Benign Paroxysmal Positional Vertigo: Present Perspective

H Bhattarai

Ganesh Man Singh Memorial Academy of ENT and head and Neck Studies, TU Teaching Hospital, Kathmandu, Nepal

Correspondence to
Dr. Hari Bhattarai,
GPO Box No.6559
Email:sukhaura@gmail.com

Objective of this article was to review the current concepts regarding etio-pathogenesis and natural history of benign paroxysmal positional vertigo (BPPV), various diagnostic and therapeutic procedures in use for its different variants and rate of its recurrence after treatment. Representative articles were selected and reviewed. Idiopathic canalolithiasis of posterior semicircular canal is the commonest pathology. Making correct diagnosis of different clinical variants is at times challenging. Once diagnosed most of these conditions respond well to simple office based particle repositioning maneuvers (PRM). BPPV can spontaneously abate in some cases. Recurrence with secondary types of BPPV is more than with that of idiopathic variety. Awareness and recognition of this condition is very important as vertigo is often treated by different specialties in Nepal.

Keywords:
BPPV, Benign Paroxysmal Positional Vertigo, Dix-Hallpike procedure and Epley maneuver.

INTRODUCTION:

Temporal bone dissection findings dramatically have changed the perception of BPPV).[1] The clinical presentation of this common condition is straightforward but the choice of diagnostic and therapeutic maneuvers at times can be challenging. [2] Although termed benign, it can be a severe and disabling problem for some of the patients. It affects the quality of life of its sufferers and can be associated with reduced activities of daily life, falls, and depression especially in geriatric patients.[3] Barany, in 1921, first described BPPV as episodic vertigo of acute onset and oflimited duration induced by change in head position in relation to gravity.[4] In 1952, otologists Dix and Hallpike described a provocative maneuver to induce positional nystagmus in patients with BPPV. Their work determined the cardinal manifestations of BPPV and helped in the lateralization of the affected ear, and its characterization as central and peripheral. In 1969, Schuknecht proposed the 'heavy cupula theory' (cupulolithiasis). Cupulolithiasis theory could explain the linear rotatory nystagmus, its latency and recurrence of nystagmus and vertigo after rest. However, it could not explain the mechanism of brief duration of the nystagmusand reversal of the nystagmus with the return to a sitting position.[5] In1979, Hall et al put forward their view on the mechanism of stimulation of the posterior semicircular canal (PSC) by free-floating particles rather than heavy cupula. This concept was later termed the 'theory of canalithiasis'by Epley.[5] This theory could explain unanswered questions of cupulolithiasis theory. This led to the evolvement of safe canalolith repositioning procedures (CRP), described by Epley in 1980 and Semont et al in 1998. This theory also led to development of a surgical approach of occlusion of PSC as described by Parnes and McClure in 1990. In 1985, McClure reported on several patients with a clinical picture was suggestive of horizontal semicircular canal (HSC) involvement. Anterior semicircular canal (ASC) variant was described by Steddin and Brandt in 1994.[1]

Incidence

Froehling et al[6] have suggested an incidence of 64 cases of BPPV per100,000 residents. Lifetime prevalence of this condition is estimated to be 3.2% in females,1.6% in males, and 2.4% overall.[1] About 20 percent of all dizziness is due to BPPV and it accounts for approximately 24% of all cases of peripheral vestibular disorders.[7] Incidence of BPPV increases with each decade and reaches the peak at the sixth to seventh decade of life.[6] Women are more affected than men, with a 2:1 ratio.[8] Right side is affected more than the left. This may be due to a subjective preference amongst patients for a right head-lying position during sleep.[8] The posterior semicircular canal BPPV (PC-BPPV) is seen in most of the cases,likely because the opening and positioning of the posterior canal is in a gravity-dependent position. BPPV may also affect the other semicircular canals (SCC) but with lower prevalence.[9] Horizontal semicircular canal (HC-BPPV) accounts for approximately 10% to 20% of all patients presenting with BPPV. [10] Anterior canal BPPV (AC-BPPV) is still considered to be a rare variant, and accounting for about 2% of cases.[11] Usually, debris within the anterior SCC is self-cleared, because its posterior arm descends directly into the common crus and the utricle.[1] Bilateral BPPV is rather rare, accounting for 6% to 26% in the reported BPPV series.[8]

Etiology

BPPV in most cases, about 50 %, is considered to be idiopathic.[9] Various predisposing factors that damage and dislodge otoconia have been associated with the development of BPPV. Sudden acceleration or deceleration of head trauma creates ruptures in segments of the macula and is more likely to be associated AC-BPPV.[1] In dental implant surgery vibration of dental turbine and forced head positioning induce loosening of otoconia.In cochlear implantation the fall of bone dust particles into the cochlea during the cochleostomy, the vibration caused while boring the cochlea and electric stimulation are sufficient to dislodge the otoconia.[12] Labyrinthitis may also cause BPPV by inflammatory effects within or near the macula or by compromise of its vascular supply.[9] The likelihood of developing BPPV may relate, in part, to metabolic considerations and the elastic and adhesive properties of the gelatinous otoconia within the maculae.[9] In migraine, spasm of the labyrinthine arteries induce local ischemia facilitating otoconial detachment.[13] It has been proposed that endolymphatichydrops damages the utricle resulting in loose otoconia. Vestibular neuronitis may simultaneously damage the utricle and detach the otoconia and bring upon BPPV.[14] Labyrinthine infarction because of blockage of anterior vestibular artery that provides the vascular supply to the ASC, HSC and utricle can also lead to BPPV.[9] Vigorous exercises as jogging, treadmill exercise and marathon running can cause BPPV. These conditions seem to lead mostly to PC-BPPV.[15] A temporal bone study in humans without vestibular disease showed a substantial decrease of otoconia in elderly persons compared with children. This finding raises the question as to whether all humans lose otoconia with advancing age and are thus prone to develop BPPV.[16] Prolonged bed rest, other ear disorders and ear surgeries are also suspected to be etiological factors.[17] Finally a positive family history has been described in some patients with BPPV.[16]

Pathophysiology

The concepts of cupulolithiasis and canalolithiasis have been the working hypotheses of BPPV for the last 30 years; both medical and surgical therapies target this site, with huge success.[1] Cupulolithiasmaintains that otolithic debris normally found in the utricle and saccule becomes displaced to the cupula of the PSCC. This then renders the organ gravity sensitive.[18] Thecanalilithiasis, the most common hypothesis, proposes that otolithic debris float freely between the ampulla and common crus of the PSCC (Fig- 1).[18] It has been suggested that the size or mass of the debris within the SCC needs to reach a critical level before BPPV symptoms develop.[19] Prolonged head-lying on one side seems to favour the entrance of more otoliths into SSC of the lowermost ear. Anatomical differences in the orientation of the SCC may facilitate or hinder the entrance of otoconia (Fig- 2).[20]

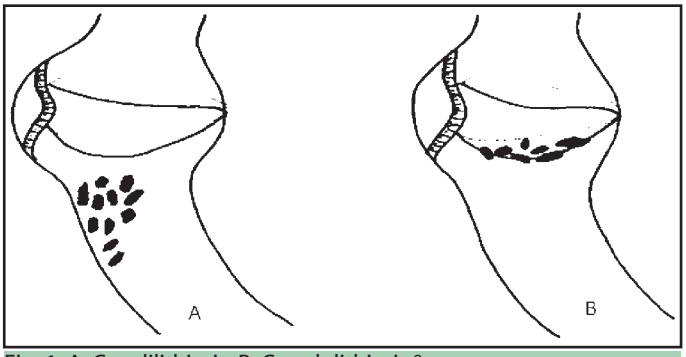

Fig: 1. A, Canalilithiasis; B, Cupulolithiasis.[9]

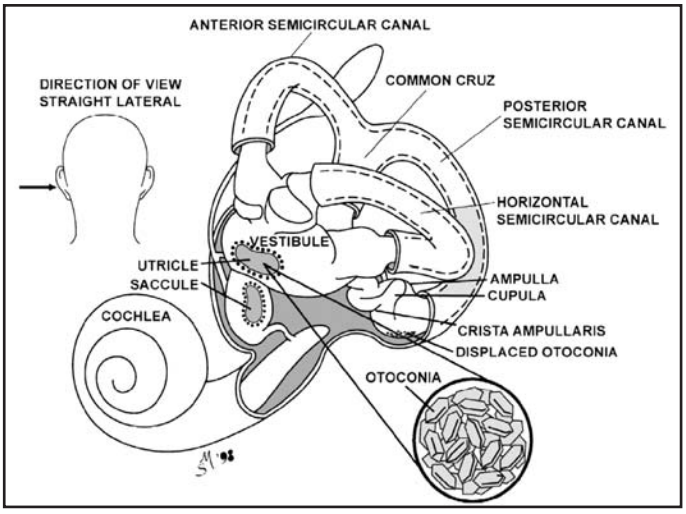

Fig: 2. Mechanism of BPPV.[9]

Occasionally, freely mobile otoconia moving within the lumen of one SSC can inadvertently move to one of the adjacent canals during the course of treatment rather than to the utricle as intended. This canal switch changes the appearance of nystagmus from that of the originally affected canal to that of the newly affected.[21] The most common canal switches are from PSC to the HSC and PSC to ASC.[9]

In recent years, two types of HC-BPPV have been recognized. One with geotropic and the other with apogeotropic positional nystagmusseen during diagnostic maneuvers.[22] The pathophysiology of the former is thought to be canalolithiasis and that of latter is still controversial, with cupulolithiasis in the HSCC being reported as the most plausible cause.[22] Two forms of cupulolithiasis, characterized by otoliths attached either on the utricle-side or the canal-side cupula, have been also identified.[23] AC- BPPV is usually transitory and most often seen in the course of treating other more common forms of BPPV.[9]

In some situations more than one canal is affected at the same time. The most common circumstance is probably bilateral posterior canal BPPV. [9] Combined HSC and PSC form of BPPV is seen especially in cases of head trauma.[24] Even after successful repositioning free-floating canaliths would be expected to re-accumulate within the PSC over time resulting in recurrent symptoms.[25] BPPV has a recurrence rate of approximately 15% per year.[20] It appears that post-traumatic BPPV has a greater tendency to recur. Of PC-BPPV successfully treated with the CRP, 44% redevelop BPPV within the first 2 years.[26] Sleep seems to be involved in the pathophysiology of BPPV.[27] Fixed positioningof the head during sleep may facilitate the deposition of particles on the cupula or in the PSCC.[28]

BPPV can spontaneously abate in some cases, but this typically occurs within the first month of onset. Asawavichianginda et alalso showed after 3 months, 84% of patients with PC-BPPV who received no treatment had converted to a negative Hallpike maneuver. [29] It can be explained by the orientations of the canals that the spontaneous remission of HC-BPPV was shorter than that of PC-BPPV. Any debris entering the PSC that hangs inferiorly from common crus essentially becomes trapped within. In contrast, HSC slopes slightly up and has its cupula at the upper end. Therefore, free-floating debris in the HSC would tend to float back out into the utricle as a result of natural head movements.[30] Geotropic HC-BPPV disappeared naturally within about 2 weeks without physiotherapy.[31]

Clinical presentation

Vertigo with nausea and vomiting is the most common symptom that occurs with change of head positionespecially while looking up, getting out of bed, rolling over in bed, bending, looking up (top-shelf syndrome) and with quick head movements.[8] Majority of patients in one study reported that their BPPV started while turning in bed during the night or while rising from bed in the morning (morning dizziness).[32] Patients do not always demonstrate the typical complaint of vertigo. It has been reported that some individuals complain of lightheadedness, dizziness or the feeling of being off-balance rather than the typical definition of true vertigo. BPPV may manifest as falls in older individuals so early diagnosis is necessary and dizziness handicap inventory (DHI) may be predictive in some cases.[32] BPPV of HSC gives rise to symptoms of episodes of vertigo that usually wakes the patient up during the night, is of a high intensity, and takes a long time to recede spontaneously, forcing the patient to remain almost immobile.[33] Side-to-side rolling in bed that causes vertigo in both directions suggests HC- BPPV but can also occur in patients who have bilateral PC-BPPV.

Diagnostic Tests

Differentiation between canalilithiasis and cupulolithiasis and the canal affected can be done with the help various diagnostic tests. Before we embark upon these tests the patient should be warned that this will provoke their dizziness but that they should remain in this position until such symptoms subside.[34]

Diagnosis of unilateral PC- BPPV due to canalithiasis, is based on the presence of an upbeating and torsional nystagmus elicited in the Hallpike-Dix position with concurrent experience of vertigo. The nystagmus has a latency of onset of 2 to 20 seconds, lasts <60 seconds, and reverses when the patient returns to the sitting position. The torsional component of the nystagmus beats towards the affected and lowermost ear while a vertical upbeating component is superimposed.[35] In bilateral PC-BPPV it is hard to decide which side is more symptomatic.[36]

It is not always easy to demonstrate observable nystagmus that is typical of BPPV and so it may be necessary to examine the patient more than once. Nunezet al have suggested that the Dix-Hallpike test can be affected by the speed of the maneuver and the plane of the occiput and that it may vary from day to day. It will be difficult to perform Dix-Hallpike procedure in obese or frail individuals, patients with stroke, anxiety states and neck trauma.[32] For such individuals various other diagnostic maneuvres have been suggested like Li Maneuvre based on subjective vertigo but not on nystagmus.[37] andside-lying maneuver.[9]

HC-BPPV is often, but not always, evoked by Dix-Hallpike maneuver. The nystagmus of HC-BPPV is distinctly horizontal and changes direction with changes in head position. The best way to diagnose HC-BPPV is by a supine head turn maneuver (Pagnini-McClure maneuver), in which the patient in the supine face-up position turns his or her head quickly to the right and left. Geotropic direction

changing positional nystagmus is right beating upon turning the head to the right and then left beating when turning the head back to the left side. Conversely, ageotropic indicates the nystagmus is right beating with turning to the left and left beating with turning to the right. The other alternative tests for the diagnosis of horizontal canal BPPV are bow and lean test[38] and Li maneuver.[37]

The Dix-Hallpike and side-lying tests used for the posterior canal BPPV can also be used to provoke and diagnose anterior canal BPPV.[9] During Dix-Hallpikemaneuver if patient has anterior AC-BPPV, the head-hanging position generates a burst of downbeating nystagmus.[26] The direction of subtle vertical-beating nystagmus underlying the torsional component is critical in differentiating AC versus PC origin. Electro-oculography/videooculography aid in accurate assessment of the vertical component for the diagnosis of canal involvement. Patients with a history of head trauma should be examined closely for AC involvement.[39]

In some situations more than one canal is affected at the same time.The most common circumstance is probably bilateral posterior canal BPPV. This can be diagnosed by the presence of typical nystagmus with Dix-Hallpike or sidelying maneuvers on both the right and left sides. Combinations of posterior and horizontal canal variants can be recognized based on the maneuver used to evoke the nystagmus and the direction of the nystagmus.[5]

Treatment

For many years, treatment of BPPV involved exercises to induce habituation and compensation.Medical treatment in the form of anti-vertiginousdrugs has been generally found to be ineffective.[39] Recent controlled studies have clearly established that physical maneuvers based on inner ear biomechanics are highly effective for PC-BPPV. In these maneuvers, the head is positioned such that loose otoconia are allowed to sediment back within the labyrinth.[41]

Appearance of a nystagmus is probably related to the amount of cupula or canal otoconia particles and might well reflect an advanced or acute stage of the disorder. Therefore, for the purpose of treatment of BPPV by repositioning maneuvers, it is not essential to observe a positional and positioning nystagmus; symptoms of vertigo connected to positional and positioning tests are sufficient.[42]

Two office procedures are very effective for PC-BPPV. Canalith repositioning treatment (CRT) (synonyms: canalith repositioning maneuver, particle repositioning procedure or maneuver, Epley maneuver or procedure, modified Epley maneuver) is the most commonly used method for treatment of posterior canal BPPV, at least in the United States. The second treatment technique is the Semontliberatory maneuver (synonyms: liberatory maneuver or treatment,Semont maneuver or treatment) more often in Europe.[9] In CRT 5th patient is moved through a series of 4 positions. With each position,the otoconia moves toward the lowest part of the canal, resulting in the movement of the otoconia around the arc of the long arm of the PC into the common crus and depositing the otoconia into the insensitive vestibule.[26] Although some advocate the use of mastoid vibrator,5or head shaking in each position,[42] these appear to make little difference, at least in most patients with BPPV.[9]

Epley maneuver may be difficult in patients with problems of cervical spine like spondylosis , disc prolapse, previous cervical spine fracture and cervical spine rheumatoid arthritis. Modified Epley with neck collar performed in an adjustable operating table is suggested.[46] Those patients who do not benefit from initial CRP may be advised self CRP at home and it is found not to affect the time to recurrence and the rate of recurrence of posterior canal.[26]

Epley recommended some posture restrictions such as sleeping in a seated position or with 45-degree elevation for 48 hours.[47] Since then, several modifications of the original repositioning maneuver and post-procedure instructions have been recommended in order to prevent otoconia from moving back into the semicircular canals. Patients are advised to avoid symptom-provoking positions, to wear a soft cervical collar,to sleep at a 45-degree angle for 2 nights and to avoid lying on the involved side , refrain from rapid head movements,[26] turning to the affected side, and flexing or extending their neck for 48 hours.[5] In the last few years, several articles have evaluated the convenience of postural restrictions.[47] However, based on current evidence; the use of postural restrictions after the canal-repositioning maneuver is unjustified.[5]

The Semont maneuver may be difficult to perform in some older or obese people because of the quick sweeping movement from one side to the other. It may be used for BPPV of the posterior canal caused by canalithiasis or for refractory cases presumed to be due to cupulolithiasis.[9]

The other procedures for PC BPPV are Brant-Darroffredistribution exercises.[32] Parnes' particlerepositioning maneuver (PRM)[47] and various modifications of Epley.However, thereis a significant number of patients who either do not respond to therapy and require repeated maneuvers in multiple sessions or who responded initially but develope rapid recurrence of their symptoms.[48] Perhaps, the most common reason for failure in these patients with an accurate diagnosis is insufficient extension of the head. If the head is not at least somewhat tilted back, the otoconia particles will not move through the canal properly. Severe kyphosis can make the positioning of the head difficult.[9]

Several repositioning maneuvers such as the Semont maneuver[49] Lempert's maneuver(360 degree barbecue rotation) and forced prolonged position (FPP, or Vannucchimanoeuvre)[48] Gufoni maneuver 50have been used for the treatment of HC-BPPV. FPP, which requires patients to lie on the healthy side for 12 hours, is regarded as the most natural and comfortable treatment.

However, the success rate of HSC-BPPV is approximately 60% to 90%; this value is significantly lower than those of PSC-BPPV. As the ASC follows adifferent trajectory from the posterior canal, maneuvers to treat AC-BPPV must necessarily differ geometrically from those described by Epley and Semont for PC-BPPV. AC-BPPV can be treated with the same maneuvers used to treat PC-BPPV but it may be difficult to ascertain the affected side unless it developed from canal switch. It is probably advisable to repeat Dix-Hallpike positioning on both sides to be sure no persisting nystagmus or symptoms are present.[51]

In this regard, there have been several non-controlled studies concerning the treatment of anterior canal BPPV. Honrubia et al[52] mention a "Reverse Epley" postural repositioning procedure. The "Reverse Semont" maneuver has also beenrecommended-as head positions with respect to gravity are identical to those of the Epley maneuver, this procedure is likely to be equally effective. Another maneuver for AC-BPPV was described by Rahko.[53] Crevits[54] described a "Prolonged Forced Position procedure'.Helminski and Hain[55] proposedusing a "deep Dix-Hallpike" maneuver for AC-BPPV. Others treatment alternatives, so far without scientific studies proving efficacy, include the Brandt Daroff exercise and the reverse Semont maneuver.[51]

If one has simultaneous PSC and HSC BPPVthe patient should be managed by first using the PSCmaneuver and then, three days later, the HSC manoeuvre.[37] Epley defined resolution of BPPV as "a negative Hallpike maneuver on follow-up exam and no further positional vertigo for one month following the last treatment."[5] Although three years' absence of symptoms is generally considered as a definitive cure.[48]

Recurrence

For PC-BPPV, Marciano and Marcelli[56] and Moon et al [57] have reported recurrence rates approximately 10% during a short-term follow-up, and Cakir et al[58] have reported even higher rates (20%) during a long-term follow-up. Despite the successful application of appropriate CRT at present, it is still controversial which factors are associated with treatment failure. Elderly patients responded less well to initial CRP treatment. This may have been due to poor cooperation, fatigue or limited mobility. Furthermore, disease may be more extensive in older patients and otoconial debris may be produced more frequently and in larger quantities than in younger patients. Patients with a history of head and neck trauma associated with the disease responded poorly to CRP treatment, with a cure rate of only 36 per cent after two treatments. Most reports generally agree that secondary BPPV has a worse prognosis than idiopathic BPPV.[59,60] It has been reported that in patients with abnormal ENG findings outcome are worse on both initial and repeat treatment. This may be explained by more extensive damage of the labyrinth in such cases.[42] Macias et al[62] found that BPPV involving any location other than a single PSC required more

than one treatment visit. Furthermore, they found that HC-BPPV responded less to therapy than did PC-BPPV. One of the causes for this low success rate is the difficulty in identifying the affected side because of recently recognized variantsof HSC-BPPV.[2] The otolith causing HC-BPPV may be adherent to the cupula or less mobile. The otolith may not be able to leave the canal because it is too big for the HSC lumen or the exit of the HSC into theutricle. We can occasionally see that the barbecue rotation causes a change from canalolithiasis to the cupulolithiasis type in several patients.[45] In addition, incorrect canalith repositioning maneuvers such as insufficient head rotation and time and failure to maintain 30° head flexion may be contributing causes.[43]

Surgery

Surgery is reserved for severe and persistent cases in which CRT is unsuccessful. Earlier destructive procedures were performed aiming at the whole labyrinth, by alcohol injection (labyrintholysis) or labyrinthectomy1but recently conservative surgery like posterior semicircular canal occlusion and ampullary nervesection[40] vestibular nerve section, microvascular decompression of the eighth cranial nerve[5]are performend . Laser occlusion of PSC for BPPV is also described.[45] For AC- BPPV plugging of the AC has been applied.[44]

CONCLUSION:

New variants of BPPV are being recognized; new, effective and simpler diagnostic tests and therapeutic maneuvers are being developed for each variant of BPPV. Management of BPPV has been moving, in part, away from treatment by health care professionals in the office and back into the home via self-treatment with either a modified Epley procedure or a modified Semont maneuver. The use of a multi axial positioning device applicable to all variants and in difficult BPPV patients shall be the subject of future publications.

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