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PROFESSOR: I want to go over the homework I will post right after the class. I wanted to talk to you about it first. These are really worksheets. It's homework that you should all get everything right. I will do a separate homework that will make up for some of the bad quiz performances. I know you requested that. That's not what this is. This is to help you review. And I selected a bunch of pictures. I'm just giving you a [INAUDIBLE].

This will all be posted. I guess you all have access to printers. If you have problems printing it, just come with us after class. We'll print it. But I'm going to show you what it is. These are the worksheets. And you can see, if we look at one of them here, this is a pretty straightforward one, and you should remember now.

Here you have places to write the names. And so, just to remind you what I want here, question on I say, in slide number one. That refers to the number of the slide is that. The number's actually written in the corner. And then I list A, B,C, D,E. I just want you to write the English and the Greek or Latin for each of those subdivision. That's what you do for that one.

And for something like this, all of these are like that. Here I ask you to name the parts that are being pointed to. Here, I ask you to color in the pathway like I did in class. And I want the local reflex channel.

Now, whereas this, the blank brain diagram. They're pretty similar to ones I used in class, but they match the text of the book very well. And then I ask you questions about them. But for those, because you have to-- if you have to draw them, notice I put it right in, most of the ones you would need printed . And for this one, too, because I'm asking for drawing.

And then I ask a question up here. Here I want you to draw a primer sensor neuron. I've already drawn it for you on the horizontal diagram there, but I want you to do that here, too. And I want you to show these final [INAUDIBLE]. And then later, I do the same thing for the dorsal column and medial meniscus. And then I do the same thing for the corticospinal.

So you can find those figures. They're all in the book. They were all on the slides, too. And I wanted you to draw them in. But if all you're going to do is find it and then go back and forth to get every detail right, you're not learning. You have to study it, then put it aside, and then try it. Try to draw it. And if you hand something in with all kinds of things crossed out because you didn't write it, that's all right. I want you to use this to learn about these things.

AUDIENCE: [INAUDIBLE]?

PROFESSOR: Some of these might be on the midterm. Or I might just ask questions about them. But, yeah, there might be things like this on the midterm, definitely. So that'll all be posted. And that is just to help you review.

You can have it due Monday, I think. And I will get another one posted where you just have-- because these are all very straightforward. You just have to find and learn them. Try to do it. Many of them, I give you the diagram, and I want you to eliminate a lot of the labels. And so, like in the one with the meninges and the glia, you should know which ones here are the astrocytes, which is the dura, what is the pia, and what is the arachnoid?

So you need to know those things, and it's good to have not just the words, but to have in your spatial memory, too. So that's why we're doing it. Use it to practise and review. And then I will give you some questions, I think, mostly on the motor system because you haven't had any homework on that. And I could give a little bit on development, things that I know you can find. And we'll use that, as I promised, to make up for bad quiz grades. We'll decide exactly how we're going to do that, but I think that's fine.

AUDIENCE: [INAUDIBLE] how many chapters the midterm will be covering? Is it one through--

PROFESSOR: 17, through the brain states chapter. And then we start sensory systems afterwards, after the break. Or wait a minute. Do we have a Friday? I guess we started just before the break.

All right, so this is where we were. It's a little bit more on the introductory chapter to the motor system, where we're talking about these multipurpose movements. There was a lot about locomotion last time. We've also talked about the other movements. All that I have of what remains is I discuss in a little bit about evolution of that ability.

And just because we know that the tectum is the dominant structure now in most animals, except perhaps in animals like humans and even monkeys, where the cortex has become so dominant over most of these functions. But in most animals, it is still very important and it has an importance even in humans, more than people realize.

So I do mention a little bit about some of the early controls. But I'm mostly emphasizing two very different functions of the tectum, one involving locomotion. We talked about escape movements. But normally, when animals are escaping, they also orient. They don't generally orient with respect to the animals their escaping from. They orient towards safe place. If they're near their tunnel, they head for their tunnel. If they're near any hiding place, any place they can crawl into to get away, they head for that. And they're extremely good at finding that very, very rapidly.

And I say a little bit about the tectum and pretectum here because I go over this in the visual system later on. If I were you, I would just read this and use it. It does help prepare you for what's coming, but right now, you need to know this, the different nature outputs to the tectum.

So a little bit more about grasping. We know why it's so important. We know what muscles are involved. We know that there's different kinds of grasping; grasping with the hands. We know in large primates, it's largely a neocortical function, but animals without neocortex can still feed themselves. They still have to grasp things, either with the mouth or with the hands, so structures evolve to do that, and they're in the midbrain. So that's what those questions are about.

And we talked about this a little bit last time, so I'm not going to over it again. But I didn't talk about the red nucleus in a little more detail. There's two major parts of the red nucleus. It is quite interesting when you compare different animals. There's a large cell part that's the caudal part of the nucleus. And then there's a small cell part, so we refer to the magnocellular red part of the red nucleus. It's abbreviated NR for nucleus ruber, and then MC for magnocellular, or NR, nucleus ruber, PC for parvocellular. Parvo means small.

So those are the two parts. And it's really interesting if you look across primates, even if you went here to primitive primates, it would be like the carnivore here. And humans, the parvocellular part, the rostral part, is by far the largest part. The magnocellular part is smaller. So, what do these two parts do? Well, the outputs are very different. Of course, their inputs are different, too.

Let's just deal with the outputs. The rubrospinal tract case. The output at the spinal cord, that comes from the large cells, the magnocellular are part of the red nucleus. So in animals where that pathway's more important than neocortex, it'll be like this. The caudal part is relatively larger. That's true and carnivores. It's true in rodents, true in most animals.

But animals that are really developed, more endbrain control of their hands, then the parvocellular part is more. So why is that? Well, what does it project to? First of all, it projects forward, not like the rubrospinal tract. This projects rostrally. Parvocellular nucleus projects to the thalamus, mostly to the ventral lateral thalamus, the part of the thalamus that projects to the primary motor cortex. And it also projects to the precerebellar nucleus. So the inputs to the cerebellum and to the neocortex depend on that for controlling hand movements, both through that parvocellular part.

And this also shows a correlation with a structure in the cerebellum. That's this structure. They just pick that out of the cerebellum. It's the lateral most deep nucleus. It's the part of the cerebellum that gets input from the very large hemispheres in human cerebellum. But any animal-- what they're not showing here is the whole cerebellar hemisphere in growing bigger in these animals at the right, in apes and humans and also monkeys. Whereas in other animals with less of this kind of control, that lateral cerebellar output nucleus is also smaller. So as the cerebellum evolves, so does the cortex, and the red nucleus changes also.

AUDIENCE: [INAUDIBLE]. Does the red nucleus [INAUDIBLE] by having visual inputs? Because I read that--

PROFESSOR: Animals, no because animals can still be pretty dexterous without so much visual input, but there is a general correspondence. If you look, there are prosimians, for example, that where the red nucleus is more prominent than it is even in humans, that have incredible dexterity, but it's mostly fixed action patterns. They use it for grabbing bugs out of the air.

AUDIENCE: [INAUDIBLE] magnocellular and parvocellular? I think the--

PROFESSOR: All the animals I mentioned that have a small one are less visual.

AUDIENCE: Magnocellular and parvocellular, there's a distinction [INAUDIBLE].

PROFESSOR: You have these different types of cells in other systems, too. But, no, there's no real strict correlation. Animals can be very dexterous even without using vision. And humans without vision can have very, very dexterous hand movements.

There's an interesting study of the projections of the red nucleus because they're concerned with control of distal muscles. Well, there's distal muscles in the forelimbs, but also in the hindlimbs. The question is, are those kept separate in the red nucleus? So you look at the projections to the cervical enlargement and the lumbar enlargement. It's just a cartoon to illustrate those two parts of the spinal cord and then the red nucleus projections.

And what this shows is neurons that project to the cervical enlargement, neurons the projected to the lumbar enlargement, and neurons that project to both; a branch to both nuclei. So if you look at the opossum, you'll see that there is some inter-mixture here. The topographic separation of the representation for hand control and foot control is it's not all that good. In the rat, it's a little better and in the cat, the separation is really good. And of course, in primates it is, too. Just an interesting thing about how you find these correspondences with function and anatomy in these descending projections.

So now I want to talk about the outputs. And I want to start with the motor neurons. So then we can talk about the pathways that connect to those motor neurons, and how the pathways, say, from the cortex and brainstem connect to the motor neurons to the spinal cord. And we're talking here about somatic motor neurons. We talked earlier about autonomic, and we said a little bit about neuroendocrine controls to the hypothalamus. We've talked a lot more about that, when we talked specifically about hypothalamus. We're just talking about the somatic system with synaptic connections to muscles, not paracrine, as in the autonomic system, and not endocrine, as in the endocrine systems.

This is Larry Swanson's characterization of the entire motor system, he says, the three motor systems. This is the somatic, number 3 here. Number one is the endocrine. And he shows this. The Dashed line indicates connections. There's no synapse at all. They're just through the bloodstream. And then the paracrine innervation of the autonomic system, parasympathetic and sympathetic. That's motor system two and motor system three.

And when he discusses that, it's interesting. He says, we don't know what are the pattern generators that are coordinating these three systems. And my comment about that is just that, we don't know if there is very much direct coordination central to these systems, but there probably is some, but it's not been specifically-- we know a lot less about that than we know about controlled pattern movements.

And this distribution of somatic motor neurons, I've had that in other pictures that I showed you earlier on. You probably didn't pay much attention, except I always show the motor neurons ventrally. So I asked this question here, this is a horizontal view, it doesn't indicate anything about dorsal and ventral. So I say, where are these neurons located in frontal sections?

So here, this is all spinal cord, where most of those neurons are, and you can see most of them are in the enlargements. Because we've many more muscles to control in the limbs, we need finer controls, so we've got more motor neurons controlling them. They're always in the ventral horn. And that's what we're going to talk about next, but these are the groups of neurons.

And these columns that aren't directly connected with each other, that we talked about when we talked about hindbrain and midbrain. And notice, the most rostral ones are in the midbrain. It did come up a couple times in a class. If you didn't remember, fine. But with the help of the quiz, you'll probably remember it now; the most rostral somatic motor neurons. If you include the endocrine system as motor, like Swanson does, then you could say, well there's another kind a motor neuron in the hypothalamus and even further forward. Secretory cells-- we're not talking about secretory cells now at all, just control of somatic motor neurons.

So I want to talk about-- it's a chapter centered on the studies of descending pathways, the anatomy, and their function. And these are the ones we just saw. I want to answer this question now. What's the basic spatial layout of motor neurons, and what are the spinal cord enlargements? How is it organized?

And then we'll talk about what connects to those motor neurons, and the main type of neuron that connects to them is interneurons in the intermediate layers of the spinal cord. Yes, there are coming directly from cortex, too. And we'll talk about those. Those connections connect mainly to those interneurons.

And this is the kind of figure you often will see in a medical school book. What does that mean? Is that Atlas holding the world up? No, it's he's holding a spinal cord up. But why do they have him pictured like that, with his arms like this? Because it's showing that the motor neurons in the ventral horns here, the motor neurons controlling the axial muscles, so the muscles of the neck or along the back, those are the axial muscles, very important in postural control. And they're most medial.

And then we talk about the girder muscles, the muscles that control shoulders or hips, separate from the muscles controlling the limbs. You find those next, moving laterally, and then the arms, and finally, the hands and feet, depending on whether you're in the cervical or lumbar enlargement. So this is the picture from the Lawrence and Kuypers study that I use in the book. It shows a section of the spinal cord. By just putting in a few neurons, they're showing you groups of neurons that are located medially and then more and more laterally.

So axial muscles, medially, then the girder muscles, and then the distal muscles. And there is some separation that you see in that other picture of the flexors and extensors as well. You don't need to worry about flexor and extensor separation because it is fairly complex. There's no need to memorize it.

But you need to know about the relation of the topography in the cord and the control of different muscles in the body. So now we know about proximal and distal representation in the cord. The next question, back to this one, is the interneurons. And, yes, they are radially arranged, so the interneurons way out laterally here do connect to the lateral-most motor neurons. Whereas the ones located medially, connect to the medial ones, controlling axial muscles.

And there's another difference. It's quite important. Some of those interneurons that connect to the medial motor neurons, project to both sides of the cord. Because when you control axial muscles, you're always dealing with movements that never-- they always involve both sides of the body. So both sides the cord tend to be involved. And the same thing is true for descending connections. Connections that are for the control of axial muscles, tend to be bilateral. That's a very important point if you're doing lesion studies, the function.

So then, in this study by-- it's a classic study by Hans Kuypers is a very well known neuroanatomist, a Dutch neuroanatomist, a colleague of [INAUDIBLE]. And his student Lawrence, that did this study back in the '60s, published it in *Brain*. And everything they found, applies pretty much to humans, as well, and corresponds to many findings in the clinic.

But what this, the upper section, show here are the patterns of termination of , first of all, at the left, the corticospinal tract, or the part of the corticospinal tract coming from precentral gyrus, the motor cortex. And it shows that it goes everywhere. If we include the postcentral gyrus, then you'll see it goes to the dorsal horn, too. So, pretty much everything in it also crosses the midline. This is just the left corticospinal tract. It goes to both sides, but only in that ventral medial area, the part that controls the axial muscles.

So then he's got pathways that travel through the-- come from the lateral brainstem, including the red nucleus, the midbrain. And they travel laterally through the hindbrain. They're joined by the axons from lateral reticular formation cells, and they terminate in this dorsal lateral part of the interneuronal group. They tend to spare the ventral medial area, for the most part, and they generally don't cross the midline.

Then, if you look at the medial pathways, medial brainstem-- I'm calling ventral medial here because they're terminating ventral medially in the cord. That's where you find the [? vectro ?] spinal pathway terminating, the vestibulospinal, the ones from the cerebellum, the vestigial spinal, and medial reticulospinal. There's a lot of different groups of axons. Center axons, always down in these medial regions of the ventral column, and they terminate in that pattern. Again, if you just look at projections from the left side here, they terminate more on the left than the right, but they terminate bilaterally.

So now we can draw pictures like this to represent these three descending pathways from cortex, in the lateral brainstem, and the medial brainstem. And I have this in color in the book, so a little easier. But all I do is show in a monkey brain with an enlarged spinal cord so we can see it a little better. Just separate the ones coming from the representation in the motor cortex of the body axis. That is the back and neck, and then, representation at the limbs.

AUDIENCE: So these aren't [? traumatic ?] motor neurons. They terminate in the--

PROFESSOR: These are not. This is a common mistake that most of your compatriots that don't take this class will make. These are not motor neurons. Remember the-- how do we define a motor neuron? And axon that leaves the central nervous system and terminates on an effector organ, usually a muscle. Those are the true motor neurons. These are pathways that, among other things, connect with motor neurons, but most of them go to interneurons.

And if it's motor cortex, they usually concerned with movement, but they go other places, too. They go to the cerebellum. They go to part of the interneuronal, the great network that now to describe. So I separate them on these two diagrams. And you just notice the ones controlling distal muscles, which I've drawn in solid line here. I shall show them. I show them in blue in the book. They're terminating mainly in the enlargements. So the two enlargements, you see them here, too. And see, the color helps a lot. It's a little harder to separate here. But the concept is very simple.

And then I show here the position of those corticospinal axons. Here they are throughout the pyramidal tract and the hindbrain. Here in the midbrain, they're in the middle portion of the [INAUDIBLE]. The other ones are going to terminate before they get to the spinal cord, a lot of them in pontine gray.

So here I show the rubrospinal axons, which cross and travel. Then they move laterally as they descend. And here they are in the hindbrain, right out here at the lateral edge. So now, the axons controlling the more distal muscles, if the anatomy is giving us the right idea, can be found in those two places; here and here. Except, if I want these to correspond, I have to say, here and here because these haven't crossed yet. These have crossed. That's a detail to keep in mind.

And then here, for the medial pathways, I show the tectospinal crossing, and then the vestibulospinal. They're uncrossed because they're hindbrain. And I could've added the ones from cerebellum. I do show a few from reticular formation here, that are joining that pathway. And those are the ones that go down through the cord and terminate in that ventromedial region. And here I show them in the cross-sections; tectospinal. I just show the tectospinal and the reticulospinal origins here. The vestibulospinal is only on this one.

So then, what are the three lesions that Lawrence and Kuypers made? Let's just go through the logic of Lawrence and Kuypers' study, very straightforward. They did try just eliminating the medial hindbrain pathways or the lateral pathways in otherwise intact monkeys. They didn't get long-lasting effects at all. So they reasoned that probably that's because the cortex is so dominant, they had spared the whole corticospinal pathway. So they all we started by eliminating the pathways from cortex. They cut the pyramidal tract. Yes?

AUDIENCE: [INAUDIBLE].

PROFESSOR: You have to talk louder. This ventilation is pretty rough.

AUDIENCE: So in the book, I remember [INAUDIBLE] there was a [INAUDIBLE].

PROFESSOR: The type, yes, you do get some recovery. But remember, there's a lot of different pathways involved here and it's very difficult to get all of them.

AUDIENCE: OK, so the--

PROFESSOR: And also, so there's various ways the brain can compensate, but it's remarkable how little the compensation is when they do a really good lesion. And then I ask the functions of these three major pathways. Well, when I introduced it with the output, you can pretty much predict the function, so we're going to go through that. And I want to know why would diaschisis effects of lesions of one of the descending pathways in the study be greater in humans than in monkeys.

So, we'll come back to that when we talk about functional effects of the lesions. This is their logic. You eliminate the corticospinal projections. They did that at the hindbrain level by coming in through the roof of the mouth. I've done this also in hamsters with a student, [? Katherine ?] [? Kalo, ?] who's been at the University of Wisconsin now for many years. I don't know if anybody else has attempted that on these little animals, but we did, and we succeeded to do it at least unilaterally.

You see this in sheep brain dissection. You can see the pyramidal tract. The problem is, you also see this huge artery called the basilar artery. So what we do-- and also, there's other problems. When you're doing that kind of surgery, you tend to stretch nerves in the neck, and that can cause them to hold their breath in and hold it because you're eliminating normal pathways involved in breathing. So what we found is if we just flood the whole area with ice-cold saline, we reduce the conduction so much in those nerves that they keep breathing just fine.

And then, we take a little spatula, nudge the basilar artery over. And we have a little spatula-like knife, very sharp. We know exactly how deep to go. And we can do what Lawrence and Kuypers did here. Here's their lesions; cut the pyramidal tract. This is the level they cut it. They're making a cut from the ventral side, just cutting the pyramidal tracts.

And usually, just above the pyramidal tract is the medial lemniscus. So they often did cut, damage, medial lemniscus fibers. So because of that, they had to have controls where they had lesions, for example, the dorsal column nuclei. So they got rid of the medial lemniscus fibers so they could see that the functional effects that they were seeing in their monkeys weren't caused by the medial lemniscus damage.

And then they allowed them to recover from that lesion as much as they could. And then they did one of these two situations. Either they cut the lateral pathways on one side. And remember, that doesn't-- it stays on one side so you can compare the two sides of the animal. Or they did the medial pathways. They tried it unilaterally, didn't get much effect. But if they did it bilaterally, then they got drastic effects.

This is what they're doing. They're going right through the damaged pyramidal tract. I mean, it's already degenerating, remember, from the earlier lesion. And they cut all those medial pathways. They all descend right near the midline. Yes?

AUDIENCE: I think Lawrence [INAUDIBLE]?

PROFESSOR: Yeah, that's a good question. There's is a little bit, and it's somewhat variable. It's not as much as in humans, but there is some lateral dominance. There is some asymmetry in the brains, too. The most drastic asymmetry they found on animals is in fish and you have venular nuclei. And they just still don't know a lot about why that is.

So here's what happens. You start with a pyramidotomy, get rid of those corticospinal fibers. You do it bilaterally. Initially, they seem almost paralyzed. And if you do this-- and here the question refers to-- this is the diaschisis effect, because they recover a lot from it. But why is that effect so much greater in humans? Remember, it's just quantitative. The pathway's bigger in humans. The motor cortex is even more important in humans.

So humans, after a pyramidal tract section, losing even their spinal reflexes for awhile. That's just like spinal shock, transection of the spinal cord. But the monkeys lose speed and strength and they never completely recover normal speed and normal strength, but they do recover a lot. Humans recover their strength. They never recover full coordination, but strength, they do pretty well. In fact, sometimes the reflexes become overactive, stronger than normal, because of sprouting that happens in the spinal cord.

But the one thing they do lose that's a qualitative difference from normal, is they can't do that anymore. They can't pick something up with single digits. They have to use their whole hand together. So apparently, those lateral brainstem pathways are quite capable of controlling fairly organized hand movements. But when it involves separation, separate control of the digits, they never really recover.

I mention here a question that I think I should give as a homework, but you will see a discussion in the book. I'll try to remember this as I'm telling it to you now. There is a discussion of fixed action patterns. It comes-- the only study I know of I did with a student, [? Katherine ?] [? Kalo, ?] the woman I mentioned. We found that they need the pyramidal tract coming from cortex to do their fixed action patterns of seed-shelling. So read about that, and then answer this question.

AUDIENCE: [INAUDIBLE]?

PROFESSOR: The corpus striatum for any kind learned movements, yes. But in this hamster study, we were looking at unlearned movements. The first time they ever see. We raised them in the lab, so you can-- they only see food pellets. They never see seeds. And yet, the first time I give a hamster a seed-- I can let them become an adult, and I can give them a seed, and they can pick it up and shell it faster than you can because it's inborn.

AUDIENCE: I thought [INAUDIBLE] fixed patterns of moving [INAUDIBLE] pattern. [INAUDIBLE]?

PROFESSOR: There are aspects of the sequence that are learned, but most grooming is unlearned, and so you don't get a permanent disruption. There's some very good studies by John Fentress. If you look his name up. He's at Dalhousie University. He's done many studies of grooming in mice, genetic effects as well as lesion effects, and showing that are fixed action patterns.

This second question, number seven, here, it refers to a study of motor system by [INAUDIBLE] lab here at MIT. So to read that, and see if you can figure out. I think I make it clear there in the book. And this one, we can deal with right now. You should know what Betz cells are. Does anybody remember?

And I want to know how it's related to the discovery of Fritsch and Hitzig. Who are they? 1870, the first guys to study motor cortex by electrical stimulation. They defined it as the area, which when stimulated-- it was a horrible time in neuroscience because they didn't have good anesthesia at all. So it was very difficult to do these experiments. They actually did some of it on humans; men in the Franco-Prussian War who had their skulls blown off, so they were dying soldiers.

They still did some of this work. Their first purpose was medical, but then they were able to do a little bit of this at the same time. But most of the work was done on dogs, and they were able to map out the motor cortex. It's where, with the smallest electrical currents, you can get movement. It doesn't mean you can't get movement from other places in the cortex. You can.

There are descending connections from visual cortex, too. They don't all go to motor cortex. Most of them, they don't go at all to motor cortex. But they do go to the brainstem. They go to the tectum, for example. So they control, orienting with this, directly from visual cortex by projecting to the tectum. So that's a type of motor cortex, too. But what we call the motor cortex is this specialized somatosensory area rostral to the primary somatosensory cortex in mammals.

Betz cells are the giant pyramidal cells that you find in that area that Fritsch and Hitzig mapped out as the motor cortex. It was four years later, 1874 that that Betz described these giant cells. They are the cells that give rise to the very long axons that go all the way down the spinal cord, and they no doubt include neurons that project directly to motor neurons.

AUDIENCE: Are Betz cells are cells in the [INAUDIBLE] area?

PROFESSOR: No, they're just the really big ones. Some of them are much bigger than others. You see Betz cells even in the hamster. When we did those sections of the pyramidal tract and we looked at the motor cortex, we wanted to know if the Betz-- the big cells or any cells degenerate. And they didn't degenerate. They just lose weight. They get smaller. They don't look as much like Betz cells. But we did cell counts, so we know that all the cells were still there. They just get smaller when they don't support a long axon anymore.

Just very quickly, the medial lesions, this is described in the book. You've got to do the lesion bilaterally. Remember the reason's because they crossover, And many axons do cross over. But you get drastic effects on writing. They did get some recovery. Why do you think they recovered? You asked me this earlier. Even after as much as 40 days, because there's other ways to write just with spinal reflexes. The input from the somatosensory system and from the joints can support some writing. but for the vestibulospinal tract is gone so they can use-- do the normal-- or tectospinal also.

They can't use visual, and they can't use vestibular input to write anymore. So when they walk, they stumble around. They don't know which way to go. I shouldn't say that. They know which way to go because they still control eye movements and you can see where they're looking. You can see where they're trying to get to. But yet, they can't make their body do it. It's a very interesting kind of, you don't really call it a paralysis, but they clearly can't control those movements well at all.

And then the lateral pathways they lose. We'll come back to this and talk a little bit next time, the way they did it. I actually have films of this, but I need to see if we can find something online so I can show it in class. The video, the film's, take forever to set up and show in the classroom. But it was a very nice study.

AUDIENCE: [INAUDIBLE] somatic [INAUDIBLE]?

PROFESSOR: Sorry?

AUDIENCE: [INAUDIBLE] motor neuron pool, were you looking for midbrain or the ocular motor?

PROFESSOR: They're both true because the ocular motor, the most rostral ocular motor nuclei are in the midbrain. So either one is correct.