Hello, everyone. Welcome to my lecture. Today, we're going to talk about the general sensory systems. In this hour, we're going to talk about the somatic sensory system, including the detailed receptors and their encoding. We're going to cover in more details about the pathways for touch and pro perception, as well as pain and temperature. These are acenin pathways, so we're going to cover from the peripheral, the receptors. And up all the way to our cribal cortex, this entire pathway. We will talk about the lesions as well as the clinical symptoms relevance. Please make sure you read the detailed objectives in the CPG, as well as the scholar brick. Back to the complex blueprint. We can see that the sensory disturbances and pain is a pretty important chapter of the blueprint. It's including tactile disturbances as well as vibration, temperature, proper section, as well as salts of pain chronic and neuropathic. Make sure you really study hard for this section. In the US MLE blueprint, very similar. You see the different types of sensory modalities being covered as well as the abnormal processes, including different types of pain syndromes, a long list of these. There are different sizes, sheets and speed of the A fern fibers. Showing here as you can see from left to right, you have different types of fibers, and so they have different characteristics. First of all, I'd like to point your attention to different diameters. You can see here, this one is very large diameter. Versus the smallest one, the C fiber, has a much smaller diameter. Also these purple aspect, these are mining sheets. The different thickness of miling sheets, as you can see from left to right, getting smaller and smaller. Then you also have the C fibers that's unique. It does not have any mining sheet. These C fibers are called melenated fibers. And the difference among this setup is actually laid at the conduction velocity. When you have increased diameter and increase the thickness of mining sheet, you're going to have increased conduction velocity. For these fibers, as you can see, these Alpha motor neurons will conduct a lot faster compared to the C fibers, mit fibers. Their speed can be about 100 fold difference among different fibers. Based on the conduction velocity as well as the exxon diameters, we could divide these peripheral fibers into myelinated and unelnated fibers. As you can see here, on the left, you have the fastest fibers on the right, you have the slowest fiber and it's correlated with the diameters. Largest diameters on the left will have the fastest velocity. The two parameters are connected very closely. And there are two different way of categorizing these fibers. You can see here, the A fibers are the myelinated fibers, and then the C fibers are unmyelinated fibers. Then these are correlating with different types of A fibers like A Alpha would be the largest diameter compared to the C fibers. You have type A fibers that's correlating to A Alpha. And then you have type four fibers that's correlating to the C fibers. You don't have to remember the axonal diameter of categorization But I'd like you to at least remember the A and C, these fiber division systems. Here are the fibers you need to remember. For the myelinated fibers, we have A and then for unmyelinated fibers, we have C. Under this A myelinated fibers, we also have fastest A Alpha fibers, that's mediating the pro prostion, as well as the motor function. If you recall at the spinal cord ventral horn, there are Alpha motor neurons, those are these type with Alpha fiber. These neurons has the largest diameter, thickest myelin sheet, so they're conducting the fastest. And A beta fibers are for the touch fiber. When we're talking about DCML, touch and per perception, they are A and A beta fibers, largest diameter fibers for the DCML pathway. For the pain and temperature pathway, dull pain and temperature are mediated by the C fibers. I want to point out one more thing for the sharp pain. Initially, when we feel something real fast, those sharp pain is actually mediated by fast conducting fibers, a Delta fibers. They're not as fast as the touch fibers, but they are thinly myelinated. They're still significantly faster than the C fibers. The C fibers also mediating and also postganglionic autonomic neurons. If you remember the autonomics, we talk about the gray mus and the white mus. The gray mus is these non Melinated, postganglionic neurons. Then for the preganglionic fibers, there are actually B fibers. This is the one that we haven't really talked about before. But you need to remember that B fiber is the preganglionic autonomic fibers for visceral sensation. Showing here, if you record from a nerve that the electrical change, the voltage change from the nerve, you would be able to see these different types of fiber and then forming different waveform. So if we gave a strong electrical stimulus to the skin, it's like an electrical shock to the skin, then you're going to be able to record three different forms. You can see the fastest one. The initial peak comes in very large peak. These are mediated by the A Alpha A Beta fibers for touch and pro perception. Then shortly after that, you're going to see the second peak, these are the A Delta fiber mediated, still fairly fast, and then you see the third a third wave, much smaller magnitude and was a very long delay. So that's C fiber mediated. So if you imagine when we're receiving some electrical shock, you would feel something real sharp. It's like a pin prick, and then you just withdraw your arm, for example. Then that's the fastest fiber mediated by the A delta fiber type of pain. Then after that, you're going to realize, oh, it's a bit burning at the skin surface too, those would be mediated by the C fiber. Faster for the sharp pain with A Delta and then slower C fiber, that would be the dull pain. And then different types of fibers mediating different sensory modality. Now, let's talk about the diversity of the receptors for the smatter sensation. By receptors, I

mean the initial very beginning of the sensory apparatus. We talked about the fibers earlier. Those either larger or smaller diameter fibers have the neurons with the cell body and then sending the ons both peripheral and central end. The receptors would be the, the apparatus that are located at the initial part of these peripheral central fibers. These apparatus can sense different types of sensory modalities. Somethings in the eyes like rods and cones, would be different from the hair cells and then would be different from the skin. You would have all sorts of apparatus to sense different types of sensory information. We're going to talk about them individually in the following slides or lectures, and you just need to know them in general for this lecture. We can categorize different receptors by their source or the types of stimuli. The source of stimuli give us the three different categories of receptors. They're called interoceptors, ter septors, and proprio sectors. You can figure out the meaning by the name. Interoceptors receiving the internal stimuli generated within our body. For example, the yalceptors that we mentioned in our autonomic lectures. It's sensing the blood pressure, to change the stretch of the smooth muscles, and those internal stimuli will activate the interoceptors. Exteroceptors are the general receptors that we had in mind. When we are sitting on something, when we're touching something, those receptors are activated. So exterior receptors are the general ones that we are all familiar with. Perceptors are the ones that sensing the change of the body positions like receptors that joints. They're special. Based on the types of stimuli, there are chemo receptors photo receptors, thermal receptors, mechanal receptors. Based on the name, you can figure out what are the sensory information they are activated by. Another special one that I want to mention is called nas sptors. Nas sptors is not limited to individual stimulus, but it's actually any kind of stimulus that are reaching the harmful or hurting level. When there's high intensity, either mechanical thermal chemical, whatever that can produce harm, then these non sceptors would be activated. Let's look at the different types of sensory sensors. First, let's look at the touch sensors. We call it touch. It's actually a little wider than touch. It's more accurately being expressed as external septors, because they are sensing the external stimuli, also called mechanal receptors, A kind of mechanical stimuli such as touch and pressure vibration. They're all included in this type of receptors. There are four different kinds that I want to highlight so Meissner's corpsals. You can see different morphology from the Merco disc and also the persnan corpsal and fini endings. They all have a different morphology at the end. Let's look at their functions. L et's look at the touch receptors in more details. As you can see here, for the receptors, you have something like shallower like Meissner's corpsle, and Merkel cells. You also have several different kinds that are deeper in the skin away from the surface of the skin. There pins corpuscle and fines endings. So Based on the position, you can imagine at the surface, they're mediating touch, like light touch and fine touch discriminative. They require smaller receptive field. The receptive field for the surface receptors are much smaller and fine tuning higher resolution. Compared to these preceding corpus or fine endings, they have much larger receptive field. Because they're mediating vibration pressure, they don't really require that high resolution, the fine tuning of the sensory modality. Then for each small or large field receptors, you would have fast adapting and slow adapting differences. Meisners cor pusle is fast adapting. You can see when you record when the stimulus is applied, you have a train of action potential and it stops quickly versus with the slow adapting the marco cells, after you give the stimulus, you're going to see a train of action potentials slowly slowing down. That's the slow adapting. And similarly for the percinan corpus, it's much quicker, have the action potentials then it disappears. Versus rapin endings, you would have a train of action potentials, slow adapting. How do you remember this? You can remember them by, alphabetical order. M and M, those are smaller receptive field. Then P and R, later ones deeper. They have the large receptive field. Then it's also alphabetical, MEI versus MER. Those are the fast versus slow adapting receptors, and P is a fast adapting, large receptive field and r is slow a large aceptic field. Hopefully that helps you to remember these The two point discrimination has been used for determining the touch sensitivity, the receptive field. As you can see here, it's basically using the two stimuli applied at a certain distance and ask the experimenter, are you able to tell these are two different dots? Then if they can, then you just keep shrinking those distance between the two points until they couldn't tell whether it's one or two. That would be the receptive field for that area. So as you can see here, our fingers has a much smaller receptive field compared to our back, you have a much larger receptive field for the touch sensation. Why is that? This actually reflects the density of those touch receptors. When you have the pack highest, that would be at the fingertips. It makes sense that during the daily life, we need a much finer sensation, be able to tell what are the different things touching our skin at our fingertips compared to at the back. You don't usually need to use the back too much in terms of sensing those stimuli. So you know, these areas that needed the most has the most density of the touch receptors to help provide that high resolution small receptor field. Let's look at the pro preceptors. These are the special receptors that transmitting the pro perception information. As you can see here, when the muscle spindle fires showing here in the red rectangle here, when the

muscle spindle fires, showing the contraction of the muscle, then the gg tendon organ that's located at the edge of the muscle spindle. Would be able to fire and transmit the information about the lens of the contra of the muscle fiber and the contraction. The joint receptors are also able to fire if there is a change of the angle of this joint. Basically, the joint receptor is able to sense the large degree of extension of the joint. Gauge tendon organs sends the force at the tendon. You can see here, these are yellowish gold. Color, these are the gauge tendon organs, and they are the free nerve endings winding through the collagen. Then showing here enlarged image like these are the tendon organs. They sense the polling force at the junction. Joint receptors are sensitive to the angle of the joint, and they're insensitive when the joint is a normal range. For example, here, at 100 degree, there is barely no firing at all in this joint receptors. When there's extremes or inappropriate directions, for example, when the degree become 140, or when there's maximum extension, then the firing goes crazily. That's the feature of the joint receptors. We mentioned that nasa spectors are special. They are actually responding to all kinds of stimuli as long as the intensity of the stimuli are beyond the normal range when it's potentially producing harm, that's when nasa spector is firing. You can see here there is two types. You have the first pain, and then you have the second pain. This is happening the first pain happens immediately, and then the second pain has a long delay and then it has a long masting painful sensation. So these are mediated by two different fibers. A Delta fiber is the one that remember it's thinly myelinated, so transmitting faster and it's giving us that first pain. And the C fibers are the slower unmyelinated fibers that give us that slower second pain. Give you a classic example. For example, when we're touching the host, and your fingers are immediately sensing that quickly sensing that sharp pain. That would be mediated by these A delta fibers. This sharp pain is actually lead to the spinal reflex. You're withdrawing your finger immediately before you even realize there's a pain and then you just have that fast response within the spinal cord. Then you realize, it hurts. Then later on after that fast pain dissipated, you're going to experience some slower pain. It's a dull aching and it lasts for a long time even during the development of the blister. That's our second pain mediated by the C fibers. We all have those sense that when you have something like a nurse injecting the vaccine to your arm, they rub the surroundings to help reduce that pain. The principle is that when you have the touch neurons activating an inter neuron here, it will be able to inhibit this projection neurons that transmitting the painful sensation from the C fiber. Basically, this projection neuron is activated by the C fiber but inhibited by the inter neuron that's being activated by the touch sensation. Therefore, the total or the sum of the signal is reduced. You have a reduced activation of the projection neurons for the painful signal. Now, the pain responses are really tightly modulated by the descending structures. For example, mid brain pons, medulla, they all have the projections sending down to the spinal. We'll go over that in the next slide. We talked about the mid brain PAG, peri acudato gray is able to activate the LC, as well as the Rafi magnus to releasenp nephrin and serotonin to inhibit the spinal cord modulate the spinal cord projecting neurons for the painful sensation. It produces profound inhibition of the pain. You can see here in cavalin being released by these projections from the PAG, LC, or RFI and release in of the interneurons in the spinal cord. That's why when the nasceptor is being activated here, the projecting neurons are receiving both inhibitor input from these interneurons and activation of the primary Afer neurons. I'd like to talk a little bit about it. Each sensation is also mediated by the C fibers. Is different from pain, but it's mediated by the C fibers as well. Why that scratching can help reduce the pain for response? I mean the itch and We actually have the similar type of theory that the pain inhibits, just like touch inhibits pain. With the collateral interneurons that produce inhibitory signals, this is similar as a gate theory as we talked about previously. The h can be either histamine sensitive or histamine independent. The histamine independent type of it is very hard to treat. Now, let's summarize our sensory receptors. Give yourself pause the video, give yourself a few minutes to fill in these blanks. Here's the answer. Hopefully, you got them all correctly. Let's do some practice questions? See if you can solve this question? Pass the video here. Here's the answer. Hopefully you got it right. We've covered the sensory receptors and the fibers in the periphery. Now let's go back and then look at the entire pathway, the ascending sensory pathways. Now, do you guys remember what are the DCML and spinosymic tracks mediating? What kind of signals do they transmit? The answer is the DCML is transmitting the touch and proper section and spinosymic tract is mediating pain and temperature sensation. These different sensations have a commonality. As you can see here, drawing, there are three neuron relays. You have this first order neurons at the peripheral, and then sending the information into the spinal cord, then it's a sent to SNFs at the secondary order neurons. In this case, in the DML case, the secondary order neurons is located in medulla. Then they will ascend again to the thalamus to meet with the third order neurons. Then the thal neurons will go ahead and go to the somatosentric cortex, which can be considered the fourth neuron, but usually we just say the relay takes three neurons. If you put together what fiber we mediate in this pathway, if you remember our touch and pro perception has these large diameter A Alpha or A Beta fibers

to mediating touch and pro perception. Their large diameter heavily myelinated fibers. Then on the receptor side, they have very specialized that's Mcener corpus, Mcal cells, and so on. That's the receptor and the special fiber that's coming in. Then for the spinothalamic tract, you also have this primary A fern neurons coming from the periphery and then C neaps right away to the secondar order neurons in the spinal cord. Then again, it has the third order neuron in thethalams. To be more specific, that would be the PL for the body. And then it reached the Sematacenry cortex neurons. We can either call it force neuron or just know it's the neurons in the Sematacentr cortex that gave us that final sensation. So in this case, for the pain and temperature sensation, the primary A fib neuron would be that C fiber, the slow malnated fibers, or the fast pain is mediated by the A Delta fiber, which is thinly myelinated fast conducting, but still slower compared to the A and A Beta fibers for the DCML pathway. All right. Here is a primary aferns that's our first order neuron. Remember we talked about the cell bodies of those first order neurons is located at the dorsal root DRGs. These DRG neurons has a special name called pseudounipolar neurons. The reason that they are pseudo unipolar because it looks like bipolar. They looks like two branches of sounds. In fact, this is pedo unipolar because they only have one sounds that from the cell body sent out to the central terminal to the spinal dorsal horn. This peripheral afferent fiber is actually not a sound. That's why it's called pseudo unipolar. And so these axons comes into the dorsal horn of the spinal cord. If there's any damage to these pathway for the primary aferent, then you would have ifs lateral deficit for the sensation. The secondary order neurons sending the axons out and always the secular order neurons are having the deposition. If you recall, we talked about the secondar order neurons for the pain and temperature sensation is located right at the spinal cords horn and then pass the mid line through the anterior and join the spinothalamic tract. The touch and perception, you have the receptors ascending is laterally without synapsing to the secular ter neurons until reaching medula. When they reach the medula, it will synapse at two nuclei at the medula level, and then use the DCML, the medial amnscs part. This is a dorsal column part. Within the spinal cord. The medial amnescs is passing the mid line to the other side, and then the signal goes up to the thalamus. The damage at the spinal level for these two different tracks are causing two different lateralizations of deficit. As we talked about in the spinal collectore, it's the lateral loss of touching perception and then counter lateral loss of pain and temperature sensation. So when you have the damage that's above the decosition right here, above the medula level, then all of these areas would generate the contralateral loss of both sensations. Now, let's go through these special sensory pathways one by one in great details. The first one is spinothalamic pathway. Spinothalamic pathway is transmitting the pain and temperature information and also each. Here I put a stop sign for the facial or syphic information, so you can ignore that at this point. We're just going to talk about the body somatosensory information. Here, the primary afferent has their cell body located in the dorsal gangln or DRG. The signals coming from the peripheral reach the cell body and the central terminal of this sensor neuron will see as at the dorsal horn and to send the signal to the secondary order neurons. The secondary order neurons. Remember, we talk about they are passing the base passing the mid line, make the deposition. These order neurons passing the anterior it commissure to reach the spinothalamic tract. This spinothymic tract is now formed on the contralateral side of where the initial central input coming in. The spinosymic tract goes all the way up to the thalamus. Do you remember which nucleus that for the body sensation for the spinal from the body in athalms. Here it is actually called VPL. VPL is for the body sensation and now reach the third order neurons, CX at the third order neurons, and these neurons will send ons out to the fourth neuron at the primary somatosensory cortex. This pathway is using internal castle. To be specific is a posterior limb of the internal castle to reach the primary somatosensory cortex. Because of the deposition happens very early on at the spinal level, therefore, the damage to the spinosymic tract almost always causes contra lateral deficit. For the DCML pathway, DC dorsal column, ML is medial bnscus, is because of the true part of this pathway. The primary Afer neurons again comes in bring the peripheral information for touch and for perception to the spinal cord. It does not see, but instead, it's going up. All the way to the medula brings the medula, and snaps here to the secondary order neurons located at the nucleus croclls, or nucleus cuneatus. The two nuclei is separated because the nucleus croclls is receiving information from the lower body. As you can see here, and the the nucleus acculeats is receiving information from the upper body. This is part of the asicul eats that receiving the information from the upper body, and now you can see the cervical spinal cord dorsal column has two parts, the faults and the faicul gracs. This is a dorsal column. Then after it passed the secular order neurons sending out the axons cross the midline. Remember, secondar order neurons is a one deficit. This neuron pass the mid line and form the medial mniscs pathway. Now goes all the way up to the thalamus. Again, the camic nuclei that's in charge of the body sensation is VPL. It snaps here to the third order neurons in thethalamus, and ethalama send out the exxon to the somatosensory cortex. That's the fourth neuron there to have the sensation for the touchin perception. Again, via the internal cale

posterior limb. If we have damage at the spinal cord level, then you would have the deficit that in the ipsilateral side of the body with attach and p perception. But if you have damage above the medula, then the deficit would be on the contra lateral side. Let's talk about the facial sensation. On the face, there are the main sensory fibers are trigeminal nerve, the cranial nerve five. There are three sensory trigeminal nuclei and one trigeminal motor nucleus. There are a total of four trigeminal nuclei. Among these three nuclei, you have the principal or main or pontine. These are the same things, different names. These trigeminal nucleus is located at ponds, it's for the touch and pro perception. And this is equivalent of the dorsal column nuclei for the DCML pathways. Those are the nucleus gracilis and nucleus cuts, located at the dorsal medula. And so the pain and temperature signal is transmitted by the spinal trigeminal nucleus. It's called spinal trigeminal because it actually extended from the medula all the way down to the cervical spinal cord. But main portion is located in the medula. So this nucleus is equivalent to the sector order neurons at the spinal cord nucleus propias for the spinothylamic tract. And so they receive the non seceptive signals of pain signals as well as temperature information. The third one is a special sensory nucleus. It's called mesencephalic. Mesencephalic meaning midbrain. This nucleus is located in the midbrain. What makes it so special is that these are actually the first order neurons. They're part of the trigeminal ganglia neurons that's supposed to be in the peripheral, but is now located inside the central nervous system inside midbrain. It's equivalent of the DRGs in the spinal cord. It has a special purpose, mainly just transmitting the pro perception of the draw muscles and periodontal regions. And these sensory fibers through the trigeminal nerve coming in without synapsing at the pose level, but just coming up to the mid brain and the cell bodies located in the mesyphalic trigeminal nucleus. Then these trigeminal nerve coming in with our mesysphalic trigeminal nucleus cell bodies, and then send out axons to snaps at the trigeminal motor. Nucleus to form this jaw jerk reflex. This trigeminal motor nucleus comes back to the jaw to produce that muscle contraction and relaxation. The whole purpose of this jaw jerk reflex is to reduce the damage potential damage to the teeth when we chew. This jerk reflex, it's a small function, but it's very useful. Let's talk about the detailed pathways for the trigeminal system. The first one pontine trigeminal pathway. This is to convey the touch and pro perception for the head and phase. It's similar to the DCML for the body, but slightly different root. Let's go through this. It also has the first order neuron or primary Af neuron, has a cell body located in a ganglion called the trigeminal gangl. Just a very similar to the dorsal gangln for the body. Trigeminal gangln has the cell bodies, and those are pseudonunipolar neurons. Then the primary a ferent to the ponds. You guys remember the cranial nerve, trigeminal nerve is at the ponds, and then enters the ponds caps to the secondary order neuron here at the principal or pontine trigeminal nucleus. And these secondar order neurons. Remember for the acndin pathways, the secondar order neurons are the ones sending out the exon and decos it. They cross the midline, goes to the contro lateral side and then ascend from there. These acndme passing the midline, passing the mid brain reaching thalamus through trigeminal lemniscus. Here at the thalamus, we'll see that to the third order neurons. And do you remember where exactly in thethalamus? We have these third order neurons for the face. If you don't remember, here's the answer, the VPM, very pretty makeup. That is for the face and head sensation in thethalamus. From VPM, will send the exons from third order neur axons A sending up again. Do you remember the pathway here, it's internal capsule. Specifically, it would be the posterior limb of the internal capule. Along the internal capule, it will reach the primary somatosensory cortex, which is located at the postcentral gyrus. Here it will snaps with the cortical neurons to have our conscious sensation of the phase. Sometimes it's called fort neurons, but rarely people refer that. Now let's look at the lesions. I want to mention specifically about medula lesion because it's special. Here, you can think about alongside this pathway. It's at the pontin level, pontin or above. If you have the lesion below the ponds, then you would not have the deficit for the touch and pererception for pace. What about for the body? For the body, you know the DCML pathway cross the midline, now would be deficit for the body, if you have the lesion at modular level. That's a unique characteristics. You have the contra lateral body touch perception deficit, but without facial or head deficits for the touching perception. Now, the even more special trigeminal pathway, the spinal trigeminal pathway for the pain and temperature sensation of the head. For these pathways, the trigeminal gangln with the cell body of the primary a ferent, sending the information into the ponds, so it enters the brain stem through the ponds level, but it does not see nes here. Instead, it descends and travel down to medula and cervical spinal level to reach the secondar order neurons at the spinal trigeminal nucleus. This spinal tritamino nucleus now send the exons across the mid line to the other side, and then go up again past the ponds mid brain, via the tritamnothalamic tract and goes all the way up to the thalamus to reach the VPM, third order neurons and thethalams. These neurons are VP, very pretty makeup. Then the third order neurons send out the axons again to the somatosensory cortex via the internal capsule posterior limb. Now if you have a lesion at the pontimdula junction, you would have a very special

deficit. Because of the phase, you would have already the pain and temperature sensation cross the mid line to the other side, already deficit. You have the contralateral deficit for the pain and temperature sensation for the phase, but then you would have this if s teral deficit for the u for the touch and pro perception, because the principal or main trigeminal nucleus are located in the pounds. So just a Now, let's look at the fourth order neuron in the primary cemeta sensory cortex at the postcentral gyrus. There is a homunculus sensory hermunculs, meaning it's a small human that's shown here. You can see the foot is at the mid line at this postcentral gyrus and then comes in a little bit more lateral is the leg and then trunk. And then on the way lateral side, you have this big representation of the hand, as well as a very big representation of the face on the very lateral side. This is a small human distribution, you need to remember it. Mainly the leg is more medial, and then trunk and then hand and face, and then the to fangs. Now we've learned the entire cemento sensory pathways from the body and face. You should be able to start from the receptors and then go through different nuclei and then thalamus, and then cortex. Now, fill in this pathways yourself. Now, here's the answer. Hopefully you were able to draw those all out. Here I'd like to mention that the body for the touch, you have thalamus VPL nucleus mediating the touch sensation of the body as well as for the pain. Both of them are VP for the body and they reach the cemeta sensory cortex. I have two additional pathways here for the pain pathways. You have the VPM. Even for the body VPM and me dorsal medial nucleus, these are reaching insular and the singulate cortex. This is for the effective component of the pain. Affective component meaning the unpleasantness of pain sensation. Similar for the phase, you have the two pathways to reach these limbic structures to mediating pleasantness of pain sensation. Shown here, why do we feel the painful response are not pleasant? Why they are unpleasant is because of those two brain areas that being innervated by the signals being sent to those brain areas. If you remember I mentioned in our limbic structure lecture, if we do the single lotomy, If we damage the single cortex, then the person would not be bothered by the painful response, but they could still tell, this is the painful stimuli, but I don't care anymore. It doesn't bother me anymore. Basically largely removed the effective component or unpleasantness of this painful sensation. Another special phenomena called phantoms of the body. The phantoms usually comes from all of a sudden, the lose or removal of the body part, usually extremities due to surgery or accident. This type of sensation is related to that last body part that you still feel like being touched, it still exists in your brain. A lot of times these sensations are painful. It's called phantom limb pain. And the phantom limb pain is quite resistant to medications. The cause of it is not super clear. Current hypothesis is that the cortical representation of this last extremity takes time to be reorganized and it takes time for the brain to learn that that limb is not existing anymore. Down here, you can see a gentleman with a mirror trying to fold the brain to say, Okay, even if he lost the right mb, but his unhurt the left leg is still there. Looking at looking into the mirror, it's telling his brain, my right leg is still okay. And that can usually diminish the phantom limb pain. It's a pretty strange phenomenon, but basically you're trying to fold the brain again to ease up that painful experience. Fortunately, the phantom limb pain diminishes over time. These are the review for the most part. How do we think about whether it's ipsilateral or contralateral deficit when you have a lesion, or conversely when you have a deficit, which side of the body, which side of the nervous system is lesion. You need to be able to figure that out by now once we went through all of these pathways. Remember the general rule, when we have lesions above the deposition, it costs a contralateral deficit when we have the lesions below the deposition, then it will cause ipsilateral deficits. If you don't remember this part, go back to our spinal cord lecture, please. Now, test yourself, if we have a injury at the specific levels here, then what exactly side of the deficit you're going to see. Make sure you differentiate both body and the head. The body and head may be different in terms of specific level for this different neuraxis. Here's the answer. I'd like to emphasize or explain a little bit more about the pont lesions that related to the pain and temperature sensation. As you can understand that the contra lateral body, this makes sense. For the face, I put both EC and contra lateral deficits if we have lesions at the ponds. This is due to if we have the pontine lesions at the lateral side. Then we most likely are only damaging the primary Ahern fibers, the primary neurons. That will cause ifs lateral facial pain and temperature sensation deficit. If the lesion is a little more medial at the pounds level, then it might damage the contralateral fibers, that the tritamopyamic fibers. If that's the case, then you would have the contralateral pain and temperature deficit and the face. If you have any questions, please reach out to me. And this is again a summary slide. Make sure you're able to fill in the blanks. Here's the answer. Here is my answer. If you have any issues and questions confused about anything, please let me know. We can definitely set up some sessions. You could come to my review sessions to clarify these. Now, pause the video to see if you can solve this question. And here's the answer. Hopefully you got it correctly. Question number three, now pause the video, please and see if you can answer this question. Here's answer. Hopefully you got it correctly. This is a pretty complex case. This is actually a round Sacar syndrome. By now

again, you should be able to figure out what the deficit which side of the lesion, this type of presentations are generated and why exactly you're seeing all of these deficits. I have explanation in the next slide. Basically, because of the loss of the sensations of this one entire segment, you would expect to have this damage to the entire side of the spinal cord. We call it hemi section of the spinal cord. Because this is a lower body that experiencing all the deficit. The damage has to be at the lumbar spinal cord, Then correlating to all these losses, all these sensory losses, we are we should be able to figure out these losses on the left side of the spinal cord, and we have all these numbers correlating with the lesions. Go over this one by one, see if it makes sense to you. That concluded today's lecture. Make sure you understand all of those information and be able to fill out the answer sheet. I know these are pretty hard materials, especially the lateralization of the lesions versus deficits. If you have difficulty, make sure you reach out to me and we can make appointment and individually. If you have any general feedback, comments, suggestions, send these along using this link. Thank you very much.