Auditory and Vestibular Complications and Legalities Following Head Injuries

Presenter: Soumit Dasgupta, MD

- [Presenter] Good morning, before we get started, I'm gonna go through the learning outcomes for today. After this course, participants will be able to explain the mechanism of head injuries and how it can affect the audiovestibular system in both children and in adults where the outcomes are different. After this course, participants will be able to describe the rigorous and comprehensive diagnostic process and treatment in a multidisciplinary team approach, noting that the management algorithm spans across disciplines and is not limited to the audiovestibular system. And after this course, participants will be able to describe the nuances and details of the legal process often arise after a head injury with audiovestibular involvement. And now I'm gonna turn this over to our sponsor Inventis.

- Welcome, everybody, and I thank you very much for being with us today. The scientific divulgation promoted by Inventis Academia and directed it to all those interested in disorders and pathologies of the hearing and balance system, never stops. Inventis is honored to announce the second seminar of the series held by an international respected expert, Professor Soumit Dasgupta, Consultant Audiovestibular Physician, Neurotologist, and the Clinical Lead in Pediatric Audiology, Alder Hey Children's NHS Foundation Trust in Liverpool. Thank you very much Professor Dasgupta for accepting our second invitation. Here with us, an old friend as well as a colleague of Professor Dasgupta, Dr. Enrico Armato, ENT doctor at AULSS 3 Regione Veneto and Inventis clinical reference for balance. Thank you Enrico, I'm so happy to see you're here with us today.

- Thank you.

- Title of the lecture is "Audiovestibular Complications and Legalities Following Head Injuries." As usual, I strongly invite all of you to write down any questions in the space provided, that you should see on the screen. After the event, you will receive a survey that I kindly ask you to fill out. The survey will help us to understand your needs and to

get your ideas in order to provide the best educational support ever. And now I leave the word to Dr. Armato first and to Professor Dasgupta immediately after. See you after the webinar. Enjoy.

- Okay, thank you Anna. My pleasure to see again my good friend Soumit. And I want to take one minute to present him. Professor Dasgupta is an award-winning consultant, neurotologist and audiovestibular physician at Alder Hey Children NHS Foundation Trust and Hypatia Dizziness and Balance Center in Liverpool, United Kingdom. He's a senior lecturer in the University of Manchester, U.K., and visiting professor at University of Siena, Italy. He's a globally acknowledged expert in pediatric vestibular disorder, leading one of the few tertiary pediatric vestibular centers in the world, and the pediatric vestibular research laboratory. He's well published in peer-reviewed indexed journal and has written several textbook chapters. He's leading the U.K. in looking into genetic hearing loss ototoxicity in the pediatric population with a dedicated monitoring protocols, and represented the U.K. in International Ototoxicity Monitoring Group, a global consortium of expert researching in ototoxicity world.

He's an international Secretary of the International Vestibular Society, the Chairman of Education, the British Association of Vestibular Physician, and an executive committee member of the British Society of Neurotology and Otology. He's an expert reviewer for nine indexed journal on neurotology and genetics, in the editorial board or for index journal, and he's an expert advisor in the General Medical Council, United Kingdom. So this is with my great pleasure, dear Soumit, the floor is yours. Have a nice talk.

- Thank you very much, Enrico, for that very kind introduction. And thank you very much Anna for taking this initiative to propagate finer nuances of vestibular sciences to a global audience. You know, we all condemn COVID. COVID was a bleep in our life, but what COVID has given us, we can't really not knowledge that is this opportunity to reach out the farthest corners of the world through this wonderful thing called either a

Zoom or a GoTo, whatever, you know, all these different platforms. So as Enrico said, that yes, I am a primarily pediatric audiovestibular physician who works in Alder Hey, but I've also got a reasonable sizes private practice where I don't see children, I see only adults.

And that's in Hypatia Dizziness and Balance Clinic, which is a standalone vestibular, audiovestibular setup with our own vestibular laboratory, which my colleague, Senior Audiologist Amy Lennox runs. So we do this, we see adult patients from all over the country out there. So today I'm going to talk to you about head injury and what can be the neurotological, the medical, and the legal aspects of head injuries. Now, why is this topic important? It is important that you will find that following head injuries in the grand scale of things because there is a obvious head injury which has got the potential of causing a brain injury, in the grand-scale of things, the vestibular system largely remains ignored.

And you would inherit patients five, six years down the line where the system has already undergone compensation, decompensation and more importantly this damage to the system might lead to a significant cognitive and a psychological aftermath. So you get the patient in a very complex type of presentation. Things are changing. We are trying to raise awareness among the legal community, because you'll get most of your patients from the legal community. So we are trying to raise awareness among the legal community that look, this tiny organ is very much there which can get hurt if you sustain a blow to your head. So things are changing. So that's why we thought it important enough discussion with Anna and I immediately agreed, that this is a largely ignored field.

You might think that I come in with all different kinds of ignored topics. For example, pediatric vestibular science is not exactly ignored, but the awareness needs to be raised similarly today, and the last lecture that I'll be doing on the 20th is also about the

thing which hardly gets mentioned. Right, so, let's have a look at this. First of all, let me break a few myths. Now you have heard about the word concussion, which is very widely used in the medical jargon, medical terminology. So any blow to the head will lead to a concussion. But concussion has got a definite definition. This was previously synonymous with a minor traumatic brain injury, which you can imagine is exactly that.

That is, there is a brain injury, but that's a minor, which follows a trauma. However, concussion now is believed to be a traumatic alteration to any brain function due to a direct or indirect impulsive force applied to the brain. Now what is an indirect force that I'm going to discuss? Very importantly when you are saying that someone has a concussion, there should be a near-total recovery or total recovery within six months. If it goes beyond six months after a head injury, it's not concussion by definition, it becomes something else. It becomes a permanent traumatic brain injury that is not the same from concussion. The effects of concussion when they continue for more than six months, then it is an indication of a permanent damage.

Now you can imagine that the legal implications of these two are completely different. A person who recovers his life completely goes back to work as if nothing has happened, obviously will get far less compensation, maybe only for the accident than someone whose life is completely messed up at the permanent traumatic brain injury stage. Now obviously it would transpire almost by default, that look, when you get a blow to the head, indirect or direct, then because the inner ear unfortunately or fortunately is located within your head, in the petrous temporal bone or just outside, including the external and middle ears, along with the vestibular system, obviously there is a potential that that same blow would have caused a damage, temporary or permanent, to the labyrinthine apparatus.

Now this might be a simple concussion where the subject completely recovers by six months or it can be a damage. So labyrinthine concussion is different from a

labyrinthine damage. Obviously if there is a damage or if there is a blow to the system, then there will be symptoms. And the classically, classical hallmark of such a symptom, if the vestibular system is damaged, is dizziness. Now you can argue that even after a traumatic brain injury because of central brain damage, there can be dizziness. Remember the dizziness which comes from a traumatic brain injury is very likely because of disruption in the central vestibular tracts. So either way, a vestibular system, peripheral or central, will generate this very classical dizziness or disorientation in space.

Note, I'm not using the word vertigo because it can be anything, it can be a thing like a brain fog or a fuzzy headedness or a muzzy headedness, et cetera, et cetera, et cetera. So it's broadly, you can call it a disorientation in space. So when there is disorientation of space following a head injury, you need to think whether there is a damage to the peripheral vestibular system or there is a damage to the brain which is disrupting the central nervous system, central vestibular pathways. So this is from, this is a internationally accepted consensus of traumatic brain injury and a minor traumatic brain injury, which is now a concussion. So as you can see, I'm not going to read through the slide, but there are some definite criteria and based on this criteria, which is the Mayo Classification, one can make a judgment or an inference whether there is a active brain injury.

Just continue reading whilst I tell you about a very important caveat. And this caveat is: traumatic brain injury is not synonymous with traumatic head injury. Traumatic head injury is a trauma to the head, which might or might not lead to a brain damage. For example, you are walking, you are in your own home and your head suddenly bumps to the doorframe. Now that is a traumatic head injury, but does that lead to a tangible brain injury? Very likely not. I mean every time you get a blow to your head there is some neuron damage, but that neuron damage must be sufficient to cause symptoms

to categorize it as a brain injury. So traumatic brain injury follows from traumatic head injuries, but not all traumatic head injuries are brain injuries.

And frequently people get caught out when they write a legal report. Because at the end of the day you need to accurately classify whether it's a brain injury or not. And just by saying it's a head injury, you are not actually classifying about the brain. So it's a very important caveat. So as you can see from the slide, that level of consciousness, amnesia, which can be a pre-traumatic, just before the accident, or a post-traumatic amnesia just after the accident, where the subject will not remember what led to the accident, or what happened after the accident, is a very important sub-criteria to diagnose a brain injury. Then, of course, you need your Glasgow Coma Scale.

There might be obvious neurological signs; changes in your mental state. Neurological deficits actually include a vestibular change, you can get intracranial hemorrhages. All these are absolutely sure signs of traumatic brain injury. But when these signs are partially present, or absent, or short-lasting, that's where the bone of contention lies. That's where the legal wrangling starts in. Because a minor traumatic injury, according to you as an expert for the claimant who is actually claiming, you will write that there is a minor traumatic brain injury. Whilst the defendant's expert, which I'm going to explain later, who is the defendant's expert, will say that, "No, sorry, there is no minor traumatic brain injury because it doesn't fulfill the MEOS criteria."

As simple as that. So this is where legal wrangling starts and you might disagree with your counterpart. But the features that I've given here, they suggest that very likely all these features which recover over a period of time is an MTBI or a concussion. And moreover, very importantly, remember an MTBI will not throw up, will not be shown on a scan. So if a judge is facing me and asking me, "What is your objective evidence?" There is no scan, there is no scan feature of a traumatic brain injury. I mean, for a traumatic brain injury you get different kind of things on the MR scan, special FLAIR or

spectroscopy or inverted recovery images. You get diffuse axonal changes, you get cortical atrophy, you get white matter changes, you get gray matter changes, you get legacies of intracranial hemorrhages.

That's a sure shock traumatic brain injury. But in minor traumatic brain injury nothing will be evident. Because all these changes I've told you, they need a certain size. Now those sizes might be very small and not shown up on the MR scan. Does it mean the person never had a brain injury? Well, that's not the case, that's not the case because of central eye movements. Central eye movements do not depend on a size. And I'm going to show you quite a few videos of these central eye movements in my next lecture. So remember, you can successfully prove that in the absence of a positive MR finding, there is still a traumatic brain injury, minor, with central eye movements.

For example, periodic alternating downbeat, they all indicate a site. So when you look into the epidemiology you can see that traumatic head injury, and this is a Korean study, traumatic head injury invariably leads to some kind of a labyrinthine assault. Not saying it's a damage, it can be a temporary concussion or it can be a damage. Out of which up to 50% show a permanent labyrinthine dysfunction at five years, of which about a quarter remain undiagnosed. Because, as I said, in the grand-scale of things, I had a brain injury, you know, I can't think straight, all my central cognitive functions are in a mess, I am emotionally labile, you know, I've got plenty of psychologicals and psychiatric issues because of the brain injury, my mobility is restricted.

So I don't think about dizziness imbalance. So my tiny vestibular organ remains largely ignored. But people need to understand that a vestibular system plays a very important part in how you react to head injuries. So this is both in adults in children, of course they behave differently from, because children have got far more cerebral plasticity than adults. So they are much better to make a recovery than adults. But even then I have found children whose lives are completely messed up. Remember one thing that

in children which do not happen as much in adults, they get or they might be, they have got the potential to develop neurodevelopmental conditions; for example, autism; for example, attention deficit disorders; for example, social communication difficulties; for example, things like, let's say that, avoidance behavior.

All these things can be generated from a head injury, which you usually do not investigate as much as in adults. But in kids I see quite a few pediatric head injuries, I need to factor in everything, it's a very kind of a holistic approach. And hearing loss is observed in up to 50% of traumatic head injuries. But today we are going to concentrate on mainly vestibular injuries, we'll touch on hearing a little bit. As a result of this lack of awareness, head injuries are often misdiagnosed. For example, a person who is very reliant on visual senses because of vestibular injury, reacts with bright light, might actually end up in ophthalmology. A person who has got hardly any balance and severely immobile, ends up with neurology.

No one thinks about the vestibular system. So they're misdiagnosed or they're never diagnosed, but they have got very important implications to the lifestyle. And if you do not treat a post-head injury vestibular dysfunction, then there is a high chance of you developing, not you of course the subject, developing a 3PD, which you know is very, very difficult condition to treat. However, I'm going to talk to you about one person whose com report I completed today, who had a remarkable turnaround because we identified a vestibular dysfunction quite quickly. And the legal implication must never be forgotten, even though you are not a legal expert appointed by the court. Even then, when you assess a person with head injury routinely referred to you either in your hospital, clinic, or privately, your report will be produced in front of the court because believe me, the majority of vestibular injuries following head injuries, they do go to court.

So you will be asked about your report, your report will be scrutinized, weak points will be taken up, they will be debated. So even when you are not seeing someone legally, you must have this legal aspect in the background, and your report should be completely watertight. So what really happens? There are two main or rather three main mechanisms of injury. It can of course be a direct blow to the head, which is quite self-explanatory. You know, you get a blow to the head, you end up with a head injury, plus minus a brain injury. However, even with a blow to the head, you might still get a head injury or a brain injury. And that is when you do not have, you do not hit your head into something.

This is when the mechanism of acceleration deceleration forces come in. So let's say you are moving in a car or you are stationary in a car. When you are moving in a car, suddenly you find an obstacle and you brake very quickly and your body is thrown forwards: one. Two: you are in a stationary car or stationary. You are hit by another car who bumps into your car, which we call a shunting energy, shunting collision. In that case, once again, your body will be thrown forwards and although you do not have a direct blow to the head, this movement of your head, on your trunk, on your neck, is enough to cause shearing forces, which will lead to alterations in your inner ear vestibular system as well as your brain.

And the reason being, these are solid organs enclosed in a very rigid bony skull, and utilize fluid. In the ear it's endolymphatic fluid, in the brain, it's CSF. So these these kind of fluid dynamics in a very rigid bony enclosure gets upset and that leads to, the acceleration, the sheer speed, the velocity of the fluid movement followed by a sudden stop is enough to cause shearing damage to the vestibular hair cells and of course the brain. And the third one is of course a blast injury, which is a combination of everything. So this usually happens in soldiers or unfortunate victims of terrorism when you get a blast, which has not only got, which not only has got a significant acceleration deceleration force, but also it has got the potential to throw you from the

ground and slam you down to the floor or the ground, which is a direct injury as well to the head.

So these are the three main mechanisms by which your vestibular system, central and peripheral might be affected. It doesn't stop at that. You might get a thing called a contrecoup injury or a coup-contrecoup. Coup injury is when you get a blow on one side of the head and your vestibular system gets messed up on that side. However, I have seen, and I would say it's about one fifth of the cases, where the side which gets the blow or which is actually is hit, is intact, whereas the other side, the vestibular system gets messed up. This is very classically recognized in head injuries. What you get is a coup-contrecoup head injury or a brain injury.

And it depends on various of subject factors and everything, and there's no way you can predict it. For example, you get a blow to one side of the head and you end up having a bilateral vestibular dysfunction. It's because the other side is also affected. It's just the sheer force. It usually happens in very significant force. The sheer force is actually transmitted across the bony skull to the other side. In many of the injuries regarding car traffic accidents, in a collision, or when you are falling from a height to the floor, there is invariably some kind of a whiplash, or transient, or a sprain of your cervical spine, which can happen. Whiplash, whether it causes a problem in the vestibular system is debatable.

But what I have found and what the literature says is, the forces which are applied to the cervical spine, they might secondarily be transferred to the inner ear vestibular system like an acceleration deceleration mechanism. So there have been a couple of papers which have said that following whiplash you do get a vestibular hypofunction. But what I would say to you today is, because I've seen many, a whiplash will not cause an isolated vestibular dysfunction, but will actually add to the dysfunction in the presence of labyrinthine damage or labyrinthine concussion because of the head injury.

So if it's complicated, I'll say it again, a whiplash, just a whiplash injury, should not affect the vestibular system. But if the whiplash injury is associated with the concomitant head injury or a brain injury, then it will add onto the damage of the vestibular system which is caused by the head injury.

So, on its own it doesn't usually, and this is a very important legal point. So what happens following the trauma? As I said, there's a gross movement of the endolymphatic fluid, which creates a shearing force and detaches the sensory epithelium of both the vestibular end organ and the cochlear end organ. There can be micro-vessel hemorrhage, there can be interference with vestibular transduction of the action potential, finally leading to a gradual degeneration of the sensory hair cells. Of course there might be a direct damage if there is a direct fracture of the petrous temporal bone, longitudinal or transverse as you know, that can really disrupt the vestibular mechanism from the structural point of view. Central vestibular tracts are usually disrupted by the diffuse axonal changes, which I said which can be overt, which is a very clear traumatic brain injury, or in an MTBI or concussion, it can be very, very subtle.

But the most important thing is, the force of the injury determines the amount of brain injury that you have. So, for example, a very significant head injury will lead to a significant brain injury. However, a very minor head injury might spare the brain, but the vestibular system can still be damaged, which means, the force of the injury does not determine vestibular damage. And frequently I argue with my counterpart experts or even with the judge or the solicitor, whoever, that look, this is a trivial injury. you know, I was sitting in a car and the car shunts me from the back at 15 miles an hour, I'm thrown forwards. How is such a small movement causing a vestibular damage?

Now the answer is: the force of the shunt or the acceleration deceleration force, is not proportional to the vestibular damage that you have. In other words, a vestibular

damage can still happen and we have found this in children as well as in adults. So what are the different functions which can be involved following a head injury? Now look, I'm saying head injury, I'm not yet saying brain injury, because I'm concentrating on vestibular system. So obviously degeneration of reflex eye movements, which is the VOR, it can be damaged, reflex muscle responses, for example, the vestibulo-colic response, the vestibulospinal response, all these reflex muscle responses might be affected. There might be a problem with eye fields, the way you are tracking, for example, with smooth pursuits and saccades.

When the light is switched off because you get dependent on your vision in order to make up for vestibular weakness when the lights get switched on, a person with a vestibular damage following a head injury might find difficult ambulation in darkness: very, very important. Vestibular system takes a good role in optic, in kind of regulation of your optic system. For example, the way the VOR is designed is basically for gaze stabilization. That might secondarily lead to an increased heightening of your somatosensory stimulation, which can happen. For example, post head injury, vestibular damage subjects might be tactile, might be very, very sensitive from the tactile point of view. Of course postural stability. Very importantly orientation, where you are at a given moment in space, and absolutely crucial to recognize is the effect of navigation.

Now as you know that the vestibular system, especially the semicircular canal but to certain extent the otoliths, they have a significant flow to the entorhinal or the hippocampus, which are very important for navigation. Your navigational skills, the way you recognize where to return to your own home or even when you're walking in your own home, you know that's a door in front of you. These are important navigational skills. And a common problem which you might find after a head injury is, you know, "It has never happened to me, but I frequently bump into doorframes." That will tell you that there could be a vestibular damage here, which is causing this. And, of course,

secondary effects of vestibular dysfunction, cognition, mood, effective states, neural pathway changes, trigeminal nucleus interactions, autonomic responses.

Did you know that otoliths have got a strong connection to the vasomotor tone, and fright and flight reaction might be regulated through your otolith system. And dream bizarreness: now I wrote this because I found it in a paper. I do not know what exactly means, but apparently following of vestibular damage you get bizarre nightmares. Now that might be a part of the PTSD or posttraumatic stress disorder or it might be linked to vestibular systems. So I am not very sure about dream bizarreness, but this is one of the things which you might find. So you must ask your vestibular patients following a head injury, or who has who have come to you for opinion about dreams that is a part of the history.

So now you know what happens, what the problem is, and what are the different systems which might be affected. Let's have a look at the clinical features. So, of course, you can get vertigo, you can get different kinds of disorientation, you can get positional vertigo, which could be BPPV like, or you can get self-motion induced when you're moving your body on your trunk or neck, you can get dizziness. Visual vertigo is an over-reliance of your visual sensors. So challenging visual targets for example, shops, crowds, even you'll hear your patients telling you, "I can't pick up a cereal which I want from a choice of 20 cereals on the shop on the superstore racks because that makes me very, very dizzy."

So any challenging visual environment will trigger off a disorientation if there is a vestibular damage. There could be, of course, imbalance, more common with bilateral vestibular weakness. There can be frequent falls. There can be drop attacks like Meniere's, Tumarkin's. There can be third window symptoms like conductive dysacusis. So post-traumatic perilymph fistula we're talking about. Of course, there can be headache, neck pain, back pain, there could be a general post-traumatic

migraine-like feature, which could be vestibular migraine or a classical brainstem migraine. Bilateral vestibular hypofunction might present with oscillopsia, blurred vision and sometimes painful eyes because you are engaging your eyes too much to make up for your vestibular system. That sometimes leads to an avoidance behavior.

You tend to avoid crowds. You tend to avoid socializing. You tend to avoid to go to shops because you know that your system will be challenged and it might decompensate. So avoidance behavior is never good because avoidance behavior leads to a significant diminishing in the quality of life. Can you imagine a person who goes to the club every evening to meet with his friends, suddenly stops going because he thinks that when talking to his mates and everything, the whole crowd atmosphere will make him dizzy. Now that has secondarily, that completely downs his quality of life from the psychological point of view. You can have a mal de debarquement-like episode. So for example, it might manifest itself after a head injury, but usually not common.

You can develop a recent onset travel sickness. So a recent onset travel sickness after a head injury is due to a labyrinthine damage, undisproved otherwise. There are problems in undertaking daily tasks, For example, climbing stairs. For example in lifts you feel very queasy because there's a rapid change of center of gravity in the lift, and in escalators you are sometimes unable to look from the top of the escalator down because of this acrophobia or a fear of heights which can develop after a post, after a head injury. So you can see that a gamut of symptoms, a wide variety of symptoms, do manifest themselves after a vestibular involvement, let's say, not talking about concussion or damage vestibular involvement after a head injury.

Now this is absolutely crucial for you to identify. Vestibular system itself leads to cognitive and psychological effects. It's very well known, right, because of its strong connections to the amygdala, to the limbic system, to the reticular ascending system.

And imagine someone who also has a brain injury, the effect is magnified. You cannot dissociate the cognitive or psychological effects from a vestibular damage. You know why? Because of two reasons. The first reason is the patient is very apprehensive and fearful of balance, which leads to an emotional problem: one. Two: the very presence of cognitive and psychological disorder, or damage, perpetuates vestibular decompensation. Your brain never gets a chance to compensate your vestibular problem, because it's very busy with all these different cognitive and psychological effects.

So it's very, very important, not only from recognition point of view, but also very importantly from the management point of view. In other words, you cannot really think of vestibular rehabilitation without a cognitive and psychological rehabilitation. So as you can see there are so many derealization, depersonalization, reading difficulty, cognitive, it looks quite a few of them. Nightmares is one of them. Post-traumatic stress disorder is one of them. Reliance on thoracic proprioceptive cues, why? Because they are overworked. Your vestibular system is damaged, your eyes are worked to the hilt by the brain. So you are very reliant on your thoracic proprioceptive cues. What would that lead to? Muscle ache, because your joints and muscles are working so much to give you balance, there is a fatigue, simple.

And it's a vicious cycle. What kind of vicious cycle? Vestibular injury, excuse me, vestibular injury, cognitive effect, brain injury, more cognitive effect, vestibular decompensation. So it's kind of in a chain of events which goes on. So this is very, very important for you to recognize. You see, when you are seeing a patient with a regular vestibular problem coming through your doors, you hardly think about this cognitive or emotional backlash. But out here in head injury you should always do that. In fact, if you can, you should always ask cognitive and emotional aftermath of any vestibular damage be it vestibular neuritis or Meniere's or whatever. So these are the

different vestibular pathologies after head injury. Variable vestibular damage for chronic vestibular syndrome.

So this is recurrent vestibulopathy with intermittent decompensating episodes, which are brought about by concurrent illness, stress, anxiety, et cetera, et cetera, et cetera. BPPV up to 60%, not uncommon because of the force physically dislodging the particles, different pathology from idiopathic variety. Up to 40% vestibular migraine and classical migraine. Central vestibular processing syndromes, where there is a central vestibular problem in about half. Meniere's disease can be found, that's what we call a secondary post-traumatic Meniere's syndrome, in about 25%. Traumatic third window lesions are not very common, about a fifth. Isolated otolith damage is very important, and this is very, very new. This was discovered by Tsu and Murofushi, I had the pleasure to meet them in Dubai IFOS, who conclusively showed that there could be just VEMP abnormalities with normal canal functions.

And recently we have found, in my series, that quite a few of these head injuries present otolith damage, therefore, the message is: always do a VEMP, always check for head heave ocular counter-rolling, and pretty much everything for utricle or saccular damage. And, of course, if there is a significant head injury, then you can have a transection of the VIII nerve itself just like as you would get in a facial nerve. A few words about post-traumatic BPPV, it is commonly bilateral and if you get a subject who is not a woman more than 55 years old, but a far younger person, man or woman, then head injury should be your first diagnosis. So it's commonly bilateral.

It can affect multiple canals, it can be followed by canal conversions quite difficult to treat sometimes and can be recurrent. Now recurrent is because that otolith anchorage mechanism is disrupted itself. As a result, even if you send the particles back to the canals, back to the otoliths, or utricle, they do not stay put but they fall back because they don't get anchored because of the disruption in the otolith cover and protein bond

inside the otolith system. So they are recurrent, and if in fact I have done six, sevens, eight times, I have done particle repositioning until I got on top of it. Now one important thing, head injury can generate a very important condition called, vertebral artery dissection.

I've seen that. If there is one contraindication, absolute contraindication for a BPPV, it's a vertebral artery dissection. You can do BPPV in obese persons, you can do BPPV in stiff necks, you can do BPPV with . And I'm sure my colleagues will agree with me in this part, the contraindication: vertebral artery dissection. So, someone with a head injury and vertebral artery dissection, coming to you with a BPPV, my question is, "How would you treat?" Would you go ahead and take the risk, or you won't treat it, or you put the person in the TRV Chair? Now of course the TRV Chair is probably a good option, but only for this, it's not very common. So one trick is basically to ask your patient, show him the Dix-Hallpike, the lateral roll, as well as the supine head hanging, and ask him to achieve his own velocity rather than you doing it.

And if there is BPPV, it's bound to manifest itself. That maneuver that you would do, once again, the patient does it. So it's an active maneuver by the patient, not a passive. So you don't do anything. You ask the patient, is most comfortable. Because you see, the patient will still move his head because you can't survive without moving your head. Does it cause a problem? It doesn't. So let him achieve his own comfortable velocity and that's a useful way of treating BPPV in a patient with a vertebral artery dissection. I've done about four, and at least on one occasion I did one and then the patient collapsed and I thought that's it, I'm going to lose my license because I have done some grievous harm.

But she picked up, that was because of vasovagal that she almost fainted. These are the inner ear hearing mechanisms which can be affected. Of course there can be a bony damage to the middle ear, to the external ear, or directly to the inner ear, to the

nerve. There could be central auditory pathway damages. Tinnitus and hyperacusis are not known, are not unknown. And this can either be generated from the inner ear itself or from the central auditory gain mechanism, which is roughly at the level of the brainstem. So they are kind of, they are encountered in varying degrees. There can be a significant hearing loss or there can be a slight hearing loss. In kids it's important because hearing loss will affect school performance, and also directly lead to a problem with his development and will affect cognition, the way he thinks.

So following head injury, I've taken a very, very long history. Now when I take a history from my head injury patients, I take one-and-a-half hours. Now this is a luxury in your clinic setup. You don't have one-and-a-half hours just for history. Remember after that comes testing, so you don't have it. But in head injuries it is recommended that if you are appearing for the court from the legal point of view, you have to ask about everything, literally everything: and not limited to the vestibular system alone. You have to ask about daily life, whether he can cook, whether he can shop, about his hobbies, about his cognitive system, about his psychological system, about his neuromuscular system, about how his quality of life, everything you need to ask.

So first thing you need to ask is details of the mechanism of the injury. Here you are trying to figure out whether there is a concomitant brain injury. Now you might ask me, I'm a neurotologist, am I trained to comment on brain injury? Yes I am. I am trained to recognize a brain injury because I've spent time with neurology department during my training to figure out whether there is a brain injury. So I'm well versed with the Mayo Classification, and it's not going beyond my expertise to comment whether there is a brain injury or not. Finer nuances, finer details, not my domain: neurologist. But I can suggest that there is a brain injury. So like, for example, loss of consciousness, which is a not a minor traumatic brain injury, but traumatic brain injury, lowering of Glasgow Coma Scale, or a PTA.

PTA stands for post-traumatic amnesia. So then you'll have to ask as to how the symptoms evolved. So immediate and subsequent symptoms up to 48 hours followed by what happened after 48 hours to three months, between three to six months, and then beyond six months. Six months is the crucial cutoff because a person will get better and will recover within six months if it's a simple concussion. But if there's a permanent damage the symptoms will linger on and continue and continue and continue. So it's very important to ask the patient this, however, if the patient is unable to remember, then you'll have to go through the evidence which has been supplied to you from contemporaneous; from hospital records and from medical records.

Often there can be complex injuries. So in order to ascertain a vestibular injury, please do not forget that this person might also have a limb injury. There might be a broken bone in the leg, in the arm, in the shoulder, there might be an abdominal trauma, closed abdominal trauma, there might be a liver laceration, there might be a spleen laceration. All these things are important because the healing period has got a direct bearing on vestibular compensation. It's a very, very, very complex field. And also if there is a skeletal injury, then obviously you can imagine it will mess up with proprioception, that will only prolong vestibular recovery. We have mentioned about psychological and cognitive symptoms. We have to ask about treatment and how his employment has been involved, past medical history.

So you have to cover pretty much the whole life of the patient, not just the vestibular injury. So key questions, I mean this is typical, I did mention this a little while back, so just have a read. This is what you would ask as you would ask for any vestibular problem, be it a neuritis, be it a Meniere's, be it a BPPV, whatever, similar kind of questioning. Look at the last one, otolith symptoms, what are they? Now what I'm going tell you today is a very interesting thing, and I'm sure many of you would know. There has been evidence that otolith symptoms differ from semicircular canal

symptoms. Semicircular canal symptoms are very classically, this classical vertiginous disorientation in space.

However, otolith symptoms are described as non-vertiginous disorientation. For example, the patient will come and tell you, "I think the ground is rushing towards me." "I think I'm floating on cotton wool." "I think I have got a feeling that I'm in a sinking ship." "I think that my brain is full of brain fog." These are very, very importantly otolith symptoms. And please note that many of them actually resembled the Barany criteria for 3PD. So I've been asked this question, "How can you say that this is otolith rather than 3PD?" Well the important thing to understand here is, an otolithic dysfunction can lead to 3PD, simple. A non-treated and ignored vestibular condition can lead to a 3PD.

In fact it is one of the commonest cause of 3PD is an untreated vestibular dysfunction. So please try to distinguish between otolith symptoms and semicircular canal symptoms so that when you test you can easily corroborate. And it becomes very important, otolith symptoms become very important in the entity isolated otolith disorder because they've got separate, different set criteria. Tsu and Murofushi, they have, Matthew and Tosh, they have got set criteria to kind of tell you that there is an otolith problem going on and the semicircular canals are not affected, and they are not uncommon after a head injury. Now we have thought about a number of reasons, is to do with utricular saccular metabolism and its location.

And this is not the topic of this lecture, but I'll be more than happy to share you thoughts as to why this happens. Then comes the examination. You'll have to go through a full examination. General examination, you'll have to look for anemia and all these things. Perform a full neurological examination, a full cardiovascular examination, including blood pressure. Remember vasomotor tone can be affected by the otolith system as a result of which you should measure lying, sitting, and standing blood pressure. And you might get things like orthostatic hypotension or postural orthostatic

tachycardia syndrome, POTS, which can be due to a vestibular problem. Musculoskeletal examination, weakness and power, et cetera: full audiological examination. And then this is not the topic of today's talk, but full vestibular assessment, no stones unturned, everything.

And this is what I call is our favorite vestibular gram, which was first propounded by the great Ulmer. So as you can see, different frequency movements, different frequency ranges of the vestibular epithelium. You know about this, you know about all the tests. For example, standard VNG, calorics, mastoid vibration, rotating chair, head shake, DVA, head thrust. And then with the utricular saccular system, the head heave, the subjective visual vertical, the off-vertical axis rotation, cVEMP, oVEMP, ocular counter-rolling, a few other tests in order to exclude other things. For example, HVIN and Hennebert's Test, the functional head impulse test and SHIMP, the Romberg, Unterberger posterior graphic, and of course neurological assessment where you look for different ectopic eye movements.

Now remember one thing, can you see this caloric dissociation? I once had a patient with head injury, vestibular system was largely intact, but very, very dizzy. Caloric system showed an inversion, caloric inversion. What do I mean by that? Instead of COWS, cold opposite, warm same, it was, cold same, warm opposite. That's called a caloric inversion. Now I question myself, why would there be caloric inversion? And that's very clearly a central lesion at the region of the brain stem. And when we scanned that patient, there was DAI or diffuse axonal injury changes in the brainstem region. So everything is important. You'll have to watch the eyes like a hawk, and that's what we'll do in our next lecture.

But it will very interesting, I can assure you. So every individual test you'll have to do, and other assessment is cardiovascular, ophthalmology, psychometric assessment, imaging, blood tests, genetic. Genetic testing is not essential for a head injury test. But

at the end of the day, if you are investigating someone with a vestibular problem in a child, then maybe genetic testing might be indicated, but only if that child presents with a hearing loss. How do we know that following a head injury, the hearing loss and the vestibular damage is because of the head injury and not because of a genetic testing? Usually in lateral. So this is something which you would not do routinely, but only in certain instances.

Vestibular spinal tests, Romberg, Unterberger, et cetera, central tests, different kind of central tests. There are 45 different abnormal eye movements, and I've got about 81 in my archive, which some of them I'm going to share you on the 20th. It will open a new world for you guys, I can assure you, the most interesting lecture that you would've heard. Static and dynamic posturography, and, of course, we have mentioned about hyperventilation. Different imaging. Now medical screening tests are important because you see, sometimes some problems might be subclinical. For example a subclinical thyroid, subclinical diabetes, because these patients might be elderly, subclinical autoimmune disorder. It's useful to do this, not to tell the court or anyone that this is responsible for the vestibular problem, but rather, these may be factors which lead to decompensation.

For example, a person with a head injury, with a vestibular damage with decompensation episodes, I do a vitamin D, and the vitamin D is very low. Now that is one of the medical reasons for decompensation, I need to treat all these things. You have to, you cannot, because dizziness is such a subjective symptom. The only way to objectify it or to put in some objectivity is by validated psychometric questionnaires. So you have got three here which I've told you; the dizziness handicap inventory, which is quite robust; the situation characteristics questionnaire, that is SCQ for visual vertigo; the Nijmegen questionnaire for hyperventilation, because these are very, these guys are very, very anxious. Now obviously it is important to do this test because you can measure this when you are assessing and following rehabilitation.

If there is a very clear improvement, then yes, you have got a case that the system is compensating. So at the end of this very extensive assessment, you should be able to diagnose the extent of the vestibular damage. You will be able to pick up vestibular decompensating factors. Many a times it's because of the cognitive and the psychological effects and the different comorbidities that will lead for you to develop an effective management plan. That's very, very important. Why are you doing all this? So that you can devise an effective management plan. Let's look at the complications. Cryptogenic. Cryptogenic means it's hidden. It doesn't come out because the patient either doesn't report it, or when the patient reports it it's very late, it's well entrenched with the full effect of all the different central processes acting on a perception of a vestibular problem.

So it's cryptogenic. And you know there is a subject out there in the world called cryptozoology, which is basically study of hidden animals. A classical example is Loch Ness, mythical creature, no one has seen it, at least some people claim, but there is no proof. So Loch Ness out there, Bigfoot in America, Yeti in the Himalayas, the Mokele-mbembe in the Congo jungles. So crypto means hidden, hidden from sight. And it's a real problem here because vestibular disorders following head injuries are cryptogenic. And the very sad thing is, this vestibular decompensation over years and years undergo a state of permanent decompensation. Because brain injuries or head injuries leading to brain injuries, they do generate other extra vestibular pathologies.

For example, the brain injury itself. For example, skeletal injury, limb injury, pain, whatever. These are all different factors which maintain the decompensation and that leads to some kind of a cognitive latching effect on your brain and you don't, and the subject is very difficult to come out, finds it very difficult to come out of the vestibular symptoms. And on top of it the vestibular problem itself generates psychological and cognitive effect setting up a vicious cycle. And it might lead to the 3PD stage. I've seen

a few 3PD, and that is one of the things which you would not want because 3PD is not easy to treat. I mean things are coming nowadays, but it's not easy to treat.

So, this is the message that I'm going to tell you, give you today, we'll have to work quick and fast. That's crucial. We are not saving a life here, but in order to make a life better, we'll have to work quick and fast. Because the more time will do, the more entrenched his whole vestibular symptom will get, the more liable it'll be to potential decompensation and mess up the person's life completely. Not only from the balance point of view, but also from the cognitive and the psychological point of view. So now armed with every information possible that you have, what I've told you, you'll have to devise an effective management plan. And post-head injury, vestibular rehabilitation is one of the most complex fields of vestibular management.

It's not someone coming to you with vestibular neuritis, you have diagnosed a beautiful vestibular hypofunction. You know, vHIT huge saccades, VEMPSs almost absent, huge asymmetry, beautiful Alexander's third degree nystagmus, and you send the person to a vestibular rehabilitation program, and there is a lot of improvement following the rehabilitation program. Unfortunately, post-head injury vestibular rehabilitation is not just that, it's far, far, far more expansive, it's far more holistic, it's far more, let's say, incorporates different disciplines to come under one umbrella and treat the patient. Now obviously as you can imagine that vestibular rehabilitation will involve not only vestibular cues but also significantly acting on the visual and somatosensory substrates as well. And that is a very intense activity from the brain.

So it's part of the holistic management, you must not tax the brain too much. Because if you do tax the brain too much that will lead to a fatigue. And fatigue, remember, is a natural complication of any brain injury. So whatever you do needs to start quickly, but it needs to be gradual and phased. In other words, you must not go bang right from day one an intense vestibular exercise regimen. No, no, no, no. You need to take it

gradually, gradually build up the tempo being mindful that all other comorbid things are also affected. In other words, vestibular rehabilitation must occur in tandem and simultaneously with neurocognitive rehabilitation and with neuro psychological rehabilitation and with musculoskeletal physiotherapy to improve the tone of your muscles, because vestibular spinal muscles play a very important role in maintaining the tone and power of muscles.

All of the functions of the vestibular system. So the first important step of vestibular rehab is, of course, accurate assessment. It has to be early but gradual and phased. Depending on what you have found, customized vestibular exercise. Treatment of visual vertigo or desensitization eyes with optokinetic exercises as well as with virtual reality. To improve your truncal tone and truncal muscles, postural stability exercises. All these are important. You have to treat the BPPV, of course. Ideally all these exercises need to be coordinated by a personal trainer, and occupational therapy needs to come in. Sometimes podiatry and sometimes behavioral optometry. For visual vertigo desensitization behavior, optometry is sometimes quite effective. And, of course, I always tell my patients, if you are keen, you can do things like Tai Chi and Pilates and all these things, Yoga, in order to reinforce your vestibular system even further.

You'll have to treat vestibular migraine and post-traumatic headache because remember they can lead to vestibular decompensation. You have to treat Meniere's disease and third window. PLF is an important complication. And sometimes I have seen in my career one case of superior semicircular canal dehiscence following a temporal bone fracture. So sometimes surgery might be offered if necessary. And very, very importantly, you cannot imagine vestibular rehabilitation without a cognitive and a psychological backup. Simultaneously the treatment. Why? Because you are trying to get rid of the factors which are responsible for decompensation. I'm sure that makes a

lot of sense. If you don't do that, just vestibular rehab, we'll have a far less favorable outcome than with all the other different modalities.

Pain is a very important factor for vestibular decompensation. You might have to ask your pain management consultant to take care of any pain. And if there's a skeletal injury, or an abdominal injury, or any kind of a hip injury, or you know pelvic injury, then the pain can be quite significant. And you need to get right on top of the pain. Of course you have to treat anything to do with hearing; tinnitus, hyperacusis. And very importantly what we do is called a situational counseling: that is to improve the self-awareness so that the patient himself can cope better with his balance problems. So this is what we have already said. So how do you assess compensation?

And this is very, very interesting and that's where my friend Enrico Armato has got a, has written a beautiful chapters in his book. So, of course, first of all, you can ask your patient, "Are you okay? How are you?" And he will tell you, right, but that's not an objective measure. But it gives you a good idea. Have I gone back to my hobbies? Can I do whatever I was doing before? Can I meet with my friends? Can I socialize with them? And, of course, some objectivity is given by the psychometric, for example, a DHI. When you first saw that patient after the head injury was let's say 82 out of 100, which is severe intrusion. And after your treatment and everything after 18 months, you have made, you have dropped it down to 40.

That's a good useful objective measure. And similar with other things. So sorry, it's not HAS, it should be HAD. You can of course use computerized static and dynamic posturography, that they are very important prognostic factors. They will clearly tell you whether the patient has got improved functional balance. Your vestibular spinal test might recover. Previously you had a Romberg, which was leading to the left, tandem gait leading to the left, but now the person can walk in a straight line. When you're doing the REHIT, a very useful compensatory indication is recovery of VOR gain with

clustering of saccades, so in the acute phase or in the decompensory phase, the saccades would be clustered, would be dispersed. The saccades slowly cluster and finally emerge into covert saccades, which are very good ways of assessing compensation, objective compensation.

A wonderful new test has come in order to assess compensation and that's a SHIMP. In SHIMP, we have found that the peak saccadic velocity when during the time of the first assessment is very, very low, less than 150 degrees per second. But following rehabilitation it climbs gradually up. And this was the patient I was telling you who I have done a report only today. His SHIMP after the injury, vestibular injury, was 123 degrees per second, dismal. And there's a paper to that, normal SHIMP values and abnormal SHIMP values after kind of vestibular pathologies. And when we did him again after a full set of rehabilitation, it has climbed up to 227, which is the lower borderline, normal range. So yes, it does improve and it's a good way.

And, of course, the asymmetry between the two sides, the bad side and the good side drops. In our laboratory the asymmetry was 15% plus is symmetrical. So previously the same patient has a 50% asymmetry, which then dropped, now at the moment it's about 20, 25%. So you can understand that objective compensation is going on. And why am I using the word objective? I'll tell you in a bit. The other very important thing, which is also identified by Tosh, is that VEMP amplitudes recover. And I'm talking about not absolute amplitudes but rectified amplitudes. This patient I was talking about, he had an absent right-sided cVEMP when we first saw him. And now after nearly two years down the line, with management, his cVEMP rectified.

cVEMP on the right side is now 2.8 microvolt. So yes, VEMPs do recover. And that is so crucial once again in order to understand objective compensation. And of course if you get a positive head shake, head heave, Alexander's nystagmus, mastoid vibration, ocular counter-rolling, all of them can show improvement once vestibular

compensation sets in. So you can make an reasonably accurate judgment that there is objective compensation going on. But, now nature, of course, throws more questions. It's not as simple. You might have a patient where you get wonderful objective signs of compensation, but the patient is as symptomatic as before. So DHI was 82, and you do all the tests, complete vestibular hypofunction, after two years, vestibular function has recovered significantly, DHI is 90, and the other way round.

So none of the vestibular function tests have recovered yet, the patient is completely symptom free. And this is because perception of balance and dizziness is a very much central cortical activity. And your central cortical activity is guided by the intensity of the brain injury that you had. So in some patients the brain injury is not significant enough, and it does lead to compensation as to how you perceive your own balance. So you might not have any symptom. And on the other hand, the opposite way is also true. Your brain injury will dictate how you perceive balance. So although your peripheral vestibular system has recovered, but your central hasn't, so you're very symptomatic. And there's a wonderful paper by Yuri Agrawal on this, who says that it's probably the tilt, you know, kind of the Romberg, the vestibular spinal tilt is the only consistent vestibular weakness that you can find.

And this is a very important legal challenge, legal point as well in a court of law, that I have demonstrated beautiful compensation from the objective point of view, the patient is very symptomatic. So I don't believe your tests. That's the way that you argue. It can happen. It's variable, it's heterogeneous, it can happen. But I must confess that in the majority, I would say more than 70%, 75%, they have got a good concordance to each other. They kind of agree to each other. And this is what I call a rehabilitation connectome, which was suggested to me by one of the best head injury vestibular specialists in U.K., Peter Savundra, who has actually taught me a lot, who has been my mentor, he has used this beautiful word called connectome, which shows you the holistic, multidisciplinary approach.

Now you might ask me that, "Well, so many different teams are involved, I'll do my bit, and that's it." No, as the neurotologist you'll have to coordinate all the teams along with your colleague neurotologist. So neurology and neurotology, the two key and pivotal experts are consultants who need to coordinate everything else. So look at the sheer amount of people involved, so many. And it goes beyond the medical domain, it goes to social services, legal services, employment, education counseling for kids. So it's a very complex topic and, you know, if you think you'll go into head injury or start seeing head injury without all this then you are not doing justice to the patient. You do have to look into everything.

Of course, for the legal bit it has to be as comprehensive. When you're seeing someone clinical, even then you'll have to take on board the vestibular damage, the cognitive and the psychological backlash, and what are the different teams involved in the care. And you need to be abreast of this, because remember at some point if it goes to court your evidence will be scrutinized. So it's not easy. It does take time and it's not just like your routine vestibular hypofunction coming to your doors due to any reason and a straight referral to vestibular rehab. The prognosis is variable and depends on the damage, the mechanism of the injury. And very importantly, as I keep on saying on the comorbidity, it must be guarded in the initial phase, but then you get a better idea after two years.

And with vestibular rehabilitation alone, there is at least a 50% chance that vestibular symptoms will recover. But if you can manage to address the comorbidities, there is at least 80% chance that it will recover. But the system will never get back to normalcy. Kids, adults, there always will be, 'cause it's a permanent damage, you know, and anything which will alter central brain function, even a simple stress, exam stress in children, that leads to dizziness and they end up in A&E. And that's not really the answer because you have to tell situational counseling that, "Look simple stress, cold,

pain might make you dizzy, take an anti labyrinth and sedative and rest it off because this is how it'll behave. Do not worry about it."

Very importantly, beyond the age of say 70 years pressed by vertigo, age-induced damage sets in. And you can imagine that this will complicate a person who's already got a vestibular damage. In fact, the chances of a person following a vestibular damage, following a head injury beyond the age of 70, they have got a 80% chance of falls. They can fall that can lead to injury, a hip fracture, a femoral neck fracture. So it's so important that you'll have to rehabilitate this persons. And not only that, increase their self-awareness, give them home exercises, ask them to do the exercise beyond the age of 70 diligently and routinely. This must be factored in the management package.

These are the factors which are adversely affecting outcome. Female gender, please don't ask me why, haven't got a clue. Previous concussion or previous brain injuries add up to the whole thing, of course. And dizziness which happens immediately after the injury and persists for more than two weeks is usually indicative of permanent damage rather than just a concussion: headache and migraine and other medical comorbidities. For example, you have checked the blood pressure if a person is hypertensive, not under control, that hypertension can lead to decompensation. So you'll have to take into account all different medical factors in order to prevent decompensation. So a few case illustrations. This was a severe road traffic accident with multiple fractures including a temporal bone fracture, which was a longitudinal one.

Complete sensory hearing loss on one side, left coup, which means similar side or ipsilateral cochleovestibular severe, severe damage. So out here what we are showing you is, so this was a left-sided vestibular damage, so this was a trace of a right-beating nystagmus, Alexander's. This is a post-head shake nystagmus. And this is a post

mastoid vibration nystagmus. So three different frequencies affected at the same time. However, on the video head impulse, it has no semi-lateral, semicircular canals. Lateral semicircular are pretty much conserved, but left posterior canal is showing very clearly low VOR gain and catch-up saccades. And I think this was the head roll, the ocular, no this was the BPPV test, lateral supine roll test, which showed right apogeotropic, less amplitude, right, sorry, more amplitude nystagmus than on the left side, which, of course, which made it a left-sided lateral canal BPPV.

So in other words, he had pretty much his cochleovestibular system messed up, and he responded quite well to treatment. Second case acceleration/deceleration shunting from behind very low velocity 10 to 15 miles, hearing is normal. And this was surprisingly a bilateral coup-contrecoup vestibular injury. Which only reiterates my point that you do not need a sufficient force to cause a vestibular injury. Brain injury, yes, but not a vestibular injury. So as you can see here, the left lateral, you get frequent, quite a few saccades. However, this is somewhat compensated because the saccade are a little bit clustered and even a normal VOR gain. There are two papers that normal VOR gain with saccades is a sign of vestibular compensation.

And as you can see over here, you do not get any oVEMPs. This is the oVEMP. oVEMP is absent on both sides and on the cVEMP it's the right-side which is absent where the left-side is normal. So this is a bilateral vestibular involvement with selective impairment of individual organs. This was a trivial injury child, he was trying to reach something from the fireplace and he fell. Hit his head at the back, immediately recovered, no loss of consciousness, no vomiting, absolutely nothing, no balance problems. Had a school entry screening where it was detected, he had a complete sensory and hearing loss on the right. This kid also underwent genetic testing obviously because we did not know.

So he had a cochlear vestibular injury. And you can see that this is a very, very good vHIT on a four-year-old, so we could see the huge catch-ups saccades. But look, anterior canal has been spared. Andrea Castellucci, my very good friend and colleague, has got a nice series about canal sparing vHIT. And this is a classical example of anterior canal sparing vHIT. But the anterior canal spared, which is mainly found in ototoxicity but also in head injuries as well. And when we looked into the CT scan very closely, there was a petrous bone fracture. You can see the fracture over here. But he compensated so well that he was completely asymptomatic from both hearing and balance point of view.

But situation counseling is important, if you do decompensate in the future, what to do. This was a very significant injury. Road traffic accident, multiple fractures, diffuse accidental injury proved by MR scan, GCS was 11, impact injury on the side of the head on the right, hearing was not affected. But, low and behold, canals were completely normal and there were central processing problems. So as you can see that this was a right-sided isolated saccular dysfunction, oVEMP was normal, and this was lack of vestibular ocular suppression, which comes right at the end. But I'm going to take you through the whole thing. So this was the first test that we did was an ocular counter-roll, when I'm trying to move the patient all the way to the right and the left, and you see that the eyes are hardly moving.

And this would be akin to a bilateral mid-frequency utricle saccular dysfunction. Static ocular counter roll is utricular, dynamic ocular counter roll is saccular. So let's have a look at the head shake. I think there is a head shake here somewhere. This is too long, so that's why I'm just, wait, let's see. Vertical head shake. And you'll see some catch-up perverted saccades. So horizontal saccades, you can see that saccades. Some horizontal catch-up saccades following vertical head shake. This is called the perverted head shake. This is roundabout the region of the fourth ventricle cerebellum.

So yes, quite a few central signs and isolated otolith dysfunction. That's very, very strange. Such a significant injury, only the otoliths were affected.

So I think I've managed to impress on you the vast complexities, the heterogeneity, and the very important caveat that part of your vestibular system might be affected after a head injury. It doesn't have to be the three semicircular canals, both otoliths, the nerve, it can be selective. And nowadays with the revolution, as Leonardo Manzari, my good friend, tells me, the revolution in vestibular diagnostics, you can make out exactly where the problem is. And why is it important? It is important to formulate your management plan. For example, that otolith dysfunction, that person will not benefit from gaze stabilization, that's classical semicircular canal. That person will develop, will respond to otolith rehabilitation, which is a separate set of nine exercises, which can be done with auditory feedback.

And there's a wonderful paper by a man called Basta. So you are diagnosing all these different kind of sites in the vestibular, peripheral vestibular system to formulate your management plan. And you are also identifying comorbid factors, psychology, psychiatric pain, medical problems, or that vestibular decompensation does not work. And of course you've got an active condition, for example, Meniere's disease or a migraine, you have to treat the active condition because active vestibular lesions they do interfere with compensation. So now let me take you to the legal domain, which if you are aware well and good, but I think many of you will not be aware. This is a very important sub-branch of vestibular injury following head injuries.

Because, you see, the vestibular injuries can mess up a person's life. And it's only fair that whoever has caused the injury is answerable and accountable for what he has done. So there is a level of compensation which is associated with vestibular injuries after a blow to the head due to any reason. So the first important thing which a court has to establish is liability. For example, you are driving in the road and I crash into you,

and it was because of my fault. So liability on my behalf has to be proved, which is, I am liable for that injury. And then once that has been proved, there is a thing called a burden of proof of causation.

So causation means, why am I having a vestibular damage? And the burden of proof usually in this country are two: one is on the balance of probabilities. So on the balance of probabilities means there's an at least 51% chance that that event has caused the injury. And the other burden of proof is beyond any reasonable doubt. Now, beyond any reasonable doubt is what you get in a criminal code of justice: for example, following a murder. These are civil cases. So your burden of proof is on the balance of probability. So on the balance of probability, my crash into your car, 51% has caused a vestibular damage. That's the second thing we need to prove. So after establishing causation, the next thing we need to look for is, how much is this vestibular damage intruding in your quality of life?

And there we introduced to important legal terms; significant limitation and restriction in life. So whatever you have been doing before the accident, you can't do. And based on how much you can't do, there are different grades of disability; mild, moderate, severe. And compensation depends on the grade of disability. I can't tell you the individual criteria because it's quite complex. But you could be in a position to exactly assess whether the person is disabled because of vestibular injury or not, taking into account everything, whatever I've told you. And they become quite complicated because they get sent to us very, very late. And the more late, the more delayed the referral is, the more complex interplay between different factors; psychological, neurological, everything, they kind of feed into the vestibular system.

So it's by no means easy. But even then with the vestibular diagnostics we have and a good history, you can still make out what's going on. And, of course, you'll depend on previous medical records. And then your role might be as a clinician, in which case

you'll still have to do a report which might get scrutinized. Or you can be appointed by a solicitor's firm as a medical legal expert, where your job as a vestibular specialist to give your expert opinion on different aspects of the vestibular damage. Where you have to answer liability, you don't have to answer liability, where you have to answer causation, that's very important. Causation according to you, the vestibular damage, the decompensation-compensation cycle, the comorbid medical factors and psychological factors, how much disabled the person is, intrusion in life, and then recommend management, and finally come up with a prognosis.

So this is a legal sequence, vestibular injury, claim for compensation. Both claimant and defendant appoint solicitors. both of us do reports. Then we agree and disagree in a joint statement. Then it goes, it can be resolved in two ways: one is outside the court, where the parties sit across the table and decide compensation. And the one which goes to court has to be fought in front of a judge with regular kind of a court process with cross-examination et cetera, which can be very, very intimidating. Because remember, if you are appearing from the claimant or the defendant, the opposite side lawyer or barrister, or whoever is fighting the case, is out to prove that you are discredited.

In other words, your evidence is rubbish, you do not know what you're talking about. It can be very intimidating. And believe me, I've been there. But what you need to do is to keep your cool. And look, most importantly, you should always speak the truth and nothing but the truth, and your honest opinion. If you think there is a BPPV, you'll say there is a BPPV. And these are the reasons that there is a BPPV, and this is the literature which backs it, and I'm going to stand by what I've said. Regardless of whether the barrister on the opposite side tries to discredit you, you mean to say there is BPPV and you can't prove it, so what kind of a doctor are you?

Yes, these questions do get asked. You must, it's an art. How to face very, very aggressive and intimidating cross-examining lawyer is an art. So you have to keep your cool for that. And the report is very, very voluminous. My average report for the court is 110 pages long with 102 reference because it has to be watertight. So there is no scope for anyone, either the lawyer or the judge to find a loophole. You'll find medical legal expert reports consisting of only 20, 25 pages. No. I have been taught that you'll have to be absolutely comprehensive and watertight. So references are very, very important: you have to discuss pretty much everything as related to the subject's life, including occupation, hobbies, et cetera, et cetera, et cetera.

So the report writing takes average 15 to 16 hours. And the overriding duty of the expert to the court is not who has instructed you or given you the commission, but only to the court, and only to the court. And you'll have to adhere to your expertise, which means you cannot comment on whether a brain injury is leading to significant cognitive problems. You can say, "Yes, there is a brain injury" because beyond that is beyond your expertise. You'll always expressly always express your honest opinion professionally with knowledge, with expertise, and by providing scientific literature: that makes your evidence watertight. And when you are cross-examined, you'll stick to what you have written. I've written that and there's a paper to it, that's it.

That's the important thing. And this is a statement which I'll have to do in every report, that my overriding duty it's towards a court. And, of course, this differs from different countries. So why do you have to become an expert? What are the pros? It's very intellectually stimulating, believe me, the two most important places, three most important places where I learn my trade, my knowledge. One: going to conferences and meeting, you know, world-famous experts, for example, Enrico, for example, Augusto discussing with them. Number two: reading updated journals and publications. And number three: doing medical legal cases. Because the moment I see something, for example, the inverted caloric, I immediately read about caloric. Pulsatile

tinnitus. The moment I find a pulsatile tinnitus, if it's not a third window, I read about the different genesis of pulsatile tinnitus.

So it increases my knowledge immensely. So it's very intellectually stimulating, it's expands knowledge and, of course, it's financially extremely rewarding. But cons, your family life might get out of the widow, very long hours, you'll have to work on weekends. So you'll have to have a perfect life and work balance. And cross-examination can be very, very intimidating. And look, I have seen this. If your report is catering to your instructing solicitor, maybe the claimant or the defendant, and you write things which you cannot substantiate just because you want to kind of do what your instructing solicitor has advise, if it's caught you will hauled towards your local regulatory body. You will complained against that you have falsified and you have lied.

And also advocacy, which means you are biased, you are not fulfilling your duty to the court but rather you're kind of more loyal to whoever has instructed you. So be very, very careful. You must not lie. You must not make up things, You must be surrendering. Look, this is what I found and I can't substantiate it. Perfectly acceptable, no problem. Yeah, you're not supposed to know everything. For example, one case I found today, the other day was, after a head shake test there was sustained square wave jerks. Never seen that in my life. He didn't have any square waves before. I've never seen that in my life. So I went through, at least spent two hours looking whether there's any evidence, there is no evidence.

So what did I write? I said, this is what I found, and I've got no idea why this happened because it's not found in the literature, and I haven't come across it with my experience. So this could be due to, he also had vestibular migraine, this could be due to vestibular migraine or deeper central, it's not established. However, if I'm appearing for the claimant, I'll say that, yes, this is a sure sign of head injury, which is a lie. And if

I'm playing for the defendant, I'll say, "It's nothing. It can be safely, clinically thrown out. It's not clinically significant." But I didn't write that. I said, "I do not know." Which doesn't mean that it's not nothing, it might still be something, but I do not know.

That's the honesty and the integrity and the probity that the court expect from you. It has to be absolutely watertight. And, you know, in my career it's very rewarding because sometimes your audio, your vestibular report can swing a judgment. Can you imagine a person with completely cooked up because of balance symptoms following head injury? No life, no work. You know, cognitively very down, emotionally upset, crying, depressed, anxious. Your report gives him the compensation which will take care for the rest of his life. You'll be only earning a pittance of that amount, but it's so rewarding that you have helped that man to go back to his life. This is an example, this is in public domain.

So this man, who's a very famous man, who was doing a program for BBC, and he was actually, he designed a rig where this, it was a kind of a carriage which was on a track, and it was going towards a collision point with a foam at about 15 to 20 miles an hour. So apparently the company he was working for did not do a safety check and risk assessment. So he did it three or four times, and immediately started having balanced problems, dizziness, cognitive, et cetera, headache, plenty of other things. There was no loss of consciousness. There was a little bit of post-traumatic amnesia, but he went completely downhill and loss of earning and capacity to work.

So his life was basically cooked, it was gone. He saw many, many, many doctors, clinically and medicolegally, and no one could agree that there was any kind of neurological injury because it was the concussion there. Scans have been completely normal. It was more than 2,000 page deliberation, and I had to go through every individual page. However, what I could find was, I confirmed an audiovestibular injury with objective vestibulometry. So his otolith function test were deranged, his later

semicircular canal tests, some of them were deranged, that's what I found. It did not settle outside the court, it went to the Royal Courts of Justice for a 14-day trial. The judgment for the patient based entirely on the evidence of an audiovestibular position.

And this is what the judge said about me, you can go to Google, type my name and find it. "It seems to me that Dr. Dasgupta doing exactly what is expected of an expert, analyzing all the evidence and prepared to reflect on the views of opposite number. I find that the claimant sustained..." Look, this is the court, and this is my language in the report. "I find that the claimant sustained subtle damage to the left utricle and semicircular canal as a result of the crash tests," established by my lab in an objective way. Now, of course, there was other things as well, pretty much, but this was the crunch of swinging the favor in front of this wonderful person who practically lost his life because of these injuries.

This is all what the judge said, "Impossible to view the audiovestibular problem in isolation. And waxing and waning because of intermittent decompensation-compensation episodes. And the reaction to the vestibular damage is disproportionate," which we mentioned, "is because of his other factors." So what was the net result? 1.6 million in damages. And that was so rewarding, you know, out of all this money coming, you might think I earned a whole lot of money after this. No I didn't. I earned very, very tiny bit. But I'm so happy for him that he could actually take this huge corporation to court and win it significantly contributed by an audiovestibular evidence. So please think about this. You are helping mankind when you're doing medicolegal work.

So just have a read. In fact, we are finished in one and a half hours, which is great, and we are certain to a certain extent. Yeah, management leads very rewarding outcome. Should not be ignored if there is strong legal implication involved. So any head injury might damage your GPS regardless of the force. And this movie beautifully illustrates it.

So this is your internal GPS system, which is essentially your vestibular system. So that's it, Enrico and Anna. So we can actually take the questions. Anna, are you with me?

- Of course, I'm here, Professor Dasgupta-

- Finished in one and half hours.

- Yes. Let me say that even this time your presentation was outstanding and inspiring.

- Thank you.

- Absolutely even this previous time. So Professor Dasgupta, if you wanted to have a look at the question from the audience. Or Enrico if you want to read the questions. It's your turn now. Enrico can you read the questions if you see the question?

- Yes, but, sorry Anna, I am not able to to see them.

- To read the question?

- No, no, sorry. No, no, no. No Windows to be able to read them.

- Okay, so most probably there is an issue, okay, a software issue.

- Yes, yes.

- Anyway, Enrico, if you wanted to attend and interact with Professor Dasgupta during the question, it would be great.

- Yes. Thank you, thank you.

- Professor Dasgupta-

- I mean, I wanted to to thank my friend for citing my mentor Erik Ulmer. Thank you very much for me, always very, very I important reference. Thank you.

- Absolutely.

- Okay, Professor Dasgupta-

- So Anna, how do I go to the questions?

- Yes, sure, please. It's your turn now.

- How do I, I just press on questions, do I?

- Sure. Just press the question folders and that's it.

- There are no questions it seems.

- No, there are a lot of question Professor Dasgupta. But if you want I can ask the question to both of you. Okay.

- Yeah, yeah please, please.

- Thanks.

- Sure.

- 'Cause I can't see anything.

- The first question is, oh my, God just a moment. Okay. I'm going to read the question as I see the question on the screen.

- Yeah. please.

- "Do you see a difference between sport-related concussions and domestic concussion on symptoms, finding and symptoms resolution?"

- That is a very, very good question. Now, the mechanism of the injury would be different in different scenarios. For example, mechanism of injury in a sport, when you are actually playing a physical contact sport is quite different from a road traffic accident or otherwise. What the general opinion is, yes, the sport injuries, when you get a head injury, that a goes more towards concussion rather than a long-term physical damage. Unless, of course, you get a solid blow to the head when you are clashing with someone in football, you both jumped up to head and all those things. However, with road traffic accidents, usually the chances of labyrinthine damage rather than a simple concussion is more. So that's what my practical experience is. And if you look into all the papers about sport related, they would always use the word concussion rather than damage.

- Okay, Enrico, if you wanted to add something.

- No. Absolutely agree. Agree, thanks. Excellent.

- Okay, so next questions. "If the vertigo changes direction, what can we do in terms of management?"

- Vertigo changes direction, means? How can a vertigo change direction? I'm not sure.

- I don't know also-

- Is it, a BP? Is vertigo changing direction? I mean, so, I mean.

- Maybe sensation. Sensation maybe is changing the sensor of the nystagmus maybe, but is not clear-

- Okay, yeah, if the nystagmus changes direction. So in other words, the eye movement changes direction. So I mean, of course, look, eye movements can change direction when you are actually checking because if it's a direction-changing nystagmus, it's a central rather than a peripheral one. So what I understand by your question is, if a nystagmus changes after a head injury. So you get something on day one, and you get something on day 15, what happens then? You'll have to make out why it has happened. For example, you know, I have seen patients where you pick up central signs over a period of time, which means that there is an evolution of a diffuse axonal injury which is causing this. So you'll have to accurately qualify and identify what this change of direction of the eye movement occurs.

And if you're talking about the direction of vertigo, which means is it a directional veer on the right or the left? Then of course, once again, you have to find out a cause as to why it has changed. And one of the important reasons why the direction of a vertigo can change if there is a concomitant vestibular migraine, because that messes up with the presentation, with the phenotype. That messes up significantly with the phenotype. For example, if you have got a right-sided labyrinthine injury after a head injury, right, and your otolith function is affected, you will get a right-sided subjective visual vertical tilt. But if the person has got vestibular migraine, the tilt might be actually on the right

side. So it puts in a lot of noise and it contaminates what your vestibular finding is. I think that's the best way I can answer it because vertigo changing direction is not clear.

- Yes, anyway, if our attendee would like to-

- Explain.

- Add more details and explain better her questions, we are here available to answer. Anyway Professor Dasgupta, our attendee Anna-Margarita Ammourine, okay, ask you, "Do you think a person with bilateral vestibular loss, even if compensated, can still be a truck driver?"

- Oh, fantastic question. Fantastic question. And this is something which the court will love, okay. Now this is a bit controversial because you see if someone has damaged the one-sided vestibular function following a head injury, right, and I'm talking about a damage, not about a concussion, so there is no recovery, then driving has to be taken very, very cautiously. And what I recommend in my report is, driving should be phased. Driving should be very, very gradual. First drive with familiar routes so that your visual engagement is not too much. Do not try complex navigational issues. And avoid nighttime driving, that's very, very crucial. So these are the things. Now, once you have started doing this over the period of two, three years with good compensation, as I have shown you how to measure compensational subjective.

If you are confident enough, then yes, you can venture out at nighttime as well with an escort. But following vestibular damage like this, you should not, or, you know, in my experience, you cannot go back to regular driving. You just can't. But some patients do. And if you are talking about a truck, no, I think that what I will recommend in my report is a permanent ban. You can't, you just, but of course remember it depends on

the extent of the injury. If you've got a mild injury, you can . I suppose we are talking about significant vestibular injury. Well, for example, if you've got only one canal involvement, just one saccular involvement, then you can go back. But just with significant vestibular injury I think truck driving is not recommended. That's me. You might disagree, you might disagree, but that's me.

- Enrico, you wanted to ask something.

- Yes, no, no. A very, very fast question. What about post-traumatic hydrops? Because I have assessed many patient with this clinical feature, but I don't know in U.K., but in Italy is difficult to create a link between the trauma and the hydrops. Many times I found other physician in the medical-legal controversy and it was very difficult to say, "Yes, it exists."

- Okay, so once again, Enrico, thank you. This is a controversial because some people believe that it doesn't happen, some people believe that it do. Now, as far as I'm concerned, I do believe it does happen.

- Me too.

- Now the onset of the secondary endolymphatic hydrops has got nothing to do with it because it can happen immediately after, or it can have happen after six months. So my take is, in order to prove to the court that it's because of the head injury, you'd have to satisfy that on the balance of probabilities rule, which I don't know what happens in Italy. So look, someone comes to me with a secondary Meniere's, some kind of Meniere's. I make a diagnosis of Meniere's, I do all the different possible investigations, I try to figure out whether it's a retro cochlear lesion or whatever, all the different tests for vestibular weakness. I do everything. And then I also run an autoimmune disorder to figure out whether there's an autoimmunity.

So I've excluded pretty much everything known. I'll still not call it idiopathic and successfully plead in front of a court on the balance of probabilities because there is a head injury. This can be a secondary endolymphatic hydrops. You see what I mean Enrico. In other words, I would say, without the head injury this could well be idiopathic. But because there is a head injury, I think based on the balance of probabilities, the chances of this, because of the head injury, is more than not. That's the way I'll plead. And I have done it in the past. For example, perilymph fistula, I've had a perilymph fistula which could be spontaneous. How do I prove it's not? I said, because the head injury is there as a result, on the balance of probabilities, there is an increased chance of this because of the head injury then not. That's the legal parlance that I use.

- Yes, thanks. Excellent.

- Thank you Professor Dasgupta. One more question from our audience, Christine Rogers asks you, "Can you give the reference for the specific otolith vestibular rehab?"

- Yes, so Anna, I'm going to send it to you.

- Sure, thank you very much.

- It's by gentleman called Basta who has proposed this, and then it was replicated and it was with auditory feedback. There are nine exercises.

- Fantastic. So Christine, it would be great if you could send an email to academia@inventis.it and tomorrow I will send you the reference as I get it from Professor Dasgupta. Thank you very much. One more question. "Can head injuries lead to central pathology or peripheral dysfunction?"

- Yeah, that's what I said. That head injury does lead to a central dysfunction. So it can lead to a traumatic brain injury where your brain function is affected, which can then affect, of course, the central vestibular processing pathways. So yes, most certainly it is a central injury. A head injury is central injury, just because the inner ear is in the head. That's why it gets caught up when someone is, has a blow to the head or because of acceleration-deceleration. Now as far as, what was the next second part of the question, Anna?

- The second part of the-

- Just read the question again please.

- Yes, unfortunately I've already deleted the questions.

- Okay, so I think this person, Christina, she was meaning whether a head injury can lead to central problems and peripheral problems. Yes it can.

- It can lead to both.

- In rehab.

- In?

- Rehabilitation.

- I mean, no, rehabilitation, it should not lead to any problem in rehabilitation. Rehabilitation can be done very effectively and successfully.

- Fantastic. So if I were attending-

- But one thing I'd like to-

- Write again the question. Yeah, one thing I'd like to insist that vestibular rehabilitation is not the only rehabilitation you're looking at. You're looking at a composite, holistic rehabilitation; vestibular, psychological, psychiatric, musculoskeletal, neuro physiotherapy, postural stability, everything.

- Okay, perfect. "Is there a time limit between head injury and the appearance of audiovestibular symptoms?"

- Okay, that's a good question again. And frequently I have entered into debates with my counterparts because many experts will say that onset of vestibular symptom, or audiovestibular symptoms, is very important because that tells you whether it's due to the head injury. For example, someone gets a blow to the head and the first thing when he comes about or if he hasn't lost consciousness, that he feels very, very dizzy, he loses his balance. So, and he also gets a very positional vertigo, for example, whenever he moves his head, tries to get down to bed, classical BPPV. In that case, because of the proximity of the symptom to the head injury, the onset, then it's very surely caused by the head injury.

It's very clear, crystal clear. No one will dispute that. However, if you get someone presenting to with a BPPV six months after the head injury, what will you say? Is it because of the head injury or is it because of a spontaneous one? Thankfully, all the vestibular complications that I showed you; Meniere's, perilymph fistula, BPPV, all of these can have a delayed onset, which means they can happen six months to a year. You know this gentleman who I showed you last, the one who won against BBC, developed a BPPV eight months after the injury. And my defendant's expert was very

adamant, this can never be linked to the original head injury. I said no, there is a paper by a man, a very, very, very, very big expert on BPPV called Balatsouras.

He conclusively said that there can be a delayed BPPV after a head injury. It's to do with otoconial gradual, otoconial degeneration. So the immediate onset is because the otoconial crystals are physically moved, right. You get a blow, bam, boom, you get a blow and otolith particles dislodge. However, the delayed onset BPPV is explained by gradual otoconial degeneration, which sheds the particles over a period of time. Same with vestibular migraine, gradual rapid nucleus firing, which happens after some time. So yes, it can happen after six months to a year. And if you can prove it with references and evidence, you can win in front of a judge. That's once again bringing me back, whatever you say, it has to be backed up by solid evidence, and you must never lose your ground. You might, you will not say that, "Yeah, this BPPV previously I thought it was because of the injury now I think it's don't." Stand your ground if you can. Does this make sense? I'm sure it does.

- Enrico, would you like to add something?

- Yes, another question to Soumit. Which the mechanism between the trauma and the superior semicircular canal dehiscence. Why?

- Yeah. That's a good question.

- A concussion without any fracture?

- That's a fantastic question. So the only superior semicircular canal dehiscence that I have found following a trauma was in a child, Enrico. And you can very clearly understand, in a child it's a developing system, so the bone is not very thick, right. It's very easy.

- Yes.

- However, in an adult it has also been reported. So I think these adults, they kind of present with the congenitally thin superior semicircular canal roof, which is liable to rupture. That's what the theory is, but I have never seen one.

- Okay

- But in kids it's easy. In kids, a 6-year-old kid, the bone is nowhere as thick as a otic capsule of a 16-year-old. So yes, it can rupture.

- Thanks.

- Thank you Professor Dasgupta. "Hi from Paris, and congratulations for the fantastic presentation. What do you think about vestibular agnosia in concussion? Well described by your colleague from London, Dr. Seemungal."

- Barry Seemungal.

- Okay, sorry.

- Barry Seemungal, yeah. Vestibular agnosia is slowly getting recognized. In fact, it was Barry's team who identified that 25% of vestibular problems are cryptogenic. And they do great work with head injuries. Yes, vestibular agnosia is definitely a possibility, it can happen. However, the way you diagnose it is still very much in its research phase. So I wouldn't kind of diagnose a vestibular agnosia without doing all those tests, and I don't have facilities for those tests. But yes, this is something central vestibular perception

that is slowly coming into the fore. And you can imagine vestibular agnosia might happen if there is a concomitant brain injury, of course it can.

- Okay, thank you Professor Dasgupta. One more question from Paulo Cardoso do Carmo. Hello Paolo. "Hello Professor Dasgupta, and thank you very much for this fantastic presentation. Is there any reference of cut points in vestibular dysfunction in CSC or otolith to limit patient work?"

- Sorry, can you repeat the question please?

- Yes, so I read the question as I can see it from my screen. "Is there any reference of cut points in vestibular dysfunction in CSC or otolith to limit patient work?"

- The first part of the question, plug points?

- Cut points. Cut points.

- What points? Can you just spell it out? I'm not getting it.

- Cut point. So Paolo said, "Cut point," C-U-T.

- Oh, cut points, right.

- Cut point, yes.

- When you are assisting a patient with a vestibular damage, whether how it has affected work and when he can go back to work, the timeframe, or when you cannot go back to work is a very careful decision you need to take. So as an example, for example, if a person has made an 80% to 90% recovery, which means a good near to

total recovery, even then 10% residual symptom will still be there. He might still decompensate in the future. In other words, it's permanent. You can never expect a complete recovery in a damage. Concussion is different. You can never expect a complete recovery in damage. So basically, as far as work is concerned, what you will recommend, he is okay to do anything he wants, but he must avoid working from heights, you know, wherever there's a significant provocation of the vestibular system.

Now, if someone is much less than that, for example, only 50% recovery, then you'll have to make a judgment as to what kind of work will be suitable for him. For example, desk jobs. For example, work not in involving manual labor where he can decompensate, he won't be able to do it. So it's a careful judgment of how exactly the patient presents and how much of his vestibular function he has recovered. But I haven't seen anyone in my cohort where they are completely jobless after a vestibular injury, however severe it might be. What I therefore write in my report is in the open job market, which is any job, this person is not at an advantage, disadvantage, but there would be jobs where he can still work.

But remember what the problem is Paolo, the problem is fine, you have cleared him from the vestibular point of view, yes. So 50% recovery, dizzy, imbalance, you're sitting in front of a computer, hardly any glare with a blue screen. You are doing some basic accounting work and stuff like that, you have got a job. But even then you can't function because remember you are cognitively messed up. You are not able to plan, you are not able to organize. So the way you write your report is very careful. You have to write from the vestibular point of view, I anticipate there will be some return of function and some possibility of taking up jobs. However, whether he will be able to continue the jobs depends on his cognitive and psychological get up, which is not my expertise.

There's nothing you can do. But from the vestibular point, if you make a good recovery, there are a few jobs which you can do, and a few jobs which you shouldn't do. So the cutoff is how much vestibular system is affected.

- Okay, I cannot see other questions from the audience. So Enrico.

- Last, one. During bedside examination, and after whiplash injury did you ever see up-beating nystagmus only under the goggle?

- Yes.

- So completely inhibited by fixation. It is not paroxystic, not never paroxystic, stationary and long-lasting, four or five degree per second velocity, mainly in the supine position, right side, left side, a little bit less in a spontaneous upright position. Because for me is frequent.

- Yes, I will tell you the trick. Yeah, Enrico. You know, the first time I saw this was in a 16-year-old person where absolutely beautiful eyes. But the moment I took the optic fixation, beautiful upbeat nystagmus. And I was at my wits end what is going on, I didn't know. Because it should be the other way, right. If it's a central nystagmus, why should it heighten the vestibular nystagmus. My point was, so this patient was sent to me with lightheadedness and a vague ataxia and some kind of intolerance to light. So I shopped around literally, I asked many people, many, many people, and then I don't remember who, but that gentleman told me that, "Have you thought about an atypical brainstem migraine?" I didn't. So I put that patient on propranolol beta blocker and the upbeat nystagmus was gone and completely symptom free.

- Completely.

- So I would say Enrico, central nystagmus, which gets heightened by optic fixation, by removal of optic fixation, is very likely to be a migraine. That's the only thing which fits. The only thing.

- Thanks.

- Try try some anti-migraine pills and you'll see the difference. I actually will, next time when we meet Enrico, I'll show you that video of that girl. Fantastic upbeat nystagmus.

- Okay Okay, thanks.

- Okay, so in the meantime, I'm getting a lot of messages just saying, "Congrats Professor Dasgupta." Thank you very much, Dr. Armato,

- Thanks.

- Thank you.

- And so forth. So I think we can conclude this session. So thank you very much Professor Dasgupta for your presentation. Even this time it has been an outstanding learning opportunity.

- Thank you.

- And thank you very much Enrico, our clinical reference-

- My pleasure.

- For your collaboration as usual.

- And thank you Enrico, my old friend, for such a kind introduction, and of course to you, Anna, for this opportunity.

- Thanks. Thanks, I really-

- Keep in mind that we'll meet Professor Dasgupta again next April 20th, for the third and final seminar of this fantastic series, "Centralized Movement Disorders: a Window to Brain Function."

- Now that will be a very interesting lecture, yeah.

- Sure.

- I'm sure, I'm sure. So for the audience, remember you receive soon a survey, which will help us to understand your needs and your suggestion. Thank you very much, Professor Dasgupta, once again,

- Thank you, Enrico.

- Thanks Anna.

- And see you next April 20th-

- Thank you. Bye-bye.

- Thank you all to all attendees, thank you. Thank you very much.

- Thank you all attendees for giving me the time, thank you. Bye-bye guys.

- Thank you, bye-bye.