

BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV) — A SIMPLE SOLUTION

'Doctor, I feel dizzy...'



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The above words send shivers down the most seasoned doctor's spine. Visions occur of a never-ending consultation with an increasingly unhappy patient and no solution in sight. However, the 'dizzy' condition described below has a definite (although theoretical) aetiology, a simple and confirmatory diagnostic test, and an effective and safe treatment.

The condition is benign paroxysmal positional vertigo (BPPV) and as its name implies it consists of recurrent bouts of transient vertigo associated with rapid changes in head position. It was first described by Barany in 1921 and expanded on in detail by Hallpike in 1952.

SYMPTOMS

The classic description is of a rotatory vertigo on rapidly changing head position, commonly noticed on turning over in bed or hanging out washing (hyperextension of the neck). It lasts less than one minute. Patients are usually completely asymptomatic between attacks, but some may complain of unsteadiness.

Most cases are considered idiopathic, but further enquiry may reveal previous whiplash or head injury. Chronic suppurative otitis media, stapes surgery and labyrinthitis have also on occasion been implicated. The condition is not associated with tinnitus or hearing loss. The presumed aetiology is fundamental to understanding the symptomatology, the diagnostic Dix-Hallpike test and the treatment options.

BPPV is due to pathophysiological processes in the semicircular canals of the inner ear. The posterior semicircular canal is usually involved. Very occasionally the lateral canals are involved and extremely rarely the superior ones. The function of the semicircular canals therefore warrants a brief review.

OVERVIEW OF SEMICIRCULAR CANAL FUNCTION

The three semicircular canals are arranged at right angles to each other in the inner ear, allowing for an assessment of the body's angular acceleration through space.

The endolymphatic fluid present within the membranous part of the canal is subject to movement as the head is turned. One end of the canal is dilated and known as the ampulla. Within this structure lies the crista, containing the vestibular receptors. The movement-sensitive cilia on the surface of these receptors are embedded in a gelatinous substance found above the crista, known as the cupula. When the head turns in the same plane as the semicircular canal, the inherent inertia of the endolymph results in a flow of endolymph in the direction opposite to the head movement. This flow in turn moves the cupula and the ampullary nerve is stimulated, resulting in the brain sensing the head movement (Fig. 1).

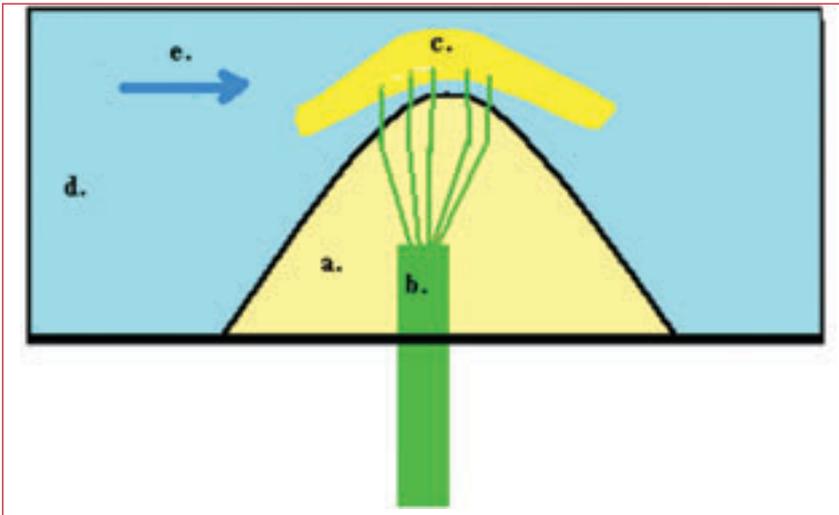


Fig. 1. Ampulla (a — crista, b — ampullary nerve, c — cupula, d — endolymph, e — endolymph flow).

PATHOPHYSIOLOGY OF BPPV

The most accepted pathophysiological explanation for BPPV was termed canalolithiasis by Hall in 1979. Here free particles (canaliths) are found within the endolymph of the semicircular canals. The canaliths are thought to be otoconial debris released from the utricle. This release may be caused by head trauma or may follow ‘injuries’ to the inner ear sense structures by other disease processes such as labyrinthitis.

The canaliths usually migrate into the most dependent area of the labyrinth, namely the posterior semicircular canal. With certain head movements

these canaliths have a ‘plunger’ effect within the narrow canal and can stimulate flow of endolymph and excite the vestibular receptors in the ampulla. A sense of vertigo is thus produced as well as a characteristic nystagmus. (Fig. 2).

An earlier theory postulated by Schuknecht in 1969 and based on his histological findings is of cupulolithiasis. The mechanism proposed here is that the particles are not free floating in the canal, but come to lie in a clump on the cupula. This exerts a gravitational force on the cupula, stimulating the embedded cilia and producing a sensation of movement (vertigo). This theory may explain some of the variant forms of BPPV.

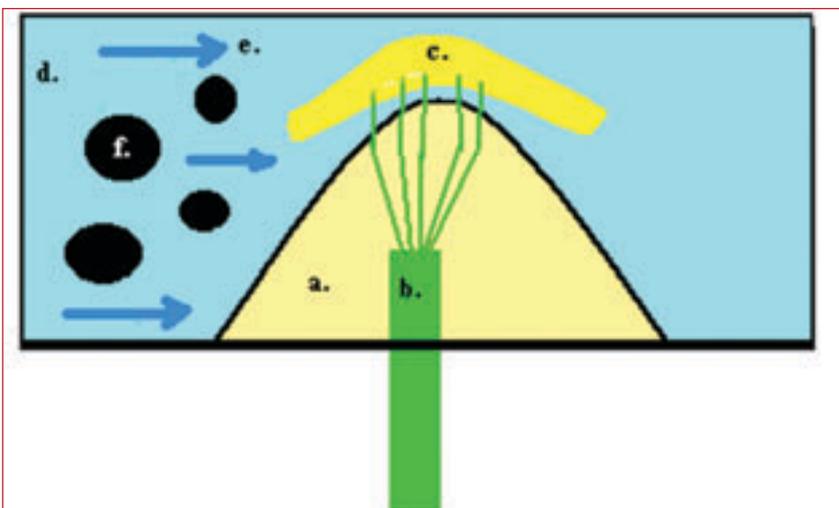


Fig. 2. Canalolithiasis (a — crista, b — ampullary nerve, c — cupula, d — endolymph, e — endolymph flow, f — canalith).

As mentioned above, the posterior semicircular canal is usually affected and all the diagnostic tests and treatments described hereafter refer to this canal.

DIAGNOSIS

The symptoms of BPPV have already been mentioned and will go a long way to establishing the diagnosis. The nystagmus is best visualised by performing the Dix-Hallpike test, which provides a diagnostic test for BPPV of the posterior semicircular canal. This test is not recommended for patients with significant neck and back problems, which should be determined before proceeding with the test.

The aim is to provoke an attack of vertigo and to visualise the expected nystagmus. First the patient is seated on an examination couch. According to Brandt the most symptomatic side should be examined first. The head is turned 45° to that side, and the patient is rapidly laid flat on the couch with the head over the end of the couch in an approximately 30° dependent position. The examiner supports the head. The patient needs to be reassured that the aim is to provoke an ‘attack’, and also encouraged to keep his/her eyes open and to avoid blinking (Fig 3).

The eyes are open and visualised for nystagmus, preferably using Frenzel’s glasses. These are illuminated and magnify the eyes, greatly enhancing the examiner’s view. The lenses of the glasses also prevent the eyes from fixating on an object. Fixation of the eyes can reduce the nystagmus produced and therefore interfere with the test. Once the symptoms and signs have disappeared completely, the patient is rapidly returned to the sitting position and re-examined for nystagmus. The test can then be repeated with the head 45° to the opposite side.

The diagnostic criteria for positive results, indicating BPPV and therefore a peripheral lesion, are as follows:

- The patient must be symptomatic during the test.

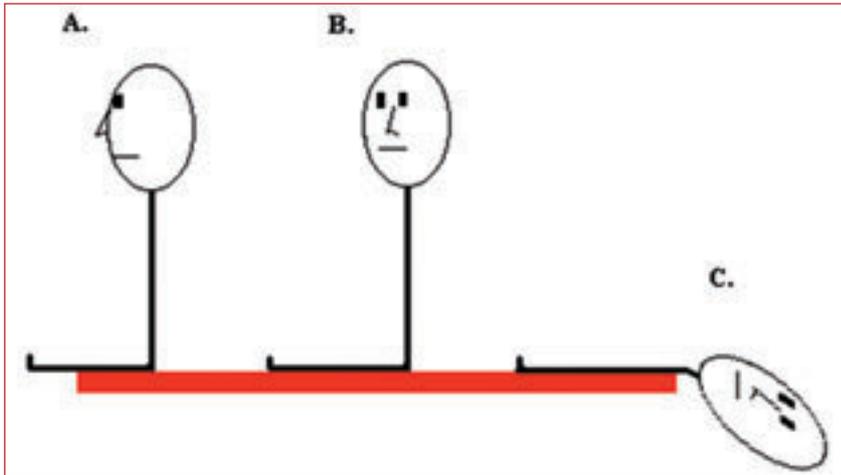


Fig. 3. The Dix-Hallpike test to the left (A — patient seated on couch, B — head 45° to side, C — rapidly into dependent position).

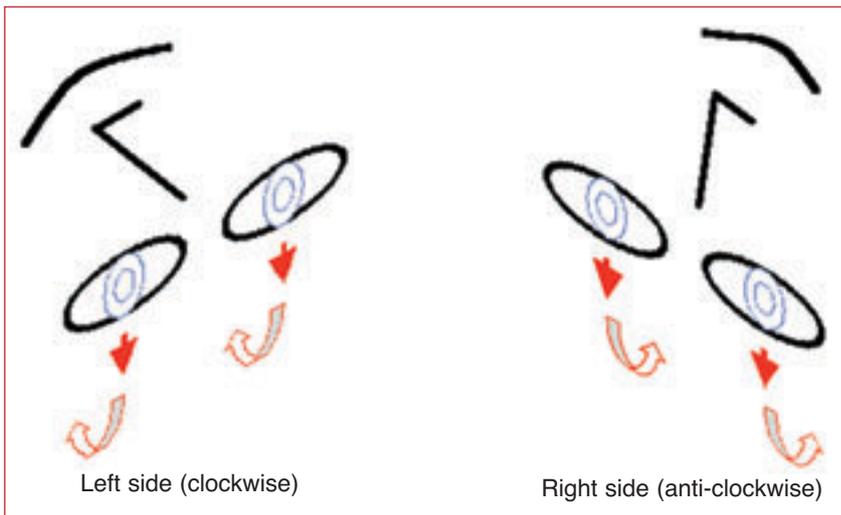


Fig. 4. Torsional nystagmus in the Dix-Hallpike test (↓ — vertical component, ↻ — geotropic component).

- There is a latent period of 4 - 5 seconds before onset of nystagmus.
- The nystagmus has vertical and torsional features. The vertical component is upward towards the forehead, and in the torsional component the upper poles of the eyes beat towards the ground (i.e. in a geotropic direction). This is clockwise (from the examiner's perspective) when testing the left side, and counter-clockwise when testing the right side (Fig. 4).
- It is of short duration, i.e. 10 - 40 seconds, and reaches a crescendo.
- The nystagmus fatigues with repetition. Each time the test is repeated the nystagmus is reduced.
- On returning to the sitting position a shorter burst of nystagmus may be seen in the opposite direction, i.e. reversal.

The diagnosis should now be established.

TREATMENT

A range of treatment options has been described for posterior semicircular canal BPPV. These vary from physical treatments, such as canalith-repositioning manoeuvres and Brandt-Daroff exercises, to surgical options of singular neurectomy and posterior semicircular canal occlusion.

Canalith-repositioning manoeuvres

The 'simple solution' alluded to in the title is the canalith-repositioning manoeuvre, the aim of which is to reposition the canaliths back into the vestibule via a sequence of controlled head position changes. It is important once again to establish that there are

no significant neck problems before attempting this.

Over the years a variety of these procedures have been described, including the Semont and Epley manoeuvres, with many modifications. The modification of the Epley manoeuvre performed at Groote Schuur Hospital will be described below.

By following the diagram in Fig. 5 the logic behind the manoeuvre can be visualised. Each position corresponds with the sequential movement of the canaliths through the posterior semicircular canal back into the vestibule.

Epley originally recommended a light tapping or application of vibration over the mastoid bone while performing the manoeuvre to enhance the progress of the canaliths. Whether this affects the outcome is debatable.

The manoeuvre is started in the Dix-Hallpike position (Fig. 5B) on the symptomatic side, as described previously. The examiner then supports the patient's head in the 45° to the side dependent (i.e. 30° over the edge of the couch) position. It is advisable to sit at the head of the couch while supporting the patient's head in one's hands. The head is then turned in 30° increments towards the opposite side, waiting for a minute after each turn (Figs 5C - E).

This is done until the head is turned 45° to the opposite side (Fig. 5F). It is important to have maintained a dependent head position throughout this stage. Here there may well be an identical burst of nystagmus as the canaliths are still moving through the semicircular canal and exercising an effect on the cupula. (The nystagmus should still be towards the symptomatic ear.)

The patient is then required to turn the whole body up onto the side through a 90° arc, sliding the shoulder that is contralateral to the affected canal underneath the body and ending up with the ipsilateral shoulder superiorly. The head remains in the same position relative to the moving body. The

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patient's face is now facing inferiorly. The patient then shuffles further back onto the couch so that the head is rest-

ing on the couch, and lies in this position for 3 - 4 minutes (Fig. 5G).

The next step is to swing the patient's legs over the side of the couch and at the same time to return the patient back up into a sitting position. With this final manoeuvre the canaliths are exited through the common crus of the semi-circular canals and into the vestibule, rendering them non-pathological. The patient's legs will end up over the side of the couch, the body facing out over the side of the couch, and the head still in the same position relative to the body, i.e. at 45° to it. The head is then returned to the neutral position in line with the body. The patient remains sitting in this final position for a few minutes (Fig. 5H).

Certain post-repositioning instructions to the patient are necessary to enhance the outcome. The necessity for these has been debated recently, but they are still part of the protocol followed at Groote Schuur Hospital. Patients are instructed to sleep in a semirecumbent position for 2 nights. Activities that result in excessive head extension/flexion, such as visiting the hair salon, are discouraged for a few days. The manoeuvre may be repeated should it not succeed in the first instance.

Brandt-Daroff exercises

Brandt-Daroff exercises are performed by patients at home. These are not single clinician-led treatments as in the canalith-repositioning manoeuvres and require patient compliance to repeat the exercises. The patient sits in the centre of a bed with the head turned 45° towards the non-affected side, and then rapidly lies down on the affected side. Vertigo may occur after a latent period. The patient maintains this position until 30 seconds after the vertigo has subsided. The patient returns to the upright position and maintains this for 30 seconds before repeating the exercise to the opposite side. Once again this position is kept for 30 seconds. The whole process is repeated until the vertigo disappears altogether.

The original recommendation was for this to be repeated up to 3-hourly for several days, and until the vertigo is absent for 2 days. At Groote Schuur Hospital the patient is encouraged to repeat the exercise thrice daily. The postulate is that these exercises cause gradual movement of the canaliths out of the canal (Fig. 6).

Surgical options

A singular neurectomy is seldom performed, but consists of cutting the singular nerve supplying the posterior semicircular canal. This is performed in the middle ear. Posterior semicircular canal occlusion was described by Parnes and McClure in 1992. The posterior canal is entered via a mastoidectomy approach and selectively occluded.

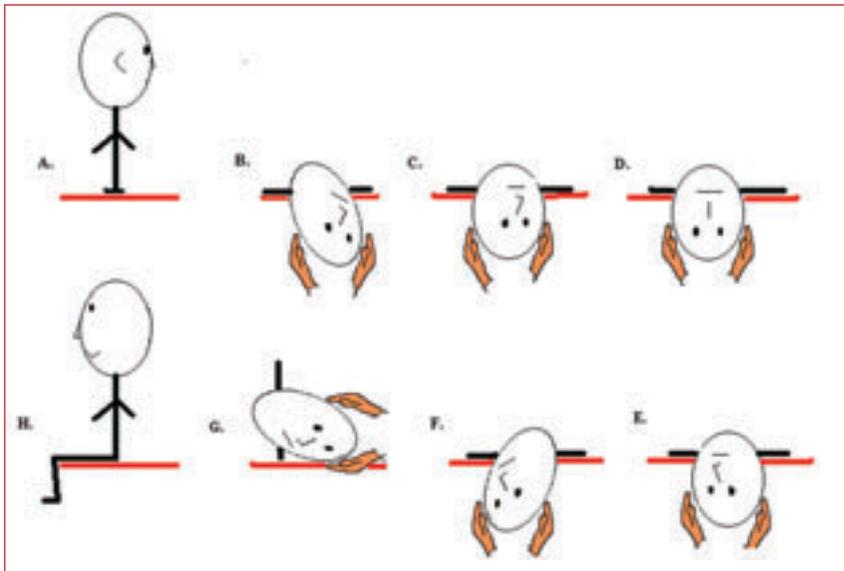


Fig. 5. Modified Epley manoeuvre for right-sided BPPV (view from head of couch: A — sitting, head 45° right, B — right Dix-Hallpike position, C-E — incremental rotation in dependent position, F — left Dix-Hallpike position, G — rotate onto left shoulder, H — sitting, looking out to side of couch).

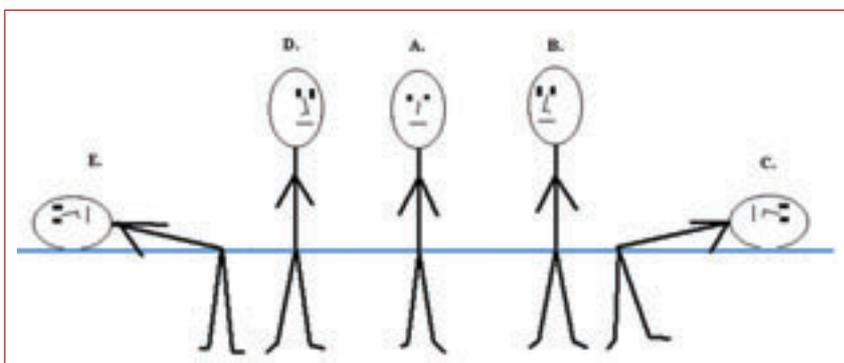


Fig. 6. Brandt-Daroff exercises (A — sitting with feet over side of couch, head neutral, B — turn head 45° right, C — lie down to the left, D — turn head 45° left, E — lie down to the right).

Further reading

Baloh RW, Honrubia V. *Clinical Neurophysiology of the Vestibular System*. (Available from SAMA - HMPG. Price: R1080.00, members: R1000.00.) Oxford: Oxford University Press, 2001.
 Brandt T. *Vertigo: Its Multisensory Syndromes*. 2nd ed. Heidelberg: Springer, 1999. (Available from SAMA - HMPG. Price: R780.00, members: R710.00.)
 Guidetti G. *Rehabilitative Management of the Dizzy Patient*. Milan: Excerpta Medica, 2000.

IN A NUTSHELL

BPPV consists of recurrent, transient rotatory vertigo and nystagmus associated with head movement in the plane of the affected semicircular canal.

It is usually idiopathic, but may follow head injury or ear pathology.

The posterior semicircular canal is most commonly involved.

It is due to loose otoconial debris (canaliths) exerting an effect on the vestibular sensors in the ampulla of the semicircular canals.

The Dix-Hallpike test is diagnostic, producing characteristic nystagmus.

The torsional nystagmus occurs after a latent period and lasts < 1 minute.

Canalith-repositioning manoeuvres are usually curative.

Brandt-Daroff exercises are a useful patient-led alternative.

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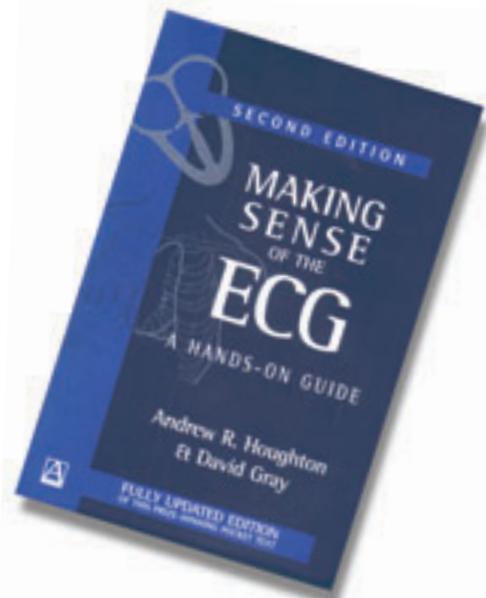
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