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Fundamentals of Video Head Impulse Test – Lesson 3: Clinical Applications and Case Studies

Presenter: Vishal Pawar, MD

Dear all, welcome back to Inventis Academia. Today we are pleased to host the final session of our three part series dedicated to the fundamentals of the Video Head Impulse Test. And let me say that it is a real pleasure to once again welcome Dr. Vishal Pawar, Specialist neurologist and Fellow of the European Board of Neurology whose clinical expertise and deep commitment to vestibular diagnostics have guided us through this journey. The title of today's session is for Fundamentals of Video heading pulse tests.

Lesson 3 Clinical applications and Case Studies. This session will focus on case based learning helping you connect the hit findings with real world diagnostic and treatment decisions. Thank you so much Dr. Pawar for being with us once again and as always thank you. Feel free to drop your questions in the question and answer box and don't forget to complete the post event survey.

Remember that your feedback is truly valuable for us. And now enjoy the webinar and see you later. Okay Good evening everyone from Dubai. I welcome you all for this final webinar on Video Head Impulse test. Thank you Anna for that kind introduction.

And so like in last two webinars we have discussed the basic concepts behind doing video and impulse test. This will be focused fully on clinical like how to, you know, report. The two main aspects of this webinar are One is V it's reporting how to report which are the parameters and what are the clinical applications like cases. We will discuss many cases in that so if we look at the VHITS reporting parameters, there are eight different parameters we should look at. It starts with velocity of the impulses, then gain of all the canals, then the corrective cuids I.e.

covert CS over sacates, horizontal dispersion of the latency. I will explain that. Then vertical dispersion of gain anti compensatory quick eye movements, whether they are present or not and whether there are any artifacts or other parameters which are there. So let us dive into this. First is high velocity head impulses.

So we know that we test horizontal canals and the vertical canals. So for horizontal canals the head velocity should be more than or equal to 150 degrees Celsius minimum. And it can go up to, you know, more than 220 degrees per second. It's always better to have, you know, different higher velocities because we can see drop in the gain at higher velocities in some of the partial vestibular deficits. Then vertical canals minimum, it should be more than 100 degrees per second and it you can go up to more than 150 degrees per second.

So if we look at this graph, these, these are the. We test the six canals and you can see so for vertical canals, minimum we should, you know, be doing. You can see the 100 degree here. So you can go more than 100 degrees. Okay.

And forward lateral canals like start from 150 degrees. You can go higher up. Okay.

So mention the head impulse velocity range used during the testing. Then why this is important? We have first time just one slide. I will show by Professor Enrico, who is, you know, my mentor. I have learned a lot of insights regarding video head impulse test in different conferences from him.

So we have learned in previous webinars that during the gauge stabilization only vestibular control is present at higher velocities. At lower velocities, visual and proprioceptive. And in medium range we have visual supplementation of the V. But at higher velocities, like ranging from 1 hertz to you know, like around 7 hertz or more, the vestibular control is there which is, which corresponds to the natural head movements. And we are, you can test mainly with higher frequencies.

And the, these values ensure that the stimulation is above the frequency range of visual pursuit system which typically functions up to 100 to 120 degrees per second. That's why we have to give high, higher degrees per second. And at these higher velocities visual tracking is ineffective to maintain the gauge

control. So the test isolates the vestibular contribution to the gauge stabilization.

So this is the first part of reporting. The first thing is number of valid impulses per canal. It automatically comes into the test report. For example, you can see the number of impulses delivered five. You need in, in the remote camera V8 system by synapses.

You just need five good impulses for, for this. So it is already there in the graph. But I'm just mentioning this. And peak head velocity reached. It should be minimum 150 for horizontal, more than 100 for degrees per second.

For vertical. All impulses exceeded minimum velocity threshold. Like obviously the equipment takes. Once you set this criteria, the equipment will take only the impulses which are of higher degrees. So make sure that you do this adjustment in the equipment so that only high, higher end impulses are, you know, are taken, you know, into consideration.

So now that was the first part. First step was high velocity impulses. Second is the gain of all the canals. So we know that we are testing six canals like right anterior, right lateral, right posterior, left anterior, left lateral and left posterior. So six canals are tested during the video Head impulse test.

And these are the canals evaluated. And we test the RALP and larp. So. So these are the six canals and bilateral horizontal canals. Now coming to the definition of gain we have seen in the last webinar.

Like I/V velocity that is our output divided by input is head velocity. We give the input in terms of head velocity and we are checking the output in terms of I velocity. So I velocity divided by the head velocity is nothing but the gain V gain how much we it should be. It should correspond to one normal gain is 0.8 to 1.1 in in case of horizontal 0.721 for vertical canals. Of course, this literature will differ from articles to article equipment to equipment.

So like for. Like in. In the equipment which I use, that is synapses, it is 0.8 for horizontal minimum and 0.7 for vertical canals. So you will get these values mean gain here of all the six canals. And what are the reporting parameters?

Let us see before that what are the advantages of remote camera V8 system is there is no headgear that it reduces the artifacts which are there from the goggles. Then binocular recording of horizontal canals is done. Because the pupillary detection happens during horizontal for both the eyes. Automatic canal detection it happens. There is no need of calibration.

You have to just take care of the distance from the camera and the eyes. If that distance is fixed like 90 cm, then automatic eye detection and pupillary detection will happen. And fewer impulses are needed. It is patient friendly. So only five impulses, good impulses are enough.

So the reporting parameters for the system, like reporting parameters for the video head impulse test are individual gain values per canal gain. A symmetry and morphology of the VOR stress one by one individual gain values. As I told you, get individual gain values here. Okay. Then asymmetry also is there.

And you have to look at the traces morphology of each traces. Whether the head and eye velocity graphs are corresponding like you know, they are uh. Overlapping with each other. If this graph is not there, you can, you know, in the reporting section you can make a table like this for horizontal gain on the right side, anterior and posterior on right side gain on the left side and normative range like you can see for the corresponding equipment. And accordingly it can be reported.

So there are six graphs which are see are there available. First graph is. This is a overview graph. That is canalogram. There are six canals, right side three canals and the left sided three canals.

And you can see the green means that it is in green zone. That is the gain is normal. Automatically the system will give a green color coding based on the gain value. So that is V it Overview graph. Second graph, which I really like is a velocity graph where you have the canalogram also and this table also along with the the velocity graphs.

Like I will the the head velocity which is given and whether the IVL eyes are corresponding to the particular that particular impulse. So you can see these six traces here, which looks amazing. Like if you see in this per this particular graph in a patient, then you like you know that the vestibular function vor is functioning well and it helps you in the diagnosis. Then position graph. Position graph means that the you are giving a high velocity head impulse.

Here the head has more. Okay. And even in normally we feel that when we give the sudden head impulse the eyes appear to be stationary. But actually they are making equal and opposite reaction equal and opposite to the head velocity. But here in the traces you can see that the eyes appeared stationary because of this quick reflex which is one of the fastest reflex in the body around with a latency of 7 milliseconds.

And that's why it appears that the eyes remain stationary despite of the change in the position of the the trace. Like the head head is mood. Then next is sim single impulse velocity graph. Like you can all these five impulses per per canal which we are delivering each impulse. You can go into the detail of each impulse and you can see the trace and you can even delete some of the traces if you feel they are noisy.

If you. You find that they are, you know, artifacts, you check, go back and check if it is really like, you know, artifact or artifactual. If you're suspecting another graph, the most important another graph which I really like is V heat gain velocity graph. This graph I really like because it plots the gain versus the

velocity head velocity. Okay, so in this graph you can see this, this.

This is the velocity in degrees per second. Here is the 100 degree. And here it is 150. So all the impulses delivered are more than 100 degrees per second for LARP and RALP plane. And for horizontal you have.

You can like keep the cutoff for 150 and you can go higher up. It's an amazing graph which gives clarity regarding whether there is a vertical dispersion which I am going to come to that later. And you can also see the V8 pictures like whether or at the end of that particular impulse, whether the eyes were in the center. And like you can see if there is VOR deficit You can see that it is not exactly in the same place. And there can be.

You can see if the patient has over second. You can see in the photograph like it will be lagging. It will be static picture. You can see and even video. Also.

You can go back and check the video of each particular impulse. You can go see the video. Now. That was the second part of the reporting. Then comes the third part.

That is covert circuits. In the corrective circuit, the first thing is covert circuit. So let us understand what are covert circuit. It occur during the head impulse. Like now this head impulse has begun from here and it ends until here.

So the. The saccades which are happening during the head impulse. They are called as covert circuits. They are invisible to naked eye. Because we can see anything which is happening afterwards from here.

When the head impulse ends here, then you can visually see if there is any over second. But this is.

This one is a covert saccade. It compensates for VOR deficits to stabilize the gaze. For example, the head velocity is this curve.

And the eyes are lagging behind. Then the brain realizes the the frontal eye field area realizes the gaze centers. Realize that it is lagging behind. And it makes a correction before the even the head impulse ends. And then until the head impulse ends, the eyes are bang on the target.

You will not be able to see this correction by the frontal eye field, gaze center, saccadic centers. So it is a kind of reflexive saccade which happens at high velocity. And it tries to correct the gaze, the VOR deficit. It is called as covert circuit. So here also you can see there is a lag.

And then there is a covert saccade. Until the head comes to the rest, the eyes are bang on the target. So clinically you will not be able to see this because it will not be perceived by the eyes. Diagnostic value is it is a marker of vestibular hypofunction. Even with normal gain.

So the one of the most important thing to realize is presence of overt or covert saccade is pathological even despite of normal gain. That is very important. Earlier there was more emphasis on the gain. But it is very important to know that the corrections can apparent gain. There is like the gain is normal because of the corrections.

So it is very important to see look at the like persistent overt or covert sac. If they are there, it is pathological despite normal gain. And so as I told improve the detection over the in clinical head impulse. You can't see this kind of covert saccade. It helps to localize the peripheral lesions.

And basically when you start the head impulse after the 70 milliseconds until the head ends. The. The. The head impulse ends. You can see the latency is more than 70 milliseconds.

As these are saccades. The latency. There is a latency more than the vestibulo ocular reflex and the amplitude. There is a criteria of amplitude and velocity for the. These circuits because they are.

They should. They should be of higher amplitude. That is more than 100 degrees per second or more than half the head velocity. More than half the head velocity. So here you can see it is began at zero and we have reached a head velocity of almost 160.

Suppose and this is time. So 50 milliseconds. 100 milliseconds. 150 milliseconds. So you have to see like this particular covert saccade.

It began here and it reached peak velocity here. So from here to here, in how much degrees it covered versus in how much time it covered that distance. That is the velocity like it should be more than 100 degrees per second or more than half the head velocity. And the clinical it should be consistent. That is very important.

It should be recurrent across impulses. And the direction should be same as the vor. Like the vestibulo ocular reflex. The head is moving and the eyes are moving. So whenever the correction like head is moving to the left, the eyes are moving to the right.

So that is the COVID circuit will be in the same direction of the vor. Vor is happening in the exactly opposite direction of the head. So whenever a saccade happens in the same direction of this vor, then it is a covert circuit. But there are anti compensatory circuits. Also because of the delayed deceleration of the vor.

Sometimes there can be circuits in the opposite direction that I will. I will cover later. Now clinical relevance of the the COVID saccades is like covert circuits with normal. Even normal gain can still be

pathological. And it is a key for identifying compensated vestibular loss.

Compensated vestibular loss can be detected by. With the use of COVD circuits. Now coming to the overt sacred. Overt means they are overt like you know, they are seen visible. So after the head impulse ends the.

If we are able to see up to 400 milliseconds. If you are able to see a corrective sacred, then that is a over sacade. These are the corrective eye movements which occur after the head impulse. They are visible in clinical exam and detected in video head impulse test. They indicate V failure to maintain gauge stability.

Diagnostic significance is they signal Vestibular hypofunction, especially with low gain. But sometimes you do. You can see it with normal gain common in acute vestibular disorder that is neuritis and repeatable. Over sacades are definitely pathological. Even if the gain is normal or borderline.

And the reporting criteria is latencies. As I told after the head impulse ends post impulse, this particular over second will happen. And the velocity is more than 100 degrees per second or more than half the velocity of head speed. The amplitude is is large enough to restore fixation. The eyes could not match the head velocity and they are lagging behind.

And that is corrected by this over sacade Consistency is they are seen in most impulses to the affected side. If you can see most of the impulses are creating this over sacade of variable latency. Their latency is variable in different different impulses. That indicates unstable vestibular lesion. It is unstable vestibular deficit.

So that I'm going to come back to that point later. Now here the interpretation tips of over sacade is over sacade with reduced gain indicates a clear vestibular deficit. And it may appear along with the

COVID Both can happen together. For example, the eyes are lagging behind. There can be covert saccade along with the overt saccade.

Both can be there in same patient and reduced gain. If you see but without any corrective saccade suspect artifacts, okay, suspect artifact or some or in patients with bilateral vestibular loss there cannot be corrective saccade. So in patients with bilateral loss it can be significant. So you have to, you know, analyze findings case to case basis and you have to compare them. You have to look at the morphology.

You have to look at the other side. So this is the, you know, this is how you can report it. You report whether the COVID CITs are present or not. Over CS are present or not. If both are there, you can report both are present.

If none of them are there, it is normal. And latencies also covert second latency and over second is O over is more than 200 millisecond and amplitudes. You check amplitude by velocity because that is a criteria whether it is really a pathological and or versus artifact. So you can differentiate based on the this particular criteria. Now, the fifth reporting parameter I was discussing was horizontal dispersion of the saccades.

Now so suppose a patient has corrective saccade. Then you have to check whether there is a variability in gain values across impulses. And in healthy there is a tight clustering. But if it is widespread, then indicates unstable or partial VOR compensation. It is often linked to inconsistent saccade.

For example, in these diagrams you can see if you are doing a head impulse and all the there is a latency and then there is you. You can see this over second. And all these over second are happening at exactly same latency. The latency is same. And on a graph, okay, superimposed graph, they will be look as gathered seconds.

They will look as gathered, which is despite of the deficit. It indicates it is a stable deficit. And good compensation is happening. But if you see that it is disorganized, the latencies are different as in every impulse that the latency is different, then it indicates a disorganized response unstable vestibular ocular deficit. And in superimposed graph you can see there are over seconds of variable latencies.

And it is a. A kind of. You can see say unstable lesion. And there is one score called as prediction and refixation score like PR score and which indicates temporal scatter of this diagram. And you can see there is a nor.

This is a normal pattern where the eye velocity and head velocities match in the superimposed graph. And normally the PR score is zero. But when these overt or covert circuits are present, you have to check whether they are gathered or they are scattered. If they are gathered, it indicates compensation. If it is scattered, it indicates uncompensated vestibulopathy.

Then the most important thing is gain decline. With high increasing head velocity. As you keep go on increasing the head velocities, then slight decline in gain is normal. But if there is marked the decline it goes below be below the threshold. Like for example lesser than 0.8 in case of horizontal or lesser than 0.7 in case of vertical.

Then it can be pathological. It can indicate, you know, partial vestibular deficit. So it's very important to expose the patient to high velocity head impulses. That is more than 220 for horizontal and more than 150 for you know, vertical. And it suggests canal hypo function.

Especially with corrective circuits. If the corrective circuits are very important as compared to just gain value. Now coming to the clinical insight. Analyze both gain and dispersion. Both the dispersion like latency dispersion means the PR value or whether they are scattered or or gathered.

It is important. Even moderate gain with high dispersion indicates possible dysfunction. So how to report this contin? This con. This particular parameter is you check whether the dispersion is low or high.

If the corrective cycles are present and if the instrument. If a PR score is available then you can mention whether the PR score low or high. The low score indicates gathered and high score indicates a scattered correct corrective saccades. Then comes the sixth parameter is vertical dispersion. Vertical dispersion.

I will. I will tell about vertical dispersion. You can see here that I gave impulses to this particular patient of in the range of around 130 degrees per second and in the LARP plane. And you can see the blue dots here. Okay.

The blue dots that is left anterior indicating the left anterior and at the same around the same head velocity. You can see there are different gain values in the. In the left anterior. Okay. And you can also see there is a horizontal dispersion also of the latency.

And you can see the vertical dispersion of the this gain versus the velocity graph. And this vertical dispersion is happening because of this particular unstable vestibular deficit. So what is this vertical dispersion? There is a variability in eye velocity across the impulses. It is seen as a spread in superimposed stresses and it indicate inconsistent VOR response despite uniform stimulus.

Suppose you are consistently giving 130 suppose degree per second impulse. There is difference in the gain values because of this unstable vestibular deficits. And it. It is because of central compensation instability. Because if the patient doesn't receive vestibular rehab the patients will have inconsistent the central compensation and it will be unstable and it will lead to dispersion horizontal and

vertical.

Then it. That's why the patient has variable overt or covert saccades in cons. And it can also happen as a part of artifact. Because if there are inconsistent head impulses that also can contribute to this and it reflects usually it reflects partial recovery or neurological involvement. So clinical relevance is healthy.

There is a tight clustering, but pathological there is a wide clustering, wide dispersion. Sorry. And it suggests unstable VOR or sometimes test artifacts and combine the gain with saccade matrix for accurate interpretation. So it's always important to interpret the gain values in light of the morphology of the corrective saccades. Now reporting tips are not present.

Symmetry and relationship to gain high dispersion with low gain is likely dysfunctional. Recommend repeat testing or monitor during compensation in a certain canal. You can go for more impulses to, you know, just to check whether they are, you know, consistent. Consistency of these particular corrective saccades is very important. And in summary the vertical dispersion indicates trace variability.

It is relevant in identifying hidden vestibular deficits. It enhances assessment of test quality and clinical accuracy. Now in the same patient, if you look at like I this patient underwent just four sessions of vestibular rehab. And you can see I repeated the test after four sessions of vestibular rehab. Look at the left anterior canal there.

There were. There was horizontal dispersion. You can see the. The. The saccades are becoming gathered even in case of the left lateral canal.

You can see the. They were earlier scattered saccades but they are gathered over saccades in this particular patient. And you can see that there was vertical dispersion in the gain gain versus the velocity

graph. But there, there is now clustering which is indicating recovery in the gain and the. The impulses are no more in producing inconsistent variable latencies there and gain pattern.

That indicates that that is the importance of vestibular rehab and that is the importance of the video head impulse test in monitoring the patient over time.

Then comes the anti compensatory eye movements. For example, what happens is when you give a head impulse some some page sometimes it can happen that there is a late deceleration of the delayed deceleration of the I'm eyes vor. And so you are giving the head impulse suppose to the left side and the eyes are supposed to go towards the right and they overshoot and then there can be a correction and that will be in the direction of the head impulse. Okay, so let us go into details of this. These are the quick eye movements in the same direction of the head impulse opposite to the expected compensatory saccades.

And they are seen in vestibular migraine and Meniere's disease not typically in central region. And they help to differentiate peripheral versus central causes. And these anti compensatory eye movements are not artifacts or saccades due to hyper gain. The gain is not hyper because it the there is a delayed deceleration of the vor. The VOR like instead of decelerating faster it is decelerating slower.

And there is eye overshoot and there is a correction on the in the direction of the head velocity. So there are again two types of anti compensatory eye movements. There can be covert or overt anti compensatory eye movement. And these are my one, this is my patient where this is a case of vestibular migraine Meniere's disease spectrum disorder where you can see the anti compensatory eye movements which are happening covert and over type. And another very important place where you will get this anti compensatory eye movement is suppose A patient has acute unilateral vestibular.

For example, this patient has right anterior and right lateral loss, indicating right superior vestibular neuritis. On the other side, you can get this anticompany quick eye movements.

During the stage of compensation. The other side is trying to compensate for the loss. And there can be, you know, these anticompany eye movements then coming to the artifacts. In what are the artifacts? There can be false signals in eye and head velocity traces caused by technical issues, patient behavior, environmental interference.

It may mimic pathology or obscure real deficits. And that is a very important area where sometimes you are, you know, in doubt whether it is really like, you know, abnormality or artifact. So it's very important to identify them and do the corrective things. What are the common types of artifacts is blinks. It can lead to spikes and interruption.

Good thing is that the machine itself disregards these particular impulses. Corneal reflex loss can lead to dropout. Poor tracking. There can be noisy traces, fixation errors. The patient fixation error.

Or there is a goggle slippage that can also cause artifacts. How to recognize is flat Traces is equal to fixation loss. Spikes is equal to blink or drift out of phase motion is due to slippage, inconsistent gain, poor stimuli or frame drops. So you have to in such cases. Now you repeat do some more impulses in those patients and whether look at the consistency and improve the attention of the patient.

Instruct the patient minimize blinking and ensure that stable corneal tracking and good lighting is present. Avoid reflections or shadows. Perform more than five impulses. In such cases. If you clear get a clear traces, then you can stop at 5.

But if there is some doubt that this is abnormal, you can just check that and you know, you can go for more impulses to, you know, gain confidence and review. Review videos and traces for confirmation.

You can always go back to the video and check whether really it is an artifact and or whether the patient blinked that time or whether there was any lack of attention. So the red flags are sudden gain drop without saccades correct. Without corrective saccades eyes move faster than head.

High trace variability across impulses should give you suspicion and video dropout or reflex misalignment. These are the various clues. Now look at this patient. The patient at baseline has this particular misalignment. And I got this particular trace.

That is, he showed abnormality in both the sides. And actually the patient's balance is not so much impaired as compared to the deficit. So we have to understand that V which vestibular the Video head impulse test is a test of whole vestibulo ocular reflex pathway starting from the semicircular canals until the extraocular eye muscles. So if the end organ that is extraocular eye muscle is V week because of some reason, then it becomes unreliable. The in.

So you can see in this particular patient the it was a patient underwent eye surgery and post eye surgery he developed squint. In such patients the V heat interpretation becomes unreliable and you have to always correlate the this particular test clinically. And in such cases you have to rely on clinical history and alternate vestibular test. So it's very important that the baseline, you know eye movements are normal in those in such cases, in patients who are having skewed aviation or squint, it becomes unreliable. So I'll give you one algorithm how to, you know, analyze VHI traces.

First check for abnormal catch up circuits, whether they are cover or over. If they are absent, look for V gain. If the V gain is normal, so V gain is normal, there are no corrective saccades, then it is a normal study. If the V gain is reduced in the absence of catchup saccades, then it could be artifact. Or if it is bilateral, it could be related to bilateral loss.

If it is bilateral. If you see abnormal catch up saccades, either covert or overt saccade, if they are present only on one side, it is unilateral lesion despite irrespective of gain value. Okay, Usually the gain will be less. But even if the gain value is normal, it indicates partial vestibular deficits. If both sides you see covert or overt saccades, then look at the morphology of the traces.

Look at the shape. If the shape is abnormal on one side, it is unilateral lesion. For example, one trace I have shown where there was deficit on one side and there was anti compensatory eye movements on the other side. Here the morphology is abnormal only on one side. So it is a unilateral lesion.

But if the abnormality is present on both sides, it is showing the deficit in the vor then it is a bilateral lesion. So the most important thing is if there are repeatable corrective circuits despite of a normal gain, it is still pathological. So this is the reporting, you know your interpretation of the results. Either it is a normal video head impulse test or it is a unilateral vestibular hypofunction. Mention the side or it is a bilateral vestibular hypofunction.

Mention about whether the Compensation is happening, that is in terms of COVID sacred, whether you noticed any anti compensatory quick eye movements which indicates peripheral imbalance, whether the artifacts are present because of any reason, then you can write that interpret with caution. And the most important thing is clinical correlation. Any test it needs to be clinically correlated. Because see the beauty is vestibular tests gives findings, they don't give diagnosis. Ultimately we have to correlate the clinically.

Whatever deficits you are seeing. I have shown you one trace in one of my patient of squint. The VOR was abnormal, like the gain was reduced in all the canals. But the patient is not having any imbalance. So the most important thing is clinical correlation.

Okay, now let us dive into clinical applications into of the video head impulse test. I have divided this particular into three parts. Peripheral conditions, central conditions and other conditions. Okay, so peripheral. Let us dive into what are the peripheral conditions in which you have to, you know which are the most important.

I have given like more slides on them. Now coming to the acute unilateral vestibulopathy. As we know that the superior vestibular nerve supplies the utricular anterior canal and the horizontal canal. The inferior vestibular nerve takes the vestibular inputs from the saccule and the posterior canal. And together there are, there is a vestibular ganglion.

Superior and inferior vestibular ganglion is located in the Scarpa's ganglion. And if there is inflammation of either the ganglion or the superior nerve or the inferior nerve or the ampullary branches, it can lead to sudden vertigo, imbalance, nausea, there is no hearing loss and it most commonly affects the superior division. Okay, now let us see what are the, what is the role of video head impulse test? First is rapid bedside confirmation of the peripheral lesion. Now let us see if you encounter a patient with severe vertigo.

Okay. Acute vestibular syndrome, acute ongoing vertigo. In such case, if you perform the head video head impulse test in the emergency department and you see that the head impulse test is normal, the patient has spontaneous nystagmus either with or without fixation or even on the gaze testing. If you find nystagmus that is with fixation or without fixation and ongoing vertigo, patient has. And if you see there is a normal heading video head impulse test or head impulse test, then it, it indicates that the patient could be having central lesion.

And if you find this characteristic finding that is involving suppose right anterior and right lateral canal, that is right superior vestibular neuritis. So it is peripheral lesion so that is the importance of video head impulse test in emergency settings. There are many centers where video head impulse test is being

used and it can help to differentiate between central and peripheral. And there is a very nice study by Professor Newman Toer in 2013 where we have just discussed that in emergency setting in patients with acute vestibular syndrome they performed the video head impulse test. And whenever the video head impulse test was normal it indicated central cause and whenever it was abnormal it indicated a like peripheral cause.

But there, there were two cases there where they found that video. The video had impulse test test was abnormal but the patient had central. But of course video impulse test is not the only clue to the central. This is just, you know, one of the clue, the most important clue are clinical bedside signs that is direction changing, bi directional nystagmus and skew deviation. So which was present in both the patients.

So it's very important that like look at the other signs. It's not that nothing is absolute in all the, if you look at all the clinical signs, nothing is absolute. You have to always look at the friends that they keep. If the friends are indicating central, then it is central. So it's very important to look at any case with respect to the company that they keep.

Okay? So this is very important that hearing loss will be present. In an acute vestibular syndrome due to IA infarction versus vestibular neuritis, the hearing loss will be present. The central signs often are present like skew deviation, bi directional gaze, evoke nystagmus and V suppression test will be impaired. Now there are situations where you will get patients with episodic vertigo and you do the interact when they come to you and interactally you are checking the video head impulse test.

It will come normal and these circumstances are central. For example, transient ischemic attack, vestibular migraine, patients with Meniere's disease, interictally. In patients with BPPV canalolithiasis in between the attacks. If you check it might be it, it will be normal. But there are of course instances in, in these disorders where it will be abnormal.

I will come to one by one. So first use of video AD impulse test in acute unilateral vestibulopathy. We saw that in acute vestibular syndrome it helps to differentiate between central and peripheral. Suppose you come down to acute peripheral unilateral vestibulopathy and you do the video ad impulse test will help you to identify which canal is defective. So for example, in this particular patient of mine, no, not my case.

In this particular patient you can see the lateral canal gain is low on left side with there is a reduction gain with over saccades. In this particular trace you can see the right posterior gain is low with over saccades. In this particular trace which we have seen earlier you can see the right anterior and lateral gains are low with anti compensatory quick eye movements on the other side and you can see in on the left side. There is a total vestibular loss in this particular patient. So you can identify canal specific deficits depending on whether there is ampullary nerve involvement, superior vestibular nerve involvement or there is a ganglion involvement or there is a trunkal involvement of the vestibular nerve.

So there is a very nice study I like by Professor Kasani which is there in the video head impulse test book written by Professor Enrico. And in this particular study, 51 patients were identified with acute unilateral vestibulopathy. And all patients underwent video head impulse test which checks three canals canals. All patients underwent OVAM to check the utricular function. All patients underwent CWAMP for saccular function.

And they noticed that the most common deficit was lateral canal, second anterior canal, third was utricular deficit, fourth was posterior canal deficit and the last was saccular deficit and which corresponds to different different involvement of different branches. So in the same study they identified various patterns. If the patient has utricular loss along with anterior and lateral canal, it is superior vestibular neuritis that they saw in 22% patients. If the patient has posterior canal with saccular deficit, it is inferior vestibular loss, pure inferior vestibular loss. They did not find in their study total vestibular

loss.

They found in 32% patients partial superior, some patients having utricular and anterior canal, some patients having utricular or lateral canal, some patient having only lateral canal, only anterior canal. And in one patient they have seen isolated utricular deficit. So that was partial superior deficit in 22%. And there was another pattern they observed was partial superior and inferior canal deficit. It which was seen in 22-24% patients where there were some involvement of the superior and some involvement of the inferior.

Isolated saccular deficit alone they did not see in any single patient. Now the next important thing is detection of the compensatory strategies. Like for example compensatory gain loss on healthy side can be seen in patients with suppose the patient has VOR gain loss. There can be compensatory gain loss on the in the canal pairs. Okay.

For example this is my patient where the patient had symptoms of acute vestibular syndrome a year ago. And then he is now presenting to me with lot of vestibular visual symptoms. And he has, he feels imbalance, he feels visually induced dizziness. And that is due to uncompensated vestibular loss which happened. He did not undergo any vestibular rehab.

And that's why he's getting this uncompensated vestibulopathy. And this is another patient, you can see where the patient underwent vestibular rehabilitation. In this patient with acute unilateral vestibulopathy which is involving left superior and lateral canal. And after the sessions of video vestibular rehab, the patient had recovery in the gain. That is the importance of vestibular rehab in patients with acute unilateral vestibulopathy.

The fourth important parameter, the important function of video head impulse test, how it can help is it is essential for monitoring the recovery of the patient. Like for example, in this particular patient the

patient had scattered over saccades and when the patient underwent the physiotherapy vestibular physio, the patient later on started getting this gathered over saccades. So the video head impulse test will, I can say it, it will motivate you and your vestibular physiotherapist test, you know, to give, you know they are on right track and it will give you objective evidence that you are on right track.

Now coming to the next is Meniere's disease. What is the role of video head impulse test? These are the five divisions I have done. First is during the attack. So during the attack of Meniere's disease there is a very nice study by Professor Yakovino.

Know they like they saw one patient and they did video head impulse before the attack. They were lucky to do this particular video impulse test before the attack. And once the attack started, they analyzed the patient 10 minute after 20 minute after 30 minutes for 60 minutes and 90 minutes. And they saw that there was the patient had clear cut V deficits. And at the end of the attack you can see the gain returned back to the normal.

And during the same attack the patient's corrective saccades normalized. Okay, so the patient had transient vestibular deficit during the attack and which recovered. And so the video head impulse test allows individual canal assessment and giving the topographic diagnosis of the vestibular injury. And this is, this is normally you will commonly see in these patients that between the attacks the patient will be normal and during the attack the patient will have a deficit. This is something similar to the audiogram, pure tone audiogram you do in a patient with Meniere's disease.

You will find there is a reversible like, you know, low frequency hearing loss which is characteristic. And you can see the similar thing happening in the well like video head impulse test, repeated attacks. There is another study by Professor Yakovino in which they studied the patient sequentially in different attacks. For example, three attacks were studied. This is attack number one which lasted for two hours.

This is attack number two. This is attack number three at different point of time. And they studied the gain on the same side in different attacks the gain went down, it came back okay. And then again during the, during the. It got normalized again during the attack it went down.

Then again it came back to normal again it went down, came back to normal. And with repeated attacks. If the patient doesn't receive vestibular rehab after the attack, if there is any residual deficit, if the patient doesn't move their head, doesn't do rehab or doesn't participate in activities, then what will happen? They will develop deficits over a period of time. And the patient next is after repeated attacks in late stages the patient can have uncompensated vestibular deficits especially in the posterior canals.

And that will. That will need definitely vestibular rehabilitation lab for recovery. In the same patient you can the like in. In one of my case I the. The patient had deficit in right and left anterior and left lateral.

Just four sessions of vestibular rehab. The saccades which were scattered became gathered here the saccades were predominant. The gain normalized. They the patient started getting developing gathered saccades. So that is the importance of rehab and that is the importance of vestibular, you know, video head impulse test where it will give you objective evidence of whether you are on right track.

So this. There are instances where you will see dissociation between video head impulse test and caloric test.

So basically video head impulse test and caloric test assess different frequency spectrum that we have, you know, discussed in detail in my previous webinar. Where video head impulse test assesses high frequency and caloric test assesses very low frequency. And the dissociation occurs when one test is normal and other test is abnormal. And the dissociation reflects frequency specific vestibular dysfunction. For example, in vestibular neuritis you commonly it affects the type 1 hair cells which which respond to high velocity which is detected by video impulse test.

And menier disease affects the type 2 hair cells which is predominantly detected by the calorics. So both tests are complementary. They are not replacement to each other. The clinical relevance in diagnosis is it helps in distinguish menius disease from vestibular neuritis. It identifies high velocity deficits missed by calorics and it impacts clinical decision making.

And that's why these two tests are complementary to each other. And they are not. Neither alone is sufficient. I will show you some instances. For example, if you get abnormal V heat and normal calorics this can happen because if the patient has selective vertical canal loss then you will definitely calorics basically assess low frequency lateral canals like horizontal canals in low frequency.

So in selective vertical canal loss weight will be abnormal. Selective high frequency loss weight will be abnormal. Central compensation you will be able to see very clearly in video and we had impulse test test early or partial vestibular loss. You will see in video ed impulse test. In those cases caloric will be normal.

And there are instances where caloric is abnormal and the video ed impulse the test is normal. For example, in early menier disease compensated neuritis where the high frequency loss recovers first and then low frequency loss recover and aging also pris bilopathy where you can see caloric abnormality and not not video ad impulse test abnormality. Then one of the very important you know role of video ed impulse test in menius disease is to confirm the if suppose the chemical chemical labyrinthectomy with gentamicin is done confirms the successful vestibular ablation in treatment resistant menius disease. It monitors the extent of canal ablation and it can help to track the compensation via gain recovery and or psychiatric reduction. Integration with other vestibular test is always important.

It is a complementary test to calorics and audiometry. Together they provide comprehensive review of the disease staging and prognosis. Okay, now let us move on to the third very important indication of video head impulse test is benign paroxysmal positional vertigo. Where we know that there there there is dislodged otoconia which go commonly in the posterior canal and they can go second commonly into the, you know, geotropic lateral canal or apogropic lateral canal. Or sometimes it can there can be anterior canal bppv.

In those cases it is the displaced otoconia. Doesn't affect the high velocity vestibular ocular reflex reflex in patients with canal liasis especially. That's why V it will be normal in typical cases of, you know, canalolithiasis where it doesn't affect the fast high velocity you know, vestibul ocular reflex. But there are certain instances where it can help in video impulse test can be positive, it can be pathological. And there are certain cases where canal dysfunction in recurrent and unresolved cases, especially post traumatic parasis like trauma patients can have canal parasis also and they can have secondary bppv.

Also patients who have neuritis they can develop for example superior vestibular neuritis affects anterior and lateral canal and utricular. Because of utricular dysfunction. It can dislodge otoconia which can go to the posterior canal which is a normal functioning utricle. And it can lead to Lindsay Hemanway syndrome. And that can be identified by you know, video impulse test useful in differentiating complex PPPV variants like APO geotropic posterior canal BPPV versus anterior canal BPPV versus canal jam.

Especially in canal jam when the autolith burden is high, it can cause canal parasis transient canal parasis which after liberatory maneuver it. You can check the the video at impulse test it will come back to normal. So evaluation post treatment also is possible in BPPV patients. It confirms the canal function integrity. It detects the canal jam temporary reduction of in VOR gain after before the maneuver which normalizes after the liberatory maneuver and it supports reversible cupido endolymph dysfunction.

Okay, so that is one of the you know advantage of video ED impulse test in bppv. Then fourth very important you know aspect of video impulse test is in patients where you are to differentiate between central positional vertigo versus peripheral positional vertigo. You can use video head impulse test test and it can be of use in these cases. Like patient suppose has persistent positional down beating nystagmus. Okay.

In those patients if you see abnormal video head impulse test involving the anterior canal it is then it is anterior canal BPPV. If it is involving the apogeotropic. If it is involving the posterior canal, it could be a posture tropical posterior canal bppv. And if there is a normal video head impulse test with positional downbeat nystagmus, you should suspect a central central cause that is a cerebellar lesion. Then the fifth very important role is it is it is gives complementary information to the positional testing.

Of course, videoed impulse test is not something you which is a preferred thing to be done first in a patient with BPPV big it will give complimentary information and to the positional testing. Positional testing is the priority test the canals which canal is involved, which side is involved. Correct that. And if if there is any issue, you can always go to the video head impulse test it which can help in ambiguous cases.

Then coming to the bilateral vestibular hypo function. This is a barony society criteria for bilateral vestibulopathy in which video head impulse test is one of the, you know, it is one of the criteria. The bilateral horizontal angular VOR gain is less than 0.6 measured by the video impulse test. Then it is one of the like, you know, it is one of the supportive criteria for the diagnosis, one of the important diagnostic criteria for bilateral lateral vestibulopathy. And in this particular patient, you can see that this particular patient of mine, he had, you know, reduced like gain in all the canals and bilaterally reduced VOR gain across all the canals.

Lack of asymmetry and gain reduction correlates with head velocity increase. As you can see, as you are increasing the head velocity, the gains are dropping. Okay, so this is the importance of this gain and velocity graph which I really like. And it detects partial versus complete, you know, vestibular loss, rehabilitation planning. So that is the importance in bilateral vestibular hyperfunction increase by vestibulopathy.

Also, if it is included in the diagnostic criteria, if the VOR gain measured by the video at impulse test is between 0.6 to 0.8 bilaterally, then it is one of the, you know, very important diagnostic criteria for these patients who are suffering from chronic imbalance. Many patients undergo MRI for cervical spine nerve studies, for the, you know, nerve conduction studies. But it's very important to check the video and impulse test in patients who are with chronic imbalance in elderly to diagnose bilateral vestibulopathy and bilateral vestibulopathy and offer vestibular rehab to these patients and which is very crucial in improving, improving the balance. The other are, I will cover them later. Then I will go to some of the central causes.

So if you look at this particular diagram. Vestibular nuclei. Yeah. See, we, we generally think that like, you know, the vestibulo, the V impulse test is analyzing only the semicircular canals, but the vestibulo, it is actually analyzing the vestibulo ocular reflex, which starts from the semicircular canal. It goes in through the vestibular nerve in the vestibular nucleus, from the vestibular nucleus through, through the medial longitudinal fasciculus.

You know, it goes to the ocular motor nuclei, it goes to the extraocular muscles. So anywhere there is a deficit, it will show up into the, into the video ad impulse test. It is so it is not isolated test of only semicircular canals. So if you look at the vestibular nucleus. I'm sorry, I'm just little overshooting because these are some of the very important things I it.

There are only I think five or six slides. I will finish. Vestibular nucleus is first central relay station for the inputs from the semicircular canals. Ocular motor nuclei is the second medial longitudinal fasciculus cerebellar control, cortical control. So all these lesions can affect the vestibular ocular reflex pathway and it can lead to the.

You know, there are certain deficits which are seen. For example, if a patient has spontane stroke or lateral medullary stroke, the patient can have impaired vestibulo ocular reflex. Similar to clinical head impulse test is positive in some of the central cases. In those cases video impulse test also will be abnormal in medial longitudinal fasciculus deficit that is Internuclear ophthalmoplasia. You can see selective impairment of contralateral posterior canal in patients who have cerebellar degeneration.

Patients can have disconjugate horizontal vestibulo ocular reflex and patient can have disorganized saccades. And these are the various, these are the various deficit disorders in which you can see gain abnormality. The higher gains which, which you can see in different problems like Parkinson's disease is some cases of vestibular migraine. You can see there is mildly increased viewer gain. Sometimes it can be due to many a times it can be due to artifact that is increased viewer gain as target distance decreases.

If the target distance decreases there can be hyper gain. So you have to be very careful mindful of the distance from the target and the eyes. Goggle slippage artifact also can cause lead to this particular hyper gain. And one of the very common abnormality which I'm finding these days is in many patients is bilateral lateral canal loss. Which is a common finding in patients with thiamine deficiency.

Even before they get MRI abnormality. Even before they get bi directional spontaneous gaze your nystagmus. Even before they get a, you know like uh, MRI abnormalities. These. So it's very important to identify these cases and you know, treat them with hyphen supplementation.

And it is rewarding actually because the canal function comes back to normal. This is one of the. My recent case of Canva where I found that the, the, the gain are less in all the. All the canals, all the six canals. And you can see that this.

These patients present with severe cerebellar ataxia. They have proprioceptive loss, they have vestibular loss. So the, the proportionately they definitely they get this ataxia which is you know more. And you can identify this by doing the video head impulse test. Then other central signs in the V hits and localization.

Uh, usually the video head impulse test is normal. So I don't want to, you know, scare like all of you. That video at impulse test is primarily it is assessing the peripheral function. But I would like to emphasize that the V pathway doesn't is not restricted to the periphery. It does go into the center in the vestibular nucleus in the media longitudinal fasciculus into the ocular motor nuclei in and it is supplying through the ocular motor nerves to the extraocular muscles.

So anywhere there is a deficit it will reflect into the VOR and it will reflect into video impulse test. But by and large the video impulse test is normal if a patient is suffering from severe acute vestibular syndrome. And if you find there is a normal videoed impulse test it indicates central cause. But if you see bilateral low gain without any cause then you should suspect Vernix as I told that is thiamine deficiency. Brain stem lesions and disorganized or scattered cycles can be seen in cerebellar lesions.

Okay. And there are host of other conditions in which you can see abnormalities like ototoxic vestibulopathy. You can see bilateral high frequency canal loss. Post cochlear implantation. You can see if there is any iatrogenic damage.

Vestibular sonoma before and after surgery you can, you know, check for canal hypofunction canal dance you it is normal. The video impulse test is normal in these circumstances. But if you want to rule

out canal paresis and you are guiding to your right diagnosis, it will help you in guiding your right diagnosis. Post traumatic vestibulopathy. I have explained pediatric vestibular disorder especially this remote camera V heat system is you know, there is no need of goggles and you can do it is non invasive tolerable test in patient, in pediatric patients also.

So these are the various, you know like indications where you can use and there are some signature abnormalities. This is my second last slide. Some signature abnormalities. That is low VOR gain indicates reduced semicircular canal function in neuritis. Bilateral vestibulopathy Vestibular neuronitis disease Unilateral Logan affects one side Bilateral Logan bilateral vestibulopathy Canvas ketchup saccades indicates you know compensation disconjugate VOR that indicates central causes high VOR gain.

Check for any, you know artifacts but it can indicate vestibular migraine or Parkinson's disease. Normal video head impulse test with abnormal calorics in Menier's disease. Posterior canal selective loss can be seen in internuclear ophthalmoplegia or or canal exam. Increase saccade frequency without gain loss. You have to check for subclinical vestibular deficits.

So this is my take home message. Video Impulse test is very important in diagnosis of vestibular disorders which affect the V pathway. It helps in monitoring the disease progression. It helps in post surgical assessment in shonomas. It helps in vestibular rehabilitation planning.

If a patient has you know particular canal deficit you can focus on that canal particular specific exercises. This pediatric evaluation it is important elderly patients to decide their fall risk pre and post cochlear implant assessment evaluation research and validation tool. It can be used as a research and validation tool. It can help in screening of various neurological disorders. So these are the various, you know benefits of video impulse test.

These are my references for this particular lecture. I have gone through this beautiful wonderful book by Professor Enrico. I recommend strongly everyone to you know go through this book and there you can gain a lot of insights through this book. And I downloaded like hun there are more than thousand one, 1,111 articles from PubMed and I am really thankful to invent this team. I I because for these this webinar series I have gone through these articles.

I gone through the diagnostic criterias on the International Journal of Vestibular Research and I would specially specifically like to thank Anna for a nice coordination. I would like to thank Mario for his you know, technical support. I would like to thank Ganesh from local team who really helped me and I really thank all of you for patient listening and attending all this webinar series. And this is my email. If there are any questions this is my WhatsApp number and with that I would like to you know end this series of our video at impulse test webinars.

And if there are any questions I'm you know if you you can put in the chat box I will answer them one by one.

So I will yes first of all thank you so much for another outstanding presentation. I would say full of clarity, depth and the practical value. In my opinion your contribution to these three part series has been truly enriching I would say and we are grateful for your dedication and expertise. And I can see 11 questions. Okay so may you be so kind to Check the question box.

Yes, and provide your feedback.

So Anna, can you hear me? Sure I can, Dr. Pawar. Yeah, but unfortunately my screen has freezed. But because of some reason I'm not able to.

If you want I can. I can read the question for you. Okay. Okay, first of all from Sarah.

How is the system calibrated to the pupils? Sir? Yes, so different systems have different, you know, methods of calibration. But a particularly remote camera, video head impulse system by synapses. What happens is.

Yeah, now it is. Okay, so in this particular system, how is the calibration? Uh, is the distance between the camera and the pupil is fixed, that is 90 centimeters. And the camera automatically, you know, detects the pupil. And once the pupils are detected and the system gets calibrated automatically.

Okay, so that's the advantage of remote camera system where you don't need like, you know, once the pupils are detected through this corneal reflex, it helps to detect the pupils in with respect to the head, you know, the position of pupils with respect to the head.

Next question is I think. Okay, so now can you see the questions? Okay, yeah, fantastic. But the question is I think typed in maybe Russian, which I can't read, but I will go for the next maybe Greece, I think. Yeah, so there.

I'm really sorry I'm not able to read that. But if I will request Ms. Elena to, you know, type this question in English for me. Then there is another question. How can I watch the previous webinars?

Yes, it will be available on Audiology on the. Yes, Dr. Pawar. Correct. So the previous webinar performed by Dr.

From Dr. Pawar are all available. Okay. On audiology online. The three first webinar on DNG are also available on the Invention Inventis website.

And the latest webinar on VHIT will be available on the Inventis website soon. Okay, so please check it out. Okay, great. So next question. Next question is does the software calculate the PR score or do you calculate it separately?

No, the. Currently the software is not calculating, but definitely in the today only I had discussion. So in the current next update it is going to calculate the PR score. Sorry. So PR score will be introducing the next version of the VHIT software schedule for March more or less 2026.

So next year. Thank you. Yeah, exactly. Then there is another question. Congratulations.

And do you think think each investigator should have their normal values for gain? Yes, that's very important because it is, you know, it depends on which equipment you are using. Equipment to equipment it might differ. And for PR score To be honest, like the currently it's not available so I. I won't be able to answer that question regarding the PR score more then another anonymous question.

Thank you for the presentation. May I ask what does it mean out of phase motion? Yes. So if it is happening out of phase means if there is like you know slippage of the goggle or when you are moving the head and there is a slippage artifact and then it will, it will lead to the out of phase is motion and it can cause artifacts in the upper traces. So then another question is what is the normal vor gain in children and adults?

I don't have much experience with children but adults vor gain which I use is. The values I use is more than 0.8 for horizontal canals and more than 0.7 for vertical canals. Canals and that is up to 1.1 for horizontal and up to 1 for vertical canals. Then like there is another question. How do you differentiate between AQEM I.e.

anti compensatory eye movements and nystagmus in acute unilateral vestibulopathy? See what happens is uh, when you are doing uh like video impulse test in a patient who is suffering from. From you know acute unilateral vestibulopathy on the other side you can get either the anti compensatory eye movements or the nystagmus. And the question is really like very important that is how to differentiate between them. So you have to check then again the corrective saccades will have its own you know the like the nystagmus will be present at regular intervals but anti compensatory eye movements will be happening as a peak together and it will be present you know at the after the end of either it can present as a covert anti compensatory or overt anti compensatory quick eye movements.

And that is due to delayed deceleration of the V. And you have, you can differentiate from the nystagmus based on the regularity. Like you know if it is in staggus it will, you know it will be present even uh, it will create noise during the traces and it you can identify based on you know, repeat repeated. Like noise will come in those cases. But aqm you will see they are present clustered.

It will be present in cluster. So what are the uh, common causes of thiamine deficiency? See common causes is the very common causes is obesity, surgery, alcoholics. These are the two very common causes of and hyperemesis gravidarum. These are the three common places where you will find thiamine deficiency.

But even in otherwise patients, also patients can have nutritional deficiency. And it is very important to keep this differential in mind. And video ed impulse test is very important in patients who are suffering from chronic imbalance or episodic imbalance. You can, you know, if you do video impulse test and you check the lateral canal deficits are present in those cases you can go for thiamine test. But what I have observed is serum thiamine level is less sensitive to detect the, the, the thiamine actual thiamine content of the body.

There are two other test. One is thiamine pyrophosphate level. And there is another very important sensitive test which is erythrocyte trans KE activity. But unfortunately see these test might, may not be available in the lab. And you have to, you know, if you do specifically these test, uh, then you can find the thiamine deficiency and you can diagnose this bilateral horizontal canal loss.

And then another comment from professor Yona Voda from Romania. A beautiful presentation. Thank you Ona. And I'm really, I really thank you for you know, giving me a lot of support and uh, I have taken some insights from you. Uh, then, uh, another question from uh, Anup Singh is how many patients with vestibular neuroid is get their neural functions restored to normal?

See, it all depends on how early rehab is started. Like in patients with vestibular neuritis, how early rehab is started that decides. And as early as you know, within four weeks the rehab that is the cut off actually. But as early as even, even the patient is on day one, just the eye movements can be started and there can be progression as per the patient tolerance. Then the patient's neural function can be restored to normal.

Then like another comment from comment from Dr. Simona that is congrats for the presentation. How many moves do you do each canal in video head impulse test? See, for the remote camera video and impulse test test which I use, I just do five impulses. In the presentation.

I have shown some of the graphs from my real cases and you would, you would have noticed that there were only 555 impulses. Of course the, the, the machine will not take the impulses which are of high, which are of low thresholds. Like the velocity is lesser than the threshold. It will not take automatically. Automatically.

It will not take the places where the patient blinks things. So but by and large, as you know, I'm, I'm also trying to gain mastery into it. I am trying to do it you know, and majority of times you end up just give. If

the patient is cooperative and even you give instructions properly then you⁷, you know, five impulses are enough. That is minimum.

And it can go especially whenever I find deficit. I go for more impulses in that particular canal to you know, to check consistency of that particular deficit.

So another you know, like Gufran the Can you send the recording via email? Yes, Anna will help and you know it is available on audiology online.

Then another question is from Chen is uh. I have the question from my practice. I always get the high gains in one pair of vertical canals but the regular gain on the other pair of canals with the same calibration. This happens among part participants. So I wonder if this is about my technique.

Many times even I have observed that you know, right. If you are dominant from right side and left side like what happens we. We tend to give impulses on one side side faster as compared to the other side. And then what happens if you are giving high velocity impulses on one side and little lower velocity impulses on the other side? There can be you know, physiological little variation you can see because even in normal patients you can see there is a little drop in the gain.

Not below the power like threshold but there could be a little drop. Stop it. It all depends on your, you know, power you are exerting. And that's the importance of gain velocity graphs. So if you plot your gains with respect to the velocity you will be able to see how much how you.

How is your pattern of you know, giving impulses. And over a period of time I am trying to monitor my pattern and I could, I could observe that earlier my impulses will where you know they were very powerful on one side and very little weaker on the other side. Then I started giving being conscious while giving the impulses and trying to deliver equal equal like velocity impulses on the other side

especially the problem comes when your left hand, you know, even in vertical canals this problem arises this but you have to be mindful of that and you have to always check the distance between the target and the patient. And that is also important. Sometimes the gain can values can be higher because of the patient displacement.

If the patient, you know, patient changes the head position it can alter the gain values. Another question by next question is by guys from Simon Shiraji. Wonderful webinars. Thank you so much Dr. Vishal.

Thank you so much. What we it finding would you expect to find in Kogan Syndrome? Any feedback to be honest like I'm yet to diagnose a case of Kogan syndrome. I am like I might have missed, I don't know. But like you can see the vestibular deficit.

But I would like to you know, read about it because I didn't go through any particular literature on like video head impulse test in Kogan syndrome. But I would definitely, you know, email you uh, like uh, about this. Then uh, when the gain is normal, but there are corrective circuits in all six semicircular canals. See it's very important that like you have to go back and check again the gain velocity curves. Check whether there, there were any artifactual uh, artifacts.

Uh, check whether the patient's attention, attention is also important if the patient is you know, attentive during the testing. Uh, because uh, if the patient is not attentive there can be some catch up circ. You can see and it's very important that there is a sustained attention while doing. You have to give rest in between the two canal, you know, in between the. Ask the patient to close eyes and then you can, you know, so that it helps in maintaining the sustained attention.

So if, if in, in such cases you have to interpret in light of the clinical scenario because as I have told there could be two, three problems in such cases. If the patient is inattentive, little drowsy, or if you have to check the patient clinically, whether it is clinically correlating, you are getting this corrective

sacreds in all six canals. If the patient has that much imbalance, like you know, in is for example one of the case which I highlighted, the patient actually had had extraocular eye movement abnormality, that is squint which led to abnormality in all the six canals. And actually it was not correlating with his degree of imbalance, the degree of symptoms with which he presented. So you have to always correlate clinically.

Could you show us again your algorithm which is first based on detection of saccades and especially the analysis in absence of saccades, I will verbally tell you you because like I have to scribble around it will like first you check for the corrective sacket. If it is absent. If the, there are no corrective circuit, check for the gain. If the gain is normal, everything is good. Okay, There is normal gain.

There are no corrective sackets. It is normal video impulse test. But if the gain is low despite absence of the corrective saccades, then you have to check whether, whether you are, you know, dealing with artifacts. Then repeat the test. If the, the, the, the abnormality is consistent and present Bilaterally then it could be bilateral vestibular loss.

Then next is corrective sacles. If they are present, check whether they are unilateral on only one direction, then it is unilateral vestibulopathy. If they are present both sides, check the morphology. If the morphology is abnormal only on one side, it is unilateral lesion. If the morphology is abnormal on both the sides, then it is bilateral lesion.

And again I would like to reiterate that is corrective saccade even in presence of normal gain could be abnormal. Mostly it is abnormal. And then again in these cases you have to check for correlate clinically it could be a compensation. There could be like partial compensation, like there is unstable vas, vestibular ocular reflex. And that's why the patient is getting the corrective sacket despite of the normal gain.

Once again the same question. How do you calculate now the PR score? Currently to be honest, I don't calculate and so I just check on the V trace visual. How is the horizontal dispersion, the PR score, what does it tell? It basically tells the horizontal dispersion of the latency that visually also you can see if there are corrective circles.

If the VOR gain is the latency, are you know, the dispersed or whether it is gathered or scattered. Visually also you can get G again insight in the overlap diagram. Then the next question is if the patient has limited neck mobility, what is the best way to still get accurate RALP and LARP impulses? See, you need at least 10 degrees of movement. 10 degrees and over a period of time.

I'm noticing that earlier I was giving little more degree movement and over a period of time you have to learn this skill. Skill of giving very small degree of head movement and trying to, you know, assess the gain, assess the vestibular ocular reflex. So you just check the patients. You know, if the patient has painful neck movements or restricted neck movements, you can avoid and you can go on by clinical parameters, you can go for other complementary tests of your function.

So best way to get accurate RALP LARP is again the same like you know, you have to go for quick and small degree of movements in such cases. And It's a woman, 75 year old. Okay, Elena has written. Okay, tell me how to interpret V8 gain corrective CS in all six semicircular canals. Yes.

So in this patient, particular patient, the patient can be having underlying mild vestibular dysfunction. And despite of the normal cane, it indicates that the, the, the patient has mild vestibular dysfunction which which the brain is trying to compensate and there is partial vestibular unstable vestibular dysfunction. That's why this over saccades are seen so in this particular patient Vestibular rehab and careful follow up will definitely helped. So I hope, I think I have answered all the questions and I'm really thankful to all of you for such a you know, interactive giving like asking me questions and I, I hope they were satisfactory in future and and those whom I couldn't answer I will definitely reply. We have your

emails, we will uh, email you.

Thank you once again Dr. Pawar for this fantastic contribution. And so today's session marked not only the conclusion of our DHIT journey but also the final Inventis Academia webinar before the summer break. So after 10 international webinar in English perform in 5 months we are taking a short pause I would say but we'll be back in September with new sessions. For this reason I kindly ask you to take a look to our social media channel in order to be always up to date on what is happening here in Inventis.

Okay, so to everyone who joined us across these months, thank you for your continued interest and support. And from all of us at Inventis, we wish you a great summer and we look forward to see you again soon. Bye Bye. Thank you. Bye.