Approach to Benign Paroxysmal Positional Vertigo

Sherin Sha S

BPPV is the commonest presenting cause of vertigo with an estimated lifetime prevalence of 2.4%. It has a characteristic history and can easily be diagnosed on examination. Treatment can be performed in the clinic with a good outcome, making it the most rewarding vestibular condition to manage.

The first clinical description of positional vertigo is attributed to Barany in 1921 and in 1952 Dix and Hallpike were the first to clearly describe the provoking manoeuvres. Dix and Hallpike coined the term 'benign paroxysmal positional vertigo' in view of the associated benign (non-cancerous) origin and momentary (paroxysmal) bursts of intense vertigo upon head movements (positional).

Pathophysiology

Otoconia are calcium carbonate crystals embedded in the macula of the utricle and saccule. They have a greater density than the surrounding endolymph thus making the macula sensitive to changes in linear acceleration and, importantly, gravity. The semicircular canals, on the other hand, are sensitive to changes in angular acceleration. [Fig. 1]

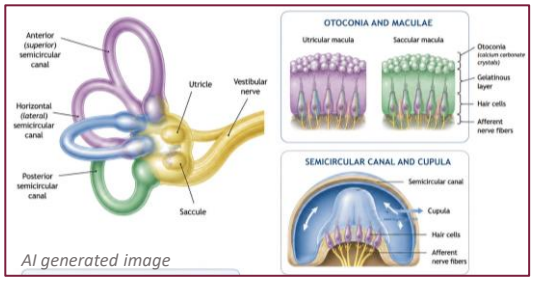

Fig. 1 Vestibular System

[1]Government Medical College, Alappuzha
Address for correspondence : Dr Sherin Sha S MBBS, MS ENT, International Vestibular Rehabilitation Certificate(The American Institute of Balance), AMVD(Assessment and Management of Vestibular Diseases)-Yenepoya University, Assistant professor of ENT, GTDMC, Alappuzha.
Email: sherinshas@gmail.com

In BPPV otoconia from the utricle are thought to collect in the semicircular canals, making them abnormally gravity-sensitive. The net result is that changes in head position with respect to gravity result in an abnormal displacement of the cupula and stimulation of the corresponding vestibular afferents. This results in the characteristic abnormal eye movements and vertigo. There are two theories of how this might occur.

Cupulolithiasis proposes that degenerative otoconia stick to the cupula making it gravity-sensitive. Canalolithiasis theory suggests that degenerative otoconial debris float freely in the endolymph of the semicircular canal. When exposed to gravity, the otoconia fall to the lowest part of the canal, causing a change in endolymph pressure with subsequent displacement of the cupula. [Fig. 2]

BPPV most commonly arises from the posterior semicircular canal (p-SCC). Much less frequently the horizontal semicircular canal (h-SCC) is involved while the anterior canal (a-SCC) is very rarely affected. The propensity for the accumulation of particles in the posterior canal is postulated to be related to anatomical factors such as the size of the common crus of the posterior

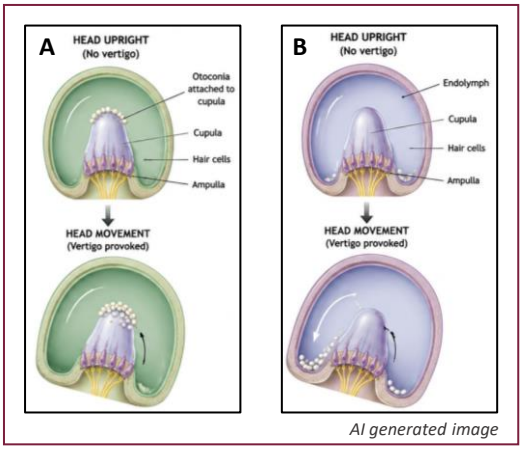

Fig 2 A Cupulolithiasis, B Canalolithiasis

and superior semicircular canals, its position below the utricle when supine and its dependent position when both erect and supine.

The hallmark of p-BPPV is vertigo lasting seconds with or without nausea and imbalance on lying down, sitting up from the lying position, or rolling in bed and when extending or flexing the neck. These symptoms can present in clusters with several attacks per day. In between attacks or shortly after successful treatment, patients are either symptom-free or experience a sensation of imbalance.

Risk factors that may initiate an acute episode of BPPV include prolonged bed rest, bending forward with the head down, and general anaesthesia, because the supine, head-down and head reclined position (e.g. during intubation) lower the opening of the posterior canal, thus promoting the penetration of particles.

Diagnosis of BPPV

Diagnosis of BPPV is made on the basis of typical signs (nystagmus) and symptoms (vertigo and nausea) provoked by specific positional tests. Understanding the characteristic eye movements during these tests will help in making the diagnosis.

Dix–Hallpike test to diagnose p-BPPV

The Dix–Hallpike test is the most important diagnostic manoeuvre for BPPV because posterior canal BPPV is the most common subtype and this test can identify the majority of affected patients. [Fig. 3]

The patient is seated on the examination couch, feet up, and the head is turned 45 degrees towards the side being tested, aligning the vertical canals with the sagittal plane. The patient is rapidly brought into the head-hanging position over the end of the couch to lie 30 degrees below the horizontal while maintaining a position 45 degrees to the side being tested. Patients should be counselled before the test about dizziness and advised to keep their eyes open for examination.

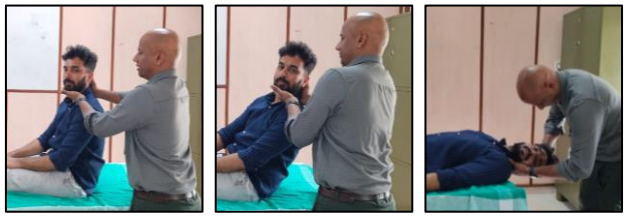

Fig. 3 Dix–Hallpike test $

The test is positive if the patient experiences vertigo and exhibits specific eye movements (nystagmus). This nystagmus typically has a latency of 5–20 seconds and is transient (lasts less than 60 seconds).

Supine roll test to diagnose h-BPPV

The patient is placed in the supine position with the head flexed 30 degrees, bringing the horizontal canal into the axial plane, and the head is then briskly rolled to one side. The same is repeated to the opposite side. In the majority of cases, the nystagmus will be horizontal and geotropic (towards the ground) and towards the ear being tested. When turned to the opposite side, the nystagmus will reverse and beat towards the under most ear again.

Straight head-hanging manoeuvre to diagnose a-BPPV

The patient is moved from the sitting position to a supine head-hanging position with the head extending 30–40 degrees below the horizontal beyond the edge of the examination couch. The position is maintained while the examiner observes for vertigo and characteristic down-beating torsional nystagmus, which is suggestive of anterior canal BPPV. [Fig. 5]

Fig. 5 Straight Head-hanging Manoeuvre $

Treatment

Treatment of p-BPPV

Epley's repositioning manoeuvre

It is considered the most effective treatment manoeuvre for posterior canal BPPV. The patient is initially seated upright with the head turned 45 degrees towards the affected side. The patient is then rapidly brought into the Dix–Hallpike position with the head hanging 30^0 below the horizontal and maintained for 30–60 seconds. The head is subsequently rotated 90^0 towards the unaffected side and held for another 30–60 seconds, following which the patient's body is

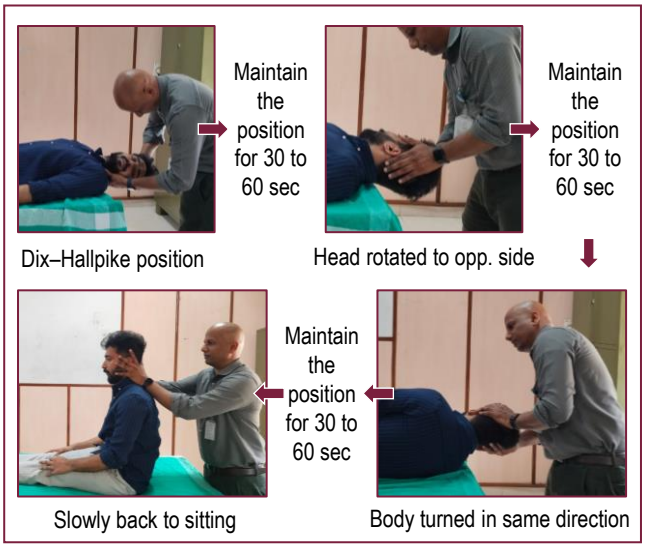

Fig. 6 Epley's repositioning manoeuvre $

turned further in the same direction into the lateral decubitus position with the nose facing downwards. After maintaining this position for 30–60 seconds, the patient is slowly brought back to the sitting position. The manoeuvre facilitates the movement of otoconial debris from the posterior semicircular canal into the vestibule by gravity.

Treatment of h-BPPV

Forced prolonged position on the healthy side

The patient is instructed to lie on the healthy side for 12 hours, facilitating gravitational movement of the debris into the vestibule by maintaining the affected horizontal semicircular canal uppermost.

270-degree 'barbecue' manoeuvre

This consists of turning the patient's head initially and then the body from the supine position in three 90-degree-step rotations (total 270 degrees) towards the unaffected ear. The body will eventually assume the prone position with the affected ear facing down, following which the patient will sit up. The rotation is performed within half a second and the head positions are maintained for 30–60 seconds.

Treatment of a-BPPV

Yacovino manoeuvre

The patient is moved from a seated to a head-hanging position with the head 30 degrees below the horizontal. After 30 seconds, the head is brought to a chin-to-chest position for 30 seconds and then to a seated position for another 30 seconds. [Fig. 7]

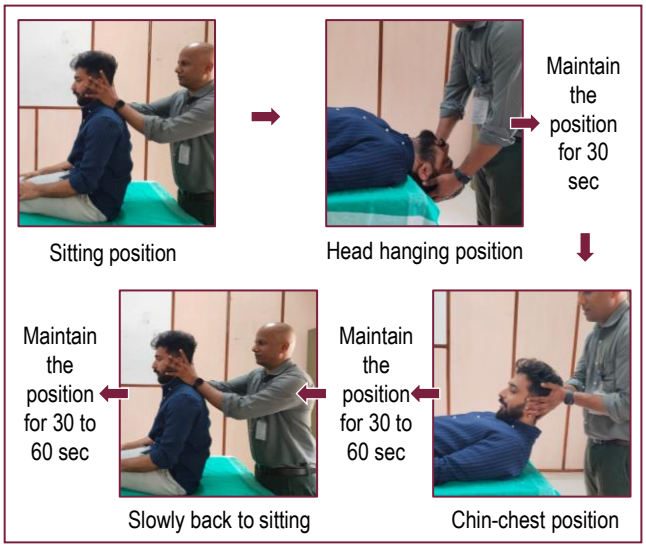

Fig. 7 Yacovino manoeuvre $

Role of imaging

Imaging of the posterior fossa is required in one of the following situations.

- Nystagmus is atypical for any of the BPPV syndromes
- Brainstem or cerebellar signs are present
- Positional vertigo does not resolve with repeated therapeutic manoeuvres

Conclusion

Benign Paroxysmal Positional Vertigo is the commonest cause of peripheral vertigo. If unrecognized or poorly treated, BPPV can lead to significant morbidity. It results from particles floating in the semicircular canals (canalolithiasis) or attached to the cupula (cupulolithiasis), with the posterior semicircular canal being most commonly affected. The vertigo is typically positional and lasts for a few seconds, and the majority of cases are idiopathic. Diagnosis is made using positional tests that produce characteristic nystagmus and vertigo, among which the Dix–Hallpike test remains the most effective diagnostic manoeuvre. Repositioning manoeuvres displace the particles back into the vestibule and are successful in the majority of cases, with Epley's manoeuvre being the most effective treatment manoeuvre for posterior canal BPPV. From a rehabilitation perspective, untreated BPPV can significantly impair balance, mobility, gait confidence, and increase fall risk, especially in older adults. Early recognition and appropriate repositioning manoeuvres can rapidly restore function and improve quality of life, while patients with persistent imbalance may benefit from vestibular rehabilitation and balance retraining.

References

1. Dix MR, Hallpike CS. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. Ann Otol Rhinol Laryngol. 1952;61:987-1016.
2. Bárány R. Diagnose von Krankheitserscheinungen im Bereiche des Otolithenapparates. Acta Otolaryngol. 1921;2:434-437.
3. von Brevern M, Radtke A, Lezius F, et al. Epidemiology of benign paroxysmal positional vertigo: a population-based study. J Neurol Neurosurg Psychiatry. 2007;78:710-715.
4. McClure JA. Horizontal canal BPV. J Otolaryngol. 1985;14:30-35.
5. Casani AP, Vannucci G, Fattori B, Berrettini S. The treatment of horizontal canal positional vertigo: our experience in 66 cases. Laryngoscope. 2002;112:172-178.

$ *image courtesy : Dr Sonu Mohan, Asst Professor, Dr Amal Chandran, Junior Resident Govt Medical College, Trivandrum*