Benign Paroxysmal Positional Vertigo - Ref 68RG

with Rudi Gerhardt

25th June 2020

TRANSCRIPT

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Steven:

I'm joined today by Rudi Gerhardt, who's joining us from Australia. So it's nine o'clock in the evening for Rudi, and he's going to be talking to us about BPPV. Rudi, welcome to the show, excuse me, for my complete inability to cope with time differences. And not only with you, but in my own country as well. Rudi, you've had 20 years, haven't you, as a lecturer in neuroscience at university level.

Rudi Gerhardt:

Yes, it's one of the subjects I taught and always enjoyed. I taught embryology and other subjects and the more hands-on practical subjects, but that was always the one which fascinated me the most.

Steven:

I noticed from your website that you've got a particular interest in vertigo. Why is that?

Rudi Gerhardt:

I work in a relatively small community and I saw the need. Often people get the run around, they go from one specialist to the other and the neurologist says, oh, it's not a neurological problem. The ENT says, no, it's a neurological problem. And nobody actually encompasses the whole spectrum, which is quite a lot of different disciplines. So, it could be cardiovascular, it could be something in the inner ear, it could be cerebellum or brainstem. So, it encompasses quite a lot of specialties andI saw that there was a need for the community, somebody to pull everything together and help those people which can be quite debilitating.

Steven:

Well, I suppose one of the questions that we might have is what is the incidence of this? You live in a small community and yet you're clearly seeing enough cases to justify putting a lot of effort into BPPV.

Rudi Gerhardt:

Yeah. BPPV is probably the, I know if you look at literature and what I see in the community is the most common vestibular or balance problem we see. And then it's confirmed in studies published as well. So, it's fairly, fairly common.

Steven:

Right. And is it readily diagnosed or does it go overlooked in say general practice or perhaps even in other manual therapy practices?

Rudi Gerhardt:

I think it's underdiagnosed often it's put down. The patients often come in and see me and say, I told my GP, that GP said it's your blood pressure, and then I said, what's your blood pressure measured in, lying down and sitting? If you think orthostatic, hypertension, for example, which can be a cause it wasn't done. It's just the patients often not serviced properly, I find. It's underdiagnosed, definitely.

Steven:

So, talk us through what you would expect to see in clinic in a BPPV sufferer.

Rudi Gerhardt:

I think we should be for understanding just go over the anatomy and physiology a bit which bridges or helps us to understand what's going on and how we treat it and how we diagnose it as well. I think that will give us a bit of basis because I think for most of us, it's probably a while ago that we covered that the area and being a lecturer, I find that there's often very little time to cover that topic. I've got 10 minutes generally in neurology to cover it under the cranial nerves, it may be another 10 minutes it's covered in the cerebellum and that's all we get out of the nystagmus system. So, our knowledge, and it's not just in the Allied Health I have friends, who are GPs and they're in the same boat, t's very little coverage, so I think it's probably good to give us a bit of a base to start. We've got quite a big model here. So, this is the left inner ear. So, this is basically the membranous part, it sits in the temporal bone. Can everybody see? That's the anterior part, so it sits in the temporal bone like this. So, this is a left inner ear. The anterior portion is the cochlea, which is for hearing, and the rest is the vestibular organ. In the middle we see the vestibule, which basically consists of the utricle and the saccule, and then we have the three canals, the posterior canal, the horizontal canal and the superior canal, and each canal basically reacts in each plane. So, if you look, the horizontal canal reacts to rotations of the head or the body, the superior canal to side bending movements, and the posterior canal to nodding type flexion/extension movements. We can see here dilations of the canals, they're called the ampullae. Within the ampulla is the ampullary crests and that's the sensory organ. Within the crest are embedded hair cells, and over the hair cells is a jelly-type hood which is called the cupula. So, if my fingers are the hair cells, the cupula sits on top. I have to mention that all the canals, including the cochlea, are filled with endolymph, which is fluid. So, when we move our head, the fluid brushes over the cupula and the hair cells and bends the hair cells. That's how we know we're moving, we're bending our head sideways or we're nodding or we're rotating. The canals react to angular type of movements, meaning rotations in each plan, the utricle and the saccule are different. Again, we have hair cells embedded, which are the sensory organ, on top of the hair cells we've got a membrane, it's called the otolithic membrane. The membrane is weighed down with crystals, they're basically calcium carbonate crystals. That's basically adding weight. So, if you go up and down in a lift or you jump, the weight on top of the hair cells bends the hair cells. That's how we know we have gone up and down. So, the difference between this and the canals is this reacts to linear acceleration, so up and down movement in linear fashion or accelerations and decelerations in anterior posterior movements. So, this is basically a gravity organ, whereas the canals don't react to gravity, purely to angular movements.

Steven:

I'm always in wonder when I look at this, because it's such a lovely, in theory, simple mechanism, but it's just beautiful the way it operates, isn't it?

Rudi Gerhardt:

Yeah, we're not aware and are all the senses are very obvious. Vision is obvious. Hearing is obvious. Touch is obvious. We're not aware of the vestibular organ unless something's going wrong. And I think it was the last sense which actually was discovered and I think we're still lagging behind on knowledge, and also on treatment approaches, because of that. So, if you look at that pathology now. I said the canals are normally not reacting to gravity, what's happening is basically the crystals

sitting on the membrane, they can loosen from the membrane and then make their way into any of the canals. And suddenly the canals react to gravity. And of course, every time you're moving your head, a common feature is people said, I'm turning over in bed and suddenly I start to spin out. The cause often is idiopathic, that's a word we often use when we don't know where it's coming from, and the other cause trauma. I receive a lot of people after concussions, for example, I had just a recent patient. So, the otoconia loosen from the otolithic membrane and make their way into the canals and then you get BPPV. So very typically if you look at the history of a BPPV patient, it's triggered so often the triggers could be the typical textbook top shelf vertigo, you're looking up and rotating the head. And if I simulate to see, can you see the rings there?

Steven:

Yeah, we can.

Rudi Gerhardt:

Yeah. And that's basically representing the otoconia inside. So, if you lie down and put your head backwards then the ring will move and then you turn the head, which actually puts the canal wall into gravitational force, and then you get movement of the otoconia inside the canal. And therefore, that's what the experience is of vertigo.

Steven:

That kind of puzzles me, Rudi. The crystals you say, are responding to gravity, I would have thought that they would have interfered with the sensation from any one of those angular receptors, but I can't understand how the crystals themselves suddenly create a new sensation.

Rudi Gerhardt:

There are conditions, for example, in vestibular neuritis where we have vestibular hypofunction due to damage to a nerve or it could be damage to the sensory hair cells, which leads to a hypofunction. Here we get a hyperfunction because the crystals have a higher density than the endolymph fluid, and therefore they've found that a lot of people had crystals, in dissections of cadavers, that a lot of people actually have crystals within the canal. But what's thought is it has to be a certain amount of crystals, which form a plaque, and then the plaque moves along and creates hyperfunction. So, an overreaction of the system, because it has a high density, then the endolymph fluid.

Steven:

Okay. That explains it then. Yes, you're right. And I was going to say are there cases where people are discovered to have had completely benign crystals in those canals. Pip Slack has asked a question, she says do the crystals degrade over time and disappear, or once they're in the semicircular canals, are they stuck there permanently?

Rudi Gerhardt:

That's a good question because what we find is there are certain cells, there's a turnover off the otoconia, or the crystals, but the turnover seems to only happen in the area where they're situated normally. And so, you have an absorption of the cells over time. So, they renew themselves, but it seems, what I gather from the literature, that the crystals only reabsorb in the utricle and the saccule, where they're normally situated, and not in the canal. I had a person just recently, a 72-year-old, who had BPPV for 25 years. Wasn't diagnosed. And I tested him, it was 10 minutes testing and the

treatment was two minutes and he stood up and said, was that it? I said, yeah. I said, come in in two days and see how you go. And he came back in two days and said, I couldn't believe it. He had this for 25 years and you do one maneuver and basically, he was symptom-free. So, it looks like that the crystals within the canals don't dissolve, they only dissolve and renew themselves in the vestibule or the otolithic organs.

Steven:

So, when somebody comes to present to you, presumably one of the things they're going to complain of, the main thing, is they're going to complain of what they might describe as vertigo, or they might describe as dizziness. Is that right?

Rudi Gerhardt:

Yeah. This is a very broad term. So, if you look at criteria it has to have to be actually vertigo. Vertigo, it depends which criteria you're reading. If you look at the Barany Society, they're saying it's movement, a perception of movement doesn't exist. That's one. Or others describe it is spinning type movements which are basically illusionary, but the person obviously experienced it.

Steven:

So, the point of the question of course is, and I suspect you were going to cover this, is when someone comes in and they won't necessarily know the correct term, they'll say dizziness or vertigo, perhaps. How do you distinguish what is treatable? What perhaps needs referral elsewhere or should be left alone?

Rudi Gerhardt:

That's a very important question because there's certain central vestibular conditions, let's say mini-infarcts in the brainstem. They're not necessarily always obvious. Infarcts strokes, are very obvious, but there are mini strokes within the posture circulation, which often affects the cerebellum or it affects the brainstem. Of course, we've got the vestibular nuclei on the brainstem, so they can be affected as well. But the obvious signs, they're not really obvious unless you look for them. And there's a distinction between what's called peripheral nystagmus and what's called central nystagmus. And to give you a few examples: a peripheral nystagmus, there's people and right as the person comes in with spontaneous nystagmus, for example- spontaneous nystagmus means without any provocation, I'm not putting them in a provocation position- the person comes in and has a left beating nystagmus. By the way, we determine the direction of the nystagmus on the fast phase, often it's diagnosed and there's slow phase and there's a fast phase. So, the direction is determined by the fast phase. So, we're talking with common language. A person comes in with a horizontal beating nystagmus and it's one simple thing you can do: you ask the person, can you look to the right? When the person is looking to the right, is the nystagmus still beating to the left or is it changing direction? Can you look up for me? Is the nystagmus still beating to the right? Or has it changed the direction of beating up? If the nystagmus is changing direction, then it's a central problem, I immediately refer them on. Whereas if it's a peripheral problem like vestibular neuritis the nystagmus doesn't change direction, irrespective of gaze, we call it gaze evoked nystagmus or GEN. Doesn't matter which direction the person looks, if the spontaneousness diagnosis was to the left, for example, and the person looks to left, it beats to the left, looks to the right, the nystagmus still beats to the left, looks up or down, it still beats to the left. So,, it's a non-direction changing nystagmus. It's a peripheral nystagmus.

Steven:

Can I ask a quick question from somebody that's just come in? Jan says, she recently started having tinnitus about three months ago and a few days ago started having dizziness and the spinning room a few times when waking up and opening the eyes in the morning. Could there be a connection between that and the recent onset of tinnitus, do you think?

Rudi Gerhardt:

Yeah, as I initially said the cochlea, which is obviously responsible for hearing and tinnitus is an auditory problem, and the vestibular organs are connected. And there's typically Meniere's disease which affects both. The theory is that the endolymph pressure is too high in Meniere's disease because the endolymph is continuously absorbed and produced. And it's still a question, is it an absorption problem or is it too much production of the endolymph? It leads to increased pressure and the destruction of hair cells in the cochlea is as well as the hair cells in vestibular system. That's one for example where the hearing can be affected and people can have a balance problems or vestibular problems as well. Generally, the hallmarks of Meniere's disease are vertigo and then of course we get the hearing loss. So, these are the two major hallmarks. Another feature people describe is increased pressure, they feel a pressure in the ears, so they can't equalise. They say, I can't equalize the pressure, like going up or down in a plane. Sometimes referred to as aural fullness. So, these are the hallmarks of Meniere's disease. So that could be the case, it has to be investigated, but that's an example where both of them can be affected. And it's not a surprise because it's continuous.

Steven:

So, do you want to run us through what you would do in clinic to carry out your diagnosis and treatment?

Rudi Gerhardt:

Yeah. If you can play the video for horizontal canal just to give people an idea of what a nystagmus looks like. This is an example of a patient who had a horizontal canal BPPV. And the beauty is we can determine which canal is involved. If the horizontal canal has crystals floating in the canal. You get a horizontal beating nystagmus if the posterior canal is involved, you get an up beating nystagmus. So, from the direction of the nystagmus, we know which canal is affected and then apply the appropriate maneuver for it.

Steven:

Okay. I'll play this on the screen behind me, because we've lost the full screen function on the video, but let's see how it goes.

Rudi Gerhardt:

That's just the test, what's called the roll test, particularly for the horizontal canal. Can you say the beating to her left, because it's a mirror image obviously? The duration is very short, as you can see, and that's what people report. They're doing the same now, test to the right. See that was much stronger to that side, to the right. So, we know the right canals involved, because it was the stronger nystagmus. And we call that a geotropic nystagmus because the head in this case was turned to the right with the patient supine and the nystagmus beats to the downward ear. So, if the patient is lying down and rotated to the right, the nystagmus was beating to the right side, so the downward ear,

towards the ground. And that's basically a term they use geotropic horizontal nystagmus. We knew that was her right canal and we knew it was horizontal because the nystagmus was beating horizontally.

Steven:

Right. It sounds like a silly question because we heard the response from that patient, I was going to ask whether a patient who is suffering from a nystagmus is always conscious of the fact that their eyes are doing that or does the brain somehow cope?

Rudi Gerhardt:

The patient most of the time is not conscious of it. They feel the spinning but they're not conscious of the eye movements, only an external observer. Some people claim, I know my eyes are moving, but generally not. It's has to be an external observer who basically looks at it.

Steven:

Right. And Christine has asked whether it's always both eyes.

Rudi Gerhardt:

Yes. There's only one condition, it's called INO, which means internuclear opthalmoplagia, that's why we abbreviate it, it's a bit of a mouthful. Basically, normally we have what we call conjugate eye movements, our eyes move always in pairs, the same direction up or down or sideways. Internuclear ophthalmoplegia, because we're talking about the nuclei in the brainstem, like oculomotor, trochlear nuclei, and abducens which basically moves our eyes about, and obviously the right and left are wired together, they're connected. And obviously if something's wrong with that connection, that's the only situation where the eyes don't do the same thing. Otherwise, they always do the same thing. But it's a very rare condition, INO, but otherwise it's always conjugate eye movements.

Steven:

Andy's asked what are your thoughts about checking the vestibular ocular reflex and its accuracy for BPPV? Is it worth doing to differentiate between horizontal nystagmus and other conditions such as cerebellar ataxia and multiple sclerosis?

Rudi Gerhardt:

No, VOR really, we use generally what's called the HIT, which stands for head-impulse test, and it's generally a test for peripheral conditionals, probably the most common one is vestibular neuritis. I've got obviously goggles which can do vestibular nystagmogram and it prints me out the gain. Basically, the VOR is the fastest reflex we have in the body so the gain is about a one. What is the VOR? If I want to look at my thumb and turn my head and don't want to lose my thumbnail and the gaze on my thumb, it's called gaze stabilisation. So, when I turn my head to the right the eyes have to move the opposite way in order for me to focus on my thumbnail. It's basic, the VOR the purpose is to have stabilised gaze. And the gain is about one, that means when I'm turning my head to the right, the eyes move almost immediately to the left, it's the fastest reflex. So, we can measure the gain. What we see in vestibular neuritis is that the gain is reduced because the nerve's obviously not functioning, it's slower. The other thing we see is what we call catch-up saccades. Normally when I'm turning my head to the right, the eyes do one quick movement to the left. And again, I can see in my print out from my vestibular nystagmogram, or in the past, when they didn't have the

technology, people said, look at the camera and turn their head, and then look at it in slow motion. You can see the eyes, rather than having one movement, they're not getting there fast enough and have a catch up. And that's what we can see obviously in the printout and the curve. But we don't find that's the problem actually in BPPV. It's almost a hyperfunction. Vestibular neuritis is a hypofunction, we have a decrease or slowing down of the VOR, whereas it's the opposite in BPPV really

Steven:

Is nystagmus always present in BPPV? A lot of people have asked that question.

Rudi Gerhardt:

It's not always, and it's a problem clinically. And sometimes I purely rely on the symptoms of the patient. Basically, you're telling me, I'm feeling dizzy, I'm getting nauseous. And often when people have it for a long time or they're on medication vestibular suppressant medication, that's a problem as well. So, I tell the people, no, you take any vestibular suppressants? Can stop the medication and come back in a week? So, the effect of the medication is reduced. These are the two examples. Or people had it for long periods of time. And there is a process which is called vestibular compensation. It's a central process where the central nervous system adapts to conditions. That's often happening in vestibular neuritis and the effect is seen within two weeks, but very rarely that much in BPPV.

Steven:

Okay. Lucy sent in a question relating to a patient. She asks, whether an incident of vertigo 15 years ago might be linked to a recent diagnosis of acoustic neuroma, vestibular schwannoma. She says the patient's presentation, it's affecting their hearing on one side and they still get unsteady, even watching children on a roundabout

Rudi Gerhardt:

There is always the possibility it depends how large the schwannoma is, that's the recent term we use now. Depending on the size you can affect the vestibular system and has to be always ruled out. Hearing loss is a problem. We see hearing loss obviously in Meniere's disease, which we just discussed before, but acute hearing loss has to be investigated. It depends. There's a frequency, in age related hearing loss it's often high frequency which we start to lose. Meniere's disease it's actually the low frequencies we start to lose, total opposite. The patients say, okay, this sounds very tinny, it sounds very thin. It's opposite to the age-related hearing loss. But acute hearing loss, we have to assume if it's very upright, that it might be an artery in the posterior circulation, which serves the organ of hearing. And that's now included in, if somebody comes in with acute vestibular symptoms, they often end up in hospital. I see them sometimes because I've got a reputation so they send them to me now, but often they're in hospital, we do basically a whole series of tests. The finger rub is one of the tests we do. We add to that and say, okay, how quickly has your hearing deteriorated? And we have to assume if it's very quickly, it might be a mini stroke in the posterior circulation. That has to be ruled out.

Steven:

So, with your very clever goggles, you showed us what your patient's eyes were doing. Could you talk us through the test that provoked that?

Rudi Gerhardt:

Yeah, I've got a video there, which is called the Dix Hallpike test. So, if you could show that. My wife volunteered. It's important to inform the patient that is provocative, that is provoking the symptoms. We're testing the right posterior canal here. We're doing the same now for the left posterior canal.

Steven:

So, Rudi, you asked your wife there, how do you feel, when she was lying down. Now clearly, if she said she experiencing vertigo, that presumably is a positive indicator. Are there other things that might give you a concern, other sensations?

Rudi Gerhardt:

Obviously, the nystagmus. She would display a nystagmus.

Steven:

When you asked her what she was feeling, is there anything other than the vertigo itself that might come out?

Rudi Gerhardt:

Yeah nausea. Nausea is very common. You get autonomic symptoms, nausea, in extreme cases vomiting. So that's very common I only had once a person vomiting, but she had multiple canals involved after a trauma. And there's only a limit of testing it can do. I basically had to break it up and say, okay, come tomorrow again. We'll do the other canal tomorrow because it was just, we're close to the buckets, put it that way.

Steven:

And interestingly that Dix Hallpike test has some similarities to the, I think, largely discredited test for vertebral artery insufficiency. And we were always told to look for nystagmus and that was a red flag for any cervical manipulation.

Rudi Gerhardt:

Yeah. Just before we go into that, if you bring the person, the sound wasn't very good, I don't know if it was on your side, into the Dix Hallpike position, the onset of the symptoms and nystagmus, what's typical is that there might be a delay of up to 10 seconds. So, you've gotta make sure you stay in the position at least for 10 seconds, because the onset of the symptoms and the nystagmus can be delayed, which is quite typical. The other thing, when the person comes up the nystagmus reverses and the next video, will show that. But to get to your question you're referring to the decline test, where we're apparently compromising the vertebral artery. There's a recent study of physiotherapists in Australia and they looked actually and the vertebral artery was more, the flow was more impeded in flexion than in extension, which actually really confirms that the test wasn't very valid. The other thing I say to people, if you're concerned about the extension rotation position, the vertigo and the symptoms will be exactly opposite. After 30 seconds, you hold the person in their position, the nystagmus disappears, we saw that in the video before, and the symptoms get better. If it would be vertebral artery and you stay in your position, the longer you stay in a position because it's impeding flow to vital structures, the more the symptoms would increase in intensity.

Steven:

Whereas in BPPV, the crystal will have come to rest and therefore it'll stop the nystagmus.

Rudi Gerhardt:

Exactly. If you look at the ring here, once you got that in position it rests here and then the person stabilizes again and the symptoms disappear. Whereas if it were VBI, then the symptoms would increase.

Steven:

Mikael has asked us whether there's any need for speed in the Dix Hallpike test?

Rudi Gerhardt:

There have been actually quite a few studies now done in different countries looking at speed of the test but also the correction maneuvers. And all those studies showed speed's not important. It's the time that you allow the crystals to settle and allow the movement of the crystals, particularly when we do the correction maneuver. So, it's more giving the crystals time to move through. Remember it's a plaque of crystals and they need a bit of time to travel through the narrow canal. So, speed we now know is not important. In the past they thought you have to do it quickly, but all the studies show, give the crystals time to move through the canal. It's more important. There were other things like tapping, vibration, totally unnecessary. All the studies show we don't need that.

Steven:

Sally's asked, I think you might've mentioned this, but Sally's asked what those crystals consist of.

Rudi Gerhardt:

It's a calcium carbonate. They lie embedded in the otolithic membrane. The crystals sit on top of the membrane. Or the otoconia, that's another word for them. So, they're basically calcium carbonate crystals. And they sit on a matrix, which is sort of a gluey substance.

Steven:

You mentioned some medications earlier on that affect the vestibular mechanisms and Pip's asked if you've got any examples of which ones do that?

Rudi Gerhardt:

Serc is often given, there's the vestibular suppressant in there. Stemetil has generally a bit of vestibular suppressant. Stemetil is mainly an antiemetic drug, meaning taking care of the nausea and vomiting, but also has some vestibular suppressants in there. They're probably in Australia the two most common ones prescribed. In the UK, they probably have different names.

Steven:

Rudy, as always, we're running out of time. We've only got five minutes left and I wonder if we could move on to your next videos.

Rudi Gerhardt:

Yeah. Let's do the Epley maneuver.

Steven:

Your wife looks very nervous.

Rudi Gerhardt:

She was brave. So, that's basically the beginning of the Dix Hallpike test. And you gotta be really there because the patient feels very disoriented. And it's important that they know you're there.

Steven:

There's a couple of things in there, Rudi. When you had your wife in the initial position you said, observe for nystagmus. So what? What if you see it? What if you don't see it?

Rudi Gerhardt:

The next video is actually showing the nystagmus that I was talking about. If you play that it'll answer the question partially.

Steven:

I think we have to go back for that one to this one.

Rudi Gerhardt:

Yep. That's actually an example of left posterior canal BPPV.

Steven:

So, the patient is currently facing to their left?

Rudi Gerhardt:

Yes. In the left Dix Hallpike position. It's an up beating nystagmus and it's a slight rotary component to the left, which tells me it's the left canal. See how quickly the symptoms disappear? So, it already correlates with the patient saying, oh, I'm feeling better. It's already slowed down.

Steven:

Okay. So once again, I would say when you put them into that initial position in the Epley maneuver, if you don't see nystagmus, does that mean that there's no point in going any further?

Rudi Gerhardt:

If you had a positive Dix Hallpike on the left side, and you've seen that nystagmus, then you would proceed. But there's a problem, because typically if you repeat the Dix Hallpike again, or you go into the Epley maneuver, which is basically like the Dix Hallpike, and then you move on. With repetition within a certain period of time, the symptoms get less. And that's a hallmark again for diagnosis that you know it's BPPV. So if, let's say, you have a positive Hallpike to the left, very obvious nystagmus, which we've seen here, and you go then into the Epley maneuver and the symptoms are less, it's normal because it's almost like a repeat of the test, so don't get discouraged. I'm still going through with the maneuver because that's diagnostic. The explanation is that because you dispersed the

otoconia and they've not formed a unique plug again. And therefore, the second time you do the maneuver, the symptoms are less and other nystagmus is less obvious.

Steven:

Before we close, Rudi, I've got a few questions, if I may, from the audience. Fiona says, she seems to be getting BPPV symptoms with increasing regularity and she's heard that similar symptoms can occur due to trigger point issues, for example, effecting the neck muscles. Is there any truth in that that you're aware of?

Rudi Gerhardt:

I think we're going in a completely different realm, which is referred to as cervicogenic vertigo or dizziness, which exists. We have now tests and we can test precision error in any joint. We can now do that in the neck as well. And cervicogenic vertigo is probably 30% of my diagnosis in the clinic and then we have really success with trigger points working on suboccipital muscles, the OAA complex. So, we can have a laser on the forehead and have a chart and ask the person to focus on a crosshair with your laser, close your eyes and then turn to the left and now go back where you think you were before. And the accuracy is normally quite good and stays within probably a two-degree angle. If I have a cervicogenic component, it's beyond four, four and a half degrees. So that's, again, we're going into a different realm here.

Steven:

Fiona says that there's a rumor that the Epley maneuver can cause the symptoms on the other side or sometimes both sides. Is that true?

Rudi Gerhardt:

No. It's very rare. If you did the Epley maneuver, then you can convert it to another canal. So not on the other side, on the same side, but you might move it into the horizontal canal, but it's very rare. Often, it's a problem with the technique, if you look at the literature, if the techniques not properly done.

Steven:

Mikael's asked about those repositioned crystals, do they reattach in their previous location or they just get broken down once they've left the canals?

Rudi Gerhardt:

The consensus is they think that they are absorbed and broken down.

Steven:

Right. And Sally says, does the Epley maneuver need to be repeated a few days later if the symptoms persist?

Rudi Gerhardt:

If you're looking at the success rate particularly with a posterior canal, which is the most common canal involved. The success rate is about 90% with one treatment. So, 10% have to come back but very rarely I see it in the clinic. It's really, most patients are blown away. They say, was that it, is there

anymore? On the first few times, I just couldn't believe in myself. It's really a very successful maneuver, in 90% of the cases only one treatment necessary. There's obviously the 10% where we have to repeat it.

Steven:

Two final ones, if I may, and I know we're over our time here. Annabel's asked a very useful, interesting question, if you've got more than one semicircular canal involved, does the Epley maneuver still work to correct the symptoms? Does it work on all the canals?

Rudi Gerhardt:

No. That's why I teach this in two days on the weekend. Primarily it works for the posterior canal involvement, which is the most common involvement.

Steven:

Which is rotational movement?

Rudi Gerhardt:

Yeah. And there's some literature showing on the anterior canal, but the anterior canal nystagmus does not have a strong rotary component. So, it's very hard to determine the side. So, there's a simple maneuver, which is called the Yacovino maneuver. Where basically we bring the head in a head hanging position in a neutral rotation and, it doesn't matter, you correct both canals. But horizontal canals have four variations. We have four maneuvers for the horizontal. Horizontal canal is very, very difficult because it depends where it is. It can be attached to the cupula, it can be the posterior portion of the canal, it can be the anterior portion of the canal, and each has a different maneuver depending where the otoconia sit. So, we have about five maneuvers. The Epley maneuver is primarily for the posterior canal, not for the horizontal canal at all.

Steven:

Mikael's asked whether there's any updated healthcare advice after you've done that? Apparently, people used to be taught that you had to sleep semi-upright and not on the treated side.

Rudi Gerhardt:

Yep. I used to give that advice in the past, but there's been three or four independent studies showing that it did not make a difference in the outcome and long-term outcome. People who were sleeping 45 degrees up or go to the hairdresser and have their head hanging back did not make any difference at all. So, I've stopped giving that advice because it didn't affect the outcome.

Steven:

Okay. Thank you. Thank you very much, Rudi.

Rudi Gerhardt:

Thank you for having me and see you soon.