Dr. Jason Barnes:

Hey there. Welcome to another episode of ENT in a Nutshell. My name is Jason Barnes, and today we're joined by Dr. Devin McCaslin, a PhD in audiology who specializes in dizziness and is the director of a large tertiary vestibular center. And we will be discussing benign paroxysmal positional vertigo, otherwise known as BPPV. We're also joined by Dr. Matt Carlson, neurotologist, who will be here to tie in clinical pearls into the otology and neurotology world.

Dr. McCaslin, Dr. Carlson, thanks so much for being here.

Dr. Devin McCaslin:

Good morning.

Dr. Matthew Carlson:

Thank you for having us.

Dr. Jason Barnes:

Dr. McCaslin, when you see a patient with BPPV in your office, what are some of the presenting symptoms and what are some questions that you ask to kind of tease out what might be BPPV?

Dr. Devin McCaslin:

Sure. These patients, when they come in, primarily complain of when they bend over, look up, any sort of head position would provoke the dizziness that they're complaining of. Most often, these patients will note that they become very, very dizzy when they wake up in the morning. That's usually the first time that they'll encounter this. Then as they go through the day, as they bend over, look up, roll over, what they'll do is they'll get this very brief vertigo anywhere from 10 to 20 seconds.

That is when queried is a true vertigo and it can be associated by a little bit of nausea. If they don't move their head, if they don't move at all, they don't provoke the vertigo. So this can continue and continue. Well, often sometimes spontaneous resolve, but then also may continue for weeks or months.

Dr. Jason Barnes:

Are there any risk factors that you ask about that could maybe tip you off that this is more BPPV than something else?

Dr. Devin McCaslin:

Yeah, no, that's a great question. The majority of cases of BPPV are actually idiopathic. Just, occasionally, they wake up and they've got it, but things we really have to sort of query is were they in a motor vehicle accident. A lot of times that is one of the common results of getting BPPV. Any sort of effect of a vestibular pathology. Oftentimes, we'll see a patient with a neuritis. We'll see a patient with a labyrinthitis.

In fact, when you look at thousands of patients, 25% of people with a neuritis or a labyrinthitis will often incur BPPV. It's also very common in Meniere's disease. Other issues like ischemic processes, autoimmune, all need to be considered. But it's pretty clear. You can differentiate from the case history pretty quickly whether or not this is BPPV.

Dr. Matthew Carlson:

I'd like to add one thing from a clinical side, from an otologic side. It's not super common, but it definitely occurs that you'll have a patient that comes back postoperatively, whether it was a cochlear implant or a stapedotomy or another procedure, and they'll be doing well for a while, and then several weeks later they'll report vertigo, and then when you ask them more specifics it will become readily apparent to you that it's more characteristic of BPPV. You'll do a Dix-Hallpike and they'll have a positive finding, as we'll talk about in a little bit.

So as Dr. McCaslin alluded to trauma or even iatrogenic just from surgery, even if the surgery went perfectly well, sometimes people report this. Naturally, right after surgery, you'd think it was a complication of surgery. They're still dizzy because you're doing something with their ear, but some cases are BPPV.

So I'd always encourage people to look for BPPV in the postoperative setting, particularly if the symptoms are really consistent with that and not a prolonged unabated dizziness.

Dr. Jason Barnes:

Dr. McCaslin, is there anything from an epidemiologic standpoint, gender, age, that kind of thing, that you see more commonly in patients with BPPV?

Dr. Devin McCaslin:

Yeah. Approximately, 6 million people present to the US healthcare system with dizziness and 20% of those are going to be diagnosed with BPPV. That makes it the most common form of vertigo that we're going to see, regardless if you're a frontline provider or even a subspecialist.

BPPV is about seven times higher in those older than 60 years when you compare them to their 18 to 39 year old counterparts. This has to do with some of the issues with aging and the otoconial aging. It's been found in 9% of the elderly population, and it's more common in women, almost from a 2:1 to 3:1 ratio.

Dr. Jason Barnes:

Dr. Carlson, when you see these patients in clinic, and we'll talk about the diagnostic maneuvers in the workup section, but what are you looking for on physical exam to tease out the etiology of their vertigo?

Dr. Matthew Carlson:

Absolutely. When you see a person in clinic and you suspect that they might have BPPV, particularly if they're giving the characteristic history of them rolling out a bed in getting dizziness or tilting their head back and feeling a vertigo for a short period of time, which is really quite diagnostic for the condition, you really want to rule out other potential causes for it. We'll get into the differential diagnosis later. But really the physical examination in this setting is to exclude other pathology.

They should have a normal otologic examination on otoscopy or otomicroscopy and at rest, without any provocative maneuver, they shouldn't have any spontaneous, symptomatic, vertigo or nystagmus on your examination. And so your full cranial nerve examination, neurologic examination, everything else should be completely normal, aside from what we'll talk about in a minute, being a positive Dix-Hallpike maneuver.

Dr. Jason Barnes:

Dr. McCaslin, as we move into the pathophysiology behind BPPV, I wanted to first talk about anatomy. The semicircular canals, I feel at face value are somewhat understandable, but there are a lot of details

to them and some of those might help us understand this pathophysiology. So could you briefly tell us about the anatomy of the semicircular canals and the vestibule?

Dr. Devin McCaslin:

Sure. It's interesting because BPPV is an interplay between both the otolith organs and the semicircular canals. So the semicircular canals, of course, are housed in the bony labyrinth. They're at right angles to each other. At one end, each semicircular canal is dilated and that's where the osseous ampulla is. That's about twice the diameter of the canal.

Each of these canals is orthogonally oriented to each other, and the lateral canal doesn't sit horizontal, but rather 30 degrees off the horizontal plane. Each ampulla contains an ampulla crest, the crista ampullaris, which consists of a thick gelatinous cap called the cupula. Many hair cells make that system up.

When the head changes position or when you move your head, what happens is the endolymph in the canals lags behind and impinges on the cupula and bends the hair cells, which activates and stimulates the nerve. The stimulation of the hair cells sends the electrical code to the brain that an acceleration is happening.

The utricle, that's an otolith organ, along with the saccule, rests in the vestibule that has hair cells that aid in appreciating the latitudinal acceleration and uses the otolith as a mass, these calcium carbonate crystals that sit on the otolithic membrane.

Dr. Jason Barnes:

So with that in mind, can you tell us about the pathophysiology of BPPV? What's going on that causes this vertigo?

Dr. Devin McCaslin:

Yeah. So as I said in the beginning, what happens is patients will typically awake in the morning or when they get up, go to use the restroom with BPPV. And this is because they use... The idea is that the otoliths from the utricle are primarily what contributes to BPPV. And when they're laying supine, what happens the otolith is oriented now vertically, and the otoliths are hanging on the otolithic membrane, and when they detach they have a direct shot right into the posterior semicircular canal.

They don't usually realize that while they're sleeping, but when they sit up, up they go, the otoliths move, moves the endolymph inside the posterior semicircular canal and triggers the vestibulo-ocular reflex, which then is their perception of vertigo.

Dr. Jason Barnes:

You mentioned the posterior canal in this context. Is BPPV only caused by otoliths in the posterior canal or what's the breakdown there?

Dr. Devin McCaslin:

Yeah, no. The posterior canal is the most common in every study that you'll read, a canal that's affected by BPPV. Again, it's because of that orientation with the utricle and the posterior semicircular canal when the patient is laying supine.

The horizontal canal comes next. That's the second most common. By far, the rarest is anterior canal superior BPPV.

Dr. Jason Barnes:

Dr. Carlson, anything to add about pathophysiology?

Dr. Matthew Carlson:

We always talk about the semicircular canals as detecting angular acceleration, but if you're moving at a constant speed you no longer detect it anymore. So it has to... Just like we would think back to physics, it's the accelerating portion that's detected and caught in the deflection.

Secondly, the detection of the utricle and the saccule and how they detect acceleration. So just remember that the saccule detects vertical acceleration and the utricle detects horizontal acceleration. That's the only thing I might add to that.

Dr. Jason Barnes:

One question we'd like to ask Dr. McCaslin is about the natural history of a disease. What happens if someone has BPPV and this just goes untreated, we don't do anything about it?

Dr. Devin McCaslin:

Oftentimes, you'll see it resolve on its own, but in other cases, it won't. And so what we will see is patients will continually complain regarding the positional vertigo. What'll happen is it can even become more severe as more otoconia become dislodged. They're very sticky. And so what the happens is the mass will become bigger and bigger and it can become more severe over time.

What you'll often see patients do is make accommodations where they will sleep in a recliner, they won't bend over. I mean, they will completely revise their lifestyle based on the idea that this head position causes them to be dizzy.

Dr. Jason Barnes:

Dr. Carlson, when we think about differential diagnosis, what else is on your list and what are you looking for in clinic?

Dr. Matthew Carlson:

That's a really critical question, because you want to most importantly distinguish it from more sinister things, things you don't want to miss. But just to talk about this broadly, when a person comes in with dizziness, whether it be BPPV or something else, I think the first questions you have to ask are what are the other concurrent symptoms they're experiencing?

So by teasing out whether or not they have a fluctuating hearing loss or fullness of the ear or concomitant tinnitus really can help you hone that in. And so concurrent symptoms and concurrent otologic symptoms and even rarely concurrent cranial neuropathy is something you want to look for and ask about.

The second thing that I think is really critical to tease out and also very, very important is the duration of the symptom. And so, characteristically, BPPV, the vertiginous portion of where the... The portion where they feel the environment is turning around or moving relative to them should not last longer than a minute. And if you're very specific to ask about that, once it starts going over a minute, and particularly when it starts going over hours, you really have to change your differential diagnosis.

I think it's really critical that when you ask them about the symptom of dizziness or vertigo or imbalance or lightheadedness or whatever term you're using, that you really help you really understand

how they're defining that because we all use these terms differently. And so, specifically, you'll want to ask them, "Is the environment moving around you?"

It doesn't have to be spinning, but it should be that the environment is moving relative to you. You should differentiate that from inner or internal vertigo, where you feel you are moving in the environment. That's also separate from BPPV. So it should be the environment moving around you and it should last for less than a minute.

If a person has concomitant fluctuations in hearing, tinnitus in their ear with oral fullness in particular, and if it's lasting for hours on end, you really have to start thinking about Meniere's disease. We have a separate, excellent podcast on Meniere's disease. I encourage you to listen to it. But, in general, with Meniere's disease, you do want to have documented sensory neural hearing loss episodes as well to help confirm that.

Overlapping with Meniere's disease, as outlined in that other podcast, is a symptom of vestibular migraine and vestibular migraine typically isn't associated with hearing loss, but rarely you can, in some circumstances, have associated hearing loss and tinnitus and other symptoms. But, also, like migraine in general and like Meniere's disease, the symptoms should last hours or sometimes even longer.

Other conditions are more rare, so you could have a stroke condition, but to have an isolated positional vertigo from a stroke would be case reportable. It would not be a typical presentation.

You can have a vestibular neuritis or a labyrinthitis. And remember, the thing that distinguishes those two are the clinical finding of hearing loss with it. So a person who has labyrinthitis has to present with symptoms of vertigo and hearing loss, and that will typically last at least a day, but many times many days and sometimes as long as a week or more. That's a labyrinthitis.

A vestibular neuritis is distinguished from a labyrinthitis because you spare your hearing. It's only affecting the vestibular system and specifically the vestibular nerves. And so they won't have any fluctuations in their hearing or their hearing won't go down, but instead they'll have an isolated longer-term dizziness that will typically last hours, days, and sometimes even weeks.

I also want to distinguish it from one other thing, just the general symptom of vestibular hypofunction. So when I was a resident seeing a lot of patients with vestibular schwannomas, they would come back and they'd say, "I was dizzy when I turned my head quickly." And I'm like, "Oh, I'm a smart resident. That's BPPV." That's not BPPV. That's vestibular hypofunction.

So patients will... We can talk about this a little bit later too, but patients with unilateral vestibular hypofunction will often have a delay between when they turn their head and they move their eyes. That mismatch is perceived as very temporary dizziness lasting seconds. And so that's another one that I think you want to distinguish.

Dr. McCaslin, do you have anything else to add to that?

Dr. Devin McCaslin:

No, I think you got it right.

Dr. Jason Barnes:

So we've talked about presentation, pathophysiology, differential diagnosis, and I wanted to next move on to workup. Dr. McCaslin, I want to talk about diagnostic maneuvers, but before then, Dr. Carlson, any role at face value for imaging, audiogram or blood work?

Dr. Matthew Carlson:

I think it goes back to your differential diagnosis. BPPV is very common and the history is extremely suggestive if you really ask the history well. And we'll talk about in a minute, but also the examination findings, the diagnostic examination finding is so telling that in that setting with all those findings and with a good interview and a good examination, you can hone in on the diagnosis very effectively and you can save people from a lot of additional unnecessary testing and extensive workup.

So in that setting, a person that you really suspect BPPV without a unilateral asymmetrical hearing loss, there's no role for MRI in that specific situation. Of course, if you have a person who has atypical findings, there's always exceptions to the rule. But, in general, a person with a history strongly suggestive of BPPV doesn't warrant an MRI or CT scan at the beginning.

Dr. Jason Barnes:

Dr. McCaslin, this is kind of the meat and potatoes of what we're here to talk about. Can you tell us the diagnostic maneuvers that you do in clinic to diagnose BPPV?

Dr. Devin McCaslin:

Sure. Yeah. Well, the first, of course, the one most well known is of course, the Dix-Hallpike, named after Margaret Dix and Charles Hallpike in 1952, when they first described this maneuver in a hundred patients at Queen Square Hospital.

What this has to do is having the patient seated upright, turn the head 45 degrees towards the side that you're intending to test. This has the effect of aligning the posterior semicircular canal with the sagittal plane of the body. What you do is the examiner brings the patient back, supporting their neck and hyperextending their head about 20 or 30 degrees over the table.

One that is key to ask is does the person have any back problems before you start maneuvering them. You really need to clear that first. But then secondly is also the head extension off the table of about 20 or 30 degrees is extremely important. Then you need to make sure you query them about, so you don't have a VBI or something along those issues. But the extension is important in that some patients will provoke the BPPV, in others it will not. And so if you don't extend the head, it's possible, you'll miss it.

When you've got them in this position, of course, what's going to happen is the canaliths, or what we call the otoliths, have gotten loose. We now give them a new name called canaliths because they are otoliths in the canal.

What this is going to do is activate the VOR and generate a nystagmus that is associated with the posterior semicircular canal. You're going to have a latency in the beginning. So you're going to bring them back. You're not going to see it right away, but give it a few seconds because of the viscosity of the endolymph, and then what's going to happen is the nystagmus is going to emerge.

What you usually want to do before you bring the patient back and you really suspect they have BPPV is warn them about what's going to happen. You want to instruct them. The one thing they don't want to do, they've been avoiding this since they've been coming in. And so you want to make sure that you understand that you're going to hold onto them and nothing's... it's going to just be brief.

When you look at the nystagmus, the character of the nystagmus is for the posterior semicircular canal, it's an upbeating, meaning the fast phase is going up and there's a torsional component rotating the eye, the upper pole of the eye towards the dependent ear. So they're upbeating to the right for the right ear, upbeating left for the left ear after the nystagmus stops. And it will.

After about 30 seconds, depending on the size of the mass, what's going to happen is let them sit for a little bit and then you're going to want to bring them back up. What you need to do is prepare them that they're going to feel the vertigo when they sit back up. If you've got goggles on or you're able to see their eyes, what you're going to do is you're going to see a reversal in the nystagmus, which again confirms the BPPV.

So the patients should then have the maneuver repeated on the same side. Again, this is a response to fatigues. And so, again, the more you do it, it's going to become less and less intense. So that's really how we identify, doing it right and left for the posterior canal.

We also evaluate the lateral canal. For that one we use what's called the supine roll test. Here, what we do is we have the patients sit as they would be prepared for a caloric or in an exam chair have them laying back but supine with the head up 30 degrees. What we do is we start with the head in the center and roll the head 90 degrees to one side.

And so we let them sit. And, again, this being the 30 degrees puts the horizontal or lateral canal in the plane of stimulation. Have the patients be there for about 30, 40 seconds. If no nystagmus, you want to bring them back to center.

Now, it's important to let them rest in the center for about 30 or 40 seconds, even up to a minute, in order to let everything equilibrate, because what's going to happen is then you're going to go to the other side, same thing on the opposite side that you didn't test. You usually start with the side you suspect. And then, center the head back up again.

The key here of letting the patient rest in between turning the head to the right and to the left is that what you're going to do is the way that you're going to identify which lateral canal is impaired or affected is by the amplitude of the nystagmus. If you don't let it rest on each side, you're going to skew the results. And so you really need to let the system settle down between head turns.

The anterior canal, oftentimes you will see this in the Dix-Hallpike. You may see some downbeating nystagmus. We don't really have a good test in what... The test, if you do suspect anterior canal, what we do is what's called the deep supine head hanging, where we just actually bring the patient all the way back and drop their head straight back hyperextended. In that way, we can actually look at and evaluate the anterior canal.

Part of the reason I think we don't see a lot of anterior canal is because we actually don't test for it. And so you start doing that and you start to see a lot more of it.

One item that is worth considering is that during the Dix-Hallpike, if a patient does have lateral semicircular canal BPPV, there will be evidence of horizontal nystagmus during the Dix-Hallpike. You'll see it even more clearly when you do the supine roll test.

Also, when you're suspecting anterior canal BPPV and then see the downbeating, it's important that you rule out other issues that may be associated with downbeating nystagmus, like cerebellar impairments or other insidious diseases.

Dr. Jason Barnes:

So we've talked about workup, we talked about the Dix-Hallpike and the supine roll test for posterior and lateral canal. I next wanted to move on to treatment, which again is a very interesting thing for providers because you can potentially provide a lot of benefit very quickly. But before we go to canalith repositioning maneuvers, Dr. Carlson, I just wanted to ask, what's the role of anti-nausea or vestibular suppressant medications for folks with BPPV?

Dr. Matthew Carlson:

That's a great question. Vestibular suppressive medications, such as Robinul or meclizine or Ativan, will certainly curb the symptoms, but really the gold standard treatment is a canalith repositioning maneuver. And so there are situations where you might have a person who has a very robust response and gets really nauseous and sick, and you're about to send them for testing or canalith repositioning. You might prophylactically give them something like meclizine just to get them through the process. But, again, the treatment isn't just to prescribe one of these medications and have them go home. The treatment is to get them into canalith repositioning and moving on down that pathway.

Dr. Jason Barnes:

Dr. McCaslin, can you tell us about these canalith repositioning maneuvers?

Dr. Devin McCaslin:

The most common one, of course, is called the Epley maneuver. In 1992, Epley was really the one that moved from [inaudible 00:22:14] theory of all the otoliths being attached to the cupula, to the idea that we could actually move them to a neutral position where they don't really bother the patient anymore.

Now, we do a lot of things that are a little bit different than what Epley did. And so the more conventional terminology that we use is called canalith repositioning procedure. And, again, they go from otoliths and once they're in the canal, we give them the name canalith. But, essentially, you'll see it referred to as Epley in a lot of the literature.

The purpose of this is really to stimulate migration of the otoconia and the endolymph of the semicircular canal back into the utricle. The patient is moved rapidly into the Dix-Hallpike position that we talked about. It doesn't have to be real rapid. It used to be that we thought you had to throw the patient all the way back, but because of the viscosity in the lymph you don't have to throw them back, but you need to go with a brisk movement towards the side of the affected ear.

The patient's head is then kept in extension and rotated in the opposite direction 45 degrees towards the unaffected ear. Now, what's key is when we're treating is time. And so how we typically do it in our clinic is we will bring them back in the first position. We will wait until the symptoms stop. And then when the patient says, "I don't feel I'm dizzy anymore," we'll wait for another minute and a half after that to allow the otoliths to continue to move through the system.

Again, like I said, we turn the patient's head 45 degrees towards the unaffected ear. Again, we ask the patient when they no longer feel symptoms, wait for another minute and a half after. We then roll them onto the side, the third position. And this is key when we roll them onto the shoulder, push, pointing their nose towards the floor. If you can't get the nose directly towards the floor, that can be problematic with regards to success of treatment.

Again, symptomatic, wait about a minute and a half. And in keeping the head rotated, we are going to have them tuck their chin down and then seat them and then have them sit up.

Now, with the Lempert maneuver, which is the AKA barbecue roll, also known as the log roll, this is the treatment for the horizontal canal. In this case, what we've done is during the supine roll test, we've identified the side that is impaired. And the way we did that is that the side with the higher amplitude nystagmus is the effected side.

And so if we had a positive supine roll test, what we would've done... And it was on the right, we'd move the head to the right. We would get a large amplitude horizontal nystagmus, bring them back to center, bring them to the left. We would have a smaller amplitude horizontal nystagmus, geotropic, right beating right, left beating left. And then, what we would do is we've identified the right side.

If it's the right side, what we do in terms of the barbecue roll, the patient starts supine when we're going to treat them, head flexed 30 degrees, and what we do is rotate towards the unaffected ear. Typically, what we'll do is go to one side, again symptomatic, minute and a half. Roll them again, symptomatic and a half, and have them go all the way around, each position being held for about 60 seconds to a minute and a half.

Dr. Jason Barnes:

Dr. McCaslin, you mentioned the word geotropic. Can you discuss the concept of geotropic and ageotropic nystagmus and how that might apply?

Dr. Devin McCaslin:

Yeah. We primarily use the terms ageotropic and apogeotropic with regards to findings that we get during the supine roll test for lateral canal. Geotropic, of course, refers to the nystagmus beating towards the ground. So if your head is turned to the right, you're going to see right beating nystagmus, fast phase being towards the ground. Turn to the left, likewise, you're going to see left beating nystagmus in the fast phase towards the ground.

That form of nystagmus suggests that the canaliths in the lateral canal are in the posterior [inaudible 00:26:24]. When you see apogeotropic nystagmus during the supine roll test, that indicates that they're in the anterior in the lateral canal and require additional maneuvers in order to successfully treat that.

Dr. Jason Barnes:

Is there any role for what are known as habituation maneuvers in these patients?

Dr. Devin McCaslin:

There are. Typically, 80% to 90% of the time with a canalith repositioning procedure done correctly, we can successfully treat these patients. There are those that will have what is known as cupulolithiasis, in cases where the otoliths are actually attached to the cupula and the idea being like Cawthorne or Cooksey exercises or some [inaudible 00:27:04] exercises can be used to remediate the cupulolithiasis.

Dr. Jason Barnes:

Dr. Carlson, is there any role for surgical management of patients with BPPV?

Dr. Matthew Carlson:

That's a very good question and interesting question. Most cases of BPPV self-resolve or are responsive to canalith positioning. And so if you really consider the people with truly refractory disease that have to go on to have additional surgical therapy, you're talking about 1%, 2%, or maybe even less than that, of the total population. So it's rare that you need to resort to that. But there are some people that have multiple refractory BPPV, and there are several surgical procedures that have been used over the years with varying levels of success.

The first primary surgical procedure that was described for BPPV, particularly that involving the posterior semicircular canal, is a singular neurectomy. That was popularized by Gacek several decades ago, and it was largely a transcanal procedure where you would drill in the region of the round window and try to ablate the singular nerve.

The singular nerve, as we can recall on imaging, travels through the singular canal, and it's a branch of the inferior vestibular nerve and it selectively innervates the posterior semicircular canal.

That was the first real description for a surgical treatment. More commonly, now, if it is being performed, it's a posterior canal plugging. This was popularized, and I think first described, by Dr. Lorne Parnes, where you would fenestrate the posterior semicircular canal and try to leave the endosteum intact where you're not actually seeing perilymph leak. And then you can occlude that with many different types of material, just as you might with superior canal dehiscence. But I think classically, it was described by obliterating it with bone pate, but any of those mediums could be used.

Essentially, you're blocking the canal, so you're reducing the risk of the movement of the otoconia and the deflection of perilymphatic current in that area to reduce your symptoms. That is met with quite good success with people with very refractory disease. But, again, it's very uncommon that you actually have to perform the procedure.

Most [inaudible 00:29:20] would say that your risk of developing a significant sensory neural hearing loss is less than 10% or 15% in the success rate of the procedure at mitigating recurrent BPPV is over 90%.

The last surgical procedure that has been described, but really is... I would say... I can't even think of a time I've heard of somebody using it for it, is a vestibular nerve section. Again, these are procedures that are labyrinth or labyrinthine sparing, so you're not actually risking sensory neural hearing loss significantly. But with a vestibular nerve section, you can perform that through a retrosigmoid craniotomy or a middle fossa craniotomy.

As you might recall, anatomically, the farther you get away from the root exit zone of the eighth nerve, the more those nerves divide, so you can more selectively ablate or divide your inferior and superior vestibular nerves. Again, that is a little bit extreme and most people would resort to these other surgical methods, if a surgical method was even required to begin with.

Dr. Jason Barnes:

Dr. McCaslin, moving on to outcomes and expectations, what are the success rates of these canalith repositioning maneuvers and how often do you see a recurrence?

Dr. Devin McCaslin:

Yeah. Well, it's probably the most successful thing we do in dizziness. About 80% to 90% of people have complete resolution.

I think it's very important to prepare the patient for the symptoms that may occur after treatment. Sometimes what they will do is complain of lightheadedness. They'll continue to experience that for up to a couple of weeks.

One of the most common symptoms that patients report, well, they feel like they're walking on the shag carpeting. I've seen that last up to a week. I think the longest has been a week. But, eventually, then that resolves.

Then there's a recurrence. And so about 30% of patients will have a recurrence. I think it's up for every clinician to really determine, is this someone that I can teach to do the maneuvers themselves or is it not? And so in elderly patients that may have risk of injury or a fall. I prefer to do it in the clinic.

If you've got a group of patient that can certainly understand it or work with their spouse to treat it at home because of the reoccurrence, then that certainly is well. I think it's a case-by-case basis.

Dr. Jason Barnes:

In terms of followup, how do you follow up with these patients? Do you give them any instructions for things they should do going home directly after a canalith repositioning maneuver?

Dr. Devin McCaslin:

Yeah. To the first question, do I have them follow up? I do. I actually ask them to give me a phone call and rate on a percentage how much better are they. A hundred percent resolved, not all the way to not resolved at all. And then in that we'll either work through it with them at home or we'll have them come back into the office.

With regards to recommendations following the treatment, this is interesting because there's a whole body of literature that says the people, when we used to send them home, we used to put them in cervical collars, tell them to sleep in a recliner. We did all of this. And then, as studies came out, it found that that really didn't make a significant difference.

What I will say to that is that I have them avoid any dramatic up or down movements of the head, bending over, doing yoga, anything like that that would put them in an unnatural position for at least two days. I've seen it reoccur within two days. Doing some of these procedures and maneuvers like yoga is a big one that came out. But after two days they're welcome to do anything that they feel comfortable with. But yeah, and I have them... Usually, recommend just sleep with a pillow, two pillows just for two nights and avoid any of those.

Dr. Jason Barnes:

Great. Well, I think this has been an awesome discussion of BPPV. Dr. McCaslin and Dr. Carlson, thanks again so much for being here.

Before I move on to the summary, is there anything you'd like to add?

Dr. Matthew Carlson:

Dr. Barnes, this is wonderful. I don't have anything to add. Thank you for organizing this.

Dr. Devin McCaslin:

No, thank you. I just think it's great that we organized this. Everybody's going to see some of these patients at some point in their life, and so it's good to be sophisticated and understand what it is and how to treat it.

Dr. Jason Barnes:

Well, moving on to our summary, BPPV is the most common peripheral vestibular disorder resulting in vertigo and patients present with vertigo and nystagmus with characteristic movements, such as rising out of bed. It's caused by otoconia that are dislodged from the utricle and sit in one of the semicircular canals, free floating in the endolymph.

The vast majority of BPPV is due to otoconia, which become canaliths in the posterior canal, so the horizontal and anterior canals can be affected. Diagnosis is made using the Dix-Hallpike maneuver for the posterior canal when it's affected and the supine log roll test when the horizontal canal is involved.

Treatment can include medication for symptom management, but it's almost exclusively treated by canalith repositioning maneuvers. As a reminder, the Epley maneuver is used to displace otoconia in the posterior canal, while the Lempert maneuver is used to displace otoconia in the horizontal canal.

Success rates are close to 80% to 90% and the disease is otherwise self-limiting for most patients. Recurrence rates of note can be as high as 30%.

I'll now move on to the question asking portion of our time together. As a reminder, I'll ask a question, wait a few seconds, and then give the answer.

The first question is, describe the anatomy of the semicircular canals.

There are three semicircular canals that are orthogonally oriented, which means oriented at 90 degrees to each other. There's the posterior canal, the horizontal or lateral canal, and the superior semicircular canal. They're filled with endolymph and at the end of each semicircular canal rests an ampulla. The posterior and superior circular canals come together to form a common [inaudible 00:35:28].

The utricle and the saccule are located in the vestibule and the horizontal canal is at a 30-degree plane from the ground or the horizon.

Next question. What is the most common cause of BPPV?

The most common cause of BPPV is idiopathic, and this is a displacement of an otolith, which becomes a canalith when it falls into the posterior semicircular canal.

Next question. Describe the most common diagnostic maneuver for BPPV.

This is the Dix-Hallpike maneuver, and it's used to identify canaliths in the posterior semicircular canal. The patient is seated upright with the head turned 45 degrees toward the tested side. This aligns the posterior semicircular canal with the sagittal plane.

The examiner manually supports the patient to quickly recline and become supine with the head hyperextended over the end of the table. The diagnosis is made with nystagmus and reproduction of symptoms.

For our final question, describe the canalith repositioning maneuver or the Epley maneuver.

Again, the Epley maneuver, or the specific canalith repositioning maneuver, is used to displace canaliths from the posterior semicircular canal. The patient is moved into the Dix-Hallpike maneuver, as previously described, towards the direction of the affected ear. The patient's head is kept in extension and in this position until the symptoms of dizziness or vertigo subside, and then the patient is rotated in the opposite direction, 45 degrees, again, waiting for symptoms to subside for over a minute.

The patient is then rolled into the side lying position toward the unaffected ear, continued with the head turned 45 degrees and with the nose pointed towards the floor. As the patient is sat up, they're asked to tuck their chin towards the shoulder as they're slowly brought into the seated position.

That wraps up our episode. Thanks again for listening, and we'll see you next time.