Benign paroxysmal positional vertigo

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§1: BPPV overview

§1.1: What is BPPV?

Benign paroxysmal positional vertigo, hereafter abbreviated BPPV, is an inner ear disorder that usually causes positionally-triggered episodes of dizziness/imbalance. It is among the most frequently encountered diseases in clinics specializing in evaluating dizzy patients.

In 1920 Robert Bárány (Bárány 1920) described a case of a young woman who had been suffering from vertiginous episodes triggered by lying on her right side. Numerous similar cases were described subsequently, and in 1952 Margaret Ruth Dix and Charles Skinner Hallpike (Dix and Hallpike 1952, Dix and Hallpike 1952) described their own cases of "positional nystagmus" (the term nystagmus refers to "jumping of the eyes"), noting that symptoms (and simultaneous nystagmus) were induced, "by a critical position of the head in space."

§1.2: When should I suspect that I may have BPPV?

In the majority of cases, BPPV presents as positionally-triggered episodes of disequilibrium that patients usually experience as a spinning sensation (sometimes associated with nausea) that can last seconds to minutes, though can be followed by many minutes of unsteadiness. The symptoms can be re-triggered when the patient assumes the same position.

In some instances (usually lateral semicircular canal BPPV, which will be discussed in greater detail below) the sensation of disequilibrium is more vague, and may be experienced as a "rocking," "tilting" or "swaying."

If you are experiencing symptoms such as these, then you may have BPPV. However, in order to be confident about the diagnosis, a clinician needs to examine your eye movements, as will be discussed below.

§1.3: What is the cause of BPPV?

BPPV is the result of otoliths ("crystals" in the inner ear) coming loose from their normal place in the inner ear (the saccule and the utricle) and entering an incorrect place (the semicircular canals) in the inner ear. The movement of the otoliths results in stimulation of motion sensors, which in turn can provoke a sensation of movement.

Notice that in order for this disease to manifest, two distinct events need to occur. First, the otoliths need to break loose; this appears to occur spontaneously, and there is very little you can do on your own physically to break the otoliths loose. Second, the otoliths need to get into

an incorrect part of the ear; this simply happens when the head is put in certain orientations with respect to gravity.

Thus, when a person goes to the hairdresser, extends her neck over the edge of the washbasin, becomes dizzy and then gets diagnosed with BPPV, it is easy to fall prey to the illusion that "bending my head backwards broke the crystals loose and caused BPPV," but that is probably not true; it is far more likely that the otoliths *were already loose* before she went to the hairdresser, and once they were loose, inclining the head backwards permitted those (already loose) otoliths to enter one of the semicircular canals, thereby provoking symptoms.

Much of the information on the internet gives the erroneous impression that BPPV is a matter of there being "one ball bearing that is loose," but actually this is more of a "grains of sand" issue — hence BPPV is a matter of degree, and can be present to a greater or lesser extent.

§1.4: Epidemiology of BPPV

BPPV is the most common cause of dizziness over the lifespan (Nedzelski, Barber et al. 1986, Parnes, Agrawal et al. 2003, Neuhauser 2007). It is less common before the age of about 40 years, and then increases exponentially in frequency (von Brevern, Radtke et al. 2007), as shown in the **Figure** below.

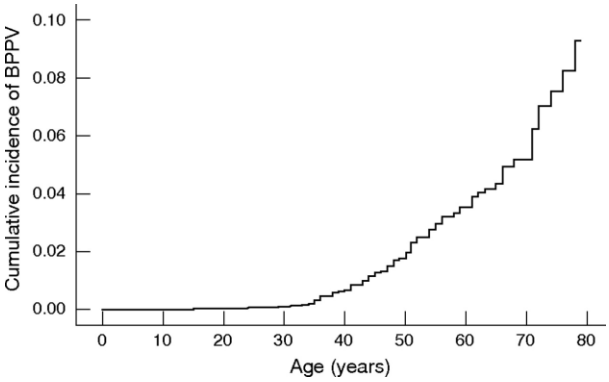

Figure: Cumulative incidence of BPPV by age (von Brevern, Radtke et al. 2007).

The recognition of this relationship led to the idea that BPPV is somehow due to "wear and tear" on the inner ear. Electron micrographs actually support this idea. The **Figure** below shows otoconia in a normal, healthy individual (Thomas, Rosseeva et al. 2012).

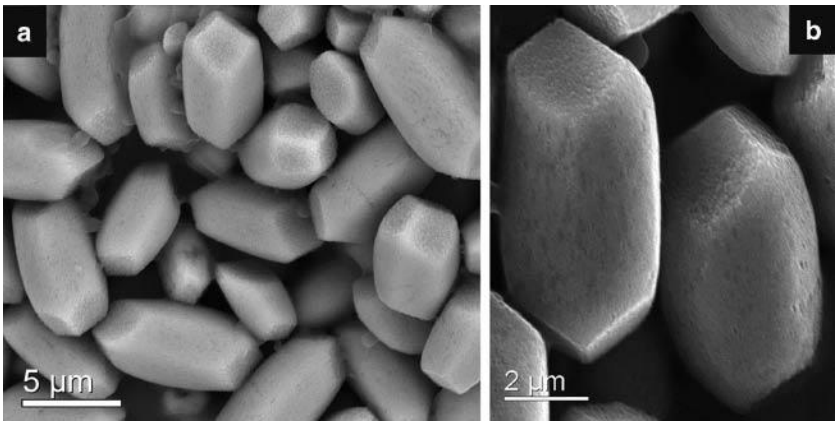

Figure: Electron micrograph of healthy human otoconia (Thomas, Rosseeva et al. 2012).

As a person ages, even if she is otherwise healthy, the otoconia begin to fragment, as shown in the **Figure** below (Walther and Westhofen 2007).

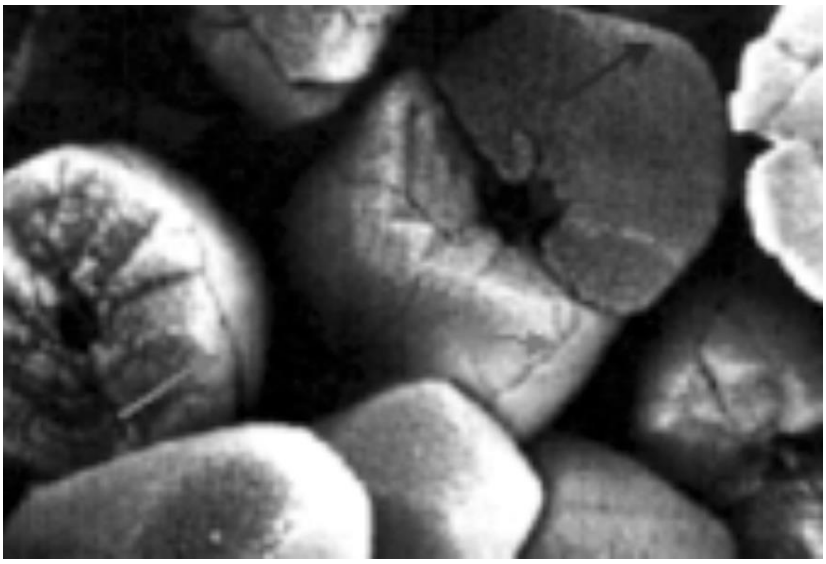

Figure: Electron micrograph of otoconia in an older individual (Walther and Westhofen 2007).

Given this deterioration of otoliths, it is not hard to imagine fragments of otoliths breaking loose.

§1.5: Risk factors for BPPV

Most cases of BPPV appear to occur without any discernible trigger — specifically, there is usually nothing in the patient's history that would explain why the otoliths "broke loose."

Probably the single most important risk factor for BPPV is a history of a prior episode of BPPV. This is because BPPV has a tendency to recur (see below).

Since BPPV occurs more frequently with increasing age (see above), older age is also considered a "risk factor" for BPPV.

There are a few other factors that appear to increase the risk of BPPV, though the literature about these is mixed. A history of migraine appears to increase the risk of BPPV (Ishiyama, Jacobson et al. 2000, Kim, Hong et al. 2019). Low bone density (Jeong, Choi et al. 2009) and vitamin D deficiency (Chen, Zhao et al. 2020) may increase the risk of BPPV. Finally, the risk of BPPV appears to be increased when a patient has a history of other ear diseases (Lee, Ban et al. 2010), such as vestibular neuritis (Balatsouras, Koukoutsis et al. 2014), labyrinthitis (Baloh, Honrubia et al. 1987) and Ménière's disease (Kutlubaev, Xu et al. 2019).

The literature provides conflicting data on the relationship between head trauma and BPPV. For example, some literature suggests that BPPV following head trauma is less responsive to treatment (Babac, Djeric et al. 2014, Pisani, Mazzone et al. 2015, Balatsouras, Koukoutsis et al. 2017); in contrast, large case series (Luryi, LaRouere et al. 2019) and meta-analyses (Aron, Lea et al. 2015) concluded that the available evidence does *not* show any difference in treatment responsiveness/outcomes between traumatic and non-traumatic BPPV. There is modest literature suggesting that bilateral posterior semicircular canal involvement (Katsarkas 1999) or (unilateral) anterior semicircular canal involvement (Jackson, Morgan et al. 2007, Dlugaiczyk, Siebert et al. 2011) are more common following head trauma. From a practical medical perspective these considerations are irrelevant in the sense that, if a patient has manifest BPPV on examination, treatment should be considered, irrespective of prior history.

§2: Diagnosing BPPV

§2.1: How is BPPV diagnosed?

BPPV can be suspected from the clinical history, but can only be confirmed by observation of a pattern of eye movements (von Brevern, Bertholon et al. 2015). The specific pattern of eye movements depends on which semicircular canal, or combination of the six semicircular canals, is affected by BPPV (see **Figure**, below). The eye movements consist of various combinations of nystagmus; nystagmus refers to a repetitive pattern in which there is a slow eye movement in a given direction, followed by a quick eye movement in the opposite direction, and then the cycle repeats.

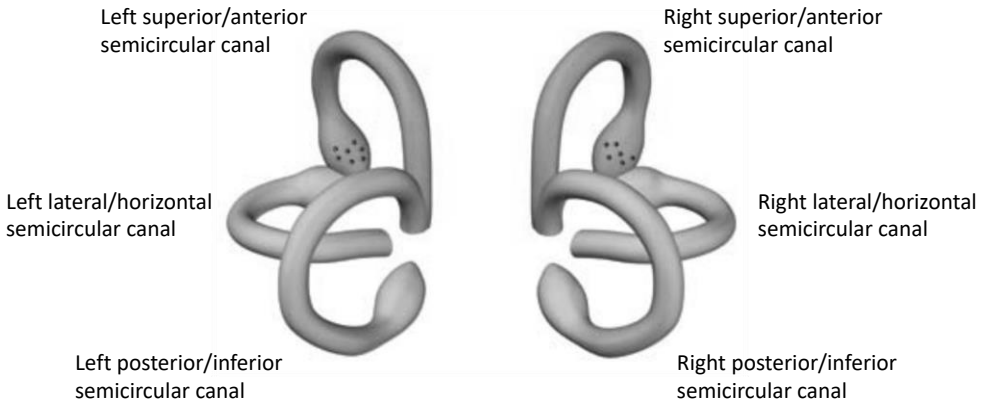

Figure: The position and orientation of the six semicircular canals, as viewed from the back of the head. Adapted from Helminski (Helminski 2012).

§2.2: Are there tests for BPPV?

Observation of eye movements IS THE TEST for confirming BPPV (von Brevern, Bertholon et al. 2015, Bhattacharyya, Gubbels et al. 2017, von Brevern, Bertholon et al. 2017).

In a patient who is already known to have BPPV, there may be some circumstances in which studying their inner ear function is appropriate. If a patient appears not to be responding well to appropriately targeted treatment, many clinicians may consider checking tests of inner ear function in order to assess whether the ears have been damaged by some other disease process, which in turn might have rendered the patient more vulnerable to BPPV. For example, there is emerging evidence that assessment of vestibular evoked myogenic potentials may be useful in this regard (Hong, Park et al. 2008, Yang, Kim et al. 2008, Korres, Gkoritsa et al. 2011, Lee, Park et al. 2013, Yetiser, Ince et al. 2014, Hoseinabadi, Pourbakht et al. 2016, Singh and Apeksha 2016, Xu, Liang et al. 2016, Oya, Imai et al. 2019, Scarpa, Gioacchini et al. 2019).

§2.3: Someone examined me and did not "see" BPPV, so do I really have it?

There are several reasons why an individual who truly has BPPV may not exhibit the associated eye movements on a particular examination.

The first reason why the diagnostic eye movements might not be seen in a person who truly has BPPV has to do with the behavior of the disease itself. BPPV is an *episodic* problem, which is why it has the word "paroxysmal" in its title. Because of this pattern, the diagnostic eye movements will not necessarily be present every time an examiner looks for them. As an example, the Dix-Hallpike maneuver is considered the "gold standard" for the diagnosis of posterior semicircular canal BPPV (Bhattacharyya, Gubbels et al. 2017); however, the maneuver has a reported specificity of 71% (Lopez-Escamez, Lopez-Nevot et al. 2000) and negative predictive value of 52% (Hanley and O'Dowd 2002), meaning that a "negative" Dix-Hallpike maneuver does not entirely exclude the presence of posterior canal BPPV; for this reason, if a patient's history is suggestive of BPPV yet her examination does not "show it," it is reasonable

to re-examine the patient at some interval. That being said, the more frequently one looks for the abnormal eye movements and fails to detect them, the more skeptical one becomes about the diagnosis.

The second reason why the diagnostic eye movements might not be seen in a person who truly has BPPV is that the examiner may be relying on an insufficiently sensitive method of detection; specifically, the examiner is not using infrared video Frenzel oculography (Frenzel 1956, Levo, Aalto et al. 2004). For example, if a clinician is only observing a patient's eye movements "face-to-face," or only using Fresnel lenses (Fresnel 1822, Strupp, Fischer et al. 2014), then some degree of vision is available to the patient; when vision is available, the brain is hard-wired to make you visually fixate on targets, and such fixation can suppress a variety of eye movement abnormalities (Sokolovski 1966, Hart 1967, Molnar and Torok 1974, Baloh, Solingen et al. 1977, Karlsen, Goetzinger et al. 1980, Chambers and Gresty 1982, Katsarkas and Kirkham 1982, Gauthier and Vercher 1990, Gizzi and Harper 2003, Hirvonen, Juhola et al. 2012), including many of the eye movement abnormalities that are diagnostically helpful.

The third reason why the diagnostic eye movements might not be seen in a person who truly has BPPV is that the abnormal eye movements (which would otherwise be present) can be suppressed by certain medications, such as vestibular suppressants (e.g., meclizine, Dramamine), or benzodiazepines.

It should also be noted that if a patient suspected of having BPPV has repeatedly normal exams (despite an adequate examination technique, and despite being off vestibular suppressant medications), then the suspected diagnosis may be incorrect. We discuss this further below.

§2.4: If I have BPPV, should I get brain imaging?

If a person's clinical history and eye movements are compatible with BPPV, and if her physical examination is otherwise normal, then brain imaging is usually unnecessary (Bhattacharyya, Gubbels et al. 2017).

§3: Subtypes of BPPV

§3.1: "Different kinds" of BPPV

Although we have been speaking of a "diagnosis of BPPV," there are several subtypes of this disease, depending on the specific semicircular canal or combination of semicircular canals involved.

§3.2: BPPV involving the posterior semicircular canal

Posterior semicircular canal BPPV is by far the most common type, accounting for over 80% of cases.

Posterior semicircular canal BPPV can be triggered in a variety of positions, some of the more common of which are given epithets in the literature; examples include the "top shelf phenomenon" (when a patient inclines the head backwards while reaching for an object above her head) (Squires, Weidman et al. 2004) and the "under the sink phenomenon" (when an individual leans forward and turns the head horizontally while trying to get the detergent out from under the sink). Other common scenarios include lying on one side, or inclining the head backwards (such as in the dentist's chair, or at the hairdresser's).

The diagnosis of posterior semicircular canal BPPV is secured by observing a specific pattern of eye movements when the patient is in a specific position. The diagnostic position is called the Dix-Hallpike maneuver (Dix and Hallpike 1952, Dix and Hallpike 1952, von Brevern, Bertholon et al. 2015). The observed eye movement is a pattern of nystagmus consisting of a combination of nystagmus in which the fast phase of the eye movement is ipsiversive (beating towards the affected ear), ipsitorsional (the top pole of the eye rotates towards the affected ear) and up beat (towards the top of the patient's head).

In cases where the posterior semicircular canal is involved on only one side, treatment typically begins with the Epley maneuver (or a variant called the canalith repositioning maneuver) on that side (Epley 1980). Multiple trials have established the efficacy of this treatment (Fife, Iverson et al. 2008, Hilton and Pinder 2014, Bhattacharyya, Gubbels et al. 2017). If an appropriate trial of the Epley maneuver fails, then some physical therapists and clinicians will consider other maneuvers, such as the Semont maneuver (Semont, Freyss et al. 1988), the Foster maneuver (Foster, Ponnapan et al. 2012) or the Gans maneuver (Roberts, Gans et al. 2006). The efficacy of these maneuvers has been assessed in meta-analyses (Hilton and Pinder 2014). All of these maneuvers are intended to treat posterior semicircular canal BPPV on one side when that side is known to be affected. Note that one cannot "alternate" treatment sides, because treating a given side will "undo" whatever had been accomplished in treating the opposite side. In other words, if an individual is thought to have posterior semicircular canal BPPV on one side but the side is unknown, one cannot "just try treating both sides." Or, if an individual is definitely known to have bilateral posterior semicircular canal involvement, one cannot alternate treatment sides.

In cases where the posterior semicircular canal is known to be involved on both sides, treatment sometimes begins with the Brandt-Daroff maneuver (Brandt and Daroff 1980).

§3.3: BPPV involving the lateral semicircular canal

Lateral semicircular canal BPPV is less common than posterior canal BPPV. Estimates in the literature suggest that it accounts for approximately 5% - 10% of cases of BPPV.

The diagnosis of lateral semicircular canal BPPV is secured by observing a specific pattern of eye movements when the patient is in several different positions. The series of positions is sometimes referred to as the "supine roll test," while some examiners use eponymous designations (Bhattacharyya, Gubbels et al. 2017), after the various investigators who provided

early descriptions of the phenomenon (e.g., McClure (McClure 1985), Pagnini (Pagnini, Nuti et al. 1989)). Essentially, when the patient's head is directly on its side (i.e., with the sagittal plane oriented horizontally, in parallel with the surface of the earth), nystagmus that is purely horizontal (with respect to the patient's head) emerges; the nystagmus is either "geotropic" (meaning that the fast phase beats towards the ground) or "apogeotropic" (meaning that the fast phase beats away from the ground). These patterns are sometimes referred to as "direction changing positional nystagmus" (McClure 1985, Baloh, Jacobson et al. 1993), because the direction of the nystagmus depends on the position of the patient's head with respect to gravity.

Because both lateral semicircular canals lie in the same plane, either side can produce the same pattern of eye movement abnormalities. Thus, physical examination alone may be able to diagnose lateral semicircular canal BPPV, but may not be able to determine the affected side. Some studies propose methods for determining the affected side (Choung, Shin et al. 2006, Choi, Oh et al. 2020), such as the "bow and lean test" (Choung, Shin et al. 2006), but practically such determination is often not possible. Consequently, when a physical therapist is confronted with this ambiguity, he or she may simply choose one side and start treating it; if after a few sessions the patient improves then it is assumed that the originally chosen side was correct; in contrast, if after a few sessions a patient fails to improve, then the therapist may switch to the other side, at which point the patient should begin to improve. One *cannot* "alternate" treatment sides, because treating a given side will "undo" whatever had been accomplished by treating the opposite side.

The first maneuver shown to treat lateral semicircular canal BPPV was described by Thomas Lempert (Lempert 1994) as the "barbecue roll;" some clinicians also refer to this as the "log roll." Other maneuvers that have been less thoroughly studied include the Gufoni maneuver (Gufoni, Mastrosimone et al. 1998) and the Appiani maneuver (Ciniglio Appiani, Catania et al. 2005).

As noted earlier, lateral semicircular canal BPPV can cause either geotropic or apogeotropic direction changing positional nystagmus. Some studies report that both variants respond similarly to the Lempert maneuver and the Gufoni maneuver (Fu, Han et al. 2020, Fu, Han et al. 2020). In contrast, some studies suggest that the geotropic and apogeotropic variants of lateral semicircular canal BPPV respond differently to different maneuvers; for instance, one randomized trial reported the Lempert maneuver and Gufoni maneuver to be effective specifically for geotropic lateral semicircular canal BPPV (Kim, Oh et al. 2012), while the original paper about the Appiani maneuver described it as efficacious specifically for apogeotropic lateral semicircular canal BPPV (Ciniglio Appiani, Catania et al. 2005).

§3.4: BPPV involving the anterior semicircular canal

Anterior semicircular canal BPPV is the rarest form of BPPV. This rarity is plausibly due to the fact that the anterior semicircular canal is the highest of the three canals, and in the course of regular activities, gravity is likely to cause loose otoliths to exit that higher canal and instead

enter one of the lower canals (posterior or lateral). Some literature suggests that anterior semicircular canal BPPV is more common after head trauma (Jackson, Morgan et al. 2007, Dlugaiczyk, Siebert et al. 2011).

Anterior semicircular canal BPPV is diagnosed using the Dix-Hallpike maneuver (like posterior semicircular canal BPPV), but the pattern of observed eye movements is different than that found with posterior semicircular canal BPPV. Specifically, in anterior semicircular canal BPPV the pattern of observed eye movements consists of a combination of nystagmus in which the fast phase is contraversive (beating towards the unaffected ear), contratorsional (the top pole of the eye rotates towards the unaffected ear) and down beat (towards the patient's feet).

Because anterior semicircular canal BPPV is relatively rare, it has also been more difficult to study. Treatment maneuvers described include the reverse Epley maneuver (Honrubia, Baloh et al. 1999), the Semont maneuver (Semont, Freyss et al. 1988), the Casani maneuver (Casani, Cerchiai et al. 2011), the Kim maneuver (Kim, Shin et al. 2005) and the Yacovino maneuver (Yacovino, Hain et al. 2009).

§4: Treatment for BPPV

§4.1: Do I need to treat BPPV?

The management of BPPV is "symptom-driven," meaning that if symptoms are infrequent and mild, some patients reasonably elect to refrain from treatment, since spontaneous resolution (i.e., without treatment) can occur. If one initially elects to refrain from treatment but then the symptoms become more aggressive, then treatment can be initiated; it is never "too late" to treat BPPV.

§4.2: Treatment for BPPV: Physical therapy

Appropriately targeted vestibular rehabilitation therapy is firmly established as the standard of care for BPPV (Bhattacharyya, Gubbels et al. 2017). The particular series of physical therapy maneuvers is chosen based on which semicircular canal, or combination of semicircular canals, is involved.

Note that the physical therapy exercises do not "put the crystals back in place." Rather, by moving the head through certain positions, these maneuvers exploit gravity to "coax" the loose otoliths out of the semicircular canals, and into parts of the ear where they have a higher chance of getting resorbed.

Note that even when BPPV has been correctly diagnosed, and even when the appropriately targeted treatment maneuver has been initiated, the response to therapy may be somewhat uneven; when observing your symptoms day-to-day, it may appear that on a given day you feel worse than the previous day; however, when observing symptoms over a longer span of time (several days in a row), the *overall trajectory* should be one of improvement. If the overall

trajectory of symptoms is one of improvement (no matter how slow it seems), then you are likely pursuing the correct treatment.

In contrast, if while performing a given exercise you find that symptoms are growing consistently worse with each day of treatment, for several days in a row, then this raises the possibility that you have suffered a "canal conversion" (in which the otoliths have exited one semicircular canal but entered a different semicircular canal; see the section below entitled, "Why is my treatment for BPPV unsuccessful?"). In this circumstance you should refrain from further self-treatment, and return to your physical therapist or physician so that they can examine your eye movements.

§4.3: Why do the physical therapy treatment maneuvers need to be repeated?

The various exercises do help maneuver the loose otoliths into a part of the ear where they have a better chance of getting resorbed, but the exercises *do not cause resorption instantaneously,* they *do not immediately dissolve the crystals,* and they *do not "glue the crystals back into place."* The crystals are *still there,* and while a few of them will get resorbed shortly after the exercises are performed, the remaining otoliths are *still loose,* and in the course of daily activities and head movements, some of those loose crystals will get back into places where they don't belong (the semicircular canals), where they will still be capable of provoking symptoms.

Therefore, where patients get the most "mileage" out of the treatment exercises is by repeating them over time. Thus, doing the appropriately targeted exercises 30 times in one night is actually *less effective* than doing those exercises 3 times per night over 10 days.

§4.4: Do I need to sleep upright after doing the treatment maneuvers?

In posterior semicircular canal BPPV, lying supine and rolling over in bed often provoke symptoms, so it is reasonable to inquire whether remaining upright, or at least remaining in a partially reclined position (between completely upright and completely supine) might help. The short answer is that remaining upright may avert symptom provocation, but *merely remaining upright does not in itself treat BPPV*.

A related question is whether sleeping upright improves the efficacy of the various treatment maneuvers for BPPV. Most literature on this topic concludes that sleeping upright *does not improve treatment outcomes* for BPPV (Nuti, Nati et al. 2000, Gordon and Gadoth 2004, Moon, Bae et al. 2005, Roberts, Gans et al. 2005, Simoceli, Bittar et al. 2005, Casqueiro, Ayala et al. 2008, Fyrmpas, Rachovitsas et al. 2009, Devaiah and Andreoli 2010, De Stefano, Dispenza et al. 2011, Mostafa, Youssef et al. 2013). A few studies suggest that sleeping upright may be helpful (Cakir, Ercan et al. 2006, McGinnis, Nebbia et al. 2009), while other studies simply conclude that further research is needed on this topic (Papacharalampous, Vlastarakos et al. 2012).

§4.5: Treatment for BPPV: Medication

Medications such as vestibular suppressants (meclizine, Dramamine) and benzodiazepines (clonazepam, diazepam) are sometimes prescribed, but their role in treating BPPV is very limited, and guidelines generally recommend *against* their use (Bhattacharyya, Gubbels et al. 2017).

It must be kept in mind that these medications may succeed in "dampening" the sensation of dizziness, but they do not actually treat the underlying cause of BPPV — specifically, they do not dissolve otoliths or reattach them in their proper place.

These medications also pose several problems. The first is that by suppressing symptoms, it makes it more difficult to assess whether the disease causing those symptoms is actually improving — in other words, it may be hard to tell whether a given treatment maneuver is effective. The second problem is that these medications also suppress eye movements, thereby making it difficult to verify the diagnosis and monitor treatment responsiveness. The third problem is that these medications tend to be soporific (cause sleepiness).

§4.6: Treatment for BPPV: Surgery

A variety of essentially destructive procedures have been attempted by otolaryngologists in the management of BPPV.

Of the surgical procedures attempted for management of BPPV the most common is "canal plugging," first reported by Lorne Parnes and Joseph McClure (Parnes and McClure 1990). In this procedure the surgeon fills the semicircular canal affected by BPPV with either bone pâté or a synthetic substance. The logic of this procedure is that plugging the affected canal "silences" it, because otoliths can no longer enter it. Subsequent studies (Shaia, Zappia et al. 2006) reported impressive success with this procedure, but it was soon realized (Kisilevsky, Bailie et al. 2009) that potential complications of the procedure included hearing loss, vestibular deficits on objective testing, and even continued dizziness. The continued dizziness was particularly concerning, since that was the symptom the procedure was intended to treat. Among other problems, it was eventually recognized (Luryi, Schutt et al. 2018) that although plugging one canal might "silence" that canal, there remain five other semicircular canals that are still susceptible to developing BPPV.

A less commonly attempted surgical procedure for management of BPPV is cutting the nerve that transmits balance signals from the ear to the brain (which procedure is called "vestibular neurectomy") or, if possible, cutting just the part of that nerve that transmits balance signals from the affected canal (which procedure is called "singular neurectomy" (Gacek 1978)). While the latter procedure (singular neurectomy) appeared promising and minimally destructive, it also turned out to be technically challenging, and few surgeons attempted it.

Taking these factors into consideration, we generally do not recommend surgery as a treatment for patients with BPPV.

§4.7: Why is my treatment for BPPV unsuccessful?

There are several reasons why treatment for BPPV may be unsuccessful.

The first is that the specific case of BPPV may be very aggressive. This likely corresponds to a situation in which a very large number of otoliths have broken loose, thereby requiring more extended treatment before resorption is complete.

The second reason by treatment of BPPV may be unsuccessful is what is termed a "canal conversion" (Foster, Zaccaro et al. 2012, Lin, Basura et al. 2012, Park, Kim et al. 2013, Anagnostou, Stamboulis et al. 2014, Babic, Jesic et al. 2014, Dispenza, A et al. 2015, Lee, Lee et al. 2019). Since all parts of the inner ear are connected, there is nothing to prevent otoliths from exiting one incorrect place and migrating to a different incorrect place — in other words, leaving one semicircular canal and entering another one. This is a problem because the semicircular canals are arranged in different orientations, so a treatment maneuver directed at one semicircular canal will be ineffective as treatment for a different semicircular canal. Clinically this may be evident if a patient who had been improving with treatment suddenly starts to deteriorate.

The third reason why treatment of BPPV may be unsuccessful is that multiple semicircular canals may be involved, and this situation is generally more difficult to treat. In some cases, at the time of diagnosis it is recognized that multiple canals are involved. In other cases, a patient may begin with involvement of only one canal, but subsequently develop involvement of a multiple canals.

A fourth reason why treatment of BPPV may be unsuccessful is that the patient has not only BPPV, but some other vertiginous disease as well. This may become evident if, during the course of treatment for BPPV, a patient's eye movements normalize (in other words, there nystagmus that is diagnostic of BPPV is no longer present on several sequential examinations), yet the patient still complains of symptoms. In this circumstance it is reasonable to suspect that an additional disease may be at play.

A fifth reason why treatment of BPPV may be unsuccessful is that the patient actually does not have BPPV. Sometimes, even when practitioners are working in good faith and to the best of their abilities, a diagnosis of BPPV may simply be incorrect.

§4.8: Can recurrences of BPPV be prevented?

Since the treatment for BPPV is a specific series of maneuvers (described earlier), many patients reasonably ask whether they should continue doing these maneuvers in order to "ward off" future episodes. This approach has been studied, and found to be ineffective (Helminski,

Janssen et al. 2008). The maneuvers only work when BPPV is "active" (i.e., when symptoms are present). When symptoms are absent, that is because there are no more loose otoliths, and in this "quiescent" state, there is nothing for the maneuvers to "act on."

The observation that vitamin D deficiency may increase the risk of BPPV (Chen, Zhao et al. 2020) leads to the question of whether supplementation of vitamin D might reduce the risk. There is emerging evidence that, after a first episode of BPPV, taking vitamin D and perhaps calcium may reduce the likelihood of recurrence of BPPV (Jeong, Lee et al. 2020). For instance, a randomized controlled trial (Jeong, Kim et al. 2020) reported that supplementation with vitamin D 400 I.U. and calcium carbonate 500 mg twice per day reduces the annual recurrence rate of BPPV. Note that increasing one's level of vitamin D (even without supplementing additional calcium) may increase serum calcium concentrations, which at least in theory can increase the risk of developing kidney stones (Letavernier and Daudon 2018), though it is unclear whether this increased risk is clinically meaningful; one might think that vitamin D supplementation should perhaps be avoided in persons who already have a history of kidney stone formation, but this does not seem to be borne out by the evidence (Ganji, Shafii et al. 2019); if you are considering supplementing your vitamin D intake (with or without calcium supplementation), please consult your primary care physician first.

§5: Prognosis of BPPV. Recurrence.

The natural history of BPPV is one of recurrence. One study (Luryi, Lawrence et al. 2018) of 1105 patients found that after a first episode of BPPV had resolved, recurrence happened anywhere from 3 to 2496 days later; 56% of patients experienced recurrence within the first year, and 76% of patients experienced recurrence within 2 years.

Recurrences usually (but not always) involve the same semicircular canal that was most recently affected. Therefore, if a given vestibular treatment exercise clearly helps a bout of BPPV, but a patient then experiences what feels like a recurrence, it is medically quite reasonable to resume exactly the same exercise that was most recently helpful, because statistically it is likely to be correct. If resuming the previously successful exercise does, in fact, help, then no further intervention is needed. In contrast, if a recurrence fails to respond to a previously successful exercise, then a patient should have her eye movements examined again in order to determine whether the BPPV is "in the same place," or whether the problem is actually still BPPV.

As discussed earlier (see the section above entitled, "Can recurrences of BPPV be prevented?"), there is emerging evidence that supplementation with vitamin D (and perhaps calcium) may reduce the rate of recurrence of BPPV.

There is a modest literature suggesting that BPPV following head trauma is more likely to recur (Dlugaiczyk, Siebert et al. 2011), though not all investigators share this viewpoint.

References

Anagnostou, E., E. Stamboulis and E. Kararizou (2014). "Canal conversion after repositioning procedures: comparison of Semont and Epley maneuver." J Neurol **261**(5): 866-869.
Aron, M., J. Lea, D. Nakku and B. D. Westerberg (2015). "Symptom Resolution Rates of Posttraumatic versus Nontraumatic Benign Paroxysmal Positional Vertigo: A Systematic Review." Otolaryngol Head Neck Surg **153**(5): 721-730.
Babac, S., D. Djeric, M. Petrovic-Lazic, N. Arsovic and A. Mikic (2014). "Why do treatment failure and recurrences of benign paroxysmal positional vertigo occur?" Otol Neurotol **35**(6): 1105-1110.
Babic, B. B., S. D. Jesic, J. D. Milovanovic and N. A. Arsovic (2014). "Unintentional conversion of benign paroxysmal positional vertigo caused by repositioning procedures for canalithiasis: transitional BPPV." Eur Arch Otorhinolaryngol **271**(5): 967-973.
Balatsouras, D. G., G. Koukoutsis, A. Aspris, A. Fassolis, A. Moukos, N. C. Economou and M. Katotomichelakis (2017). "Benign Paroxysmal Positional Vertigo Secondary to Mild Head Trauma." Ann Otol Rhinol Laryngol **126**(1): 54-60.
Balatsouras, D. G., G. Koukoutsis, P. Ganelis, N. C. Economou, A. Moukos, A. Aspris and M. Katotomichelakis (2014). "Benign paroxysmal positional vertigo secondary to vestibular neuritis." Eur Arch Otorhinolaryngol **271**(5): 919-924.
Baloh, R. W., V. Honrubia and K. Jacobson (1987). "Benign positional vertigo: clinical and oculographic features in 240 cases." Neurology **37**(3): 371-378.
Baloh, R. W., K. Jacobson and V. Honrubia (1993). "Horizontal semicircular canal variant of benign positional vertigo." Neurology **43**(12): 2542-2549.
Baloh, R. W., L. Solingen, A. W. Sills and V. Honrubia (1977). "Caloric testing. 1. Effect of different conditions of ocular fixation." Ann Otol Rhinol Laryngol Suppl **86**(5 Pt 3 Suppl 43): 1-6.
Bárány, E. (1920). "Diagnose von Krankheitserscheinungen im Bereiche des Otolithenapparates." Acta Oto-Laryngologica **2**(3): 434-437.
Bhattacharyya, N., S. P. Gubbels, S. R. Schwartz, J. A. Edlow, H. El-Kashlan, T. Fife, J. M. Holmberg, K. Mahoney, D. B. Hollingsworth, R. Roberts, M. D. Seidman, R. W. Steiner, B. T. Do, C. C. Voelker, R. W. Waguespack and M. D. Corrigan (2017). "Clinical Practice Guideline: Benign Paroxysmal Positional Vertigo (Update)." Otolaryngol Head Neck Surg **156**(3_suppl): S1-S47.
Brandt, T. and R. B. Daroff (1980). "Physical therapy for benign paroxysmal positional vertigo." Arch Otolaryngol **106**(8): 484-485.
Cakir, B. O., I. Ercan, Z. A. Cakir and S. Turgut (2006). "Efficacy of postural restriction in treating benign paroxysmal positional vertigo." Arch Otolaryngol Head Neck Surg **132**(5): 501-505.
Casani, A. P., N. Cerchiai, I. Dallan and S. Sellari-Franceschini (2011). "Anterior canal lithiasis: diagnosis and treatment." Otolaryngol Head Neck Surg **144**(3): 412-418.
Casqueiro, J. C., A. Ayala and G. Monedero (2008). "No more postural restrictions in posterior canal benign paroxysmal positional vertigo." Otol Neurotol **29**(5): 706-709.
Chambers, B. R. and M. A. Gresty (1982). "Effects of fixation and optokinetic stimulation on vestibulo-ocular reflex suppression." J Neurol Neurosurg Psychiatry **45**(11): 998-1004.
Chen, J., W. Zhao, X. Yue and P. Zhang (2020). "Risk Factors for the Occurrence of Benign Paroxysmal Positional Vertigo: A Systematic Review and Meta-Analysis." Front Neurol **11**: 506.
Choi, S. Y., S. W. Oh, H. J. Kim and J. S. Kim (2020). "Determinants for bedside lateralization of benign paroxysmal positional vertigo involving the horizontal semicircular canal." J Neurol.
Choung, Y. H., Y. R. Shin, H. Kahng, K. Park and S. J. Choi (2006). "'Bow and lean test' to determine the affected ear of horizontal canal benign paroxysmal positional vertigo." Laryngoscope **116**(10): 1776-1781.
Ciniglio Appiani, G., G. Catania, M. Gagliardi and G. Cuiuli (2005). "Repositioning maneuver for the treatment of the apogeotropic variant of horizontal canal benign paroxysmal positional vertigo." Otol Neurotol **26**(2): 257-260.
De Stefano, A., F. Dispenza, L. Citraro, A. G. Petrucci, P. Di Giovanni, G. Kulamarva, N. Mathur and A. Croce (2011). "Are postural restrictions necessary for management of posterior canal benign paroxysmal positional vertigo?" Ann Otol Rhinol Laryngol **120**(7): 460-464.
Devaiah, A. K. and S. Andreoli (2010). "Postmaneuver restrictions in benign paroxysmal positional vertigo: an individual patient data meta-analysis." Otolaryngol Head Neck Surg **142**(2): 155-159.

Dispenza, F., D. E. S. A, C. Costantino, D. Rando, M. Giglione, R. Stagno and E. Bennici (2015). "Canal switch and re-entry phenomenon in benign paroxysmal positional vertigo: difference between immediate and delayed occurrence." Acta Otorhinolaryngol Ital **35**(2): 116-120.
Dix, M. R. and C. S. Hallpike (1952). "The pathology symptomatology and diagnosis of certain common disorders of the vestibular system." Proc R Soc Med **45**(6): 341-354.
Dix, M. R. and C. S. Hallpike (1952). "The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system." Ann Otol Rhinol Laryngol **61**(4): 987-1016.
Dlugaiczyk, J., S. Siebert, D. J. Hecker, C. Brase and B. Schick (2011). "Involvement of the anterior semicircular canal in posttraumatic benign paroxysmal positioning vertigo." Otol Neurotol **32**(8): 1285-1290.
Epley, J. M. (1980). "New dimensions of benign paroxysmal positional vertigo." Otolaryngol Head Neck Surg (1979) **88**(5): 599-605.
Fife, T. D., D. J. Iverson, T. Lempert, J. M. Furman, R. W. Baloh, R. J. Tusa, T. C. Hain, S. Herdman, M. J. Morrow, G. S. Gronseth and A. A. o. N. Quality Standards Subcommittee (2008). "Practice parameter: therapies for benign paroxysmal positional vertigo (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology." Neurology **70**(22): 2067-2074.
Foster, C. A., A. Ponnapan, K. Zaccaro and D. Strong (2012). "A Comparison of Two Home Exercises for Benign Positional Vertigo: Half Somersault versus Epley Maneuver." Audiology and Neurotology Extra **2**(1): 16-23.
Foster, C. A., K. Zaccaro and D. Strong (2012). "Canal conversion and reentry: a risk of Dix-Hallpike during canalith repositioning procedures." Otol Neurotol **33**(2): 199-203.
Frenzel, H. (1956). "[Practical methods of a systematic study of otorhinolaryngology]." Munch Med Wochenschr **98**(29): 972-975.
Fresnel, A.-J. (1822). Mémoire sur un nuveau système d'éclairage des phares. Paris, Imprimerie Royale Lu à L'académie Des Sciences.
Fu, W., J. Han, N. Chang, D. Wei, Y. Bai, Y. Wang, F. He and X. Wang (2020). "Corrigendum to "Immediate efficacy of Gufoni maneuver for horizontal canal benign paroxysmal positional vertigo (HC-BPPV): A meta-analysis" [Auris Nasus Larynx (2020) 48-54]." Auris Nasus Larynx **47**(1): 171-172.
Fu, W., J. Han, N. Chang, D. Wei, Y. Bai, Y. Wang, F. He and X. Wang (2020). "Immediate efficacy of Gufoni maneuver for horizontal canal benign paroxysmal positional vertigo (HC-BPPV): a meta-analysis." Auris Nasus Larynx **47**(1): 48-54.
Fyrmpas, G., D. Rachovitsas, A. B. Haidich, J. Constantinidis, S. Triaridis, V. Vital and M. Tsalighopoulos (2009). "Are postural restrictions after an Epley maneuver unnecessary? First results of a controlled study and review of the literature." Auris Nasus Larynx **36**(6): 637-643.
Gacek, R. R. (1978). "Posterior ampullary nerve transection for benign paroxysmal positional vertigo." Trans Pa Acad Ophthalmol Otolaryngol **31**(2): 190-193.
Ganji, M. R., Z. Shafii and M. S. Hakemi (2019). "Vitamin D Supplementation and Risk of Hypercalciuria in Stone Formers." Iran J Kidney Dis **13**(1): 27-31.
Gauthier, G. M. and J. L. Vercher (1990). "Visual vestibular interaction: vestibulo-ocular reflex suppression with head-fixed target fixation." Exp Brain Res **81**(1): 150-160.
Gizzi, M. S. and H. W. Harper (2003). "Suppression of the human vestibulo-ocular reflex by visual fixation or forced convergence in the dark, with a model interpretation." Curr Eye Res **26**(5): 281-290.
Gordon, C. R. and N. Gadoth (2004). "Repeated vs single physical maneuver in benign paroxysmal positional vertigo." Acta Neurol Scand **110**(3): 166-169.
Gufoni, M., L. Mastrosimone and F. Di Nasso (1998). "[Repositioning maneuver in benign paroxysmal vertigo of horizontal semicircular canal]." Acta Otorhinolaryngol Ital **18**(6): 363-367.
Hanley, K. and T. O'Dowd (2002). "Symptoms of vertigo in general practice: a prospective study of diagnosis." Br J Gen Pract **52**(483): 809-812.
Hart, C. W. (1967). "Ocular fixation and the caloric test." Laryngoscope **77**(12): 2103-2113.
Helminski, J. O. (2012). "Differential Diagnosis and Treatment of Anterior Canal Benign Paroxysmal Positional Vertigo." Vestibular SIG Newsletter **Special edition: BPPV**.
Helminski, J. O., I. Janssen and T. C. Hain (2008). "Daily exercise does not prevent recurrence of benign paroxysmal positional vertigo." Otol Neurotol **29**(7): 976-981.
Hilton, M. P. and D. K. Pinder (2014). "The Epley (canalith repositioning) manoeuvre for benign paroxysmal positional vertigo." Cochrane Database Syst Rev(12): CD003162.

Hirvonen, T. P., M. Juhola and H. Aalto (2012). "Suppression of spontaneous nystagmus during different visual fixation conditions." Eur Arch Otorhinolaryngol **269**(7): 1759-1762.
Hong, S. M., D. C. Park, S. G. Yeo and C. I. Cha (2008). "Vestibular evoked myogenic potentials in patients with benign paroxysmal positional vertigo involving each semicircular canal." Am J Otolaryngol **29**(3): 184-187.
Honrubia, V., R. W. Baloh, M. R. Harris and K. M. Jacobson (1999). "Paroxysmal positional vertigo syndrome." Am J Otol **20**(4): 465-470.
Hoseinabadi, R., A. Pourbakht, N. Yazdani, A. Kouhi and M. Kamali (2016). "The effects of abnormality of cVEMP and oVEMP on rehabilitation outcomes in patients with idiopathic benign paroxysmal positional vertigo." Eur Arch Otorhinolaryngol **273**(3): 643-648.
Ishiyama, A., K. M. Jacobson and R. W. Baloh (2000). "Migraine and benign positional vertigo." Ann Otol Rhinol Laryngol **109**(4): 377-380.
Jackson, L. E., B. Morgan, J. C. Fletcher, Jr. and W. W. Krueger (2007). "Anterior canal benign paroxysmal positional vertigo: an underappreciated entity." Otol Neurotol **28**(2): 218-222.
Jeong, S. H., S. H. Choi, J. Y. Kim, J. W. Koo, H. J. Kim and J. S. Kim (2009). "Osteopenia and osteoporosis in idiopathic benign positional vertigo." Neurology **72**(12): 1069-1076.
Jeong, S. H., J. S. Kim, H. J. Kim, J. Y. Choi, J. W. Koo, K. D. Choi, J. Y. Park, S. H. Lee, S. Y. Choi, S. Y. Oh, T. H. Yang, J. H. Park, I. Jung, S. Ahn and S. Kim (2020). "Prevention of benign paroxysmal positional vertigo with vitamin D supplementation: A randomized trial." Neurology **95**(9): e1117-e1125.
Jeong, S. H., S. U. Lee and J. S. Kim (2020). "Prevention of recurrent benign paroxysmal positional vertigo with vitamin D supplementation: a meta-analysis." J Neurol.
Karlsen, E. A., C. P. Goetzinger and R. Hassanein (1980). "Effects of six conditions of ocular fixation on caloric nystagmus." Arch Otolaryngol **106**(8): 474-476.
Katsarkas, A. (1999). "Benign paroxysmal positional vertigo (BPPV): idiopathic versus post-traumatic." Acta Otolaryngol **119**(7): 745-749.
Katsarkas, A. and T. H. Kirkham (1982). "Failure of suppression of post-caloric nystagmus by fixation." J Otolaryngol **11**(1): 57-59.
Kim, J. S., S. Y. Oh, S. H. Lee, J. H. Kang, D. U. Kim, S. H. Jeong, K. D. Choi, I. S. Moon, B. K. Kim and H. J. Kim (2012). "Randomized clinical trial for geotropic horizontal canal benign paroxysmal positional vertigo." Neurology **79**(7): 700-707.
Kim, S. K., S. M. Hong, I. S. Park and H. G. Choi (2019). "Association Between Migraine and Benign Paroxysmal Positional Vertigo Among Adults in South Korea." JAMA Otolaryngol Head Neck Surg **145**(4): 307-312.
Kim, Y. K., J. E. Shin and J. W. Chung (2005). "The effect of canalith repositioning for anterior semicircular canal canalithiasis." ORL J Otorhinolaryngol Relat Spec **67**(1): 56-60.
Kisilevsky, V., N. A. Bailie, S. N. Dutt and J. A. Rutka (2009). "Lessons learned from the surgical management of benign paroxysmal positional vertigo: the University Health Network experience with posterior semicircular canal occlusion surgery (1988-2006)." J Otolaryngol Head Neck Surg **38**(2): 212-221.
Korres, S., E. Gkoritsa, D. Giannakakou-Razelou, I. Yiotakis, M. Riga and T. P. Nikolpoulos (2011). "Vestibular evoked myogenic potentials in patients with BPPV." Med Sci Monit **17**(1): CR42-47.
Kutlubaev, M. A., Y. Xu and J. Hornibrook (2019). "Benign paroxysmal positional vertigo in Meniere's disease: systematic review and meta-analysis of frequency and clinical characteristics." J Neurol.
Lee, G., S. G. Lee, H. S. Park, B. J. Kim, S. J. Choi and J. W. Choi (2019). "Clinical characteristics and associated factors of canal switch in benign paroxysmal positional vertigo." J Vestib Res **29**(5): 253-260.
Lee, J. D., M. K. Park, B. D. Lee, T. K. Lee, K. B. Sung and J. Y. Park (2013). "Abnormality of cervical vestibular-evoked myogenic potentials and ocular vestibular-evoked myogenic potentials in patients with recurrent benign paroxysmal postitional vertigo." Acta Otolaryngol **133**(2): 150-153.
Lee, N. H., J. H. Ban, K. C. Lee and S. M. Kim (2010). "Benign paroxysmal positional vertigo secondary to inner ear disease." Otolaryngol Head Neck Surg **143**(3): 413-417.
Lempert, T. (1994). "Horizontal benign positional vertigo." Neurology **44**(11): 2213-2214.
Letavernier, E. and M. Daudon (2018). "Vitamin D, Hypercalciuria and Kidney Stones." Nutrients **10**(3).
Levo, H., H. Aalto and T. Petteri Hirvonen (2004). "Nystagmus measured with video-oculography: methodological aspects and normative data." ORL J Otorhinolaryngol Relat Spec **66**(3): 101-104.
Lin, G. C., G. J. Basura, H. T. Wong and K. D. Heidenreich (2012). "Canal switch after canalith repositioning procedure for benign paroxysmal positional vertigo." Laryngoscope **122**(9): 2076-2078.

Lopez-Escamez, J. A., A. Lopez-Nevot, M. J. Gamiz, P. M. Moreno, F. Bracero, J. L. Castillo and J. Salinero (2000). "[Diagnosis of common causes of vertigo using a structured clinical history]." Acta Otorrinolaringol Esp **51**(1): 25-30.
Luryi, A. L., M. LaRouere, S. Babu, D. I. Bojrab, J. Zappia, E. W. Sargent and C. A. Schutt (2019). "Traumatic versus Idiopathic Benign Positional Vertigo: Analysis of Disease, Treatment, and Outcome Characteristics." Otolaryngol Head Neck Surg **160**(1): 131-136.
Luryi, A. L., J. Lawrence, D. I. Bojrab, M. LaRouere, S. Babu, J. Zappia, E. W. Sargent, E. Chan, I. Naumann, R. S. Hong and C. A. Schutt (2018). "Recurrence in Benign Paroxysmal Positional Vertigo: A Large, Single-Institution Study." Otol Neurotol **39**(5): 622-627.
Luryi, A. L., C. A. Schutt, D. I. Bojrab, M. LaRouere, J. Zappia, E. W. Sargent and S. Babu (2018). "Causes of Persistent Positional Vertigo Following Posterior Semicircular Canal Occlusion for Benign Paroxysmal Positional Vertigo." Otol Neurotol **39**(10): e1078-e1083.
McClure, J. A. (1985). "Horizontal canal BPV." J Otolaryngol **14**(1): 30-35.
McGinnis, P. Q., M. Nebbia, L. Saez and K. Rudolph (2009). "Retrospective comparison of outcomes for patients with benign paroxysmal positional vertigo based on length of postural restrictions." J Geriatr Phys Ther **32**(4): 168-173.
Molnar, E. M. and N. Torok (1974). "The effect of ocular fixation on the caloric nystagmus." ORL J Otorhinolaryngol Relat Spec **36**(2): 76-84.
Moon, S. J., S. H. Bae, H. D. Kim, J. H. Kim and Y. B. Cho (2005). "The effect of postural restrictions in the treatment of benign paroxysmal positional vertigo." Eur Arch Otorhinolaryngol **262**(5): 408-411.
Mostafa, B. E., T. A. Youssef and A. S. Hamad (2013). "The necessity of post-maneuver postural restriction in treating benign paroxysmal positional vertigo: a meta-analytic study." Eur Arch Otorhinolaryngol **270**(3): 849-852.
Nedzelski, J. M., H. O. Barber and L. McIlmoyl (1986). "Diagnoses in a dizziness unit." J Otolaryngol **15**(2): 101-104.
Neuhauser, H. K. (2007). "Epidemiology of vertigo." Curr Opin Neurol **20**(1): 40-46.
Nuti, D., C. Nati and D. Passali (2000). "Treatment of benign paroxysmal positional vertigo: no need for postmaneuver restrictions." Otolaryngol Head Neck Surg **122**(3): 440-444.
Oya, R., T. Imai, Y. Takenaka, T. Sato, K. Oshima, Y. Ohta and H. Inohara (2019). "Clinical significance of cervical and ocular vestibular evoked myogenic potentials in benign paroxysmal positional vertigo: a meta-analysis." Eur Arch Otorhinolaryngol **276**(12): 3257-3265.
Pagnini, P., D. Nuti and P. Vannucchi (1989). "Benign paroxysmal vertigo of the horizontal canal." ORL J Otorhinolaryngol Relat Spec **51**(3): 161-170.
Papacharalampous, G. X., P. V. Vlastarakos, G. P. Kotsis, D. Davilis and L. Manolopoulos (2012). "The Role of Postural Restrictions after BPPV Treatment: Real Effect on Successful Treatment and BPPV's Recurrence Rates." Int J Otolaryngol **2012**: 932847.
Park, S., B. G. Kim, S. H. Kim, H. Chu, M. Y. Song and M. Kim (2013). "Canal conversion between anterior and posterior semicircular canal in benign paroxysmal positional vertigo." Otol Neurotol **34**(9): 1725-1728.
Parnes, L. S., S. K. Agrawal and J. Atlas (2003). "Diagnosis and management of benign paroxysmal positional vertigo (BPPV)." CMAJ **169**(7): 681-693.
Parnes, L. S. and J. A. McClure (1990). "Posterior semicircular canal occlusion for intractable benign paroxysmal positional vertigo." Ann Otol Rhinol Laryngol **99**(5 Pt 1): 330-334.
Pisani, V., S. Mazzone, R. Di Mauro, P. G. Giacomini and S. Di Girolamo (2015). "A survey of the nature of trauma of post-traumatic benign paroxysmal positional vertigo." Int J Audiol **54**(5): 329-333.
Roberts, R. A., R. E. Gans, J. L. DeBoodt and J. J. Lister (2005). "Treatment of benign paroxysmal positional vertigo: necessity of postmaneuver patient restrictions." J Am Acad Audiol **16**(6): 357-366.
Roberts, R. A., R. E. Gans and R. L. Montaudo (2006). "Efficacy of a new treatment maneuver for posterior canal benign paroxysmal positional vertigo." J Am Acad Audiol **17**(8): 598-604.
Scarpa, A., F. M. Gioacchini, E. Cassandro, M. Tulli, M. Ralli, M. Re and C. Cassandro (2019). "Clinical application of cVEMPs and oVEMPs in patients affected by Meniere's disease, vestibular neuritis and benign paroxysmal positional vertigo: a systematic review." Acta Otorhinolaryngol Ital **39**(5): 298-307.
Semont, A., G. Freyss and E. Vitte (1988). "Curing the BPPV with a liberatory maneuver." Adv Otorhinolaryngol **42**: 290-293.

Shaia, W. T., J. J. Zappia, D. I. Bojrab, M. L. LaRouere, E. W. Sargent and R. C. Diaz (2006). "Success of posterior semicircular canal occlusion and application of the dizziness handicap inventory." Otolaryngol Head Neck Surg **134**(3): 424-430.
Simoceli, L., R. S. Bittar and M. E. Greters (2005). "Posture restrictions do not interfere in the results of canalith repositioning maneuver." Braz J Otorhinolaryngol **71**(1): 55-59.
Singh, N. K. and K. Apeksha (2016). "Efficacy of cervical and ocular vestibular-evoked myogenic potentials in evaluation of benign paroxysmal positional vertigo of posterior semicircular canal." Eur Arch Otorhinolaryngol **273**(9): 2523-2532.
Sokolovski, A. (1966). "The influence of mental activity and visual fixation upon caloric-induced nystagmus in normal subjects." Acta Otolaryngol **61**(3): 209-220.
Squires, T. M., M. S. Weidman, T. C. Hain and H. A. Stone (2004). "A mathematical model for top-shelf vertigo: the role of sedimenting otoconia in BPPV." J Biomech **37**(8): 1137-1146.
Strupp, M., C. Fischer, L. Hanss and O. Bayer (2014). "The takeaway Frenzel goggles: a Fresnel-based device." Neurology **83**(14): 1241-1245.
Thomas, A., E. Rosseeva, O. Hochrein, W. Carrillo-Cabrera, P. Simon, P. Duchstein, D. Zahn and R. Kniep (2012). "Mimicking the growth of a pathologic biomineral: shape development and structures of calcium oxalate dihydrate in the presence of polyacrylic acid." Chemistry **18**(13): 4000-4009.
von Brevern, M., P. Bertholon, T. Brandt, T. Fife, T. Imai, D. Nuti and D. Newman-Toker (2015). "Benign paroxysmal positional vertigo: Diagnostic criteria." J Vestib Res **25**(3-4): 105-117.
von Brevern, M., P. Bertholon, T. Brandt, T. Fife, T. Imai, D. Nuti and D. Newman-Toker (2017). "Benign paroxysmal positional vertigo: Diagnostic criteria Consensus document of the Committee for the Classification of Vestibular Disorders of the Barany Society." Acta Otorrinolaringol Esp **68**(6): 349-360.
von Brevern, M., A. Radtke, F. Lezius, M. Feldmann, T. Ziese, T. Lempert and H. Neuhauser (2007). "Epidemiology of benign paroxysmal positional vertigo: a population based study." J Neurol Neurosurg Psychiatry **78**(7): 710-715.
Walther, L. E. and M. Westhofen (2007). "Presbyvertigo-aging of otoconia and vestibular sensory cells." J Vestib Res **17**(2-3): 89-92.
Xu, H., F. Y. Liang, L. Chen, X. C. Song, M. C. Tong, J. F. Thong, Q. Q. Zhang and Y. Sun (2016). "Evaluation of the utricular and saccular function using oVEMPs and cVEMPs in BPPV patients." J Otolaryngol Head Neck Surg **45**: 12.
Yacovino, D. A., T. C. Hain and F. Gualtieri (2009). "New therapeutic maneuver for anterior canal benign paroxysmal positional vertigo." J Neurol **256**(11): 1851-1855.
Yang, W. S., S. H. Kim, J. D. Lee and W. S. Lee (2008). "Clinical significance of vestibular evoked myogenic potentials in benign paroxysmal positional vertigo." Otol Neurotol **29**(8): 1162-1166.
Yetiser, S., D. Ince and M. Gul (2014). "An analysis of vestibular evoked myogenic potentials in patients with benign paroxysmal positional vertigo." Ann Otol Rhinol Laryngol **123**(10): 686-695.