Fundamentals of Videonystagmography: Lesson 1: Oculomotor tests: Saccades, Smooth pursuit and Optokinetic

Presenter: Vishal Pawar, MD

Welcome to today's course titled Fundamentals of Video. Nice lesson one. Before I turn it over to our presenter today, I would like to run through the learner outcomes. The learner outcomes for this course are as after this course, participants will be able to identify and describe the different functional classes of I've movements, including saccades, smooth pursuit and optokinetic movements, and their significance in oculomotorotor control. After this course, participants will be able to describe various eye movement recording techniques such as video nysteganography to capture and analyze eye movements, enhancing diagnostic precision for vestibular and neurological disorders.

After this course, participants will be able to explain VNG protocols effectively in clinical practice, facilitating the assessment, ongoing monitoring and management of conditions affecting the vestibular system and eye movement regulation. And now I'm going to turn this over to our presenters. Greetings everyone. We extend a warm welcome to each one of you and express our gratitude for joining us today. Inventis is a privilege to introduce the first of three lectures of the course, fundamentals of video nysteganography, theory, practice and clinical cases, featuring an internationally respected expert, Doctor Vishal Pawar from dubious.

Doctor Pawar is a nicely skilled specialist neurologist with over a decade of experience in neurology and autoneurology. Doctor Pavlov's expertise lays in vestibular medicine and headache disorder, and he has actively contributed to patient care, academics and research in these areas, earning recognition as a fellow of the European Board of Neurology and establishing a dedicated vertigo clinic. Thank you very much, Doctor Pavlova, for accepting our invitation. Well, this comprehensive course is structured to empower participants with essential knowledge and practical skills. It spans three online sessions followed by one hands on session in dubious on June 8, attendees will gain invaluable insight into the intrigue cases of VNG procedures.

Today, first session will be discussing oculomotorotor test. So stockade, smooth pursuit, optokinetic and so forth. As usual, I strongly encourage you to jot down any questions in the designated space on your screen. Following the event, you will receive a survey and we kindly request your participation. Your feedback will enable us to tailor your educational support to meet your needs effectively.

And now I leave the word to Doctor Pawar. Enjoy the webinar and see you later.

So thank you so much, Anna, for that kind introduction. So, I'm Doctor Vishal Pawar and today I'm going to talk on fundamentals of video stigmography, theory, practice and clinical cases. Today, like, I will cover the saccades, smooth pursuit and optokinetic so the outline of my talk is, basically, I will start with why we move eyes and how do we move eyes. Then I will go to functional classes of eye movements. Then I'll go to various eye movement recording techniques, which are the recording techniques available.

Then what are the different VNG protocols like in VNG, what are the tests which are possible? And then I'll touch upon saccade, smooth pursuit, and optokinetic. So, to begin with, why do eyes move? Like, why we move eyes? So, basically, if we look at our vision, like vision and retina, there is one area of the retina where, you know, we have maximum concentration of cones, which is present in area which is called as macula.

So I would like to request the audience to outstretch both your hands, okay. And with the thumb pointing upwards. And don't interlock the fingers. And keep looking at the left thumb. And start moving right thumb away from the left thumb.

And keep looking at the left thumb. Okay. So you will notice, like, can you put in the chat box what happened? Like, when you are focusing on the left thumb and then you are moving the right thumb away, then you might have noticed that your clarity of vision of the left right thumb keeps on reducing. As you move away from your macular vision, the clarity of the vision reduces when you are moving the thumb.

So that happens because we have something called as macula or fovea, where the maximum concentration of cones is present. That's why our visual acuity is maximum in the center. And as we move away from the macular vision, then we are moving towards the peripheral vision, and the visual acuity drops. Like, as we move away from the foveal vision, in the parafocal region, it drops further, and in peripheral vision, it drops further. So when the object of regard is here, it will, we will see it clearly.

But when the object of regard is suppose moves here, or another object of regard comes. So to bring this foveal vision from here to here, we have to move our eyes. So ultimately. So why we move eyes is to bring the clarity of that object of regard. We have to move the eyes towards the foveal vision.

Like, if the object of regard should be focused on the macula to have the clarity. So that is the reason why we move the eyes. Okay? That. So that is the one purpose of eye movement.

So if we look at the, like, extraocular muscles, so these, they are subserving this function. So basically, the eyes serve the function of vision, but the extraocular muscles are at the service of vision for doing this, extraocular eye movements. So that's how we have a clarity of vision. Now, if we look at the peripheral vision, like the peripheral retina, whenever an object comes into the peripheral vision, then peripheral retina acts like a guard. And when an object of regard falls upon it, it shout, who?

Who? It shouts who goes there and calls the fovea to the spot. So with the eye movement, it will help to, like, you know, focus the fovea on the object of regard. Now, let us see how we move eyes, like we have seen why we move eyes. Then let us touch upon how do we move eyes.

So, as I told, extraocular muscles are at service of the vision. Then there we need something which will move the extraocular muscles, that is, nerve supply. So the ocular motor nerves are at service of extraocular muscles. Then we need something which controls the ocular motor nerve, that is, ocular motor nucleus controls that impulses which are going through the ocular motor nerves. And the ocular motor nerves are under control of internuclear and the supranuclear connections, like supranuclear pathways and the internuclear connections, they are controlling these eye movements.

And whenever we are moving the head, we need to do adjustment in the eye movements. So that is done by the vestibulo ocular applique. So the vestibular sensations go to the extraocular, like, nuclei of the ocular motor nuclei, and they give them input. That's the head is moving this way. So you move your, move the eyes this way, so that that is the adjustment.

What is done in the eye movement based on the vestibular sensation, that is angular or linear head movements.

So if we look at the ocular motor control, we can divide it into four parts, that is, extraocular muscles, infra nuclear control, inter nuclear control, and supranuclear control. So let us go one by one into each one of them. Now, looking at extraocular muscles. So there are like four recti and two oblique muscle and one levator polyprion superioris. These are the extracurricular muscles.

So you can see there is one inferior rectus. There is a lateral rectus, there is a medial rectus, which cannot be seen from here. You can see after cutting the cutting open, the lateral rectus. Then there is a superior rectus. Then there is inferior oblique, which is, as the name indicates, it's obliquely present in the inferior part of the eyeball.

And there is superior oblique, which is present like this, like it is superior oblique. Suppose my head is right eyeball. The superior oblique is like this and this is superior, uh, levator palpebrae superioris, which is, uh, for the elevation of the eyelid. So, uh, there are four recti, two oblique muscle and one muscle which is acting in on the lead. And like I would like to clarify a few things before we start.

Like the muscle action, there is lot of confusion about the muscle actions, extraocular muscle action. So there are variety of actions which the extraocular muscles have. One is primary action, that is robust action seen in the center gaze. So for example, superior rectus has action of elevation as well as intorsion. So that is primary action.

Then the secondary action is action seen in abduction. And for example, the superior rectus. In abduction it is elevated and in adduction it is interted. So that is secondary action. And the tertiary action is action seen in oblique gazes, the oblique gazes, the, the same muscle can have some other action and coming to the field of action.

So the field of action is nothing but the major action seen when the eyeball is aligned to the muscle axis. For example, the superior rectus, the major, the field of action is elevation. And the field of action for the superior oblique is, uh, like intorsion. When the field of action for the, uh, superior oblique is intorsion. So these are the, this is the field of action.

Now let us, I have some, I have done some created some animations to explain this concept of, you know, extraocular eye movement. It is very important to understand it. So what I will do, I will show you one by one.

So when the eyes move towards the right, that is right gaze, what happens is the right eye, there is abduction, which is done by right lateral rectus. And the left eye, there is adduction, which is done by the left medial rectus. Okay? Now, in the abducted eye, if there is elevation, like when we do elevation in the abducted eye, that is done by the superior rectus on the right side and on the left side, it is done by the left inferior oblique. Then the depression in the right gaze is done by inferior rectus on the right side.

And left superior oblique does the depression in the like. Adducted eye. Adducted eye depression is done by the superior oblique. Now, these are the actions of these muscles. Like, okay, now suppose person looks towards the left side, the left gaze, the left lateral rectus and the right medial rectus are the yoke pair of muscles which help to do that.

So in an adapted position, adapted position. Suppose you look at the right eye right eyeball, okay? And in the adducted position, the intorsion is done by the right superior rectus. And in the abductor position, okay. The extortion is done by the left inferior oblique.

The same muscles are acting differently in different gazes. You have seen the superior rectus was elevator in abduction. But the same muscle is interferer in adduction. And inferior oblique, which was, uh, like elevator in adduction. It is extortor in the abduct abducted position.

So these are the, like, you know, um, primary action, second reaction. And, you know, this is what is happening with the muscle. That's why it becomes little complicated. Because it is not about only the primary action. Each muscle has a secondary action and field of action.

Also that there is primary action of that. Like, you know, major action of that muscle. Now, coming to the superior rectus. It causes elevation in abduction. And it causes intorsion in the adducted position.

Right inferior rectus causes depression in the abduction. And extortion in adduction.

And left superior oblique causes depression in the adducted position. And intorsion in the abducted position. And inferior oblique causes elevation in adducted position. And extortion in the abducted position. And there is one way to remember this.

All inferiors. If you look at inferior oblique or inferiors rectus, they are extorter. Inferiors are extorter that you can remember. Medial rectus, lateral rectus, it is very easy to remember. Medial rectus causes adduction.

And lateral rectus causes abduction. And the superior rectus is easy to remember. It causes elevation. And inferior rectus. It's easy to remember.

It causes depression. And superior oblique is an intortor. All superior are interpreters. So superior oblique and superior rectus are the interpreters. Only thing is, superior oblique is a depressor in the adducted positions.

And in adducted position, inferior oblique is an elevator that we need to remember.

So this is in the center gaze. As I was telling the superior rectus has two actions like elevation and intorsion. Superior oblique has two actions, that is depression and intorsion. Inferior rectus has two actions, that is depression and extortion. And inferior oblique has two action, that is elevation and extortion.

So if we look at the yacht pair of muscles like left superior oblique and the right inferior rectus. They have similar action but in different eyes. For example, the superior oblique is a depressor and into. And similarly, the inferior rectus is depressor and extortor. Because extortion is nothing but a conjugate.

Like, you know, it is nothing but like similar action which is seen in the other eye means, for example, if a person wants to do like action in the muscles, for example, here in this right eye, it will be extortion. At the same time, there will be intorsion in the left eye. So that is yoked pair. They are yacht with each other. And if we look at the vestibulo ocular reflex, the posterior canal on the left side is connected with the superior oblique of the same side and inferior rectus of the other side, that is vestibulocular reflex.

And we have discussed about the right gaze, what are the depressors and like, what are the, uh, extorter? Extortion and intortion. So these are the actions of the muscle. The medial rectus causes adduction, lateral rectus abduction. Superior rectus causes elevation.

Inferior rectus causes depression. Uh, superior oblique causes intorsion. Inferior oblique causes extortion. Now, coming to the like, the infra nuclear control, the third, fourth and 6th cranial nerves are important. They supply these extraocular muscles, and they arise from brainstem nuclei.

And let us see how they arise and how they supply. So this is midbrain, okay? And from midbrain, the superior part of the midbrain, the third, new third nerve cranial nerve arises, and it supplies all the extraocular muscles except the superior oblique, which is supplied by fourth nerve, which arises from inferior part of the midbrain. And the 6th nerve, which supplies the lateral rectus, which arises from the inferior, like lower part of the pons. Okay?

Now, this is overall the ocular motor control. That is, ocular oculomotorotor nerve arises from the upper part of the midbrain and supply all the extraocular muscle except the superior oblique, which is supplied by the trochlear nerve, or the fourth nerve, which arises from the lower part of the midbrain. And the lateral rectus, which is supplied by the 6th nerve. That is abducen's nerve, which arises from the lower part of the pons. So, innervation, if we look at is all extraocular muscles are supplied by the third nerve except superior oblique, which is supplied by this fourth nerve, lateral rectus, which is supplied by the 6th nerve.

And here one distinction has to be made is oculomotorotor. And ocular motor is not same. Oculomotor refers to the third cranial nerve, and ocular motor refers to third, fourth and 6th cranial nerve. So whenever you call extraocular movements, that is nothing but ocular motor movements or ocular motor control and not oculomotorotor. Oculomotor denotes to only third cranial nerve and there are two other cranial nerves also.

So we should use oculomotor, ocular motor instead of oculomotorotor.

Then coming to the internuclear control. Now, internuclear control means how these three nuclear control, okay, how this third nerve, third nerve nucleus, fourth nerve nucleus and the six nerve nucleus are controlled now. So they are controlled by variety of pathways. For example, if we look at the control of third nerve and the 6th nerve

nucleus, and here fourth nerve nucleus is not shown, but there are variety of pathways or variety of inter nuclear connections which control this nuclei. That is third nerve nucleus, fourth nerve nucleus and six ner nuclear, the major being, major pathway being the medial longitudinal fasciculus.

But the others are omnipause neurons, excitatory burst neurons, inhibitory burst neurons and neural integrator. This concept I'm going to explain in a while. When I'm going to discuss about the saccades, I'm going to talk more about the internuclear control. And the supranuclear control is nothing but the cortical centers, cortical and subcortical centers which control the this ocular motor nuclei, and these nuclei are under control, voluntary control of these pathways. And that's how the supranuclear control is achieved.

So there are so many things, so many pathways are important. So we began from vision, then extracurricular muscles, then cranial nerves, cranial nerve nuclei, internuclear connections, and then the supranuclear pathways, supranuclear control of the eye movements. So this is how the ocular motor control is organized.

Now coming to the functional classes of eye movements. Like if we look at the functional classes of eye movement, I am going to show this slide again at the end, and I'm going to ask all of you to write in the chat box about, like, you know, what are the, what is the each eye moment? What, which functional class of eye movement is needed for each of these purposes? For example, let us see the first one. First one is hold images of the visual world steady on retina during brief head rotations or translations.

Now, like for example, suppose I am doing head movements and I want to keep looking the presentation like. And even if I'm doing my head movements, my eyes are steady on the screen. So that is achieved by which moment? So that, so that is

achieved by vestibulo ocular reflex despite of angular or linear head movement. The eyes, we can hold the images of the visual field steady on the retina during brief hold, head rotations and translations.

That is, that is vestibular ocular reflex. Then the second purpose of eye movement is hold the image of a stationary object on the fovea by minimizing ocular drip. Now, suppose I'm not moving my head. My head is fixed, and I want to focus on the presentation. I want to minimize my eye movements.

So the fixation mechanism is needed for this purpose. So the fixation neurons and fixation gaze fixation is needed. Now, let us see the third purpose. The third purpose is hold images of the visual world steady on the retina during sustained head rotation or translation. It is sustained.

So the difference between the first, that is vestibulocular reflex, it was a brief moment. So if we look at here, it was a brief head movement, and the vestibule ocular reflex gets fatigued in 30, 30 seconds, 30 to 40 seconds. So here the it, we need a sustained, whenever there is a sustained head rotation or translation, for example, I'm sitting in a car as a passenger and I'm looking out, or I'm sitting in a train and looking out. So what is happening? I'm translating and translating in a continuously, in a sustained fashion.

And then the visual field is continuously changing, and I have to fixate my gaze. So I will follow the visual field with a slow movement. And then again, my, there is a fast correction. So that's how, so slowly, I will try to see the, like, you know, the environment, and try to scan, look at one particular thing. And again, there is, there will be a saccharidek moment.

So this is done by the optokinetic reflex. Now, the next purpose is hold image of a small moving target on the fovea, or hold the image of a small target on the retina that

is close to the head during linear motion with optimal aids, gaze stabilization during sustained head rotation. So the first example I will take in this, for example, there is one bird, okay? And the bird is flying. And if you want to focus on the bird, and the bird is moving, okay, moving in a certain speed.

And if I have to fix it on the bird, like povia, then my eyes will still so slowly start tracking that particular, like, object or bird, and that will be done by smooth pursuit. So this move, the movement which is needed to carry out this particular purpose is smooth pursuit. Now, the next purpose is bring images of objects of interest onto the fovea. For example, I'm looking here, and suddenly there is something happening in the peripheral visual field. And then I move my eyes from this direction to there suddenly.

So that is. That is that that purpose is served by saccades. So bring images of object of interest onto the fovea. That is done by the saccades. Now, the last purpose is move the eyes in opposite direction so that the images of single object are placed and held simultaneously on the fovea of each.

On the fovea of each eye. Okay, so, for example, if something is moving towards my, you know, nose and I have to converge my eyes. So the I have to move the eyes in opposite direction so that the images of a single object are placed or held simultaneously on the fovea of each eye. The same thing can happen when that there is a divergence of eyes is less apparent to the eyes. But for the distant object, if an object is moving away, there is little divergence of the eyes.

Convergence is more predominant, like prominently. We can see it. Okay, so these were the purposes. And these are the functional classes of eye movement. So one by one, I will show you animations which I have done.

The first one is sake. Okay. So here you can see one object of regard. Okay. And then when it moves towards the.

Towards your right side of the screen, then your eyes are moving from left to right. Then again your eyes are moving to the left. Again, your eyes are moving to right, left, right, left. So this is nothing but the sake. This is a saccadic moment, which is helpful to bring the object of regard on the fovea.

Okay, now let us look at the second movement.

That is smooth pursuit. Now, suppose here is the object of regard. And it starts moving slowly towards the right, then to the left, then to the right, then to the left. So when it is moving from the left to right, your eyes are tracking from left to right. When it is moving from right to left, your eyes are slowly tracking it from right to left.

So there is a sinusoidal movement which is created. It is nothing but the smooth pursuit. So the purpose of this movement is hold the image of a small object. Okay. Or target on the fovea.

Small moving target. It is moving. Okay, now let us look at the vestibulo ocular reflex. What is happening during the vestibulo ocular reflex? Now, suppose my head is looking straight ahead at some object.

And then my. I move my head towards the right side like this. Okay. Now, I still want to fix it here. But my head, initially it was looking here.

My head was also looking in the direction of the gaze. But I moved my head suddenly, but still I want to fixate on the same target. I want to look at the screen. Then vestibulocular reflex, what it does is when there is a head movement, it simultaneously,

there is an equal and opposite movement of the eye. For example, when my head moves to the right, the eyes are moved to the left, equal and opposite.

But that, see, that is what is vestibulocular reflex. But let us look at another situation. Suppose I choose not to fixate on the screen, but I choose to move the eyes along with the along with the head. That is nothing but vor separation. Like vestibulocular reflex separation.

For example, I am looking straight towards the screen, okay. Screen. And I move my head and the eyes towards the right side. Earlier I was keeping my eyes here and I moved my head here, okay. But now I will move my head and eyes together, both of them together towards the right side.

That needs separation of this vestibulo ocular reflex. So that is called as vestibulo ocular reflex separation pathway. Whenever the vestibule ocular reflex is not able to, we are not able to suppress. Because of the lesion, we are not able to suppress. That will lead to saccadic smooth pursuit.

Because during the pursuit movement, we have to inhibit the vestibule ocular reflex. If we are during the smooth pursuit, if we are not able to suppress the vestibule ocular reflex, it will lead to psychedelic smooth pursuit. Now, coming to the optokinetic reflex, during optokinetic, we have seen whenever there is a linear, sustained environmental motion, either from left to right or right to left or from down to up or up to down, there is a kind of nystagmus which is seen in the eyes and that is called as optokinetic nystagmus, which is a physiological nystagmus. It is not a pathological nystagmus, it is nothing. Is trying to give the clarity of like, you know, vision, despite of the ongoing optic flow, the moment of the visual field, despite of the moment of the visual field, it is trying to give the clarity by, by doing a smooth pursuit and then cycle.

Smooth pursuit and smooth pursuit and cycle. So optokinetic nystagmus is nothing but a combination of smooth pursuit and circuit. Now, coming to the vergence versions means like when an object is moving towards the eyes. Now keep you, if you are looking at object. Now try looking at the eyes.

What is happening to the eyes? Okay, now see what is happening to the eyes. There is a convergence. The eyes are converging. So the eyes are moving like, because of the medial rectus, there is a convergent center, which is located in the midbrain.

And that helps to converge the eyes. When the object is moving towards the like nose or towards the eyes, then there is a convergence as opposed to the divergence. In case of divergence, what happens when the object is moving away from. From the eyes? Look at the eyes.

What is happening? There is a mild divergence. Okay, so if we. If you look at the eyes, there is mild divergence of the eyes. So both lateral recti are bringing out this divergence.

Okay, now that was about the virgin. Now look at this central, this spherical, the one circle which I have drawn in the center. If which. If you keep fixating at it, which mechanism are you using? You are using fixation, like visual fixation, which is an active process.

If you are not fixating, like, you know, if you don't decide to fix it, then eyes can produce unwanted circuits, micro circuits, which is like, you know, happening passively. But if you want to fixate, you have to actively suppress all these unwanted circuits. And that is done by the fixation neurons, which we are going to discuss. So, visual fixation is an active process. It is not something which is passively we are doing.

We have to actively focus on something and fixate. And neural integrator plays a role in the fixation. So coming to the functional classes of eye movement, the sachets are important for bringing the images of object of interest onto the fovea. Smooth pursuit is important to hold the images of small moving target on the fovea. Vor is needed when the visual world to keep it may keep it steady during the brief head rotation.

Brief is very important word here. And optokinetic is nothing. But during sustained head rotation and translation, to keep the images of the visual world steady on retina. So retina and this is also important. And fovea is nothing but smooth pursuit and saccades.

As well as in case of visual fixation, we are keeping the object on the fovea. Even in case of versions, we are keeping it on the fovea. Okay, so in vergence, the eyes are moving in opposite direction. And visual fixation, we are maintaining a steady fixation. So if we look at the these eye movements, which are gaze shifting and gaze stabilizing.

So gaze is shifting. We are shifting our gaze for sacred. We are shifting our gaze for smooth pursuit and invergence. But in gaze stabilization. Suppose I want to see here.

But despite of the head movement, that is gaze stabilization, optogenetic. Despite of the visual motion like optic flow or translation. The. We are trying to stabilize the gaze in the visual fixation also then coming to the sensory input needed for the vestibulo. Ocular reflex is vestibular.

And all other other eye movements are visually guided. For example, saccade is by something in the periphery. Smooth pursuit is because we are seeing some object and then we are following it visually. Smooth pursuit is not possible without a visual target. Saccharides are possible.

For example, there are auditory guided sachets, also anti sachets, also without a visual stimulus like the auditory circuits. So that can happen with in case of circuit, but not in case of smooth pursuit. Optokinetic visual stimulus is must. Vergence is also it is mass. And visual fixation is on a target.

So it cannot be achieved with eyes closed or some other stimulus. Now, if we look at all the extraocular like these movements, they are all conjugate except versions, which is disconjugate. Okay? And if we look at all the like just now we discussed that this point also all of them are foveal except the vestibulo ocular reflex. And optokinetic reflex, which causes retinal.

Like this is nothing but a retinal. We are maintaining the like, you know, the object of regard on the retina. In case of optokinetic and vestibular ocular reflex. Now we can inhibit most of them, like all of them can be inhibited. We can inhibit saccharide, smooth pursuit, vestibule ocular reflex separation, virgin separation and visual fixation.

But optokinetic reflex is a very difficult to suppress. Of course, with practice it can be achieved. But there is no naturally, you know, natural pathway for optogenetic reflex separation. And if we look at the voluntary versus involuntary, we can have produce sakes voluntarily and involuntarily. Also smooth pursuit, also voluntarily and largely involuntarily.

For example, in optokinetic, then the vestibulo ocular reflex is name itself is reflex. So optokinetic and vestibular ocular is a reflex. Vergence is large, you know, voluntary. But it can be sometimes reflexive. And visual fixation is clearly a voluntary phenomenon.

So all these reflexes which are visually mediated or vestibular mediated, they go to the brain. And what is the purpose of all this eye movement is to maintain clarity of vision.

Why we are doing all this is to maintain a clarity of vision. When we are head is fixed, object is fixed, we want clarity. When head is moving, object is fixed, we want clarity.

Bivestibular replacement when head is translating or the object is regard or the is translating in a continuous, sustained manner. For example, in optokinetic, we have mechanism to have clarity. So when the object of regard is coming or moving away, then there is, there is a vergence mechanism. So all these mechanisms are designed to have a clarity of vision.

So why are we studying eye movement like, why are we so much interested in eye movement like this? If we look at the pathways which control the eye movement, they encompass the whole brain. Like, you know, cortex, subcortical areas, brain stem, cerebellum, all of them are involved. So the extraocular movement, eye movements are the pathways are located diffusely all over the brain, cerebrum and brainstem and cerebellum. So any lesion in any of these areas will manifest with eye movement.

So that is the purpose. Why we are studying eye movement is only because we are trying to localize where is the lesion? Is it in the brain stem? Is it in midbrain, pons, medulla? Is it in the cerebellum?

Is it in the cerebral cortex? Is it in the occipital lobe, frontal lobe? So what we are trying to achieve by studying the eye movement is just, we are trying to see where is the localization by mapping and deciding what is the, which eye moment abnormality we are seeing and which area is needed for that particular function and which is not functioning. So that is how it is done. For example, like a person has a gaze evoke nystagmus, the patient looks to the right, there is a right beating nystagmus.

Patient looks to the left, there is left beating stagnant. So this gaze evoke nystagmus is caused by leaky neural integrator, because the gaze stabilization mechanism is done

by the neural integrator. And so neural integrators for the horizontal gaze is located in the lower pons and the upper medulla. So we can localize the lesion in these areas. And they're the controlling.

There are certain areas which control these areas. That is, cerebellum controls this area. So that's how we can localize the lesion. By looking at the eye movement and abnormality, we can, it can help in the localization.

Now we are going to see which are the eye movement recording techniques. So there are three techniques which were, which are available. Like, first one, older one is electron steganography, scleral search coil and video nysteganography. So the electron steganography, what it does is there is a cornea, retinal, potential. The cornea has a positive potential and the retina has a negative potential.

So the electrodes are placed in such a way that whenever we are moving the eyes, if the positive, uh, like the cornea moves towards the electrode, it will be positive deflection. And the cornea moves away from the electrode, it will cause negative reflection. And so that's how it was designed. The electron stalling graphene and where we have to place the electrodes and we can record the eye movements. Then scleral search coil, where the coil is placed as a, like a contact lens on the eyes and which can be detected by a magnetic field, which is created by the strong magnets and the person sits into the magnetic field.

And whenever there is any eye moment, that eye movement is picked up by the magnetic field. So this is how the scleral search coil, a portable version of the scleral search coil was also designed recently. But it is still not that, you know, user friendly. Now coming to looking at the video and steganography, video style graph, your video oculography. It is nothing but a video recording along with the eye movement graphs.

And it is practical and easy method in the clinical setup. Okay. And I will show you the differences between all of them. So the electron stigma graph is the electrical potential. VNG is nothing but tracks eye movement with a video camera.

And scleral search coil is it helps in like, you know, the magnetic field is needed for this. In terms of accuracy, the highest accuracy is clearance search coil and. But it cannot. It can be used only in research setting. The high accuracy can be achieved by videonysteganography.

Typography portability is like good in case of electronics tomography and the cost is moderate in Eng and vanguard data captured is all the three types of eye movements can be captured in the VNG. For example, torsional can be seen in the video and horizontal and vertical can be seen on the graph. Of course, there are some VNG systems which can record torsional also. And all eye movements can be recorded in scleral search coil. There are of course, limitations of every equipment.

The invasive. The scleral search coil is invasive and it requires specialized equipment and it is applicable in only research setting. In case of VNG, camera resolution is very important. The field of view requires good head stabilization and the application is we can use it for most of the ocular motor movements we can use the VNG. Another commonly like, you know, like nickname for video oculography is video nysteganography.

Like video oculography. Is more appropriate word because the, this equipment helps in like taking care of all the type of eye movement, not only nystagmus, it helps to see the saccade, smooth pursuit, any other type of eye movement, not only the nystagmus, but you know, the VNG name is more popular and so it is synonymously used for the Vog.

If we look at the various, like, you know, what are the various options available in VNG? There is a video frenzel and it can also be used to do positional testing, caloric testing and ocular motor examination. All the ocular motor examination that is circuit smooth pursuit, optokinetic testing, gaze testing with fixation without fixation, head shaking test, rotary chair test. All these four test protocols can be done. So what are the various VNG test protocols that is saccade, smooth pursuit, optokinetic testing center, gaze testing in light, gaze testing in light, all the testings without fixation, head shaking test, positional testing, hyperventilation.

Nystagmus can be done in the like, you know, video frenzel mode and vor separation testing, caloric testing and rotary chair testing. So it's a scalable system where, you know, you can add the protocols based on the other things available today. In the today's talk, we are going to cover saccharidede, smooth pursuit and optokinetic as far as possible. And you know, like with. There are two more talks where I can cover further things.

So psychotic system, if we look at the purpose of saccharidedes, we have discussed in depth, that is to place the object of interest to place on the phobia object of interest which often have been first registered by the peripheral retina. That is the purpose. And the psychedelic eye movement are used to inspect a complex scene. So when we look at one object, we feel that we are looking at a whole scene. But our macula, the foveal vision is constantly moving and that is causing the saccades.

But the rest of the features are seen in the peripheral vision. Even though when we are looking at something we, our macula is focused at some point and it keeps shifting its focus with the help of sacades. So there are variety of circuits which are created while looking at a complex scene. Now, what are the types of sakes? It can be visually guided.

It can be memory guided. For example, when you come at home, you know, where are the things located. So suppose somebody tells you something, you immediately look at that particular area. Suppose somebody tells you to bring something, like you will. You can immediately shift your attention to that area because of the memory of that particular object.

Then, for example, now you are currently looking at the screen. And if you buy any reason, you look away from the screen and your prediction, like something is going to come on the screen. So you, the predicted circuit will happen onto the screen. Then antisaccade means like for example, if you are focusing on something and suppose something comes in the peripheral vision and you move the focus away from that, or you don't move your eyes towards that particular novel object of regard. That is, that is antipsychotic task.

And many a times, you know, when we are, as I told, fixation is an active process. So when you are not fixating, there are a lot of spontaneous sacred which are called as microsaccade and reflexive. Sacred means like something coming in the periphery. And suddenly you reflex, you look at it or something, some sound comes suddenly and you look at that particular sound, that is reflexive or reflex. Auditory guided circuit.

Now, the simplistic pathway of, because this pathway, you know, there are voluntary centers for saccade, involuntary saccade centers. But I try to keep it very, very simple. And the pathway, it starts from frontal eye field for the voluntary saccharidede control, that is frontal eye field. And it goes to the PPRF, that is paramedian pontine reticular formation on the opposite side. And then from there, these are nothing but the premotor neurons for the 6th nerve.

And then the impulses go to the 6th nerve. And from 6th nerve there are internuclear connections with the, with the help of medial longitudinal fasciculus, with the

contralateral third nerve. And this third nerve supplies the medial rectus on this side. This supplies the lateral rectus on this side. And that leads to adduction of the this eye and abduction of this eye.

So frontal eye field area of this. Suppose this is left, is needed for right circuit. Okay, circuit towards right is done by the left frontal eye field area. So this is how it is like right frontal eye field is needed for the left saccadic moment. And left frontal eye field is needed for the right saccadic movement.

Both frontal eye pill and superior colliculus for this vertical sacking.

Now, coming to the role of attention now, you are here in the webinar from last 1 hour. You are attentive and you are trying to grasp how many things possible. So that is a door for cognition. So your attention is door for the cognitive function. And it is, you know, it is a key for the cognitive.

Cognitive functions are not available unless this door is open. If the door is closed, it's not available. So there are various doors of cognitive function. For example, first thing, when you are now attending the webinar, so somebody is feeling suppose sleepy. So you will not be able to, you know, comprehend the webinar.

If you are fully awake, then you will be able to comprehend it more. So the cognitive functions are open when you are awake, then the second door is attention, even if you are awake. But you, if you are not attentive, then you will not be able to grasp many things. So attention is important even if you are awake. Then the third thing is language function.

Suppose I start talking in some language which you don't understand, even if I might be giving a good information. But your language, the language, if it is you are not able

to comprehend it, you will not be able to understand whatever I say that that is the third door of cognitive function. And the fourth door is motivation. I would like to appreciate whole audience for their motivation to be here with me from last 1 hour. And that is fourth door of cognitive function.

So these are the four doors you can see, you can think it as a screen lock for your mobile app. Mobile. For example, before opening the mobile and going into various apps, which are nothing but cognitive functions, different functions of the mobile, you cannot use unless you unlock it. So similarly, our brain has these four locks. You can say sleep, wake, cycle, attention, language function and motivation.

And while doing VNG test, also these things are important. Patients should be awake, attentive, understand your commands and motivate it. Because what happens if the patient is sleepy inattentive, it will lead to a lot of abnormality in saccades and smooth pursuit. So inattention is a very common cause of saccadic and smooth pursuit abnormality. Now like what are the mechanical properties of saccades?

Like brain makes use of sensory map of visual environment. And what happens is this sensory map of the visual environment and coordinates of the orbit. And it decides where it's like, you know, where I need to look and what is the desired position, where I need to look and what is the actual current position and desired position. And then may it makes a circuit accordingly, it calculates the pulse and step. I'm going to explain what is, what is pulse and what is step.

Now, if we look at these graphs, okay. When the. During the VNT testing, this green one is a stimulus, okay? So the stimulus is moving from left to right, okay. And then the eyes move from right eye moves from left to right.

So right eye, there is a movement from the left to right. And the left eye moves from left to right. Okay? Similarly, when the stimulus moves from right to left, okay, then the eyes right eye moving from right to left and the left eye moves from right to left. Okay?

So that is how sacred happens in case of a while testing. And this is a step where, you know, suppose I move from my eye from left to right support and I stay there. That is step. And this is pulse. And I, I stay there.

That is a step. Then this is pulse. Then I stay there. That is a step. Then this is another pulse from left to right, right to left pulse step, left to right, pulse step.

So that's how the pulse step is organized. So the pulse to the right side and pulse to the left side, left gaze, okay? And the pulse is nothing but a phasic discharge. There is a sudden ballistic discharge. You can see there is ballistic discharge from left to right.

That is pulse. And this is step means it stays there. Your gaze is staying there for some time. That is step. And again there is a pulse from right to left.

Again there is a step. So that is how it is organized, okay? So what this pulse does is it overcomes the resistance of orbital tissues and inertia of the globe, okay? And you can you see this latency? So there was, there is a stimulus here, okay?

And. But the actual saccadic response started from here. So this is nothing but the latency. Despite the stimulus coming here, there was, it started here. So that is nothing but the latency.

Latency means the supranuclear pathway generating the cortex, generating a pulse command. It is coming to the PPRF and then going towards the reverse neurons. And

then it is. It causes firing of the ocular motor nucleus. The time taken for this pathway is nothing but the latency.

So the latency is prolonged in case of, usually in case of cortical. Cortical lesions, which are help, which are important for generation of the sake. Now, the step step is nothing, but, you know, it stays there. The eye stays there. The staying is very important.

You know, we keep the eyes suppose on the right gaze, it is not something which is happening without any energy, but it is happening at the expense of, you know, they. The eyes have to overcome the elasticity of orbital tissue and you have to keep in the new position. It is also active process. And the transition between the end of the pulse and the beginning of the step is not always abrupt. It is gradual.

So always there will be one step, pulse, slide, step, which be there like, you know, this. This thing will be there like there will be a pulse. Then slide and step. Okay. Pulse, slide, step.

So one step is, uh, like needed to, like, you know, accurately achieve that particular, uh, area. Like, particular, like, you know, object of regard moving to that particular site. So in case of horizontal circuits, like, okay, suppose our gaze, you are looking at the screen right now. You are fixating. So the omnipause neurons are very important for fixation.

They have to suppress all the burst neurons and try to keep your eyes fixed on the screen. Now suppose, like, and they are, these omnipotents neurons is nothing but nucleus Raphael interpositus. And they avoid unwanted states and they cause inhibition of the excitatory burst neurons and inhibitory burst neurons. And then there is something called latency. Like even if the stimulus has come, there is a latency.

That latency happens because, like, you know, there is firing from the frontal eye field coming to the PPRF and then excited reverse neurons. And then there is the pulse, this saccadic pulse, which is, which happens in the case of horizontal saccade because of the ipsilateral 6th nerve and contralateral third nerve, medial rectus muscle. Okay? And at the same time, these excitatory burst neurons, when they are exciting ipsilateral 6th, contralateral medial rectus, at the same time it has to inhibit the like, you know, the contrasting muscles, that is ipsilateral medial rectus and contralateral lateral rectus, if they are inhibited, then the movement will happen very smoothly. Okay.

So at the same time, excitatory burst neurons are stimulating one yawn pair and inhibitory burst neurons is inhibiting the antagonist muscles to the, to the burst neurons. To the excitatory burst neurons. So there is a pulse slide step, which is a normal phenomenon. Once slide step is normal, then there is a role of neural integrator to maintain the gaze on that particular target. It's very important that the neural integrator comes into picture and this velocity command is converted into a position command.

In that position, eyes have to stay there. That is staying is because of the neural integrator. So the neural integrator for the horizontal gaze is located in the medial vestibular nucleus and the nucleus prepositus hypoglossi, excitatory burst. Again, when the saccade happens from, from right gaze towards the left gaze, right toward the left, again similar thing happens. But another group of nucleus activates another group of excited reverse neurons and inhibitory burst neuron activate.

Again there is a pulse slide step. And then again omnipresent neuron come into picture in the center gaze. Like, you know, in the center gaze, it inhibits excitatory and inhibitory burst neurons. Similarly, in case of vertical gaze, there are variety of centers which I am going to show in the next table, in the next slide. So this is how it is like, you

know, gaze centers are located for horizontal in the caudal pons and vertical and torsional in the midbrain.

And these are the various gaze falsies which can be seen. A neural integrator function. For horizontal gaze, it is nPh, mVn, or in. It is interstitial nucleus of kohl. In case of vertical and torsional and gaze yoke nystagmus is seen because of the lesions in the neural integrator.

Excitatory burst neurons are located in for horizontal and vertical in different areas. And if there is any lesion in excitatory burst neurons, it can lead to slowing of the saccades. Inhibitory burst neurons are important because if they are not functioning well, then it can lead to saccadic intrusions and omnipotents neurons, which are present in nucleus raphae interpositors. It can, if they are not inhibited, inhibiting the unwanted saccades, then it can lead to, you know, ocular flutter or oculoclonus can happen because of the lesion in omnipotent neurons, which happens in paraneoplastic syndromes, autoimmune encephalitis. Okay, then coming to the like, the basic thing is cerebrum decides when and where to move.

When to move, for example, my brain will decide when to move. Eyes, for example. Currently you are focused on the presentation. Your brain is deciding not to make unwanted circuits. But if something comes in the peripheral visual field, still it will decide whether to move or not.

It will decide and where to move. Even if the. Suppose there are two stimuli, one in the right and left. So which side to move? It will decide.

So that is the function of cerebrum. But the brainstem function is how to move. Like reflex action is by the brainstem.

Now, coming to the how it is recorded, I will show you how the sachets are recorded. You can see the sake towards the left side. This is towards the right. This is towards the left. Okay, once again I will show you this sake to the left.

Right, left, right, left. Okay, so. And you can see in the graph the eyes are moving from left to right. Then there is a step. Step means the eyes stay there, then comes from right to left.

Then it stays there, then comes from here to here. So here, if you. You can see there is hypometric saccharidede. Why? Because if the attention is not perfect, it can lead to hypometria and it can lead to a series of hypometric saccade.

But that's the importance, you know, if you see another saccade which is done correctly, then that means that here the attention was not okay. So it's very important during psychiatric testing to tell the patient to be very attentive and look at continuously keep following whatever is coming in the, on the screen. So what are the parameters which we can see here in the psychedelic, you know, you can see here, this is a step, okay. And this is one uh, pulse of the sacred. And this is another pulse of the circuit.

Okay. And you can see there is a latency whenever the stimulus is starting. Okay. The green one is a stimulus, after that the saccade is starting. So there is a gap between these two is nothing but the saccharidek latency.

Okay. And this is for the uh, you know, this is for the horizontal circuit and this is for the vertical circuit. And the parameters which we check in case of circuits is latency, velocity and accuracy of the circuit. And which can be, which is given here, I will tell you the normative and here in a graphical format, beautifully. It shows the velocity and accuracy of the circuit.

The velocity plotted on the y axis and the accuracy on the x axis. And most of the circuits, if you can see they are falling in this uh, you know, normative, uh, data. So this is for the vertical, all these circuits are falling into the uh, this vertical circuits are normal. So these are the three parameters which I was talking about. The latency.

Normally the latency is less than 200 milliseconds. Uh, and velocity is ranges from 300 to 700 seconds. If you can see here, 703 hundred degrees per second. And the precision is 8200. You can see here, precision is around 8200.

Okay. So that is precision. And accuracy is same. So if you look at the pathways which are needed, supranational pathways are needed for the latency until it fires. Then the velocity, the accuracy, the precision is dependent on the cerebellum and the velocity is dependent on the brain stem.

So each of these parameter like is important for different functions.

Now let us see, variety of lesions, precision, latency. There can be problem in the velocity, trajectory and conjugacy. So as I was describing, the latency is decided by the particle and sub particle areas. So if whenever there are neurodegenerative disorder, there is a delayed initiation of the saccade and it leads to increase in the latency precision is dependent of the cerebellum. That is, if there is any cerebellar lesion, it leads to dysmetria, that is imprecision, and it is due to various cerebral pathologies, and it can lead to overshooting and undershooting.

And if there is a problem in the velocity that is excitatory and inverse neurons or the premotor neurons, there is a brainstem or ocular motor dysfunction. It can also happen due to muscle disorder or oculomotorotor motor palsy or brainstem lesions. And there is impaired signal transmission or muscle weakness that is the anatomical substrate for

this problem. Slower sachets are generally seen in elderly as compared to the, you know, the velocity goes down in elderly as compared to the younger people, drowsy. That.

That's why I was saying sleep wake cycle. People who are sleeping, the sachets are slower, those who are inattentive and medicated. And these are the variety of cortical lesions which can lead to, you know, the increase in the latency of the saccades.

Now, coming to the smooth pursuit. Yeah, I will take another five minutes, like, you know, to complete, and then we will go to the question answers. In case of smooth pursuit, the object of regard should fall on the fovea. The retinal sleep is the major stimulus for it. What is retinal sleep, I'm going to show detected by the visual system.

So it is a visually guided moment, eye movement. And in case of optokinetic nystagmus, it is a smooth pursuit. Combination of smooth pursuit and the saccade velocity of the smooth pursuit is around 30 to 40 degrees per second. Only more than 40 degree. It has to do a catch up circuit.

Okay, but up to 30 to 40 degrees per second, if the object is moving slowly up to this velocity, then it will lead to smooth pursuit. Faster moving object sake, even faster moving, you have to move the head also. So first, initially, if the object is moving slower, it is smooth pursuit. If it starts moving faster, you will do a psychedelic moment. And when it moves away, this.

This point of your circuit, you have to move your eyes head along with the eyes. So this is what is retinal slip. Suppose, uh, this is, uh, suppose a fan. Like, you know, it is, uh, it is like, you know, uh, rotating in a circular fashion. And as the speed increases, you can see the retire.

Because of the retinal slip, the clarity reduces. So whenever the object of regard is moving, okay, if it is a slow velocity, it is smooth pursuit. If it is higher than that, it is sacked. We have to catch up and then we have to, you know, go for the head moments also. But if it is a continuous, linearly accelerating or continuous movement, then it will combination of smooth pursuit and circuit.

That is a optokinetic nystagmus. So the control of smooth pursuit is nothing but from the frontal eye field, parietal eye field areas. And then it goes to the pontine nucleus. The command goes to the pontine nucleus. Fontaine nucleus communicates with the cerebellum and then decides the.

This output of the motor nuclei. Ocular motor nuclei. So let us see how this smooth pursuit is performed. This object is moving from right to left, left to right. And you can see the eye movements, okay?

The eyes are moving to the right, to the left.

And you can see the tracker that keeps tracking. The pupillary tracker helps in tracking the eye movement. This you can see the smooth pursuit eye movement. Okay?

Again, for the vertical smooth pursuit, the object is moving up and down and you can see the eye moments which are happening up and down. Okay?

So here can you see the eye moments which are happening up, that is smooth pursuit and down. And you can see the graph here, graph of like, smooth eye movements. So this is recorded in a patient where you can see the smooth pursuit, like the green one is the stimulus and you can see the red one, that there is a smooth eye movement, which is exactly tracking the. That's why the gain, you can see the 1.01 almost. It is equal to the moment.

And you can see nice graph gain. Graph here. That is rightward gain and left work gain. And so this is for the vertical. And this is for the vertical.

And this is for the horizontal. Okay. So the normal gain is 0.8 to 1.1. And usually the vertical gain is lesser than the horizontal gain. That is commonly what we see.

Okay. Because the vertical eye movements, we are not much trained into horizontal eye movements. We use more commonly than vertical eye movement. And these are the abnormalities in the smooth pursuit. Like smooth pursuit abnormalities.

During. While doing the smooth pursuit, there can be saccadic intrusions because of the various lesions. There can be reduced gain because of, again, because of the vestibular, cerebellar or other lesions. There can be asymmetry in the arms of the pursuit, for example, like right arm left to right or versus right to left arm. There can be sometimes, you know, one arm can be saccadic as compared to the other.

So that is very common in unilateral vestibular loss or unilateral cerebellar dysfunction. Or brainstem lesions. Then there are two types of saccades which are seen during the, like smooth pursuit, ketchup saccade and catch back. Saccade. Like catch up saccade is nothing.

But when the stimulus is moving, okay, suppose the eye lags behind it, catch catches up with the sake. Again, it lags behind, then it catch catches up with the sake. So this is a catch up type of, you know, saccadic, but it is catch up type. Then there is another one which is catch back type. Like, catch back type means it, the stimulus is moving like this, but the eyes move overshoots, then catch catches back.

Overshoots, catches back, overshoots, catches back. So catch up is more common. And this is seen in cerebellar brainstem and vestibular. And this is also seen in the disorder of cerebellum and brainstem. Then there is.

There can be some gauge u of nystagmus during the pursuit. Many times patients have pure torsional nystagmus, horizontal nystagmus, and that is reflected in the, uh, during the smooth pursuit testing. And there can be pursuit. Intermittency means interruption of smooth pursuit by phases of fixation or loss of tracking. Uh, because there can be sometimes inattention, attentional disorder, patient is drowsy, or neurodegenerative disorders can lead to this.

So impaired Vor separation, as I have told already, that Vor suppression, if it is impaired, then it leads to psychiatric smooth pursuit and cortical hem. Like, you know, lesions, like hemisphere otomy, can lead to impaired smooth pursuit. Then coming to the legions of supra, this smooth pursuit pathway can be supratentorial or infratentorial. We will not go into the details of this, but by and large, you have to remember that psychotic smooth pursuit can be seen. It has a diffuse localization.

It is not like localizing to one particular point. For example, you see unidirectional nystagmus following Alexander's law. It is peripheral variety of nystagmus. It has a precise localization. You see a beating torsional nystagmus in the right dix-hallpike.

It is right posterior canal be ppv. But when it comes to, you know, lesions, which the supranuclear movements, like smooth pursuit saccade, which has got diffuse control, the control areas are different. So many areas are controlling it. So that's why it has a diffuse localization and sustained attention is needed even while doing the smooth pursuit. That's why psychedelic smooth pursuit can be seen in migraine patients, sleeping sleepy patient patients who are medicated or may various disseminated disorders.

Now coming last to the optokinetic quickly, I will finish this. This you can see Professor Enrico here during one of the meeting in dubious and one of his slides I can show here. So when the object of interest or head head is moving faster, okay. While like, you know, when we are walking or active hair movements of the head or running, then our vestibular reflex. Vestibular ocular reflex come into picture.

And to maintain the stability of the retinal image, okay? But at lower frequencies, along with the vestibulo ocular reflex, optokinetic reflex is there, which supplements the vestibulo ocular reflex at little lower frequency and at very low frequencies, you know, the cervical ocular reflex comes into picture, which is helpful, you know, for stabilizing the vision. For example, you imagine a person gets vestibular lesion. Like, you know, the vestibular ocular reflex is abolished. Then these primitive reflexes come into picture.

The cervical reflex becomes more predominant. And that's why many people with vestibulo, vestibular lesions, they have neck pain, mainly because the activation of the cervico-ocular reflex, which tries to stabilize the region by stabilizing the neck. Okay, so as we, we have discussed, the Vor fatigues in after 30 seconds. And that's why we need another system. Different system is needed, is required to maintain the gaze stability with continuous head motion in the same direction.

And so optokinetic nystagmus is ocular stabilization system with a succession of moving stimulus, there is optic flow, which is generated and we create. We. This is a nystagmus, which is a physiological nystagmus, which is combination of smooth pursuit and saccade. Suppose something is moving fast from my left to right. Then I will slowly pursue it through a rightward smooth pursuit.

And there is a sachet towards the left side, smoothly, I will pursue. Then there is a sacred smooth pursuit. Sacred smooth pursuit. Sacred. So, uh, the, there will be a left beating, stagnant, okay.

When a stimulus is moving from left to right, there is a left beating, stagnant stimulus is moving to. From right to left, there is a right baiting. Similarly, uh, it happens, it is also called as relevant stagnant or a parade nystagmus. It helps in cracking of targets in succession.

You can see that this is optokinetic stimulus. And there is an sphagnous, which is seen in the eyes. And this is how it is seen in the eyes. You can see there is a left beating stagnus because the object is moving from the left to right. So there is a, you can see the nystagmus here.

The slow phase is towards the right side and the fast phase is towards the left side. Slow phase is towards the right. Fast phase is towards the left. Slow phase, fast phase. Nystagmus is named after this fast component.

That's why it is a left beating nystagmus. So here you can see it is a horizontal optokinetic stimulation. Vertical, there is nothing. And it is recording the right eye and the gain is 1414.5 degrees slow phase velocity. So the gain is calculated automatically, that is 0.7.

And there is very mild minimal vertical gain which is seen. And here you can see during the horizontal optogenetic stimulation, the horizontal nystagmus which is seen which is again left beating nystagmus, left right, slow phase, fast pace towards left. Right, slow phase, fast phase towards left. And during the vertical optokinetic stimulation you can see the gain is usually there lesser as compared to the right horizontally. And you can see the vertical nystagmus and horizontal is not seen.

There is no, not much moment. But you can see some square wave jerk which are, which are seen in this horizontal trace. So this is the table of, you know, this is again optokinetic nystagmus which is beating towards the left.

So what are the various clinical significance? Like clinical, clinically, how relevant is optokinetic? So whenever there is anterior virtual pathway defect, there is a reduced optotypic response in parietal occipital lesions. Also it can show reduced response in unilateral vestibulopathy. There is a directional preponderance.

Like for example if a person has right unilateral vestibular path, he will have left beating nystagmus. So whenever the paper send the stimulus in, the optokinetic moves from left to right, the left beating nystagmus will be stronger as compared to the right beating stagnant. When the stimulus moves from right to left. Then visual activity in infants can be assessed by doing optokinetic testing.

Psychological blindness. I had one patient, she was having dizziness and suddenly she started saying me that I can't see anything, doctor. Then I put her on Vng, I started optogenetic and she had optokinetic nystagmus. Optokinetic nystagmus is a visually driven nystagmus. It has to, vision has to be present for optokinetic nystagmus.

So psychological blindness in reduced level of conscious consciousness. It can be used reversed. Optokinetic nystagmus can be seen in congenital or infantile variety of nystagmus. Subtle iron o can be picked because of the like, you know, optokinetic nystagmus testing and convergence retraction. Nystagmus becomes prominent in perinatal syndrome in case in like by doing the optokinetic testing.

So this was again as I promised. I will show the that slide again. Now I will ask you to put into chat box like which eye movement is necessary for, you know, holding the

images of visual field visual world steady on the retina during brief head rotations or translation. Can anybody put into the chat like which, which moment eye movement is needed for this?

Okay, so I will tell this. This is vestibulocular reflex which is needed for this particular moment. Then hold the image of a stationary object on the fovea minimizing ocular drips. This eye movement is nothing but fixation. We have to.

We need fixation to hold the image on the stationary object. Then the whole images of the visual world steady on the retina. During sustained head rotation or translation. It is optimal. Then the hold the image of a small moving target on the fovea.

It is smooth pursuit and bring the images of the object of interest onto the fovea. That is done by the sakes move the eyes in the opposite direction so that the images of the single object are placed or held simultaneously on the fovea of each eye. This eye movement is nothing but vergence.

So this is my team. I would like to thank my audiologist Mister Muhammad Shaukat. He is instrumental in, you know like our vestibular function testing, VNGs and video head impulse test. And he has helped me to prepare this presentation by, you know, we. While doing the testing we have shot some videos and graphs.

She's my dedicated nurse, Reshma. And we have three vestibular therapies. Like they are helpful for the vestibular rehabilitation. And I would like to again remind all of you about the next session. That is on the 20 March.

I will be discussing further aspects of the video and stereography. And the third session will be on the 3 April and we will be having an in person hands on session on the 8

June in dubious. That is a full day session. And I will be demonstrating all the ocular motor testing how it is done. And with that I thank you all for patient listening.

And this is my email and phone number in case of any doubts. And I would like to invite all of you, you know, in the chat box, you can comment, like how if there are any questions which are related to the today's presentation. So thank you so much, everyone, to spare one and a half hours with me. I would like to like, if there are any questions, I will be happy to answer.

Yes. Thank you. Thank you very much for your presentation. A fantastic learning opportunity. If you check the question box, I can see in this moment eight questions from the audience.

Answer. Oh, okay. Okay. I was seeing, seeing another chat box. Okay, no problem.

Question. And yes. Yes. Oh, they have answered also. So Angela has answered VOR fixation virgin.

Thank you so much. Then AsMa is asking when to suspect pathological square. Square jerky eye movement. Yes, it's a very important question. Many a times, physiologically, you know, when the patient is anxious, we can see square wave jerks.

But how to differentiate this square wave jerks which are happening physiologically versus pathologically? There are two, three ways. How you can differentiate. One is by looking at other extraocular eye movement abnormalities. If everything is normal.

And if you can look at the patient, patient is anxious. So that can be a, you know, like, physiological like response. That is anxious patient. But if the square wave jerks

morphology is also very important. The pathological and physiological square wave jerks, the morphology is different.

And in my next talk, I'll be talking about what is the square wave morphology, a jerk morphology in physiological and pathological. This thing, I will give you some, you know, table which will help to differentiate. And the third is the frequency. The frequency is very high in case of pathological, as compared to, you know, in case of physiological, the frequency doesn't go to, like, you know, beyond certain frequency. That is how many, uh, square jerks in a minute.

I I hope I answered your question. Then I will move to another question. Can we get recording of this? Yes. Uh, Anna will help you in that.

You can follow the inventive website. They. It will. It will be available for that. Then?

Um, yes. Yeah. So then another, I spoke about the recording. Then can you explain Alexander's law? Where is it applicable, like peripheral or central pathology?

Yes, it's a very important question. For that, I would like to explain you. This is my right vestibular system suppose. And this is left. Okay?

So the right vestibular system and the left vestibular system, normally they have equal and opposite output. Okay. Output normally at rest. Also, they. They have resting discharge.

So the right vestibular system is trying to push my eyes, head and body towards the left side, okay. And my left vestibular system is trying to push my eyes, head and body towards the right side, okay. So normally the system is in balance, okay. Because of

the equal and opposite output, normally the system is okay, is in the center. My eyes are in the center because my.

Both the vestibular systems are firing equally and opposite. Suppose I have right vestibulopathy, what will happen? The left side, which is normal, it will start pushing my eyes towards the right side. That is the slow phase. But my brain, the left cortex frontal eye field area will detect it and it will make a saccharidene movement towards the left.

That is a left beating nystagmus. So in right vestibulopathy, there will be a left beating nystagmus. So in vestibulopathy, what happens, happen is whether I look towards the right side or in the center or towards the left side. Patient will have unidirectional nystagmus because the push from the right vestibular system is absent. That's why patient will have unidirectional nystagmus, which will be beating maximum in the left gaze as compared to the center gaze and as compared to the right gaze.

Okay, so this is the Alexander's law. What is Alexander's law? A patient with vestibular lesion will have grade three nystagmus, that is vestibulopathy, will have grade three nystagmus. Unidirectional nystagmus, which will be maximum in intensity when it beats towards the normal ear. When eyes are looking towards the normal ear, normal ear, then that side, it will be maximum in intensity, little lesser in the center and even lesser or absent when it is looking towards the affected ear.

Alexander's law is applicable for peripheral vestibular lesions. But. But there is a caveat. Okay? Structure.

See what happens? The, like the vestibular pathway, the peripheral vestibular pathway doesn't end at the brainstem, okay? Like brainstem, when you at the Cp angle, it goes through it, through the fascicles, okay? The root entry zone, and then it reaches the

vestibular nucleus. So still the peripheral sensations are unadulterated, unchanged until it reaches to the vestibular nucleus.

So there are certain central regions, strategic lesions in the central center, which can mimic, like peripheral lesions. Suppose a person has a brainstem encephalitis affecting the root entry zone of the vestibular nerve. It can still mimic, like a peripheral lesion, which might even follow the Alexander's law. Law. So don't decide the localization based on the like, you know whether it is following Alexander's law.

But if a patient has unidirectional nystagmus following the Alexander's law, largely it is peripheral. Look for any other central signs. If there are no central signs, then it is peripheral. Like for example, look for the head impulse test. Head impulse test is positive, pathological.

Then it is more in favor of peripheral. Again, that is also there. That is another caveat. We have to check for other central signs. If the other central signs are absent, it is peripheral.

If the other central signs are absent, it is peripheral. So one dictum you remember, central signs are always central and peripheral signs can be central as well as peripheral. Until proved otherwise, even peripheral signs can be central. So it's very important to look at the whole picture. Okay, then I think it seems there are no more questions and I think that we have arrived at conclusion.

Other questions from Doctor Pavlov before ending this first session of the course.

No? Okay, so Doctor Pavlov, I think that we have arrived at conclusion. Thank you so much for your presentation. A fantastic learning opportunity. We look forward to

reconvening with Doctor Vishal Pavlov on March 20 for the second lecture in this captivating series.

The session will focus on vestibular test. The final online appointment with doctor Vishal Powar takes place on April 3. The session will cover positional testing for the treatment of common pathologists. As usual, I strongly encourage you to stay tuned to inventis social media channels and register for upcoming events hosted by inventis academia. Please remember to watch out for the upcoming survey and we greatly appreciate your input and suggestion.

Thank you very much doctor Pawar, see you in two weeks. Thank you very much my audience and thank you for your participation. See you next time with the second part of the course. Bye.