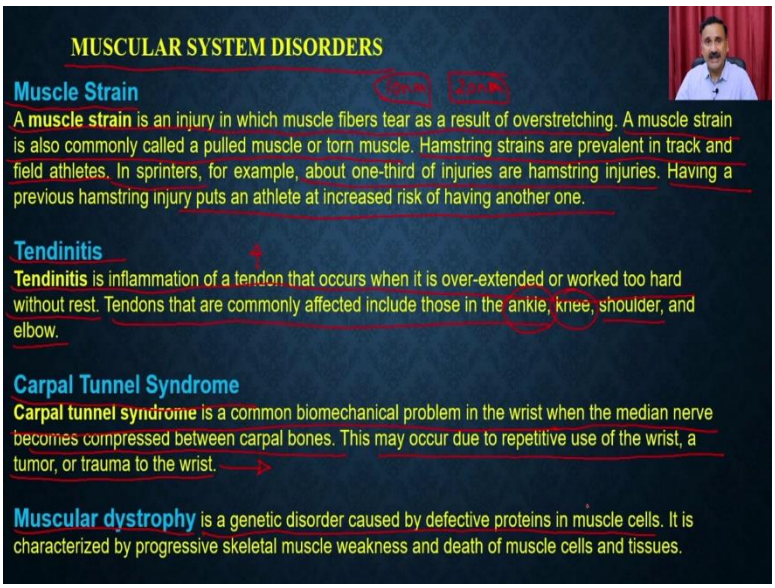

So, you can have the muscle strain, okay. You can have the Tendinitis, you can have the Carpal Tunnel Syndrome, you can have muscle dystrophy, you can have the other kinds of diseases also. So, muscle disorders are very very common because, whether it is the minor injury like the muscle strain or whether it is very very serious issues like the muscular dystrophy. So muscle strain, muscle strain, a muscle strain is an injury in which the muscle fibre tear as a result of over stretching.

You might have seen that when you actually slips or sometime when twist your hand very sharply or very fast you actually feel the pain because what happen is that the muscles actually cannot go beyond that particular limit. For example, if a muscle cell can stretch only up to 10 nanometres and if you are forcefully do the contraction and which it goes up to like 20 nanometres for example.

If you do that, then the muscle is actually going to be damaged, because they cannot go beyond the 10 nanometre stretching but you have stretched it up to 20 nanometre, and in that case there will be an injury and that injury actually is going to damage the muscle cells and that is going to be responsible for the muscle strain.

A muscle strain is also commonly called a pulled muscles or the torn muscles. Hamstring strains are prevalent, so one of the classical example is the hamstring strain which are very much prevalent in the track and field athletes and in the splinters, about one third of the injuries are hamstring injuries, okay.

Having a previous hamstring injury puts an athlete at the higher risk of another one. So, that is why the hamstring injury is a muscular strain and that happened because the athlete is running very fast or athlete is trying push up to an extreme and that is how it actually going to have that kind of injury.

Then we have the tendinitis. So, tendinitis is a, tendinitis is a inflammation of the tendon that occur when it is over-extended or worked too hard without rest. So, tendon is joint, so joint and inflammation of the tendon is going to be responsible for these particular conditions. Tendons that are commonly affected include those in the ankle, knee, shoulders, and the elbow, okay.

Then we have the Carpal tunnel syndrome. So, carpal tunnel syndrome is a common biomechanical problem in which the wrist when the median nerve becomes compressed between the carpal bones. And this may occur due to the repeated use of the wrist, a tumour or the tunnel, okay.

You might have observed, sometime when you use the keyboard, when you use the mouse, when you use the, you know some of your fingers very long then you might have feel that there is a pain actually and that pain is because there is a too much usage of that particular muscles and that happens very often when you use the computers. So, that is a classical example of the carpal tunnel syndrome.

And then we have the muscular dystrophy. Muscular dystrophy is a genetic disorder which causes the production of the defective proteins in the muscle cells. So, if you have a defective proteins like the defective myosin, defective actin, it is characterized by the progressive skeleton muscle weakening and the depth of the muscle tissue. So, if you do not have the very strong these kind of proteins, then you are actually going to develop these weak muscles and then the because the muscles are susceptible for the damages and susceptible for the death and that is how you can actually develop the muscular dystrophy.

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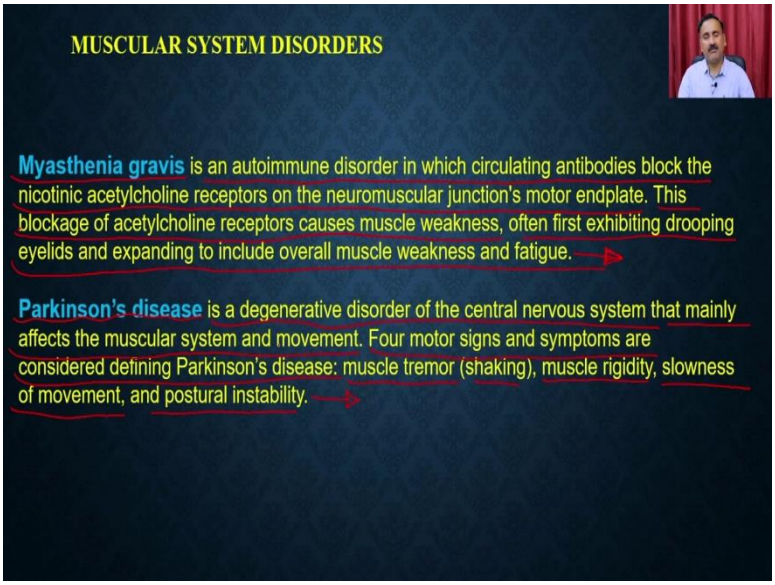

Apart from that we can have the Myasthenia gravis. So, Myasthenia gravis is also a autoimmune disorder in which the circulating antibodies blocks the nicotinic acetylcholine receptor onto the neuromuscular junction motor endplate. And just now, I think we have discussed that if there will be a blockage of the signal from the brain to the neuromuscular junction, this blockage is actually going to not allow the muscle cells to respond to the signal what they are getting from the brain. So, this blockage of the acetylcholine receptor causes muscle weakness. Often first exhibiting the dropping eyelids and expanding to include overall muscle weakness and the fatigue.

So, in, if there is no strong signal what the muscle cells are going to happen, the muscle cells are not going to be get excited into the contraction stage and they also will not go into the relaxation mode and because of that it is actually going to overall develop the muscles which are actually going to be very weak.

Because you remember that if you use the muscles very often, if you use the muscles on a regular basis, the muscle cells are actually going to be keep getting the good supply of blood,

good supply of nutrients, good supply of everything, and that is how they actually are going to be strong. But if you do not get with the muscles they are not going to contract because there is a neuromuscular disorder, there is a junction problem, so they do not, they do not get signal, the muscle cells will not going to grow and that is how they are actually going to be very very weak.

Then we have the one of the serious problem is the Parkinson disease. So, Parkinson's disease is a degenerative disorder of the central nervous system that mainly affects the muscular system and the moment. So, Parkinson's disease is actually a problem of the brain but it also affects the muscular system, it also affects the person's ability to remember so many things it also affects the person's ability to movement.

So, four motor signals and the symptoms are considered defining the Parkinson's disease, such as the muscular tremors like the shaking of the hands, then we have the muscular rigidity, slowness of the movement and the postural instability which means you cannot actually be able to sit in a particular posture for very very long time.

And there are many many celebrities which actually got the Parkinson's disease and ultimately they have developed these kind of symptoms and that is how they cannot be able to perform the daily activities and ultimately once you have the very loose or weak muscles, you are actually going to affect the visceral organs, you are actually going to affect many different types of other activities.

Like for example if there will be a loss of peristatic motion, if there will be loss of other kinds of motion in the elementary canal, and so on, these are actually going to overall affect the health of that particular individual like for example, if there will be a weakening of the cardiac muscle, that is actually going to affect their heart to pump the blood into the blood, into the body.

And that is why the Parkinson's disease is actually a disease of the brain but it affects the overall development of the body, okay. And you might have heard about the many of the celebrities who actually got the Parkinson's disease and ultimately, they die not because of the complication of the central nervous system, but they die because there is a complication of the muscular system or there will be a complication of the other organs in the body.

So, with this what we have discussed? We have discussed about the molecular mechanism of the muscular motions, how the different players are actually interacting with each other, and

they are responsible for the contraction of the muscle, and the relaxation of the muscles, and then we also discussed about the different types of muscular system disorders which are going to happen.

And so, with this brief discussion about the muscular system, I would like to conclude today's lecture. In next lecture we are going to discuss some more aspect related to human physiology. Thank you.