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Central Eye Movement Disorders: A Window to Peripheral and Central Function

Presenter: Soumit Dasgupta, MD

Welcome, everybody, and thank you very much for being with us today. We have come to the end of this cycle of three seminars held by Professor Soumit Dasgupta, consultant audio vestibular physician, neurotologist and clinical lead in pediatric audiology elder a children's NHS Foundation Trust in Liverpool. Professor Dasgupta, the time flew by. Let me say.

What impressed me most about Professor Dasgupta is the great passion for this subject. The enthusiasm Professor Dasgupta puts into describing every detail is contagious, let me say so. Title of the lecture is central eye movement disorders, a window to brain function. To date, more than 50 different eye movements are known, all of which tell a story and leads to a concrete diagnosis of neurological pathologies. This lecture discusses the desired movements with the author's personal collection of more than 80 abnormal eye movement.

This is a topic to which Professor Dosgupta is particularly fond of. Here with us, a valuable collaborator of inventist academia and I can also say a dear friend, doctor Andrea Castellucci, ent consultant at Santa Maria Nuova Hospital in Reggio Emilia here in Italy. As usual, I strongly invite all of you to write down any questions in the space provided that you should see on the screen. And after the event, even this time, you will receive a survey that I kindly ask you to fill out. The survey will help us to understand your needs, to get your ideas in order to provide the best educational support ever.

And now I leave the word to doctor Castellucci first and to Professor Dasgupta immediately after. Enjoy the webinar and see you later. Thank you, Juana, thank you very much for your introduction. Good evening to everyone. Good morning, everyone.

To everyone, depending where you are. I'm Andrea Castellucci from Italy. And today I'm here because I'm honored, I'm honored to introduce Professor Dasgupta to all of

you. Okay. He doesn't need my introduction because he's popular neurotologist in particular for his skills in pediatric audio vestibular science.

And I'm very happy to introduce him because we are friends. We are friends. We met. Maybe we got to know each other since one year ago, no more than one year ago, we met in Bourhani society meeting in Madrid. Thanks to our common friends, Professor Marco Mandela from Siena, he's the head of the ENT in Siena.

And Professor Dasgupta, he's a visiting professor in Siena. So he comes to Italy quite often, but we immediately start to be friends. And the thing that impressed me most about soumit is his enthusiastic enthusiasm as Anna said in his lectures, the passion and the things that he's teaching, that he's studying in his research. And I learned so many things from him, in particular about audio vestibular science in pediatric population, but also so many other things in the interpretation of eye movements due to central nervous system disease. So today he will have a very interesting webinar about this topic.

I will immediately leave the podium to summit, and I would like to tell to everybody, have a nice webinar with him. Goodbye. Thank you very much, Andrea, thank you very much for your very kind introduction. And once again, it's an honor and privilege to be here. I was talking to Ana about this inventive academia initiative, which is absolutely phenomenal, and it kind of reaches to the far corners of the world.

So this is the last lecture in my series, and this is about eye movement disorders. Now, originally, when Anna and I, we were talking about what I will lecture about, I came up with central eye movement disorders, a window to brain function. But as I was preparing the lecture, I suddenly discovered that to recognize central eye movements, you need to have a very good knowledge about peripheral eye movements. Because right at the beginning, let me tell you one thing, and it will become apparent and

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intuitive, really, that anything or any eye movement which is not peripheral is central unless proved otherwise. That's a very useful aid, the memoir, or a very useful tip for you guys.

So if you see an abnormal eye movement and it doesn't exactly confirm two peripheral eye movements, then it would be central, and then you can fine tune it, you can detail it even further. So that's why, with Anna's permission, I'm doing this lecture as a window to peripheral and central function. Different kind of eye movements, abnormal and normal.

So, first of all, let me talk to you about the importance of the eyes. Now, your eyes, people say, and this is biblical, are the windows to your soul, right? I remember the day that I fell in love. I was looking at my girlfriend now my wife's eyes, and I could read her eyes, which told me that she has got feelings for me. If you look into anyone's eyes, you'll be able to make out whether that person is deceitful, telling you lies, or whether that person is the truth.

So eyes are everything. They are the most expressive part of your face. And that is also very, very applicable when you are actually looking into the eyes of your patient. As a doctor, those eyes tell you a lot. It's almost like doing a blood test, which tells you a lot.

Your eye movements tell you so much about your periphery and your center. And to highlight this, I have selected this video from the movie Galileo and Chaim Topol, who acted in the movie fiddler on the roof, which I'm sure many of you have seen. Chaim Tupol was the main protagonist. Galileo. This is what he says about the eyes.

I wanted people to understand that nature gave them eyes to see her works, but also brains to make them capable of understanding.

So you see, this is very, very important that you have got eyes, your patients have got eyes. With your eyes, you need to look at your patient's eyes. But that's not enough. You need the knowledge, you need the wisdom, you need the brains to understand what is going on behind those eye movements. You know, when I went to Pisa for this trip arranged by professor, as I was telling you that in the duomo, I was pointed out towards a chandelier.

So I asked, what, what is so special about the chandelier? And I was told that this chandelier is the very same chandelier under which the same man, Galileo Galilei, watched it move and discovered the theory of pendulum. So once again, this is all to do with observation. So with this introduction, let me carry on.

This is the history of the study of eye movements from a wonderful article by NJ Wade. As you can see, there are quite a few, some well known and some not very well known figures. For example, Aristotle understood that the ways the eyes move are very important not to your brain function, but also to your spiritual function. That is an index of the soul. You have seen the name of Perkinje.

You see the name of Mac. That's Mac, that's Mac of endolymphatic flow fame. You see crumb brown, same. You see Hering, who devised the herring bra reflex in the lungs. So this has been researched.

But I'd like to highlight one man who for the very first time, actually figured out that the way your eyes move might be a problem with your ears or your brain. Among all these, the earliest one, and this is this man, Alexander Crum Brown, who was scottish in Edinburgh. In fact, he was Arthur Conan Doyle, the creator of Sherlock Holmes, teacher. And he was so animated that the way Sherlock Holmes has been portrayed was partly derived from Alexander Crumb brown. And then we published this paper about Alexander Crumb Brown, because he's not very well known.

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So we tried to resurrect him. As you can see, he, in one of his lectures described. We fancy that we can move our eyes uniformly. That by a continuous motion like that of a telescope, we can move our eyes along the skyline, in the landscape, the cornice of the room. But we are wrong in this, however determinedly we try to do so.

What actually happens? That our eyes move like the second hand of a watch. Jerk and a little pause, another jerk, and so on. Only that the movement is not so regular. These jerks are sometimes of greater or less angular amount, and the pauses vary in duration, although unless we make an effort, they're always short.

This is a classical scientific description of a saccade, which is the prime and the most important eye movement, which is stabilizing your gaze, which is giving you orientation in your space, which is doing everything very much a brain activity that Alexander Crumb Brown described, but it remains forgotten. So I would please ask you to remember that this man, who hardly came out of Edinburgh, accurately described what was later, what was later known as eye movements. Scientific expansion of eye movements. So today, more than 50 different types of aberrant eye movements are identified. They all tell you a story.

Now, there are two types of aberrant eye movements. One is when the eyes move abnormally, and one is when the eyes don't move. And there is a very strong distinction in this lecture. Unfortunately, I can't cover when the eyes don't move, when there is paralysis or gaze pulses, because then it becomes huge. So today, when I say 50 different aberrant eye movements, I'm talking about abnormal eye movements.

So the eyes are moving, but abnormally, not when they are not moving. We collect these movements in all the hay. So till date, I have got about 81. As of last week, 81 different eye movements. Probably just count, has gone up a little bit.

90, I would say. Whenever I see an abnormal eye movement, I try to record it. And we have built up a museum. I call it the Alderhey Eye Movement museum. Now, here you are going to see five different museums which you must visit if you haven't done so already.

The Hermitage in St. Petersburg, the New York Metropolitan Museum of Art, the Louvre in Paris, the uffizi in Florence, and the Prado in Madrid. These are the five must see museums that you have to visit. And to that, I've got the cheekiness, or I've got the impertinence to add my own. Alderhey museum.

Eye movement museum. This is where I work. This is where my lab is, and this is where I collect all my eye movements. So this, as you can understand from this introduction, that this is a subject of my own heart. And this is unfortunately not very well percolated in the otology or even the neurology community, except, unless if a neuro is a neurologist, they get trained in this.

But we who have come from an ENT background or from any other background other than neurology, we don't get trained in this. And that's unfortunate because they tell you so much and they lead you to the diagnosis. So the first important thing which I'd like to impress today is if you want to study eye movements or their abnormalities, you need a very good knowledge about neuroanatomy and neurophysiology. That's a given. And we get special training when we were actually training as residents.

So in this lecture, out of the 89, 90, you would see 44 different eye movements. Because I don't have time. I wish I could have shown you everything and anybody coming to UK, especially to all the hay, I'm more than happy to take you through all our eye movements. I've already invited Andrea to this and Andrea is probably visiting me

next year. So here I'm going to tell you 44 different eye movements which are not infrequently found.

Some of them are common, some of them are not that common. But you'll get an idea as to what we are dealing with here. They are highly sensitive and specific indicators of disorders of the optic system, because it's eye movement, at the end of the day, the vestibular system, what creates the vestibular ocular reflex and the neurological subsystem, that is a central indicator, central integrator. And these three systems, if they get go wrong, then obviously you'll get an abnormal eye movement and you do not need high tech software. You see, this is my lab.

This is my vestibular, pediatric vestibular lab. This kid is wearing VNG goggles. Even if you don't have VNG goggles, you can use frenzels or what I've been taught recently, you catch the eye movements with your mobile phone, magnify them, crop them and then play them in slow motion. That's all you need. Everybody has got a mobile phone nowadays.

You'll have to take the patient, you have to take the patient's permission or the parents permission, and they can be seen without the necessity of removing optic fixation. Because remember, optic fixation does not abolish central eye movements. They make peripheral eye movements heightened, especially vestibular eye movements. But central eye movements remain unchanged. There are some exceptions to the rule, which I'm going to talk later.

It needs, therefore, a very, very significant experience and a full and detailed examination of the neurological oculomotor as well as the ophthalmological examinations are compulsory. So all your nine different positions of gaze, an ophthalmoscopy and a complete neurological examination, in addition to a full vestibular examination.

So what are the fundamentals of these eye movements and why do they move abnormally? So you know that the objective of the oculomotor system is to maintain a stable gaze of the environment on the retina. Now, this is very important because that tells you where you are at a given moment in space. And if you do not have this attribute, then you. It's not really compatible with life because you are just a tiny, tiny speck in the vast, infinite universe.

Space is infinite. You need to know at any point of time where you are. And how does the oculomotor system do it? It does it by fixation. That's a visual system mediated retinal drift correction.

So when you move your head or when you are actually physically moving your whole body and your head is moving with it, your retina must get a non confusing, non confounding image on both sides which need to fuse together. And that's called fixation. That's very much driven by the vestibule ocular reflex and the ocular motor system with the brain. As we have mentioned in the next point, gaze holding is a centrally mediated mechanism by which pool of senses, suspensory and extraocular muscles, that try to pull the eye away from retinal fixation. Because in body physiology, everything has got an antagonistic effect.

They are continuously counteracted by the opposite ocular muscular activity. And the fine balance is reached even when you are trying to move your gaze. It's also used for low velocity, high movements, for example, when you are using your smooth purse suits and saccades. So obviously, these are the three cardinal principles. They might get deranged or disrupted if your system, either in the brain or in the eyes or in the vestibular system, are not working properly.

And the achillometer eye movements are very complex. Now, I am going to send this sent to Anna, a directed reading. And this is a wonderful article by our good friend Michael Stroup, who has beautifully explained central eye movement disorders. And these oculomotor eye movements are very complex. There are definite five levels, level one, as you can see in this figure, and that's indicated by the blue line.

This is what we call supranuclear control. So, over the nucleus of the oculomotor eye muscles, as you know, that there are three of them, that is the oculomotor nerve itself, the trochlea and the abusive nerve. So the upper right supranuclear area, it initiates a saccade in the frontal eye field, which is in the cortex, or it can initiate a smooth pursuit attribute or a smooth pursuit paradigm in the postero parietal cortex, which is just, you know, to the dorsolateral ponti nucleus. So from there, the impulses connects to the nucleus, that's the ipsilateral 6th nerve and contralateral third nerve nucleus. The DL pen connects to the extraocular eye muscles.

As you can see that this is with the yellow line. The extra ocular muscles to eyeball form the lower motor neuron in its wake. The level four is cerebellar connections, which is purple, which is this bit is the cerebellum continuously modifying. Because, you know, cerebral is very important for coordination of vestibular output, and this is best given this is also connected to the vestibular system. So something needs to modulate this.

And the last level is vestibular neurons from contralateral to brainstem and oculomotor neurons. Now, remember one thing, eye movements, erratic eye movements are complex. However, paralysis of eye movements are probably as complex because different types of gaze pulses when the eyes do not move can either be due to an infra nuclear lesion or it can be due to a brain supranuclear lesion. And there are many of them, at least 20 to 25. Each of them tells you a story in the brain.

But that's not the content of this lecture today. And this we have mentioned. The prime movement of oculo eye movement is the generation of a saccade, which Alexander Crumb brown very, very beautifully described way back, I think it was in 1873 or 1874. And the saccades, once they are generated, they are modulated by so many different areas of the brain. You know, you don't have to remember all these areas.

If you do, that would be great. But basically, the saccharideideedes are modulated by different neurons in that second generation tract. So there might be an IBN, which is an inhibitory burst neuron. So there are neurons which inhibit saccades, there are excitatory burst neurons, EBN, there is an omnipause neuron, which is in between the two, and there is a transitory neuron which is transiently firing. They're all in the brain stem formations.

And these are highlighted in this text here, which I'm going to put a box in. So these are the different. So TN also stands for tunic and transitory neurons. So these are. This is very much controlled by the brainstem region, but the final modulation, remember, is coming from your cortical center.

So this is well researched, and still plenty of research is going on. It's highly, highly complex, and you need a lot of time to understand what's going on. But if you're interested, it is absolutely fascinating and opens up a new world altogether. And the final balance, perception, is mediated through the vestibular and ocular motor, and it's essentially a central function. So, as you can see, this is very well known to you for those who are into neurology.

So the central connections of the vestibular systems are absolutely huge, but the main, most important area is the PIBC, the peri insular vestibular cortex, which is consisting of a number of centers which get heavy efference from the vestibular nuclei. So all these together, they generate your eye movements. And the most important attributes,

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which I've also mentioned is gaze fixation, just gaze, or vestibular ocular reflex, as we have mentioned before. And obviously, therefore, if the things go wrong, the etiology would be in these three areas. So you can understand that a problem in either the vestibular system or the brain or the eyes, either alone or in combination, will generate different types of eye movement abnormalities or no eye movement, which is basically a parasis or a palsy.

And because they are so site specific, they will tell you exactly where the problem is, where otherwise, everything else can be normal. This will become clear in a few slides. So you can see that at the end of the day, one thing. You may think that I'm very biased, and I'm not, but if a person is disorientated in space, which is manifesting, as disinhibit go, lightheadedness, fuzziness, brain fog, whatever, whatever, whatever. And if there is, the peripheral system is normal.

That is the visual system in the inner ear, that is the vestibule system. And normal, you will. Most people would call it. It's a brain problem. It's a central.

Now, remember, central actually, inherently, by default means, it's a central vestibular integration problem, because at the end of the day, all these lesions, they would affect the pathways and the tracts, which are essentially getting a heavy contribution from the vestibular nuclei. So if I say that the vestibular system, peripheral vestibule system is very important, but the central vestibular system is also equally important, please don't think that I'm biased. I mean, I have had arguments with my neurological colleagues. But finally, they had to understand and they had to appreciate that at the end of the day, any disorientation in space or unsteadiness or balance, if it's not peripheral, it's a central vestibular integration problem. Of course, it can be caused by varieties of neurological disorders.

That goes without saying. So so far, therefore, you can imagine that eye movements or abnormal eye movements, they might be of five types. This is by deduction. So supranuclear lesions, mediated in the cortex and the brainstem regions will generate abnormal eye movements. Nuclear lesions in the oculomotor nuclei itself will generate abnormal eye movements.

Infra nuclear lesions, there is an intracranial part and there is an extracranial part. When it comes out of the skull, through the different foramina, they will generate abnormal eye movements, disrupting the vestibule ocular reflex, which we all know, either intranuclear or extra nuclear vestibular lesions. Remember? And this is a very important thing to understand, you get a peripheral vestibular lesion, which gives you classical peripheral vestibular signs, right? We hit wimp, etc.

Etc. Etcetera. But do you know that if you get a problem selectively in the vestibular nuclei, you might also get peripheral vestibular signs? So the notion that peripheral vestibular signs is a peripheral vestibular sign in some instances, might be generated by a central pathway. At the end of the day, it's still vestibular.

But the central vestibular lesions in the extra ocular eye muscles. Of course, that goes without saying. If the eye muscles themselves are not working due to a restrictive mechanical structural problem or a nervous lesion, a disruption of the nerve, or due to any neurological problem, this is where you do not get aberrant eye movements. You get a paralysis, and when you get a paralysis, you get a squint or a strabismus, given oculomotor, isolated oculomotor, extracranial or intracranial lesions. However, if you get a problem in any other system, the brain of the vestibular system, you get deficits in saccharideidee generation, which we will call nystagmus.

When it's periodic. Regular repeats itself sometimes gets fatigued or when the eyes move erratically. Do not follow a pattern. We would call it oscillation. There can be a microsychotic oscillation.

There's a macrosychotic oscillation. macroschizophrenic oscillations are easy to see. micropsychotic is only when you need a good vng and measure peripheral ophthalmological disorders not involving the extra ossicular muscles. So let's say there is no gaze palsy or gay species, but peripheral ophthalmological disorders can still generate some very bizarre oscillations, some of them which might look like nystagmus. And I'm going to show you examples of everything.

And as I said, today's lecture is about eye movements and not about strabismus or gaze pulses, due to any reason, because that becomes even more onerous if Anna permits me, then one day maybe we will go through that as well. So, as I said, let's talk about the peripheral vestibular eye movement disorder, because, as I said, if the nystagmus or the oscillation doesn't follow a peripheral vestibular pattern, it is a central vestibula, unless proved otherwise.

Now, I have asked many residents and many, many fellow, well, my fellows, and when I actually go to lecture, as to how do you define a nystagmus, especially a vestibular nystagmus? Any jerky eye movement of the eye is not a nystagmus. And the nystagmus that you see from the vestibular system is usually lateral semicircular canal, but there are some movements which are not coming from the lateral, but the vertical canals. So, as you know, I'm quickly read through this. It's usually horizontal, with the fast and the quick phase, which you can easily see.

It's sustained and it does not change direction on gaze eccentricities. Sometimes it may fatigue. If it's roughly, on an average, about more than a minute, it's abolished,

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decreased by optic fixation. So removal of optic fixation increases the nystagmus. It does not.

It never rebounds on spontaneous gaze. It has different intensities in different positions of gaze, which is defined by Gustav Alexander in 1903. It's called fixed intersectic intervals. The intersectic intervals never change in vestibular nystagmus. Now, obviously, how would you measure if you don't have a VNG software?

You can still, if you're very, very observant, you can play back on your mobile phone. You'll see that the inter second intervals of the quick and the fast phases, they are different. That is not a vestibule anystagmus. And because the vestibule of basement membrane is very tonotopic or frequency specific, you can provoke all these different frequencies with different tests. For example, caloric, you know, that's non physiologic, ultra low frequency chair, about one to 2 hz.

Mastoid vibration, similar head shake, mid frequency hyperventilation. To find out whether there is anything in the cerebral point, an angle v hit. That's also a ketchup saccade. That's not exactly a typical vestibule or nystagmus, but you can provoke in order to bring out circade. And it must agree with the history, because the phenotype for central vestibular disorders is different from that of at the periphery.

So let's have a look into the vestibular nystagmus. Our first video today. As you can see, it follows Alexander's law. I don't have time to discuss Alexander's law, but it's to do with the amplitude on gaze. So as you can see, this nystagmus is central, spontaneous.

It has got a clear, quick and the slow phase. And you can understand that it's on your left, that the quick phase and the slow phase is towards the right. So it is in

compensatory period. This would be a right sided lesion, and it never changes direction. It's still beating to the left, and it doesn't rebound, it's still beating to the left.

This is a fundamental nystagmus. Now, this nystagmus, if it's subtle and well compensated, will not be picked up on the bright, proper light. So you would need to abolish optic fixation. But if the person is decompensating, then absolutely you can see it even with just a pen. Mid frequency, the head shake.

You all know about this. Once again, horizontal, quick and slow phase. After a passive head shake, you are shaking the head. Mid frequency vestibular function. You saw, I shook the head.

Now you see a very clear nystagmus. This is not localizing. Sometimes you find that this might be localizing in some articles, but in my experience, you should not make a judgment as to which side, because you are basically shaking the whole head, which is stimulating both vestibular systems. But when asymmetrical, it is an indication of peripheral vestibular problem, mastoid vibration. This is now very popular.

Once again, you put the mastoid vibrator. Now I'm not allowed to use the word vibrator. I'll call it a mastoid shaker in your mastoid process. And once again, you're stimulating both sides, so the direction is dubious. So once again, it would provide you with an asymmetry.

So, as you can see, I've touched the mastoid, and now you will see the nystagmus, which is appearing, which is the horizontal nystagmus, classical jerk nystagmus. And you can accurately measure it with. With the VNG software, the head impulse test. You all know about this. This is an adult, as you can see here.

So you will see, I don't have to tell you which direction it's coming from. So as you see, I'm moving the head. You can't see that well, because I took this with a mobile phone. But you will make it, make it out. So as you just wait for it.

I've slow mo'd it.

Nothing there you see the clear ketchup suck. So this is an adult, this is a child.

You see the beautiful ketchup saccade, only two years old. On the right with the hearing loss, you see the beautiful ketchup saccade. In fact, bilateral vestibular hypofunction. And this is what you see with a Vng magnification. You see the clear ketchup saccade.

These are all real patients, by the way. See the saccharideideede there? Nothing on the right. Beautiful left sided saccharideideed. This is a very, very wonderful test.

First diverged in 1989, which can be easily seen by. In the clinic, even with complete optic fixation. The reason being it's mediated by the type one, very fast neurons, and the brain doesn't get enough time to kind of do any central modifications. So you'll see the saccharide. The saccharideidee itself is a central modification, but brain doesn't get time for the visual system to compensate for it.

So you'll find it and you don't need abolition of optic fixation. Hennebet sign, very interesting. Hennabert sign in the presence of perforation. Perforated tympanic membrane, you call it a fistula sign. So this is a very classical third window sign.

So talking about semicircular canal dehiscence, then dilated vestibular aqueducts, any inner ear, bony canal dehesins and so on and so forth, and also in the early stages of many years. So you just put pressure on the tragus or ask your patient to do a valsalva

and you wait for the nystagmus after the maneuver, not during. So, as you can see here, I'm pressing the Tragus and the patient will also complain about dizziness. I've left it, let it go. And you can see the beautiful, how would I call it?

Anti clockwise torsional nystagmus, which is appearing on the eyes. This was a 27 year old with a superior semicircular canal dehiscence with a severe autophony.

Next is. This is uncommon, but I managed to catch this. Look what happens, eccentricity. Nothing central, no spontaneous nystagmus.

And as it turns to the left, you can see this nystagmus appearing, which he told me that it's pulsating with my heart. This was once again a very classical superior semicircular canal dehesins. You get a falsetto torsion.

Now, hyperventilation induced neck sigmas. As you know, it's to do with calcium metabolism. One sec, sorry. It's to do with calcium metabolism. In cerebral.

Upon an angle, you provoke this nystagmus by asking the patient to hyperventilate for 30 seconds. Should never do it in epilepsy, and not very much in kids, they indicate a cerebral pontine angle lesion, either for with the cross arterial compression where the anterior inferior cerebral artery, and in some rare instances, the Picard, they actually impinge on the vestibule of ochlea nerve and cause this hyperventilation. Indispens. And also, of course, in tumors as well. This was actually a cerebellar pontine angle acoustic shoanoma.

So you ask the patient to hyperventilate and as usual, once again, you ask, you record the eye movements after the hyperventilation. So we have done the hyperventilation and you see it's a beautiful jerk nystagmus. And the wonderful thing is this nystagmus

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reverses direction. You know why? In the early phases, when it is limited to the intracanalicular, when the nerve is still not myelinated, it will be towards one direction.

But when the nerve, when the tumor expands, it will be to the other direction. This is a useful way of monitoring whether your schwannoma is increasing in size before mister. So in our, in UK, generally, we would do interval scans to monitor very small intracranial acoustic schwannoma. But there would be instances where if your hyperventilation induced nystagmus changes its direction, you might be tempted to ask for a scan sooner than a couple of years.

Head heave is the physiological counterpart of the head thrust. So this is high frequency utricular function. So I'm moving the head, not a thrust, not in an angular plane, but in a translational pain. And you see what happens here?

You'll see the kiss occur. I don't have to show it to you. There we go. Nothing. There we go, nothing.

There we go. This is not head thrust, by the way. This is a head heave, which is utricular function, ocular counter roll. Very interesting. You ask your patient to move his head all the way and touch the shoulder.

What the eyes will do is generated by the triclocular system. The eyes will move on the opposite direction rollingly. So there would be a torsional movement of the eyes on the opposite direction. So see what happens and see the beautiful movement of the eyes. Rolling of the eyes.

The important thing is the actual rolling is a dynamic counter roll, which is done by the secular. And the displacement from point a to point c, point b, is the static ocular

counter role, which is served by the utricle. Now, that was a normal one. You see an abnormal one here. I'm doing the same thing.

But can you see any static counter roll at all? You see a little bit of dynamic counter rule and a very, very tiny static counter rule. So this was a utricular function dysfunction again. And nowadays, I'm sure inventors has got it as well. You can physically measure this.

This wasn't measured when I took these videos, but now you can physically measure this BPPV. Nothing to say. The commonest variety. You get the posterior canal, which is very classically upbeat and torsional. So this is posterior canal.

This is a classical geotropic of a lateral canal. Of course, you can get apogeotropic lateral canal as well. And this is anterior canal, or common crux, where you get a positional downbeat on the deep head hanging positions. You'll see that when I'm putting the patient in a deep head hanging position, there will be a downwind nystagmus. That's the downwind.

He's in the deep head hanging position. So this could be common crux, or anterior canal, which is the rarest variety. But positional downbeats need to be scanned. There are some clinical pointers just by the maneuver as to when it is a positional or one is a central one. For example, a positional anterior canal or common crux will not last for more than 30 seconds, whereas a central positional downwind will carry on and on and on.

And that's an important point of distinction. But I would recommend that if you get a positional down weight, it's always good to scan, and my friend Marco will tell you the same. So now you're in a good position to understand that. How does a peripheral

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vestibule, nystagmus, or a peripheral vestibular system generated saccade looks like the dictum is anything which doesn't follow. This is central unless proved otherwise.

And central means the brain central means central vestibular integration deficits. Let's have a look. So they do appear as normal abnormal acculomotor movements and they may coexist. So in the same patient, you might see a central and a peripheral eye movement, and both of them you can make out every individual abnormal eye movement suggests deletion in a definite sight, which is subtle and which will predate predate scanning. So you might have a situation where you pick up a central eye movement problem and then you scan the patient and the scan is perfectly normal.

You should repeat the scan after a year or two and I can guarantee you you'll find something, because scan needs a certain size. That's a drawback of a scan. Scans are wonderful, but they need a certain size of the lesion to attain before you can physically see what is going on. Central. The oculomotor eye signs, they can occur at a very, very subtle level.

And I've got many examples here. When I have recommended to my neurological leagues that please do interval scans and you will find out the lesion they might present with the vestibular phenotype. You know, they might present with vertigo, they might present with anything. Although vertigo is very classical of peripheral vestibule, that is. But if it's near the region of the vestibular nuclei, you'll feel, you will perceive what I go.

And neurological signs and symptoms must be discerned. That is why it is so important that you have a full assessment of the neurological system. Everything, tone, power, reflex, cerebral sensations, everything. You have to be well trained to do it. And not only that, you need to know if something is abnormal, what does it mean?

So you see the amount of neurological knowledge which you have. Now in UK, the audio vestibular medicine program, five year residency, will train the trainees with neurological phenotypes and neurological conditions. And that is very important message today, that for people who would do vestibular neurotology, they need a very solid training in neurology as well, because the distinction between the two system is quite blurred. So these are the different types of abnormal size movements and the list is not exist. Exhaustive.

For example, I haven't put the oculo gyric crisis, but these are the ones which I have seen. So we have seen different vertical nystagmus, pendulum torsional rebound, gaze evoked nystagmus, which changes its direction in the middle direction, changing vertical head shake. Perverted head shake. What is a perverted head shake? I'm going to explain square waves, inverted caloric nystagmus, for example, you know that calorics will classically give you the cows direction, which is cold, opposite, warm, same.

What if cold becomes same and warm becomes opposite? Then you might get a report saying, the clinical significance of this is uncertain. But you'll know that along with historical presentation and along with other signs, this is a case for central vestibular integration, very likely at the junction of the medulla and pons. And you scan the patient and you find out there's a demyelination or something going on there, even an arachnoid cyst pressing on it. So this is the way you do it.

You need to know the exact site. So that's why I keep on saying you need to know the topography and the anatomy of the brain abducting ion, nystagmus, opsoclonus flutter. And I'm going to show you most of them. Pulsatile nystagmus, as you saw. I've already shown you one, but this can also come from the rostral medulla as well.

Vor cancel saccade when you cannot suppress your Vor properly. All these I'm going to show you right now in this lecture. So these are the ones which I have selected for you. But this is not an exhaustive list. There are many more seconds you all know.

So when you are trying to use the random paradigm, you can do it with your finger, you can do it with a toy in pediatrics, you can do it with anything, or you can slap in a VNg goggles and put some lights which move. And this is when the person is unable to track those fast moving, fast moving dots or fast moving targets. And you might get an overshoot, you might get an undershoot. As you can see here, there are some undershoots and there are some overshoots. That's an overshoot.

And this, when you get saccades, it comes from the vestibule cellular pathways, the extrapyramidal system, for example, the basal ganglia, the median longitudinal fasciculators, the parapontan nuclear formation in case of supranuclear lesions, and from the cerebral cortex, when the DLPM doesn't work, the dorsolateral pontine nuclear. So, you see, the point is, do you have to remember all these different sites now, at this stage, because I've been doing eye movements for the last ten years, I'm so fond of this, I have made a special effort of remembering the different sides so that I can write to my neurology colleagues where the problem is for them to take over. This is one thing. Do not try to treat a central neurological or a central vestibule intuition problem yourself. Your neurology are there to help you, because they are the ones who will deal with neurological pathologies.

You are not. You are an inner ear otologist, but you must be able to diagnose. So do you need to remember all these? Well, what I would say is, if you are very passionate, if you want to seek the knowledge, and if I'm, you know, today's lecture, I have managed to inspire you enough, then, yes, you will remember this. Otherwise, all you need to remember is this is coming from somewhere in the brain.

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And let it go. Let it. Leave it for your neurological leaks to do the rest. But the fun, actually, guys, is it's a lot of intellectual stimulation when you can accurately pinpoint. And it's very rewarding.

You know, when I see all these eye movements, I send for scanning. When I see that it is backed up by some scanning result, it just makes your day that you have learned something very, very worthwhile. Smooth pursuits once again. Now, one thing about Sakajan. Smooth pursuits always do horizontal.

Never forget vertical. Vertical. Smooth pursuits and vertical circuits are better indicators of central dysfunction than horizontal ones. Because you see smooth pursuits, horizontal can be normal in very young and very old. So how do you make a distinction?

You can make a distinction because it has to be backed up by other signs and other symptoms. So this is a smooth pursuit, which you can see. It's clear, you know, he can't smooth, he can't track an object smoothly. You see this broken up. So what we call dysmetric, smooth pursuits horizontally can happen from intoxication, from the cerebellum, from the cortex and the extrapyramidal system.

They are that specific? Very, very specific. Now, this is. I sometimes do this as a teaser. Teaser to my, to my residents and fellows that just describe what you are seeing.

And, you know, just, it's become so easy and so apparent. It's not rocket science, really. The anatomy is rocket science, but not this. You see, no quick and fast phase. It's equal excursions on both sides, just like the ticking of a pendulum, which Galileo discovered long time back.

So this is once again coming from vestibular cerebellar pathways, cerebellum and brainstem, especially the vestibular nuclei. So you get a pendulum nystagmus spontaneously. And then you also manage to quantify a VHIT that might be positive here. You might ask me a very interesting question, which is entirely pertinent and which is entirely relevant if you have got an added central eye movement, will not that put some noise in your peripheral vestibular quantification? Absolutely.

But there is a trick, because all these central eye movements, they have got what we call inter saccadic intervals, all of them. These are seconds, right? You'll have to capitalize on the intersecting interval to give you a vestibular stimulus. For example, congenital ionist nystagmus. You need to wait for the null point and then execute your peripheral vestibular provocation maneuver.

This one, for example, this pendulum nystagmus will go on and on. You wait in between the two, for example. I'll show you the way to do it. You see, when the eye is absolutely still, from one swing to other, that's when you need to execute. Maybe you'll have to do 25 to 31st in order to achieve three or four positive ones, but you can still do it.

And if you can quantify, if you can show a vestibule, and this is, of course, an art, this takes a lot of practice. So I wouldn't ask you if you're not practicing enough to make a judgment immediately. But at the end of the day, you know that this is coming from the vestibular cellular pathways, so maybe it will show or stimulonuclear lesion, for all you know. This is a pure torsional. This is from lateral medullary syndrome, and it comes from anywhere in the brainstem because that's where the IBN, the EBN, the Tn are all located.

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And any saccharideidee generator or modifier problem can generate a pure torsional. In clinical practice, pure torsional is very rare. So this is not common. What happened there? Yeah, should play.

Yeah. So this is beautifully torsional.

This is not ocular counter rolling, by the way. This is spontaneous.

Upbeat. Now upbeat. HorizoNtaL nystagmus is quick phase and fast phase. The beat is always determining the direction of nystagmus. So if nystagmus is beating towards the top vertical axis superior, then that's an upbeat.

When it's beating to the down vertical axis inferior, then it becomes a downbeat. So you can have one in spontaneous. So look at this one. So, as you can see, spontaneous, there is a slight upbeat, but look what will happen. And this is a very interesting case, one of the most interesting cases in my life.

This PersoN presented to me with dizziness and Postural instability.

So you see, this has become more. And, you know, when this has become more, this is when I moved Optic Fixation. So you have got a central nystagmus, which is actually heightened by ReMOval of optic Fixation. Now, this person also presented to me that with a significant episodic headache and associated with autonomic symptoms, for example, visual blurring and all those things. But the dizziness was not episodic.

So I was foxed, I didn't know what to do. So I asked a colleague, who is a very big name in America, who told me that this is very classical of brainstem migraine propranolol. The thing went, we did every possible scan. So migraine can generate upbeat. In fact, I've got quite a few.

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So, upbeat nystagmus can come from cerebellum, brainstem migraine, or it can be normal. It can be normal in the very young, it's usually, it can be normal in smokers. Don't ask me why, please, I haven't got a clue. Maybe nicotine has got something to do with it. So this is spontaneous gaze, upbeat.

Now, you might not have an upbeat on spontaneous gaze, but when you move gaze. So either on gaze eccentricity or when you move your eyes up or down. So look at this one. There is hardly any nystagmus in spontaneous case, but when he tries to look up, or maybe this was down, I don't remember, you will get this very typical upbeat nystagmus. Just wait for it.

There's nothing really good eye movement and the phenotypes are very typical. All my patients remember come to me with some kind of disorientation space. That's the, that's my main source of. Ah, there, there we go. So you see, once he moves his eyes up, there would be a downbeat.

Miss upbeat mistigmas. There we go, there we go. Very, very subtle, but it's very much there. And when he looks down, it will be similar. Wait for it.

Now, of course, we can measure all this, but you don't have to see that movement.

So this is gaze evoked vertical nystagmus. That's a hectic, which I'm doing, by the Way, and that abolishes the nystagmus, but that's a different story. Opposite is downbeat. This one is classically downbeat. This, you can easily see that this was a foramen magnum metastasis from a breast cancer.

Downbeat. Remember, upbeat nystagmus can be normal, but downbeat nystagmus is never, never, never normal. The downwind nystagmus is serious business and, well, it

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can come from the skull base and it's not uncommon in foramen magnum or peripheramen magnum space occupying lesions. As I said, this one came to me with, you know, about fifties lady who had a breast cancer treated 30 years back. Came to, went to ent first, saying that, you know, I'm feeling lightheaded, and I have got a severe, severe, severe ear pain on the right side.

So they did everything. They did a flexible scope. They couldn't find any area for referred pain. They did a whole set of vestibular function tests, and they didn't find anything. Finally sent over to me, I picked this up.

There was some peripheral numbness. This, along with the downwind nystagmus and a disorientation space, suggested to me that it's a foramen magnum lesion. And when I scanned her, she had this metastasis from a breast cancer treated 30 years ago, and she was dead very, very quickly. But I could take away her ear pain. Nothing was working.

No paracetamol, no nsaids, nothing I could take of her ear pain with carbamazepine, because it was a neuralgic pain, she was comfortable. And that is a big, biggest reward. This one, again, was an episodic ataxia type picture where you get a torsional downbeat. So the downbeat is not only downbeat, but you also have a torsional component. Can you see the.

Clearly the torsion. So downbeat and torsional. So these are kind of mixed. I'm just showing you this for a few seconds. I do know that it doesn't do justice to what you actually see.

This is just to give you an idea. But believe me, if you see more and if you keep your eyes and mind open, then you will be able to pick all this up. So this was an episodic ataxia. In our series, square waves. They look exactly like a vestibular jerk dystagmus.

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See? Slow phase, quick phase, but the period between the slow phase and the quick phase, or one cycle of slow and quick phase, and the next one is quite prolonged, more than at least 20 milliseconds. And that is when you say that this is a jerk. Horizontal nystagmus with prolonged intersacadic intervals, which might change in vestibular nystagmus, intersaccharideideede interval never changes and is finite and definite. However, here it can change, and it's at least 20 milliseconds or more.

That gives it the definition of a square wave jerk, which you can clearly see when you plot it. Even if you can't, you can still make out. Just look at the duration or the interval between two beats of nystagmus, and it can come from cortex cerebellum, it can be normal, it can come from basal ganglia. In fact, Parkinson's patients might have a square wave jerk.

Now, gaze evoke nystagmus. You know that when we are looking for Alexander's nystagmus, right, you are asking the patient to move towards one gaze eccentricity, right or left. Ideally, Alexander's nystagmus is best seen when it's midway between your extreme and midpoints, around about 45 to max, 60 degrees. Gaze evoke nystagmus is a nystagmus which is generated when you look towards the extreme of gaze, completely towards the extreme. This is very, very important, because physiological nystagmus is also a kind of gaze evoke nystagmus.

But there are obvious difference. This is. I'm going to tell you that when I show you a physiological nystagmus. But remember, if there is a gaze evoke nystagmus, so central, spontaneous, hardly anything, you might say. There are some random gaze, random oscillations.

But look on this gaze eccentricity, there is a gaze dependent direction, which is the right beating nystagmus. Look at this, Clara, which goes on and on and on. It goes for

a very long time. In physiological nystagmus, it's no more than five to ten beats here. It goes on for minutes if you are patient.

But as a rule of thumb, about 20 to 30 seconds would be enough. Then a funny thing happens when he brings the eyes back to the central position. Look what will happen. You see a spontaneous jerky, stagnant. That's called a rebound.

This is coming from vestibular cerebellar pathways, from the medulla, so not the pons as such, and from the interstitial nucleus of Cajal, which is an important site where you might pick up neurological lesions. And gaze evoked nystagmus is also on the other side. It will once again beat to that side. So right sided gaze eccentricity, right beating, left sided gaze eccentricity, left beating, direction changing, cannot be vestibular, although looks like vestibular, doesn't it? Horizontal jerk.

This is a gaze evoked nystagmus as well as a downbeat nystagmus. So I did tell you that you can have a mixture. This can come from the brainstem or the cortical. This was a brainstem demyelination. As you see, there is a gaze evoked as well as there is a downbeat, and clearly see the downbeat there, that's a downbeat.

And on the gazebo, there's a downbeat. And same here. So in other words, you'll have to look for nystagmus in all nine different positions of gaze. Stop that one. Play this one.

This is a gaze evoke. So you can see the gaze evoke. Look at the sclera. Sclera is moving and there is an upbeatness thickness. So this is coming not from the metal, this is coming from the ponds.

So this was a pontometal relation, or it can also come from the cerebellar pontine junction. So roughly where the cerebellum joins the pons. So this is gaze evoke

nystagmus. You will not call Alexander's nystagmus gaze evoked, you will call it gaze positional nystagmus, because the gaze doesn't bring on the nystagmus. The nystagmus is brought on by vestibulocular reflex derangement.

Here, the gaze itself brings on the nystagmus. So it's gaze evoked provocation. Now, this I haven't seen. I haven't seen any periodic alternative distinguish in my life. Probably a region.

One of the reasons, because it comes with plenty of gaze palsies. Now, obviously, I don't get referral for gaze palsies. I've seen a few because they usually go to neurology or ophthalmology and they don't come to you with dizziness as such. So this has been borrowed from Professor Timothy Hayden in Chicago, where if you can make out, then the direction of the nystagmus changes. So this begins with the left sided direction, but after about 30 seconds it becomes right sided.

So it's a direction changing nystagmus in the same sitting, which is the name, suggest that that's when it becomes a right sided one. It's difficult to appreciate like this, but that is the idea that a nystagmus which changes his direction in the middle is a periodic alternating nystagmus which comes from the Pontumari junction, or what it hit, shake. So horizontal head shake generating horizontal nystagmus is peripheral. Vertical head shake generating a vertical nystagmus is central cerebral or brainstem. However, sometimes you get situations like you move horizontally and you get a vertical nystagmus, or you move vertically and you get a horizontal nystagmus.

This is called a perverted head shake. Once again, it's brainstem and it's very classical of the perihypoglossal nuclei region in the brainstem so this one was one of our patients who had a brainstem lesion. So I've done the head shake. Now look what happens.

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Nothing good. Maybe one beat of up. Now I'm doing the vertical. Now see what will happen.

Always do the vertical electric, please. You see that nystagmus appearing?

So this is a perverted vertical heat shake generating. A horizontal nystagmus is coming from somewhere in the. Where it's, let's say, the metal medullary region rather than from the top oculus cube. Nowadays you can measure it. This is a very wonderful video I borrowed from YouTube.

No names because he only calls him the doctor in Kuwait.

So skew deviation is malalignment of retinal axis or pupillary axis. One is up, one is central. Three different types of skew. One of them is coming from the intriculus circular system. And that's a wonderful period paper by Thomas Brunt.

But otherwise it comes from the brainstem of cerebellum and it is part of the hints paradigm, which is now often done for stroke patients. Skew is a central sign, unless of course, it can still be a peripheral autolytic lesion. Obsoc. This is classically very site specific, coming from cerebellum, vestigial nucleus, due to any reason, and this is taken from any GM. So as you can see, this is off the clonus.

So remember, ankle clonus sustained non tetanic contraction.

This is not horrid. This is not horizontal jerk. So it's not definitely vestibule. It has to be central. It takes some rest and then starts all over again.

continued

And it's quite bizarre. Quite bizarre movement. Now, this was a pair case which was published. This is a very weird osteosclerotic lesion at the Glyphos region and see what happens. You can only describe this as a flutter.

Flutter.

Now, as he has grown, this flutter has disappeared. I checked him about two, three months back. He's much better. It's just because of central cerebral compensation that he has come out of this. So that's an ocular flutter.

It just looks like a flutter. Rapid oscillations. This is when you do the VOR cancelled test. So put the patient in a chair, this is a target, and ask the patient to follow the target. It should be a smooth eye movement, but if he can't suppress, then there would be a ketchup saccade, which you very clearly see here.

There we go. You see the saccades there? And surprisingly in my cohort haven't published it. This has got a reasonable correlation with the number of shim saccharideideedes that you produce. So if you see a patient with hardly any shim saccades, that means there's a serious problem with the way that person is suppressing the voR.

Because shim, at the end of the days, that's what it says, you are suppressing your vor. This one has got a reasonable kind of correlation with cerebral of f and low. I've got at least ten to twelve which we haven't written up. I'm just waiting for someone to give me a hand because I've got too much in my plate. This is very interesting.

This person came to me with vestibular migraine, typical vestibular migraine. And he also had this postural instability, not exactly ataxia, but ataxic, semi ataxia. So not a

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very gross problem, but definite unsteadiness and postural instability. And this is what we found in his eyes. Look at the eyes of your patient lovingly.

Okay, I'll just fast forward it. So far, so good. Now look, upward gaze. It will come. Wait for it.

It's broken up. Anyway, the thing is upward gaze. And you see these upward beats? You see the gross upward beats. Upward gaze.

Not enough convergence. That's nystagmus. You see, that is classical of a convergence retraction, nystagmus. And that is a hallmark of a dorsal midbrain lesion. When I scanned him, I found that he has got what we call a pari nose syndrome because of a stenosis of the aqueduct of.

This was a neurotological diagnosis from my clinic. And then subsequently he ended up with a shunt because he had developed hydrocephalus. And you know, the other thing which struck me was he was very, very emotionally reactive to his vestibular migraine. I've never seen someone who reacts to a vestibular migraine in such a negative emotional way. He was a complete wreck.

So I asked myself the question, why is he so emotionally challenged? And this is because his lesion was close to the emotional center, slightly removed, but some kind of thing is going on which is kind of marrying his vestibular migraine to his emotional nuclei, the amygdala, the corded nucleus, and everywhere else which are not far. So the migraine firing is actually also affecting his emotional limbic system nuclei. And as a result, you are reacting to it very, very emotionally. That's what I thought.

And we have given him CBT and everything else, and I managed to take out his vestibular migraine with topiramate completely. So plus he has got a shunt now, and,

you know, the way I'm monitoring him. I spoke with my neurological colleagues and we said, we decided, neurosurgery colleagues and we decided that we are going to watch his fury. Nose is tegmus. Whenever he feels symptomatic, I quantify his convergence retraction nystagmus with an increased amplitude, and I sent him to neurosurgery, who then adjusts the shunt.

So this is, once again, you can get away without doing any imaging just by monitoring the paranormal nystagmus. And we also have got ophthalmology colleagues with us as well in this. It's a multidisciplinary meeting, but at the moment he's doing quite well.

Abducting eye is pathognomonic, almost of inter nuclear ophthalmoplegia. So look what happens when he turns the eyes to the left. The right eye will not adduct, so adductor palsy, but the left eye will throw into an nystagmus. So it's called an abducting eye nystagmus, that I will abduct. The left eye will abduct and will have an nystagmus.

The right eye will show paralysis of adduction. This is a classical abducting eye nucleus, which can be the first presenting sign of multiple sclerosis. These are macrosychotic oscillations. It can be normal. You know, you might not have any pathology, but you see this doesn't follow any pattern of any nystagmus.

It's completely bizarre. Very, very random eye movements. It can still be pathological if there are other features. And seesaw is quite diagnostic of a chiasmal lesion, as you can see, when you go down to seesaw like that, that's what it is, and it can come from the midbrain as well.

Excellent. We have just over 1 hour. So you see all those different eye movements where they do not conform to a classical peripheral pattern, and you make a diagnosis of a central lesion, a central vestibular integration lesion, and you saw the sight

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specificity of these different eye movements. Now, please remember one thing. This kind of diagnostic eye, when you understand what's going on, doesn't come in one day.

You have to train quite hard, you have to read a lot, and you have to continue to see the eye movement. So today's objective is so that it opens up a window in your heart, it opens up a window in your medical algorithm, medical expertise, that yes, there are movements which can tell you a lot, and then you expand on it, and that's when I will feel that my talk to you is vindicated if I inspire you into this wonderful world of looking into your patient's eyes. You know, it's almost like stargazing. You know, you love looking at stars, so look at your patient's eyes.

So problems with your eyes, do they generate nystagmus? They do, but it's called dissociate nystagmus, which means a nystagmus which has got all components, and they might be jerky nystagmus, a quick and slow phase. They might, but there has to be a null point. So all dissociate nystagmus must have a null point because that's the only time the person can see. Otherwise, he can't.

Monocular visual loss due to any reason can generate a low velocity amplitude horizontal jerk nystagmus, but only when you cover the eyes and bring out the visual loss. So this is called a latent unilateral nystagmus. Nystagmus with mild points is typically pendulumr and congenital or infantile, and they always present with focal ophthalmological signs. And your ophthalmoscope will tell you a lot. And many ophthalmological conditions, of course, present with different types of central dystagmus, and also because you remember I told you about the optical system as well.

And of course, they would present with many types of, uh, gaze pulses or strabismus. So this is a congenital nystagmus. Interesting one. So facet person is born with it. It

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can be generated from multiple slides, usually from the saccharide generator, uh, brain stem performations.

And it can be genetic. It can be anything, really. For example, oculocutinous calvinism. There are many conditions. So look what happens here.

Oops. Why is it doing this? I don't know. Anyway, I'm sure it can play so rapid. Pendulum jerks, but, but, but wait for it.

Suspense. Stop.

Stop. That is the null point. You have to identify the null point, which can come after a minute, and that's what is keeping you alive. And I have been told by many, many people that how on earth can you do a vestibular quantification such. Such a continuous eye movement?

Wait for the null point, and you won't find this any textbook. This is experience. Wait for the null point and then execute your head thrust. Wait for the null point and then execute your head heave. Wait for the null point you measure, you have to wait.

And sometimes, as I said, it can be up to a minute. This is a manifest latent. So from oculomotor paralysis, from cataract, from oculomotor malfunction.

Now, you see the nystagmus, which is a pendulum nystagmus. Actually, it's not a jerk nystagmus, but it's only apparent once you cover the eyes and then it stops. And obviously, I have taken this from Professor Digry, who is an ophthalmologist, not a neurotologist, because they have got a large collection of these ophthalmological instagram, not me, dissociate. This was retinal lesion in one of my patients. You just watch the eye movements.

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It's very, very bizarre. You will get some upbeat, you get some horizontals, you get some downbeats, you get pendulum, you get torsional, you get all sorts. And this was a retinal disorder. It was a genetic retinal disorder.

So next we come to mixed movements, oculomotor eye movements, displaying both peripheral and central component. In the same examination, you can make out that tells you that whatever is happening has affected the peripheral vestibular system as well as the central vestibular integration. Now, the first one is physiological gaze extreme. And you see typical gaze evoked, but not gaze evoked. It's gaze positional because it never lasts for more than 10 seconds.

You see quite significant movements, and you see it many times. And this is a mixed movement because it will change direction, which is central, so it moves, it beats towards direction of the gaze. It's peripheral vestibular, because this nystagmus is generated by a peripheral vestibular system. The moment you look to the right, that's when your right sided input increases from your vestibular nuclei and your left side is damped. And the brain tries to compensate for it with this nystagmus, but then soon the system is restoring, or the entire input from either side balance is reached and the nystagmus stops.

You might get a thing called an exaggerated physiological response, in which case it can be a pathology, like a gaze evoked nystagmus. So I think I have managed to, for you to appreciate that vestibular nystagmus is characteristic and it's got some features, and you should be in a position to identify when an nystagmus is not vestibular optokinetic. This is from an ophthalmologist as well. Supranuclear and pariet occipital lesions. So you follow a rotating drum and they measure the gain, obviously, their abnormalities.

Well, I would use optokinetic stimulation in my clinic with a drum sometimes or with a computer target. However, I wouldn't. I usually do not incorporate into my regular vestibular test battery, because optokinetic system is what you are endowed with. And unless you've got special software, it's difficult to kind of make a judgment whether it's abnormal or not. But you can, you're most certainly, because by this time, you have picked up.

In other words, you won't find a central vestibular integration problem where you get only an optokinetic. Optokinetic usually comes with something else. This is an interesting one. This is called a branch distinguish. I'm lucky to catch this.

So this presented to me with a complete sensory neural hearing loss and a complete loss of vestibular function. So the hearing loss went first, thought it would come back. Never did vestibular function. Very dizzy, lost balance after three or four days back on his feet, and he thought that he has recovered. He came to me because during exam times, he was getting very stressed, stressed and dizzy.

So is there ever still a problem? Look at the eye. So, first let us appreciate that he is having a beautiful pendulum beat.

That's pendulum. But look what will happen after some time. It converts to a jerk. Still pendulum, you'll have to be patient. One thing, when you're watching your patient's eyes, you have to be very, very patient.

That's when it becomes a jerk.

So you can see the jerk, which is beating to the right, to the left, rather. He's having huge gaze evoked excursions as well. So on the same eye, you have seen a pendulum converted to a jerk. Huge gaze evoked. As you can see, huge gaze evoked.

This can never be physiological, and you'll see a rebound when he comes back.

So that told me a lot that some misleading is going on in. The best part of the mixed lesion is either the region of the fourth ventricle or at the ceremonial pontine angle. So I scanned him, and there was a huge 36 mm cerebral pontine angle tumor, which ended up with a stereotactic gamma knife surgery. So let me ask you a few questions. Please be kind enough to write it down with chatlin.

This is not an exam, is just to make it more interesting. So, as you have seen, vestibular nystagmus is usually horizontal. Can you name any nystagmus that is not horizontal. That can arise from the vestibular system. First question.

Second question. This patient presented with bilateral vestibular ear weakness, ataxia and bilateral hearing loss. Where is the site of the lesion? What are the likely pathologies? Just watch carefully.

We'll go back to it. So this was what we found.

And I've still got plenty of time, so let this run through. Please, just watch. As I said, you'll have to be patient. Two, three minutes. Easy.

By the way, we quantified the vestibule of white weakness. With the vhit and a vampire, they were non existent. Both sides counter rolling.

Let me tell you. See at least six different eye movements, and they all tell you a story as to what the pathology is. I mean, if this is not Sherlock Holmes, then what is? This is the head heaven.

Okay, I think we have played for nearly two minutes. Finally, the conclusions. Sensitive and specific indicators. Some of them are normal. They have defined characteristics if they are peripheral.

Anything which is not peripheral is central. And you need a robust knowledge of peripheral vestibule, the ophthalmological system and the neurological system in order to understand eye movements. It doesn't come overnight and it needs a lot of reading. It leads a lot of experience in order to come to this stage. However, it is highly doable if you have got the interest and the passion.

So beautiful quotations. And this is my favorite, Galileo Galilei. Again, the Bible says, our eyes don't lie. They are windows to the soul. And Galileo said, gentlemen, I beseech you in all humility to trust your eyes.

This was when he was being disbelieved by the Medici court that the sun is not the center of the universe. When the sun is the center of the universe. So he said, well, I have seen it through my telescope. I can't disbelieve my eyes. I can't disbelieve.

I can't trust. I have to trust what I've seen. I wish you the same. But obviously they didn't believe when he was called up to the inquisition and he recanted. And the middle one is.

That's Gupta's comment. Your patient's eyes don't lie either. They are windows to very, very complex disorders. So I end there. And hopefully I've managed to inspire you even tiny bit and take this up, because this is very much ignored in the.

In the neurotrological clinic fraternity who does not have neurological training. But you can inculcate this, and you can, you can be absolutely on the ball, figure out what's

going on thank you very much. And you're more than happy. Welcome to get in touch with me if you want. Thanks very much, Anna.

That's it. I'll end my slideshow. Thank you. So, eye movements tell a story, and our eyes don't lie. Correct.

Amazing. Simply amazing. Sorry. Now, as I told you that, you know, eyes. Eyes are really everything.

Yeah. Everything is eyes. Yeah. And I'm getting loads of compliments, Professor Dasgupta. I think that this lesson was inspiring in outstanding learning opportunity for our audience.

And let me say again that I really would love to have you as speakers for a future educational session. And I'm sure that our audience feels the same way. Absolutely. Please do. Definitely.

So, about questions. I'm getting a lot of questions as well. Professor Desgukta, can I ask you some questions from the audience? Yeah, yeah. We have still got half an hour.

Yeah, absolutely. Go ahead. Fantastic. Let me read all the questions exactly as I see them on my screen. So, Melanie Graze is asking, can a posterior circular stroke manifest with a bilateral positive head impulse test?

That's a good question. So, normal human anatomy is the AICA, which supplies the vestibulocochlear artery, in less than 20%. The pica may also supply the vestibulocochlear artery. So if you get a bica stroke where it's unfortunate enough to supply the vestibulocochlear artery, then most certainly you'll get a bilateral vestibular weakness or unilateral vestibular weakness, whatever that is. Right.

The second thing is, in some instances, the posterior inferior cerebral artery does supply the vestibular nuclei as well. So, depending on what exactly the stroke is and where the embolus is, you can have selective peripheral vestibular hypofunction due to a central cause. Yes. So the answer is yes, but it depends on human anatomical variation. Okay, fantastic.

Thank you.

Ramish. Say superb. And then. Okay, sauza, I don't know if I might pronounce you correctly. Your name.

Professor Rosguksa, in your case, experience, what is the most common finding in DMG tests in patients with vestibular migraine? Okay, so we have actually published. Now, look, I am mainly pediatric, so I can talk to you about pediatric vestibular migraine patients. The commonest. The commonest central sign in pediatric vestibule, because we have found all sorts.

The commonest pediatric. In the pediatric series, the commonest vestibular sign that we have picked up is a gaze evoked nystagmus in vestibular migraine but saying that, you know, this is quite non specific, you do pick up many central signs. In vestibular migraine, you pick up upbeat, you might pick up direction changing, might pick up vestibular nystagmus, jerk, nystagmus. But the commonest that I have found is Gen.

Did you get that? Yeah. Anna? Yes. Fantastic.

Then another question from Jerry. Jerry Kaplan, can you please review. He said the difference between normal and abnormal. Square wave jar. Okay, that's very good.

Look, there are many papers which tell you that the length of the inter saccadic interval will tell you whether it's pathologically normal or abnormal. So, as I said, as a rule of thumb, the intersect interval is about 20 to 200 milliseconds. So if it's beyond this, then it becomes pathological. However, what I'll tell you is, just by this intersecting interval length, you should never make a judgment. Your judgment will be based on what you have found.

So the phenotype, the presentation, other signs, the age is quite important. For example, I see 78 year olds with square wave jugs, which is entirely normal, but they will be abnormal. If the same child has got other eye signs or other neurological problems, then it doesn't become normal. So I would say that because majority of you, not majority, some of you will not be having this measurement device where you can measure the intersecting interval. I mean, at a certain stage, you can just trust your eyes.

Once again, you trust your eyes and you can make out just by eyeballing or just by looking, whether the intersecting interval is very prolonged. If you do not have that facility or that expertise, then go by how your patient is presenting and what other signs are there. So if the patient is not conforming to, let's say, central vestibular integration deficit or a peripheral vestibule, yet he has got square waves, it's very likely normal. But you are going to get patients referred to you where there is a problem. And there, if you find a square wave, if you think that there are other things to back it up, then it's pathological.

Otherwise, if you can measure the intersecting interval duration, that's fine. That's an added bonus. But please go by what the clinical picture is.

For example, let's say that square waves may be normal in the elderly population, too. But if I get a elderly person coming to me with a head injury and has got a square wave

and has got a vertical and has got an upbeat, then I'm 100% sure that square wave is pathological and not normal. So do you see that the way you kind of marry the context of what you are seeing. Does it make sense? Anna?

Yeah, it makes sense. Of course. Then one more question, Professor Dasgupta. Roxana says, I see many patients with positional, horizontal geotropical nystagmus, but with no vertigo and no history of positional vertigo. Is it central?

Okay, so it's important. What do you actually see? So if you see someone with a classical history of a BPPV with this, then as you rightly conclude, that there is a BPPV lateral, however, if the patient is not getting any positional dizziness yet, you are picking up a positional geotropic or even an apogeotropic. I think the most important distinction to make out whether it's the central or peripheral is the duration of the nystagmus. See, if the duration of the nystagmus peters down or kind of fatigues out or stops by 20 to 30 seconds, it's very likely physiological, actually.

However, if it carries on for 30 seconds or more, it becomes central. This is one thing which I've also seen, but I haven't put down in my list of central movements. So please check the duration and very importantly, the amplitude as well. If it doesn't. What did.

What I did say that in the beginning. And once again, I'm reiterating, if a peripheral nystagmus does not agree or if it's. If it deviates from the peripheral characteristic, it has to be central, unimproved, otherwise. So, yes, if that nystagmus persists, if that nystagmus is not confirming to a typical BPPV yet you pick up a nystagmus, then that would be an indication for a scan, neurological. But if it's just for a few beats, you know, 10 seconds, 15 seconds, then I don't think it's clinically significant.

Perfect. Let's move on. Okay. Using DNG, what is considered an insignificant nystagmus degree, for horizontal and vertical nystagmus. Okay.

For vestibular nystagmus, I would say anything from, let's say, three to five degrees or more. Now, three to five degrees are very, very tiny nystagmus, and they are usually seen best with vanguard, because otherwise the visual system compensates for it. If a system is vestibular, system is uncompensated, and the person is very symptomatic, you will get ten to 15 to even 20 degrees of nystagmus. So I would say, as a rule of thumb, and I see this in plenty of head injury patients, very, very central. The system stabilizes around about three to five degrees horizontal.

In vertical, it can be anything. Even, remember, even three or four beats of one to two degrees. Very subtle. Nystagmus is important. Important.

Not upbeat. It can be normal, but downbeat, even three or four bits is important.

Fantastic. Okay, then, Sausan, Professor Dasgupta answered to all your questions. So would you. Okay, now let's go back to the quiz. Let's.

No one has answered the quiz.

Okay. Can I share my screen again? Is my screen showing? Yeah, sure. I'll just go to the two questions.

Yes. This is very interesting. That's why I put it in. Right. Okay.

The first question is. First question is, what are the instances where you can have a vertical listing generated by the vestibular system? I was asked this in an international conference, and because I wasn't expecting it, I was taken aback. So my mind worked at a supersonic max speed. And then there are.

And it's a very, very good question. So the first important condition, which you will think where a vestibular pathology will generate a vertical nystagmus is your very classical BPPV. So in case of your posterior canal, you'll get an upbeat. In case of a common crux anterior canal, you get a downbeat. The next one is, which I showed you in the slides.

This was, this is when you get a pulsatile downbeat, which is coming from the. Which is coming from a semicircular canal. Dehiscence. The third one is also quite interesting. When you do a head thrust, we hit or clinical.

You know, you do vertical head thrust, don't you? What do you think your results show you in the vhit gear? It shows you shakades if it's abnormal. Now, these shakals are the horizontal and vertical, obviously they are vertical. And you can pick up these vertical saccades when you do it clinically without any vng.

In fact, I would strongly recommend that all of you please perform a clinical head impulse test in the vertical canals in the ral pen. Larp. So that's a third instance where you might get a peripheral vestibule, even kind of a vertical nystagmus, in case of a vestibular nystagmus. So there are instances where you can get vertical nystagmus when the system is generated, when the problem is generated by the vestibular problem. So that's the first one.

The next one is very interesting. Right? Have a look. I'll take you through each individual eye movement, spontaneous gaze upbeat, clear, beautiful. Upbeat, spontaneous.

Upbeat. Doesn't depend, do not depend on gaze.

Gaze evoked, right. Huge excursions, beating to the right. So gaze dependent, gaze evoked nystagmus. Look at the sclera. Naked eye, look at the sclera, come back.

Rebound, upbeat is gone. So you've got upbeat, you have got gen, you have got rebound, and you can see that there's a slight torsion as well. That's a torsion. So now you get a torsional component.

Other side, gaze. Once again, huge direction changing. Gaze evoked, rebound, upbeat is gone.

So, gen, rebound, upbeat, torsion.

Okay, just random movements is not following my head shake.

Horizontal nystagmus persists. That's vestibular, vertical horizontal nystagmus persists. Ocular counter rolling on the right, hardly anything on the left. Yes, there is ocular counter rolling, but you see it's broken down. So I call this saccadic ocular counter rolling.

And we have got a nice series which we intend to publish at some point. So gaze evoked, rebound, upbeat torsion, positive horizontal head shake. Head heavy is okay. Ocular counter rolling, hardly any static. Counter rolling on the right.

Usually bilateral vestibular weakness as quantified with vhit, no wimps. So you get a lesion which has given you loads of central signs and some peripheral signs with the head shake and everything else. So likely lesion, you marry everything. The only place which this can be unified together is the vestibular cerebellar pathway, which is bordering the brainstem. The vestibular system sends out plenty of reference to the vestibular vermis, vestigial nucleus everywhere.

So what can be the etiology? You have identified the site. What can be the etiology? It can be a tumor, it can be a demyelination, it can be a hereditary atarcic syndrome. So hearing loss here becomes crucial.

What syndrome generates this kind of picture? We send the kid, 16 year old, young adult. We send a young adult for genetic typing. And this was spinocerevular ataxia, which explains everything. So just by looking into the eyes, you have located the exact lesion by these different eye movements.

And that is what the beauty of gazing into your patient's eyes. I think that kind of concludes my talk, and I'm sure that you have now been inspired. And I have two more questions. Professor? Yes.

Okay, fantastic. So I can go on all night if you want.

Thank you. So, upbeat nystagmus observed in spontaneous nystagmus positioning and positional test in DNG and low gain with phase leads in rotary chair is it indicates central or peripheral? Once again, you know, you have to take everything into account. So if your tests, you know, if your rotatory chair phase gain and everything else sha, it does not follow a peripheral pattern. It has to be central.

So I can't tell you unless I actually see the raw trace. You'll have to make out whether this trace or whether this actually what you're finding is peripheral. Because as I told you before, peripheral has got definite, certain defined characteristics. And if it's not peripheral, it has to come from somewhere. And by default, is usually central.

But I agree with you. Sometimes rotary chair tests, they do generate this non characteristic features, in which case they are central. So do not ignore them. For

example, the cows bit, which I told you in calorics. So if you see there's an inversion or a reversal of caloric, please do not say that.

Please do not believe or infer that this is clinically inexplicable or not significant. It is central. Same thing happens in auditory chair as well.

Okay, then, can vestibular compensation generate perverted head shake? Nystagmus? No. If it's an isolated vestibular lesion, then peripheral vestibular lesion, when it's compensated, will not generate a perverted head shake. However, if this peripheral vestibular lesion, like the one which I showed you just now, if it coexists with the neurological disorder, then of course, vestibular compensation or not, the perverted head shake will remain.

But isolated peripheral vestibular disorder in the compensatory phase should not generate a perverted headache. Why would it do that? You know, if it. If you do see one. So your original diagnosis is a peripheral vestibular hypofunction.

And then once it's compensated, no symptoms or hardly any symptoms, you pick up a perverted head shake. Very, very importantly, you'll have to scan that patient. It's not coming from the peripheral system. It's central. And who knows?

That might unearth some problem which was not there when you first saw him. Highly possible. So that's how you have to keep your eyes and mind open. It does happen. Science can evolve.

This is a fantastic question. Science can evolve. You might not find it today, but you might find it later on. So always we'll have to keep. And the other thing is, when you are looking into eye movements and quantifying all these things, there is no shortcut.

You'll have to do everything every time. You can't say, I've done this before. You know, I have seen downbeat nystagmus evolving in head injuries and in demyelination. First time, no downbeats, nothing. Six months, one year, downbeats have evolved.

And the other way around. There were downbeats before because of central cortical damage or because of central brainstem damage. But after about a year, with good compensation, the downbeat disappears. How would I know unless I check them? So you'll have to do everything.

No shortcut, professor. But Sheila. Sheila said I would thought that is possible for vertical head shaking. Generate horizontal nystagmus. Illuminatoral peripheral hip function.

Are you saying this is incorrect? No, no. Peripheral unilateral peripheral infarction. The. When you say infarction, I might ask you, do you mean an infarction of the vestibular cochlear artery or the anterior inferior cerebral artery or you are calling infarction of the inner ear labyrinth?

I need to know that. What does infarction mean? Because infarction, by definition, means that it's a lack of blood supply leading to ischemic changes. What exactly do you mean? Do you mean a simple vestibular damage or an infarction of the blood supply?

I need to know that, professor. Sheila is a shame. I mean labyrinth. Labyrinth. So what you have been taught is horizon.

Sorry. So what has she been taught? It's a horizontal vertical head shake generating horizontal nystagmus. Yes. Well, a vertical headshake generating a horizontal nystagmus is perverted headshake.

CONTINUED

It has to come from central. It has to come from central. It does not come only from the periphery. So with what you have been taught. Sorry, I do not know.

But that is how logic and rationally works. Okay, so maybe. Maybe. Maybe. Yeah, maybe.

The studies which have done, they have really not looked into the central nervous system in detail. They were only following vestibular compensation and they picked this up. And that's my point. That if you do see, in a peripheral vestibular lesion, a head shake becomes a vertical head shake generating horizontal nystagmus. You need to think, is there a problem in the central nervous system?

That's what I'm telling you. Fantastic. I think that makes a lot of sense. Yeah. Okay.

Thank you, professor. Then I keep getting compliments. Thank you. Excellent motivating. Thank you for this excellent presentation and especially for all the videos.

Oh, you liked it? End of question. So we are finished, yeah. No, one more question. Professor Dasgupta, what is the maximum duration of acute vestibular neurotrace?

nystagmus ah, yeah. So 20 degrees. Up to 20. Up to 30 degrees. Huge excursions in acute vestibular Nyx.

You know, in my experience, I have seen it for at least 30 to 40 to 50 seconds. But after 50 seconds, something happens, and the nystagmus loses some of its amplitude. But, yeah, it can go on. It can go on for some time. Okay.

Because that's when the brain is at a very, very delicate position. It doesn't know what to do. But over a period of time, the nystagmus starts to fatigue, and the patient starts to feel better. Okay. And I'll just like to tell.

I'll just like to tell Sheila as well that, you know, if she can please give me some references where it says very categorically that investibular compensation, a horizontal head shake generates a perverted nystagmus or the other way around, I'll be very happy, because, look, I don't know everything. I give you that. So I learned something. If you can please furnish me some details with that case, I'm going to talk to the author and have a constructive debate about it. Yeah, absolutely.

I'm unopened about this. Okay. So, Sheila, if you will do. She said, sheila, if you want to email, so academia inventis. Okay.

With the references, I can put you in contact directly with Professor Dasgupta. Okay. Just send me an email. Okay. Thank you so much.

Okay, professor, no more questions. Okay? This is the end, the real end of this fantastic series. Very, very inspiring. And let me say one more time that we really would love to have you as speaker for future educational sessions.

Thank you. Thank you. I'll be delighted. And thank you for being here and not getting bored. That's all I can say.

No, absolutely. Thank you so much. Okay, we have a long time. Yeah. See you soon.

See you. Bye bye, everybody. Bye bye. It.

continued