Etiology of Vestibular/Balance Disorders in Children

Presenter: Soumit Dasgupta, MD

Welcome to today's course, the Etiology of Vestibular Balance Disorders in Children.

These are the risks and benefits associated with today's course. This will be available in the handout if you would like to review it at a later time and these are the learner outcomes that are also associated with today's course. This is also available in the handout and you can look through what you're going to learn today if you want to, in greater detail in the handout. And at this time I'm going to turn the presentation over to the presenter. Okay, I think we can start.

Welcome everyone and thank you so much for joining us today. It's always a pleasure to have you here for another Inventis Academia webinar and let me say that we deeply appreciate your engagement. Today I have the privilege of introducing Professor Sami Dasgupta, who truly needs no introduction. However, for those who may be less familiar with his outstanding career, Professor Dasgupta serves as a consultant, odovestibular physician, neurologist and clinical lead in Pediatric Audiology at Alderheigh Children's NHS Foundation Trust in Liverpool. His expertise and contributions to the field of vestibular and pediatric cardiology are widely recognized, let me say, making him a leading authority in this domain.

I'm also pleased to announce that Professor Dasgupta will be delivered in a total of three webinars for academia this year. Today's session is just the first in the series, with the next two scheduled for March 13 and April 16. Before we begin, Professor Dasgupta will be introduced by his colleague and friend, Dr. Amy Boley, who is eager to share a few words about his work and contribution to the field. Today's lectures, titled Etiology, Including a Genetic Disorder Causing Vestibular Hypofunction in Children, will provide a comprehensive overview of of the causes of vestibular disorders in children, emphasizing how understanding gender line etiology is crucial for effective management.

As always, I encourage you to ask your questions at the end of the webinar and please remember to fill out the survey we'll send afterward. Your feedback. Help us continue to provide high quality educational

content. Amy, the stage is yours. Enjoy the webinar and see you later.

Thank you Anna. And thank you Inventus for inviting both myself and Prof. Subit Dasgupta to be here today. So Anna's given a great introduction for Prof. Sumat Das Gupta, but I just wanted to add a few personal notes.

Working with Prof. Is wonderful watching him work with both the children and adults in the vestibular field which is a very difficult but passionate area for him. He has done a lot of groundbreaking work, especially in older Hay Children's Hospital, changing those children's lives. Truly a great friend and great person, but unfortunately supports Man United, which is a bit of a problem because I'm a Liverpool supporter. So I'll hand the stage across to you.

Prof. And everyone enjoy this session on vestibular and balance disorders in children.

Thank you very much, Anna and Inventis for inviting me as usual. I've been doing this for a few years now and it is a fantastic initiative from Inventis academic academia point of view to do all these lectures and reaching out far corners of the world. And I can see nearly 130 attendees today, all waiting to delve into this fascinating world. And thank you, Amy, my comrade in arms. We run clinics together.

She's always been very kind to me and she is a legal authority in her own field. We have done some wonderful work and we continue to do such work. So today the first series of three lectures is on etiology of vestibular balance disorders in children. Why does it merit a completely separate lecture? Will become apparent because there is somewhat of a trend, at least there was a trend that children will share the same pathologies as adults as far as the vestibular system is concerned.

But this is not the case. The way children present with vestibular weakness or vestibular disorder, for that example, any balance disorder is different from adults. So just extrapolating adult kind of findings

and adult caveats to children is not the right thing to do. And that's why this deserves, on its own merit, a complete lecture that Inventis has very kindly arranged.

So before I begin the real lecture, some fundamental basics, how children differ from adults. So we need to specially consider children as a different group of cohort. Many of you, no doubt, just like me when I started my career, would see adults and children. And in those instances, at least, I can talk from my side. The mindset to approach an adult is completely different from a mindset to approach children.

And this for the simple reason children will not communicate the same way as adults would. And this is important because, remember when we trained, we were told one thing, that history, what the patient tells you subjectively is absolutely supreme in making a vestibular diagnosis. Now, instances where you don't have someone talking to you and describing the different features, this becomes very difficult. So, so the two groups are completely different. And switching from the adult mindset to the child mindset, when you're interviewing a child is quite different.

You have to keep this in mind when you are approaching a child. In other words, you just can't have the same practice in children as you would have in adults. So as I said, history might not be forthcoming. So you'll have to depend heavily on the parents or the carers, whoever are accompanying the children. You'll have to glean out vestibular behavior, for example.

I'll give you an example. You know, a child will not tell you that I'm dizzy, I'm spinning, I'm vertiginous. In many instances, I find this word being used. Lectures being done on pediatric vertigo. Now, you can call it an adult vertigo because adults can narrate what a vertigo is.

Vertigo is a symptom, but a child, even I've seen older teenagers, they cannot describe the way they feel. So vertigo in children cannot be qualified. Therefore, the term pediatric vertigo should be slowly

phased out. But this is frequently used. This is just an example.

So we need to ask the parents. And sometimes when the parents are not forthcoming, you have to improvise questions. I remember I was sent a patient. I was sent a child 7 or 8 years old, with problems in balance. No dizziness, no lightheadedness history, nothing from either the parent or the child was sent to me.

And yes, conventional questions. There was really no affirmative answer. But then I asked the mum what happens when he goes to a playground. And mom said that when he comes down on a slide, when he lands on his bottom after coming down on a slide, he is unable to pick himself up and has a dazed expression on his face. Now, that dazed expression actually equates to dizziness.

And that gave me my vital clue that this could still be vestibular. There are millions of questions like this. And these questions come to you very instinctively and reflexively when you start interviewing a child. Will you ever ask an adult this question? What happens when you go to, you know, go to the.

Go to the fair? Will you ever ask them? No. So once again, it's very important that you have a different mindset and doing vestibular stuff, that is talking to the children, assessing them, formulating management guidelines. Everything takes lots and lots of improvisation.

Fun. It can be fun. You'll have to physically get down to the level of kids. For example, today itself, I had a couple of autistic kids who refused to cooperate. I had to go down to their level and lo and behold, we could manage to get the entire spectrum of vestibular function test objective, by which they not only wore the goggles, they Also did the vs, they did everything.

So it's just a matter of getting into the child's psyche. And for that you do need pediatric training. Not just see a few kids in your practice. You need pediatric training at the ground level. And we have got

that training.

Therefore we do have that insight to actually understand the child and what the child's carer is thinking. Just like audiology, you might need a second distractor. And the test that you will choose will depend on the age of the child. That is the same as audiology. For example, you would do a VRA only in a group of children up to a certain age.

And pure tone and performance similar with vestibular function tests as well. It depends pretty much on the development of the child. For example, under the age of two years you wouldn't be able to do a conventional headband, video head impulse test. You can only do the remote control, which inventors has in the synapses we hit, which was before. So yeah, you have to tailor your tests based.

But you'll get plenty of information anyway, whatever tests you do. And the next point is absolutely crucial. I have seen many publications where when they are doing a cohort of children, let's say they have got some vemp values, they compare it with adult established values. That's not the right thing to do. Because remember, a child's vestibular system is developing, it's in a state of flux.

It's a dynamic development. So vestibular function achieves maturity over a period of time. So obviously the norms will change and the norms will not be reflected in adults until a certain age. So for vestibular function tests, you need to get your own norms in your own laboratory by doing a group of normal healthy children. You know, the minimum, the minimum number of children you need, the power would be about 40 or so.

But anything between 30 to 40, you need to get your own norms across an age range. That's the first thing you need to do when you are checking children and the presentation of the phenotypes, the etiology, they are different because they, as I said, they, they kind of follow a different natural history. We should not limit ourselves just to find out a diagnosis what is going on? What is the vestibular

weakness? But we should also ask ourselves a singularly most important question.

Why is the child symptomatic? Because you do know that a child's brain is extremely plastic with a lot of activity as a result, vestibular weakness, even up to moderate vestibular weaknesses, sometimes even knocked out vestibular system, they compensate brilliantly, and there are no symptoms. The child presenting with a symptom means there is something going on with decompensation. And my job and your job will be to figure out why the child is decompensating. It can be a medical cause, it can be a simple thing like vitamin D deficiency, anemia, whatever, or it can be a mental cause, for example, anxiety, stress, a psychiatric problem, a psychological problem.

And that's why you need all this training to go to different pediatric disciplines and find out what's going on. And you'll have to know a lot of neurology, you know, because neurology and neurotology are very, very closely linked. And most of my patients are referred by my neurology colleagues because that's where they first end up. You need a very good and solid grounding of Neuroanatomy 1 and Neurophysiology. And if you know the basic premise that, look, a child's brain is a developing brain, it's highly metabolically active.

Neurotransmitters and neurosynaptic junctions are accelerated or their formations are accelerated as compared to adults, right. As a result of which, if things go wrong, the manifestation will also be different because in adults it's at a steady state. So you need to know neurophysiology, neuroanatomy, and you need a very holistic approach and as I mentioned, a very good knowledge in different branches of pediatrics. Ideally, I would recommend you spend some time in these departments. Development, pediatrics, pediatric, ophthalmology, pediatric rheumatology, pediatric oncology, cancer, for example, pediatric metabolic disorders, pediatric endocrines, pediatric A and E.

You know, if a child presents to you with acute symptoms, how to approach the child, this will only enlarge your, enlighten you and enlarge your horizon to do what you need to do as far as vestibular

system is concerned. Because remember, vestibular system is all pervading it. It encroaches on pretty much every individual developmental aspect of the child. So, and this awareness we are trying to raise throughout the world that it's just not balanced, no, no way. It takes part in your autonomic control, vasomoto tone.

There is a connection from the vestibular nuclei all the way to medulla blancata. So Iwasamoto toe number one, number two is cognitive development, number three, overall development, muscular activity. Everything is encroached by the vestibular system. It's probably the most complex and most elaborate organ in the whole body. Due respect to all my other medical colleagues who would think otherwise, but to me it is one of the most complex and most intricate organs in the whole body, more so in a child.

And the reward outcome is very rewarding. If you can manage to do a correct diagnosis, find out a cause, then the treatment has got very rewarding outcome, because treatment is basically based on symptom relief and improving your compensation, which means you're addressing the brain. So you are trying to establish new neuronal circuitry and synaptic connections with rehabilitation, which will address the decompensation. Decompensation that is going on. So you need the training.

Absolutely. And this is very, very true, that one third of children with hearing loss will also present with the vestibular problem, hand upon heart. How many of us actually check vestibular system in a child with a hearing loss? But it should be done in all cases. And I was just writing a paper and you know something?

This one third of children with hearing loss presenting with a vestibular deficit was identified a very long time back in 1930 by a very famous otologist, Shambo. So for the first time he said a third of children with hearing losses will have vestibular deficits. At that time, the only test available for children was the caloric test, and that too through Eng, not VNG. So what he discovered in 1930, 100 years later, recent

studies have also replicated him. So these are what I call absolutely seminal discoveries.

So before we delve into etiology proper, a brief look into the development of the vestibular system. So the vestibular system anatomically starts forming at the age of 6th week in utero, and is fully developed at the age of 21 weeks. However, they are not functional, which throws up vast possibilities. Are they really not functional? Because look what happens in utero.

There's an amniotic fluid, an amniotic sac, where the fetus literally floats. There is hardly any gravity, but there is some maternal gravity. So does the vestibular system prime itself in utero? We do not know that, because I have looked a lot and I haven't found any studies. However, just for the time being, understand that the vestibular complex is fully formed.

The synapses and neural connections are formed, but they are very basic formation. The labyrinth becomes completely ossified. And yes, there is evidence that otoconal mechanotransduction has already formed. But canals, no. So just what I told you just now in when I talking to about the amniotic fluid and the way the kit floats with almost no gravity.

Autoconal function is established before semicircular canal function quite naturally, because otonia, if you look into the phylogene and the whole thing, out of the five organs, the two organs, cul and sacu, they are the most important vestibular organs because gravity is ubiquitous. That's how vestibular system evolved. And this at this time, during this entire period in utero, they are. The vestibular system is heavily, heavily influenced by genetically controlled mechanisms. And 38 genes exclusively responsible for vestibular development have been identified.

So this is a very important point, that this is a genetically controlled vestibular system. Development after birth what happens? First dawn of light, the child cries, takes a gush full of air, and then suddenly thrown into this vast infinity called space. And the child needs to know where he is at this point of time.

And please forgive me if I referred the child as a he.

Now, in this age of political correctness, it should really be they, but I know this very well that he can be used from both male and a female angle. So after birth, what happens is the vestibular system starts to become primed with environmental stimulation. And that's very, very important. So the vestibular system learns. So in the beginning, the vestibular system in its learning, obviously it wouldn't behave the same way as it would it would in an 18 year old, a young person, it's primed.

And this priming takes time. And this development is a gradual process. That's why you get different stages of balance. A child can't walk the moment he's born, that takes time. He can't stand the moment he's born, that takes time.

And that because of vestibular system and other inputs coming in, that is the brain and everything else. So this is the period, remember, of stimulus control development. And it's this time there's the most heavy expression of neurotransmitters, of synaptic connections, of neurochemicals. Remember these, that this is the time when there is an explosion of all these different physiological processes and that if they go wrong, will have an effect on the child with a vestibular disorder. And this is a very important physiological difference between the adult and the child, which explains pretty much everything.

And you will appreciate this as I go along.

So this whole chart list, I am doing it here for the second time. I have compiled this and the literature is given below as to how does the physiology differ and why is it important to know? Because, you know, physiology is normal. Right. Anything which is not normal is pathology, which is disease.

So unless you know the normal, it's impossible to know what is not normal. So first thing is, we understand the normal. So as you can see, a child's vestibular system, the brain, in fact the whole

body, has got a very high cellular metabolism and a high mitotic rate. You know, why do we start from a very tiny human being to a 6ft 5 strapping person? We have grown.

So this has to be mitotic activity all the time. But in a child in the developing stage, it's a high mitotic rate with a high energy demand. What does it mean in etiological terms? Etiological terms, what it means is because of this, because of high metabolic. The classical viral infection that you get in vestibular neuritis does not happen that often because it's just the sheer metabolic metabolic activity which does not let the virus to colonize.

As a result, acute vestibular neuritis, the ubiquitous, one of the commonest causes of vestibular problem in an adult, you know, acute vestibular neuritis, which you all are very well aware of, that hardly happens in children. A children might still present with an acute vestibular event, which is a sudden dizziness, just like acute vestibular neuritis in adults. But that's usually due to a secondary cause and not the classical viral neuritis that you would get in adults. And there's a continuous crosstalk between metabolism and immune system. So it's almost like the metabolic processes, they talk to the immune system that, look, I am here, you need to be my friend and accompany me.

And the immune system says the same thing. As a result, because of high demands and high kind of activity, both of them talk to each other and prevent colony colonizations with a virus and all these things. Even bacterial colonization is not very common unless complicated with meningitis and all these things. I mean, septismic bacterial colonization of the labyrinth in a child is not as com as as much as in adults. And this, this time the immune system is also prime.

So it gradually builds up, builds memory. All of these prevent B festival and neuritis of the viral nature. Very importantly, now it's well established that the otoconia are anchored to the gelatinous layer with proteins which are genetically expressed. One very important protein is otoconin. And now they are seeing that otoconin levels can be measured in blood in adults, in bbb, but that's a different thing

altogether.

So otoconal anchorage proteins, they slowly start to mature in childhood. What does it mean? It means that they're very strong and robust, and after a certain period of time, from the age of maybe 40, 45, they start to degenerate and generates BPPV. However, in children, BPPV is practically impossible or almost unheard of. It can still happen.

Not absolutely absent, but when it happens, it's usually post traumatic. The striavascular is a spiral ligament which secretes endolymphatic fluid actively. They are also very highly metabolically active, as a result of which, your classical menier disease, which is idiopathic in adults, is hardly found in children. If it's meniers like the disease, it's usually a syndrome due to a secondary cause. When you are hit on the head, not you.

Generally, when someone is hit on the head, the way the skull reacts to that blow or head trauma is quite different in adults than in children, because in children there is the different cerebral perfusion, the anatomy is developing, the skull bones are not as hard as adults, and intracranial pressure is quick to change as a result. A child with a head injury, there is an advantage. There is a disadvantage. The advantage is the skull bones are very, very kind of. They rebound, they are plastic, so they act as some kind of cushion.

But on the other hand, because of intra. Intracranial changes, it can be far more serious than in an adult. And once again, we come back to the synaptic and neuron circuitry which is continuously happening. Why do you think vestibular migraine is so common in children? It's because it's such a high activity, activity with synaptic connections and with neurotransmitter expression that if things go wrong or if they become haphazard or random, they will generate a vestibular migraine.

Focus. So already, you know, three important things in children as far as etiology concerned. One, vestibular neuritis of the viral type is very uncommon. Two, BPPV is very uncommon. Three, many diseases very uncommon.

And four, vestibular migraine is the commonest. So already, by just noting the mechanism and the difference between child and adult physiology, you know 50% of the etiology. And because you know why it happens, you'll never forget this, right? So that is. That is why I have prepared this slide for you guys.

So now let's delve into hardcore etiology, pediatric vestibular disorders. Now, these are different studies who have used different kinds of equipment to come to the conclusion. For example, O'Reilly from Wilmington, Delaware, 2007 is one of the Pioneers of vestibular pediatric vestibular science, as is Silver tweener Vacher in 2008. And then came the VHIT. So different kinds of categorizations are there, but as you can see, peripheral vestibular disorder and migraine and BBBC are the commonest of which vestibular migraine is the commonest.

We published in 2017 but our at that time we just started. So now it's. Now our finding has changed because now we all believe vestibular migraine is the commonest cause of pediatric vestibular disorder. It can be central, it can be peripheral. We still don't know.

Periphery can be affected, but usually it's a central nervous system which is affected in vestibular migraine. So there are loads of other conditions, for example third windows, central neurological disorders, genetic, as I said, some we do not find out a reason. But in the vast majority of vestibular weaknesses in children, we do find out that there is a problem or an etiology, because etiology is king. Etiology tells you quite a lot about future natural history of the disease, which will enable you to formulate your management guidelines and protocols and also counsel the parents as to what can go on in the future. So if you do not know what you are dealing with, then you are stuck.

There are many disorders, especially in adults, where you do not find a reason to there you treat that person empirically. In other words, you get rid of the symptoms without knowing what's causing the symptoms, which is not the ideal way of treating someone. So Decorah in 2007 this first published this beautiful etiological paper, different causes. I'm not going to read them off, but as you can see, quite a few of them which we successfully replicated with the VHIT in 2017. And the incidence is not something which is infrequent.

And they can create a lot of morbidity in children develop, involving or influencing their development and overall cognitive behavior and everything else. 35% of pediatric balance disorders, not the majority, but a significant chunk by balance I mean balance, not vestibular. So you know things like falls, trips. Balance could be due to orthopedic problems, cardiological problems, ophthalmological problems. About a third of pediatric balance disorders are due to a problem in the vestibular system.

So let's start with congenital or genetic vestibular disorders. So as you know that a labyrinth might not be formed properly. So some kind of assault damage could be alcohol, could be chicken pox in utero, in the first trimester will Lead to an arrest of development of the inner ear labyrinth, cochlea and vestibule. You get different combinations and that's what we are seeing here. These are the ones which I have seen.

I have seen complete aplasia, no labyrinth, no cochlea, no vestibular system, nothing. Just a hole. That's called a Michelle Sea, by the way. Different kind of cochlear deformities, third windows, dysplastic lateral semicircular canals or posterior semicircular canals. Dysplastic utricul and secure.

Arnold Chiari is a structural deformity where the cerebellum herniates by about more than 5 millimeter through the foramen magnum all the way to the spine track, causing traction of the vestibulocochlear nerve bundle, generating a vestibular hypofunction from the nervous origin, not from the inner ear

labyrinth origin. So structural deformities, that's a broad term for this. And this is the stage of development of the otocyst, that is the labyrinth or the cochlear vestibular organ. First six to eight weeks is the most difficult time. Now, the structural deformity, if it's not due to a maternal substance abuse or alcohol or an infection, it can also happen because of genetic mutations.

And these are highly, highly heterogeneous. They almost invariably always affect the vestibule and the cochlea. Varieties of presentations and syndromes, quite a few of them are autosomal dominant. There are some which are autosomal recessive and the autosomal dominance are usually post lingual. Now you might ask, this is a little bit contradictory because at the one hand I'm saying that the cochlea is not formed yet.

They have got good speech. It's because these losses can be progressive in nature that a child develops speech. But then the hearing loss problem process and you scan the year and you see all these different kinds of deformities.

So when you get a genetic mutation, but it's only limited to the inner ear, you get different kinds of. When it's not limited to the inner ear but to different body organs, we call it a syndrome. Right? So these are all the syndromic cochlear vestibular structural deformities. I'm sure you have seen quite a few of them and the gene is given on the side.

So again, on the one hand you get a bor branchio to renal spectrum, you get a charge, you get a golden heart, which with gene we do not know it's a clinical diagnosis. You can get a pendride with a thyroid problem. Different kind of chromosomal abnormalities. Trisomy 21 is Downs Noonan is male. Turner Turner is XO Wilder Wank another genetic syndrome and craniofacial syndromes with typical craniofacial features apart Crusoe Pfeiffer Schaetrich.

Now Schatz Schen is a very interesting condition with a twist one mutation. And I need to tell you about this because you see you will get instances in third windows. That's one of the diagnostic criteria. Dehiscence enlarge vestibular aqueduct where you get a conductive airborne gap with normal middle ear. Otoscopy is normal, completely normal.

Plus tympanometry is completely normal. So it is an inner ear problem which is manifesting as a conductive hearing loss shin. What happens is the vestibular cochlear apparatus is not very well formed but there is a sizable conductive earborne gap because the inner the middle ear ligaments are not very formed. Not the ossicles, not anything else. The ligaments are not very well formed which prevents the oicles from moving properly.

So this is another instance where you'll get normal tympanometry, normal mid layer in the presence of a conductive airborne gap. So remember two conditions and two conditions only. Third windows and Schatz Hun which can present with normal middle ear but with a conductive hearing loss gap. Now obviously third windows do not present with craniofacial deformity. Sch does.

So if you see a craniofacial deformity with conductive hearing loss where you get a normal yellow look. Glue here is very common in in all these craniofacial syndromes because of the way the official skeletons form. But you have eliminated glue by proving there is no glue with a normal tip but craniofacial then either this kid could be having semicircular canal de facial nerve canal dehiscence, carotid artery canal dehiscences or it could be a SH3 surgeon and recently we have actually isolated and we have discovered one. So that's why this knowledge is very fresh within me and it's a very interesting condition shin. Next we come to syndromic genetic hearing losses.

But the cochlear vestibular system is completely normal from the structural point of view. So these are Yang and Nielsen which is a potassium genopathy Alpo al and all this including Usher 2 the Ataxia Group, all these ataxic syndromes spinoceretaxia episodic ataxia, the autosomal recessive ataxia of

Sharro Savini big name but we call it Aracas. They present with cerebellar ataxia and with vestibular hyperfunction. Children do not get canvas which you get in adults but you can get this ataxic group and the hereditary sensory motor neuropathy group of which the prototype is Charcot Mari tooth. Here when you scan the ears, the ears are completely pristine and beautiful looking.

Then we come to the non syndromic variety where the problem is only in the inner ear. And you don't have to remember this but remember quite a few genes have been identified which affect not only the cochlea but also the vestibular system. A third I would say roughly. So these are the different genes and these are the different genes which I have seen in my clinics. I haven't seen a DFNA 28 but the others I have.

And indeed this is because of the new genomic service which NHS has recently introduced. So we are in a position to check for all these genes. There are some non syndromic genes where you do get some kind of a structural deformity in the vestibule and that is the DFNB4 which also presents with DVA which is part which which could be a pen red and you get a very classical X linked gusha which presents with so many different cochlear vestibular deformities. So that's an X linked genetic condition and quite a few other genetic condition which presents with vestibular problems. Familial Meniere's disease, motion sickness, X linked hypophosphatemic rickets, Von Hippel Lindau syndrome.

If you want to know more about this just type my name and type these conditions. Von Hippel Lindau Metabolic Accelerator we have done, we have published on this Deadmoid is an interesting condition. It's also called the Wolfram zg, the Wolfram syndrome because of the genetic mutation in the Wolfram gene. So DI stands for diabetes insipidus, DM stands for diabetes mellitus, OA stands for optic atrophy and D for deafness. Very tragic condition.

Longevity up to 40 years and they're usually mid teenage manifestations I have diagnosed and it's gutting and devastating to tell the young person all these things might happen in the future. That's

another thing why you need to know the etiology you need to tell your patient and if you patient doesn't understand you need to tell the carers there are familial menas disease, but thankfully they manifest much later. They do not manifest at a very early age. And some genetic mutations have been found for vestibular migraine which is also associated with cerebellar ataxia, for example, the CAC NA1 gene. We have seen this as well.

It's not, it is uncommon, but it can happen. So how do these children present congenital disorders? Now obviously because the vestibular system strongly influences motor development. There is a motor developmental delay that's a no brainer. A third of them will present with the hearing loss, a third of hearing loss present with vestibular loss.

And usually these are almost always bilaterally symmetrical vestibular hypofunction. You might get unilateral in some instances, for example the pen red or the bor, but usually they are bilateral. And if they're bilateral, their main phenotype or their main presentation is not a disorientation in space, but unsteadiness and an ataxia. Difficult moving incoordination and navigational difficulties. My child drops things, my child bumps into door frames.

My child cannot resolve space. He can't look down when he's on the top of the skeletal navigation is strongly influenced by semicircular canal and otolith input at the level of the entorhinal cortex. This is very, very important piece of history navigational difficulties and they overlap strongly with sensory processing. For example, children with autism and ADHD will also have navigational difficulties. And today and Safa, she's here, my resident, we have seen a case with navigational difficulties with central eye movement problem.

So the navigating navigational difficulties are because of central vestibular integration as well as deficit at the periphery cognitive delay. We said that very important behavioral difficulties that has an effect on the psychology of the patient. Everybody is going on the trampoline. My child can't and that has got a

psychological back defect. You need to tell them why he can't go on the trampoline.

They present with different syndromic stigmata and usually bilateral vestibular diagnostic markers but also central vestibular diagnostic markers. Vestibular system vestibular weakness can lead to learning problems and yes of course because to vestibular spinal reflexes they control or modulate muscle tone. They can lead to muscle weakness, my children and they end up with neurology with proximal myopathy like syndromes and it's actually a vestibular problem. We have got quite a few of these cases. So the first example of a congenital disorder.

So this child with a late onset mixed hearing loss, motor developmental delay, neurodevelopmental issues in coordination etc, very apprehensive, doesn't have much of a life learning deficits and rehabilitation changed. Now as you can see here, this child has got a very clear unilateral vestibular hypofunction as you can see with the V and vhiddy. And when we scan you see this Christmas tree appearance of the cochlea without any modulus and semicircular canal dysplasia. This was classical of X linked gusher. Now this is one of the.

We have got two kids like this, one of them has got unilateral vestibular hypofunction. One of them was a bilateral vestibular hyper function. Now I asked myself why is this the case? Why is asymmetrical pathological involvement? This is because human body is not symmetrical on either side.

There is, there are minor differences, always will be the differences. So this asymmetry is something which is inherent and involved. So and that's probably because the way one side develops is slightly different from the other. So you can have as I said unilateral involvement but usually they are bilateral. This is the second example.

Child with late development of motor skills and unsteadiness with the hearing loss and there is a motion sickness and fear of heights which is classically autolith, severe vestibular weakness as you can see

WIMP is knocked out sick, semicircular canal hypofunction. Again excellent results. And this child came from the mental health services because this child was a complete recluse. He didn't have a life because he couldn't participate in any playground activity. And let me see if the video plays.

Yeah, that's a classical lateral canal V hit, not a VHIT standard hit impulse test as you can see clearly the catch up seconds on either sides. So this is the second example of congenital disorders. And finally we managed to hone down the gene. This was a MYO7Amutation. The genetic diagnosis came much later because now we have got the facilities.

So there might be many autological conditions with generator vestibular hyperfunction. It could be infective inflammatory coming from the middle ear. Only 1% to be labyrinthitis meningitis, autoimmune disorder due to drugs due to trauma due to third window endolymphatic hydrops, not many years secondary endolymphatic hydrops, secondary BPPV and in syndromes metabolic conditions where There is a lot of hyperviscosity in the blood which leads to a relative ischemia of the end artery. The vestibular cochlear artery is an end artery. If the blood flow is slow and stagnating, then over a period of time this might lead to a chronic ischemia generating a bilateral vestibular hyperfunction.

It can be unilateral too, and that's in diabetes mellitus, thyroid disorders, vitamin D deficiency, electrolyte, mesa, anything can affect the vestibular system. I have diagnosed leukemias coming to my clinic, very low grade leukemia with bilateral vestibular hyperfunction. So remember, blood flow or vascular vestibular weakness in children is something we need to look out for. This is an acute vestibular event. Remember nowadays we do not call it acute vestibular neuritis.

Even in adults. We call it an event because it might not be a neuritis, it might be something else. In other words, it can be a tumor. It's much less common and it's usually secondary to space occupying lesions, autoimmune disorder, bacterial labyrinthitis, ototoxicity, intracranial radiation, etc. And the classical phenotype is characterized by a sudden onset of dizziness without without hearing loss, with

near total recovery.

And then depending on compensation, whether there are recurrent vestibulopathy, which is not common because children compensate extremely well. And OME can generate either an acute phenotype. So typical gluya get giving rise to a typical acute vestibular event. But this is rare, as I said 1% and that's because the endolymphatic fluid ion balance is reversed and then once it goes to a chronic state, so acute vestibular event and then to the decompensation stage. So decompensation can still happen, giving rise to recurrent vestibulopathy.

These are chronic presentation with episodic spells lasting for seconds. They can present with dizziness or provocation movements, short lasting, strongly reliant on visual senses for compensation. So they don't like to go to places with plenty of people or bright lights and fear of heights is possible. We have to find out a cause for decompensation. They can be pretty morbid because the child loses confidence in balance.

However, this is not rare, this is not common, as I said, because cerebral plasticity is very robust in a child.

So lots of inflammatory reactions can occur in the vestibular system. It can be acute or recurrent, they can be bacterial, they can be virus, spirochetes, syphilis, very uncommon. But autoimmune is a very important part because we do unearth autoimmune disorders with the first manifestation of an acute vestibular event. And it's my job to figure out which autoimmune condition. And we have diagnosed rheumatoid arthritis, juvenile rheumatoid arthritis, sle we have diagnosed all sorts.

We have even diagnosed gluten sensitivity. Can you imagine? Gliadin sensitive autoimmune ear disorder. We have even diagnosed that. You just have to keep an open mind and the knowledge that these things can happen.

Lifestyle is severely affected. So yes, you do need to treat them in certain instances. This child with acute imbalance. These are all real cases by the way, all from my series. Child with an acute balance, joint pain, hearing loss almost overnight.

Seen by pediatrics. Hated the light. They thought it was photophobia. Couldn't move the head, had to walk with support, holding on to mom or the nurse did not. So could not look at bright lights, but did not want the lights to be shut off completely because she thought she would fall the moment it's dark.

The balance improved dramatically over a period of three to four days, but hearing did not. So that's when I saw her almost as an emergency. And I found out that one side of the vestibular system is knocked out and the vemp is severely affected on that side too. And when I did this test, what I found was once again a positive head impulse test which I'm showing you now. There we go.

Beautiful, beautiful, beautiful. All inflammatory markers raised. So we did a run of all the inflammatory markers and everything. We treated her with steroids which salvaged the hearing somewhat from moderate to mild. It brought up mild and then we did vestibular rehab with fantastic outcome.

She's back to her normal life and she, touch wood, hasn't decompensated. Eventually she was proven to be juvenile rheumatoid arthritis. Diagnosed from an autologic clinic. This was an infection. So this child was treated with external cancer with proton beam therapy.

Presented with an acute labyrinth like picture. And that was because the inner ear, the cochlear labyrinth system was affected by the proton beam leading to an opportunistic infection. Once again a very acute presentation. And before anything happened she actually this was interesting, accurate presentation. Admitted to the hospital, they did everything.

They couldn't find a reason, then discharged because recovered. However, when she entered a school entry hearing screening at the age of four, she was found to be completely deaf on the left side, no hearing. And that's when she was sent to me and we picked this up. We scanned her labyrinthitis, ossificants, the whole labyrinth was ossified. And this was a head shaped test which I did on her.

As you can see, head shake, shaking the head and you see a beautiful nystigmus which is middle frequency vestibular asymmetry doesn't tell you the side because you're stimulating both sides. So it's a middle frequency vestibular asymmetry. So this was a classical acute vestibular event provoked by previous proton beam therapy. Space occupying lesions can be tumors, can be cysts, can be aneurysms. The commonest CPA tumor is vestibular schwannoma associated with NF2.

But I haven't seen many, to be frank. I have seen meningiomas and lipomas, but I haven't seen that many NF2. And they can be cysts, granulomas, cross. Arterial compression is very important. The internal carotid artery in the posterior fossa is so tortuous that it presses on the vestibulocochlear nerve and you get vestibular paroxysmic spells.

Malignant tumors are in 10%. If the tumor or the cyst or anything bleeds, then you get an acute presentation. Acute vestibular event like picture and the SOL itself, as it grows, it maintains decompensation. It's very important the posterior fossa. We have launched a study with posterior fossa vestibular presentation.

Previously, none of our posterior fossil tumors underwent vestibular examination or investigation, but now they are because we recognize that the vestibulocochlear nerve can be affected by the tumor at any stage. One, when it's growing, two after you remove it because post surgical fibrosis might put attraction on those and three, following radiation, that is cranial traditional radiotherapy or even with a proton beam. And they might present without a hearing loss. That's very interesting. This is an example.

A very young, young boy presented with an acute vestibular event. No balance, no hearing on one side. And you pick up the classical vestibular nystagus, which is Alexander's Nystagmus Grade 3 to begin with. One side of the system was knocked out. And when we im imaged her, imaged him.

This was a big arteriovenous malformation or aneurysm in the posterior fossa which was threatening her lowest. His lower six cranial nerves ended up with emergency stereotactic gamma knife surgery. I met him last week in my canteen. He's a grown up man now and he is doing excellently. There is a problem with swallowing which is still persisting because of Vegas.

But he is doing very well and he absolutely loves me to bits because I've been involved with him for a long time and he's one of my favorite patients.

He presented with paroxysmic spells, very brief lasting spell. And you know what, the way I treated him, he was given quite a few labyrinthine sedatives like cinerazine, proclozine. Nothing was taking it out. But because this is a neuralgic, so it was an irritation of vestibular nerve by the avm, what I did was I put him on an anti neuralgic medication, carbamazepine, which you also use in tribal neuralgia. And his paroxysmic spells was completely gone, disappeared.

So that's why once again let me stress how important is to how important it is to make an etiological diagnosis. Plenty of phototoxic drugs, aminoglycosides, gentamicin is mainly vestibulotoxic rather than cochleotoxic. Remember this, it's not the other way around. But once again, hand upon heart, how many of you have been asked to check the vestibular system following gentamycin therapy? We get hearing requests, but what about vestibular?

Platinum is very important. I see that frequently in my practice because we are a big oncology, oncology department. And of course it can be an acute vestibular event or a gradual buildup of chronic

damage with recurrent vestibulopathy. And it's always bilaterally symmetrical unless there is a unilateral posterior fossa problem which imparts it with an added hearing loss and quite a few susceptibilities. We're trying to identify giant.

We haven't gene, we haven't got around it as yet. Extremes of age, kidney problems, poor nutrition, so and so forth. And remember these kids are very, very ill so they are prone to develop ototoxicity. That's why it's very important you get a, you get a robust autotoxicity protocol in your unit. This is an example.

All six canals knocked out, no autolith response. This was after cis platinum chemotherapy with a COP2. So the hearing loss was limited from 4, 4, 6 and 8 kilohertz to about 40 decibels or 45 decibels. Not a great deal of hearing loss, but the vestibular system and we took him through a whole set of rehabilitation and he regained his balance after the Cessation of chemotherapy. Very, very well.

Trauma, as we said, 80 to 100% of children with a trauma to the head will have some kind of vestibular hyperfunction, but the majority of them will never present with one because the brain compensates. 25% of them might present later when they undergo, when they go to a stage of recurrent vestibulopathy. So it can be a direct blow or it can be acceleration, deceleration, that is a moving head suddenly stopping. They are often missed. Often, often missed.

And there is a strong legal implication because the child who is affected with, and also adults, actually they can demand compensation. And one thing, remember, very important and frequently I had to fight it in a court of law and thankfully I've won so far. The force of the injury is not proportional to the labyrinthine injury. I have seen kids and adults, and Amy knows this very well, with Glasgow coma scale of 3 to 4 or 5 with hardly any vestibular involvement, whereas a Glasgow coma scale of 14 and 15, normal, trivial injury, the vestibular system is knocked out. In fact, I've got two or three kids, very, very tiny, tiny injury, fell and then was dazed, took it, taken to the hospital, nothing wrong, presenting with complete bilateral vestibular hypo function.

So the impact force of the impact is not proportional to. Because this is because of the way the labyrinth, the bony labyrinth is protected, which is hugely variable in the human being. Hugely variable. Your, your labyrinthine bony protection is completely different from mine.

And chronic symptoms or chronic legacies of trauma include quite a few things. As we said, recurrent vestibulopathy, bppv. It can happen, yes. If there's a BPPV in a child, almost invariably it is linked to trauma, which our colleague Jake Brodsky from Boston Sick, he has done a beautiful paper on this. Vestibular migraine is a sequel vestibular processing of central vestibular integration difficulties.

Endoliphytic hydrops after trauma is a legacy. You might get a frank breach of the semicircular canal, which is perineum fistulas, which are third windows. And what is more important is nowadays we are seeing more and more and more patients. In fact, we intend to do a paper. Amy and I isolated otolith damage.

That is the semicircular canals are completely intact, but the otoliths are gone. Previously this was not recognized because previously WIMs were not as advanced as now. But now with VEMS and all other otolith tests, we can say the semicircular canal is completely intact. But the otoliths are damaged and there is a criteria for isolated otolith dysfunction by sue and Morofishi. Good colleagues from Japan and South Korea have clearly delineated the criteria of isolated otolith dysfunction.

Why am I telling you this? If you see any child or an adult even following a head trauma, never forget to perform otolith function tests. If you do only your semicircular canal function test and say vestibular system is normal, that is not the right thing to say. You'll have to do your wims and head heaves and ocular counter rollings and pretty much everything else. Example of a trauma, very, very trivial trauma.

15 out of 15 Glasgow Kumas kid. What? So this kid was trying to reach something from the top of a top of a fireplace at a height, stood on a stool and then fell from the stool and hit the back of his head, dazed a little bit vomiting, came to the hospital completely fit and fiddle. So how did we pick this up? Because he was routinely screened once again in school and found that there is no hearing on the right zero, no hearing and vestibular system, huge saccades.

Look at this. Traumas can spare the anterior semicircular canal. And there is a reason which I'll explain if I've got time. So trauma can spare the anterior semicircular canal. And the only other instance where you find an anterior semicircular canal sparing vestibular problem is ototoxicity.

So these are the two instances where you can get an antic in sparing so they don't affect. So once again this is a classical head shake and out here what you'll see is also we picked up a post traumatic semicircle dehiscence that was the head shake one. So no intervention was needed because there is no recurrent vestibulopathy. There is nothing except situational counseling things to avoid in the future.

Third windows. We have diagnosed more than 100 third windows. Our threshold is extremely low and the hallmark is conductive hearing loss with normal middle ear plus different autolith symptoms like conductive diseases, misophonia, pulsatile, tinnitus, autophony, Tulio's phenomena, Henna's phenomena. But remember, these are all historical, historical kind of narratives which a child might not be able to tell you, but the parents are very, very astute. Parental sensitivity of picking of vestibular weakness is more than 80%.

So always believe the parent or the carer. Final diagnosis by hydration, CT scan and of course the commonest one that you will come across is an enlarged vestibular aqueduct. And because of their oddity of symptoms, you know, a child who child was singing in a choir and suddenly withdrew completely. Why? I don't like the way I sing.

Well, come on, you are being too kind of being a perfectionist. That can't happen. We'll all love your singing. No, I don't want to sing because I hate my own voice. Kept on insisting nothing found, went to quite a few specialisms and then finally ended up with mental health services because she was reacting to it.

And finally they sent her to me and she had a bilateral superior semicircular canal lesions, bilateral, not unilateral, which was surgically closed. And now she's back to singing. So as I said, these are very rewarding. These are different third window examples. The ones with yellow are the ones which we have picked up.

So different kind of bony lesions. Third window, remember, essentially is a bony lesion, right? It is not a soft tissue lesion, it's a bony lesion, something in the bony labyrinth. So dehesences, enlarged vestibular aqueduct, dilate internal automat is quite a few things. This is an example that girl who I was talking about.

And as you can see in this vestibule of. As you can see, no, this is another one actually. Sorry. These are the one who also presented with autophon. So as you can see, there's one sided vestibular hypofunction.

There was a conductive airborne gap in a mixed loss and she had a dilated antonitus and a deficient facial nerve canal. So facial nerve canal dehiscence with direct internoitus which generated a classical third window. And she did not require surgery but good counseling and some rehabilitation which did the trick. So we have spoken about this and the reason why they are different in md because most trivascular is difficult to elicit criteria. Hearing loss is variable, vestibular function test variable.

And in children it's almost always secondary and very, very slow progressing. And we have also mentioned about bppv. This was a classical Menier syndrome example, Just a tiny bit of right posterior

cohort saccades. And this was. This was anus during acute attack which her dad took with a mobile phone and sent it to me.

This is not very common. So I treasured this video and this is with the full permission. So she fulfilled Barbara Barani's criteria and everything. So she had a steroid responsive autoimmune disorder, not a systemic one, but a steroid responsive because her symptoms dramatically went with steroid management.

So the neurological disorder spectrum is huge and I'm just going to touch a little bit on it. But anything in the central nervous system can lead to central vestibular integration generating a vestibular phenotype with unsteadiness, with dizziness, with vertigo. If the child can discuss, if they describe. Now remember, sensory processing disorders might present with central image vestibular integration problems. And today, in fact, today itself, in my clinic I saw two of them with autism and adhd.

So present with variable neurological features. It's not essential you'll get a classical focal neurological sign, but they do present with movement disorders and incoordination ataxia. The vestibular symptoms might be quite variable and heterogeneous, but the prime difference between, between a peripheral and a central vestibular lesion is in peripheral lesion, when you shut the eyes, your dizziness gets worse. In central lesion, when you shut the eyes, your dizziness gets better. So improvement with eye closure.

If someone tells you or if you see that the parents are hinting at that, then what you do is you immediately think that this could be a central problem. Fainting episodes, positional dizziness, not the BPPV dizziness, but positional dizziness. And you might pick up central oculomotor eye signs and navigational difficulties, as I said, are extremely, extremely important. And these children quite on a few occasions present with cognitive and psychological difficulties. Vestibular migraine is the commonest.

You know, the reason we see so many, they usually follow Barani's criteria, but they can be atypical and they are very, very morbid. They, they, they, they have a significant drain in quality of life. Variable vestibular function test during the ictal phase or just post ictus, you might pick up peripheral vestibular problems. But one of the criteria is vestibular tests are usually normal in the interictal or the inter attack phases. You do in many instances get centralized signs and they coexist with endolymphatic hydrops and sometimes with bppv.

So child with an acute disease spell followed by recovery, but persistence of episodic spells, photophobia, phonophobia difficulties. Vestibular function tests are pretty normal. But when we did her vng, what we found was upbeat nystagmus on removal of optic fixation, which was quite bizarre. Why would a central nystagmus appear once you remove optic fixation? It should be there with and without optic fixed.

So this immediately told us that something very atypical and rare thing going on with these Symptoms we made a diagnosis of vestibular migraine. And these young persons or children, they respond brilliantly to medication with lifestyle modifications. And I always do a scan.

So the first example, second example of neurological disorder child with proximal myopathy like syndrome sent to me after neurology had a full look. Positional dizziness feels better in dark frequent falls. There was some migraine features with headache, quite obese and simultaneous vestibular migraine. The vestibular test battery was completely normal and this is what we found on the eye test.

So central spontaneous gaze, nothing but huge gaze evoke nystagmus. If you don't have goggles, always look at the sclera that will give it away. And look what will happen when he brings it back to central big movements. It's not physiological, it goes and on and on. And physiological is not that huge amplitude.

So look what will happen when it brings it back.

He was undergoing genetic testing at that time. So I didn't have the verdict. But after all this I wrote to my neurology colleagues that this looks like this kind of genetic syndrome going on. And he that was then confirmed and you see a rebound. So now in the central spontaneous gaze you get this nystagmus.

So this was eventually proved out to be CACNA1 mutation migraine and cerebral atasia. We were successful in typing the gene.

So child with postural stability and ataxia vestibular function completely normal. Positional dizziness. And when we did vestibular suppression test Vs, you see this catch up saccard. So the problem was in the central central cerebelo vestibular tracts, mainly in the floc nodular. No, but we have published this.

See first time we have seen that when you are doing a shimp you do not get these anti compensatory saccades because the saccade generated in the cerebellum is a problem. And that is extremely interesting. I think no one else has actually reported this but us and we have found quite a few actually. So this was cerebellar demyelination once again picked up from our otology clinic. So once again vestibular migraine.

He had a shunt for hydrocephalus. We did not know the reason vestibular function was normal, but a low peak psychic velocity. And when I checked his eyes with vng what I could find was a convergence retraction nystagmus which is basically an upbeat nystagmus with convergence. So you see that nystagmus and this is a very useful nystagmus because the intensity of the nystagmus tells you whether his condition is behaving or not. Finally, the diagnosis was a third ventricle aqueduct of Sylvia's stenosis.

We do not know a reason, but he responded very well to anti migraine medication. Vestibular function tests were normal. So these are very interesting cases actually. So it's called a parino syndrome, by the way. We do not limit ourselves to just vestibular causes.

Look, cardiovascular causes, metabolic disorders, musculoskeletal disorders, ocular abnormalities, psychogenic disorders, they can all generate a vestibular manifestation. Right? How do we know which is which? The only way to know is to do a very thorough clinical examination. That is the only way of doing it.

You develop an instinct sometimes to. At my stage now, I don't have to do everything because I pretty much know that this is not is what is happening. But when we are beginning, it's very important to do everything and have that knowledge that these things can also cause balance problems. For example, I sometimes measure the leg length discrepancies in my kids because leg length discrepancy telepes genu, then coxavera. All these things can lead to a balance problem.

We are not specialists in all these disciplines, remember, but we are trained to take a judgment or decide whether it's coming from the vestibular system or not. In other words, we will be able to say this is a musculoskeletal cause of imbalance, this is an ocular cause of imbalance, there's a cardiovascular cause of imbalance and there's a metabolic cause of imbalance. We would be able to say, but we wouldn't be able to treat them. We would delegate them to our specialist colleagues. So you can imagine the amount of holistic knowledge that you require if you are really wanting to do pediatric balance.

So finally I show this slide to everybody. It's a very beautiful slide. And because I'm British, I love, I love the stories of Sherlock Holmes. Well, non British also love it. So how do we make a diagnosis for vestibular stuff?

Because it's so complex, spanning across so many disciplines, so many overlaps. Let me tell you this story. So Sherlock Holmes, the very famous detective, and his assistant, Dr. Watson, were once summoned to the Dorset coast where a prized racehorse called Silver Blaze was abducted from his stable and his keeper, or the stable keeper, was killed. There was practically no clue whatsoever.

And this was the conversation between Sherlock Holmes and the local police, who are absolutely morons. As in any other detective story, as you very well know, Inspector Gregory. Let me just take this out. Is there any point to which you wish to draw my attention, Holmes? To the curious incident of the dog in the nighttime.

Gregory. The dog did nothing in the nighttime. Holmes. That was the curious incident. So why do you think this was so important to Holmes?

So let me introduce you to this thing which I call inductive reasoning. There are two types of reasoning, right? Deductive reasoning. Inductive reasoning. Deductive reasoning is when you do research, hypothetical question, null hypothesis, and then you gather the evidence to prove or disprove the null or the alternate hypothesis.

Deductive inductive reasoning, you've got the evidence in front of your hand, in front of you. You need to make a decision or a judgment as to what this evidence tells you. And every little bit is important in this story. That was the curious incident. The dog did not bark because the dog knew the person who abducted Silver Blaze and killed the stable keeper.

I love to sing in a choir and I can't. Yeah, what is the curious story here? This girl loves to sing in a choir, but he can't. Something is going on. It's just not mental or psychological.

So for vestibular diagnosis, you need to continuously exercise this power of inductive reasoning, which is backed up and supported by your expertise, your wisdom, your knowledge, your approach to the child, your mindset of interacting and integrating with the child. It's not easy and it has to come from a great deal of experience, but it's eminently possible. So it's very, very complex, challenging and stimulating and rewarding because it's a continuous intellectual race in your own mind. And once you make a diagnosis and treat, that's the biggest reward because children have got better central compensation. They bounce back like anything.

And it's still very limited awareness, but this is changing. I'll tell you why. And they are quite different, as we have seen and we have appreciated why. And you know, the classical typical presentations and the commonest and the least and the less common vestibular etiologies in children. And this is what we are absolutely over the moon.

The meeting happened last Wednesday. Ten of us from all over the world, all passionate and dedicated pediatric vestibular specialists. We floated this organization called International Pediatric Balance Network within a week. We have floated a website, www.ipbn.net. we are sending invitations to pretty much many people across the world, more than 150, 200, who all do some form of pediatric vestibular stuff, to become members of this.

And we plan to do this In a very, very integrated multidisciplinary as well as in a very kind of a very active and energetic way. So these are all my colleagues, they're absolutely giants, especially Silver. She's like our mentor who introduced me to the field in 2006. So this will raise awareness. In fact, it's already done.

Not this, but already there is interest in this field because we are now getting invited more and more to international conferences where pediatric dizziness, pediatric vestibular things were never even considered. Never. Nowadays we are getting about an hour or two in a three day conference. That's not

very much. But we are still getting there and we are very happy.

And this will actually accelerate the whole thing. And of course you are most welcome to my Alder Hay October pediatric vestibular course where Amy teaches and I direct. And it's a beautiful city of Liverpool. It's one of the most beautiful cities in the world. But it has.

I do apologize for this. It's not got a very good football team because I come from Manchester and Manchester United is the only football team to me. This is my lab, by the way. Okay, right, so that's it. Thank you very much.

I'll stop sharing now and open to questions.

Open to questions, please.

I've just taken 15 minutes because I started a little bit late. Yeah, one hour. No worries, Professor. In the meantime, thank you so much for this fantastic session. Professor, I think that your insight and expertise are truly invaluable in my opinion.

And we deeply appreciate your contribution to in. To inventis academia. So thank you very, very much. So we are open for the question question time. So please, in case of question, this is the right time.

Thank you so much. We have got one question, let's have a look. Okay. Professor, it'd be possible to get the PowerPoint. Yes, you will be sending them PDFs would you?

And professor, our first question is from my colleague Mario. Mario says thank you so much, professor, that's good to see you tomorrow. See you tomorrow. Mar forward man. Looking forward.

So I have got one from anonymous attendee, you know. Please introduce yourself. That would be great because we are all friends here. May I ask you more examples? Like the child in the slide showing signs of dizziness, balance problems.

How many more examples do you want? It can be tens or hundreds or thousands. So that's why I show you just a snapshot.

All the children in the slide showed signs of dizziness and balance problems.

If patient feels they are spinning themselves compared to room spinning. Does this an importance in adults? Probably in children, they would never be able to tell you. But my personal take is I am spinning and the room spinning. The latter one is more vestibular than the former one, but overall, you know, it can happen in both.

So to me it's not a terribly important question, but it's useful but not it doesn't give me much information.

Yes, trauma can cause deins most certainly you it can. And I have shown you the I have shown you the picture and trauma can cause cause dehiscence in about roughly 4%, I would say. There has not been any studies, so my evidence is from reading a number of publications. But yes, not only in children I've seen it. Amy and I in adults have also seen it because we deal with plenty of trauma cases.

That's a good point from Ruth Marin. Vestibular migraine at the age of about 18 ideally, pediatric vestibular migraine should disappear by the age of 18. So if it's a childhood migraine, it should completely disappear. And you can understand the reason why. Because neurotransmitter activity, the randomness gets less and less and less as you reach adulthood.

So at the age of 18, once you are fully full adult, your neurotransmitter activity is very, very stable. However, it is still prone. So yes, there is a chance it might come back not as a vestibular migraine, but as a classical migraine at about the age of early 40s. But your son should be free of vestibular migraine maybe if not already, maybe in the next few years and then should be free of vestibular migraine up to the age of mid-40s. But then again, it might not happen.

Yes, I ask MRI for every child. When I first started it Ram, my radiology colleague said you are way over the top. Why are you asking an Mr. Scan for a simple dizziness. I said no, dizziness is simple and 2 to 3% of dizzy kids will have a positive Mr.

Finding only the other day. Vestibular migraine child, Ms. Scan a sizable arachnoid cyst pressing on the posterior fossa. Yes, it pays. Always be safe rather than be sorry.

So my radiology colleagues have now completely appreciated and yes, I do scan for every individual child who presents with the dizziness, even though I'm sure it's a vestibular migraine.

Will there be an upcoming webinar that reviews treatment protocols for do you mean in children? In that case, it's up to Anna. Not this year, but I'm more than happy to do this next year.

Ah, anonymous attendee, Nurul Asia. That's a great question, Nurul. So, as you know that recently, about a month or two back there has been a beautiful paper and I think it came out, it, it actually came out from one of my colleagues, Ruth and Lean. You saw, I, I kind of quoted them in my vestibular network thing. They have done a beautiful study on this as well as some.

And there was another study where I was actually the reviewer. This is very important. I think we should perform vestibular screening on children, but not peripheral vestibular screening as much, but central vestibular screening. I mean, my resident will tell you that today we diagnosed two kids, both of them

with central eye movement signs, so. Absolutely.

But these are not pathologies as such because these are more, you know, kind of developmental delays and immaturity and these kids slowly grow out of their balance problems as they grow older. That's my experience. But yes, I think there's a lot of merit in the premise that you should be checking the vestibular system in kids with ASD and adhd. And it's coming slowly. We are trying to do it as much as possible in Alderheim and there have been publications.

Yes, sport related injuries. Well, my colleague Jake Brodsky in Boston, he sees a lot because they're a tertiary sports trauma center. I frankly don't see many sport related injuries exclusive to sports. No, but I see different kinds of trauma, but not specifically sports related. But there are centers who do it.

For example, I know of a center in Auckland who does it and they've published. And yeah, if you just type Brodsky B R O, D S, K Y. He has got fantastic data on post concussion sports. Post sports concussion vestibular legacies in, in children and young adults. Ah, Oana.

Hello. How are you? We met in Romania. Yeah, fantastic. Yeah, it's directive work in children.

Just like Sherlock Holmes. You got it.

Okay, that's a good point. Anonymous attendee. Again, in a clinical setting with limited resources with clinical examination should be prioritized for a child with dizziness. So you mean which clinical examination as a very basic. That you need to do.

So limited resources. Let's say you do not have bng, but you have got your mobile phone. So what you need to do is to get information on at least one frequency range and at least one autolith test. So for the autolith test you do a subjective visual vertical. If you don't have anything, you don't need the bucket,

you just use a protractor which you will find in any child's geometry box anything more than 10 degrees is significant.

And for the clinical examination, ask someone to shoot the. To photograph the eyes of a child. Do a static examination, that is spontaneous nystagmus. Do a head check middle frequency and do at least a lateral semicircular canal head impulse test. You get low frequency, middle frequency and high frequency information.

You do some. You do some static posturigraphy. If you don't have a foam cushion, use a pillow and ask the child to stand and gently jog with eyes closed, with arms outstretched. That's vestibular spinal. And for the, and, and, and of course this backed up with the history will give you good information.

In many, many cases. It's not a complete examination, but that will lead you to the correct path. In most of the cases. That's how I started.

There is no one test which is most reliable. So we do everything. However, if you ask me what is the most important test, and I would say, because we are very high frequency dependent animals, you know, we hardly move ahead like this. Do we move our head like that? It is the head impulse test.

That is one test that you'll have to do.

Good point. Children of witch etiologist. I think you know the answer, Elki. When you are unable to treat the decompensation, a child who is decompensating because of significant anxiety and significant stress. If you do not treat the anxiety and stress, this child will never respond to physiotherapy.

No. So treat decompensation, get a very good result. So that's why it's so important to treat decompensation. So it's not just child comes to you. We do all possible tests, pick up a vestibular

weakness and then give him vestibular rehabilitation.

I covered this in my management module somewhere that vestibular physiotherapy is not the answer for everything and it should not be a blanket thing. No. You'll have to understand what is going on, the etiology and why this child is decompensating. A medical or a, or a mental cause or a physical or a mental cause.

Okay. That is fantastic. I did a lecture on in casino about this Morasha. How would you assess its effectiveness? So there are two types of effectiveness assessment.

One is subjective where you can do a pediatric DHI score or a bird balance scale or even just ask how is the. How is the child? From the subjective point of view, how is the child doing in school? There are varieties of question. Objective testing is very interesting.

Quite A few. Okay, so the first one is if you have done a mastoid vibration, the mastoid vibration test will disappear. There will be no post mastoid nystagmus. If you have got nystagmus, spontaneous Alexander's nystagmus converts from degree three to degree one and then no nystagmus. If you're looking into middle frequency, you can do a dynamic visual equity test where the three lines which were dropped will recover.

Or when you're doing the head shake test, post headshake nystagmus will disappear. And finally the cream and the creme de la creme is the video head impulse test. When you are doing a video head impulse test, your saccades from dispersion will be clustered, vor gain will go up. And finally you get only covert saccades. And what we have shown in our publication, the suppression head impulse test where the peak saccadic velocity of the anti compensatory saccades as well as the asymmetry between the two sides improve.

So quite a few. However, remember, subjective sensation might be disproportionate to objective evidence. Which means objectively nothing has changed. Subjectively, the patient is feeling all over the moon. Brilliant.

Or the patient is feeling absolutely horrendous. But objectively everything is normal. So you can get both ends. It entirely depends on the patients get up the cognitive reaction and psychological reaction. But there are many ways by you can use you can actually measure the effectiveness.

Ruth Marin, are you asking me to read the question in full? Because I think I did, but let me go back to your question.

Well, I did answer should he outgrow this? I think I told you very categorically. Maybe for a year or two he might still have it, but then outgrow it and then probably might be recurrence round about the age of mid-40s, I think answered it.

Absolutely. Anonymous attendee, please do a HR ct. You need to know the size of the eva. Oh, child is three years old, right? Yeah, ct.

He might be a little bit young for ct. So what I do is I usually don't do CT before the age of seven or eight. But you can do it if you are pressed. So what I usually do in such an instance, three year old child with an Mr. Scan.

So you did a CT and now you want to do a HR ct. I don't think there is any need if you've got a CT report. I thought it was an Mr. Scan. So you have, you have diagnosed enlarged vestibular aqueduct with a ct?

Yes. So EVA means That you'll have to adequately counsel him as to what will happen if he gets a sudden blow to the head. In that case, report to the nearest Hospital in 72 hours for a steroid salvage which we successfully do in Al and two, of course, lifestyle changes, for example, contacts, avoid contact sports and everything. Apart from these two, there's not much you can do. The youngest you can do a VNG is six months.

Ah, why? The anterior canal is spared in autotoxicity and this will take a lot of time. I'll come back to you. In fact, I'll explain it to you tomorrow. But remember, it's the way the anterior semicircular canal is orientated and the vascular supply to the anterior semicircular canal, the vestibulocochlear artery, the way the vascular supply is drug delivery to anterior semicircular canal is less than lateral and posterior semicircular canals in brief, but it's a far more complex Beta blockers work like magic.

Betahistine also works like magic in recurrent vestibulopathy and chronic vestibulopathy. Betahistine does not suppress vestibular compensation. On the other hand, it promotes vestibular compensation. That's recent evidence. So betahistine was much condemned and maligned before, but now it's not.

How early can you perform SEMs? Six months?

No, when it's an active vestibular migraine, when the spells are very frequent, then vestibular rehab will fall flat. You know why? Because vestibular rehab is for vestibular weakness. And in vestibular migraine you do not get a vestibular weakness per se. But.

But vestibular rehab has got a role to play with vestibular migraine, especially with chronic patients where you'll have to provide with vestibular rehab. After you control the vestibular migraine and the. And the other reason is of course vestibular migraine with itself. Maintain vestibular decompensation if there is a vestibular weakness. How would vestibular migraine be managed in kids?

Cognitive approach, the diagnosis, the etiology all needs to be counseled, followed by pharmacological management. There are seven different drugs you can try. Start with the one which has got the least side effects and finish with the magic bullet which is gabapentin. Lifestyle changes, right to the school, engage the school, engage the parents, adequate hydration, avoid migraine triggers if you can and they all get back to a normal life. Very good prognosis.

Yes, there is a beautiful paper. Anterior canal Sparing. I don't remember the name as to who has written it, but if you just type anterior canal sparing and autotoxicity, this paper will come. So it's a very beautiful paper.

Ah, skull vibration Indus is a fantastic test and the kids love it. The problem is that it only yields a low frequency caloric like information. You see, it does not tell you with the most practical frequency, which is a high frequency, which is the head impulse test. So if you can manage to use the SVINT along with the head impulse test, then you have got your case. So what I suggest is you do the svi, you use the mastoid vibration, let's say you don't have goggles or anything.

Once again, record it and then do a clinical hit impulse test. If there is a significant vestibular problem, then there will be. You'll find findings, I'm sure about it. Because look very significant and severe vestibular lesions, they do not compensate. So you will find them even under full proper light once they start compensating with vision.

That's why you don't get these signs and you can only get them once you remove optic fixation. But if it's a significant problem, then yes, I think we have done it. All done.

Well done. Professor Dasgupta, thank you very much. One more time and thank you all for attending. I take this opportunity to remind you that the second webinar in Professor Dasgupta's series will take place on March 13, titled Assessment of Vestibular function in Children, Including Compensation after

management. Ah, there was a question like that.

So yeah, the next one. Yeah, I'll show you so many different examples how you can do this. Oh yeah, Good. Well, thank you. Yeah, okay, fantastic.

Professor, thank you very much once again. Thank you. Have a lovely evening guys. Thanks. Bye.

And additionally, just one staff. Our next webinar is scheduled for February 19th and will be hosted by a new and prestigious addition to our speaker lineup, Dr. Nicolas Perez Fernandez, joining us directly from yes, Clinica Universidad de Navarra in Spain. Thank you, Amy. Thank you, Professor.

Thank you. Bye. Bye guys. Bye. And stay tuned for more updates by visiting the Academia section on our website site and following us on our social media channels.

Thank you all once again and see you at the next session. Bye.