Fundamentals of Videonystagmography: Lesson 2: Spontaneous Testing, Gaze Testing, Head Shaking, and Hyperventilation

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Greetings everyone, and we extend a warm welcome to each one of you and express our gratitude for joining us. Today, inventis is privileged to introduce the second of three lectures of the course, fundamentals of vision theory, practice and clinical cases, featuring an internationally respected expert, Doctor Vishal Pawar from Dubai. Doctor Pawar is an eyelid skill specialist neurologist with over a decade of experience in neurology and auto neurology. Thank you very much, Doctor Pawar, 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 Dubai on June 8. Attendees will gain invaluable insight into the intricacies of vng procedures after discussing the oculomotor tests. Today's second session of the course will focus on vestibular tests, particularly on spontaneous nystagmus in light and dark, edge shaking and hyperventilation tests. We 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 our educational support to meet your needs effectively. And now I leave the word to Doctor Pavar. Enjoy the webinar.

Thank you so much Anna, for the kind introduction. Good evening everyone from all parts of the world. Good evening from Dubai. So, welcome to the second session of this course where we are discussing the fundamentals of video stagnography. Last time we covered saccade, smooth pursuit, optokinetic testing.

This time we are going to see spontaneous testing, gaze testing, head shaking and hyperventilation. So I'll those who are attending this, those who missed the previous webinar, I'll just revise the previous session in few slides and then I'll go to the today's topic.

So last time we have discussed there are six functional types of eye movement, like supranuclear eye movements, where there is supranuclear control over different group of eye movements. And we even discussed about the purpose of eye movements, like there are the purpose we discussed. So one by one I will tell you about different purposes. The first is.

Yeah, the first is hold images of the visual world steady on the retina during brief head rotations or translations. So when suppose I'm moving my head briefly. Suppose I'm moving it right or left. So during that brief rotation to maintain the. Suppose I want to keep looking at the screen and I move my head briefly to right or left.

That movement is brought about like by vestibulo ocular reflex. So, vestibular ocular reflex helps us to maintain the stability of the gaze, despite of the head rotations, which are brief. Then the second purpose is hold the image of a stationary object on the fovea, minimizing ocular drifts. Suppose you are looking at the screen and you don't want to, you know, you want to avoid the saccadic movements or microsaccades you want to fix on the screen. Then that is done by the visual fixation.

The third 3rd purpose of eye movement is hold images of the visual world steady on the retina during sustained head rotation, or translation means. Suppose you are translating in a train. Suppose you have, you are sitting in a train and you are looking outside to the scenery, or you are on the platform and looking at the, at the train. So when, whenever there is, you are either translating or the, the visual, you know, whatever you are looking at is translating in a fixed direction. What happens is to keep the visual world steady, there is a reflex called as optokinetic reflex.

So, so that helps in keeping the like, you know, this particular, in this particular scenario, keep the visual world steady on the retina. Then next is hold the image of a

small moving object. If there is a small moving target on the fovea, and if it is moving in a slow speed and it is at long distance, then what happens is slowly the eyes are able to pursue that target with the help of smooth pursuit. So the nav itself indicates that we can smoothly pursue the target because of this smooth pursuit movement. Then the next purpose of the eye movement is bring images of objects of interest onto the phobia.

Means, like, you know, for example, if I'm looking here and suddenly something appears in the peripheral visual field, and then if I have to look at that particular object, then I will suddenly move my eyes towards that direction. That is particularly done by the saccades, and move the eyes in opposite directions. So that images of a single object are placed or held simultaneously on the fovea of each eye is brought about by virgins. For example, an object moves towards the eyes. Then the both the eyes will converge to look at the near, near approaching object.

And if an object is, you know, moving far away, there is a little divergence, little divergence of the eyes. That is, you know, that happens because of simultaneous contraction of the lateral rectus of both the eyes. Now, last time we discussed about how the saccharine testing is performed. Now, if you look at here, okay, the saccades are nothing. But from here, the eye is moving to the left, moving to the right.

Okay. And you can see the Sacchari graph from left to right here. Then it is maintained steady here. And then it goes from right to left. Okay.

So the stimulus is moving. You can see the green one is the stimulus. And like the, this one particular is, there is a latency. There is a latency of milliseconds, like up to 200 milliseconds. And then there is like, you know, saccade from left to right.

Then there is, you know, it is maintained in that position. And then it comes, it can either come to the center or it can go to the left side. So here you can see now the subject is sitting, wearing the goggles. And the object, this thing, like it is moving from one area to the another area directly. It move, jumps from here to here, left to right.

And then jumps from right to left. Okay. So this kind of movement is called as saccadic moment. And this, like, you know, this is a horizontal circle, horizontal circuit.

So the cerebrum decides, we have seen last time when and where to move. And the brainstem determines how to move the eyes with the help of the various gaze centers. Now, we have also seen last time about the smooth pursuit eye movements where you can see the other object is moving, swinging from right and left, right to left. And it is not disappearing. It is smoothly moving in the screen.

And if you can see here, the eyes are also smoothly tracking the object from right and left. And you can see they. Here the graphs are recorded. Smooth pursuit. Graphs are recorded.

Okay, so this is about the horizontal smooth pursuit. Now look at the vertical smooth pursuit. The object or the object of regard is moving up and down and the eyes are pursuing vertically. Okay. And that moment is this vertical smooth pursuit.

And it will give the gain right to left gain, rightward gain, upward gain and downward gain. It will give, in case of horizontal, it will give rightward and leftward gain. And they are falling into the normal lay. The precision is normal. Like, uh, so this is again, uh, regarding the vertical smooth pursuit video.

The eyes are moving up and down. And you can see the graph is recorded here. Okay. The smoothly smooth pursuit graph is recorded here. Next.

Yes. Last time we have seen the optokinetic stimulus is given. Okay. This is optokinetic stimulus. And you can see the optokinetic nystagmus which is recorded here on the graph.

And the eyes also is showing. This is a normal nystagmus. This is a physiological nystagmus. You can see which is a left beating, stagnant left beating stagnant you can see when, like, you know, the stimulus is moving from left to right, left, beating, misdiagnosis seen. So that was revision of the previous session.

We discussed three testings, saccharic testing, smooth pursuit and optokinetic. So this time we dive into the spontaneous testing, uh, the first part. So in this, basically when you look at the eyes of the patient, uh, with the help of, you know, the video nystagnography, we are planning, we are, you know, aiming to check for the nystagmus. But there are other things which also we can examine. Uh, that is skew deviation, which is a very important central sign.

Ptosis of the eyelid, like that is grouping of the eyelids. And pupillary dosimetry also is very important because it is a. Another central sign. So, like. So main focus of spontaneous testing is to check for any nystagmus.

So let us go into the details of the nystagmus.

So, yeah. Yes. So let us dive into the nystagmus. So before going into the nystagmus just now, I have showed two things, like two pictures where this. You can see the video nystagnography goggle when the visor is open.

Okay. It is. We are testing with fixation. The patient is fixating. That is, it is in light.

So patient is fixating on the target object of regard. And you can see the visor on, like that is the lead is on. And you can see me in the background. So this is without fixation and SPN in dark. This is spontaneous testing in dark.

Like patients are. Is completely cut off from the visual stimulus. And here you can see. One good thing, like about this picture is you. This is, you can see in this photograph, this thing.

This area is very clear and you can see the. This background. Even my face is looking blurred. This is how our peripheral vision is. Now.

You are now, currently you are focusing on the screen. And if you keep your focus on the screen and try to look at the objects in the periphery, they appear blurred. Okay. That is because only the screen is on the fovea and parafoveal region. And the other part, other parts you are able to see is in the peripheral retina.

So that's how. That's why my image is blurred. That is in the peripheral retina. And actually in the photographs also we can see the blurred background. So that's a peculiarity of this photograph.

Then, like regarding the nystagmus, it's very important to understand the nystagmus because that is the most crucial aspect of video oculography. That's why, you know, even it is named as video nystagnography instead of video calligraphy. The, ideally, the name should be video oculography because it helps to check all types of eye movements, not only nystagmus, but because we have more, you know, emphasis on the nystagmus. So it's very important to understand what is nystagmus, what are the attributes of nystagmus and what are the why we are trying to look at the nystagmus. So let us go into that.

If we look at the nystagmus, it is involuntary moment, it is not a voluntary moment. Second, it is a rapid movement. Third is it is rhythmic, there is a rhythm involved in it, and it is oscillatory moment. It's, these are oscillations, like to and fro oscillations. And it, it must have at least one slow phase.

Either it can have one slow phase or it can have two slow phases. If it has both the fast phases, it is not a nystagmus. It is nystagmus like moment, which is nothing but some type of saccade. We are going to see that. So nystagmus is underutilized in the diagnosis.

It is used only 5% of the times in the emergency rooms and many a times when it, whenever it is labeled, it is labeled as nystagmus is present. But it is very important to characterize the type of nystagmus that gives the diagnosis. Why we are trying to do video nystagnography or video oculography is basically to know what is the type of nystagmus, what are its features, how it is variating with the gaze position, how what is the variation without fixation. So we are trying to look into the eyes very in detail, like video nystagnography, I would say is nothing but looking in, looking at eyes under a microscope. We are trying to, you know, categorize each and every eye moment so that we get the diagnosis, we get the localization.

So nystagmus is a common diagnostic challenge. Causes of nystagmus can be physiological, as we have seen, like optokinetic nystagmus, or it can be life threatening, for example, like some types of nystagmus, like bronze nystagmus, rebound nystagmus, which is of cerebellar pathology, down weakness, diagnose. So. And most important thing is the differentiation between central and peripheral variety of nystagmus. If you look at the most common type of nystagmus is vestibular type of nystagmus, peripheral vestibular.

But of course, there are central vestibular type of nystagmus, and there are other types of other varieties of nystagmus. And one of the major area of concern is, am I missing a central pathology when I look at nystagmus? The most common type of nystagmus is peripheral, but poor central nystagmus is very important to identify central nystagmus. And if nystagmus is present, it is of central variety, and MRI comes normal, then it creates a lot of diagnostic confusion and anxiety. So, let us try to simplify this topic.

So I'm going to cover the basics, and there are 47 different types of nystagmus. So we are not going to go into the details. I'm going to just cover the basics for the audiologist who are attending the webinar. I'm just going to touch upon the basics so that you get the basic concepts right. Then it will be very easy, you know, to do this testing.

Why we are doing this testing. We will be able to understand that. Now, coming to the practical aspects of nystagmus. Like, you know, there are the why we are trying to do the nystagmus testing is the most common type of nystagmus is peripheral vestibular. Peripheral vestibular.

Either it could be, or it could be originating from the brain stem, or it can be originating because of the cerebellum or less commonly, the supratentorial structures. Okay, so why we are doing this nystagmus testing? We are trying to localize the region. Now coming to the nystagmus attributes. Like, it's very important to understand when we are recording the nystagmus by video nystagnography with the help of video equipment, VnG equipment, we need to understand some terms are very important and crucial.

The first thing is we must be able to differentiate between jerk nystagmus and pendulum nystagmus. If. If a nystagmus or if a like, you know, eye movement abnormality, you can say, or eye movement finding is having one fast phase and one slow phase, then it is called as jerk nystagmus. So there are three types of jerk

nystagmus with a linear slow phase, then with the exponentially reducing velocity, exponentially increasing velocity, slow phase. So there are three types of jerk nystagmus.

Then if the movement, like the extracurricular eye movement, is showing both the slow phases, it is called as pendular nystagmus. And if it, if there are both the fast phases, there is not a single slow phase. Then it is called as nystagmus like movement. It is not nystagmus. Then there are variety of types like macrosaccadic oscillations, like ocular flutter, like micro square wave jerk.

Macro square wave jerk. So these are the types of eye movements which are nystagmus like moments. I am going to show schematically each and every of these types. So for example, jerk nystagmus. So it begins with a slow, this is a slow phase of the nystagmus.

And so have you seen the slow component? The slow component was going towards the. So this is the right and this is left. Okay. And this is right eye.

This is the left eye. Now, again, you look at this slow phase. It started from here and it is going towards, from right. It is going towards the left. Now look at what happens.

The slow phase is because it is the genesis of the nystagmus. It is actually the nystagmus begins because of the slow phase. And then the brain realizes that this is abnormal movement and it tries to correct with the frontal eye field area. Just now, have you seen the, there is, there was a fast component which, which beat from the, towards the right side. So in the video, in stagnography, the graphically it will be recorded like this.

This is a fast component and this is the slope. Okay. Then fast component is a corrective component. It is a secondary component, not the primary. But unfortunately the nystagmus is named after the fast component because it is visible.

Why it is named after fast component is because we, our eyes cannot appreciate the slow component. We will be able to appreciate this fast component. So conventionally, the direction of nystagmus is defined by the direction of the fast component. So now how will the nystagmus will look?

Okay, so can you visually, like, you know, appreciate that there is a slow phase from right to left, but there is a fast phase which is beating towards right side. So the nystagmus is named after the fast component. So then this nystagmus is right beating nystagmus, right beating jerk nystagmus because it has one fast component and one slow component. It's very important to know that. And the graphical representation will be like this.

Okay, so the graph will be recorded from like this.

So this is a slow component and this is a fast component. This is a slow component of the nystagmus. This is the fast component which is on the graph. And you can see the slow component which is going towards the left, and fast component which is beating towards the right. Okay, so I hope EveRYone is able to appreciate that.

Now, let us look at the pendulum nystagmus. So the slow component from left to right and graphically it is looking like this. And there is another slow component from right to left. Okay, there is another slow component.

So like this, this nystagmus will be seen like the pendulum is diagnosed. Now let us look at the difference between what is meant by nystagmus trajectory. What is meant

by nystagmus direction. Okay, now if you look at this particular nystagmus, try to appreciate what is the, you know, the direction of the nystagmus. The beating of the nystagmus is towards the right side.

This is the right side.

This is the left side. The nystagmus is beating towards the right. Okay, now let us look at what is the, what is meant by trajectory and what is meant by direction. What is the starting eye position? If you look at the starting eye position of the fast phase, not the slow phase.

So the fast phase is suppose from here the green line and the ending eye position is here. Okay, so the pathway which is followed the path taken from the starting eye position to the ending eye position during one half of the cycle of the moment is nothing but the trajectory. So there are three trajectories which are possible. One is horizontal trajectory, second is vertical trajectory and third is torsional trajectory. Either.

Okay, so this is trajectory. Now let us look at the direction. Okay. In horizontal trajectory, there are two possibilities. Okay.

One is either it can be towards the left side. Now here you can see that is fast phases towards the left and the slow phase is towards the right. So this is nothing but the left beating nystagmus. The first one is nothing but the direction is left beating nystagmus. And the lower one, the direction is right beating nystagmus.

If you can see the fast phase is towards the right and the slow phase is towards the left. Okay, so there are two types of horizontal trajectory. One is left beating and one is right beating. Now, on the video nystagnography equipment, how we record this is

slow phase towards the right and fast phase towards the left is. This is a left beating trajectory.

Left beating nystagmus is recorded like this.

So you can appreciate in the graphically, the left beating is like this and right beating will be like this.

The fast face towards the right, slow face towards the left, fast face towards the right, slow face towards the left. Now, there are two types of vertical, like two directions of vertical trajectory. The first one is now I would like you, all of you, to appreciate, like can you appreciate that the fast phase is up and the slow phase is to download. So nystagmus is named after the fast component. So this is a upbeating nystagmus.

And if you look at in the down, uh, like, you know, the lower part, if you look here, the fast phase is towards the down and the slow phase is towards up. Okay, so nystagmus is named after the fast component. So that's why it is a down beating nystagmus.

And if you look at the, like, you know, this, I didn't tell about this. This is a horizontal trajectory and this is vertical. In the recording, if you video in stagnography tracing, this is horizontal and this is vertical. So if you pupillary tracking, if you see in the horizontal trajectory, it is almost, you know, it is flat line. But in case of this vertical treasury, this thing, you can see a down beating stagnant.

Because this, this slow phase is up. Then it is fast phase, low phase is up. There is a fast phase, low phase, up, down beating stagnant will be seen like this. And so in up beating nystagmus, what will happen is the fast phase is up, then there is a slow phase. Fast phase is up, then there is a slow phase.

This will be seen in the vertical tracing, not in the horizontal. Horizontal tracing will come as flat unless there is oblique nystagmus. If a person has oblique nystagmus, even this stress will show some nystagmus. In the, if it is a pure vertical, it will clearly show only in the pure. In the recording of vertical, if it is a horizontal nystagmus, it will show only in the horizontal tracing.

If it is oblique nystagmus, anywhere in between this, then both the graphs will show some movement. Like, you know, the nystagmus, depending on which side it is more like up, is it more predominantly vertical versus horizontal. So similarly, in case of torsional, there are two trajectory, two directions which are possible. Either the upper pole can beat towards the right. Okay.

From the observer's perspective, if you look at, if you are looking at it now, you are feeling like it is moving like this. Okay. So the, is this clockwise or anti clockwise? If it is moving like this, okay, it is moving like this. Sorry.

Here, it should not be like this. It should be moving like this. So it is moving anti clockwise direction. Now I will tell you what is the fallacy of this particular. From the patient perspective.

If you are a patient, if you. From the patient perspective, actually, this is clockwise for the patient himself or herself. That is the problem with this clockwise and anti clockwise terminology. That's why Barani society came up with the consensus that we should avoid the terminology like clockwise and anti clockwise. And the terminology used should be upper pole of the eye beating towards the right side or towards the left side.

Okay. So either it can be a dextro cyclo-rotatory, that is upper pole beating to the right, or it, we should mention it as levocyclo-rotatory, that is upper pole beating to the left.

Okay, so these are the two directions which are possible in torsional nystagmus. So there are three trajectories and six directions.

Now, there is very important concept of reference frame. Now, if we look at the nystagmus, either it can follow eye reference frame or it can follow. Follow the head, head and canal reference frame is same because the canals, semicircular canals are fixed into the head. The semicircular canals are fixed into it. They move along with the head.

So both these frames are equal, like semicircular canal, semicircular canal frame and the head frame are same. And the earth reference frame, the nystagmus can be even labeled based on earth reference frame in some settings. Not in every setting, we should label it as in the earth reference frame. So eye reference frames are like, there are three eye reference frames. If the eyeball, there are, there is a vertical axis, anteroposterior axis and horizontal axis, then I won't go into the details of that because, you know, I will try to keep it as simple as possible.

In case of head reference frame, there are planes like, this is sagittal plane, this is coronal plane. And this is horizontal plane, horizontal. And the canal reference frame is similar to the head reference frame. But the canals are not exactly in the plane of, you know, sagittal plane. For example, the horizontal canals are not exactly horizontal.

It is the. It is making an angle of 30 degree. Okay. The anterior end and the posterior end are not exactly, you know, in. In one horizontal line, the anterior ends are 30 degree to the horizontal.

Like, you know, it makes an angulation. And the posterior canals are not exactly posterior. They are making an angle of 56 degree with the sagittal plane. And the

anterior canals are not exactly anterior. They are making an angle of 41 degree with the sagittal plane.

Let's look at the gravitational reference frame. That is earth reference frame. Now, in the earth reference frame, you can see for all of us, the it towards the gravity, that is like towards the gravity, towards the center of the earth is nothing but geotropic. And if it is away from the gravity, it is apogeotropic. It's very important to avoid the term a geotropic.

It is apogeotropic.

Now let us, I will give an example. Suppose this is the head of the patient. This is the bed. Patient is lying down. You are looking the patient from the head end.

And we turn the patient, suppose towards the right side. Okay. Then this is the right horizontal canal. And this is the ground. Okay.

And the eyes are, eyes will be like this. The left eye is here. Right eye is here. Okay. So once again, I'll show you.

Okay, so patient turns towards the right side. This is the left eye, this is the right eye. And the patient turns towards the right side. And suppose the patient has this kind of nystagmus. So it is beating towards the ground.

It is a right beating nystagmus. The fast phase you can see is towards the right side. In this context, we should not, we can call it right beating nystagmus also, but in this, you know, position, it is beating towards the ground. That's why it is called as geotropic nystagmus. Here the special term earth reference frame is used and we call it as a like, you know, geotropic nystagmus.

And if it beats away from the ground, away from the ground, it is called as apogeotropic nystagmus. So the geotropic and apogeotropic term is used specifically in the context of lateral canal BPPV. And sometimes it is used for posterior canal also like there is apogeotropic variety of posterior canal, which I am going to explain in our third session because that is reserved only for the positional testing, positional maneuvers.

Now let us look at what is meant by straight ahead position, which is also called as center gaze.

So whenever we are looking straight ahead, that is in the plane, frontal plane, we are, we are in the frontal plane, but in the anteroposterior axis, the eyes are looking forwards. That is nothing but the straight ahead position. Earlier it used to be called as a primary position, but Barani society recommends to use this woo terminology, straight ahead position or center gaze position.

Then the next attribute of the nystagmus is whether it is originating. The nystagmus is coming from one eye versus both the eyes. Okay, so if a nystagmus is originating in both the eyes, it is called as binocular nystagmus. If the nystagmus is seen only in one eye, which can happen in case of internuclear ophthalmoplegia, it can happen sometimes in migraine, it can happen in epilepsy, it can happen in glaucoma. So there are certain etiologies where it can happen that there is adduction, paralysis.

Adduction is not happening and there is abduction, stagnant. So in this case it could be a case of internuclear ophthalmia. So there are a variety of causes which we are not going to go into details, but you should be able to identify that the anastomosis is happening in one eye versus both the eyes. Okay.

Then there is another issue, that is whether the nystagmus is conjugate versus disconjugate. Conjugate means it is happening equal in in the direct, in the same direction and with the same velocity and amplitude. Then it is called as conjugate misdiagnosis. For example, this is a right beating example of right beating nystagmus. And it is happening equal intensity in the the size more or less equal intensity.

It may not be exactly equal because there are two different muscles which are acting. Here there is a lateral rectus. Here there is a middle rectus. The nerve supply is different. The number of fibers, muscle muscle fibers in that muscle are different.

So it may not be exactly equal, but by and large it is in the range of 30% equal, like 30% variation from one to another side. But if there is a different velocity or amplitude of the nystagmus, more than 30%, it is this conjugate the type, the type of nystagmus even though the direction is same, okay. But the velocity and amplitude is different. It is called as dissociated variety of dis conjugate nystagmus. Okay, so one eye is moving faster and one eye is moving slower.

It is a dissociated nystagmus. And there is another type of disconjugate which is called as disjuncting nystagmus. That is, one nystagmus is beating towards the left side and one eye is beating towards the right. It is a horizontal variety of disjunctive, that is convergent divergence nystagmus. Then there is a vertical disjunctive nystagmus.

It is called as c saw nystagmus. This is beating up. This is beating down. It is called as season. And there is a third variety that is cyclo-vergent.

Nystagmus, one is beating to upper pole, beating to the left. This side, upper pole is beating to the right. cyclo-vergence nystagmus. So these are the types of dis, conjugate disjunctive nystagmus.

Now, coming to the slow phase velocity, which is a very important parameter. For ExAmple, you look at the, both the nystagmuss and, and just see what is happening.

Both nystagmus are beating towards the right side. Both are beating towards the right. Okay, this is the right side. Can you appreciate that the fast phases are towards the right side in both the cases. But I'm sorry, the lower one, the right sided eye, this is looking like a monocular nystagmus.

But that animation is not working. But this is both sides. Just assume that this eye is also moving in this slow phase velocity. So if you look at the both the eyes, this eye, this right beating, is faster as compared to this right beating nystagmus. So the upper panel having higher velocity as compared to the lower panel.

Okay? So that is the importance of the velocity. So, and the slow phase velocity, if it is higher, then the fast phase velocity is, uh, like, you know, is proportionately higher. So that's how these two nystagmus, you can differentiate, then coming to the frequency, how many beats are happening in a second. Okay, that is the frequency in Hertz.

So if we look at the upper panel versus the lower panel here, the both the eyes are working. It is a binocular nystagmus. If you look at the frequency, it is higher in upper panel as compared to the lower panel. So it is higher frequency in the upper versus the lower frequency in the lower panel.

Now coming to the amplitude of the nystagmus. Now look at the nystagmus. This is fine nystagmus, which is higher in, you know, frequency, but amplitude is short. The amplitude is short, okay. The frequency is high.

The amplitude is short, and it is. That's why it is called as a fine nystagmus, as opposed to. Look at this nystagmus. The amplitude you look at is, is bigger here. The amplitude was smaller, okay.

As compared to this amplitude. Okay. Smaller amplitude. This amplitude is larger. Okay.

And if you look at the frequency, also, the frequency is faster here as compared to the, this side. So this is a coarsene, stagnant. Okay. This is a fine nystagmus, and this is a coarseness diagnosis. If you see a patient with, like, suppose when the patient looks toward the right side and gets fine nystagmus, and patient looks towards the left side, and the same patient gets a coarse nystagmus.

Then it is called as bronze nystagmus. Okay. And this is say, seen in Cp angle lesion. And the lesion is on the side of the porcelain because the vestibular nerve gets damaged here, it leads to the left unilateral vestibulopathy. So the left unilateral vestibulopathy will cause right beating nystagmus.

Fine nystagmus, which is seen on the right beating. And because of the compression of this tumor over the cerebellum, it leads to the gazebo nystagmus, which is a coarse nystagmus. It is beating towards the right side, which is beating towards the left side. So this coarse nystagmus is due to the neural integrator issue.

Now, coming to the spontaneous nystagmus testing in the spontaneous nystagmus, what happens is you can either see spontaneous horizontal nystagmus in the center gaze, you have to check in the center gaze. From the center gaze. The nystagmus is beating towards the left side, so it is a left beating nystagmus.

So if you look at this nystagmus, this is beating down. It is a spontaneous down beating, stagnant. Why it is called spontaneous because it is in the center gaze.

Now, if you look at this particular nystagmus, the upper pole is beating towards the right side, so it is a torsional dextro cyclo-, spontaneous, pure torsional nystagmus. Okay.

If you see a patient in the center gaze, okay. If the patient has center gaze, this is left beating nystagmus and the patient either may not have nystagmus on gaze testing or may be present the same nystagmus, or it can get increased in one direction, or it can get decreased in one direction. So it can, all the variations can happen, and that decides whether it is central or peripheral. So I'm going to come to that.

And another peculiarity I will tell you. If, if. Suppose nystagmus has all the three components. Suppose a patient, nystagmus has a beating component, upbeating torsional component, and upbeating. Suppose oblique, like all the three components are there, which is classically seen in peripheral in this patient, if you, if you ask the patient to fix, say, the horizontal component and the vertical component will get suppressed, but the torsional component is very difficult to suppress even with fixation.

So it might appear that it is a pure torsional or peripheral nystagmus. Can appear as a pure torsional in the center wave. But it's very important, that's why it is very important to remove the fixation and check the same patient, and then we can appreciate the vertical and horizontal components. So that's why it is so important that we check the patient without fixation. And with the help of video calligraphy equipment, it is very easy to put the flap and put the flap, put the, this, you know, like that visor.

And that helps to block the fixation.

So these were the different attributes of nystagmus. Now I'm going to talk about the influencing factors. What can influence the nystagmus one is gaze position, can influence visual fixation, can influence vergence, means convergence, can change the nystagmus. And it's very important to identify what pattern of nystagmus a particular patient has. Let us look at the gaze positions, like effect of gaze positions.

Now, if the patient is looking straight ahead, this is a center gaze or straight ahead gaze. If the patient looks towards the right, patient can look towards the right, or the patient can look towards the left. Okay, so there are two gaze positions horizontally, either to the right or to the left. Right or left.

Now let us look at the recording. This is the object of regard. It is moving, move towards the left. It stays there and then it moves towards the right. And you can see in the.

Here it is. The eyes are moving to the right, moving to the left. And the visor is open. This flap is open. So it is a test with fixation.

It is, it is in light. We are doing it in light. That is with fixation. Okay, so in vertical gaze testing the same object of regard, we have to ask the patient to follow. It will move up, it will move down.

It will stay there for the up gaze. It will stay there for the down gaze.

It moved up, now it will go down. And if you look at the eyes, they move up, then it goes down. So the graph also, if you can see it, when the eyes move up, it will go up, then the stimulus is steady. It stays there and then it moves down. Either it can come to the center gaze or it can go towards the left, to a down also.

And then like this, the graphical representation of the particular, this particular gaze testing can be recorded.

Again, you can see here with fixation. Now, in with fixation, there is no visual cue to the patient. So we have to give auditory cue. Like we have to tell the patient, look towards the right or look towards the left. Look up, look down for the horizontal testing, we have to tell the patient look to the right, look to the left.

For the vertical testing, ask the patient, look up or look down.

Now, what is meant by gaze evoke nystagmus? It is a burning question. Like gaze evoke nystagmus is a very hot topic as far as nystagmus is concerned and it is alarming sign. So now if you look at this nystagmus, now conventionally, this is right, this is left. Okay.

So when the patient is looking towards the left, the nystagmus is beating towards the left. Okay. When the patient is looking towards the right. Now what is happening? You see the nystagmus is beating towards the right.

Okay. So look at me now in the video, when I am suppose looking towards the right, it beats toward the right. And if I look towards the left, if it beats towards the left, then it is called as gaze evoke nystagmus. Okay? And that is a central sign.

It's a very important central sign. As soon as you see this gaze evoke nystagmus. In an acute vestibular syndrome, you should suspect central pathology.

Now there are two possibilities. Either in the center gaze the patient may not have any nystagmus and have gaze u of nystagmus. Or in the center gaze, the patient has nystagmus and the patient has, you know, in the center gaze, if you see the patient has

left beating. When the patient looks towards the left side, there is a left beating, stagnant. When the patient looks towards the right side, there is a right beating nystagmus.

So this is nothing but gaze yoke nystagmus. Combination of gaze evoked and pure horizontal nystagmus. In such cases, it is very important to check for periodic alternating nystagmus. In these patients, it's very important to check the nystagmus for 90 seconds and check whether the nystagmus is altering its direction after 90 seconds. Now look at this particular nystagmus in case of peripheral nystagmus.

In case of vestibular neuritis, patient suppose looks towards the left side. Suppose there is a left beating nystagmus and if the patient looks towards the right, it is not right beating. It is still remains as left beating. Then it is called as unidirectional nystagmus. Despite of the change of the gaze, the nystagmus is remaining left.

The direction is not changing. So it is unidirectional nystagmus, which is classically seen in peripheral etiology. But in case of central. Just see what is happening. Look right.

When I ask this patient to look to the right, it is a right fitting nystagmus. Look left. When I ask the patient to the look to left, the patient is having left beating.

I will play this video again so that you appreciate the gaze yoke mis fragments. Look right.

You're seeing double. Look left.

So in this patient, when the patient looks to the right, right beating. Patient looks to the left beating. Of course there is a down beating component. Keep looking up. So that is typically a gaze evoke nystagmus.

It was a patient of cerebellar downbeat nystagmus syndrome and the patient had cerebellar paraneoplastic pathology. Now look at this patient. When the patient is looking towards the right side, there is a right beating nystagmus. Patient looking to the left, there is a left beating nystagmus. Patient looking in the center, there is a pendular variety of nystagmus.

In the center.

When the patient is looking up, it is still beats horizontally. It is beating horizontally in even in vertical direction. Even in the down gaze. Also it is beating horizontally. Okay, so this is a typical case of infantile variety of nystagmus.

Okay. Congenital, stagnant. Now let us look at the fixation like methods I use for fixation Vng. Obviously the visor is provided with the VNg equipment to close then observing the patient in dark, which like even the patient can't see and we can't see. That's why it's very important to use some method.

Check this. Frenzel glasses is a traditionally used method. M glasses, like Professor Michael Spruce glasses and rohiwal glasses. Like one indian professor from, he's a neurotologist. He has, you know, modified the friends and glasses into like type of, you know, it can be used for without fixation.

Like it can be used to remove the fixation. So with the help of the video oculography equipment, this is with fixation. And this we are you, we are blocking the vision. And this is without fixation.

So whenever the nystagmus, okay. Is present without fixation and it gets suppressed by fixation, then it is usually peripheral. And usually the central nystagmus doesn't get suppressed by fixation. That is central. But the central has liberty of having both the types of nystagmus.

In general, central has liberty of having even peripheral signs. So one dictum you always remember, if you see a central sign, it is always central. And if you see a peripheral sign, it can be either central or peripheral. This dictum you just remember because if you see a central sign like gaza, ok, nystagmus, it is central, but if you see unidirectional nystagmus, usually it is peripheral but it can still be central. So that's very important.

So like there another influencing factor is convergence, like which, you know, the infantile variety of nystagmus gets suppressed by convergence, then pattern recognition is very important. For example, these are the nine cardinal gases. And in this patient, suppose there is non.

I have just used a schematic diagram. Suppose you get this type of nystagmus in the center gate that is up beating and torsional upper pole beating to the left side and this is in the torsional upbeating in the center gaze, and on one side, if the patient looks it is more upbeating and on other side it is more of a torsional and it increases in the up gaze. It is typically a peripheral variety, that is posterior canal, bppv upping torsional nystagmus. Then if you see with fixation a patient, and suppose you find this variety of nystagmus, a patient is having left beating nystagmus in the left gaze, left beating in the center gaze, and there is no nystagmus in the right gaze. Suppose this is with fixation and without fixation.

If suppose in the same patient, if you find enhancement without fixation, which is increasing on the left gaze and it is still there, it is still left beating on the right gaze. So it is unidirectional variety of nystagmus.

It is following the Alexander's law. So that is it is a peripheral typically seen in peripheral, unilateral, acute unilateral vestibulopathy. Now if you find a nystagmus where you know there is no nystagmus in the center gaze, when the patient looks to the right, there is a right beating nystagmus and patient looks to the left, there is a left beating, it is a gaze u of nystagmus. If suppose a patient has horizontal nystagmus in the center, right, left, up down, it is a infantile variety of nystagmus. Congenital nystagmus.

If a patient has down beating in the center and down gaze and side pocket nystagmus in the side gazes, then it is a typically a downbeat nystagmus. It is a central nystagmus. Now the reference frames I have discussed. The central nystagmus follows the eye reference frame. Okay.

The peripheral nystagmus follows the head or semicircular canal reference frame. And for lateral canal and posterior canal we use the earth reference frame. Of course in central nystagmus also earth reference frame can be used because there are there is apogotropic variety of nystagmus can be seen in some central pathologies. It is not restricted to central or peripheral. These are the reference frames.

All frames are equally valid. Selection of frame is linked to the mechanism of nystagmus. Specify the frame. And again this is the that same infantile variety of nystagmus. So the central nystagmus, remember three important features.

There is no separation on fixation. If you see a gaze evoke nystagmus, it is central. If you see a pure variety of spontaneous nystagmus in light until crude. Otherwise it is a

central status. Now let us go to this provocative testing like the last another ten minutes we will discuss the provocative testing.

So there are various provocative testings for the nystagmus and I have divided them into three parts. The one which localizes to the central one which is there are three which localizes to peripheral and either it can be central or peripheral. There are provocative testing. So the two of the tests are which are very promising. Is head shaking test and hyperventilation test.

Let us discuss. So the one of the very important thing I would like to tell is if whenever we are doing vestibular testing it gives findings. We have to interpret the diagnosis based on the clinical scenario. The aim is to estimate the presence of a disorder rather than to verify or quantify it. And the vestibular test battery indicates vestibular test system abnormality rather than the lesion localization.

For example, if there is gaze evoke nystagmus versus a unidirectional nystagmus it will give, you know, abnormality. But lesion localization we have to do based on the other associated finding and the clinical scenario. So head shaking test, what happens? It is a test which screens the vestibular system abnormality by inducing stagnant to active or passive head shaking. The patient's head is taken right and left.

It is shaken at 2 hz frequency. I'm going to show the video and then we have to check for whether there is any nystagmus which is seen. That is post head shaking nystagmus. Now let us see. He is our audiologist.

We can call him vestibulogist because he is now expert in vestibulology also. So he is performing the head shaking test, you can see the head shakes. Okay. After the head shake, he has stabilized the head. And then we watch for the nystagmus.

I will play this video again.

He's shaking the head at 2 hz frequency. That is two times in a second. And if you can see there is a head shaking waves followed by if there is non stagnant, it is a straight line. If there is a left beating, there is a left beating. Or if there is a right beating, there will be right beating stagnant.

So this is how you know this. Or it can be even up beating down beating nystagmus also. So that is the head shaking test, how it is performed. We shake the head for 20 times. That is 10 seconds, 2 hz frequency.

It can be shaken for ten to 20 seconds. And then you can see the beautiful graph that is head shaking graph is here. And you can see the horizontal trace here and the, uh, vertical stress. And you can see here there is, uh, horizontal head shaking, not vertical. So vertically there is little because you know, exactly.

We have to maintain the exactly. It should be moving the horizontal position. And then we can see the mean velocity, maximum velocity. And we have to check for the spontaneous nystagmus. Not spontaneous.

It is a head shaking induced nystagmus. There are certain limitations of head shaking test, but it is sensitive. Sensitivity is moderate. Specificity is also they're relatively high. It has some limitations.

Equipment. We can use it with the video oculography. And we have to check for the, basically it is a test to check the vestibular asymmetry. We are checking the vestibular asymmetry by giving the jerks. So now I will tell you very important thing about head shaking.

Like suppose, like what we have to do while doing the shaking is you little bend the head by 30 degrees so that the horizontal canal become exactly horizontal. When you shake the head towards the right side, the right semicircular canal is activated. When the head is shaken towards the left, left semicircular canal is activated. Suppose you consider that one moment is a stimulation of one plus. So when I move towards the right side, there is one plus stimulation of the right left side, one plus right, one plus left, one plus.

So suppose you check 20 times, there is a 20 plus and 20 plus on right, 20 plus on the left. If both the vestibular systems are functioning equally, there is a known stagnant. Suppose the right side vestibular system is not working. Then you move to the right, zero move to the left, one move to the right, zero move to the left, another one. So 20 times you move it is 20 plus here and zero here.

This 20 plus will get accumulated because of the velocity storage mechanism. And there is a stimulation of the normal side, left side. It will fire. And it will fire. That is slow phase towards the right and fast phase towards the left.

So the nystagmus will be towards the left side. That's how paralytic nystagmus will be seen. And there are again, there are varieties of nystagmus in that biphasic variety, uniphasic variety. So that is about head shaking nystagmus. If you see vertical nystagmus in a horizontal after horizontal head shaking it is called as cross coupled nystagmus.

It is a sign of central. But of course sometimes it can be even seen in peripheral theology. But like you know, until proved otherwise it is central. Coming to the hyperventilation testing. In hyperventilation testing it is the only test which unmasks the vestibular deficit without testing.

Dynamic properties of vestibulocular reflex means dynamic properties means head shaking. We are doing is a dynamic aspect of the vestibulocular reflex. But hyperventilation involves just hyperventilation breathing to elicit the vestibulocular reflex abnormality. Now look at this test, how it is conducted. Again.

Here is our vestibulogist, mister Shauka. He is performing now the patient is doing the hyperventilation.

Deep breathing with open mouth. One breath per second. One inhalation. Deep inhalation per second, one exhalation 1 second.

So that this is how it is performed for 60 to 70 seconds. That is a minute. And you can see after the hyperventilation we are checking if there is any nystagmus. We are checking for the traces after the hyperventilation. Okay, so basically what hyperventilation does is it causes hypocapnia, alkalosis, biochemical changes, neuronal hyperexcitability.

Vestibular system gets stimulated. Central compensation mechanism get decompensated. And that leads to hyperventilation induced either parity or excitatory nystagmus. And after the hypocapnia goes away, then again it returns to normal. And again the hyperventilation induced nystagmus can convert to the spontaneous, either spontaneously stagnant nonstop.

Then the hyperventilation test is useful. You know, it can be seen in 22% of the vestibular patient. It is capable of revealing latent nystagmus. It can help to find out vestibular neuritis, caustic neuroma. Some central vestibular disorders.

And there is a very nice study by Professor Luigi where he has, you know, came up with an algorithm where if the nystagmus duration is more than 48 hours and the

patient has excited nystagmus, it is inflammatory in origin. And if it is parasitic, consider central. So it is a very nice study. I would like, you know, request everyone to go through it. It is to differentiate whether the acute unilateral vestibulopathy is inflammatory or vascular.

So, you know, this, this, like, you know, patients with a caustic neuroma shows high incidence of hyperventilation induced nystagmus. Patients with neuritis show high incidence of nystagmus. Vestibular migraine can show like hyperventilation induced nystagmus. Patient with multiple sclerosis can show. Cerebellar disease can show.

So if you see a hyperventilation nystagmus, it is not sign and con, with central variety, it can be seen in peripheral. It can be seen in central. So it is very important to check whether other signs are there. So none of the findings, you know, they can localize. You have to consider the clinical context.

You have to consider other signs to come to a conclusion. So there are variety of lesions which can show the hyperventilation induced nystagmus. So that is about hyperventilation induced nystagmus. And a short last slide about the peripheral variety of nystagmus. It can be, it is the most common variety of nystagmus.

And I have chalked down these types of nystagmus. In anterior canal BPPv, there is a down beating torsional nystagmus, posterior canal BPPv, a beating torsional lateral canal BPPV, it can be opposotropic or geotropic. In vestibular neuritis, it is horizontal torsional nystagmus. And in cases of, you know, differentiating regions, like specific regions of some canal, it can show specific variety of nystagmus. So this is our team.

He's our audiologist from vestibulogist, Mister Shoka, our physiotherapy team, dedicated nurse. I'm really thankful to Mister Shoka for helping me daily, on a daily basis for diagnosis and treatment of patients. Plus for this presentation, he was really

helpful to shoot the videos. And with this, we have completed the second online session for the video and stagnography course. The third lesson that is mainly dealing, going to deal with the positional testing will be on the 3 April.

And we have this practical hands on session. That is going to be very, very important because this is theoretical aspects we are dealing with, but practically how to perform stereography testing, what are the different protocols, how to interpret? I am going to discuss in detail on one whole day, on 8 June in Dubai. So with that, I thank you all for patient listening. In case you have any doubts you can put into the chat box, I will answer your questions.

So I will go into the question answers and start answering the questions one by one.

Yes. The first question is by Wayne harbor. In case of old neuritis, remaining head shaking test nystagmus means there is no compensation occurring. Negative HST means it has been compensated. Thanks.

Yes. So what happens is if the nystagmus disappears after the, you know, after the vestibulopathy, then definitely the tone of the both vestibular nuclei is same. That means, for example, if. Suppose a patient, normally the tone is this much, okay, normally both have equal tone. Suppose the.

Suppose I got right vestibulopathy, the right vestibular nucleus tone goes down in the acute stage and it recovers a little, and then there is compensatory strategy, and the left sided lowers down its tone. And suppose there is a compensation, and both sided tone is equal, but it is after the compensation. Okay, as opposed to another patient where the vestibular nuclear function is completely recovered, like the both, the tone are equal. In that case, you will not be able to see head shaking nystagmus in the

follow up. But as opposed to a patient where it is compensated, it is, you know, the vestibular system is trying to use its reserve to compensate.

In that case, you do head shaking, and that will decompensate the central mechanisms, and it will bring. It will bring out the head shaking induced nystagmus in the recovery. And if it is stronger, that means there is less compensation. And if it is less stronger, that means there is a less comp. Like, you know, the compensation is okay.

But still there is a vestibular asymmetry, which is getting masked by other mechanisms, again by the same, uh, you know, like, in case of fresh right neuritis, for example, which sense would you practice? Optokinetic reflex triggering, right or left nystagmus? Uh, so what happens in case of optometric, uh, you know, in case of neuritis? Suppose. Suppose, like, you know, we can give the stimulation either, like, from right to the left or left to the right.

Both stimuli should be given to the patient. It's going from right to the left and left to the right. So the gain, the gain. Suppose normally the gain of right beating nystagmus when the stimulus is moving from the right side to the left side, the slow phase will pursue from the right to the left and the fast phase will be towards the right side. So there will be right beating nystagmus when the optokinetic testing is performed from the right to left and there will be left beating nystagmus when the this optokinetic stimulation is given from the left to right.

Normally the right gain of the right beating status and left beating stagnant same. But suppose the patient has right unilateral vestibulopathy. Then what will happen is the patient will have a spontaneous left beating nystagmus. The right vestibulopathy patient will have spontaneous left beating nystagmus. So the gain of the left beating

nystagmus on optokinetic stimulation will be stronger as compared to the gain of right beating nystagmus.

So that's the answer. I hope you I answered your question the next what do you think of patient with right spontaneous nystagmus and then left head shaking test?

I didn't understand the question correctly, but. But I will try to answer. Suppose a patient, what he means to say is patient has right beating nystagmus. It is beating towards the right and the patients I feel that he is asking whether only left sided head shaking test is done. I'm not sure what he means to say, but that will reduce the intensity of the nystagmus.

Obviously, because you are stimulating the other side. This side you are trying to stimulate and we are trying to negate this effect of the right beating nystagmus. Because suppose in the left head shaking test you are stimulating the left side and the left side stimulation will lead to the left beating nystagmus and the patient has baseline right beating nystagmus. It will try to reduce it. I hope I answered his questions then.

Like is there a gender specificity in the type of nystagmus? As far as I know, there is no gender difference, like in patients when they have nystagmus, like whether they have nystagmus of neuritis, migraine, vestibular migraine, or central pathologies. There is no gender difference as far as I know of. Then anonymous attendee has asked, how many sessions of a police manure does it take to show improvement? To be honest, this is this question is related to positional testing, which I'm going to deal with next session.

But I will answer you. It depends on the debris. Like, suppose in one maneuver, if you are able to, you know, clear the debris, one manure is sufficient. In my practice, 80% to 90% of the times, single manure is effective to clear the debris. But sometimes, you know, some patients can need repeated maneuvers.

And if you are doing repeated maneuvers and the patient is not recovering, that means there are variety of, you know, possibilities. It could be a canal jam. It could be, you know, it could be like vestibular migraine. It could be vestibular paroxysmia. It could be other central pathologies.

So there are. And it could be light cupula syndrome. So there are variety of things then, like, for gaze evoke nystagmus, nystagmus urdan. For gaze yoke nystagmus, does it have to beat both towards the left and right to be classified, that is, bilateral? Or can it also occur, only occur unilateral and beat towards one direction upon gaze direction?

Very important question. So that's why the nystagmus is graded into three grades. Like, you know, the grade one nystagmus is beating towards only one direction. But suppose the nystagmus is beating towards the, like left gaze, it is beating towards the left. It is still left the gaze.

You have nystagmus. Okay, then, uh, if the patient, that same patient has left beating nystagmus in the center, it is grade two nystagmus. If the same patient has left beating nystagmus in on right gaze, it is grade three nystagmus, which is commonly seen in peripheral. And we should specify that it is present on only left gaze versus bi directional. In this patient, you will not call it as bi directional.

You will call unidirectional. It is seen only in one direction. Instead of calling it gaze evoked, we will call left gaze evoked nystagmus, which is seen only in one direction, versus a bi directional nystagmus, which is left beating and right beating also. Then Miss Shifali is asking, can you please explain cross coupled nystagmus. So, cross coupled nystagmus means what you are doing in normally, in case of head shaking, is you are shaking the head horizontally.

Okay. You are shaking the horizontally. You are stimulating the right and left lateral canal. Alternatingly, you are stimulating, you are not stimulating the vertical canals, but. And you are expecting that if there is a vestibular asymmetry, the patient should get either horizontal nystagmus, that is right beating, or left beating.

But suppose the patient gets up beating or down beating nystagmus after horizontal head shake. It is called as earlier it used to be called as perverted nystagmus. But the current terminology for this is cross coupled nystagmus. Usually it is seen in central pathologies. But there was a recent article where they found cross coupled nystagmus even in peripheral etiologies.

So it is not cyan cion of central. Until proved otherwise, it should be considered as central. And if central etiology is ruled out, then only it should be called as peripheral.

Like my colleague, I am my good friend. Like Miss Noor Salman. She is a very good vestibular therapist from Cleveland clinic. She is asking just a thank you for amazing information. Thank you so, so much, Noor.

Thank you so much for joining. Mister Abhishek is asking if I is beating towards left than problems in left side or right side. If I is beating towards the left, then problems in then problem left side or right side? See if the eye is beating towards the left. Okay.

Then you have to check whether it is unidirectional, stagnant or bidirectional. Okay. By checking in the center gaze and in the right way. So if the patient has unidirectional status which is suppressed by fixation, then the patient has right unilateral vestibulopathy will cause left beating nystagmus. But suppose the patient has neural integrator problem.

Still the patient can have left beating nystagmus. So that's how it is. Like left gaze will cause left beating, right gaze will cause right beating. It depends on the localization.

Miss Maria is saying. Thank you. My question. How about perverted nystagmus by Mister Abdel Wahab. I have answered that question.

The earlier name for cross coupled nystagmus was perverted. The terminology itself is perverted. So they have, you know, they. It is. It happened because of cross coupling.

What is cross coupling is you are stimulating horizontal canal but somehow there is a miswiring. You are trying to stimulate the horizontal canal, but vertical canals are getting stimulated or inhibited. And that is leading to nystagmus. That's why it is called as cross coupling. Okay.

And anonymous attendee, what could be the VOR gain for patients with chronic renal failure? CKD? To be honest, I have not done any study specifically in chronic renal disease. I have not come across with patients with chronic renal disease. And I have not done like, you know, VOR studies in this group of patients.

So I am. I don't have any experience. So maybe I will read about it and maybe I can give you answer in the next session. Mister Ricardo is asking, in the measurement of the caloric provoked nystagmus, how do you manage to evaluate the data if the patient deviate the gaze in just one of the ear stimulation? To be honest, Ricardo, we didn't discuss about caloric stimulation here in today's talk, or the last talk.

I will reserve this for, you know, in the further presentations. Then, would you give us some information about velocity storage? Mister Abdel Wehab is asking. So, velocity storage mechanism is nothing. But suppose the vestibular system is tuned to detect the acceleration, that is, change in the velocity over time, if there is a constant velocity.

Suppose, like, for example, you consider this as, you know, hair cell. Okay, so when the car starts. Suppose the car starts and you start moving forward, the. Then there is a hair cell goes backwards, you know, and that detects. You know, that detects the.

We have started like, there is detection of movement. Now, suppose after some time, you attain a nor same speed, then it will remain. The hair cell will remain in the steady state, and this velocity will be stored in the brain. The brain has velocity storage mechanism, which will. That we are moving in this much velocity.

And then when it is breaks are applied, then the hair cell will go forward, and the change in the velocity that is acceleration will be detected by the hair cell. So the velocity storage mechanisms are actually trying to store the velocity and they are trying to, you know, we are. It is helpful for the spatial navigation. It is helpful to store the linear acceleration. It is helpful to let us know about how much speed and with how much speed we are accelerating, like, in a linear fashion.

So that is the velocity storage mechanism. Then what could be the adverse effect of an elevated creatinine on vestibular function? This is, again, out of context. Like, I don't have any experience in this. I haven't done study specifically in patients with chronic kidney disease or patients with elevated, like, creatinine.

Yeah. Has had experience in neurocystic sarcoidosis. Neurocystic sarcoidosis is a CN's infection. It is because of a parasite. And it can affect any part of the brain.

It is. It may not affect only the cerebellum or in the brain stem. It can affect the cerebrum. And usually it manifests with a seizure. It's.

It manifests with a seizure. It manifests. It can manifest with headaches. So if the neurocystic sarcoidosis is strategically located in one of the centers, like. Like central vestibular pathways or in the cerebellum then it can lead to a specific variety of nystagmus.

If it is located in the midbrain, it might lead to abating nystagmus. If it is located, you know, in the cerebellum, it can cause down beating, it can cause in the. If it is causing the affection of the NPH, it can lead to, uh, gaze, evoke nystagmus. So the location of neurocystic sarcoidosis will decide the type of the nystagmus. And if it is located in the insular region, that also can, you know, it is a vestibular cortex, it can lead to, uh, seizure and it can lead to a stagnant in that area.

So it depends on where it is located. So I think I have answered, like most of the questions which are present here, and I thank all the attendees for having patience to stay till the end of the webinar. And I hope you got some concepts out of this webinar, and I'm trying to keep it simple and like, you know, in subsequent session, we will discuss about the positional testing and we will also have the practical session. I hand it over to Anna. Sure.

Just a moment, I need to set up my feed editor, Pavar. Something is changing.

Oh my God, what's happening?

Okay, now, that's fine. So, so many questions that are provided. Webinars. I think the webinar was really well received, but now we have arrived at the conclusion. And so thank you very much, Doctor Pavar, for your presentation.

Even this time, it was a fantastic learning opportunity. So we look forward to reconvening with Doctor Vishal Pavara on April 3 for the final online lecture, or this

fantastic series. The session will cover positional testing for the treatment of common pathologies. We encourage you to stay tuned to invent social media channels and registers 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 once again, Doctor Pawar, for your participation and see you next April 3 for the final online lecture of this fantastic series. Bye bye. Thanks for nowhere.