

Benign paroxysmal positional vertigo



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Jane Doe, an active 67 year old woman, rolled over in bed, ready to begin her day, and opened her eyes. The familiar sight of her bedroom greeted her, and then everything around her began to spin in a dizzying array of color and movement. Jane quickly closed her eyes and rolled back over, a feeling of panic rising within her. She waited a few moments behind the safety of her closed eyes and then she cautiously cracked one eye open. Everything appeared in order. The room remained still and Jane felt her panic subside. To make one hundred percent sure that everything was alright, she tentatively rolled over again. A few seconds passed and then the room began to spin. Quickly she rolled onto her back, wondering what was wrong. With a sinking heart she made an appointment with her doctor, who informed her that she needed to see an ENT. It wasn't long before she was informed that she was suffering from BPPV, or benign paroxysmal positional vertigo, a condition characterized by a false sense of spinning and dizziness caused by contradictory sensory information sent to the brain by our eyes, muscles, and vestibular systems (vestibular. org).

First described by Robert Barany, an Austrian otolaryngologist, in 1921, BPPV was first referred to as a, “ syndrome of episodic vertigo, induced by sudden movement of the head,” (Pearce, 2007). It wasn't until 1952, when Dix and Hallpike presented a more in depth study of the disorder, that the syndrome was given the name, benign paroxysmal positional vertigo (Pearce, 2007). Each part of its name describes another aspect of the disorder. ‘ Benign’, means that although it is unpleasant and in some cases debilitating, BPPV is not life threatening. The word ‘ paroxysmal’ describes the short and sudden nature of the symptoms during a classic attack of BPPV. Because the

symptoms of BPPV are triggered by changes in head position, Dix and Hallpike included the term ‘positional’ in their name for this syndrome. Lastly, they included the most common and characteristic symptom of the disorder-‘vertigo’ (vestibular. org).

The vertigo of BPPV is caused by a disconnect in the processing of sensory information. The human brain uses messages from multiple sources to understand the world around it. In order to know where the body is in space, the brain combines sensory input from our eyes, muscle joints, and vestibular systems to create our sense of proprioception (vestibular. org). The bulk of the information is sent from the vestibular systems of both ears, located deep within the head inside the inner ear. This extremely sensitive system is made up of two parts, the vestibule and the semicircular canals, and is responsible for sensing head movement and rotation (mayoclinic).

The vestibule is divided into the two otolith organs, the utricle and saccule, which are responsible for sensing, “acceleration in the horizontal and vertical planes respectively,” (Reches, 2019) and are divided by the striola, a line marking the division in the orientation of the hair cells (NIH ²). Inside the otolith organs is the macula, a strip of sensory epithelial tissue that contains hair cells, which project into a gelatinous layer. This layer lies directly below the otolithic membrane, which has embedded within it small calcium carbonate crystals called otoconia. The otoconia make the otolithic membrane heavier than anything in its surroundings, which causes it to move with gravity when the head tilts. “The resulting shearing motion between the otolithic membrane and the macula displaces the hair bundles,”

which sets off the chain reaction needed to pass the message up the eighth nerve to the brain (NIH ²).

The second part of the vestibular system is the semi-circular canals, a series of three endolymph filled ducts that sit perpendicular from each other different planes. Each canal is named for the plane in which it sits. The first is called the superior or anterior canal, which senses back and forth head movement. The next one is the posterior canal, which senses up and down movement, and the final one, the horizontal or lateral canal, senses left and right head tilting. At the end of each canal is the ampulla, “ a bulbous expansion,” at its base, that house the cristae, hair cells that project into the cupula, a gelatinous mass separating the ampulla from the semi-circular canals (NIH ¹). When the head is rotated, the endolymph in the canal sitting in the plane of the rotation moves, distorting the cupula, which triggers the hair cells that send a message to the brain for processing (NIH ¹).

Although the vestibular system communicates with the semi-circular canals through the utricle, for the most part the two systems remain separate. However, sometimes a piece of the otoconia can break off the otolithic membrane and enter the semi-circular canals, where it wreaks havoc in this highly sensitive system, causing benign paroxysmal positional vertigo, or BPPV (mayoclinic).

Patients diagnosed with BPPV, the most common cause of vertigo, complain of dizziness, light headiness, nausea, vomiting, and a false sense of their surroundings spinning (mayo clinic), “ when their head is in the offending position,” (Reches, 2019). BPPV can be caused by numerous issues including <https://assignbuster.com/benign-paroxysmal-positional-vertigo/>

but not limited to head trauma (21%), inner ear disorders, such as vestibular neuritis (7%), Meniere's disease (7%), sudden sensorineural hearing loss (2%); acoustic neuromas (2%), ear diseases (29%), such as otosclerosis (4%) and otitis media (9%); and diseases of the central nervous system (11%). However, most often cases of BPPV have an idiopathic or unknown etiology (39%) (Li, 2018). Because BPPV is common in middle aged women, some researchers have postulated that it might be caused by a hormonal imbalance, explaining that lower estrogen levels might cause problems with the internal structure and connectedness of the otoconia to their gelatinous matrix, making it more likely that they will break off and enter the canals, resulting in BPPV. Other researchers point to the low, "mineral density scores," and high cases of osteoporosis found in both men and women with BPPV, which may indicate that a, "deranged calcium metabolism," might be a factor in causing BPPV (Journal of Clinical Neurology, 2010). A correlation has also been found between, "vestibular nerve...degeneration," and BPPV (Hamid, 2001).

In addition to researching the cause of idiopathic BPPV, scientists have also studied the two types of BPPV, which are characterized by the action and location of the broken pieces of otoconia. The first type, suggested by Harol Schuknecht, MD, in 1962, was termed cupulolithiasis, which means, "heavy cupula," (Li, 2018). In this type of BPPV, the otoconia exit the ampulla and adhere to the cupula, which makes it difficult for the cupula to remain in a "neutral" position, causing it to shift, which activates the hair cells and sends a false signal to the brain. Although today Schuknecht's theory is accepted, at the time not all researchers agreed, and in 1980, Epley suggested an

alternative theory for BPPV called canalolithiasis. His theory proposed that the loose otoconia actually enters into the semi-circular canal itself causing the endolymph, which is usually only sensitive to head rotation, to move with the crystal as it reacts to gravity (Li, 2018). This also causes false messages to be sent to the brain, which contradicts information sent by our eyes and muscles, causing a person to feel as if they or their surroundings are spinning (vestibular. org). Today, both theories are accepted by the medical community (Reches, 2019).

When a patient complains of dizziness, an ENT or audiologist will begin by performing several test maneuvers that involve systematically positioning the patient's head in several positions in an attempt to cause the otoconia to shift and cause the symptoms of BPPV to be present. This is done so that the physician, usually through the aid of frenzel goggles, which magnify the eye and reduce eye fixation, can observe the resulting nystagmus, or, " reflexive rhythmic... eye movements," (Bhatnagar, 2018) which is used for differential diagnosis of which canal is affected and whether canalolithiasis or cupulolithiasis is present (sciencedirect).

Characterized by a slow and fast phase, nystagmus is the result of the vestibular reflex, which enables humans to retain focus on their environment as the head rotates, gone wrong. When BPPV is present, the body is still but the misinformation sent to the brain causes the eyes to begin to move as if the body was turning, resulting in nystagmus (Bhatnagar, 2018). Nystagmus can be described as horizontal, vertical, and rotational, which can be ageotropic, which means that the eye is moving in the opposite direction that the head is turned in, geotropic, which means that the eye is moving

towards the ground, or apogeotropic, which means the eye is moving away from the ground (Li, 2018). Because there are other causes for nystagmus, such as over-ingesting alcohol and drugs, it is important for clinicians to be aware of the unique symptoms of BPPV induced nystagmus, such as a, “slightly delayed onset, provoked by movement or position of the head... reduced with repeated positioning, and accompanied by vertigo,” (Gleason, 2012). Only if these symptoms are present during a Dix-Hall Pike maneuver can the nystagmus be considered as a sign of BPPV.

Because BPPV in the posterior canal is most common (60-90%), the Dix-Hallpike maneuver, which will trigger, “up beating and torsional nystagmus,” if the patient is suffering from PC-BPPV, is performed first (Journal of Clinical Neurology, 2010). This maneuver brings the patient from an upright position to a supine one, with their head angled at 45° and their neck extended 20°. The clinician then holds the patient in that position and watches for nystagmus (American Academy of Otolaryngology, 2017). If the eyes move counterclockwise, it is the right ear that is affected, while if they move clockwise it is a sign that the BPPV is stemming from the left ear (Hamid, 2001). When checking for PC-BPPV cupulolithiasis, clinicians performing the Dix-Hallpike should watch out for abnormally strong and long-lasting nystagmus that fatigues with characteristic, “eye jerks,” that are symptomatic of constant cupula irritation (Hamid, 2001).

Less common than PC-BPPV, is BPPV in the horizontal or lateral canal (HC-BPPV). Because no nystagmus will be present during a Dix-Hallpike if the patient has HC-BPPV, clinicians should perform a supine roll test, a clinical maneuver characterized by moving the patient’s head 90 degrees to both

the left and then the right sides to observe the resultant nystagmus, which is typically longer lasting than found in PC-BPPV, as well (Journal of Clinical Neurology, 2010). The nystagmus observed during the supine roll test, which can be either geotropic or apogeotropic, give clinicians a clue as to where the otolithic debris is. For example, when the otoconia are free floating in the long arm of the lateral semi-circular canal, the resultant nystagmus will be horizontal and geotropic. On the other hand, if the otoconia are stuck to the cupula, resulting in cupulolithiasis, the nystagmus will be apogeotropic (American Academy of Otolaryngology, 2017). Nystagmus can also be observed in patients with HC-BPPV in either a supine position or when the patient is sitting upright with his head bent forward. 80% of the time the nystagmus in each of these positions will be moving in the opposite directions (Journal of Clinical Neurology, 2010). Because it is the highest up, which makes it difficult for otolithic debris to collect in the canal, Anterior canal BPPV (AC-BPPV) is extremely rare and its, “ existence has been challenged.” However as time goes on more and more cases of AC-BPPV have been diagnosed. For AC-BPPV a, “ downbeat nystagmus with ipsitorisional component,” is seen (Journal of Clinical Neurology, 2010). Another rare type of BPPV is the mixed canal type. Affecting only 1. 5-5% of cases, mixed canal type is usually a combination of the posterior and horizontal canals in the same ear, but in some cases it can be different ears (Journal of Clinical Neurology, 2010).

Most cases of BPPV will resolve by itself, with PC-BPPV lasting approximately 39-47 days and HC-BPPV lasting only 16-19 days (Journal of Clinical Neurology, 2010) when the endolymph flowing in the semi-circular canals

dissolves the otolithic debris (Hamid, 2001). However, because of the debilitating symptoms, many clinicians are tempted to provide patients with vestibular suppressant medications, such as anti-histamines and Benzodiazepines. Although many people have become accustomed to solving their problems by swallowing a pill, researchers at the American Academy of Otolaryngology have warned against this practice, claiming that such medications may only be given to reduce symptoms, such as nausea and vomiting, in severe cases (2017). However, these medications should never be used in lieu of the primary treatment for BPPV, canalith repositioning maneuvers, which clinicians have devised to successfully speed up the process of otolithic removal from the semi-circular canals (American Academy of Otolaryngology, 2017).

The most popular repositioning maneuver for treatment of PC-BPPV, with a success rate of 80% after one treatment (Journal of Clinical Neurology, 2010) and 90% after 1-3 treatments, (vestibular.org) is the Epley maneuver, named for Dr. John Epley, which focuses on moving the loose otoconia in the affected canal through head rotations that slowly shift the loose floating crystal into a harmless position at the bottom of the canal or return it to the utricle (Reches, 2019). For patients who have difficulty moving or extending their neck due to spinal injuries or surgeries, an alternative maneuver, called the Semont's Liberatory or the 'slam-dunk procedure', can also be performed (Journal of Clinical Neurology, 2010). For this treatment, patients are moved quickly from one side-lying position to the opposite one while keeping their head still (webmd) The Semont is also used to treat cupulolithiasis. The quick movements involved in the maneuver shake the

otoconia free from the cupula, which then allows the clinician to guide to now free floating otoconia out to the canal (Reches, 2019). Other clinicians have created hybrid maneuvers, such as the Gans repositioning maneuver (GRM), that combine the theories behind or the movements of the Epley and Semont maneuvers to treat BPPV in patients with extreme physical limitations because of disorders such as cervical spondylosis and back problems (Gans et al, 2006). In a 2006 study, Dr. Richard Gans et al proved that 80. 2% of patients treated with the GRM exhibited no symptoms following one treatment and 90. 6% of patients were cured after two GRM treatments. HC-BPPV requires a different set of repositioning treatments, including the Lempert 360° roll maneuver, also known as the barbeque roll, which involves having the patient lie supine, turn their head to the unaffected ear, and then roll until they have completed a 360° roll. Unlike the Lempert roll, which only treats geotropic HC-BPPV, the Gufoni maneuver, originally published in 1998, can treat geotropic and apogeotropic HC-BPPV. The maneuver involves the patient lying on their side and then quickly positioning their head 45°-60° towards the floor. To treat apogeotropic cupulolithiasis with the Gufoni maneuver, the patient's head is positioned nose up 45°-60° from the floor for 1-2 minutes, which shakes the otoconia free from the cupula (American Academy of Otolaryngology, 2017). To treat AC-BPPV, most researchers suggest performing the reverse Epley maneuver, which involves initially positioning the patient on the side of the healthy ear and then moving to the side of the affected ear (Journal of Clinical Neurology, 2010).

Although the canalith repositioning maneuvers used to treat BPPV have a high rate of success, recurrence is common. According to the Journal of Clinical Neurology, “most recurrences (80%) occur within the first year after treatment,” with 15-37% of patients reporting symptoms following their initial treatment (2010). And about half of patients will suffer at least one relapse during the next ten years. Most of these recurrences can be treated with additional repositioning maneuvers. However, for specific cases, other options, such as surgery, may be appropriate.

Although repositioning maneuvers, such as the Epley and the Semont Liberatory, have been shown to be the least invasive and most effective methods for treating BPPV, there are several surgical treatments that have been developed to cure persistent cases of, “intractable BPPV,” (Journal of Clinical Neurology, 2010). These surgeries should only be considered as a last option for cases where all other methods have failed and the, “persistent spells of disabling...vertigo,” is severely reducing a patient’s quality of life (Journal of Clinical Neurology, 2010). For such patients there are several options, the most popular is either a Singular Neurectomy or a Posterior Canal Occlusion (Li, 2018). Originally recorded in 1974 by Gacek, a singular neurectomy has been described as a, “transection of the posterior ampullary nerve innervating the posterior canal,” (Journal of Clinical Neurology, 2010). By cutting the nerve passing the message from the vestibular system to the brain, this surgery prevents the misinformation created by the otolithic debris from being passed to the brain where it triggers the symptoms of BPPV. Unfortunately, this also results in, “significant post-operative balance issues,” in many patients (Li, 2018). It is

for this reason, coupled with a dearth of surgeons who have been trained in performing this surgery (Li, 2018), that cause many patients to choose the second surgical option, posterior canal occlusion, also known as, “canal plugging,” (Journal of Clinical Neurology, 2010). During PC- occlusion, the surgeon, drills through the bony labyrinth until he reaches the membranous labyrinth of the semi-circular canals and then compresses the semicircular canal causing the flow of the endolymph through the canal to be disrupted. This prevents the otoconia from entering the canal and causing symptoms of vertigo (Li, 2018).

Since its original presentation in 1921 and naming in 1952, there has been extensive research into the causes and treatments of benign paroxysmal positional vertigo. However, as listed in their published guideline, the American Academy of Otolaryngology pointed out several areas that still need to be researched. For example, research is needed to determine the optimal number of canal repositioning maneuvers needed to successfully rid the patient of all symptoms with the least chance of recurrence and how much time should elapse between each maneuver. The foundation also stresses the necessity of studies determining the correlation between BPPV and fall risk. Mastoid vibration, which some practitioners still use and others claim is useless and may actually cause additional otoconia to be shaken loose (Reches, 2019), also needs to be assessed for effectiveness (American Academy of Otolaryngology, 2017). This small sampling only covers several areas of general research still required to be performed on the medical community’s continued quest to fully understand benign paroxysmal positional vertigo.

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