Hello. My name is Gonzalo Tassu, and today we will talk about the neural anatomy of the visual system, including I functional anatomy and vision pathways. Students should be able to describe the neural muscular and vascular anatomy of the eye. You should also be able to describe the neural processing within the eye. You should be able to treat visual pathways, underlying visual perception, and you should be able to relate visual deficits to these functions to specific portions of the visual pathway. The star with the. The i has three consecrissue layers and a lens. The first layer is the sclera. In front of the pupil, this first layer is transparent, the can go through it and on the other parts, is not transparent, it's called sclera. Now, the second layer is the chord. The Corid includes a vascular layer, the ciliary body, and the iris. And the ell layer of the eye is the retina. The retina is divided in two parts. There is the retinal pigment epitlium, which in this section, we can see it around here, which this space this contains the blood supply to the neural retina where the photo receptors are located. Here. Now, the space around the pupil is divided in two spaces. There is the anterior chamber and the posterior chamber. The anterior chamber is the space from the iris to the corn, and the posterior chamber is from the iris to the lens. The anterior and posterior chambers are filled with cus uma. Also, it's important to remember that in looking at the retina, that the retinal pigment epithelium is where the blood supply is. Where the neural retina separate from the retinal pigment epithelium, the blood supply to the nerve cells will be compromised resulting in neural death and blindness. This condition is called retinal detach. They this is an optic element, the shape of the eye is very important to have proper focusing of light. The way contrary to the cameras built by humans, which are rigid, the eye shape is maintained due to the interocular pressure. So Here, the ciliary body is going to be secreting the aqueous Umer into the posterior chamber, which can be seen here. This is the posterior chamber, and this a Umer flows from the posterior chamber into the anterior chamber. The trabecular meshwork, which is similar to the arachnoid granulations at the docornal angle are responsible for the drainage of the cus um. The endotum lined scleal venous sinus, which is also called the canal of schlem communicates directly with the venous drainage of the eye. Now on the other part of the eye close to the retina, here, the ts is filled with a gelatinous material, so pressure on the anterior i is transmitted to the rest of the eye. The pressure is controlled here and is applied. The excessive intraocular pressure can is decremental and that condition is called glaucoma. There are two types of glaucoma. One is the open angle glaucoma in which the flow of fluid is maintained. However, the problem is in the trabecular meshwork, that there is not enough or not fast enough drainage of this fluid, and then the pressure increases. Now, the second form of glaucoma is the close angle glaucoma here mechanically, the ris blocks the drainage angle formed by the cornea and iris, blocking detrabcular metwork. This is a medical emergency. It is characterized by redness and the eye, nausea, and headache associated with blood is. The cornea and the lens focus the images on the redine. Focus in an image require refraction that is bending of light across one or more interfaces when there is a change in the refractive index. For example, you can see this in this glass of water. The cus and virus mo refractive indexx are only slightly lower than the refractive index on the lens. The lens, although we might think that the lens is the main optical element in the eye, actually most of the eye's refractive ability is at the air interface at the surface of the corn. However, the lens can provide some accommodation. The eye accommodates for close vision by tightening the ciliary muscles, allowing the pliable crystal in itself to become more rounded. When the ciliary muscles contract, and we can see the ciliary muscle here in red, the ciliary muscles contract, then the sul fibers, which connect the ciliary muscles to the lens here. The sul sul fibers relax, and the lens acquires a rounded shape. A more rounded shape is going to allow the light to bend more. In this case, the accommodate the close vision. When the ciliary muscles are relaxed, the soul fibers are more intension and the length is flat. You can see this is look like a window, so there's not going to be as much bending of the light. When the light comes from faraway objects is going to focus on the retina. This process is called accommodation. The other optical element is the iris, which is the aperture here that permits the light to reach the retina. The pupil is the aperture in the middle of the iris. The pupil can close, and that is under parasympathetic control, or the pupil might dilate and that is under sympathetic control. There are different set of muscles, but different control. We have these radially dilator muscles. When they contract, there's going to be there's going to be opening of the pupil. There is also a spin like where the constructor muscle is located around the pupil when they contract, this is going to be like a spinter, and it will close the pupil. Smaller pupil, has some advantages in that it produces larger depth of field and we'll explain that this later. But the con is that there is less light entering the ice. The aperture is smaller, they're going to be less lighting, less light coming into the retina. However, there is some advantage to that. This is a little bit complicated, so bear with me. The how com P contraction provide depth of feel of view. Here we have a diagram of of the i with a lens with is n. Let's imagine that there are two objects, x and y. Here we have a large pupil. In the lend, the object x is in focus. All the lie that comes out from x is going to be focused at the red. However, object y it's not on the focal plane,

it's somewhere else. What happens is you can see by the geometry when the pupil is large, there's a more light going on. But the focus is going to be happening away from the retina. But the actual passage of this y object is going to create a large object. We'll see x in focus and y will be completely out of focus blur. However, But on the other hand, a lot of light is coming in. Now, if the pupil is small existe focus, that doesn't change. However, y because of the smaller pupil, is going to be still going to be out of focus, but much less sol. The size of the object is going to be smaller. You look more as in focus. Smaller pupils allow objects that are not exactly on the plane of focus to appear focus. We'll have a deeper field of view with a smaller pup. This is known. We know this from cameras so that the iris is plays the same role in photography at the aperture. If they have a large aperture, only the objects that are on on the focal plane appear crisp and the rest appear blur. However, if we close the aperture, both the objects that are focused and the objects that are away from that plane of focus are going to appear more in more crisp. However, if we close the aperture, there's going to be also less light coming in. The brain should be able to adapt the aperture depending on what do you need. When we are focusing on a nearby object, three things happen in a reflex manner. The convergence of the two I, because of geometry. You contract the ciliary muscle, so you produce accumulation of the length and your pup contracts. Allowing larger depth of the field of view and then allowing more of these objects to appear in fox. Cortex and cebalu play an important role in this near reflex. L et's now describe the neural processing within the eye. Here in the retina, in the retina, we have photo receptors. These photo receptors are going to respond to light. Then the information from the photo receptors is going to travel through the bipolar cells. Here we have the photo receptors. Then we have the bipolar cells, and the bipolar cells are going to provide input to the ganglion cells. The gangln cells are going to provide the output of the eye to the brain. There's also an internal processing in the retina. There's amacrine cells. Here and horizontal cells. You can see that these cells are going to be combining information across multiple photo receptors and it's going to modify the information flow. Something that is also important to notice is that the retina, the location of these cells in the retina is reverse to what you will suppose. Actually, the light has to cross through the ganglion cells to the microcell bipolar holisontal cell to finally reach the photoreceptors. Light goes this way, but information flow goes the other way. It goes from the photo receptors to the glaglu. There are two types of photoreceptors. They are the rods and the cones. This is actually a descriptive name, the rods are rod and the cons are. These photo, characteristic they have very large membranes. The reason to have large membranes is allow to collect more light because the sensitive elements in the photo receptor cells are in the membrane. There are different opsins in the rods and cones. Rhodopsin is in the roads, and the con pigments are in the codes. The o the opsins by to 11 cs retinal, the light is going to summarize the 11 cs retinal to all trans retinal that is bound to the opsin. The light inducing isomzation of the retinal causes a conformational change in the opsin. This activates transducin, which in turn activates an enzyme, phosphodiesterase that hydrolyzes cycle GMP, and decrease availability of cycle GMP causes the cycle GMP gated cation channels to close. And the cells hyperpolarize. That's interesting, contrary to other receptor neon. This photo receptors You can see that the membrane potential becomes more negative when they are activated by light. Now, also, you notice that the rots are larger than the cones. This means that the rots are going to be more sensitive to light, so that you need less light because there's more membrane to activate. But you'll have better resolution with the cons, we have smaller but less sensitive. Also notice that because of the cells hyperpalyzin in response to light, photo receptor reduce their neurotransmitter release in response to light. The rods are more sensitive, but they saturate here you can see an electrical trace of the response of rot to different amounts of light, and you can see that there is saturation, the level of light is increasing, but the output of the rot is not increasing, there is saturation. In contrast, the cons there seem to be less sensitive. You can see that the responses are smaller, but there is no saturation. So and these rods and cones play different roles in the red. Because of this specialization of the different type of cell, the retina is not uniform. Here is a endoscopic view of the left retina. You can see here the optic disc. The optic disc is where the optic nerve is going to be attached to the retina. You can see that the arteries and veins come from the optic disc. This optic d has no neither cones and rods, it's actually bland. There is no light sensitive cells here. The opposite can be say about phobia. Here in the phobia. You can see that there is no blood vessels there. Is only going to have photo receptors there. There is a high concentration of cones in the phobia. This is the place where we have the most accurate vision. Remember the cones are smaller and they are sensitive for color. There is also high concentration of rots outside the phobia. Which is interesting is that the phobia, which is the highest resolution area because they are not many rods there is not the most sensitive part. Sometimes when you, for example, are looking at the night sky and there is a very dim star that you want to look at. If you look directly at it and put the image on the fobia, you won't be able to see it. But if you move slightly your eye slightly away, then you're going to put that image onto parts of the retina that have the rods which are more sensitive, and then

you'll be able to see that dim star. We say that the near the edge of the optic dis is a circular portion of the retina, about 5 millimeters in diameter, that's called the macula lutea. The center of the macula is a depression called the phobia, which we talk about, which is particularly rich in cons, color vision. The central phobia is specialized for vision of the highest equity. All the neurons and capillaries are present elsewhere, and the periphery, you have cons and roads, so you have black and white and color vision. Other Another region of the retina is the optic papilla, as the optic papa, optic nerve penetrate the retinal layer and leave the eye. And the retinal blood supply enters and exit. This creates the blind spot in the retina. The blind spot is located 15 degrees temporarily from the that seem like absual space, and we are not aware of this blind spot because the nervous system fills. Here is a demonstration. If you now cover your left eye and starter the plat in the green square, and now move your head closer to the screen and at one point the circle will disappear and your brain will fill it in with yellow. You have at that point, you have put this circle into the blind spot, but we don't have a conscious perception of the blind spot. Our brain is smart enough to fill it out based on the surrounding images. Now, remember, what we were talking about the flow of information is is in opposite direction of the flow of light. However, that having the light, travel to the ganglion cells, and these other parts of the retina would be detrimental for the phobia because we want to have the most accurate vision there. There, what happens is there is a high density of cones, but there are no blood vessels or other cells in the light pad. The phobia is the place where the light directly binges onto the photo receptors, and that's where we have our maximum equity. What's the side of our phobia? You extend your arm in front of you and now look at the side of your thumb nail, that's the size of your phobia. That's the side where you have highest equity. But we are not aware of that. We have this illusion that our whole field of view has high equity, but that's not true. What happens is that we are always moving our eyes like this famous picture where when we look at the path, we have the impression that we are looking at this pace and we have high equity all over the path. But what's happening if we look our eye movement here, we keep moving our eyes around on this cade, and then we our brain creates this high resolution image. But that is an illusion. So that is mostly bring into attention by conditions like H related macular degeneration, AMD, AMD causes damage to the macula, but spares most of the retina. So if you think about it, extend your arm, think about that and think about the size of your to um nail, you're saying, Okay, that part of the retina is damaged, no big deal. However, and you are not blind, but you lose the ability to see because the loss of the central vision in AMD interfere with simple every day tat such to the ability to see faces, to drive, to read, write, and do close work, such as cooking or fixing things around the house. Although the affected area of the writing is pretty small, the effect is quite large. Now there are three type of cones. Cones are going to provide us with the color vision. We have the S cones, we have the M cones and the cones. S comes from small, medium, and large. Here we are plotting we're seeing the wave length. Longer and shorter wave lengths. Shorter wavelengths are more towards the violet or and the longer wave length, some of 600 nanometers are for the red colors. If you remember your quantum mechanics, you will know that the longer wave lengths have less energy. The quanta of these longer wavelengths have less energy. It's actually hard to design detectors that to have detectors that detect that low energy. In fact, you can see that both all the even the elks that are responsible for red vision, its peak absorption is yellow green. But hitting their tail, they are they still respond to red, and they are going to be given the perception of red colors. Now looking at the output cells of the retina. They are the ganglion cells, the ganglion cells respond to contrast. They actually have a center around structure, they are more responsive, for example, for a little dot s dark dots surrounded by light. There are different type of gangln cells. The pumerically dominant class of small gangl cells are sensitive to color and form, and they are large gangln cells that are more sensitive to movement and contrast. Oh. Now we will look at the visual pathways underlying visual perception. Visual field and retinal fields. The visual field of each eye is the region of space that the eye can see looking straight ahead without movement of the head. Now you can look, close one eye. That is the visual f of one eye, close the other eye, and now that's the visual field of that eye. The phobia of each retina is going to be aligned with a point called the fixation point in the visual field. Peripheral images fall on the nasal hemiretina. We have the two visual fields, this is a left half and a right half. This is the visual field. Now, what happens is that the peripheral images fall on the nasal hemiretina. Here now we are in the retinal space, and here you can see that there is a cross. There is a cross on where the image are projected. That's not a neural effect. This is just an optical effect. Remember that we have lenses and the prenss of the lenses causes the images that are projected in the retina to be inverted. This inversion that you are seeing here is produced by the optics. The end result is that the peripheral images are going to be projected into the nasal hemiretina. The part of the retina that's close to the nose. This is the periphery here and here on the nasal hemtin. Now let's just to be clear, let's focus what happened on the left eye in the binocular field, which is the part of the visual field that is seen by both eyes. Images in the left half of the visual field fall on the nasal

hemretina of the left eye. Images present in the right half of the visual field of the left eye fall on the temporal hemo retiny of the left eye. So Let's see what happens when we all are looking at an object. Let's imagine that we're looking at this arrow and here we have our retinas. From the point of view of the left eye, you can see how this peripheral object is going to be projected onto the nasal hemiretina. Now in the retina, we have the ganglion cell axons, which travel to the optic iasm. There's going to be partial decation of the optic nerve fibers into optic tracks at the optic iasm that is going to permit by nucular vision. If you think about it, you can see how this part of the arrow is in the temporal hemiretina of this eye, but is on the nasal hem retina of this other ei. These two. In order to create binocular representation, we need to bring together these two images that are on different eyes. What happens? A fibers from the nasal half of each retina cross decosate, to the contralateral optic track. In that way, the images from both eyes of the same part of the visual field can come together. The All fibers from the temporal half of each of each retina, are going to pass through the lateral portion of the optic asm without crossing and entering the ipsilateral optic tract. In the optic asom anatomically, the decosting fibers from the nasal hemiretina that are going to be representing the peripheral vision are going to be in the middle. What happens is that the optic asm is on top of the putatoric gland. A tumor of the putatoric gland will cause pressure here on the optic i asm and what is going to be mostly affected is going to be the meatal hemiretina projections that are going to correspond to the peripheral visual field. Here is an example where we can see an MRI of normal MRI, and you can see here a patient that has pituitary adenoma. You can see here the opticasm, and you can see how the optic asm is going to be be pressed here in the center, which is the part where you have the fiber accusation. In that case, the symptom that you will get from this pressure is going to affect the temporal feel of view of both dyes. You're going to be having peripheral vision loss. The after the optic asm. Most of the fibers in the optic tract make synapsis onto the lateral genicular body, the LGN of the talus. You can see that here in this section of the brain, you can see the optic tract, and the optic i asm, you can follow this, and this is going to go into the lateral genic, the LGN. Neurons in the LGN are contacted by fibers orientated from one i and have monocular receptive fields. Las 14 and six are the most superior ones. Received from the contralateral I and layers two free and five from the xteral. There is a separation in the LGN depending on which layer, from which I that input came from. There are also magnocellular layers one and two that respond to contrast and movement, large, magno large, and the parvocellular layers, parvo respond to color and forms. You have higher resolution, lower resolution areas in the LG. Now you also have the LGN. The Talamul has to project to the visual cortex. Now, the visual cortex, as you probably are aware, is located in the occipital lobe. There is a genicular calcarine tract which goes from the LGN to the primary visual cortex, which borders the calcarine sulcus. There is a radiation which are the actions that go from the GN, and these actions radiate in a broad sheet around the lateral ventricles. There are two main pathways for that to reach. One part of the visual field is going to be to traveling through the mayor's loop that will correspond to the superior visual field, and that goes through the temporal loop. One way to remember this is remember in vision, everything is going to be reversed. So the mayor loop travels through the temporal lope. The temporal lope is more inferior. Of course, that's going to represent the superior visual field. So you have damage to the temporal lope, then you may affect the mayor loop, and you have the defect that is called Pi in the sky, where the person is going to complain of loss of vision on the upper visual field. Contralateral to the loss of field of view. There is also the upper optical radiation. And which goes that passes under the parietal lobe, and this is called the inferior optical field. You have loss of the inferior optical field, that might indicate that you have damage in the parietal lobe. When you have blockage of the middle cerebral artery and its branches, it might affect the male loop or the upper optical radiation. Individual fibers carry information from only one eye. Damage here often results in deficits that are overlapping but are not identical in the two eyes. Now, in the primary visual cortex, the primary visual cortex is located in the occipital lobe, as always in the visual field, everything is reversed. The inferior visual fields of the inferior part of the word pre to the cortex above the calcarine sulcus, and the superior superior visual field pre to the cortex below the sulcus. Now, the macula the fobal vision is represented more posteriorly and peripheral fields of view are represented more anteriorly. The blood supply to the ox to this primary visual cortex is provided by the mostly by the posterior cibal ar. Now let's look at the visual deficit that appear. And this may be a little bit complicated, but this is something that you will see a lot of questions for boards. Let's look at it. So Just a little bit of language, hemianopia means loss of half of a visual field. Quadrantanopia means loss of quarter of a visual field. This visual losses can be monymous, where the visual field losses are similar for both eyes. They could be congruous, identical or non congus, overlapping, but not exactly the same. Heteronymus means that the two eyes have non overlapping field losses. Here, you have the visual fields, the retinal fields, the accusation of the optic asm, and then you have the two tract, genic the two paths of the genicul calculate tract, you have the mayor loop,

or you have the other path that goes to the paralo. You have damage to the optic nerve, you have total loss of vision in one eye, in this case, for this example for the right eye. Now, if you have damage to the optic sin as we thought before. Now the bod nasal hemiretins, vision is going to be affected, and peripheral is going to be affected. But this is not symmetric because it's highly unlikely that the tumor or whatever is pathing against the sin is perfect. You're going to have no homonymous bitemporal hemianopia. It's loss of the peripheral. Now, when visual defects can be grossly divided if they are anterior from the optica or posterior. If they are anterior from the optica acid are going to be by, the deficit is going to affect each independently. Once things go posterior from the optic am, the f is not going to be per, but the deficits are going to be per field of. You have now on the optic track a deficit, you have contralateral homonymous heminopa. You lose the f of the optic track it affected, you lose vision coming from both eyes. Remember that there has been an accusation, but it's going to affect the full fill. Now, you have now damage in the temporal lobe in the mayor loop, you'll have the Pi in the sky disorder. You have superior homonymous quadrant anopia. The pal view, the upper part of the fill of view, which is coming from both eyes is going to be lost. Now, if the deficit is localized to the partal lope, you have inferior homonymous quadrantanopia, the Pi in the floor disorder. If the whole genic calcarine tract is affected, you have contralateral homonymous heminopa. You'll lose both the upper and the lower field of view. Now, if now when we go to the cortex, you have either the inferior bank of the calcarine fate is affected, then you'll have superior left homonymous cotinopa. I the superior bank of the calcarine fissure is affected, then the field of view that will be affected will be the inferior. Now inferior left inferior homonymous quadratinopia. If both banks of the calcarine sulcus are affected much larger than you have contralateral homonymous hemenopa. Now, you can see that in the visual cortex deficits, there's macular sparing, which means that the vision in the In the fobial vision, the hyp resolution vision tends to be preserve. Let's talk about a little bit about that macular sp. Macular sparin. Remember that the entire visual cortex is supplied by the calcanine artery, which is a branch of the posterior cellular artery. The more caudal parts of the vital cortex tend to receive also blood supply from collateral branches of the middle cellular artery. There are two route for blood supply for the fobial vision. One is coming from the posterior cerebral artery, but there is also from the middle cerebral artery. Also, remember that the fobia is very important. There is an expanded representation in cortex. The cortex is going to assign a very large area, many many cells are going to be representing that fi because that important. It is unlikely that the damage or stroke is going to just block the whole thing. That's why you have that macular spar. The macular sparing is clinically important because it permits to differentiate medial cereal arter infratation that affect the geniculcal cin tract, which there won't be no macular sparing from posterior ceral arter infraction, where there will be macular sparin. There is macular sparing, means that the cortex is enough. Another important feature about the visual system is that this condition called papdma. The optic nerve is part of the central nervous system embryologically. The menial coverings are similar and continuous to the other areas of the central nervous system. The sclera is continuation of the dural when it's lining inside by the acid and the pa. The Sabar noise space is continued with the sabra noise space around the optic nerve. If there is elevated intracranial pressure, that elevated intracranial pressure is going to be transmitted through the optical nerve. N is going to be visible in the pendscopy exam. If there is a compression of the optic nerve, that condition is called papildma, is going to be characterized by a swelling of the optic des. This is a non invasive tool that might indicate elevated intracranial pressure. Using a endoscopicic. We don't need to make a spinal tap or something We have been focusing mostly on the path from the eye to the cortex, but there are other additional optic cluck branches, the superior colliculus, where we have head orientation, eye movement, but this path is more important than lower animals. They are also accessory optic nucleus, that are important for retinal image stabilization. Also the hypotham of the Casmatic nucleus, it's related to our circadian rhythms. There is input, part of that carcaa rhythm is determined by the time of day, which we determine by the amount of light, of photic input regulates our circular. How is visual information process in the cortex? Visual information is actually separated into different features. This is done in a distributed manner. They are part of the brain that respond more to orientation. They are part of the cortex that respond to color or they respond to or to motion. Each different part is going to analyze its information simultaneously. And it is sought that this is a much faster way to analyze all parts separately and integrated into a final image and each column on cortex responds to a certain type of stimulus. Here we have vision picture of the brain. Here the ocyptal lobe, where we have our primary visual cortex, and there are two paths leaving the visual cortex. One is the dorsal part, which is also the we path, which is going to be it's going to be represented mostly location and motion. There is a ventral pathway. The what pathway, which is going to be related to form and color. Remember that there were these magnocellular and cellular differences at the level of the lateral genic nucleus, the magno were more about movement that separation and the part were more about

detail and color. The part we more related to the what pathway and the magno are more to the were pathway. There's a separation from the LGN about these two different parts of the visual perception. We can have different loss of visual capabilities. If we damage the posterior brain, that's going to affect both the optic radiation or the stride cortege, the visual cortex, will have full visual loss in some aspect of our visual field. But however, as we go if the damage occurs more advancing the w and pathway, the deficits are going to be more specific, which is one deficit that could appear is motion blindness. For example, we have a 40 for woman, suffer bi lateral infers of lateral parts of the parietal occipital on posterior temporal lobes. Visual feels, color vision, de persion, and rhythm were unaffected. However, she still cancels moving sounds and feel moved on skin, but she's not able to detect visual motion. Motion that's a dorsal stream So On the Power Point, you should be able to see a video. But people that have motion blinded is actually dangerous. For example, if they want to cross the street. They won't see the cars moving, but they'll see blurr image, not blur image, but they'll be jumping images. They'll be car in one position, and they'll try to cross, but the car is actually moving, but they don't perceive that movement, so it's dangerous. There's also color blindness on proapnosia. So this is example of a man that has abrupt onset of headache and confusion one evening. He doesn't lose consciousness, but subsequently remember nothing that occurred during the next 12 hours. He was taken home and help to bed and when he awoke the next morning, he became aware of several visual defits. There are some visual field deficits. However, he lost the ability to recognize color, which is called achromatopsia, and he also lost the ability to recognize phases, prospnosa. This is not about this is not the pathway. This is the what pathway. This would indicate lesions in the ventral stream. The posteropartal cortex plays an important role for localization, and there are multiple association area for multiple sciences is a processing center, damage to the left side. Is usually less frequent and transient compared to the right side damage. R hemisphere is dominant for spatial attention. After left hemisphere damage, the right hemisphere can direct attention to both the coralateral and ipsilateral side. But after right hemisphere damage, the left hemisphere can only manage to attend to the contralateral side. The left side of the word is neglected. That condition is called hemi neglect. Imagine that you're seeing a 50 year patient in your office. He's accompanied by his wife. He had a right sided stroke last year and is currently undergoing physical therapy to recover some of his motor control. For the last num, he has been able to feed himself. His wife interjected ding your HMP stating that for some reason, his plate of food looked like this when he's done. You can see that the person is only perceiving the right side of the word and he doesn't even perceive the left side of the word. These patients, you can ask them to copy like a clock and he's only going to draw the right side of the house of the clock of the flower. This is classically associated with right posterior palatal cordcleion. This is damage to the processing of the image is not the same a hemianopia, is that the subject actually is ignoring the existence of the left part of the word. Another interesting condition is blind sign where you have lesions in the primary visual cortex. The person becomes cortically blind. They cannot see. They cannot consciously see. However, when they are asked to guess aspect of visual stimulus, they are significantly better than. The person say no, I cannot see and then can you try to put this letter to this lot, but I cannot see. And then they'll do it right. They don't have a conscious perception, but there are other parts of the brain that are not cortical that are taking over and permitted to do some actions. However, we are not conscious of that. That will be all. Thank you very much for your attention.