REVIEW ARTICLE

BENIGN POSITIONAL PAROXYSMAL VERTIGO (BPPV)

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ABSTRACT: The most common peripheral vestibular disorder generally is agreed to be BPPV. The hallmark of the disease is brief spells (lasting seconds) of often severe vertigo that are experienced after specific movements of the head.

- The head movements that most commonly cause symptoms are rolling over in bed and extreme posterior extension of the head as if looking under a sink.
- Current understanding of this disease has evolved such that specific therapies based on accepted theories have been developed and proved successful in controlling symptoms.

HISTORY:

- This disorder was first described by Barany in 1921. He documented the various components of this disorder as 1. Nystagmus, 2. Fatiguability of the nystagmus and 3. Vertigo. He failed to correlate the onset of nystagmus with specific positions of the head.
- Dix & Hallpike 1952 described the Dix Hallpike maneuver for eliciting the nystagmus. They also described the unique features of nystagmus accompanying this disorder. These features were 1. Very short latency, 2. Directional features, 3. Brief duration and 4. Reversibility on returning the patient to a seated position.
- Schuknecht postulated that BPPV was caused by loose otoconia from the utricle which in certain positions displaced the cupula of the posterior canal. (Schuknecht theory). He later modified his theory and proposed that it was due to the deposition of otoconia on the cupula of the posterior semi-circular canal. He termed this theory as cupulolithiasis. The cupulolithiasis theory proposes that calcium deposits become embedded on the cupula making the posterior semi-circular canal sensitive to gravity.
- Hall & Ruby suggested that BPPV could result from deflection of the posterior canal cupula caused by debris within the posterior canal. This theory became known as the canal lithiasis theory. In this theory the calcium debris doesnot become adherent to the cupula but float freely within the canal. Head movements like looking up, down, or rolling over to the affected ear may result in the displacement of the sludge causing the classic symptoms.
- Hall & Ruby described 2 types of BPPV: 1. BPPV with a fatiguable nystagmus, where the deposits are freely mobile within the cupula of the posterior canal,
- BPPV with a non-fatiguing nystagmus where the calcium deposits are fixed on the cupula of the posterior canal.

Anatomy and physiology: The vestibular system monitors the motion and position of the head in space by detecting angular and linear acceleration. The 3 semicircular canals in the inner ear detect angular acceleration and are positioned at near right angles to each other (Fig. 1). Each canal is filled with endolymph and has a swelling at the base termed the "ampulla" (Fig. 2).

The ampulla contains the "cupula," a gelatinous mass with the same density as endolymph, which in turn is attached to polarized hair cells. Movement of the cupula by endolymph can cause either a stimulatory or an inhibitory response, depending on the direction of motion and the particular semicircular canal. It should be noted that the cupula forms an impermeable barrier across the lumen of the ampulla; therefore particles within the semicircular canal may only enter and exit via the end with no ampulla.[3]

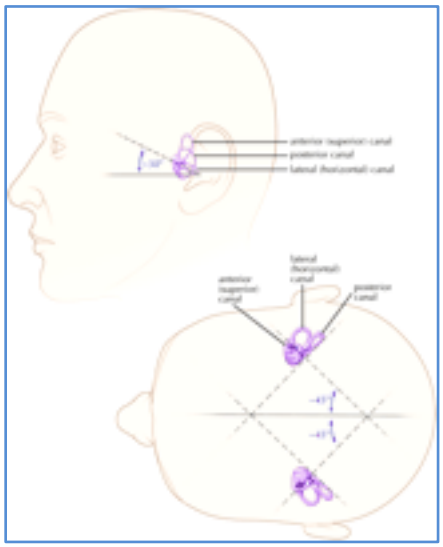


Fig. 1

Fig. 1: Spatial orientation of the semicircular canals. Note how the posterior canal on 1 side is in the same plane as the contralateral superior canal. Both lateral canals are in the same plane, 30o above the horizontal. Photo: Christine Kenney.

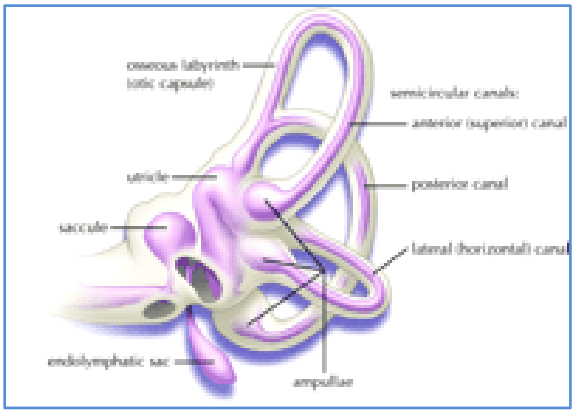


Fig. 2

Fig. 2: Osseous (grey/white) and membranous (lavender) labyrinth of the left inner ear. Perilymph fills the osseous labyrinth external to the membranous labyrinth, whereas endolymph fills the membranous labyrinth. Photo: Christine Kenney.

"Ampullofugal" refers to movement "away" from the ampulla, whereas "ampullopetal" refers to movement "toward" the ampulla (Fig. 3). In the superior and posterior semicircular canals, utriculofugal deflection of the cupula is stimulatory and utriculopetal deflection is inhibitory. The converse is true for the lateral semicircular canal.

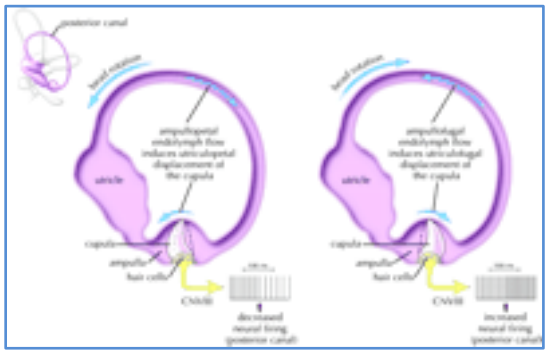


Fig. 3

Fig. 3: Schematic drawing of the physiology of the left posterior semicircular canal.

In the image on the right, note the excitatory response (increased neural firing) with utriculofugal cupular displacement. The same excitatory response would occur in the superior (anterior) canal with utriculofugal cupular displacement, whereas the opposite (inhibitory) response would occur with utriculofugal cupular displacement in the lateral canal. The same rules would apply to the image on the left. CNVIII = vestibular nerve, ms = millisecond. Photo: Christine Kenney

"Nystagmus" refers to the repeated and rhythmic oscillation of the eyes. Stimulation of the semicircular canals most commonly causes "jerk nystagmus," which is characterized by a slow phase (slow movement in 1 direction) followed by a fast phase (rapid return to the original position). The nystagmus is named after the direction of the fast phase. Nystagmus can be horizontal, vertical, oblique, rotatory or any combination thereof. "Geotropic nystagmus" refers to nystagmus beating toward the ground, whereas "apogeotropic nystagmus" refers to nystagmus beating away from the ground.

Mechanism: BPPV can be caused by either canalithiasis or cupulolithiasis and can theoretically affect each of the 3 semicircular canals, although superior canal involvement is exceedingly rare.

Posterior canal BPPV: The vast majority of all BPPV cases are of the posterior canal variant. The pathophysiology that causes most posterior canal BPPV cases is thought to be canalithiasis.

This is probably because most free-floating endolymph debris tends to gravitate to the posterior canal, being the most gravity-dependent part of the vestibular labyrinth in both the upright and supine positions. Once debris enters the posterior canal, the cupular barrier at the shorter, more dependent end of the canal blocks the exit of the debris. Therefore, the debris becomes "trapped" and can only exit at the end without the ampulla (the common crus)

The mechanism by which canalithiasis causes nystagmus in the posterior semicircular canal was described by Epley.[9, 10] Particles must accumulate to a "critical mass" in the dependent portion of the posterior semicircular canal. The canalith mass moves to a more dependent position when the orientation of the semicircular canal is modified in the gravitational plane. The drag thus created must overcome the resistance of the endolymph in the semicircular canal and the elasticity of the cupular barrier in order to deflect the cupula. The time taken for this to occur plus the original inertia of the particles explains the latency seen during the Dix–Hallpike manoeuvre, which is described later.

In the head-hanging position, the canalith mass would move away from the cupula to induce ampullofugal cupular deflection. In the vertical canals, ampullofugal deflection produces an excitatory response. This would cause an abrupt onset of vertigo and the typical "torsional nystagmus" in the plane of the posterior canal. In the left head-hanging position (left posterior canal stimulation), the fast component of the nystagmus beats clockwise as viewed by the examiner. Conversely, the right head-hanging position (right posterior canal stimulation) results in a counter-clockwise nystagmus. These nystagmus profiles correlate with the known neuromuscular pathways that arise from stimulation of the posterior canal ampullary nerves in an animal model.[11]

This nystagmus is of limited duration, because the endolymph drag ceases when the canalith mass reaches the limit of descent and the cupula returns to its neutral position. "Reversal nystagmus" occurs when the patient returns to the upright position; the mass moves in the opposite direction, thus creating a nystagmus in the same plane but the opposite direction. The response is fatiguable, because the particles become dispersed along the canal and become less effective in creating endolymph drag and cupular deflection.

OFFICE TREATMENT OF BPPV: The Epley and Semont Maneuvers

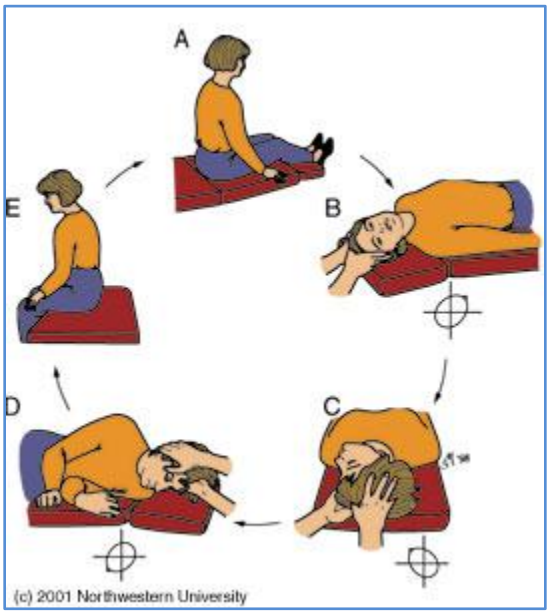
There are two treatments of BPPV that are usually performed in the doctor's office. Both treatments are very effective, with roughly an 80% cure rate, according to a study by Herdman and others (1993). If your doctor is unfamiliar with these treatments, you can find a list of knowledgeable doctors from the Vestibular Disorders Association (VEDA).

The maneuvers, named after their inventors, are both intended to move debris or "ear rocks" out of the sensitive part of the ear (posterior canal) to a less sensitive location. Each maneuver takes about 15 minutes to complete. The Semont maneuver (also called the "liberatory" maneuver) involves a procedure whereby the patient is rapidly moved from lying on one side to lying on the other. It is a brisk maneuver that is not currently favored in the United States.

The Epley maneuver is also called the particle repositioning, canalith repositioning procedure, and modified liberatory maneuver. It is illustrated in figure 2. Click here for an

animation. It involves sequential movement of the head into four positions, staying in each position for roughly 30 seconds. The recurrence rate for BPPV after these maneuvers is about 30 percent at one year, and in some instances a second treatment may be necessary. While some authors advocate use of vibration in the Epley maneuver, we have not found this useful in a study of our patients (Hain et al, 2000).

After either of these maneuvers, you should be prepared to follow the instructions below, which are aimed at reducing the chance that debris might fall back into the sensitive back part of the ear.

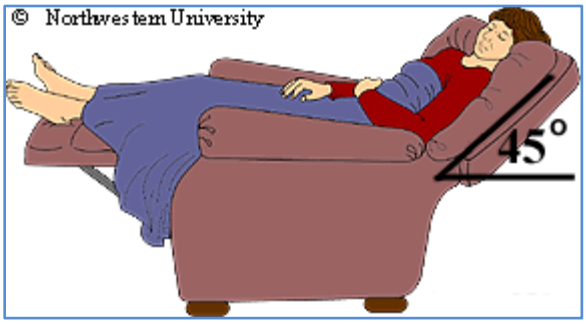


INSTRUCTIONS FOR PATIENTS AFTER OFFICE TREATMENTS (Epley or Semont maneuvers)

1. Wait for 10 minutes after the maneuver is performed before going home. This is to avoid "quick spins," or brief bursts of vertigo as debris repositions itself immediately after the maneuver. Don't drive yourself home.
2. Sleep semi-recumbent for the next two nights. This means sleep with your head halfway between being flat and upright (a 45 degree angle). This is most easily done by using a recliner chair or by using pillows arranged on a couch (see figure 3). During the day, try to keep your head vertical. You must not go to the hairdresser or dentist. No exercise which requires head movement. When men shave under their chins, they should bend their bodies forward in order to keep their head vertical. If eyedrops are required, try to put them in without tilting the head back. Shampoo only under the shower.
3. For at least one week, avoid provoking head positions that might bring BPPV on again.
    - Use two pillows when you sleep.
    - Avoid sleeping on the "bad" side.
    - Don't turn your head far up or far down.

Be careful to avoid head-extended position, in which you are lying on your back, especially with your head turned towards the affected side. This means be cautious at the beauty parlor, dentist's office, and while undergoing minor surgery. Try to stay as upright as possible. Exercises for low-back pain should be stopped for a week. No "sit-ups" should be done for at least one week and no "crawl" swimming. (Breast stroke is OK.) Also avoid far head-forward positions such as might occur in certain exercises (i.e. touching the toes). Do not start doing the Brandt-Daroff exercises immediately or 2 days after the Epley or Semont maneuver, unless specifically instructed otherwise by your health care provider.

4. At one week after treatment, put yourself in the position that usually makes you dizzy. Position yourself cautiously and under conditions in which you can't fall or hurt yourself. Let your doctor know how you did.



Comment: Massoud and Ireland (1996) stated that post-treatment instructions were not necessary. While we respect these authors, at this writing (2002), we still feel it best to follow the procedure recommended by Epley.

WHAT IF THE MANEUVERS DON'T WORK?

These maneuvers are effective in about 80% of patients with BPPV (Herdman et al, 1993). If you are among the other 20 percent, your doctor may wish you to proceed with the Brandt-Daroff exercises, as described below. If a maneuver works but symptoms recur or the response is only partial (about 40% of the time according to Smouha, 1997), another trial of the maneuver might be advised. The "habituation" exercises are also sometimes useful in the situation where all other maneuvers (Epley, Semont, Brandt-Daroff) have been tried -- in essence these consist of a more intense and prolonged series of positional exercises. When all maneuvers have been tried, the diagnosis is clear, and symptoms are still intolerable, surgical management (posterior canal plugging) may be offered.

BPPV often recurs. About 1/3 of patients have a recurrence in the first year after treatment, and by five years, about half of all patients have a recurrence (Hain et al, 2000; Nunez et al; 2000). If BPPV recurs, in our practice we usually retreat with one of the maneuvers above, and then follow this with a once/day set of the Brandt-Daroff exercises.

In some persons, the positional vertigo can be eliminated but imbalance persists. In these persons it may be reasonable to undertake a course of generic vestibular rehabilitation, as they

may still need to compensate for a changed utricular mass or a component of persistent vertigo caused by cupulolithiasis. Fujino et al (1994) reported conventional rehab has some efficacy, even without specific maneuvers.

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