Assessing Vestibular Dysfunction After Concussion

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- Our title for our course today is, "Assessing Vestibular Dysfunction After Concussion". And it is my pleasure to introduce Tonya Fuller to AudiologyOnline. Tonya is the founder of Dynamic Mobility and Balance Center in Inglewood, Colorado, which is a private practice that specializes in treatment of neurological injuries, brain injury, and vestibular disorders. She received her master of science in PT in 2001 from Texas Women's University in Dallas, and she began her career at Baylor Institute for Rehab in Dallas, where she also started treating vestibular disorders in 2006. Throughout her career, she has participated and assisted on various research projects and has developed and presented a multitude of lectures to physicians and peers. So at this time, I'm gonna go ahead and turn the microphone and the classroom over to you, Tonya, and we'll get started, here.

- [Tonya] Thank you, Calista. And thank you, everyone, for joining me this afternoon, on this wonderful Friday afternoon, to discuss vestibular dysfunction after concussion. Here are my disclosures for today's presentation. And as Calista had mentioned, I've been treating brain injury for quite some time, as well as the vestibular issues. I'm not an audiologist, so hopefully the information that's presented today will be beneficial to you in your practice. The learning outcomes for today, after this course, you should be able to identify three vestibular disorders that're common after concussion. We will not be discussing everything that is possible after concussion, but we will talk about the more common disorders. Be able to explain how to perform testing for vestibular dysfunction after concussion using appropriate tools, we'll talk about computerized objective tools, we'll also talk about just clinical tools that you can do in the practice, and describe how to determine a central versus peripheral pathology after testing.

So with that, we will get started. Dizziness and imbalance accounts for 40 to 60% of symptoms after head injury, and the prevalence is 23 to 81% in the days after injury. Many times, people will make a full recovery in the first few days after an injury. But for those that don't, these're the more persistent symptoms. Dizziness is identified as one

of the most common symptoms in high school and collegiate athletes, and it's second only to headache in symptom frequency. It is also a predictor of prolonged recovery. It has a sixfold increased risk for developing post-concussion syndrome compared to those without dizziness. It's an independent predictor of failure to return to work. Persistent dizziness and imbalance were the predominant symptoms after three months in one study done in 2015.

And as I mentioned, many people will make a full recovery within the first week and have no lingering symptoms. But for those that do, headache and dizziness tend to be the most common symptoms. Statistic-wise, 50% of concussed athletes report dizziness, and 40% report a balance disturbance. Positional vertigo, things like benign paroxysmal positional vertigo, which we talk about quite a bit in here, occurred about 28% of the time. Vestibular migraines about 41% of the time. Interestingly, vestibular migraines do not always have pain associated with them, as we think of migraines. Most of the time, vestibular migraines present with dizziness, and spatial disorientation occurring about 19% of the time are commonly reported. BPPV, or benign paroxysmal positional vertigo, is the most common cause of dizziness after concussion.

And its prevalence is anywhere from 10 to 57%, and then 30% of patients will have a combined peripheral and central vestibular dysfunction. So the vestibular system begins where cranial nerve number eight exits the brain stem, and goes all the way to the ear. That is considered the peripheral vestibular system. Everything from the entrance of the nerve into the brain and the connections there are more of the central, the central vestibular system. So what causes dizziness after concussion? Well, a direct injury to the vestibular system would be a primary cause. So a direct blow to the head that causes a fracture of the bony casing of the vestibular system. Anything that might, when we talk about BPPV, which we do quite a bit in this course today, the calcium carbonate crystals within the system are knocked loose because of that direct

blow, or even an indirect blow where there's enough force to knock them loose, that can cause the crystals to come loose.

And then, of course that's a, would be a direct injury to the vestibular system. And it could be just damage to the structure itself. There're also micro-structural injury to the central nervous system. So oftentimes, peripheral and central injuries will occur together. So you can have processing problems within the brain because of injury to the central nervous system, but also have a peripheral problem such as a loss of function on one side, or even BPPV. There're also injuries to the visual system and the cervical spine, which can affect the vestibular output. So our visual system, our cervical spine, and our vestibular system all work together to tell us where our head is in space, and tell us what's upright.

And so, injuries to any of those systems can affect the connections that it has to the vestibular system and interfere with the outputs from it. And then, of course, psychological factors can increase dizziness, such as anxiety or depression. The anxiety and stress pathways actually overlap with the vestibular pathways, so you can get interference from anxiety and stress that can create vestibular problems. When we talk specifically about vestibular injury, injury to the peripheral vestibular sensory organs or the central vestibular pathways can produce things like dizziness and imbalance, which is what we've been talking about. But also postural instability, as our vestibular system is one of our primary balance systems that tells us that we're upright. And also visual blurring because of the direct connection to the visual system from the vestibular system through a reflex called the vestibulo-ocular reflex, which we will talk about.

Other mechanisms that can occur, there can be a shearing effect on the root of cranial nerve number eight, which is our vestibulocochlear nerve. There can also be direct injury to the otolith organs, the semicircular canals, and the entire vestibule. You can

have a disruption of the endolymphatic duct, which changes the pressure within that system, and it creates a vestibular problem called Meniere's disease. And also, there could be a fistula that's causing perilymph fluid to leak within the system, causing dizziness as well. Those mechanisms, we will not be talking about as much. We are gonna look at injuries to the otolith organs, the semicircular canals in the vestibule, but things like Meniere's disease, perilymph fistula, these're more rare vestibular events, so we won't be discussing those today.

The more common vestibular issues are going to be, as I mentioned earlier, benign paroxysmal positional vertigo; things like a labyrinthine concussion, so a concussion of the entire vestibular system, which generally leads to loss of function either on one side or both sides depending on the injury. You can also have something like visual motion sensitivity due to decreased functional ability of the vestibular system, which causes the visual system to overwork. And then, also vestibular migraine. All right, so BPPV is the most common vestibular, the most common peripheral vestibular disorder. And this is in all cases of dizziness. It actually accounts for 50% of all dizziness over the age of 65. But in concussion as well, it is the most common peripheral vestibular disorder.

It accounts for up to 57% of dizziness in concussion. With concussion, you do have a higher incidence of atypical canal, which is your anterior canal. And again, we talk about this when we look at the anatomy. A higher incidence of having multiple canals involved, having it occur bilaterally and be recurrent, meaning it goes away when it's treated, but it also comes back with recurrence. One study of military patients found that with concussion, 1/3 had dizziness... 1/3 of the people with dizziness after concussion had evidence of BPPV, and they found that prompt treatment resulted in more rapid return to duties. So it will be very important to get them to a specialist who treats vestibular disorders. The labyrinthine concussion, or a hypofunction.

You can have auditory and/or vestibular dysfunction of the inner ear without a bone fracture. So this can be damage that's directly impacting the hearing organ and the vestibular apparatus. Compression and vibration forces are transmitted through the skull with a hit to the head or with enough force with whiplash to the inner ear. And they believe the pulsation of the inner-ear fluids may damage the inner-ear hair cells. And that's what causes laceration or hemorrhage of the membranous labyrinth, which is the soft structure of the vestibular system. Also, the macula of the otolith organs may be more vulnerable to pressure waves. And then, both otolithic organs are susceptible to injury with a hard enough force.

This is a peripheral injury. And again, you can have damage on one side, which would present as a unilateral hypofunction; or damage on both sides, which would be a bilateral hypofunction. And they are treated differently in the clinic, so it's important to know if one side or both sides is affected. Visual-motion sensitivity and spatial disorientation is the ability to centrally integrate the visual and vestibular information. So our vestibular system provides us with information about our head position in relation to gravity. And so, it is the primary balance system that keeps us upright, especially when we can't see. So things like walking in the dark, closing your eyes in the shower, washing your face, what keeps you from falling over is your head-position information from that vestibular system.

Our visual system also has the ability to tell us that we're upright when we can see, because we can compare ourselves to our environments and see that we're vertical, compared to doors, and poles, and things like that. So what happens when you're not centrally integrating the visual and the vestibular information together because of an impairment of the vestibular system, then what you get is an increased awareness of visual stimuli. So things that our brain would typically ignore, such as the leaves moving on the trees, somebody walking down the sidewalk with their dog, our eyes

become more aware of that stimuli. And so, symptoms will increase with that visual motion, and they become specifically worse in busy environments.

So airports, restaurants, grocery stores, things like that, tend to make people feel much more off-balance and disoriented. Visual vestibular deficits are often persistent symptoms, so they're very hard to break. And they tend to be symptoms that linger for an extended period of time. And these are predictors of prolonged recovery. And then lastly, vestibular migraine. This's a central injury. So as I mentioned, headache is the most common reported symptom after concussion, and vestibular migraine is the second most common cause of vertigo after BPPV. And again, this is kind of in general. However, it is very common to have vestibular migraine with vestibular symptoms after head injury. It is the most common cause of spontaneous vertigo. So people might just be sitting still, and suddenly they have this episode of vertigo that may last only a few seconds.

This would be, likely, a vestibular-migraine issue. However, vestibular migraine must have dizziness associated with 50% of headaches. So if there is pain, 50% of those headaches do have to have dizziness associated with them. However, vestibular migraines do not always have a headache component. Sometimes it's just the dizziness that occurs. Concussed athletes with a family history of migraine were at an increased risk for post-traumatic migraine following concussion compared to athletes with no history of migraine. So obviously, they're more sensitive to migraine if they have a history of migraine, or even a family history of migraine. And athletes with post-traumatic migraine experience worse neurocognitive and vestibulo-ocular impairment following concussion. So our vestibulo-ocular reflex, we'll talk about this, here, shortly, holds the images still on the retina while the head is moving.

And so, people with post-traumatic migraine tend to have more difficulty with stabilizing their eyes while the head is moving. And then central injuries, you can have a

direct injury to the brain stem or the cerebellum. And this's where the vestibular nuclei are located within the brain stem, and where the central pathways are. So any direct injury to those areas can also create vestibular dysfunction and dizziness. Abnormalities and oculomotor function can indicate damage to central nervous system pathways. So anywhere in the cerebral hemisphere's the cerebellum, and the brainstem. These central pathways will, from the vestibular system, project up into the cerebellum to the eyes and to the spine. And so, many times, if there's injury there, we get loss of balance.

We get difficulty with stabilizing the eyes, we get coordination problems, which all occur in those areas. And because of the connection of the vestibular system to the eyes, I think the important thing to think about when you're looking at some of the testing is a lot of times it can look like there's an issue with saccades, when the real problem may be a stabilization issue. So really trying to determine, is this a central-pathway issue that's causing dizziness? Is it a vestibular problem such as gaze stabilization? Many times with gaze-stabilization issues, people will overshoot with saccades, because they can't stop their eyes and fixate with their head. Now, concussion and auditory symptoms, 50 to 92% of concussion patients, including those that have non-blast injuries, report auditory issues.

And many times this's things like tinnitus, noise sensitivity, and auditory-processing difficulty. Hearing loss can occur. But a lot of times, these symptoms occur in the absence of any hearing loss. So we know the cochlea and the vestibular apparatus share the same cranial nerve, number eight. So those symptoms can overlap. Many times, abnormal signals from the vestibular system cause an overlap into the cochlear portion of the nerve, and it creates tinnitus. We see many times that people without concussion, just vestibular problems including BPPV, will also have tinnitus and noise sensitivity, simply because of the abnormal signals that're crossing over into the

cochlear portion of the nerve. Okay, so we're gonna do a quick cover of the anatomy and physiology of the vestibular system.

And a quick question, how do anxiety/depression pathways overlap with the vestibular pathways? Without going into the anatomy there, I'd have to go back, actually, and tell you, I couldn't tell you off the top of my head. But these pathways, as they are moving through the brain, a lot of times those pathways of the vestibular system are very close. And so, you can, as I mentioned, vestibular abnormalities can overlap into the cochlear portion of the nerve. Many times, you get some interference that occurs from there. So a lot of times, when we look at pure vestibular problems, there's something called persistent perceptual postural dizziness or 3PD. This is an anxiety-based dizziness. And so, it's treated with anti-anxiety medication, so...

Okay, well, we will move on to anatomy and physiology of the vestibular system. So the vestibular anatomy, the vestibular apparatus is a membranous labyrinth, and it is housed within a bony casing. And that all contains the auditory and vestibular organs. So this's your cochlea, and this's your vestibular apparatus that're all contained in that bony labyrinth. This's all supplied by cranial nerve number eight. So when we look at a picture, here, you can see the bony casing, here, that shows the three semicircular canals in the vestibule, there, in the cochlea. The membranous labyrinth contains three semicircular canals. You have an anterior canal, a posterior canal, and a horizontal canal. And your semicircular canals're going to sense angular acceleration.

So if I turn my head to the right, that is the semicircular canal that's giving information to my brain about head movement to the right, that angular movement. These canals're aligned at right angles to one another, with the horizontal canal sloping down from anterior to posterior by 30 degrees. So from your face to the back of your head, the horizontal canal slopes down at 30 degrees. It also contains two otolithic organs. There's the saccule, that detects motion in the vertical plane; the utricle, that detects

motion in the horizontal plane. And these sense linear acceleration. So if I'm walking forward, backward, going up and down in an elevator, it's my otolithic organs that're giving me information about my position.

And they also sense head tilt. And when we talk about head tilt, it's a small tilt to one side or the other. It's not a full movement of the head all the way down to the shoulder. With that head tilt, what it triggers is an ocular tilt in the opposite direction. So it will right my eyes to a certain degree, so the world still appears vertical to me, or I still feel upright in my surroundings. The semi-circular canals provide sensory input about head velocity, so how fast my head is moving. This enables that vestibulo-ocular reflex to generate an eye movement equal in velocity to the head in the opposite direction. So this stabilizes the eyes for clear vision.

So an example of that is if I turn my head to the right, but I keep my eyes forward, my semicircular canals are sensing that my head is moving to the right, it's sending a signal to my ocular motor system to turn my eyes to the left so they can stay fixated. And the VOR is really important with walking, running, riding in a bumpy car, so that our vision is able to stay clear. The semi-circular canals contain a fluid called endolymph, and this fluid has a certain density, which allows it to provide that information. We talk, in a few minutes, about the cupula. There's also coplanar pairing and sensory redundancy. So we have a vestibular system on each side of our head, and they're mirror images of each other.

And because of the way that the canals are lined up from right to left, what we get is this coplanar pairing and sensory redundancy in the sense that whatever my right side is telling my brain, my left side is giving the same information. This becomes important if we do have a hypofunction, that we lose a portion of that vestibular system, the other side can still be giving accurate information as to where our head is in space. This's provided through a push-pull relationship, meaning that a push on one side of one

vestibular system in the semicircular canals will create a pull on the opposite side of the vestibular system semicircular canals. And that's what provides some of that sensory redundancy.

As I mentioned, they have perpendicular relationships to each other, so the right anterior canal is in the same plane as the left posterior canal, and that goes for the left anterior and right posterior being in the same plane, and the right horizontal and the left horizontal are in the same plane. At the end of each canal is something called the crista, and the crista contains the cupula and the ampullary crest. And a push or a pull on the cupula is what provides us with that information about head position. The otolith organs, they're sheer forces from the otoconia that provide us information. The otoconia are those calcium carbonate crystals. They are very dense, and very heavy, and they sit on top of this otolithic membrane, which is like a jelly-like material.

They're somewhat embedded into that jelly-like material. And so, what they do is they have... There're sheer forces from the otoconia when there's a pull of gravity that gives information about movement. Forward movement, backward movement, up, down, et cetera. Again, this is linear motion. So acceleration, deceleration information comes from the otolith organs. Utricle detecting information in the horizontal plane, saccule detecting information in the vertical plane. And this contains the macula. And the macula is all of that information together. So here we see a picture of the semi-circular canals. The ampulla contains the cupula. The endolymph will either push or pull on the cupula to give us information about movement. Over here on the right, we have the macula, which contains the otolithic membrane, the otoconia.

And that's gonna give us information based on the pull of gravity on our movement in a linear direction. And then, real quick on the physiology, how does all this work? Well, we get information from the three primary balance systems, which include our visual system, our vestibular system, and our somatosensory system, which is sensory and

proprioception. These're processed in the brain stem in the cerebellum, and this provides motor and perceptual outputs, and those motor output're mediated by the VOR and the VSR, which we'll get to in just a second. Primary vestibular afferents originate at the hair cells in the vestibular apparatus. They come down the nerve and they enter the brain stem between the pons and the medulla.

From there they proceed to the cerebellum, which's where some of the balance and coordination information comes from; or the vestibular nuclei, which're four areas within the brain stem that send signals all other places. There're four nuclei. Each semicircular canal and each otolithic organ has its own neural network to provide this information. So the four vestibular nuclei, you have a superior vestibular nuclei that's gonna send information to the eye muscles for that gaze stabilization through the vestibulo-ocular reflex. The lateral vestibular nuclei sends information to the spinal cord via the lateral vestibulospinal tract. And that's going to give information to the vestibulospinal reflex, which's what keeps us upright. That's our postural reflex. The medial vestibular nuclei goes both to the spinal cord and the eyes via the medial vestibulospinal tract.

And the inferior vestibular nuclei goes to the reticular formation. And this area's a little less understood in terms of what it does, but we do know that it's where a lot of the abnormal signals may go that create the dizziness, the nausea, things like that, with vestibular problems. We talked briefly about this. Vestibular inputs are used to hold images stable on the retina during head rotation. So what happens with the vestibulo-ocular reflex is the semi-circular canals will signal how fast the head is rotating. The oculomotor system responds by rotating the eyes at an equal velocity in the opposite direction. So this's important with, as I mentioned before, walking, running. If we're running and our head is moving up and down, what we don't want is our eyes doing the same thing that our head is doing, we need to be able to see.

That's where the vestibulo-ocular reflex comes in. So you can determine if there're problems with the vestibular system just by a very simple test of the VOR. The vestibulo-spinal reflex, the system provides the central nervous system with information about movement and position of the head with respect to gravity and those inertial forces. So the vestibular system does require gravity to function. This causes excitation of the extensor musculature of the spine. And this's what helps to stabilize our body. So this reflex is... The vestibular system as a whole, through this reflex, is responsible for keeping you upright when you can't see. So what happens with the vestibulo-spinal reflex is you get rotation of the head, which creates a weight shift in the body.

And the vestibular system responds by making sure you don't fall over. So you can see this with things... One of the things I kind of noticed the most is, like, skiing. So if you're coming down a hill and you're moving from side to side, you'll notice that people always have their head upright. And keeping that head upright helps the vestibular system respond by keeping the body balanced. As I mentioned, the reticular formations's not very anatomically well-designed, because it includes neurons that're located in a lot of different parts of the brain. But these neurons are excited by a variety of sensory stimuli that come in from the somatosensory, auditory, visual, and the visceral sensory systems. The visceral sensory systems are where we're getting a lot of the symptoms from vestibular dysfunction.

This reticular formation is responsible for general arousal or alertness. So when you get up in the morning and sit up, your system kind of... Your whole brain sort of wakes up. That's what the reticular formation is primarily used for. It's also responsible for vital functions, attention, it can adjust transmission of pain information, and it helps with consciousness levels. So I have normal signals from the vestibular system into that vestibular nuclei and out to the reticular formation are what produce dizziness; nausea; sweaty, clammy feelings that could come on. All of that is coming here from those

visceral symptoms. So in summary, the vestibular system, the function of the system is posture and balance control through the vestibulo-spinal reflex; knowledge of self-movement through the saccule, the utricle for linear movements; semicircular canals for angular movements; and visual fixation during head movement via the vestibulo-ocular reflex.

So the system is... I think the thing to keep in mind, this system is responsible for self motion. It's really the only system in our body that can tell us if we're moving through space or not, where our visual system can provide us with information that something is moving. It can't tell us where motion comes from. So people that rely heavily on their visual system because of vestibular dysfunction end up with that visual-motion sensitivity. Okay, so we're gonna talk a little bit about medical testing, and this's where audiologists tend to do a lot of the work. So some of this may be complete review for many of you, but where we get our information helps vestibular therapists a lot when they're seeing an audiologist having testing done ahead of time to give us a better idea of what's happening.

So we're gonna talk about the video nystagmography, this one's typically used more now. I'm not even sure they use electro nystagmography anymore, because of the fact that the visual system has the ability to override the vestibular system. If the eyes are open and in room light, they can sometimes stop the nystagmus from occurring. So they use, now, the video nystagmography. The cervical vestibular evoked myogenic potential, or the cVEMP. There's also the ocular VEMP and then a CT scan. So these're things that would typically give us information after concussion, in terms of some of the things that might be wrong within the vestibular system. So the VNG is a battery of tests to identify causes of dizziness.

There's ocular motor function, so smooth pursuit is tested, saccades is tested, optokinetic nystagmus is tested. This's all looking at more central areas of the brain to

determine is there a problem with one of these. And remember, saccades very possibly can show up with abnormalities when there's a gaze-stabilization problem. So it's important to really kind of look and see how severe it is, to some degree, and are there other central pathologies, or, central abnormalities that also're showing up. It also looks at positional testing through the Dix-Hallpike. There are also head-position tests that occur with this, and then calorics. And I think, also, to keep in mind with people when they age, smooth pursuit and saccades naturally declines and slows down.

So if we're looking at someone who's fallen, has a head injury, has dizziness from that regard, who might be 70 years old, you might see abnormalities in that strictly from aging, and not necessarily from a true dysfunction. The calorics of the VNG is the gold standard for identifying peripheral unilateral vestibular hypofunction, although it can detect bilateral hypofunction as well. This uses a temperature gradient to deflect the cupula of the horizontal canal, and it generates nystagmus by blowing cold, or more likely, ice air, and warm air into the ear. So if the system is working normally, then there should be nystagmus that occurs during the test. If there is an abnormal test, then what is seen is a weakness on one side or both of less than or equal to 25%.

Now, I will say, many of the EMTs I work with, when they do these tests, if they see a weakness of 20% and have no other way of, or no other explanation for the dizziness, many times they do believe that that could be causing the dizziness. So it's only clinically significant, though, if there's a weakness of greater than or equal to 25%. What's important here is this only examines the horizontal canal function. That's supplied by the superior vestibular nerve. So if the inferior vestibular nerve is involved, it will not show that, and the test will come out normal. So one thing I didn't mention is as cranial nerve eight exits the brain stem, when it gets to the ear, it splits.

You have the lower portion, which supplies the cochlea; the upper portion, which supplies the vestibular system. That upper portion splits again when it gets to the

apparatus to the superior vestibular nerve and the inferior vestibular nerve. And so, a VNG only tests the superior vestibular nerve. So again, if there's inferior nerve involvement, it's going to show up normal, and that's where the VEMP comes in to play. So the VEMP is performed to determine pathology in the otolith organs, but it also looks at inferior nerve involvement. So the cervical VEMP is performed by placing electrodes over the sternocleidomastoid muscle. Your vestibular system sits right behind the mastoid bone behind your ear lobe. So this muscle must be contracted during the test.

An abnormal result would be absence of muscle contraction, which would also indicate the side of the lesion. So what happens, these electrodes're placed on the sternocleidomastoid muscle, which attaches at the clavicle, the sternum, and the mastoid. There's something put into the ear to create a click. That click triggers the nerve, causes a contraction of the muscle, and if there's no muscle contraction, then an abnormal result would say that there's inferior vestibular-nerve involvement. This has fair-to-good test-retest reliability, so not the best test, but not bad. The ocular VEMP is another way to test the superior vestibular nerve involvement. So this's performed by placing electrodes over the inferior oblique eye muscle. Patients must be looking up so that it brings the muscle closer to the electrodes.

There's also a click that's done, and an abnormal result would be absence of muscle contraction on that same side. So this actually has excellent test-retest reliability. So this's a very good test to do as well, without having to do a full-on VNG. Couple of things interesting about the VEMP is that it's a useful tool for diagnosing, also, two other vestibular problems, like a superior canal dehiscence and Meniere's disease. So a decreased threshold and an increased amplitude of response could possibly detect a superior canal dehiscence, which basically is just a thinning of the bone on the upper portion of the vestibular system; and also Meniere's disease, because of the pressure within the system. VEMPs are often absent in healthy elderly, and it's believed that

they're weaker muscles, so there're less forceful contractions that occur with these tests.

So many times, in people who are very healthy but are elderly, you may not see a positive VEMP, you may not see the contraction in there. So I thought that was interesting when you're considering the age of the person that you might be testing. And then a CT scan is an x-ray technique to determine fractures or thinning of the bones. So it can be used to detect temporal bone fractures, the vestibular system's located behind the temporal bone. So if there's a fracture, there could be disruption to the vestibular system. As I mentioned, it's also used to detect thinning of the bony labyrinth of the superior canal, which's also your anterior canal. And that would be a sign of superior canal dehiscence.

Okay, so now we'll talk about some clinical testing that can be done without having to do the full-on testing. And this is, a lot of times, what we as therapists will do in the clinic. But many times, if patients're unable to tolerate some of these tests. Which I'm sure, if you're doing VNGs in your office, you know there're many times people can't lay flat, they can't do many of the things because of visual issues or other things. And so, sometimes these clinical tests're much easier to perform. They take less time to perform, and there's less anxiety, I think, around them. So one subjective test is a Tinnitus Handicap Inventory. This's the most widespread and validated questionnaire for measuring severity of tinnitus.

This is a subjective measure, however. So scores range from zero to 100. A higher score would result in more perceived handicap. But interestingly, a minimal clinically-significant change is only seven points. And then, if there's a highly relevant improvement, that would be 17 points. And this's very easy to perform. You just hand them the test, they fill it out, you get a lot of information in that. We also use in the clinic something called the Dizziness Handicap Inventory, which is very much the

same. It just gives different possibilities, functional, physical, emotional components, that may be creating dizziness. So you can break down where some of the problems may be coming from in that particular test. It says things like, "Does rolling over in bed increase your dizziness?"

So if it does, then we may think that there's a positional issue that could be BPPV or something like that. The other thing I wanna say here is that many times the dizziness is addressed in therapy, but the tinnitus is minimally improved because they're still getting some kind of interference with the cochlear portion. And so, a lot of times, or there's damage that's occurred, that creates the tinnitus as well. So this's also another way that if the dizziness is now better but the tinnitus is not, that's something that needs to be addressed beyond the dizziness piece. The head impulse test, this used to be called the head thrust test. This's an unpredictable, small-amplitude, rapid head thrust.

Patients with vestibular deficits have to make a corrective saccade when they do this test to refix on the target. So the way the test is performed, the easiest way in the clinic is that you put your hands on the side of the patient's head, you sit in front of them, you have them focus on the tip of your nose, you turn their heads side to side, and you do a quick, rapid, small-amplitude thrust to one side. So what we're ultimately looking at is the VOR. Are they able to stay stabilized and focused on the target, or do their eyes come off the target and have to come back to correct for that? So again, this's a amplitude, maybe 10 to 30 degrees of motion, and it's almost like a wrist flick is kind of how I think of it.

Just this really quick motion where you just flick their head to one side really quickly. You'll see it right away, their eyes come off, have to come back to the target. And then the affected side is a positive test in the direction of the head thrust. This's caused by a low gain of the vestibular system. So typically, the head and the vestibular system

move at the same speed. If there's a low gain, the head moves first, the vestibular system needs a second to catch up. This has been shown to be a reliable, and actually, the best bedside test to determine hypofunction. The study was done outta Johns Hopkins University, in the hospital, and they found that the ability to determine hypofunction was much higher with this easy test versus putting somebody through all of the other testing.

The other important thing is to remember with this particular test is that every single time you do the test, it will be positive. If they have a true hypofunction, every time you thrust their head to the affected side, it will be positive every single time. So there's no correcting for it, there's no fatiguing, or anything like that. So here's kind of what could happen if they thrust their head to the left if their VOR is impaired, suggesting a low gain of the vestibular system, their eyes will go to their left, and then they'll come back to you in a corrective motion. In a normal response, their eyes would stay fixed no matter how fast you move their head.

Okay, now let's talk quickly about BPPV. I know we're coming up close to the end of time. BPPV signs and symptoms are dizziness with positional changes. So things like rolling over in bed, looking up, bending over, people typically will get vertigo, with these positional changes, that will last anywhere from 10 to 30 seconds. most of the time. They could last longer, they could last less, but it's this vertigo, the room spinning, with these positional changes. A lot of times they'll have nausea due to excessive dizziness. So if they're coming into your office getting a test for BPPV, or for VNG, if they've got a lot of nausea, it's really hard to do some of the testing. They also tend to have balance issues.

They feel like they're floating, they feel like they're swimming. A really simple way, and one of the things that's done within the VNG is the Dix-Hallpike test. This is the classic test for BPPV. The downside to the Dix-Hallpike is that the horizontal and anterior canal

do not have a significant positional change in this test if it's done by the textbook method. And so, you may not see a response if there's a problem with those tests. So we'll talk a little bit about things that you can do to potentially pull in those other canals. Because it may look like they don't have BPPV, but then they come into the clinic with us and we test them, and they have horizontal canal BPPV but you don't see it on a VNG.

I always say they have to keep their eyes open, obviously we've got to see what's happening with their eyes. It's important to explain that procedure thoroughly, and the expected results. This is considered the gold standard for the diagnosis of posterior-canal BPPV. So as I mentioned, many times you don't get the anterior canal or the horizontal canal in this. You always need to make sure you test both sides because of that coplanar pairing, and that sensory redundancy. You can get a response with BPPV on both sides, but you're looking at the one that's more commonly affected. And the posterior canal is affected more than 88% of the time. It's the closest to the opening of the utricle, so it's the one that, typically, the crystals will fall into too.

On this particular test, oh sorry. The patient will begin in long sitting, and it doesn't matter what their feet're doing, they can cross their legs, it doesn't really matter. The head is turned 45 degrees to the side being tested. If you are hanging their head off of the edge of a mat, then it's important that you support their shoulder with your forearm, you're holding the side of their head. You wanna make sure you're also supporting their neck. You wanna have them lie down as quickly as possible. Now, when I say to lie down as quickly as possible, they don't have to throw their bodies down. But if you think about in a normal situation, if you lie down at night, kinda the speed that you lie down if you have no problems.

No back issues, no neck problems, anything, you just lie down. That's about the speed that they need to lie down. So just a normal speed, it just can't be really slow. If they go too slow, then the crystals could be moving at the same speed they are, and you won't get a response. So it's important that they lie down at a pretty good speed. You wanna extend the neck 30 degrees, and that's gonna give enough of a tip of the posterior canal to allow the crystals to move. And you wanna have them focus on something, unless you're using infrared goggles or Frensel lenses, then you just wanna ask them to keep their eyes as still as possible.

Importantly, you need to maintain that position for 45 seconds, because it can take up to 25 seconds for the otoconia to traverse just 1/4 of the canal. Also, the endolymph thickens as we age. So it can take longer for clusters to move. So once you put them down in the position, it may take a little bit of time for gravity to kinda get a pull on that, and to move it through a thicker endolymph. If you want to include the anterior canal in this test, then you have to dip the head an additional 20 to 30 degrees. So the anterior canal, because it's mostly upright, if you add an additional 20 degrees of neck extension, so you're getting 50 to 70 degrees of neck extension is ideal, then you actually get the anterior canal to tip down far enough that you'll get movement of the crystals within the canal.

So you can actually, if you get enough neck extension, can test both posterior and anterior canal in this one test. So many times, if you've done the VNG in your office, and you know that people have neck problems, they have back problems, and they can't lie flat, they can't extend their neck back, they can't do the test, then you, you know, you ultimately just kinda say, "Unable to test because of patient pain," or "patient limitations," or those sorts of things. So the sidelying test is actually an alternative test to the Dix-Hallpike, it's a very valid test. The results're very comparable. So you can actually do this with the VNG. If they can't get into the traditional

Dix-Hallpike test, then you could put them into a sidelying test and get the same results.

So this's very adaptable to patients with limited range of motion, specifically in their cervical spine. And for those who have difficulty relaxing, if you're trying to put their head and hang it down over the edge of a mat and they won't let you tip their head down, it's hard to get a result. So many times, those who have difficulty relaxing, this's a really easy test that can be done in place of that. So again, this is primarily testing the posterior canal. But again, that's the one that's most commonly affected, upwards of... Well, more than 88% of the time, it's your posterior canal that's affected. So the patient begins by sitting on the edge of the mat, they turn their head 45 degrees away from the side being tested, and then they lie down to the side being tested.

So if I'm testing the right side, they're gonna sit on the edge of the mat, turn their head 45 degrees. If I'm testing the the right side, they're gonna turn their head 45 degrees to the left, and they're gonna lie down on their right side. So we're gonna get a tip of the posterior canal a little bit, there. Again, you're not gonna get as much, but you are still going to be able to get some posterior canal. That position is held for one minute. So again, it could take upwards, it needs to be held for at least 45 seconds. The instructions within the textbooks say that you need to hold the position for one minute. And you're looking, again, for any eye movements, anything that would suggest that there's a problem.

In the Dix-Hallpike, if the posterior canal is involved, you would get up-beating torsional nystagmus to the affected side. So if I'm doing a right Dix-Hallpike and the nystagmus that I see is up and to the right, then that's the side that's affected. Now, if I'm also getting up-beating torsional nystagmus on the left, when I test the left side, then that could be a bilateral problem. If it's the anterior canal, what you typically would see is a down-beating torsional nystagmus to the affected side. But you can see pure

down-beating nystagmus. This's atypical BPPV, where there's no torsion associated with it. It just shows pure down-beating nystagmus, 'kay? And so, if it is positional in nature and there's no spontaneous nystagmus associated with it, this is likely not central.

It is likely the anterior canal, You can't rule out that it's not central, but positional nystagmus pure down-beating could be anterior-canal BPPV. And if there's horizontal nystagmus, then you would do a roll test to confirm that. There're two forms of BPPV. There's a canalithiasis, those're symptoms that last less than 60 seconds. And there's a cupulolithiasis, where the crystals're stuck on the cupula. Those symptoms would last greater than 60 seconds. To determine horizontal canal, you can do a roll test. Same idea, they have to keep their eyes open, they must stay in the provoking position. The difference is, patients experience vertigo and nystagmus in both directions due to debris moving back and forth within the canal. Because you're turning both horizontal canals at the same time.

To do this, they lie supine with their neck flexed 30 degrees. This's to position the horizontal canal perpendicular to the horizontal plane. You can turn their head 90 degrees to the affected side or one side as quickly as possible. You're having them focus to see if you can see any nystagmus, unless, of course, they're wearing goggles. And then you would repeat that to the other side. If the patient is unable to turn their head 90 degrees, they can turn their entire body. It does not matter if it's just their head or just their body. Again, the results're slightly different for horizontal canal, but there are two forms. Canalithiasis basically means that the crystals're floating freely within the canal. So if that's the case, you would have a geotropic nystagmus, means if they laying on their left side, the nystagmus is beating toward the ground, or their right side is beating toward the ground.

If it's a cupulolithiasis, the nystagmus is going to be beating away from the ground. That means the crystals are stuck on the cupula. And that's just important for therapists, because we treat those differently. Canalithiasis, cupulolithiasis have completely different treatments in the clinic. And so, if that's something that is available, then that just makes it easier on the therapist. So real quickly, let's talk about recovery. So BPPV should resolve quickly if you test it, if you get the right canals involved, and the type, the form as well. Then the symptoms should resolve quickly. So many times, if they get to a therapist and the therapist treats the BPPV, many times, one treatment is all they need. And a lot of times, things like the canalith-repositioning maneuver, that is about 97% effective after two treatments.

So a lot of times, that can be very quick. And as we mentioned earlier, they found that prompt treatment of BPPV showed a quicker return to work, play, or duty in the terms of, for those military patients with blast injuries. However, recovery after a concussion can take months. So again, when we're looking at the different types of of issues, pediatric-patient recovery took an average of 40 days if they had vestibulo-ocular dysfunction. So if there were visual-motion sensitivity, if there were issues with VOR and a low gain of the vestibular system, those patients took longer to recover than those that did not have any vestibulo-ocular dysfunction. So their eyes, they were able to stabilize, et cetera, those patients only took upwards of 21 days for recovery.

Patients with fixed peripheral vestibular deficits should be improved by three months. So these're going to be people who have just BPPV. That should be a pretty quick recovery. These're gonna be people that have just a unilateral hypofunction. So I tell people with hypofunctions, if that's the only problem that they have, that the average recovery is gonna be six to eight weeks. So, many times, I'm sure, when people're coming into you and they're getting this testing done, they're very anxious, they're very scared, they're not 100% sure what exactly is going on, how long is this gonna take to

recover, what're my tests show, all of those things. But if it's a fixed peripheral vestibular deficit, it should be improved pretty quickly.

If there're central issues, peripheral issues, visual issues, auditory issues, all of those things, obviously that is prolonged recovery that can occur. So in summary, dizziness is the second most common symptom, after headache, with concussions. So people that do have head injuries, most likely, they're going to have a headache. Dizziness's gonna be the second problem. Visual issues also're gonna play a role in there. It's a predictor of prolonged recovery. However, vestibular rehabilitation has been shown to reduce recovery times and improve overall function. So there's a shorter time to, a shorter time to intervention will improve the outcomes faster. So one of the things that I always encourage is I work very closely with a lot of ENTs in my community, and the audiologists that do the testing for these ENTs.

So I think it's a great idea if you can find a vestibular specialist. I know that most physical therapy practices say they treat vestibular rehab, but many times they may treat BPPV in one form. They may do some balance therapy, but they're not doing the pure vestibular rehab on that. So I always encourage for you to find a vestibular specialist in your area. If you need to find a vestibular therapist, there is a website called vestibular.org. I have no affiliation with them, but you can find a provider on their website. And those're people that do have to go through and talk about what their credentials are for being a vestibular therapist. And so, you can usually find someone in your area.

But getting people who have dizziness and head-injury issues, visual issues after dizziness, that come up positive on all of these tests, getting them to a vestibular specialist will improve their recovery significantly. So, I hope that everybody got some good information. I don't have any questions, so I will let everyone go. Thank you again for joining me on this Friday afternoon.

- [Calista] Well, thank you so much Tonya, and thank you, everyone, for attending. We'll go ahead and officially wrap up. Have a great day, everyone.