Evaluation & Management of Benign Paroxysmal Positional Vertigo (BPPV) : Review of Guidelines with Updated Summary

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ABSTRACT

Benign Paroxysmal positional vertigo (BPPV) is one of the common diagnoses encountered by an otolaryngologist. Because the symptoms and presentations of BPPV closely resemble those of central lesions, including brain tumors, diagnostic pitfalls and mistakes of judgment may occur during diagnosis. A review on position statements about the evaluation, diagnosis and management of BPPV is presented here along with a synopsis on each topic.

Keywords: Benign Paroxysmal Positional Vertigo, BPPV

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Benign Paroxysmal Positional Vertigo (BPPV), was first described by Barany in 1921; with a life time prevalence of 2.4% it is the most common cause of vertigo constituting about 1/3rd of the total cases.

The episodes of intense spinning sensation associated with repeated vomiting triggered by change in head position in the classical presentation are so dramatic and frightening for the patient that it is not uncommon to see active agile individuals being reduced to nervous wrecks paranoid even to move. Gone are the days when prolonged drug treatment to pacify and suppress the angry inner ear was the standard treatment; the therapeutic maneuvers now available for BPPV can completely cure the patient without the need for prolonged treatment regimens or fancy surgeries. So it is of vital importance that the patient is diagnosed early and appropriate treatment started for his benefit.

Though very common, diagnostic pitfalls and errors of judgment may arise in evaluation because symptoms and presentations of BPPV closely mimic those of central lesions which includes intracranial tumors. Position statements related to evaluation, diagnosis and management of BPPV are presented below with a summary of discussion on each.

a. Dizzy spells on change in head position are NOT always can be classified as positional vertigo

- The common perception is that a history of triggering vertigo with change in position of the head is sufficient to make an assumption of positional vertigo, be it BPPV or central origin. However, on deeper analysis it should be clear that worsening of vertigo on head movements can occur in any vestibular disorder by virtue of the vestibular system being the prime organ destined to perceive linear/ angular acceleration and direction of gravity. The fact is that to make a diagnosis of typical positional vertigo there should be a reorientation of the head with respect to the gravity vector. For example, lying down from standing or sitting position reorients the head with gravity, and should

signify BPPV; this is also applicable to turning over in bed, sitting up after lying down, extending neck to look up or bending forward.[1]

- However, this cannot be spoken of about standing up from sitting, since orientation of the head remains unchanged with respect to gravity. Here, a diagnosis of orthostatic hypotension or neurological lesions causing gait disturbances need be entertained rather than positional vertigo.

b. Typical history may not be elicited in every patient; there are alternate presentations for BPPV

- Typically, an attack of BPPV lasts for 5-20 seconds only, and never longer than a minute. However, after paroxysms of recurrent positional vertigo, history given by the patient would be prolonged dizziness and imbalance lasting for days. The Importance of positional nature of vertigo may not be recognized by the patient and that history may not be volunteered; hence, leading questions may be necessary.
- Though the illusion of body movement is usually rotary it can also be felt as body tilt, imbalance or oscillopsia.
- It is not uncommon that the only presentation may be nausea and repeated vomiting without any vertigo.
- Since the trigger of a dizzy spell is head movement patients may also adopt compensatory postures to minimize head movements like holding the neck stiff which might as well be a presentation.
- By definition BPPV is not associated with auditory or neurological symptoms; however, the paroxysms of severe vertigo may create strong anxiety or phobic behavior which may mimic such symptoms and has to be tactfully recognized.

c. Converse to statement b, typical positional vertigo need not always signify BPPV

- Positional vertigo can be caused by central vestibular disorders like cerebellar infarction, brainstem tumors and degenerative diseases of the CNS. The symptoms include positional vertigo, nausea, vomiting or oscillopsia which can occur in isolation or in combination. There may be additional complaints like diplopia, dysphagia, dysarthria or incoordination.
- Moreover, patients with other vestibular lesions like vestibular neuritis, Meniere's disease and migraine may develop BPPV due to the damage induced by these primary conditions on the labyrinth. Diagnosis and treatment of the associated BPPV certainly result in better symptomatic improvement during management of these conditions..

d. Conditions other than BPPV presenting as positional vertigo are not uncommon

- Vestibular migraine (VM) is the most common cause of recurrent spontaneous vertigo and is the second most common vestibular lesion after BPPV. Vestibular disturbance in migraine may be varied and include recurrent spontaneous vertigo, positional vertigo or head motion discomfort provoked by head movements. These different presentations can occur in isolation, simultaneously or sequentially. Attacks lasting for hours or days may manifest initially as spontaneous spinning vertigo, changing in course of time to pure positional vertigo aggravated by head movement, followed by head motion intolerance and ataxia dying off eventually. There may be other vestibular symptoms like nausea, vomiting and imbalance. Only a few have migraine headaches as a regular accompaniment of the vertigo while others have attacks with or without headaches and some have never experienced the symptoms together. Interestingly cochlear symptoms like hearing loss and tinnitus are present in 10- 40% of patients with VM.[2]
- As explained earlier any vestibular lesion gets worse on head movements; vertigo aggravated by movements of the head can be a feature of vestibular neuritis (neurolabyrinthitis) which is the second most common peripheral vestibular lesion after BPPV. There may be associated canalolithiasis due to inner ear damage caused by the primary disease with resultant BPPV coexisting with these conditions.
- Other conditions which present as positional vertigo includes Perilymph fistula, alcohol induced positional vertigo, head extension vertigo due to cervical osteophytes and vertebral artery occlusion which are not uncommon.
- Pure central lesions causing positional vertigo are relatively rare and includes structural lesions (including tumors) of the pontomedullary brainstem or the caudal cerebellum; these can become life threatening and should be considered in the differential diagnosis of all positional vertigo.[3]

e. The gold-standard in diagnosis of BPPV is positional testing

- Despite advances in modern technology the only definitive method to make a diagnosis of BPPV is by doing a positional test. Initial evaluation is always by the Dix-Hallpike test since it evaluates the posterior semicircular canal which is the commonest canal involved. The purpose is to provoke vertigo and observe the type of nystagmus in the patient to arrive at a diagnosis. The importance is not just diagnosis of BPPV but also ruling out central lesions which has a greater significance from the patient perspective.[4]

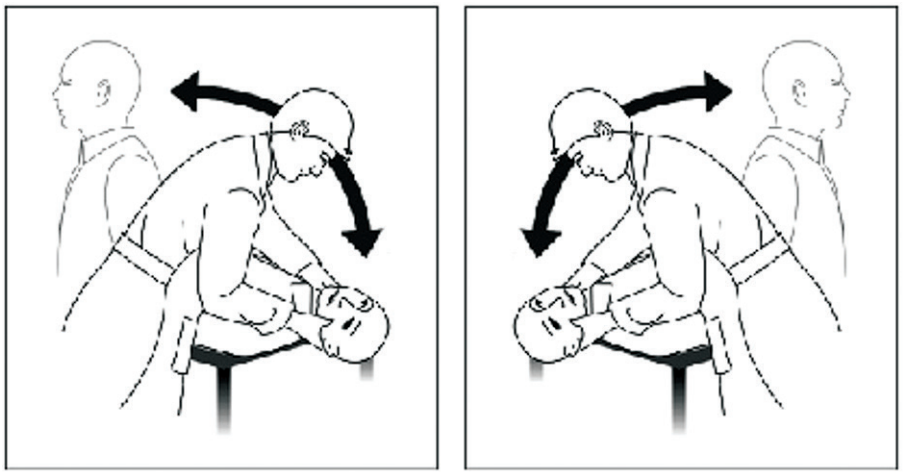

Figure 1. Dix-Hallpike test

f. Added positional testing should be chosen depending on the semicircular canal involved

- The diagnosis of BPPV is confirmed by observing the typical torsional nystagmus after Dix- Hallpike test where the posterior canal is rotated in a plane parallel to gravity **(Figure 1).** Diagnosis of BPPV can be established if the nystagmus thus induced appears only after a latent period, is torsional, geotropic (directed towards the ground), short lasting, and fatigable on repeated testing. There is reversal in the direction of nystagmus when the patient is returned to the upright posture from supine. Immediate onset, persistent nystagmus with Hallpike test especially if purely vertical signifies a central lesion which has to be investigated accordingly.
- Torsional nystagmus signifies BPPV due to stimulation of the posterior canal (pc- BPPV). However, sometimes during Hallpike test a pure horizontal nystagmus is observed, when it signifies canalolithiasis involving the horizontal canal (hc-BPPV).
- Diagnosis of hc-BPPV can be established by the Supine head roll test **(Figure 2).** A horizontal nystagmus after a latent period, which is fatigable and beating in the direction of the abnormal side is diagnostic of hc- BPPV after a Roll test. When the head is rotated 180 degrees to the opposite side change in direction of the positional nystagmus is observed.
- For testing anterior canal BPPV which is very rare, Hallpike test with the head placed as low down as possible may be done. In the left head hanging position Hallpike test stimulates the right anterior canal and vice versa due to the co planar orientation left anterior and right posterior canals and hence results have to be interpreted accordingly.

g. Special precautions are necessary for best results while doing Hallpike test

- As mentioned, positional tests provoke vertigo; it is unpleasant and for best results has to be explained beforehand to the patient to gain his confidence.
- The patient should be warned that even if he feels vertiginous, he should not close the eye and should look at one point on the examiner's face.

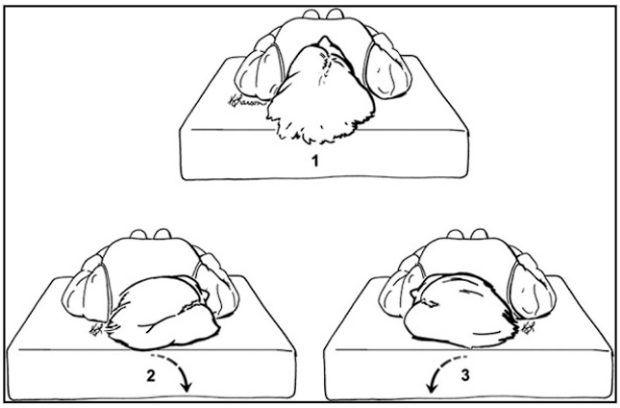

Figure 2. Supine Head Roll test (for hc-BPPV)

- If the eyes are not in the primary gaze and wandering around, observation of nystagmus is difficult. The examiner can use his free hand to keep at least one eye of the patient wide open since the intensity of vertigo may provoke the patient to close the eye or sit up.
- The patient has to be maintained in the head down position for some time; though in most of the cases the nystagmus is observed after a latent period of 5-10 seconds, some cases of canalolithiasis exhibit extremely long latencies (up to 20-30 seconds). If the patient is made to sit up before that interpretation of results may go wrong altogether.
- Hallpike test and the treatment maneuvers for BPPV involves some neck strain; it may be extremely difficult or even impossible to perform this in patients with cervical spine pathology like severe spondylosis. In such a situation the best option is to perform this on a couch where the upper half of the body can be lowered by 20- 30 degrees which obviates the constraint of head rotation.
- Care should also be exercised in patients with cervical stenosis, severe kyphoscoliosis, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, ankylosing spondylitis, spinal cord injuries, and morbid obesity.
- In patients who are obese it may be difficult for a single examiner to fully support the head through the maneuver, and additional assistance may be required.

h. Induced nystagmus after positional test need not always be of short duration in BPPV; it may last longer in some situations

- Although most cases of BPPV are due to Canalolithiasis (freely mobile otoconia within the lumen of semicircular canal), Cupulolithiasis is a form of BPPV due to otoconia getting attached to the cupula of semicircular canals. Positional testing in these patients results in nystagmus that may persist for >1 minute.

i. Video-Nystagmography (VNG) need not be routinely used for evaluation of nystagmus in Hallpike positional testing

- Nystagmus in the commoner posterior canal variant of BPPV is very strong, well noticeable and torsional, a plane in which suppression of visual fixation may not be very effective. VNG hence offer no added advantage.
- It may be of some benefit in cases of BPPV where the nystagmus characteristics are suggestive but not very clear, since better interpretation is possible after recording. Patients with horizontal canal variant may also benefit since it produces horizontal nystagmus which is aggravated after visual suppression. However, as we are aware, the horizontal canal variant is less common accounting for only less than 20% of BPPV.

j. Positional testing is needed even for evaluation of vertigo other than BPPV

- There is no vestibular disorder where a positional testing is purely worthless! BPPV can be ruled out with absence of findings or atypical findings on a Hallpike testing.
- Presence of immediate onset persistent nystagmus on positional testing points to a grave condition like cerebellar infarction, degenerative brain disease or tumors.
- This may also be an incidental finding in clinical examination of a patient without any positional symptoms but known neurological disease.
- As discussed earlier even other common vestibular disorders like Meniere's disease, migraine and vestibular neuritis may be associated with BPPV due to labyrinthine degeneration diagnosis of which needs positional testing.

k. Negative positional testing does not conclusively rule out BPPV

- The patient may experience vertigo during Hallpike test, but there is no nystagmus. In such cases it is possible that only minimal otoconial debris was present within the canal and patient has spontaneous remission.
- Minimal debris may also not be recognized on positional testing especially if the patient is uncooperative. A therapeutic maneuver on the symptomatic side is often found to be beneficial in such patients.
- Alternatively, the fallacies in execution of a Hallpike test can also be responsible; the eye may not be in

the primary position of gaze to evoke nystagmus or adequate latent period was not applied before looking for nystagmus.

- Speed of head movement and angle of head turn while testing does have a bearing on interpretation of Hallpike test.
- Sometimes the positional test may be negative when a clot of debris may be blocking the canal after getting lodged somewhere within. Particles may also be sometimes adherent to the canal wall. It may be worthwhile to try gentle head percussion to dislodge the clot and repeat the testing after some time.
- Alternatively, this may be due to movement of the clot next to the canal wall which prevents relevant endolymph shifts while the particles are moving. Repeated testing is then likely to provoke nystagmus and vertigo.
- The canalolithiasis may also be on a different canal than the one being tested; for eg. If Hallpike test is negative, try supine head roll testing for the horizontal canal.

I. No imaging is required in patients with typical BPPV

- In patients with typical BPPV no imaging is necessary since the diagnosis is straight forward especially if posterior canal BPPV.
- Even when the BPPV is atypical, for eg. horizontal canal origin, if associated with typical nystagmus without added neurological deficit imaging need not be advocated immediately. A trial of treatment with repositioning maneuver is prudent before considering imaging.

m. Patients with positional vertigo should be advised imaging in the following situations

- Atypical positional vertigo associated with abnormal nystagmus and focal neurological deficit definitely need imaging.
- Atypical nystagmus even with a typical history is an indication for cerebral MRI, for eg. ageotropic nystagmus with Horizontal canal BPPV.
- Associated Neurological symptoms like headache (especially recent onset), projectile vomiting, diplopia, facial paresthesia and dysarthria also merit imaging.
- MRI brain should also be advised in cases of positional vertigo not resolving with repeated therapeutic maneuvers.

[It would be worthwhile to remember that about half of the atypical cases of BPPV are of central origin and of them 3-5% intracranial tumors]

n. Normal imaging need not conclusively rule out central vestibular lesions

- Normal brain imaging should not be a yardstick to confirm diagnosis of BPPV. A patient with central positioning nystagmus with normal imaging can be due to Vestibular migraine or drug effects like Amiodarone and alcohol. Vestibular migraine is extremely common as detailed earlier. Positional vertigo related to migraine tends to occur earlier in life and is usually associated with migraine symptoms during the attack. There may be a history of vertigo being precipitated by migraine triggers as well. Response to antimigraine medications clinches the diagnosis in this situation.

o. Canalith Repositioning Procedures (CRP) are the mainstay of treatment in most of the cases of BPPV

- These maneuvers work directly on liberating the adhered otoconia on the cupula and/or by moving free-floating otoconia out of the involved semicircular canal and back into the vestibule.
- Epley and Semont maneuvers **(Figure 3)** are the therapeutic options in posterior canal BPPV which induce the return of the otoconia from the posterior semicircular canal back to the utricle.[4]
- The Epley maneuver involves the rotation of the posterior canal backwards close to its planar orientation by a series of successive head positioning, each of about 90-degree displacement.
- The frequent cause of failure in Epley maneuver is insufficient reclination of the head of the patient during transition between one head hanging position to the other. Treatment failures may also be due to the particles sticking on to the cupula converting into cupulolithiasis. Gentle head percussion, vibration or head shakes dislodges the otoconia from there.
- The Semont maneuver is a rapid 180-degree swing of the head in the plane of the posterior canal. The patient is brought from the ear down position on one

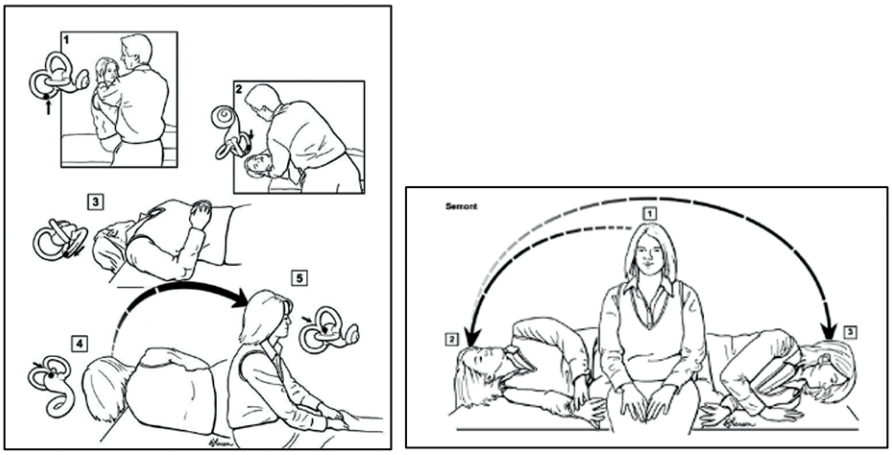

Figure 3. Supine Head Roll test (for hc-BPPV)

side to the eye down position on the other. Frequent cause of failure is insufficient head rotation and slow execution of the swing.[5]

- Both the maneuvers are highly effective in BPPV when performed properly achieving success rates as high as 75% for even a single maneuver, which increases to 90% if repeated. If there is treatment failure with one maneuver the other on can be tried with definite chances of success.
- Sometimes the cause for failure may be conversion of pc-BPPV to another type say hc-BPPV due to accidental shifting of the particles during the maneuver into the horizontal canal. In such situation the specific maneuver for horizontal canal should be tried.
- For the horizontal canal the Barbecue roll maneuver is utilized, where the supine patient is rotated by 360-degrees in steps of 90-degrees in the plane of the horizontal canal towards the healthy side. Alternatively, Gufoni maneuver is used which involves lying sideways onto the uninvolved side and then turning the head into the terminal nose down position **(Figure 4).**
- Patient diagnosed to have hc-BPPV also may be made to lie down sideways with the healthy ear lowermost for 12 hours to allow the particles to gravitate out of the affected horizontal canal.

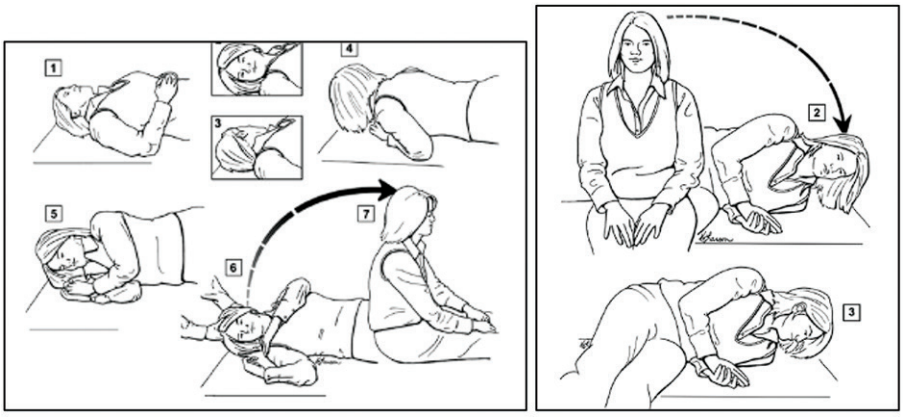

Figure 4. Barbeque Roll maneuver & Gufoni maneuver

- Some patients may complain of instability for a few days following the treatment maneuvers; typically described as a pitching sensation; the ground appears to oscillate gently like a boat or floating barge. This is otolithic syndrome due to the particles returning back to the utricle – subsides eventually.
- Patients with physical limitations including cervical stenosis, Down's syndrome, severe rheumatoid arthritis, cervical radiculopathies, Paget's disease, morbid obesity, ankylosing spondylitis, low back dysfunction, retinal detachment, carotid stenosis, and spinal cord injuries may not be candidates for CRP or may need specialized examination tables for performance of the procedure.
- Finally, it is not uncommon for patients with BPPV to develop phobic postural vertigo due to the anxiety about recurrence and the clinician finds it extremely difficult to differentiate this from recurrence. Making matters worse phobic vertigo may be resistant to therapy and patient continues in the state of anxiety self-perpetuated by myriad variety of dizzy spells; it usually merits Cognitive behavioral therapy and drugs like tricyclic antidepressants.

p. Vestibular Rehabilitation Therapy (VRT) is an alternate option in management of BPPV

- VRT may be offered to patients who fail initial CRP attempts, who are not candidates for CRP, have

additional impairments and/or who refuse CRP.

- Vestibular rehabilitation refers to a broad designation of therapies that include habituation exercises, exercises for gaze stabilization, balance retraining and facilitation of sensory and motor integration, gait retraining, fall prevention, relaxation training, and conditioning exercises. Examples include Cawthorne-Cooksey exercises and Brandt-Daroff exercises.

q. Routine prescription of vestibular suppressants is not indicated in BPPV

- Vestibular suppressants are not effective, either for primary treatment or as a substitute for repositioning maneuvers.
- Apart from the potential harmful adverse effects these drugs can interfere with central compensation for a vestibular insult.
- Vestibular suppressant medications may be used for the short-term management of autonomic symptoms, such as nausea or vomiting or in a severely symptomatic patient.

r. **There is limited role for surgery in BPPV**

- Surgery may be the only option in those exceptional minority (< 1%) of patients with confirmed diagnosis, who do not respond to repeated appropriate therapeutic maneuvers.
- Surgical options include denervating the posterior canal through the middle ear by a singular neurectomy or plugging the posterior canal via a transmastoid approach.

END NOTE

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