BENIGN PAROXYSMAL POSITIONING VERTIGO: CUPULOLITHIASIS/CANALOLITHIASIS

- Most common cause of peripherally induced vertigo. Second most common cause is vestibular neuritis.

**DEFINITION**

- Benign paroxysmal positioning vertigo is a disorder characterized by brief attacks of vertigo, with associated nystagmus, precipitated by certain changes in head position with respect to gravity.

**AETIOLOGY**

- BPPV occurs due to the inappropriate stimulation of SCC hair cells by sequestered otoconia.
- Otoconia are crystals of calcium carbonate that are normally found embedded in the gelatinous otolithic membranes of the utricle and saccule.
- If free-floating Otoconia find their way into the duct of an SCC (canalolithiasis) or attach themselves to the cupula of an SCC (cupulolithiasis), changes in head position in the plane of that SCC will result in displacement of the cupula, resulting in vertigo and nystagmus in the plane of the stimulated SCC.
- BPPV may occur as a complication of
  - Head trauma
  -Vestibular neuritis
  - Meningitis
  - Cogans syndrome
- F > M, 0.1 % incidence, Elderly
- MC is posterior SCC BPPV.

**CLINICAL MANIFESTATIONS**

- History of brief recurrent episodes of vertigo that occur following certain changes in head position with respect to gravity.
- The most common provocative maneuvers include rolling over in bed, getting in or out of bed, pitching the head forwards while bending over.
- Each episode of vertigo typically lasts 10-20 seconds.
- The vertigo is intense and may be accompanied by nausea and, occasionally, vomiting.
- The characteristic clinical sign of BPPV is nystagmus following a Dix Hallpike maneuver.
- During **DIX'S HALLPIKES** ➔ on making patient lie down (head turned 45 degree and lowered 20 degree) the midpoint of the posterior SCC duct is lowermost and any Otoconia in the duct will therefore move away from the ampulla and come to rest at the midpoint of the duct. The clinician will observe, after a latent period of several seconds, vertical torsional nystagmus, with the quick phases directed upwards and towards the lowermost (affected) ear (as we testing posterior SCC).
- The nystagmus typically lasts for less than 30 seconds and is associated with such intense vertigo that patients are often inclined to shut their eyes.
The clinician must encourage the patient to keep their eyes open and to avoid blinking as much as possible, as the characteristic nystagmus must be observed to confirm the diagnosis.

If the patient is returned from the supine to the upright position, the nystagmus may recur but the direction reverses.

In LSCC – Horizontal nystagmus

In some patients with lateral SCC BPPV, the Otoconia are adherent to the cupula (cupulolithiasis) and both the vertigo and nystagmus persist following a Dix-Hallpike maneuver.

**DIAGNOSIS**

- BPPV is a clinical diagnosis
- Most patients with BPPV will have no abnormalities on vestibular and auditory function tests.

**DIFFERENTIAL DIAGNOSIS**

- Migrainous vertigo – young and if asso with other migranous symptoms.
- Rarely posterior fossa tumour, malformation or degenerative condition.

**MANAGEMENT OPTIONS**

- BPPV can be effectively treated by relocating Otoconia from the SCC duct into the vestibule using the **EPLEY MANOEUVRE**
- Aka **CRP = Canalith Repositioning Maneuver**
- C/I in neck disease, severe carotid stenosis.
- Best immediately after dix hallpike’s.
- Lateral SCC BPPV does not respond as well to the Epley manoeuvre as does posterior BPPV.
- The patient should be instructed to remain upright for 24 hours after the treatment and to avoid sleeping on the affected side for the following week, to reduce the likelihood of Otoconia finding their way back into the duct of the posterior SCC.
- In the patient with severe, intractable symptoms that do not respond to repeated manoeuvres, surgical occlusion of the posterior see can be highly effective in relieving symptoms.
- Alternatively, division of the posterior ampullary nerve, a technically difficult procedure, may be considered.

![Diagram of the Epley repositioning maneuver for left posterior SCC BPPV](image-url)

*Figure 240c.10 The Epley particle repositioning maneuver for left posterior SCC BPPV. The patient is rapidly reclined into the left Dix–Hallpike position (a) and remains in that position until both the vertigo and nystagmus have totally disappeared and the otocional particles have settled into the lowest portion of the anterior SCC duct. The patient’s head is slowly turned by 90° into the right Dix–Hallpike position (b–f), so that the particles are guided into the crus communis. Then the patient slowly rolls on to the right shoulder (g) and the head is turned another 90° so that the particles fall via the crus communis back into the vestibule. The maneuver is completed by sitting the patient upright. Adapted from Klima-Moldawer GM, Cremen SE. Assessment and treatment of dizziness.*
MODIFIED EPLEY’S FOR SELF-TREATMENT

BRANDT DAROFF POSITIONAL EXERCISES

SEMONT’S LIBERATORY MANOEUVRE

FACTORS INFLUENCING OUTCOME

- BPPV due to head injury – Poor prognosis
- BPPV secondary to Vestibular Neuritis – Better prognosis
- Posterior BPPV can become Horizontal or anterior BPPV.

SURGICAL TREATMENT

- <1 % patients with BPPV may require surgery
- Psce Occlusion Or Singular Neurectomy

SPECIFIC TREATMENT FOR hBPPV

- Forced prolonged position on the healthy side (12 hrs lying on healthy side)
- 270 degree barbecue maneuver
- 360 yaw rotation
- Liberatory Maneuvers

SPECIFIC TREATMENT FOR aBPPV

- Very rare
- Like Dix Hallpikes only
- First, sit the patient on an examination couch with the head rotated 30° toward the unaffected side; second, take her/him backwards to ahead-hanging position while keeping the head turned 30° to the side and wait for 30 seconds; third, sit her/him up again.