# Identifying Vestibular Loss in Head and Brain Trauma

**Presenter: Amy Lennox-Bowley**

Sam hello and thank you for joining our E Seminar today. Whether you're a regular attendee or new to the Hearing and Balance Academy, you are very welcome and we hope you get a lot out of today's session. Just some brief housekeeping advice to get us started. You've joined today's session as a listening and viewing participant only, but we strongly encourage you to use the Questions Box facility located in your GoToWebinar widget on the right hand side of your screen. This session will run for approximately one hour, including some time at the end for some of your interactive questions.

If we don't have time to get to all your questions, please don't worry as we'll make sure you're contacted after today's seminar. If you're joining us from a mobile or tablet device today, you may need to swipe right or left to view the presentation. There is also a copy of today's presentation in your handout section. On behalf of the Hearing and Balance Academy and Natus Medical Incorporated, welcome to today's online seminar Identifying Vestibular Loss in Head and Brain Trauma. My name is Chris Versakeli and I'm the Global Medical Education Manager at natus.

We are delighted to welcome our guest lecturer today, Amy Lennox Boley. Amy is the founder of Hypatia Specialist Clinics and Hypatia Training, which aims to help improve vestibular care by providing innovative clinics and training for clinicians worldwide. After completing her Bachelor's degree in Audiology at the University of Manchester in 2009, Amy worked in the UK National Health Service until 2011 when she had the opportunity to emigrate to Australia. Here she completed her Master's equivalence and became Audiology Lead in Queensland, looking after patients in the public and private sector. In 2012 she joined Otometrics as Clinical Lead for Australia where she worked on research and development projects for the ICS IMPULSE and produced the interpretation guide for Head Impulse testing.

Alongside working for otometrics, she developed the Vestibular Forum, an online community for multidisciplinary clinicians, and regularly hosted vestibular and electrophysiology lectures at La Trobe

and Flinders University. She is currently researching visual vertigo, otolith dysfunction and vestibular injury in NTBI and has been providing patients with innovative techniques to help relieve these symptoms and is evolved in a number of multicentered research projects. Thank you for joining us today, Amy. Please feel free to start whenever you are ready.

Wow Chris, what amazing welcome and introduction. Thank you so much. And thank you so much for inviting me to be here. So, yeah, I don't need to introduce myself anymore because Chris has already done such a great job. It's so weird hearing everything back that you've done in the past.

So, yeah, let's get into it. So I've been asked just to go over some of the work that I've been doing at one of the clinics, well, my clinic in Liverpool in England.

So there's some exciting stuff because there's like a lot of new and innovative things in this industry that you can use and apply to these patients. And it's been quite a journey, I have to say. So let's start by looking at our learning objectives for today.

So we're going to examine different forms of head injury and how that they're categorized. Identify common types of vestibular disorders associated with head trauma and the mechanisms behind them. Discuss the complex nature of patients presenting with both NTBI and vestibular disorders. Review and evaluate any vestibular assessment strategies and the usefulness and implement. Talk about implementing a dedicated and personal vestibular rehab plan for this patient cohort.

Now, there's a lot to go into in an hour, so I have summarized a lot of stuff, but you are very, very welcome to contact me if you want any more details of any of the topics that we discussed today. So I'm just going to start by playing you a video that was created by Syntropy Studio. Instead of me going through the anatomy and physiology in 2D, they've put a really good 3D video. It's about, about a minute and a half long. So we'll just play that for you, Sam.

It's so a beautifully done video there by Syntropy Studio. And what's important to remember is that you can have head injury that is both on the coup side, so the side that you've had the direct injury, but you can also get contra coup as well. So often we find patients where the hit of the head has been on this side, but actually all the injuries are on this side, both vestibular and brain. So let's talk about the causes of tbi. And I just want to point you to the pie chart on the right because I think a lot of you will be working with vestibular patients already and look at the biggest cause, it's falls.

And so, you know, we often get patients post fall, of course, but I always think it's very important to talk about prehab and not just rehab. And there's obviously a need there with falls prevention to try and reduce the number of falls to reduce the number of TBIs caused by it as well as the other injuries. Motor vehicle accidents, that's probably the biggest cohort that come through my clinic. But we also get a lot of headed like we've had a few where poles have fallen on heads or had shutter fallen on ahead assaults struck by and against and then other or unknown on the left. I thought this was interesting to put in because the DoD in the US is very interested about TBI and they talk a lot about blasting.

So it's being recognized as an important form of traumatic brain injury in both civilian and military populations. And the recent estimates indicate that 10 to 20% of the 2.5 US military service members deployed to Iraq and Afghanistan have been affected. And to go into a bit more further detail of that, here's the numbers from 2000 to 2020 2022. So you can see I've broken down. Well, they've broken down here the different levels of traumatic brain injury.

And I'll explain how to you in a second. How they're all classified, but you can see that's 472. That's nearly half a million people who have been affected by a traumatic brain injury. Now, thankfully, the biggest number there is mild. But even if you have a mild traumatic brain injury, your symptoms can be still quite severe.

It affects your sleep, your mood, your cognition, your memory, your planning, etc. So even though it's mild, and thankfully most people in the TBI world are mild, there's still significant hurdles for these people to overcome.

So I just want to talk a little bit about the different definitions and what is a head injury? I'm sure many of you have spoken about or read about concussion as well. And then there's traumatic brain injury. So head injury. Head injury talks about where there's damage to the head but not necessary damage to the brain or disruption to the brain function.

So if you bang your head and you get a bit of a laceration and there's no obvious damage, well, there is no damage to the brain, then that's just a head injury. Concussion. Concussion is a mild traumatic brain injury. So they do overlap. And then traumatic brain injury can be both concussion or moderate severe and impermanent brain injury.

Traumatic brain injury. What's the definition of traumatic brain injury? So it's traumatically induced structural injury and physiologic disruption of brain function by the result of an external force. And it can be categorized by both a loss of level of consciousness, so loc, or if a patient has poor post Traumatic amnesia, if there's any alteration in their mental state at the time of the injury, that is measured on the Glasgow Coma Scale, which is actually the gold standard of how you grade the levels of traumatic brain injury. And often, if you're getting a patient referred to you, you might see what their original Glasgow Coma Scale was.

So here, this is how it's categorized. The physician at the time needs to monitor the patient's eye opening, their best verbal response and their best motor response, and then scores it based on what they get from these categories here. So anything 13 plus would be a mild TBI or a concussion patient is usually well enough and like awake and functioning. And this covers 75 to 80% of all traumatic brain injuries. If the patient has a GCS score of 9 to 13, this is classed as a moderate TBI.

Patient is normally initially quite lethargic or straporous, bit confused, bit more dazed. So this is showing more kind of severe signs than someone who is more awake and chatting at the side of a football pitch, for example. Then we go on to talk about severe traumatic brain injury. So this is the very unwell patients, they're usually comatose, unable to open eyes and follow commands, and they are very high risk for secondary brain problems, including hypertension, hypoxia, brain swelling. Often they can be put in induced comas.

And unfortunately, there's a direct linear relation on poor outcomes for these patients. And if your patient is over 60 years, that's an increased risk of, again, poor outcomes. But what I'd like to say here is we have had patients that have had these scores and have been over 60 and they've come into the clinic and been very poorly, not feeling like their quality of life is very good. And after a year or so of dedicated therapy, they've probably got their life scores up from maybe a 2 out of 10 to more 6, 7, 8 out of 10. So people can improve.

And you're not to write people off just because of the grade of the traumatic brain injury, for sure, no matter how bad they seem when they initially come in to see you. We've seen huge changes over time.

So we've talked about the Glasgow Coma Scale, which is kind of the gold standard of how you categorize traumatic brain injury. But let's just look at the loss of conscious and the post traumatic amnesia a little bit more, because those measures have been argued to be better at predicting your patient outcomes than the Glasgow Coma Scale. So for mild.

For mild traumatic brain injury, a loss of consciousness of less than one hour and PTA of less than 24 hours and then usually pretty much all the time, have normal imaging. With moderate TBI, it's defined as lost conscious between 1 and 24 hours and PTA of 1 to 7 days, they could have normal or abnormal imaging and then via TBI is less conscious for more than 24 hours and then PTA for more than a week.

And again, they might have normal or abnormal imaging. So what sort of injuries cause a traumatic brain injury? So there's an obvious one.

So blunt force trauma, direct trauma. So hitting their head on a car dash, for example. Then you have the acceleration deceleration injury. So three, think about like a whiplash type thing. So patient's got a seatbelt on, they don't actually directly hit anything, but the brain moves and has trauma from the brain being moved within the skull, and the brain might bang itself against the skull.

Then there's the shockwave injuries, which is more to do with like blast injury. So explosions, etc. Direct, again, that's repeated from what I've just said, Indirect whiplash is a good example. Another good example of indirect is, you know, say, for if a sport like a rugby player, if they are tackled and say they're tackled by the ankles and they fall forward, they might not necessarily bang their head, but the shock wave that goes through their body from such a aggressive tackle can actually cause a traumatic brain injury. And I think over the years there's been plenty of research and people talking about, especially in America, the NBA players getting repeated concussions or mild traumatic bone injury from these indirect.

Indirect traumas. Okay, we're nearly there with the brain injury and the different types and how you categorize them. So, so focal, if you read about focal injury, that again, is just a direct injury. So it's in a specific location. So the ball hits them here and then they have.

The damage is measured and found here. Diffuse injury occurs across a more widespread area, and it's common for both focal and disfuse damage to occur as a result of the same event. And my experience in patients who have vestibular damage, and they're seeing me at my stage, which is quite a late on stage, they usually always have a diffused injury. I really love this slide. This is from Mayfield Brain and Spine.

And it just shows you about the different areas of the brain and how if there's normal function, what you can kind of expect those areas of the brain to be responsible for. And then if that area of the brain is injured, what can happen? So I think, because we're talking about dizziness, we can accept that there'll be dizziness and that's usually coming from the brain, sten and cerebellum. But a lot of our patients come in and they have terrible cognitive issues like planning their day. We have special letters of how we write out to the patients, talking to them about what to expect from their appointment.

So their, their appointment letters are actually quite long and it talks, there's like pictures of the clinic, what to expect, pictures of the people that they might meet.

I've noticed that a lot of these patients can be quick to emotion or quick to temper. So that's probably, that's linked to the temporal lobe. So I think you just have to be aware that there's, especially with the diffuse injury that could be across multiple parts of the brain, that you've got to be kind of sympathetic to all these different impacts that the patient might be struggling with. And we've had patients just not turn up to their appointments because they've just totally forgot and then they'll go, oh, I did write it down, but I still forgot. So you just got to be aware of this.

And we've got some systems in place now to help help minimize those type of problems. But if you're going to start taking on patients with traumatic brain injury, you just have to do a lot more pre planning with them with your department, with your service, and also have maybe more time for them when they are in the room as well. Right, so let's get into why we're here. So dizziness and TBI. So it is a high complaint in acute TBI.

So, so 83% is the figures that are put in the research.

But after five years there's consensus that there's still patients that will talk about non rotational dizziness, lightheadedness imbalance in about 37% of patients. There are the ones that I grab at my

clinic. I seem to get the ones that are two plus years post injury and they tend to have kind of those more vague symptoms of dizziness with tbi. And if you look at the TBI studies, it says more than any other symptoms, dizziness and vertigo has independently been linked to the poor recovery and disability outcomes of TBI patients. So it is very important that those of us who have capacity to measure the vestibular system and to help rehab it are aware of these links between the TBI and dizziness because I'm not sure that that's always the case.

So I'm going to show you another video now that it's only again, two minutes long. And it's just to overview the vestibular system. So I'm sure most of you watching this are very familiar with the vestibular system, But I just want you to really focus on when it starts talking about the otoliths and think about that shearing that you saw when the brain has an injury. When you seen the original video about tbi, look at the otoliths and how they work and think about how like a acceleration deceleration injury might affect those organs. So yeah, we'll play this now.

The human ear is divided into three the external ear, middle ear and inner ear. The inner ear contains the spiral shaped cochlea where sound waves are transduced into neural signals and the vestibular complex which contains the receptors for our sense of equilibrium. The central egg shaped cavity is the vestibule, which contains a pair of membranous sacs, the saccule and the utricle. Inside the utricle and saccule are hair cells similar to those in the organ of Corti. The hairs are clustered in the macula, where their processes are embedded in a gelatinous mass and lie under a thin layer of crystals called otoliths.

When the head tilts, gravity moves the crystal mass and distorts the stereocilia of the hair cells. This is how the saccule and utricle provide information about position with respect to gravity.

Behind the vestibule is the third portion of the bony labyrinth known as the semicircular canals. The canals project from the posterior region of the vestibule and are responsible for the detection of headphones motion in three spatial planes. The anterior duct senses forward and backward motion.

The posterior duct detects moving up and down. The lateral duct senses moving left to right.

Each canal contains a membranous semicircular duct where angular momentum is sensed. At the base of each duct is an expansion called the amplitude.

Within the ampulla, long stereocilia of hair cells are embedded in the cupula, which sticks out into the endolymph. When your head moves, the endolymph moves the cupula and stimulates the stereocilia.

Okay, so now I want to talk to you about specific vestibular conditions that are known to be linked to traumatic brain injury. So the first one is post traumatic bppv. So many of you will know what BPPV is. But just to go over it, in the otolith organs, the utricle and saccule are otoconia. And post a head injury, those otoconia can become displaced and fall into the semicircular canals.

Now, they shouldn't be there. And what happens is when they move, they stimulate that canal. And patients will report things like they go dizzy when they roll over in bed or they go dizzy when they move their head in certain positions. Now, BPPV is the most common cause of dizziness in most demographics of patients. However, in the TBI demographic, probably not so much.

And when you go ahead and test for bppv, what you're doing is you're putting the patient into a triggering positioning position depending on the canal that you think is the problem. As a result of that, you'll get a nystagmus, which I will play an example here for you. So you'll see in a second, I'll tell you when the patient's being put in position. So lots of eye blinks, the eyes moving as expected. Now they're being put back.

So there was a few jiggles, but that's nothing so far. And in a second you're going to see an upbeating rotational towards the left ear nystagmus. So that's a left posterior canal bppb. It'll fatigue over time, which is another diagnostic factor. So if it fatigues, you can be pretty confident that it's canalothiasis,

which basically means that the otoconia is free floating within the canal.

If this was your patient, you would then hopefully go on and do an Epley manoeuvre. So there's different repositioning manoeuvres that you can do to treat them. So in my clinic, we love the Apley manoeuvre. There's many different manoeuvres you can do. Now, if you look up all the manoeuvres, I think there's about 64 or up to.

So, yeah, so that's post traumatic BPPV. How common is it in the TBI population? I tried to find prevalences for you. Research is very limited on people specifically looking at TBI and vestibular, so I didn't have many studies that I could pull, so you all need to get to work. But this one was really good.

This is from colleagues in Kerala. They took 128 patients over two months and they monitored kind of. Was there a BPPV present for these patients?

They had 17% of post traumatic BPPV was found and then what they. So they treated that and then what they did is they followed them up in six months time and when they reviewed them, 40% of those patients, unfortunately it had reoccurred. Now What I've noticed in my clinic is if a patient does have a bppv, that seems to be our highest prevalence of reoccurrence as well. So I'm not saying they all reoccur, but they are definitely more anecdotally, they are definitely more likely to have a recurrent. And I don't know whether that's to do with underlying damage that's happened in the utricle.

Who knows? If anyone knows, email me and please tell me the reason. Then there's labyrinthine concussion. There is a lot of studies talking about labyrinthine concussion, and this is damage to the inner ear due to head trauma, but there's no obvious skull fracture there. Now, most of these patients, well, all of these patients will have a sensorineural hearing loss, but it can come with or without vestibular symptoms.

So they're not always. Doesn't always mean that they'll have a balance problem. What's interesting with labyrinthine concussion is that the effects may be immediate or they could happen days after the event. So when you're doing your history and you're trying to pull out from the patient when things started, if there was a head injury and they tell you, oh, but my hearing loss was days after, don't rule that out. That that wasn't linked for sure.

There's loads of interesting studies trying to talk about the different mechanisms of why a hearing loss would appear after tbi. I think that all of them pretty much have rationale and I think it just, again, depends on how the force happened. And so you can read things up from reverse traveling waves that affect the outer hair cells to cochlear cyanotopathy. So again, there's a lot of detail there that you can go and have a little read about yourself. Probably again, just too much detail to cover in this hour, but the information is out there.

So do have a look. And this study that I've quoted here at the bottom is a good one. That kind of overviews all the different reasons that people have talked about why this might happen.

Traumatic endolymphatic hydrops or post traumatic Meniere's disease. So this might happen because of a few reasons. A fistulization of the bone labyrinth which causes disturbance in the relationship between the parilymph and endolymph. Makes sense, right? Direct injury to the membranous lamp labyrinth, which causes fluid in the cochlear duct from irritation, inflammation.

That seems to be maybe more of a temporary problem rather than a. Like a permanent Meniere's disease or endolymphatic hydra and then injury to the endolymphatic fluid drainage system, which. Where the temporal bone fracture might come across the vestibular system. And so that can be more permanent.

Now, when I was reading about this, I was like, why have I not seen any of these? So I then panicked and started looking through all my series of patients to see if we'd missed any. But we definitely haven't. There's only seven publications since 1962, and those publications only talk about one or two patients. They will present exactly the same as a typical Meniere's disease patient.

And I think maybe these patients are probably quite easily picked up in emergency ENT or in an ENT department because everyone knows about Meniere's disease, right? So I think that these, these might actually fall within just typical Meniere's disease demographic and might not always. The trauma might not always be linked if it's just been an obvious, you know, there's fluctuating oral symptoms, a hearing loss and dizziness. Okay, so let's talk about superior semicircular canal dehiscence. So this is known otherwise as a third window syndrome.

And this is where there's like a thinning or a hole in the, usually the superior semicircular canal. Now, when TBI happens, it does happen in the superior semicircular canal, because what happens is you get a temporal bone fracture that cuts through the superior canal. And you can see that on that image with the arrow pointing at it. That's where the crack is, just above the superior canal. Patients will present, usually with very unusual trigger symptoms.

So they'll go dizzy if they laugh, sneeze, cough if they lift any heavy. I've had patient who's been in the shower and they've heard the shower drops on their head. Echoes. If you stick a tuning fork on these patients ankles, they'll actually hear it in the head because it's attenuated through the bone conduction. So they will often have a conductive loss, usually low frequency.

The example I've given here is actually from this study, but often you'll see like a low frequency conductive and then it becomes sensorineural. And for those of you who don't work with vemps, what usually happens is if there's a conductive loss, hearing loss, you won't get a vemp. And then in these patients there's a conductive loss, but you do get a vemp. And that's because the sound for the vemp is

attenuated. By that bone conduction rather than.

Rather than the middle ear suppressing the sound so the sound can't get through for us to get our vemp response. Again, it's been very rarely reported in TBI literature. I think there's one or two studies that talks about it specifically, but it definitely does happen.

Perilymph fistula. So the Morani Society do not have a diagnostic criteria published yet for perilymph fistula, but this is probably the most sensible proposed criteria. So you'll see there's a fluctuating or non fluctuating hearing loss. They can have tinnitus, oral fullness. What's this sound like?

Sound a bit like Meniere's disease. Endolymphatic hydrops. Right. And you'll see that they need. It needs to be preceded by one of the four following events.

So barotrauma, direct trauma, which is our tbi. And then they need to fulfill criteria A or B. So laboratory testing or observation of perilymphatic leakage in the middle ear. So that was always the gold standard. The gold standard was open them up and have a look and see.

Can you see it there?

And now you can actually use laboratory tests. What laboratory tests can you use? You can use imaging and you can use vemp sometimes will pick it up and also the fistula sign or the Hennebert sign, which I'm going to show you in a second, that can also pick it up. The problem with perilymph fistula is it can vary. So the perilymph will drain out of the cochlea into the middle ear and then so that once it's drained, you might have like the hearing loss, but then it'll come back once it refills.

So it's very up and down as to whether you'll get the same test results one day to the other, or you'll get the same imaging results one day to the next. And this was also why the argument of opening

somebody up is not always the right answer. Because if you open them up on a day where it's not leaking very much, you might not see the leakage as well as other reasons. So if I just show you this lady down here, she's got a positive heading, birth or fistula sign. So basically what you do to elicit this is you close off the suspected ear.

So you're going to close it with some pressure. So hopefully you'll see the doctor here. This is Dr. Sian, so thank you for this video. So he's going to press in here and you'll see a nice stagmus. It'll beat towards her right ear that she's pressing now.

It's not very clear at the moment, but he'll zoom in on her eyes and you'll be able to see it much better in a second.

So, yeah, so that's a positive Hindenburg sign.

Otolift dysfunction. So otolith dysfunction seems to be quite linked to TBI and studies are looking around 30% for TBI and around 52% for blast trauma injuries. The military are very interested in this. NASA are very interested in this as well. If a patient has isolated otolith damage, so there's no other damage to the vestibular system, they can have very non typical vestibular symptoms and can often be missed.

Maybe on a typical vestibular clinic because of that expectation of episodic or rotary. The histories are often very vague. They're just generally unwell, they're highly anxious. Spatial awareness is a bit off.

Symptoms are there. Very 3 PD actually. Very 3 PD. Here is the proposed criteria for isolated otolith dysfunction. So you can have a otolith dysfunction that is associated with the semicircular canal, but this is in terms of isolated otolith dysfunction.

And again, the Bahrani Society haven't finalised what the criteria is, but this is where we're at the moment. So you can see that there's laboratory findings that indicate an otolith dysfunction, but normal semicircular canal function. So it's usually proven with femps. Then the patient will have normal caloric and V HIIT tests. Yeah.

And symptoms are like non spinning, tilting, waxing, waning, swaying, bobbing, rocking. They'll feel well in the car, but when the car stops, they might feel like the car's still going.

Yeah. So just all those kind of more vague symptoms. And let's not forget there are non vestibular causes of dizziness. So if they have a continuing central pathology and if there is damage that brainstem or cerebellum, they are definitely going to have ongoing balance problems. Medications.

A lot of them are on medications and will have to be on them for the rest of their lives. But are those medications causing the problem? Often, if the damage is significant enough, they'll have vision changes. They might not be able to see as well or process their sight as well. Partial hypertension.

So again, the damage to the brain might mean that they cannot now monitor the. Or the homeostasis in the body is not as good as managing their brain blood pressure. Levels as they stand up. And so they're more likely to kind of get that when they stand up. Post traumatic headache.

And this also is like pain as well. So I've noticed the patients, if they've got a post traumatic headache, they tend to have like a headache that doesn't leave them and they're in constant pain. And I've noticed that if they're in constant pain, you can't rehab them. You need to try and manage that if possible. It's not always manageable, but if there are things that can be done for them to reduce any headaches or any pain, do it, because then their outcomes will be much, much better.

And then remembering if they have been in some sort of motor vehicle accident, they may have lost a leg or had fractures all up their legs. We've had people who've been in traction and so their sensory input through their vestibular spinal reflexes will be completely different. And so the brain's not only got to compensate for maybe a vestibular loss or cerebellum loss, but then also a issue with the proprioception as well. So moving on to the experience that I've had. So we've been working with these type of patients for maybe four years and I think we've got about 90 patients that we've seen roughly haven't done all the numbers, but I see them further down the line than maybe many of you will.

So usually about 18 months post injury. And what that means is probably the BPPVs have been picked up and it's more kind of those ones where it said at the beginning we said, you know, 30% of them will have continuing issues. They're probably the ones that land at my door. What we typically see, if there is no bpv, they always come in with vague non rotational dizziness. There's a post traumatic headache.

I would say in a high percentage of them there'll be anxiety, low mood, auditory issues, speech and noise issues, tinnitus, hyperacusis, over sensitivity to sound as well. A lot of them do describe poor spatial awareness and often they don't actually know what they're describing. But because we've heard it that many times, we can usually kind of now put it together that it is a poor spatial awareness, personality changes. So again, a lot of them will admit that they're quick to temper or they feel very emotional quickly. A lot of big guys that come in, they said, oh, I never used to cry and now I see a puppy on the TV and I cry.

Poor memory, planning and cognitive functions, which I think we've talked about. Agoraphobia, unfortunately, people don't want to go out. They used to be. A lot of them say to me, oh, I used to be so bubbly, I had so many friends. And now I just can't face going out and see them.

And I think it's because they get so overstimulated with vision and sound and their balance that it's easier for them to stay. In general, a lot of them have got maladaptive techniques, so they restrict their

movement. So it's that typical vestibular truncal movement. So instead of like looking to the left and right like this, they turn their whole body like this.

Postural instability, brain fogs are busy. Big biggie. And then visual vertigo is very, very common. I'm going to just do a few slides on visual vertigo before I go into kind of what typical test results that we get just because it is so common. Patients.

So visual vertigo is defined as the result of being overstimulated by a complex visual environment. And it's demonstrated here by my lovely friend Fluffy. So he sees the movement and he feels the movement. So usually if you have normally functioning ears and brain, your ears will be equal and or opposite. But as soon as, let's just make it easy, there's a unilateral vestibular weakness.

Your ears are no longer equal and or opposite. Your brain detects that. That's not quite right. And what it will do is it will now prioritize the information that's coming from your eyes, from your ears. So for somebody who has an abnormality, if they see movement, they will feel movement.

Me, who doesn't have an abnormality, if I see movement, I don't feel movement because I'm ear driven, not eye driven. And these patients will struggle in environments. You all know supermarket syndrome, where they often describe that they find that the supermarket so overstimulating, airports, the tube, train stations are all nightmare and also devices. So they tend to be a little bit better on, not big screen, but like a bigger screen, like a laptop without fast moving web pages. And we have the TikTok outcome measure in our clinic.

So how quickly can you go through your TikToks? Shows how dizzy you're feeling. But yeah, these patients will struggle even to go to the cinema. So, you know, if they go and see an action movie in the cinema, they will. As the cars and the superheroes are flying around and Groots doing this thing that will all stimulate them too much.

Their eyes will tell them that they're moving so they will feel constant movement throughout. And it's that vestibular visual mismatch that's the problem.

Okay, so back to the clinic. So our typical test battery is something like this. It's tends to be three to three and a half hours. Our history is quite long. It can be one to one and a half hours.

So, yeah, we do really, really take the time. And with these patients, you do have to give them breaks as well. So depending on what they say and their information that is given to you, you're going to always do pure TO audiometry and speech audiometry and then spit speech and noise tests if indicated. So if you think that there's some auditory processing problem, it's worth doing it. Tympanometry and reflexes.

Tympanometry is actually really important for your vemp test. So if you are going to do a vemp, and I've briefly mentioned this before, you want to make sure that sound can conduct through the middle ear. So it doesn't matter if there's a hearing loss, it's just, is there a conductive problem? Can sound get through to the cochlea? If it can get through to the cochlear, whether there's a hearing loss there is negligible.

I'm not testing hearing here. I'm using sound to test the vestibular system. And so I need to make sure that there's enough sound intensity for me to shake that vestibular organ. Because we're using a very loud sound, 85. Sorry, 95dB to get that vestibular response.

Then you're going to do your positioning manoeuvres because you want to. If there's a BPPV there, you want to address that and treat that straight away. So it hopefully doesn't have an impact on your other tests that you're going to do. VHITs, Chimp, VVR, VORs, they are all mostly peripheral tests. I do think there's a bit of overlap with both the shimp and the bvors into the central system, then CVEMPs

and O VEMPs.

So we tend to do a 95 comparison on both sides. Sorry, I'll send to myself. So 95 comparison on both sides. We're looking for any asymmetries. If both are symmetrical, then we'll see that it abolishes by 75 dB.

If it doesn't abolish, then we will suspect that third window. That's superior semicircular canal dehiscence problem, maybe fistula as well. We'll then do VNGs and our VNGs will look up both the central. So this is our neurovestibular testing battery. So smooth pursuit is a central test.

So it's saccade. SVV is an otolith test. Gaze central spontaneous vestibular head shake. Both head heave looks at the otoliths, ocular counter roll looks at otoliths and then the Hennenbert sign if indicated. If you think again that there's a fistula or a third window present.

Calorics are a funny one. Like, like it's really important to do calorics. Calorics are brilliant if you can get them but these patients have usually central problems and if you do a caloric on someone who has a central problem, you can often get a hyper responsive result which is not pleasant for them at all and they wouldn't be able to sit through for those irrigations anyway. Also if they are highly anxious and I've got the information that I need, I probably wouldn't put them through a caloric. If you're doing medical legal cases, the courts do love a caloric, but your patient comes first, right?

And their comfort. So you know, always have your discussion with the patient of, you know, the impact of calorics, how they they'll feel about them and often if you say, you know that it's important for your case for us to do it, they'll want to have a go at least. So just take your time. And I teach them the breathing. So in through your nose, out through your mouth throughout the test, try and keep the anxiety levels to the minimum.

We mess around with different questionnaires. I've used the VRBQs etc but DHI seems to be kind of my go to and then the visual vertigo scale as well. They're my two favorite questionnaires. And so what are our typical outcomes? So I am meaning to write up all of our patients but what I did for this presentation is I pulled up the latest 20 that we've done just so you can get a feel for kind of what we're seeing.

So for BPPV three for sure out of 20. So then positive V hits only in four which probably surprised some of you with positive VEMPs 16 and then positive vestibular VNG findings in 17 of those patients, positive central signs TBI. Right, 14. So kind of makes sense. Positive calorics is eight.

Six of those 20 patients that we didn't do, we said that they were contraindicated. So that leaves how many six normal and then visual vertigo. 19 of 20 of those patients have visual vertigo. It's.

There's definitely something with that visual vertigo. And TBI patients, what were their results? What did we categorize them as? So BPV3, third window was two of those patients. Only one of them came out to have only central findings in a TBI demographic.

So I just think that's really interesting. There's definitely something in this. Peripheral only was 3 and then central and peripheral vestibulopathy across 16 of them. Out of these 20 patients, most common abnormalities in this series were smooth pursuits, saccades, shimps and any measurements looking at utolith organs and actually those, I was comforted to see those results because that kind of fits in with what is being written in the literature. So there is a general agreement that saccade smooth suits and fixations are often impaired in TBI patients.

And abnormal vertical smooth pursuits is definitely a marker for those who've suffered a moderate severe tbi. Now, when you read this stuff, do remember that people are testing them in acute phase. So there's lots of studies that talk about what the results are with eye movements within the first seven

days. So there's talk about mild MTBI eye movements being affected, but those studies were done very quick after the event. Whereas when we've had mild TBI in their central eye signs are pretty much normal.

So I do think there's a time and importance there. There seems to be no clear diagnostic pattern. So I can't sit here and say to you, oh, all your patients vemps will be abnormal or all their smooth pursuits will be abnormal. And that's why I think it's so important that if you are seeing TBI patients, that you do have a wide range of artillery in your clinic so that you can do lots of the different tests and you can see. So a lot of clinics that are claiming to be vestibular clinics are doing VHIT only.

And you can see in those that cohort of patients only four that were positive. And I do know services that would then discharge that patient. So just be aware of that. With chronic tbi, most of the literature does rely on caloric and horizontal VHIT and abnormality rates on those tests are less than 25%. So I got four out of 20.

So that pretty much matches up right.

Otolith function has been mainly studied using CVEMPs and rates of saccular function range in between, depending on who you read. Otolith function has been mainly studied using CVEMPS with dysfunction range between 0 and 52%, that's a huge difference. But I definitely think that if you are doing TBI stuff, you do need to be looking at otolith function and especially with those patients, like if you're dealing with military patients or those that have blast injury. Use of OVMs to report utricular dysfunction has been rarely reported. We do ovums all the time and I do think they're a very important part of the vestibular test battery and I would urge you to do them a lot of services that do CVEEM will tell me that they don't do OVMs because they're a bit tricky to do.

They are tricky when you don't know how. But if you sat with somebody who does know how, they actually end up being easier than CVEMPs promise. So do your ovamps. So what sort of recommendations might we say for these patients? So hopefully if someone's been referred to you post brain injury, they will already be part of a multidisciplinary team and it's definitely a multidisciplinary team effort.

On the odd occasion, I have just picked up a few people who've just because they've thought that they were unbalanced and they didn't associate it with the tbi, they didn't have a team in place and they've been just struggling along. So that does happen to be aware that. But the majority, they do come to us. There's a case manager in place and there's already a team.

So what is the order of what we kind of would do? So if you see any bppv, treat that straight away, get that sorted if you can. And then look at that post traumatic headache. Can you get them out of pain? Sometimes?

I mean, medications with the overseeing doctor, cognitive support is really, really important and often I will refuse to start vestibular rehab with patients until there's cognitive support in place, especially if I know that they've got TBI because they are struggling cognitively and I know that happy brain is a healing brain. A stressed out, overwhelmed brain will not heal. You cannot teach vestibular compensation to a brain that can't gather itself. So it's really, really important. Often I've had patients who come to me and they've said, oh, I've done vestibular rehab, it doesn't work.

And then when I've explained to them the mechanisms of how it actually does work, they're like, oh, I didn't have those support systems in place. I wasn't ready for the vestibular rehab and then we've got them that support in place, they've had that treatment or management and then they've done the vestibular rehab and it's been a success.

If there's any auditory issues. So over sensitivities, tinnitus, hyperacusis, can you do anything to treat that alongside? Because anything that, like, supports the system as a whole is going to help. You may need to reach out to occupational health. Now, don't get me wrong, occupational health doesn't mean in the workplace.

Often many of our patients work, they're just too poorly to work. Occupational health still applies to the households as well. So, for example, they might need modified baths to get in the bath now because they'll fall if they climb over a bath to get in. And so getting occupational health in to make recommendations on the home and if they are working, the work environment is really, really good. So personal trainers are really useful resource to access if.

If the patient is not having regular physiotherapy, and that might be for multiple reasons. So they might be on a long waiting list and you want to get them some physical therapy asap, or they might have a physiotherapy plan in place. But a personal trainer is a handy tool to kind of keep things topped up. It's always important to kind of have communication open with the personal trainer just to make sure that they can support the patient safely. But we've had really, really great outcomes.

And actually, what a couple of the physiotherapists that I work with, they always have personal trainers now just to keep things ticking along. And we've had really great outcomes. So I can give you an example of one patient. He come in, he couldn't walk, he had a moderate traumatic brain injury and he loved bike riding and Love football. And 18 months ago, he couldn't even imagine doing either of those things.

And with the support of a personal trainer, he is now, obviously, they got him on a static bike and he's now going out with his personal trainer on proper bike rides as well as he's now kicking a ball and he can get it through the cone. So that's really good. So what we do is we line up the cones and get him guiding the ball around the cones and he's shown that he's got like that real good control of the ball,

whereas, you know, he couldn't stand 18 months before. So, you know, it's good, it's good, consistent work. Visual vertigo treatment is very important, but it's very difficult, but very can be very, very successful.

And so I would always say do Visual Vertigo last. You want all the foundations in place before you start attacking the visual vertigo. If you do it too soon, you're going to overwhelm the patient too much. So you've got to make sure they've got the coping structure, is the ground and they understand cognitively, they know that they need to push themselves, they're not maladaptive, etc. Etc.

Etc. So, yeah, visual Vertigo is the last kind of piece of pie that you're going to put in when you play Intriguing Pursuit with MTBI patients for sure. Okay, so I just wanted to talk about these three amazing women who've put together some really good resources for patients and a lot of them are free. So you've got Dr. Yona Arthur and she's put together a online CBT esque course for people to do. She's done loads of YouTube videos as well.

And then we've got Joey Remini. So she's written the Rocksteady book and again, she's got some stuff online that people can access. If you go to her website, you can see how easily it is to get her book. In your countries there'll be a form of a main brain injury group and I would really urge you to go and look at those resources and get yourselves involved in a multidisciplinary group because you'll have your skill sets and especially with these patients because they're so complex, it's good for you to bounce off professionals who have different skills to yourself. The Dizzy Cook so this is Alicia Wolf and she was diagnosed a few years ago with vestibular migraine.

Now, I'm not saying that all patients with tbi, we haven't really even talked about vestibular migraine, but I do think there is something in the stuff that she talks about. And she's written lots of recipes for people who are dizzy and she's got lots of good information on her website. So I would really urge you to look at that or recommend especially patients who have headache now. Thank you, ladies. If you

ever hear of this presentation and you happen to be watching it or any of their friends, just send them a big thank you because you really, really helped a lot of my patients.

Thank you very much. Do urge your patients to go back to their childhood a little bit. What do they love to do? Some music, art, craft. All those things are really good therapies for patients.

And do you remember I said before, a happy brain is a healing brain. And it sounds quite flippant and whimsical. Yes, they've had very serious injuries and I understand that. But if you can get their brain in a better overall mindset, they will start to heal. And so what can you do to do that?

So often it's. There is some research into kind of very fine motor stuff can. Can be quite mindful and bring you more centered. So like doing Lego, Plasticine, arts and crafts, anything like that. There's some work I think, is it Dr. Jill Davis talks about, you know, teaching patients who have more of a processing, auditory processing problem if they start learning piano.

Doesn't matter what age they are, they seem to improve. I've actually seen that anecdotally with my patients. So they might say to me, oh, I used to play the trumpet, but I've not played it since I was kid. Or piano, like, get it out, get one out and have a go.

So, yeah, I do think there's something in that, treating them as a whole so that then you can then actually then heal or manage the vestibular and the central symptoms. Case studies. Let's just whiz through some case studies. So what I've done is I'm going to present three case studies. One of them is acute and not mine.

And then two of them are more chronic and mine. So this one was published by Vivi Moochie. So this is a professional ice hockey player, 25 years old. He's had three concussions, 2015, 2017 and 2018. He had visually induced dizziness as vid, reduced gaze stabilization.

And he was treated with a. Excuse me, a combination of vestibular rehab and OPK trait retraining. Opk, if you're not familiar with that, it's where the black and white striped lines are shown and then they'll either move leftward or rightward, up or down as well. And so what you do is you train the patient to follow those lines. This is what he had.

So you can see he only had five days of therapy. There was a break in the middle after day two, but it's not that much therapy. And he had the combination. And there are the different exercises that he was given alongside some OPK training.

Now here are his outcome measures. So what's fascinating is if you look at where I've circled here, look at the prescores. This is the VVAs, the visual vertical analog scale. So you can see that the prescores are quite High. So 10 is bad and 0 was good.

So you can see pre, moderate to High numbers. And then look at day five, he's all like zero point something scores which is amazing, amazing improvement. So his visual vertigo completely dipped down and he got better, went back to Sports.

Patient number two is one of mine. This is Paul, he's 29 year old male, lovely person, loved fishing. That's why I put a picture of fishing in the background. I can't use real pictures because a lot of my patients are going through litigation. So I can't, I can't.

I'd love to get videos and pictures for you, but I can't do that. He was diagnosed with a right vestibulopathy post a car accident and he hadn't received any pre treatment or any vestibular investigations until he came to us. And that was, that was two years after, not prior. Well he hadn't received treatments two years prior to us finding us him. And then he has continuous dizziness and unsteadiness, generally feeling unwell, he had brain fog, highly anxious, many cognitive issues.

He felt like a 3 PD patient to me is the best way I could describe him. Look at his DHI scores. So again, if you're not familiar with the DHI, 100 is bad, 0 is good and he's way up at 72 which would be classified as severe. So here's Paul's vestibular lab results. So if we start by looking at the vhit, the VHIT results are at the top right hand corner.

Now if you're not familiar looking at the VHIT results, what you need to think about is if you look at that middle column and you see at the top it says left lateral. If you can see that they're normal. So you want those kind of peaks to follow each other and then the smooth ISH line after that peak. If you look at the final column and it says right lateral on the top, they're his right semicircular canals and you can see that there's quite a difference between the peaks and then there's these spikes. So they're catch up saccades.

So they're showing that the VOR has a different deficit. Underneath there you can see VEMP results. So we've got a normally function left vemp and then a completely flat line on the right. So the right vemp is absent. Right positive vhit, they match each other.

We also have numerous positive signs on the VNG which I've listed on the left hand side for you to have a look at. So there's lots of stuff going on there which would explain Paul, Paul's symptoms. So what did we do with him? So he read the Rocksteady book because there was a long wait for him to get to see anybody. He started CBT therapy and we kind of spoke to him about kind of because he was maladaptive, he wasn't moving.

So I did say to him, you know, you need to feed your body movement and that helps it heal. So he started going the gym quite a lot and that was before he got to his physiotherapist. And he went to the gym and did go with personal training advice and went very easy. Then we did some gay stabilization OPK work and then we also started on some virtual reality as he started to show improvement. So I'm

going to show you some objective results of pre and post.

And again, doesn't really matter if you're not used to looking at these type of tests. I want you just to focus really on that. If you're not used to these, look at the right laterals. So at the top that's your pre. And can you see it's kind of a bit all over the place.

There's a biggish gap between the green, the orange bad. And then if you look at the gain scores in the, in the kind of the tables on the left hand columns, look at where that red average X is. It's not fantastic. Now if you look at the, at the bottom one, look at the right lateral. Can you see those purple spikes are very much more time locked.

So they're on top of each other, which is good, means they become time locked. Then look at the crosses on the left in those tables. You can see that they've come up as compared to the pre. So that's all really, really positive findings. And objectively it's showing us that we're doing something to improve the vestibular ocular reflex.

And his dhis, sorry, his DHI scores have come down significantly as well. And I'm really sorry, I should have put them on this slide. I can't remember any exactly the number but they were significantly reduced. His shimp results. Now, shimp is still in early stages of kind of the research and literature, but the top shimp is pre rehab and the bottom shimp is post rehab.

And I think the important things to look at are those downwards purple spikes. So they're kind of the program saccades and how the brain kind of makes the assumption to move and catch a target. And you can see that in the post they're kind of really scattered and all over the place. And then in the after rehab, which is the one at the bottom, you can see that they become more gathered. And look at that asymmetry ratio.

So pre rehab it's way up at 40%. That's a very big difference between when you're moving the left to the right, left ear versus right. And then look at the asymmetry ratio. Post rehab, it's down to 23%. Okay, last case study.

I want to bring Emily in because Emily's nine and yes, paediatrics do suffer traumatic brain injury. So this isn't Emily. She had left vestibulopathy and vestibular migraine. She only came to us when she was nine year old, but she actually had the incident when she was an infant. And she's struggling with extreme fatigue, extreme dizziness and falls.

She'd always felt dizzy, like she felt that she never had escaped from it. She doesn't have any fluctuating oral symptoms. Neurology and imaging is clear. One to two headaches. Weak.

Bless her. She feels terribly anxious because of symptoms. Her VFT vestibular function test. Sorry, were normal. She did have an absent left CVEMP and reduced gain on her shimp, which is that last test that I've just showed you for Paul.

Her pediatric DHI was 36, so moderate. And her pediatric Visually Induced dizziness score was moderate. Visual vertigo and motion sensitivity. Otherwise she's fit and well. She is a little rock star and she really wants to be a doctor when she grows up.

She really takes school seriously, possibly a bit too seriously, but she's just finding lessons so exhausting. So just trying to make things a little bit easier for her. So what did we do? So she had some pharmacology for migraine, she had neuropsychology, eight interventions every two weeks. And then she had physiotherapy and she had that at school.

So she was quite stressed about missing school. So the physiotherapist agreed come into the school and she would have sessions there. After having the eight interventions with physio, we decided to give

her a we go of the virtual reality and see how she would cope with it. Now what you're doing with virtual reality is basically you're teaching the brain that mismatch signals are safe and okay, you're normalizing it and you're also desensitizing the eyes to any motion. So remember I said before, if they see motion, they feel motion.

So with Emily, we would start off very easy playing videos like here and so you can see there's not much background noise, in a sense. There's no. There's a bit of snow, but not a lot. Very plain colours, not fast movement.

And she would sit in a VR headset, fully immersed in this type of environment. And you can encourage them to look around as they're doing this. And this would be kind of like the starting level video, a bit like the gaze stabilization effort exercises. You start off easy and then you would advance it over time. You gradually increase it.

You would introduce more colors, more things going on. People walking through a. Maybe a busy market in Monaco, for example.

And then to make it even trickier, this would probably be more like the kind of the top level type of video. You can see there's an umbrella bouncing at the top, there's loads of snow, the head's moving left and right, there's lights, there's cars, there's lots of color. This is very visually stimulating. Some of you might even feel a bit stimulated watching this on the screen. So imagine it in a virtual reality environment.

It can be quite tough, especially with somebody with vestibular problem. You can do further things, especially with children, they like the game, so you can introduce certain games for them to do and that introduces secondary coordination tasks as well. So that's really good for them too. And it does mean that you can make it very specific. So Paul, who liked fishing, I said he could do a fishing game and he

did, and he used the system pretty much every day.

This is Emily's shimp results. Her V hits were normal, so there's nothing really to show pre and post, but her shimps weren't. So you can see the top shimp is pre treatment. So again, you look at those purple squiggles that are going down, they're all over the place. She got an asymmetry of 49% and after six months, you can see that they've gathered.

Her asymmetry ratio has come down 6%. Her DHI has come down from. From 36 to 20. She still said she had visual versico, but it was much milder and she still had motion sensitivity. But we can keep working on it and keep going.

So, yeah, there's three examples to hopefully show you that there is light at the end of the tunnel for these patients.

I just want to close with a video from Vida, which is about sports players or youth sports players and concussion. It's a really good video to close on. They did a great job of putting this together, but it does show the importance of vestibular rehab with these patients and I suppose to summarize, I think I would just like to encourage anybody who works with TBI or in vestibular that there is an overlap of these two, and we need to be working together to make sure that the patients are appropriately looked after.

I took a slap shot in the face, and it, you know, injured my right inner ear. The force went right out here, and I had rain, geez, for months. By the end of the season, I started noticing something was off, and I didn't know what it was. I just thought, hey, you know what? I'm just getting old.

We went to down a path that really delayed me finding out that I had a vestibular issue. And, you know, a couple months went by and just, you know, okay, Bryce, you know, you should be back. You should be fine. And, you know, you go through all the protocols and you're checking off all the boxes, but

you're saying, no, I'm not right. I know inherently that I'm not right.

It's almost a mixed blessing that concussion is so in the news these days right now. Concussion, on the plus side, it has brought it to light. You know, it used to be the dark subject that no one wanted to deal with.

So I had loss of balance and base. Barely any balance, which I've always had, really good balance because I danced, and I did play soccer and cheerleading and all this stuff. I couldn't remember anything. I could barely concentrate when someone would talk to me. I couldn't even, like, understand what they were saying.

Well, it's been very difficult because she, as a high achiever, decided that she was going to get all the symptoms that a concussion would give. And she had very severe headaches, which, of course, affects her mood as well. She's miserable. Memory, cognitive ability can really be affected, and when that's affected and children can't read or they can't go to school, they can't go to class, they can't, you know, interact with their friends. That affects their mood.

She had lack of concentration, lack of sleep, and the balance issue as well. So it was very difficult for her, which, of course, makes it difficult for me to see her that way. One of the most important things is that if a child is feeling the effects of a concussion, certainly for several they're after, we really want them to see a healthcare provider.

Turn your head to face the same one that you're looking at. So vestibular rehabilitation has really come into its own over the past several years. We have a much better understanding of how to measure a vestibular problem. We can measure what part of the system is actually not functioning correctly and then we put you through a course of rehabilitation, depending on what part of the system is not functioning properly. So much of my senior year cheer, at least, was taken away from me.

I couldn't even cheer at my senior game. I came afterwards for the ceremony, but I couldn't cheer at it, which I hated. Team. Team sports in particular. The key is keeping them involved with the team.

Coaching and teaching techniques have become really important. Teaching kids as they're progressing through the stages of development of how to properly take contact or give contact. There's a push towards equipment. There's a lot of analyses being done on helmets and different styles and types of helmets, better neck control for prevention of neck injuries.

I think that sports are vital in a child's development. I see that there's progress being made. We want all kids to play the sport that they want to play.

So I think, to summarise, I just would like to emphasise the importance of us communicating. If you're working in TBI or if you're working in vestibular or Both these patients do need, I think, both sets of care and I really would love for my message to be that vestibular needs to be taken seriously in the realm of brain injury. I would love to thank you so much for your attention and your time. I know that somewhere around the BPV section my head was drifting off camera, so do apologize for that. You are very welcome to email me any questions that you have.

If you can't think of any now, I am going to pass back to Chris. Thank you for having me. And here are my references if anyone needs to see them. There you go. Thanks, Chris.

Well, I don't suppose that the thanks is mine, really. I mean, that presentation was astonishing. What an insight into a category of patient that is so unfortunate. And yet, hopefully, if they see you in your clinic, very fortunate. We've had a wonderful insight into the advances in virtual reality and so many other things.

So I just can't thank you enough, Amy, very, very much. But I must say we have had some questions through from our online colleagues that will go through, if I may. So I'm going to crack on very quickly. I'm just conscious of time. So the first question that we've got here is, is there any guidance on how you categorize mild to penetrating tbi?

I know you mentioned the Glasgow Coma Scale, but is that the same thing as actually how you categorize? What is the difference between a mild Moderate severe traumatic brain injury. Yep, good question. So the Glasgow Coma Scale is kind of gold standard. So that's what you will see mostly in literature or if you get referral to somebody who has a known tbi, you might get given kind of what the Glasgow Coma Scale is or was at the time of the injury.

But the loss of consciousness so the time that they were unconscious or the length of time at the post traumatic amnesia has been linked to, to the outcome. The prognosis basically of those as well, so dependent, I mean mostly you'll see it Glasgow Coma Scale. But having the information about loss consciousness and post traumatic amnesia is also valuable and valid. Perfect, thank you very much for that. The next question is, is there anywhere central that colleagues can start sending post traumatic BPPV data to improve data pools and white paper content?

You can send it to me and we can put it together if you want. For sure. I mean, I'm all about multidisciplinary work and for sure, I don't know anybody who is specifically focusing on that at the moment. But I mean, we all need to be talking to each other and there's a few reasons for that. It's for discovering the new stuff, but also not reworking the wheel because I bet you there's some stuff that I figured out here that was also figured out years ago in another country, you know, so just an open forum where people can chat and you know, I'm always open for that.

So. Yeah. Thanks, Amy. The next question I think comes from an example on a slide you had about endolymphatic hydrops. Can you explain why the hearing loss seems to be conductive?

I think it's referencing why the hearing loss seems to be in the lower frequencies. So the audiogram that I've put on that slide is actually from that study that is quoted the. They have Mark 250 and it has a slight in the UK light conductor, but 512 is not conductive. I wouldn't worry too much about that conductive element on the 250 for sure. Brilliant.

Thank you. So would your recommendation be that it's often a sensorineural hearing loss? Yeah. So within endolymphatic hydrops the expectation is a sensory to or loss. For sure.

Brilliant, thank you very, very much. Next question. Are there guidelines for defining a headache? There is, and I don't know them off the top of my head. You can go to the international classification for headaches and you just need to make sure your patient fulfils that criteria.

There's Also, of course, there's the Bahrani Society's criteria for vestibular migraine. Now, what I do want to point out, and I'm kind of hijacking your question because this isn't what you asked, but I do want to point out that vestibular migraine may or may not be associated with a headache. Doesn't always mean that there's a physical headache. And I think that can be quite confusing for clinicians and patients. That's fair enough.

Thank you very much. Next question. Is there a questionnaire you'd recommend to use for patients referred with known TBI both pre clinic and in clinic appointments? So, yes, very good. DHI is my kind of go to.

We've tried a few of the other questionnaires and we are not also diagnosing the TBI at this point. What we are doing is we're seeing if there's an association with the disease, dizziness and balance problems. So our patients are already diagnosed with tbi. However, what I would say is I am not aware of the TBI questionnaires and how you measure those outcomes. TBI only.

So if you are somebody who is looking into being involved in these patients at an earlier stage, it's sensible to suggest that probably ask TBI specific questionnaire for those patients. Brilliant. Thank you very much. And the last question we'll take for today is actually more of a comment. So they've said we don't have access to vemp testing.

Would you say it was a good argument that TBI is a good reason to invest? I feel like I'd have my right arm chopped off if someone took my vemp away from me. And actually my vemp needs pull fixing. Hint, hint. If you could get that organized.

So, no, I'm joking.

You cannot see what the otolith organs are doing really without a VEM test. There are some tests that kind of help glean information from the otolith. So for example, ocular counter roll head heaven skew sometimes as well. But none of them are as sensitive and specific as the bemp. But also with tbi, I think the sensitivity and specificity of all tests is all over the place.

So I think to be a responsible clinician, you need to have access to as much as as is possibly available to you. Yep. I think that's a very diplomatic answer. And I think if anything we've learned from your teaching today is that you must, must, must make sure you have a complete plethora of diagnostic testing available to you, especially for patients who've been struggling a long time. So, again, thank you so much for your time and your teaching today, Amy.

We've really appreciated it. For everyone, the recording of today's seminar will be available on the Hearing and Balance Academy within the next few weeks. Just before we let you all go, please, please, could you take two minutes to complete the survey that automatically comes up at the end of the seminar so that we can continue to provide further education to all of our amazing colleagues around

the world? But that does conclude our E seminar for today, so it's a massive thank you from me, Chris, and a great thank you from Amy, I'm sure. Thanks so much, guys.

Bye. Take care, everyone. Bye for now.