Neuroscience of Human Movement
Indian Institute of Technology, Kanpur

Lecture - 51
Primary Motor Cortex Part - 15

So, welcome to this class on Neuroscience of Human Movement.

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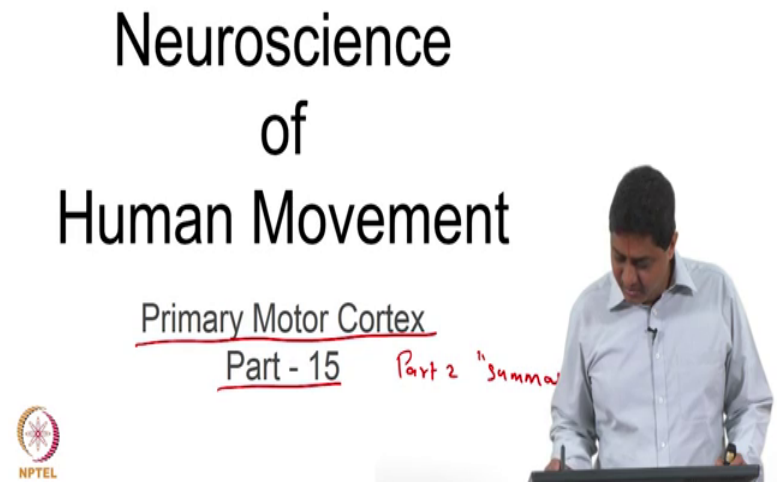

This is part 15 of our discussion on Primary Motor Cortex, and this is the last lecture on this topic. This is also a part 2 of the summary on Primary Motor Cortex. So, we have discussed the various works in Primary Motor Cortex, but we also discussed was lesions and brain machine interfaces in the previous classes.

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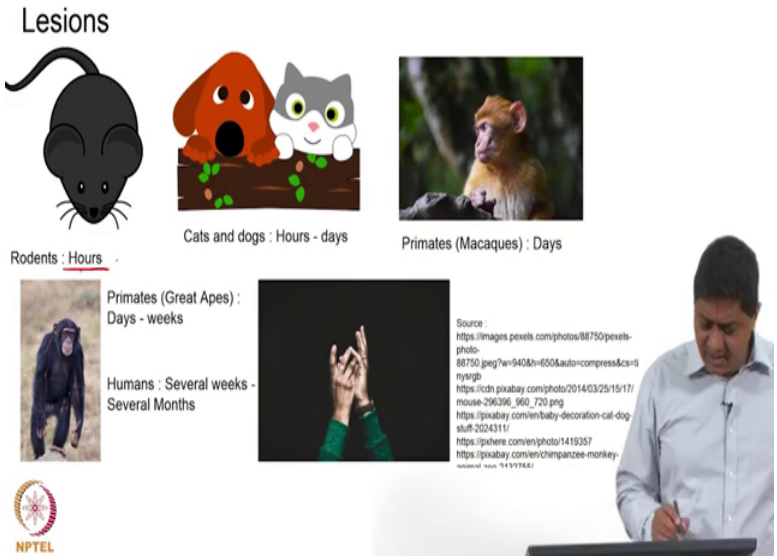

So, I am going to summarize that in today's class. We discussed the case of lesions how different animals behave post lesion. So, and how much time is taken by different animals to recover from a lesion, rodents take a few hours we said that. And cats and dogs hours to days and primates as in macaques days, and great apes days to weeks. And in humans this may be several weeks, to several months.

And as you go in this direction and in this direction for example, the potential for recovery reduces and the time for recovery increases. And also it seems that there is a critical period during which motor training must be imparted to people who have had a lesion. A question is what is this critical period is this critical period the same, these are open questions in research; people are still trying to understand what this is. This must obviously, be related to the size of the lesion, to the location of the lesion, to the persons previous experiences to the actual specific case.

So, it is a very unique thing because it turns out as we discussed in the previous class there is this duality between brain structure and brain function, how we perceive the world at a particular point in time is determined by your brain. But so that means, if I am seeing this if I am seeing this class of students if I am seeing the camera that is determined by my brain and how I perceive that. But the experiences also change my brain not just functionally, not just notionally; actually there is a structural change within the brain. So, this leads to a situation where each brain is different and the response of

each brain to injury will also be different. This is not an excuse that we are giving this is a fact. We will have to find solutions despite this truth.

So, there must be complete acceptance of this truth that there is going to be a requirement to deliver patient specific therapy, then with that fact accepted then therapy is expected to be more effective for patients. So, open question us to what kind of therapy is useful. Several questions are open, so I hope there is going to be more questions that come at the end of this course than answers. I hope that is the, that is achieved.

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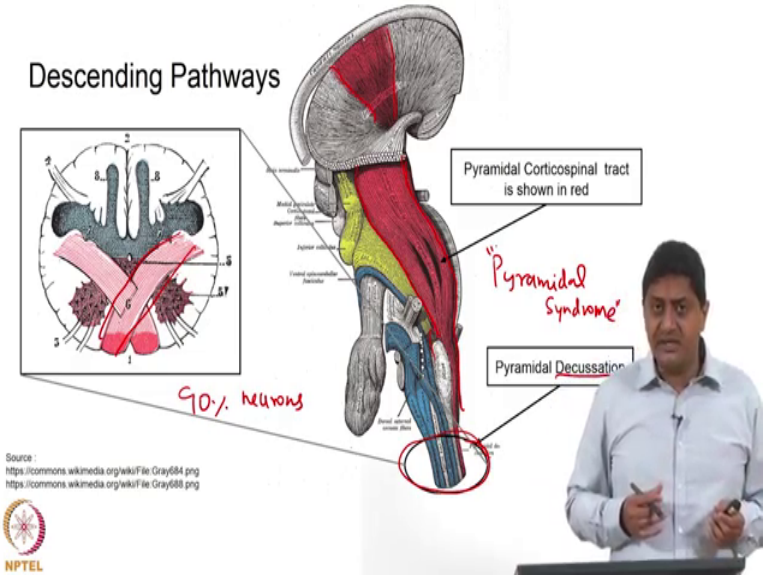

But we also saw was a pyramidal tract and there are several descending pathways of corticospinal pathway, in the corticobulbar pathway etcetera. So, the pyramidal tract is shown in red here, right, just going through the internal capsule down here and it is zoomed out here. So, this is where the decussation happens, right. So our crossing over of about 90 percent of the neurons to the contralateral side which means that there are 10 percent of the neurons that project either if cilaterally or bilaterally right; about 80 to 90 percent of these neurons cross over to the other side in that area, so that is called as a pyramidal decussation.

And we said that transacting this or injuries to the pyramidal tract cause typical symptoms that have come to be called collectively as pyramidal syndrome. We said that

this is a misnomer several other problems also cause similar symptoms; we said this we discussed this in detail in the previous classes, ok.

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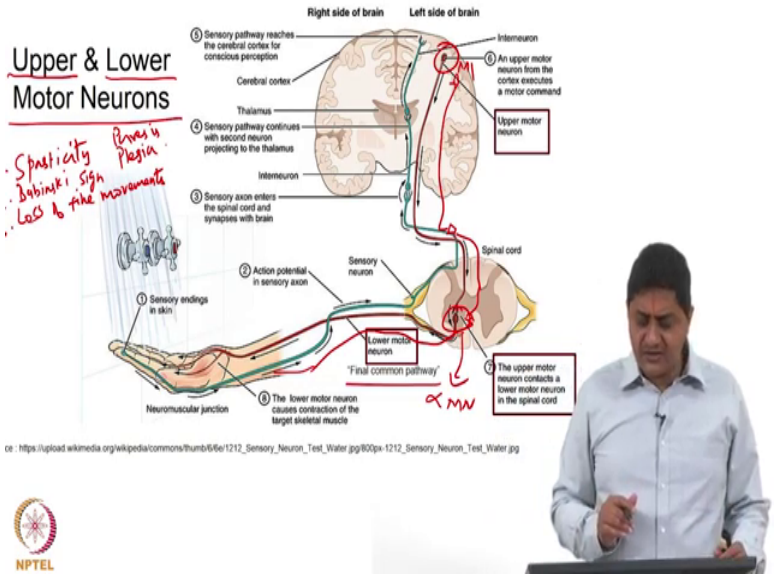

And we also said there are upper motor neurons or the cardio motor neurons in M 1 and then there are the lower motor neurons in the spinal cord we call this as an alpha motor neuron. And the alpha motor neuron muscle pathway is called as a final common pathway please find out who said this. This is the final common pathway and some neurons alpha motor neurons they receive direct M 1 projections or monosynaptic M 1 projections.

So and there are others that receive inputs through inter neurons. There are others that receive inputs through you know through an inter neuron, and to some other alpha motor neuron etcetera, right, to more proximal parts of the body. And we said what lesions to these two are upper and lower motor neurons can do fundamental differences in mechanisms, right. When the lower motor neurons are injured there is actually denervation right, because of this there is going to be a trophy, there is going to be a flaccidity, there is going to be weakness.

When the upper motor neurons are injured the descending inputs from the M 1 region are lost, the influence of this descending input is quite complicated, but at least the what it does to the system is that it results in an increase of tone or hypertonia, actually this hypertonia is preceded by a hypotonia. And an increase in the reflexes brisk and

heightened reflexes right we said this brisk and heightened deep reflexes are hyperreflexia and spasticity. So, what are the classic signs of the upper motor neuron syndrome? There are 3 classic signs, spasticity. What is spasticity? Rate dependent rigidity.

So, a rigidity is felt if load is applied very fast, if the same load is applied relatively slowly the rigidity is not felt or relatively small, right. So, this is spasticity this is different uniquely different from the general rigidity found in other problems, ok. What is the other sign? Classical sign, Babinski sign, what does this refer to? Babinski reflux, Babinski sign. What does this refer to? This refers to the case where the fanning of toes is observed when a blunt object is used against the foot.

Suppose this is the foot this is the hand actually, but assume that this is the foot of an adult right, and you are using a blunt object to perturb like that perform this moment. This in a healthy adult will cast flexion of the toe suppose these are toes assume that the fingers or toes this will cause flexion of the toes including the great toe, right.

But in a person who has had upper motor neuronal relations, this stimulus will cause fanning of toes with an extension of the great toe. This is called as Babinski sign such a sign is considered normal in infants, but abnormal in adults so, classic sign.

What is the third problem? Loss of fine moments, we said this earlier. These are the classical problems that come with upper motor neuron relations in lower motor neuron relations you have, flaccidity, denervation or trophy, and hypotonia. Whereas in an upper motor neuron relations you have hypertonia, you have hyperreflexia and Babinski sign and spasticity not flaccidity, spasticity. And in depending on the lesion there is going to be a graded response there can be either paresis or plesia, paresis refers to weakness whereas, plesia refers to paralysis.

So, this also we discussed in detail the differences between the two cases. I have just presented some points here just to remind you for this. So, that termed here is to summarize the situation, right.

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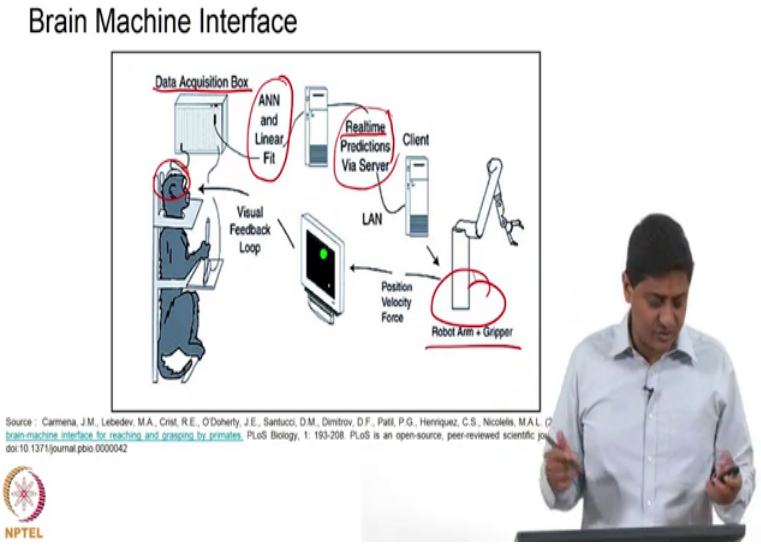

And then we discussed the case of brain machine interfaces how to record from live behaving animals. Hopefully this technology will improve sufficiently enough for it to be implemented in humans. What are the challenges that lie before us, that stand between and the actual implementation of this in humans: many too many actually to discuss.

So, that is good and bad simultaneous it is good because it provides us an opportunity to solve this problem in a way that is good. It is bad because well we still do not have solution to the problem, so that is bad. Despite so much technical progress commercially viable, a relatively affordable brain machine interface that can be used that is scalable and sustainable or still eluding us, despite substantial progress in this field.

So, what are the main components of data acquisition from the brain and computation? And then, that is given to a machine which is a robot arm and gripper which is controlled, right. So, this must happen the key word is real time like it would happen in a live human being. That means great challenges in computation, great challenges in instrumentation right. So, the opportunity is there. So, the window of opportunity is there for all of us to contribute; how many of us are willing to take up that opportunity; million dollar question.

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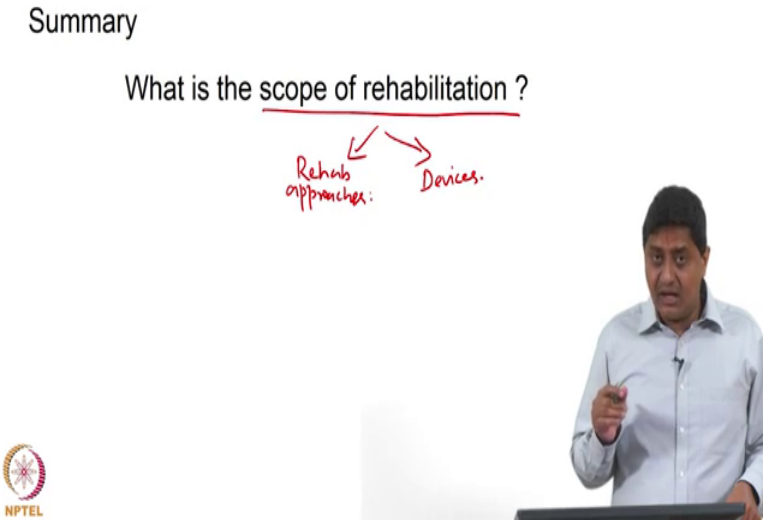

So, in summary the question is what is the scope of rehabilitation in humans, to what extent can we rehabilitate? Well, the hope is that we can rehabilitate to begin with. Two is that: what are the appropriate approaches of rehabilitation that remains the question.

One is rehab approaches or therapy, physical therapy, two is devices both of this need to improve and catch up with the problem. In the United States 3 quarters of a million people are affected by stroke every year. In India it is about 1.2 million people every year, so 12 lakh individuals every year and 750000 individuals every year in the US, 12 lakh individuals in India. How many of these people are going to be helped by us and by our innovations? Questions are open, there are many problems, there are many solutions. I leave you with questions than answers. Hopefully there will be some of us who will contribute to change this world.

Thank you very much.