

# Diagnosis and management of benign paroxysmal positional vertigo (BPPV)

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## Abstract

THERE IS COMPELLING EVIDENCE THAT FREE-FLOATING endolymph particles in the posterior semicircular canal underlie most cases of benign paroxysmal positional vertigo (BPPV). Recent pathological findings suggest that these particles are otoconia, probably displaced from the otolithic membrane in the utricle. They typically settle in the dependent posterior canal and render it sensitive to gravity. Well over 90% of patients can be successfully treated with a simple outpatient manoeuvre that moves the particles back into the utricle. We describe the various techniques for this manoeuvre, plus treatments for uncommon variants of BPPV such as that of the lateral canal. For the rare patient whose BPPV is not responsive to these manoeuvres and has severe symptoms, posterior canal occlusion surgery is a safe and highly effective procedure.

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Of all the inner ear disorders that can cause dizziness or vertigo, benign paroxysmal positional vertigo (BPPV) is by far the most common. In 1 large dizziness clinic, BPPV was the cause of vertigo in about 17% of patients.<sup>1</sup> It is a condition that is usually easily diagnosed and, even more importantly, most cases are readily treatable with a simple office-based procedure. Bárány<sup>2</sup> first described the condition in 1921:

The attacks only appeared when she lay on her right side. When she did this, there appeared a strong rotatory nystagmus to the right. The attack lasted about thirty seconds and was accompanied by violent vertigo and nausea. If, immediately after the cessation of these symptoms, the head was again turned to the right, no attack occurred, and in order to evoke a new attack in this way, the patient had to lie for some time on her back or on her left side.

Since this initial description was written, there have been major advances in the understanding of this common condition. In this paper, we review the normal vestibular physiology, discuss the pathophysiology and causes of BPPV, and then go on to discuss diagnoses, office-based management and, finally, surgical management.

## Anatomy and physiology

The vestibular system monitors the motion and position of the head in space by detecting angular and linear acceleration. The 3 semicircular canals in the inner ear

detect angular acceleration and are positioned at near right angles to each other (Fig. 1). Each canal is filled with endolymph and has a swelling at the base termed the “ampulla” (Fig. 2). The ampulla contains the “cupula,” a gelatinous mass with the same density as endolymph, which in turn is attached to polarized hair cells. Movement of the cupula by endolymph can cause either a stimulatory or an inhibitory response, depending on the direction of motion and the particular semicircular canal. It should be noted that the cupula forms an impermeable barrier across the lumen of the ampulla, therefore particles within the semicircular canal may only enter and exit via the end with no ampulla.<sup>3</sup>

“Ampullofugal” refers to movement “away” from the ampulla, whereas “ampullopetal” refers to movement “toward” the ampulla (Fig. 3). In the superior and posterior semicircular canals, utriculofugal deflection of the cupula is stimulatory and utriculopetal deflection is inhibitory. The converse is true for the lateral semicircular canal.

“Nystagmus” refers to the repeated and rhythmic oscillation of the eyes. Stimulation of the semicircular canals most commonly causes “jerk nystagmus,” which is characterized by a slow phase (slow movement in 1 direction) followed by a fast phase (rapid return to the original position). The nystagmus is named after the direction of the fast phase. Nystagmus can be horizontal, vertical, oblique, rotatory or any combination thereof. “Geotropic nystagmus” refers to nystagmus beating toward the ground, whereas “apogeotropic nystagmus” refers to nystagmus beating away from the ground.

“Canalithiasis” describes free-floating particles within a semicircular canal (Fig. 4). The concept was first described in 1979 by Hall, Ruby and McClure,<sup>4</sup> and the phenomenon was first demonstrated in vivo by Parnes and McClure in 1992.<sup>5</sup> “Cupulolithiasis” describes particles adherent to the cupula of a semicircular canal (Fig. 4). This term was coined by Schuknecht<sup>6,7</sup> in 1969.

## Mechanism

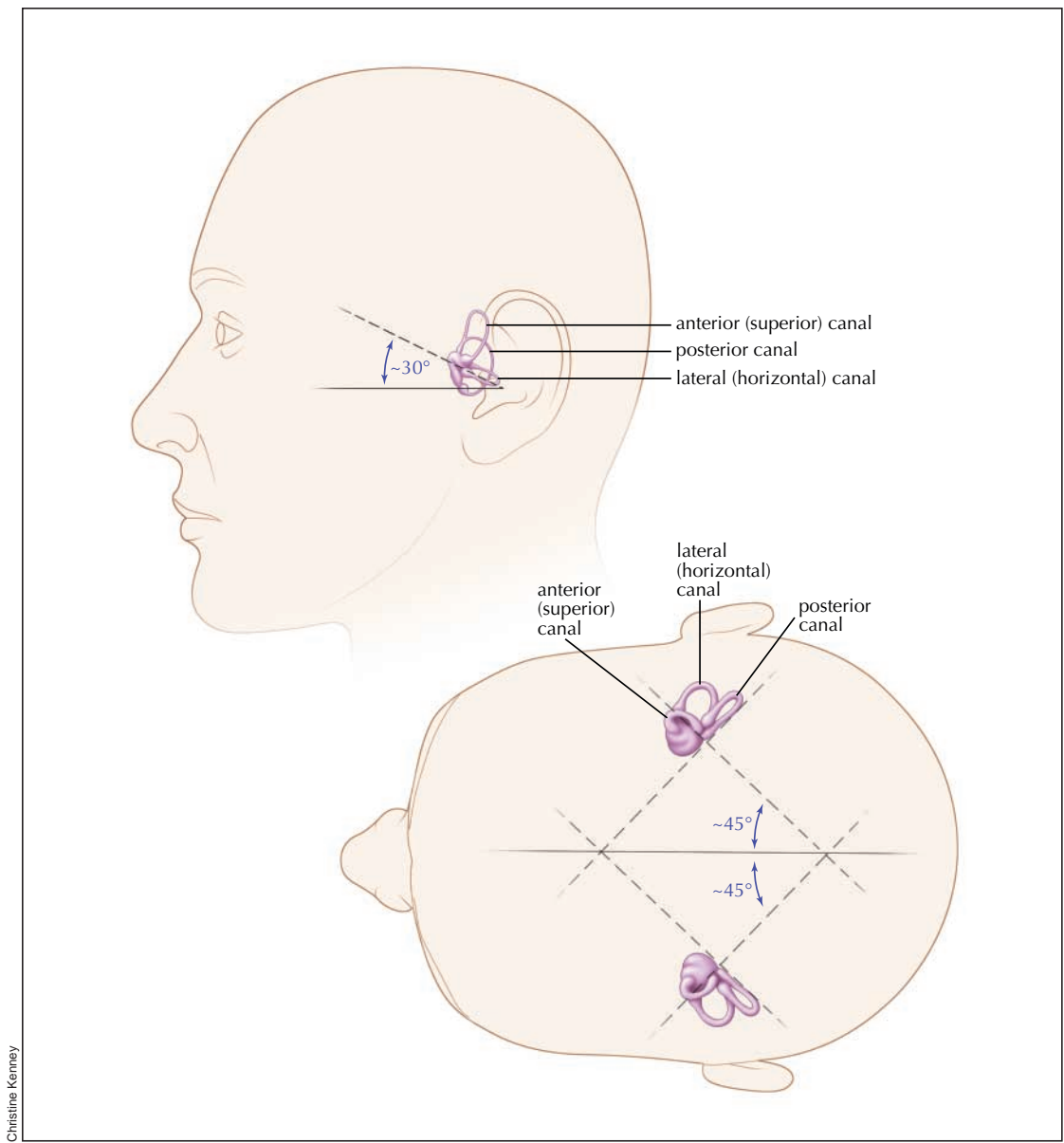
BPPV can be caused by either canalithiasis or cupulolithiasis and can theoretically affect each of the 3 semicircular canals, although superior canal involvement is exceedingly rare.

**Posterior canal BPPV**

The vast majority of all BPPV cases are of the posterior canal variant. The pathophysiology that causes most posterior canal BPPV cases is thought to be canalithiasis. This is probably because most free-floating endolymph debris tends to gravitate to the posterior canal, being the most gravity-dependent part of the vestibular labyrinth in both the upright and supine positions. Once debris enters the posterior canal, the cupular barrier at the shorter, more dependent end of the canal blocks the exit of the debris. Therefore, the debris becomes “trapped” and can only exit at the end without the ampulla (the *common crus*) (Fig. 4). Agrawal and Parnes<sup>8</sup> found obvious free-floating en-

dolymph particles in 30% of ears operated on for posterior canal BPPV (Fig. 5).

The mechanism by which canalithiasis causes nystagmus in the posterior semicircular canal was described by Epley.<sup>9,10</sup> Particles must accumulate to a “critical mass” in the dependent portion of the posterior semicircular canal. The canalith mass moves to a more dependent position when the orientation of the semicircular canal is modified in the gravitational plane. The drag thus created must overcome the resistance of the endolymph in the semicircular canal and the elasticity of the cupular barrier in order to deflect the cupula. The time taken for this to occur plus the original inertia of the particles explains the latency seen during the Dix–Hallpike manoeuvre, which is described later.



Christine Kenney

**Fig. 1: Spatial orientation of the semicircular canals.** Note how the posterior canal on 1 side is in the same plane as the contralateral superior canal. Both lateral canals are in the same plane, 30° above the horizontal.

In the head-hanging position, the canalith mass would move away from the cupula to induce ampullofugal cupular deflection. In the vertical canals, ampullofugal deflection produces an excitatory response. This would cause an abrupt onset of vertigo and the typical “torsional nystagmus” in the plane of the posterior canal. In the left head-hanging position (left posterior canal stimulation), the fast component of the nystagmus beats clockwise as viewed by the examiner. Conversely, the right head-hanging position (right posterior canal stimulation) results in a counter-clockwise nystagmus. These nystagmus profiles correlate with the known neuromuscular pathways that arise from stimulation of the posterior canal ampullary nerves in an animal model.<sup>11</sup>

This nystagmus is of limited duration, because the endolymph drag ceases when the canalith mass reaches the limit of descent and the cupula returns to its neutral position. “Reversal nystagmus” occurs when the patient returns to the upright position; the mass moves in the opposite direction, thus creating a nystagmus in the same plane but the opposite direction. The response is fatiguable, because the particles become dispersed along the canal and become less effective in creating endolymph drag and cupular deflection.

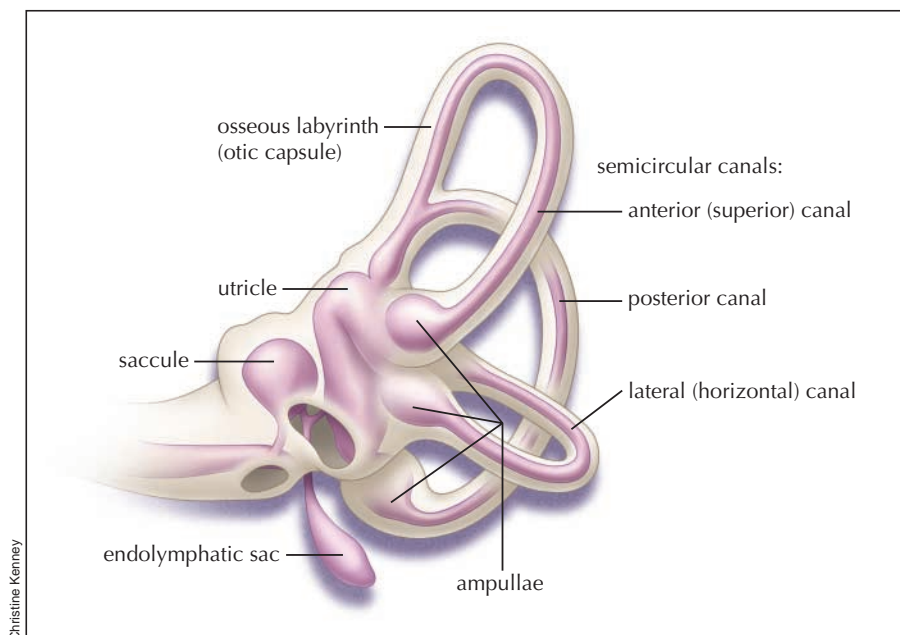
### **Lateral (horizontal) canal BPPV**

Although BPPV most commonly affects the posterior semicircular canal, 1 report suggests that up to 30% of BPPV may be of the horizontal canal variant.<sup>12</sup> In our dizziness clinic, the horizontal canal variant accounts for less than 5% of our BPPV cases. However, our findings may be

biased by the long wait for an assessment in our clinic (> 5 months), as it has also been our experience that lateral canal BPPV resolves much more quickly than posterior canal BPPV. These observations are understandable when one considers the orientations of the canals. The posterior canal hangs inferiorly and has its cupular barrier at its shorter, more dependent end. Any debris entering the canal essentially becomes trapped within it. In contrast, the lateral canal slopes up and has its cupular barrier at the upper end. Therefore, free-floating debris in the lateral canal would tend to float back out into the utricle as a result of natural head movements.

In lateral canal canalithiasis, particles are most often in the long arm of the canal relatively far from the ampulla. If the patient performs a lateral head turn toward the affected ear, the particles will create an ampullopetal endolymph flow, which is stimulatory in the lateral canal. A geotropic nystagmus (fast phase toward the ground) will be present. If the patient turns away from the affected side, the particles will create an inhibitory, ampullofugal flow. Although the nystagmus will be in the opposite direction, it will still be a geotropic nystagmus, because the patient is now facing the opposite direction. Stimulation of a canal creates a greater response than the inhibition of a canal, therefore the direction of head turn that creates the strongest response (i.e., stimulatory response) represents the affected side in geotropic nystagmus (Table 1).

Cupulolithiasis is thought to play a greater role in lateral canal BPPV than in the posterior canal variant. As particles are directly adherent to the cupula, the vertigo is often intense and persists while the head is in the provocative posi-



**Fig. 2: Osseous (grey/white) and membranous (lavender) labyrinth of the left inner ear.** Perilymph fills the osseous labyrinth external to the membranous labyrinth, whereas endolymph fills the membranous labyrinth.

tion. When the patient's head is turned toward the affected side, the cupula will undergo an ampullofugal (inhibitory) deflection causing an apogeotropic nystagmus. A head turn to the opposite side will create an ampullopetal (stimulatory) deflection, resulting in a stronger apogeotropic nystagmus. Therefore, turning away from the affected side will create the strongest response (Table 1). Apogeotropic nystagmus is present in about 27% of patients<sup>12</sup> who have lateral canal BPPV.

### Epidemiology

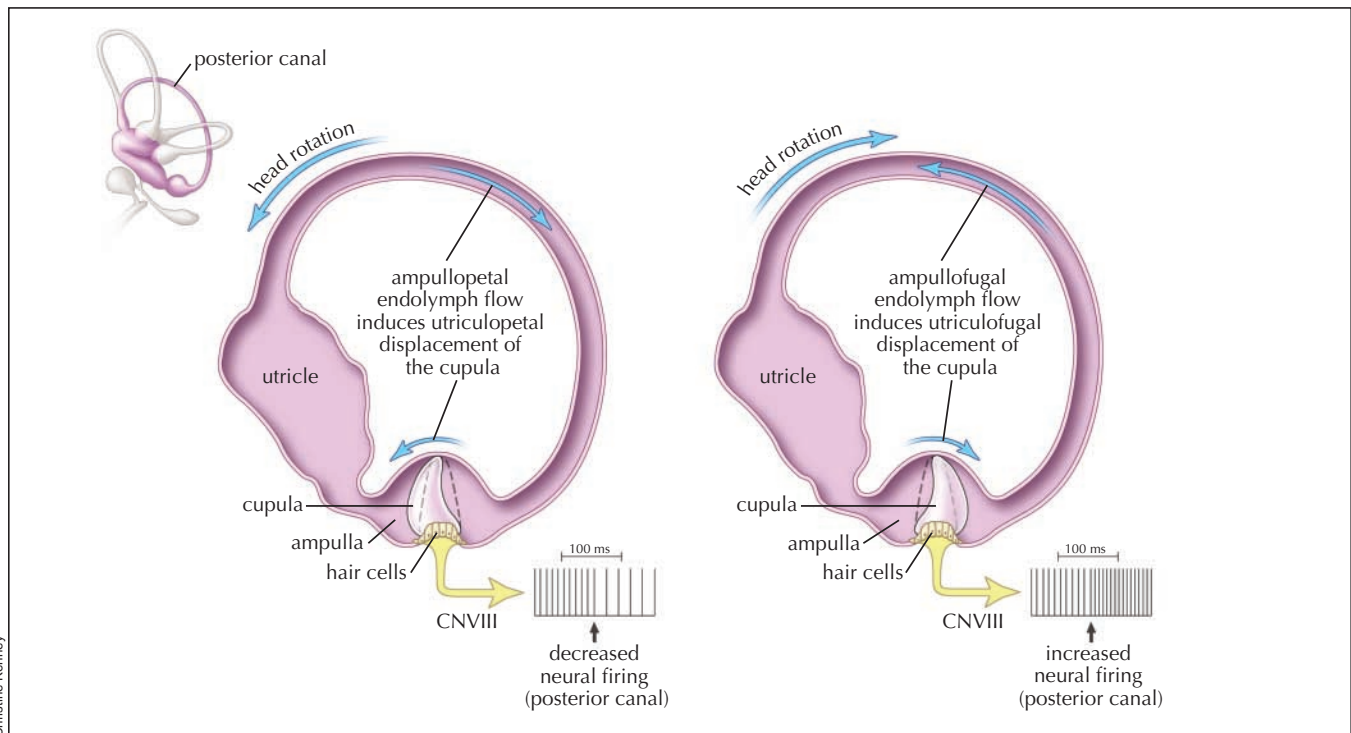
BPPV is the most common disorder of the peripheral vestibular system.<sup>13</sup> Mizukoshi and colleagues<sup>14</sup> estimated the incidence to be 10.7 to 17.3 per 100 000 per year in Japan, although this is likely to be an underestimate because most cases of BPPV resolve spontaneously within months. Several studies have suggested a higher incidence in women,<sup>14-16</sup> but in younger patients and those with post-traumatic BPPV the incidence may be equal between men and women.<sup>1</sup> The age of onset is most commonly between the fifth and seventh decades of life.<sup>14,15,17</sup> Elderly people are at increased risk, and a study of an elderly population undergoing geriatric assessment for non-balance-related complaints found that 9% had unrecognized BPPV.<sup>18</sup> BPPV most often involves a single semicircular canal, usually pos-

terior, but may involve both posterior and lateral canals in the same inner ear. Posterior canal BPPV may convert to lateral canal BPPV following repositioning manoeuvres. Head trauma is the most common cause of simultaneous bilateral posterior canal BPPV.

### Causes of BPPV

In most cases, BPPV is found in isolation and termed "primary" or "idiopathic" BPPV. This type accounts for about 50%–70% of cases. The most common cause of "secondary" BPPV is head trauma, representing 7%–17% of all BPPV cases.<sup>1,15</sup> A blow to the head may cause the release of numerous otoconia into the endolymph, which probably explains why many of these patients suffer from bilateral BPPV.<sup>1</sup> Viral neurolabyrinthitis or so-called "vestibular neuronitis" has been implicated in up to 15% of BPPV cases.<sup>15</sup>

Ménière's disease has also been shown to be strongly associated with BPPV. There is large variation in the literature regarding what proportion of patients with BPPV also have the diagnosis of Ménière's disease. Estimates range from 0.5% to 31%.<sup>19,20</sup> Gross and colleagues<sup>21</sup> found that 5.5% of patients with Ménière's disease had "certain" posterior canal BPPV. The causative mechanism is not well understood but may be the result of hydroppically induced



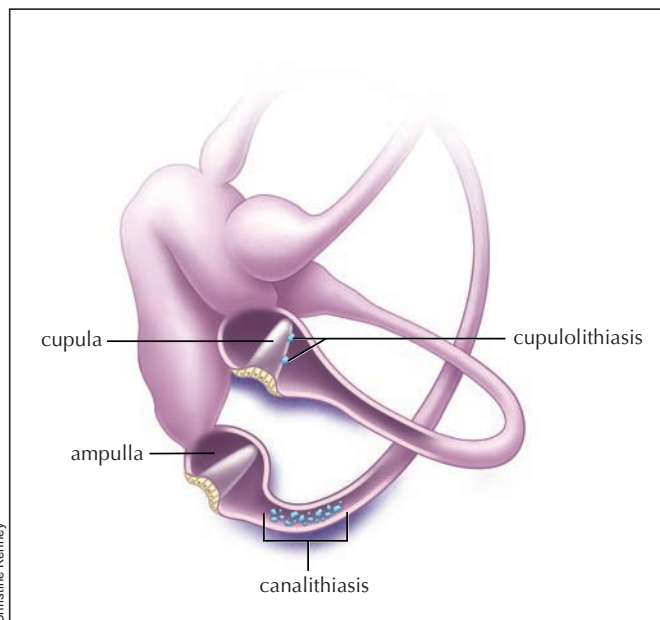
**Fig. 3: Schematic drawing of the physiology of the left posterior semicircular canal.** In the image on the right, note the excitatory response (increased neural firing) with utriculofugal cupular displacement. The same excitatory response would occur in the superior (anterior) canal with utriculofugal cupular displacement, whereas the opposite (inhibitory) response would occur with utriculofugal cupular displacement in the lateral canal. The same rules would apply to the image on the left. CNVIII = vestibular nerve, ms = millisecond.



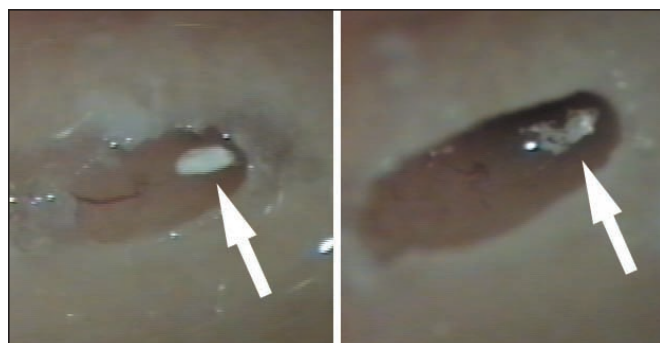
damage to the macula of the utricle or by partial obstruction of the membranous labyrinth.<sup>21</sup>

Recently, migraines have been found to be closely associated with BPPV. Ishiyama and colleagues<sup>22</sup> and Lempert and colleagues<sup>23</sup> found an increased incidence of migraine in patients with BPPV and higher recurrence rates of BPPV after successful positioning in patients with migraine. It has been suggested that spasm of the inner ear arteries may be a possible causative mechanism, because vasospasm is well documented in migraines.<sup>22,24</sup>

Secondary BPPV has also been described after inner ear surgery.<sup>20,25,26</sup> The cause is thought to be linked to utricular damage during the procedure, leading to the release of otoconia.



**Fig. 4: Left inner ear.** Depiction of canalithiasis of the posterior canal and cupulolithiasis of the lateral canal.



**Fig. 5: Sequential computer-regenerated photographs taken from an intra-operative video of a fenestrated posterior semicircular canal.** Note the single white conglomerate mass within the membranous duct (arrow) (left). Note how the mass has fragmented into tiny particles 2–3 minutes later, after the membranous duct has been probed (right).

## Diagnosis

### History

Patients describe sudden, severe attacks of either horizontal or vertical vertigo, or a combination of both, precipitated by certain head positions and movements. The most common movements include rolling over in bed, extending the neck to look up and bending forward. Patients can often identify the affected ear by stating the direction of movement that precipitates the majority of the attacks (e.g., when rolling over in bed to the right, but not the left, precipitates dizziness, this indicates right ear involvement). A study by Kentala and Pyykko<sup>27</sup> reported that 80% of patients experience a rotatory vertigo and 47% experience a floating sensation. The attacks of vertigo typically last fewer than 30 seconds, however, some patients overestimate the duration by several minutes. Reasons for this discrepancy may include the fear associated with the intense vertigo along with the nausea and disequilibrium that may follow the attack. The vertigo attacks occur in spells; patients have several attacks a week (23%) or during the course of 1 day (52%).<sup>27</sup>

In addition to vertigo, many patients complain of lightheadedness, nausea, imbalance and, in severe cases, sensitivity to all directions of head movement. Many patients also become extremely anxious for 2 main reasons. Some fear that the symptoms may represent some kind of sinister underlying disorder such as a brain tumour. For others, the symptoms can be so unsettling that they go to great lengths to avoid the particular movements that bring on the vertigo. For this reason, some may not even realize that the condition has resolved, as it so often does over time without any treatment at all. BPPV can be described as self-limited, recurrent or chronic.

As the name implies, BPPV is most often a benign condition, however, in certain situations it may become dangerous. For example, a painter looking up from the top of a ladder may suddenly become vertiginous and lose his or her balance, risking a bad fall. The same would hold true for underwater divers who might get very disoriented from acute vertigo. Heavy machinery operators should use great caution especially if their job involves significant head movement. Most people can safely drive their car as long as they are careful not to tip their head back when checking their blind spot.

**Table 1: Lateral (horizontal) canal BPPV — side of origin and mechanism based upon direction and intensity of nystagmus**

Intensity of nystagmus	Side of origin and mechanism of BPPV	
	Apogeotropic nystagmus	Geotropic nystagmus
Stronger on left side	Right cupulolithiasis	Left canalithiasis
Stronger on right side	Left cupulolithiasis	Right canalithiasis

Note: BPPV = benign paroxysmal positional vertigo. Lateral canal BPPV side of origin and mechanism are based upon the direction and intensity of nystagmus in the 2 lateral head positions.

Although 50%–70% of BPPV is idiopathic (with no identifiable cause), a history should be taken regarding possible secondary causes of BPPV. These include head trauma, viral labyrinthitis or vestibular neuronitis, Ménière's disease, migraines, and otologic and nonotologic surgery.

### Diagnostic manoeuvres

The use of the Dix–Hallpike manoeuvre to diagnose posterior canal BPPV was first described in 1952.<sup>28</sup> As shown in Fig. 6, the patient is initially seated in position A and then lowered to position B, and the patient's eyes are observed for nystagmus. After the head is lowered, the typical nystagmus onset has a brief latency (1–5 seconds) and limited duration (typically < 30 seconds). With the eyes in the mid (neutral) position, the nystagmus has a slight vertical component, the fast phase of which is upbeating. There is a stronger torsional component, the fast phase of which has the superior pole of the eye beating toward the affected (dependent) ear. The direction of the nystagmus reverses when the patient is brought into the upright position and the nystagmus will fatigue with repeat testing. Along with the nystagmus, the pa-

tient will describe feeling vertiginous, the intensity of which parallels the nystagmus response. It should be emphasized that the 2 posterior canals are tested independently, the right with the head turned right and the left with the head turned left.

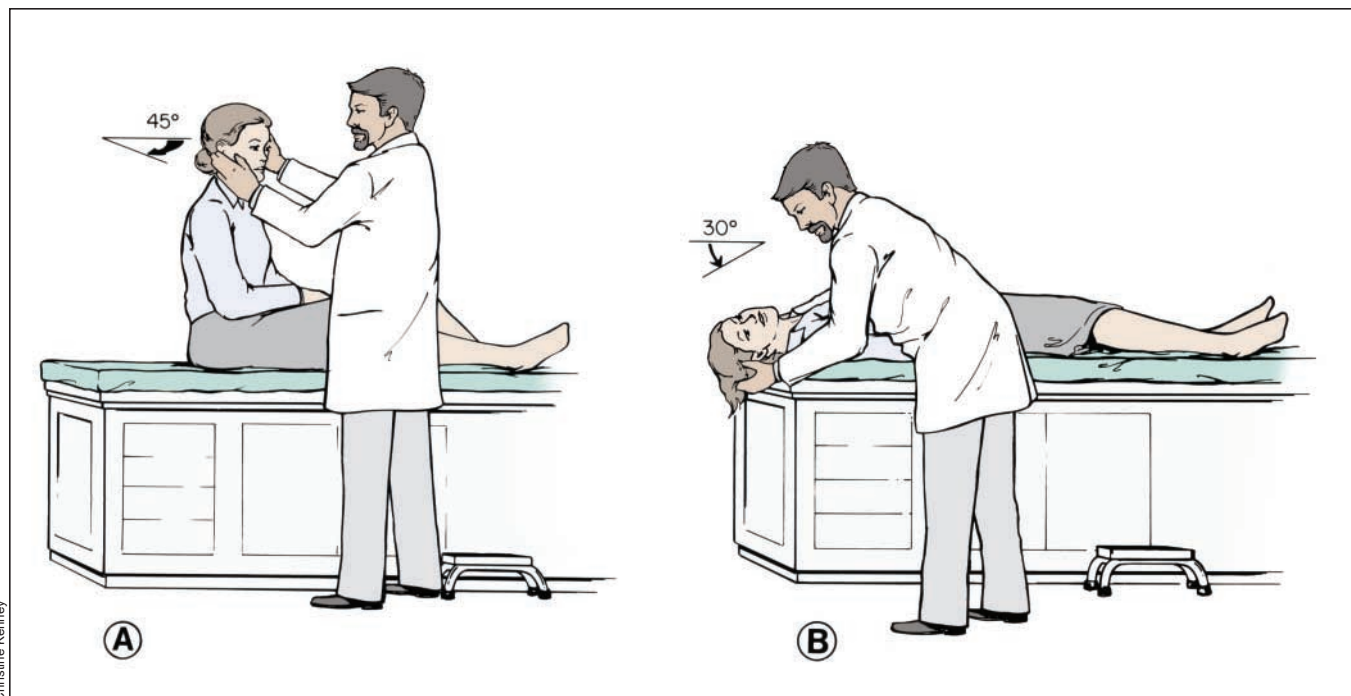
Testing for lateral canal BPPV is done by laying the patient supine and then quickly turning the patient's head (and body) laterally toward the side being tested. A purely horizontal nystagmus occurs that is geotropic (fast component toward the lowermost ear) in the majority of cases, but may be apogeotropic (toward the uppermost ear) in 27% of cases.<sup>12</sup> Compared with the

vertical–torsional nystagmus of posterior canal BPPV, this horizontal nystagmus has a shorter latency, stronger intensity while maintaining the test position and is less prone to fatigue.<sup>29</sup> Both sides are tested, and the direction of nystagmus coupled with the direction of roll that causes the greatest nystagmus intensity often identifies the affected side and the mechanism (Table 1).

Overall, the history and eye-findings during positional testing are the gold standards for diagnosing BPPV. Additional testing is not normally necessary. Because electronystagmography (ENG) does not record torsional eye move-

#### Causes of BPPV

- Primary or idiopathic (50%–70%)
- Secondary (30%–50%)
  - Head trauma (7%–17%)
  - Viral labyrinthitis (15%)
  - Ménière's disease (5%)
  - Migraines (< 5%)
  - Inner ear surgery (< 1%)



**Fig. 6: Dix–Hallpike manoeuvre (right ear).** The patient is seated and positioned so that the patient's head will extend over the top edge of the table when supine. The head is turned 45° toward the ear being tested (position A). The patient is quickly lowered into the supine position with the head extending about 30° below the horizontal (position B). The patient's head is held in this position and the examiner observes the patient's eyes for nystagmus. In this case with the right side being tested, the physician should expect to see a fast-phase counter-clockwise nystagmus. To complete the manoeuvre, the patient is returned to the seated position (position A) and the eyes are observed for reversal nystagmus, in this case a fast-phase clockwise nystagmus.

ments, it adds little to the diagnosis of BPPV. More recently, infrared videography has allowed for direct eye observation during the testing manoeuvres, but 3-dimensional eye movement analysis<sup>30</sup> is not common in clinical practice. Rotational-chair testing and posturography have no role to play in this disorder. Imaging with CT scanning or MRI is unnecessary unless there are atypical or unusual features to the assessment.

### Subjective versus objective BPPV

A certain subset of patients may not demonstrate the typical nystagmus during the Dix–Hallpike manoeuvre, but they may still experience the classic vertigo during positioning. This has been termed “subjective” BPPV, and several studies have found repositioning manoeuvres to be highly effective in this group of patients. Haynes and colleagues,<sup>31</sup> Tirelli and colleagues<sup>32</sup> and Weider and colleagues<sup>33</sup> found that patients with subjective BPPV who were treated with various repositioning manoeuvres had response rates of 76%–93% overall. Proposed theories to explain the lack of nystagmus in patients with BPPV during the Dix–Hallpike manoeuvre include the following: subtle nystagmus missed by the observer, fatigued nystagmus from repeat testing before the manoeuvre and a less noxious form of BPPV that elicits vertigo but with an inadequate neural signal to stimulate the vestibulo-ocular pathway.<sup>31</sup>

### Differential diagnosis

There are very few conditions that can even remotely resemble BPPV. In Ménière’s disease, the vertigo spells are not provoked by position change, and they last much longer (30 minutes to several hours). Furthermore, there is accompanying tinnitus and hearing loss. The vertigo in labyrinthitis or vestibular neuronitis usually persists for days. The vertigo may be aggravated by head movements in any direction, and this needs to be carefully extracted from the history so as to not confuse it with specific position change–evoked vertigo. In addition, the Dix–Hallpike test should not induce the burst of nystagmus seen in BPPV. Very rarely, posterior fossa tumours can mimic BPPV, but there have been no reports in the literature of a tumour that has perfectly replicated all of the features of a positive Dix–Hallpike manoeuvre. As mentioned previously, BPPV can be secondary, so as to occur concurrently with, or subsequent to, other inner ear or CNS disorders. Furthermore, being so common, BPPV can often be a coincidental finding with other disorders.

### Nonsurgical management

The management of BPPV has changed dramatically in the past 20 years as our understanding of the condition has progressed. Traditionally, patients were instructed to avoid positions that induced their vertigo. Medications were pre-

scribed for symptomatic relief, but 1 double-blind study showed that they were largely ineffective.<sup>34</sup> BPPV is self-limited, and most cases resolve within 6 months. As the theories of cupulolithiasis and canalolithiasis emerged, several noninvasive techniques were developed to correct the pathology directly. An earlier method used habituation exercises and, although some benefit was achieved, the effect was not long-lasting and the exercises proved to be too burdensome for many patients.<sup>35,36</sup>

### Liberatory manoeuvre

In 1988, Semont and colleagues<sup>37</sup> described the “liberatory manoeuvre” (Fig. 7) based on the cupulolithiasis theory. It was believed that this series of rapid changes of head position freed deposits that were attached to the cupula. The manoeuvre begins with the patient in the sitting position and the head turned away from the affected side. The patient is then quickly put into a position lying on his or her side, toward the affected side, with his or her head turned upward. After about 5 minutes, the patient is quickly moved back through the sitting position to the opposite position lying on his or her side with his or her head now facing downward. The patient remains in this second position for 5–10 minutes before slowly being brought back to the sitting position.

In their series of 711 patients, Semont and colleagues<sup>37</sup> found an 84% response rate after 1 procedure and a 93% response rate after a second procedure 1 week later. Several other case series have had response rates of 52%–90%,<sup>31,38,39,40</sup> with recurrence rates of up to 29%.<sup>31</sup> There has been no difference in efficacy shown between the liberatory manoeuvre and particle repositioning manoeuvre, which is described in the following section, in randomized studies by Herdman and colleagues<sup>39</sup> and Cohen and Jerabek.<sup>41</sup> In our opinion, the lib-

#### Diagnosis of BPPV

##### History

- Rotatory vertigo
- Lasts < 30 seconds
- Precipitated by head movements

##### Dix–Hallpike manoeuvre (posterior canal BPPV)

- Brief latency (1–5 seconds)
- Limited duration (< 30 seconds)
- Torsional nystagmus toward downmost ear
- Reversal of nystagmus upon sitting
- Fatiguability of the response

##### Lateral head turns (horizontal canal BPPV)

- Geotropic nystagmus
- Apogeotropic nystagmus

##### Subjective BPPV

- Classic vertigo during positioning
- No nystagmus seen — repositioning manoeuvres still effective



eratory manoeuvre is effective, but is cumbersome with elderly and obese patients, and shows no increased efficacy compared with the simple particle repositioning manoeuvre.

**Particle repositioning manoeuvre**

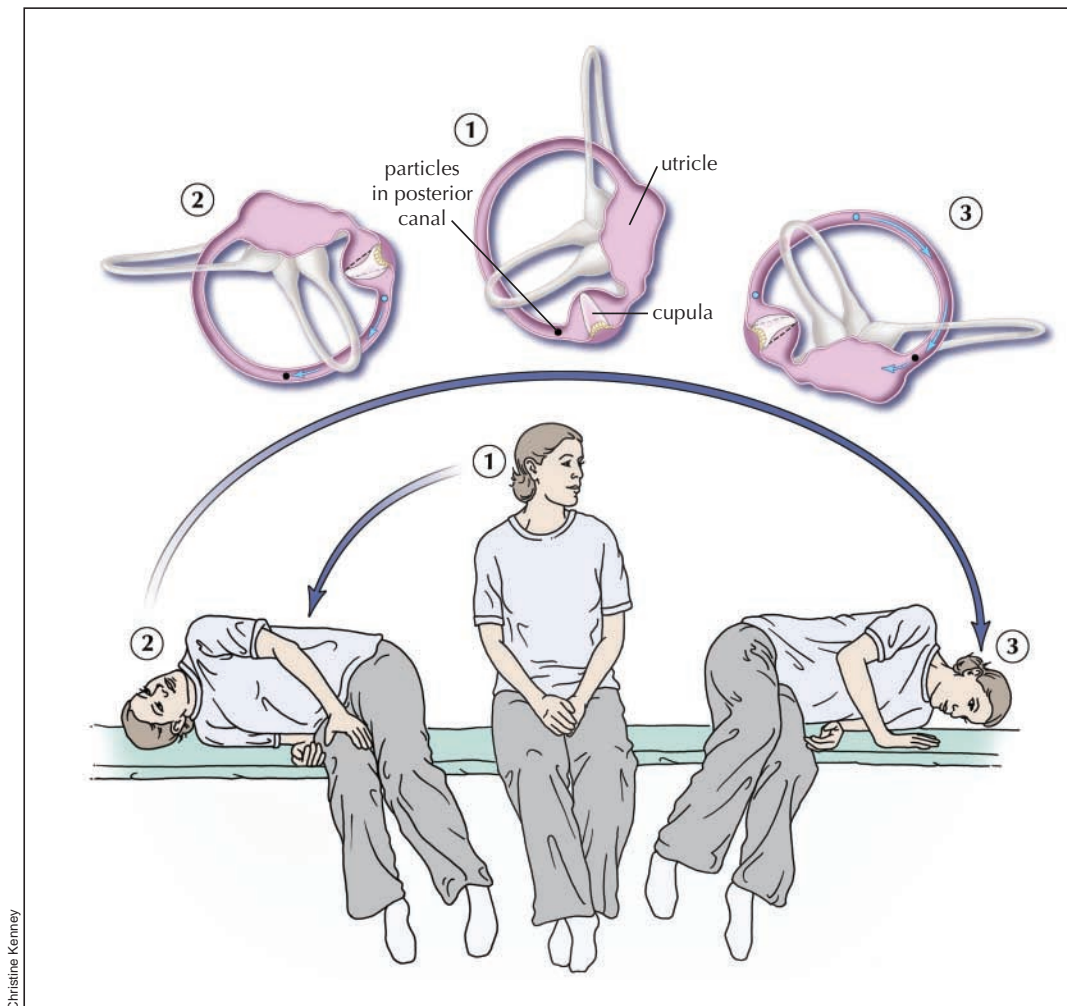
Although he had been teaching his technique for many years, it was not until 1992 that Epley<sup>42</sup> published his first report on the “canalith repositioning procedure” (CRP). This highly successful “Epley manoeuvre” is performed with the patient sedated. Mechanical skull vibration is routinely used and the patient’s head is moved sequentially through 5 separate positions. Epley postulated that the procedure enabled the otolithic debris to move under the influence of gravity from the posterior semicircular canal into the utricle. Most clinicians today are thought to use a modified version of the CRP.

One modified CRP is the particle repositioning manoeuvre (PRM) which is a 3-position manoeuvre that elim-

inates the need for sedation and mastoid vibration<sup>43,44</sup> (Fig. 8). With proper understanding of inner ear anatomy and the pathophysiology of BPPV, various appropriately trained health professionals, including family doctors and physiotherapists, should be able to successfully carry out the PRM in most straightforward cases. Atypical cases or cases that do not respond to this manoeuvre should be referred to a tertiary care dizziness clinic.

In the PRM:

1. Place the patient in a sitting position
2. Move the patient to the head-hanging Dix–Hallpike position of the affected ear
3. Observe the eyes for “primary stage” nystagmus
4. Maintain this position for 1–2 minutes (position B)
5. The head is turned 90° to the opposite Dix–Hallpike position while keeping the neck in full extension (position C)
6. Continue to roll the patient another 90° until his or her head is diagonally opposite the first Dix–Hallpike position (position D). The change from position B, through



**Fig. 7: Liberatory manoeuvre of Semont (right ear).** The top panel shows the effect of the manoeuvre on the labyrinth as viewed from the front and the induced movement of the canaliths (from blue to black). This manoeuvre relies on inertia, so that the transition from position 2 to 3 must be made very quickly.



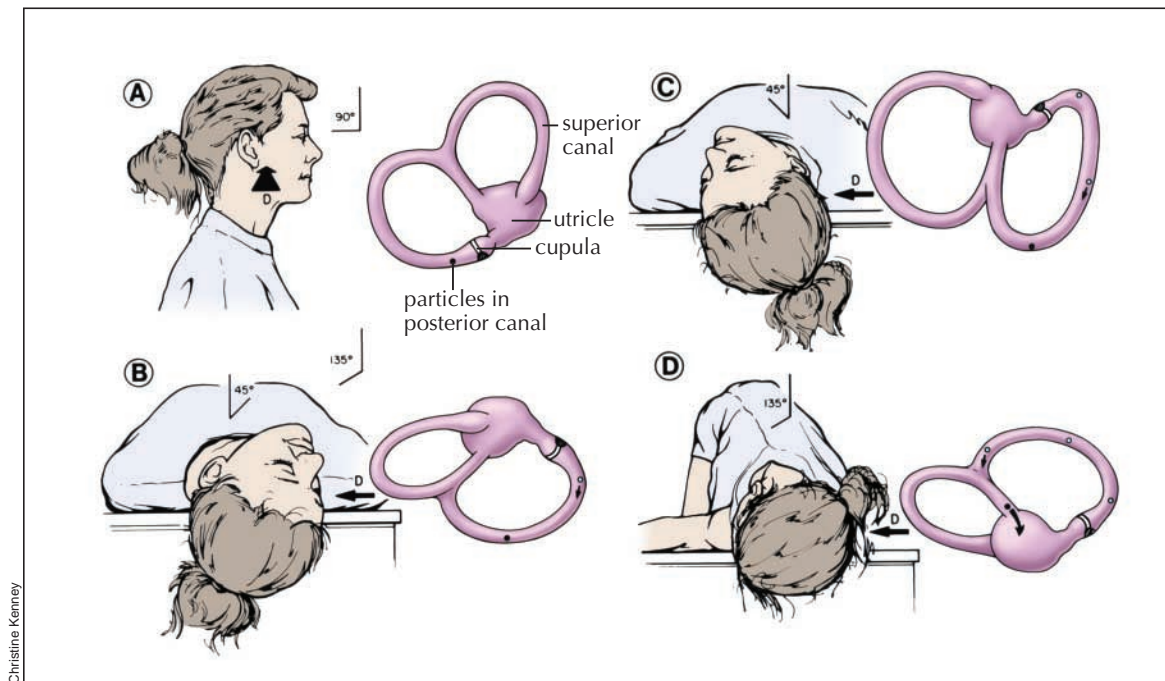
- C, into D, should take no longer than 3–5 seconds
7. The eyes should be immediately observed for “secondary stage” nystagmus. If the particles continue moving in the same ampullofugal direction, that is, through the common crus into the utricle, this secondary stage nystagmus should beat in the same direction as the primary stage nystagmus.
  8. This position is maintained for 30–60 seconds and then the patient is asked to sit up. With a successful manoeuvre, there should be no nystagmus or vertigo when the patient returns to the sitting position because the particles will have already been repositioned into the utricle.<sup>43</sup>
- Overall, the PRM should take less than 5 minutes to complete. Patients are then typically asked to remain upright for the next 24–48 hours in order to allow the otoliths to settle, so as to prevent a recurrence of the BPPV.

It is difficult to compare studies that use the repositioning manoeuvres, because they vary considerably in the length of follow-up, number of treatment sessions, number

of manoeuvres per session, the use of sedation and the use of mastoid vibration. The efficacy and treatment protocols of many trials in the literature are summarized in Table 2.<sup>31,33,39,42,43,45–52</sup> The overall response rates range from 30% to 100%. Most of these studies are case series, but Lynn and colleagues<sup>46</sup> and Steenerson and Cronin<sup>45</sup> provide good evidence from randomized studies.

### Lateral canal BPPV positioning techniques

Several positioning techniques to treat lateral canal BPPV have been developed. Perhaps the most simple is the “prolonged position manoeuvre” developed by Vannucchi and colleagues.<sup>53</sup> In cases involving geotropic nystagmus, the patient lies on his or her side with the affected ear up for 12 hours. They had resolution in more than 90% of their 35 patients. Six of their patients converted to posterior canal BPPV for which they were successfully treated using standard repositioning manoeuvres.



**Fig. 8: Particle repositioning manoeuvre (right ear).** Schema of patient and concurrent movement of posterior/superior semicircular canals and utricle. The patient is seated on a table as viewed from the right side (A). The remaining parts show the sequential head and body positions of a patient lying down as viewed from the top. Before moving the patient into position B, turn the head 45° to the side being treated (in this case it would be the right side). Patient in normal Dix–Hallpike head-hanging position (B). Particles gravitate in an ampullofugal direction and induce utriculofugal cupular displacement and subsequent counter-clockwise rotatory nystagmus. This position is maintained for 1–2 minutes. The patient’s head is then rotated toward the opposite side with the neck in full extension through position C and into position D in a steady motion by rolling the patient onto the opposite lateral side. The change from position B to D should take no longer than 3–5 seconds. Particles continue gravitating in an ampullofugal direction through the common crus into the utricle. The patient’s eyes are immediately observed for nystagmus. Position D is maintained for another 1–2 minutes, and then the patient sits back up to position A. D = direction of view of labyrinth, dark circle = position of particle conglomerate, open circle = previous position. Adapted from Parnes and Robichaud (*Otolaryngol Head Neck Surg* 1997;116: 238–43).<sup>45</sup>

The “barrel roll” was described by Epley<sup>10,54</sup> and involves rolling the patient 360°, from supine position to supine position, keeping the lateral semicircular canal perpendicular to the ground. The patient is rolled away from the affected ear in 90° increments until a full roll is completed. This is believed to move the particles out of the involved canal into the utricle. For less agile patients, Lempert and Tiel-Wilck<sup>55</sup> proposed the “log roll.” Here, the patient begins with his or her head turned completely toward the affected ear. The patient is then rapidly turned away from the affected ear in 90° increments for a total of 270°, with the head being held in each position for about 1 minute. Only 2 patients were in the study, but both were completely relieved of their vertigo.

### Controversy

Despite the excellent results from repositioning manoeuvres, there has been some controversy as to whether they actually have an effect other than central habituation, that is, when the brain adapts to repeated vestibular stimuli over time. In 1994, Blakley<sup>48</sup> published a trial of 38 patients randomly assigned to a particle repositioning group and a no-treatment group. No significant difference was found between the treated and nontreated groups at 1 month and, together, 89% showed some improvement. Blakley concluded that the manoeuvre was safe but did not provide any treatment benefit for BPPV. Buckingham<sup>56</sup> examined hu-

man temporal bones and attempted to demonstrate the possible paths taken by loose otoliths under the influence of gravity in different positions of the head. He found that although loose macular otoliths would tend to fall into the lumen of the utricle, they would not be returned to their original position in the macula of the utricle, which has a higher position in the vestibule. He concluded that a mechanism other than the repositioning of otoliths is responsible for the relief of BPPV seen in repositioning manoeuvres.

Although most cases of BPPV are self-limited, a number of randomized studies have shown that repositioning manoeuvres are highly effective. One group in Thailand performed a 6-month efficacy trial comparing the CRP with no treatment in patients with BPPV.<sup>57</sup> At 1 month, vertigo resolution was significantly higher in the CRP group (94%) versus the no-treatment group, although this difference was not seen at 3 and 6 months. Lynn and colleagues<sup>46</sup> randomly allocated 36 patients to either a PRM group or placebo treatment group with assessment at 1 month by an audiologist who was unaware of the patients' treatment allocation. Resolution of vertigo was significantly higher in the PRM group (89%) compared with the placebo group (27%). Steenerson and Cronin<sup>45</sup> randomly allocated 20 patients into either a PRM or vestibular habituation group and 20 patients into a no-treatment group. At 3 months, all patients in the treatment group had resolution of their symptoms, whereas only 25% of the no-treatment group were symptom free.

**Table 2: Efficacy of the particle repositioning manoeuvre for posterior canal BPPV**

Reference	No. of patients	Success rate, %	Recurrence rate, %	Treatment sessions	No. of manoeuvres per session	Post-manoevrue instructions	Mastoid vibration
Epley <sup>42</sup>	30	80	30	Single	Multiple	Yes	Yes
Epley <sup>42*</sup>	30	100	NR	Repeated	Multiple	Yes	Yes
Epley <sup>42</sup>	14	93	NR	NR	NR	Yes	Yes
Li <sup>47</sup>	10	30	NR	Single	Single	Yes	No
Li <sup>47</sup>	10	100	NR	Repeated with vibration	Single	Yes	Yes
Li <sup>47</sup>	27	92	NR	Single	Single	Yes	Yes
Blakley <sup>48</sup>	16	94	NR	Single	Single	No	No
Smouha <sup>49</sup>	27	93	NR	Multiple	Multiple	No	No
Wolf et al <sup>50</sup>	102	93	5	Single	Single	Yes	No
Herdman et al <sup>39</sup>	30	90	10	Single	Single	Yes	No
Parnes and Price-Jones <sup>43</sup>	34	88†	17	Multiple	Multiple	Yes	No
Weider et al <sup>33</sup>	44	88	9	Multiple	Multiple	Yes	Yes
Steenerson and Cronin <sup>45</sup>	20	85	NR	Multiple	Multiple	No	No
Welling and Barnes <sup>51</sup>	25	84	NR	Multiple	Single	Yes	No
Harvey et al <sup>52</sup>	25	68	20	Multiple	Single	Yes	No
Lynn et al <sup>46</sup>	18	61	NR	Single	Single	Yes	No

Note: NR = not reported. Table adapted from Haynes et al.<sup>31</sup>

\*Multiple entries from the same reference indicate data extracted from a single study that used different treatments for different groups of patients.

†Excluding patients lost to follow-up.

## Factors that affect repositioning manoeuvres

### Number of manoeuvres per session

There are variations in the literature regarding how many repositioning manoeuvres are performed in each treatment session (Table 2). Some performed a set number of repositioning manoeuvres regardless of response.<sup>58</sup> However, the majority of groups are divided between performing only 1 manoeuvre per clinic visit and performing manoeuvres until there is a resolution of nystagmus or excessive patient discomfort. Our objection to repeating the manoeuvre until there is a negative Dix–Hallpike response is not knowing whether the response is abolished because of a successful manoeuvre or because of a fatigued response that occurs naturally with repeat testing. From the literature review, there does not appear to be any significant difference between these approaches in terms of short-term effectiveness and long-term recurrence. Therefore, in our clinic, repeat manoeuvres are reserved for those patients who do not demonstrate an ipsidirectional secondary stage nystagmus or those who have a reverse-direction nystagmus at position D<sup>24</sup> (Fig. 8).

### Skull vibration

In Epley's original description of the CRP,<sup>42</sup> he used mechanical vibration of the mastoid (skull) bone thinking that it would help loosen otolithic debris adherent to the membrane of the semicircular canal. In 1995, a study by Li<sup>47</sup> randomly assigned 27 patients to receive the PRM with mastoid vibration and 10 patients to receive the PRM without mastoid vibration. He found that the vibration group had a significantly higher rate of improvement in symptoms (92%) compared with the nonvibration group (60%). These results do not, however, compare well with the literature (Table 2) where the majority of authors who did not use mastoid vibration achieved much higher success rates. In 2000, a larger study by Hain and colleagues<sup>58</sup> reviewed 44 patients who had the PRM with mastoid vibration and 50 patients who had the PRM without mastoid vibration. There was an overall success rate of 78% with no difference in short-term or long-term outcomes between the 2 groups.

### Postmanoeuvre instructions

Another area of divergence among experts involves the use of activity limitations after repositioning manoeuvres. Epley<sup>42</sup> asked his patients to remain upright for 48 hours after the CRP. In addition to remaining upright, certain investigators also request that their patients avoid lying on their affected side for 7 days. A study by Nuti and colleagues<sup>40</sup> examined 2 sets of patients following the liberatory manoeuvre. One group of patients were asked to remain upright for 48 hours, whereas a second group of patients were not given

any postmanoeuvre instructions. These 2 groups were compared retrospectively and no difference was found in short-term vertigo control. This finding is consistent with an earlier prospective study by Massoud and Ireland,<sup>59</sup> who also demonstrated that post-liberatory manoeuvre instructions were not efficacious.

## Surgical treatment

BPPV is a benign disease and, therefore, surgery should only be reserved for the most intractable or multiply recurrent cases. Furthermore, before considering surgery, the posterior fossa should be imaged to rule out central lesions that might mimic BPPV.<sup>60</sup>

### Singular neurectomy

Singular neurectomy, or section of the posterior ampullary nerve, which sends impulses exclusively from the posterior semicircular canal to the balance part of the brain, was popularized by Gacek<sup>61</sup> in the 1970s. Although initial reports by Gacek<sup>62</sup> demonstrated high efficacy, there was a significant risk of sensorineural hearing loss,<sup>63</sup> and the procedure has been found to be technically demanding. It has largely been replaced by the simpler posterior semicircular canal occlusion.<sup>29</sup>

### Posterior semicircular canal occlusion

Parnes and McClure<sup>64–66</sup> introduced the concept of posterior semicircular canal occlusion for BPPV. Obstruction of the semicircular canal lumen is thought to prevent endolymph flow. This effectively fixes the cupula and renders it unresponsive to normal angular acceleration forces and, more importantly, to stimulation from either free-floating particles within the endolymph or a fixed cupular deposit. Until the advent of this procedure, invasive inner ear surgery was felt to be too risky to otherwise normal-hearing ears. However, Parnes and McClure<sup>67</sup> laid the groundwork for this procedure in an animal model by demonstrating its negligible effect on hearing.

The procedure is performed under general anesthetic and should take no longer than 2–3 hours. Using a 5–6-cm postauricular incision, the posterior canal is accessed through a mastoidectomy. With the use of an operating microscope and drill, a 1-mm × 3-mm fenestration is made in the bony posterior canal. A plug, fashioned from bone dust and fibrinogen glue, is used to occlude the canal. Most patients stay in hospital for 2–3 days after this procedure. Because the occlusion also impairs the normal inner ear physiology, all patients are expected to have postoperative imbalance and disequilibrium. For most people, the brain adapts to this after a few days to a few weeks, with vestibular physiotherapy hastening this process.

In 2001, Agrawal and Parnes<sup>8</sup> published a series of cases of 44 occluded posterior canals in 42 patients. All 44 ears

were relieved of BPPV, with only 1 having a late atypical recurrence. Of the 40 ears with normal preoperative hearing, 1 had a delayed (3-month) sudden and permanent profound loss, whereas another had mild (20 dB) hearing loss.

Further studies by Pace-Balzan and Rutka,<sup>68</sup> Dingle and colleagues,<sup>69</sup> Hawthorne and el-Naggar,<sup>70</sup> Anthony,<sup>71</sup> and Walsh and colleagues<sup>72</sup> have supported the safety and efficacy of this procedure. In most otology clinics, posterior semicircular canal occlusion has become the surgical procedure of choice for intractable BPPV.

## Conclusion

Patients with BPPV present with a history of brief, episodic, position-provoked vertigo with characteristic findings on Dix–Hallpike testing. Whereas a variety of positional manoeuvres have been described, PRM (Fig. 8) is a simple effective treatment for most patients with objective or subjective BPPV. Current evidence does not support the routine use of skull vibration with repositioning. Although most clinicians are still advising patients to remain upright for 24–48 hours after repositioning, recent evidence suggests that this is unnecessary. In addition, the literature is equivocal regarding the ideal number of repositioning manoeuvres to perform per treatment session. To date, no factors have been identified to indicate an increased risk of BPPV recurrence after successful repositioning, however, the association between BPPV recurrence and migraine warrants further investigation. For the small group of patients with classic posterior canal BPPV who do not respond to repositioning, posterior canal occlusion is a safe and highly efficacious procedure.

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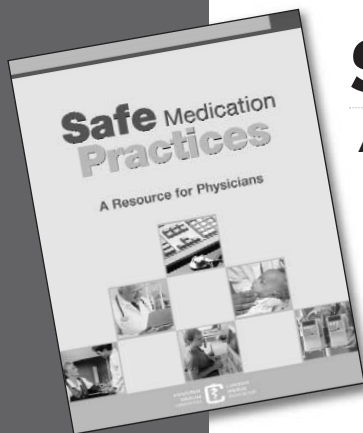
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