

An Overview of Benign Paroxysmal Positional Vertigo



Jason A. Beyea, MD, PhD; and Lorne S. Parnes, MD, FRCSC

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Benign paroxysmal positional vertigo (BPPV) is the most common disorder of the peripheral vestibular system, with the most frequent age of onset between the fifth and seventh decades of life. Overall lifetime prevalence is reported as 2.4% (3.2% for females and 1.6% for males).¹ However, the true incidence is difficult to determine as most cases spontaneously resolve within months. BPPV was first described in 1921 by Bárány, through the report of a patient who demonstrated rotatory nystagmus to the right when she laid on her right side, accompanied by vertigo and nausea, lasting about thirty seconds.

Causes of BPPV

Primary (Idiopathic) (50% to 70%)

Secondary (30% to 50%)

- Head trauma (7% to 17%)
- Viral labyrinthitis (15%)
- Ménière's disease (5%)
- Migraines (< 5%)
- Inner ear surgery (< 1%)

Pathophysiology

Most cases of BPPV typically arise in one of the posterior semicircular canals (SCC), and this

Mary's case

Mary, 71, presented to her family physician with episodes of vertigo. She described nightly episodes during the previous month. An episode of vertigo was brought on by rolling over in bed to the right. The vertigo lasted about 20 seconds each time, and was accompanied by nausea. After an attack, she had a feeling of uneasiness for about two minutes. She did not have a history of otologic, vestibular, or neurological disease, or head trauma. The Dix-Hallpike manoeuvre (figure 1) was performed, which was negative on the left but positive on the right (rotatory counter-clockwise nystagmus). She was diagnosed with right posterior semicircular canal BPPV. A particle repositioning manoeuvre (figure 2) was performed. At her one month follow up visit, she claims to have been vertigo free since the manoeuvre at her last visit.

will be the focus of this manuscript. The posterior canal is most affected because it is the most gravity dependent part of the inner ear. The understanding of BPPV is evolving, with the canalithiasis theory prevailing at present. This postulates that free-floating debris/endolymph particles (probably dislodged and displaced otoliths from the utricular otolithic membrane) migrate down into and become "trapped" in the posterior SCC. When the orientation of the canal is moved in the gravitational plane, the



Figure 1. The Dix-Hallpike manoeuvre (illustrations: Christine Kenney; Reprinted with permission).

resulting inertial changes in the canal's perilymph and cupula gives rise to the observed rotatory nystagmus and vertigo.

Diagnosis

History

Patients describe sudden severe vertigo, precipitated by particular head positions and movements. Typical movements are rolling over in bed, looking up, and bending forward. Associated symptoms include lightheadedness, nausea, and imbalance. Although most BPPV has no identifiable cause, a complete history should include a search for secondary BPPV causes.

Physical exam

The primary diagnostic manoeuvre for BPPV is the Dix-Hallpike manoeuvre (Figure 1). The patient is seated so that the head will extend over the edge of the table when supine. The head is rotated 45°

toward the ear being tested which brings that posterior SSC into the anterior/posterior axis and the contralateral posterior SCC into the transverse axis (A). The examiner lowers the patient to the supine position, with the patient's head hanging 30° over the edge of the table. Eyes are observed for nystagmus, and the patient is questioned about feeling vertiginous (B). After a minute or two, the patient is then



Dr. Beyea is a Resident, Department of Otolaryngology, Schulich School of Medicine and Dentistry, University of Western Ontario, London, Ontario



Dr. Parnes is a Professor, Department of Otolaryngology and Neurological Sciences Schulich School of Medicine and Dentistry of Western Ontario, London, Ontario

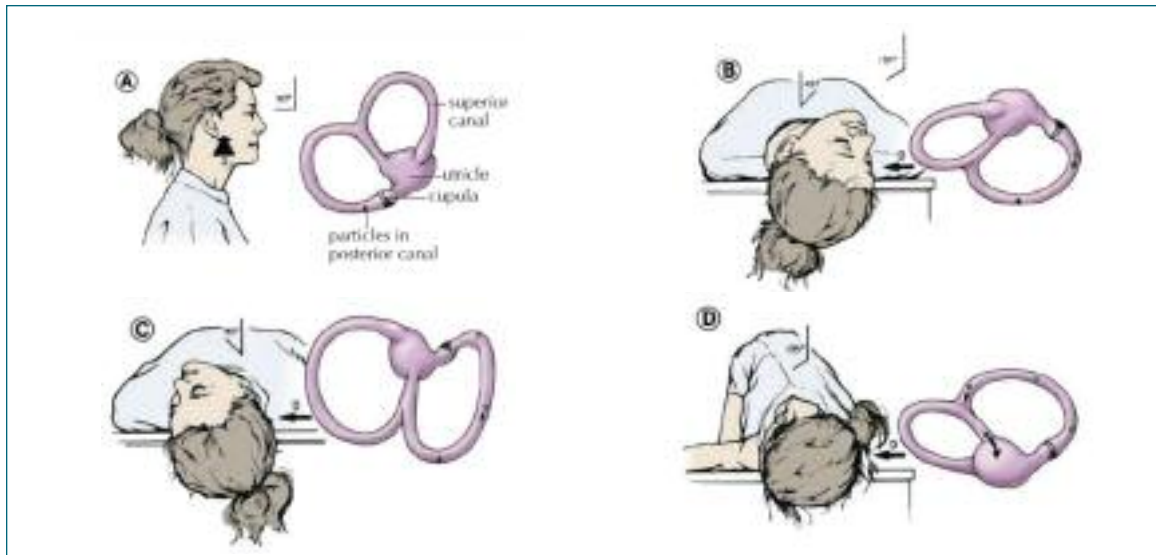


Figure 2. The Particle Repositioning Manoeuvre (illustrations: Christine Kenney; Reprinted with permission).

returned to the seated position (as in A), and the eyes are observed again for nystagmus. In a positive response during the head back position, the fast phase of the nystagmus has the top of the globe beating toward the ground. In Figure 1, the patient's right side is being tested so the physician would expect to see counter-clockwise rotatory nystagmus when the patient is supine. When the patient is returned to the seated position, the opposite endolymph flow and cupular displacement occurs in that same posterior SCC resulting in a clockwise rotatory nystagmus. The exact converse occurs if the particles/debris are in the left posterior SCC, that is clockwise rotatory nystagmus in the head back position converting to a counter-clockwise nystagmus after sitting back up.

Management

Primary management for BPPV is the particle repositioning manoeuvre (PRM). Figure 2 shows the important head positions for a right

posterior SCC PRM and a cartoon of the corresponding inner ear and moving particles. D marks the angle of view of the inner ear in that particular position. For a left-sided posterior SCC PRM, all figures and movements would be mirror images. The key issue in understanding the manoeuvre is that the cupola forms a barrier to the movement of the particle so that the only way the particles can get in and out of the posterior SCC is through the opposite end of the canal where it merges with the superior SCC (the so-called crus-communis).

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The patient is seated on the table (A). The head is turned 45° to the right side of the mid-


- BPPV is a benign disease, and most cases resolve spontaneously.
- Over 80% of patients will be controlled with one particle repositioning manoeuvre.
- Cases that are atypical, or do not respond to the particle repositioning manoeuvre, warrant referral to a tertiary dizziness clinic.

line. The patient is then lowered into the supine position, with the head hanging 30° over the table edge (B), and observed for nystagmus. Position B is maintained for 30 to 60 seconds. The head is then rotated to 45° left of the midline, maintaining the neck in 30° extension (C), and the motion is continued through into position D. The transition from position B to D should take no longer than 3 to 5 seconds. Position D is maintained for 30 to 60 seconds and the eyes are again observed for nystagmus. The patient is then asked to sit up, to position A, and the eyes are again observed for nystagmus. The patient should remain seated for at least 5 minutes, as sometimes there is a delayed vertigo response. There are no specific post maneuver instructions/restrictions.

Counselling, medications and follow-up

Clinicians should counsel their patients regarding BPPV and its impact on their personal safety. For example, a tree trimmer or house painter should not work until their symptoms have completely resolved. Vestibular suppressant medications (antihistamines, benzodiazepines) are not effective. The patient should be followed up in clinic in one month's time to assess for symptom resolution.

When to refer?

One properly done PRM controls 83.8% of cases². Atypical cases, such as horizontal canal BPPV, which causes horizontal nystagmus with supine lateral head turns, or cases that do not respond to the PRM should be referred to a tertiary Otolaryngology – Head and Neck Surgery Dizziness clinic. For intractable cases of BPPV not responsive to the PRM, surgical transmastoid posterior SCC occlusion is very safe and highly effective. 

References

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Resources

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