Benign Paroxysmal Positional Vertigo (BPPV): Diagnosis and Physical Treatment

Abstract

Benign paroxysmal positional vertigo (BPPV) is one of the most common causes of vertigo. It is easily diagnosed and treated. BPPV is due to the presence of otoconial debris within the semicircular canals, usually the posterior canal. The vertigo is intense but brief, usually triggered by seating, lying down and turning over in bed. The diagnosis can only be made by observing the typical nystagmus during positional manoeuvres such as the Hallpike manoeuvre or equivalent. The nystagmus occurs when the affected ear is in the side down position and is mostly torsional (rotatory) beating towards the undermost ear. On observing the typical nystagmus in a patient with a typical history one can proceed immediately to physical treatment with one of the repositioning manoeuvres. The Semont manoeuvre is described here, essentially consisting of one big swing whereby the patient is taken from one end of the couch (the ear down side) to the other (the eye down side). These manoeuvres 'reposition' the debris back to the utricle and cure patients of their current BPPV in 70-90% of cases. Links to a webpage showing videos of the diagnostic and treatment manoeuvres are provided.

Introduction

Up to 10 or 20 years ago the diagnosis of vertigo was just an academic exercise. Identification of the various causes of vertigo was largely irrelevant as all patients ended up with the same antivertiginous drugs and with little benefit. Physical therapy and rehabilitation have been at the centre of a refreshing change in dizziness treatment.

We now understand that the centre piece for the treatment of the patient with a single episode of vertigo (eg vestibular neuritis or 'labyrinthitis') and the patient with long term dizzy symptoms is rehabilitation.[1] Setting up a vestibular rehabilitation service does not require complex equipment or prolonged training. A physiotherapist, particularly a neuro-physiotherapist, or an audiologist, particularly one with an interest in vestibular disorders, can quickly acquire the necessary skills. Indeed, a recent study has shown that a GP practice nurse, with an appropriate one-day training session, can make a significant difference to the outcome of dizzy patients in primary care.[2]

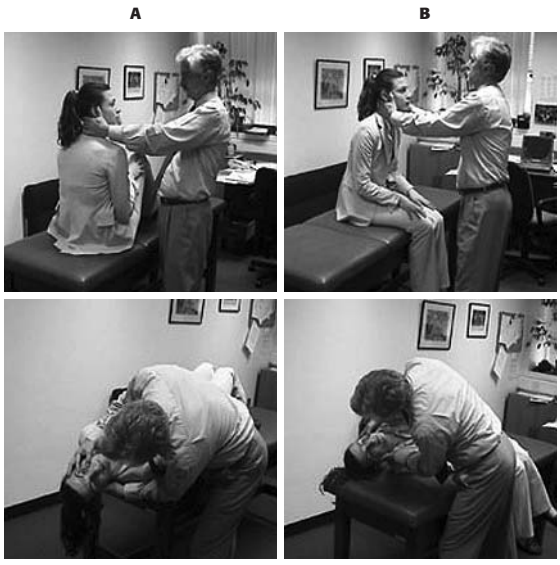


Figure 1: Two positional manoeuvres for eliciting positional vertigo and nystagmus. In this case the right ear is being investigated. Both manoeuvres are equally successful in inducing positional nystagmus and should be conducted briskly. Note how the clinician can help the patient keep the eyes open for full visibility of a positional nystagmus. A: the Hallpike manoeuvre in which the head finishes in a head hanging position. B: a trunk-sideways positional manoeuvre.

In this review, however, we will concentrate on the diagnosis and physical treatment of one specific condition, benign paroxysmal positional vertigo or BPPV. BPPV is one of the most common causes of vertigo. Both the diagnosis and treatment are straight forward and yet many patients have many drugs and expensive investigations instead of what they really need, namely a Hallpike manoeuvre for diagnosis and an Epley or Semont manoeuvre for treatment.

Symptoms

BPPV is by far the most common cause of positional vertigo, accounting for about 90% of patients. Moreover, BPPV is the number one vestibular disorder, causing 20-30% of referrals to specialised dizziness clinics.[3,4] The prevalence of BPPV increases with advancing age; women are affected almost twice as often as men. BPPV may involve each semicircular canal, with BPPV of the posterior canal being by far the most common variant. All subtypes of BPPV can be diagnosed on the basis of clinical observation during the positional manoeuvre.

Patients with BPPV complain of brief episodes (<1 min) of vertigo that appears in specific head positions, e.g. on lying down or sitting up, after turning in bed from one side to another, with head extension or bending forward. Frequently, however, the positional and brief nature of the vertigo is not recognised by the patient, even after direct questioning. Therefore, positional tests should be performed in all patients with recurrent or episodic vertigo.

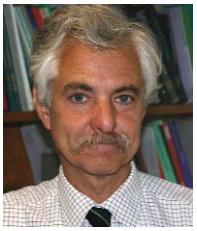
Patients are usually aware that certain head movements precipitate attacks of vertigo. They often develop strategies to avoid vertiginous attacks, e.g. sleeping upright or holding their neck stiff, which may lead to immobility and prolongation of the natural course of the disease. Indeed many BPPV patients with stiff neck and head movement-induced vertigo are often told that the problem originates in the neck – the myth of 'cervical vertigo'.[5] Another presentation is the patient with BPPV who, due to the terrifying nature of the vertigo in BPPV, develops a secondary anxiety disorder – the myth of the 'it's all in your mind' syndrome.

Each single BPPV attack lasts a few seconds but after a series of attacks, patients may complain of prolonged dizziness and imbalance lasting from hours to days. Typically, BPPV manifests itself with symptomatic episodes lasting from a few days to several months, which are interspersed by asymptomatic intervals of several months to years duration. Most cases of BPPV are idiopathic, but about 25% develop after head trauma or on the background of a pre-existing labyrinthine disorder such as vestibular neuritis or Menière´s disease. Bilateral BPPV is more common in post-traumatic patients.

Examination

Conventional clinical examination as performed by GPs, neurologists or ENT surgeons is negative in BPPV. Provocation of vertigo by positional testing and observation of typical nystagmus is the only way to make a diagnosis of BPPV. Patients must have this clearly explained to them before the positional manoeuvres. Positional vertigo is terrifying but brief - if the patient closes his/her eyes in response to the vertigo the examiner will not be able to make a diagnosis.

The most popular test for provocation and confirmation of BPPV is the Hallpike manoeuvre (Figure 1A). With this procedure the head is rotated with respect to gravity in the plane of the affected posterior canal. Alternatively, a lateral



Adolfo Bronstein is Professor of Clinical Neuro-otology at Imperial College London and a Consultant Neurologist at Charing Cross Hospital and at the National Hospital for Neurology and Neurosurgery, Queen Square, London. He heads the Department of Movement and Balance in the Division of Neuroscience at Imperial College and is the lead Editor of the first and second (2004) edition of the book 'Clinical Disorders of Balance, Posture and Gait'.
www.imperial.ac.uk/medicine/dizziness

Correspondence to:
Prof Adolfo M Bronstein MD PhD FRCP,
Professor of Clinical Neuro-otology and Consultant Neurologist,
Head, Department of Movement & Balance,
Division of Neuroscience & Psychological Medicine,
Imperial College London,
Charing Cross Hospital,
London W6 8RF.
Tel: 020 8846 7285,
Fax: 020 8846 7577,
Email: a.bronstein@imperial.ac.uk

tilt of the trunk and head from a sitting position can be performed with the head turned 45° to the opposite side, which positions the head with the lateral aspect of the occiput onto the couch (Figure 1B). This latter manoeuvre is the only one that can be performed when the couch is placed between walls or cupboards and the patient's head cannot reach the head hanging position. In any case, with both manoeuvres, the final position of the posterior semicircular canal is identical (compare Figures 1A & B). The patient is instructed to keep their eyes open, to watch the examiner's forehead or eyes and to stay in the final position even if vertigo occurs. It is useful to help the patient keep their eyes open with your own fingers, as some patients find it difficult to keep their eyes open when the vertigo develops. Frenzel´s glasses are not necessary for observation of the nystagmus.

The nystagmus in posterior canal BPPV is mostly torsional (often called 'rotatory'), with the upper pole of the eye beating towards the undermost ear (Figure 2). In addition, there is a smaller vertical skewing upbeating nystagmus component, most prominent on the uppermost eye. Typically, nystagmus and vertigo start a few seconds after the precipitating head position is reached (latency). Nystagmus intensity increases rapidly and then decays (adaptation), usually lasting 10 to 20 seconds. On returning to the sitting position, a transient nystagmus of lesser intensity beating in the opposite direction can be observed (reversal). With repeated testing, vertigo and nystagmus decrease with repeated positioning in most cases (fatigability).

A patient with a typical history of brief rotational vertigo on lying, seating or turning over in bed and with a transient torsional nystagmus as described above does not require any further investigations. One should proceed to repositioning treatment straight away. A similar clinical history can be due to the rarer horizontal or anterior canal variants of BPPV. The former has horizontal nystagmus and the latter downbeat nystagmus with a torsional component. However, unless the clinician is conversant with positional nystagmus, an MRI is advisable to rule out cerebellar-brainstem disease whenever a positional manoeuvre induces a nystagmus atypical for posterior canal BPPV.

Pathophysiology

BPPV appears when dislodged calcium rich particles from the utricular otoconia fall into the posterior semicircular canal. These debris, due to gravitational forces, move within a semicircular canal and cause inadequate endolymph flow after changes of head position (canalolithiasis). There are five factors predisposing to BPPV, namely advanced age, head trauma, a preceding inner ear disease, migraine, and general anaesthesia. These predisposing factors act by a combination of age related or ischemic utricular degeneration and head reclination (for intubation during anesthesia).

Figure 3 shows how these otoconial debris move within the posterior canal. Once otoconia have entered the posterior canal they tend to sink to the most dependent point. When the patient is upright, they are located at the base of the cupula and do not have any effect. During the Hallpike test, the head is rotated backwards in the plane of the posterior canal, inducing movement of the particles within the canal away from the cupula and thus activation of the canal's hair cells. The nystagmus subsides after the particles have reached the most dependent point of the canal and the cupula has returned to the resting position. Agglomerates of otoconia may disperse with repeated positional manoeuvres which may explain BPPV fatigability. Although the canalolithiasis concept is supported by several histological and intraoperative findings, the most convincing proof for canalolithiasis comes from the efficacy of positioning manoeuvres, which clear the affected canal from mobile particles.

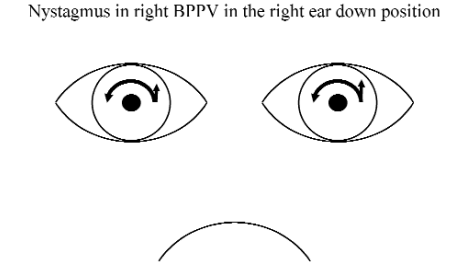


Figure 2: The nystagmus observed in a case of right sided BPPV in the right ear down position. The arrows indicate the predominant beat (fast phase) direction: torsional beating towards the right ear with a minor upbeating component. This nystagmus pattern is the result of the excitation of the right posterior semicircular canal.

Treatment

The rationale of the treatment is to redirect the otoconial particles back to the utricle where they do not cause BPPV symptoms. First of all the patient is informed about the benign course of BPPV, its mechanism and rationale for repositioning treatment. Patient cooperation is vital during the treatment as further vertigo is unavoidable during the manoeuvres. There are essentially two repositioning treatments, Epley´s and Semont´s manoeuvre. Patients should keep their eyes open for observation of nystagmus, since a positional nystagmus beating in the same direction with respect to the head indicates successive movement of the particles towards the utricle and predicts a favourable outcome to some extent. Both these therapies are highly effective in terminating an acute episode of BPPV but recurrences after several months or years are not uncommon.

Epley has introduced the canalith repositioning procedure, in which the posterior canal is rotated backwards close to its planar orientation. The manoeuvre consists of a series of successive head positionings each of about 90° displacement and several reviews illustrate clearly how to carry it out[3,4]. My personal impression is that, unless the doctor or therapist applies this manoeuvre frequently, Epley's manoeuvre is more difficult to remember than Semont's, so the latter will be described and illustrated here.

The Semont manoeuvre involves a 180° swing of the head in the plane of the posterior canal (Figure 3). The examiner guides the manoeuvre by standing in front of the patient who is seated on a couch with the head rotated 45° away from the affected ear. Then the patient is brought with a fast movement to a lying position on the side of the provocative ear (Figure 3 - 1,2). This initial part of the manoeuvre is in fact the diagnostic phase equivalent to a Hallpike manoeuvre or, more precisely, the sideways variant Hallpike manoeuvre described under Examination and illustrated in Figure 1B. In this position vertigo is triggered and torsional nystagmus beats toward the affected (undermost) ear. After being kept in this position for approximately a minute (so all debris falls to the bottom), the patient is swung rapidly onto the opposite side of the couch (and stays there for another minute) (Figure 3 - 2,3). The manoeuvre should be executed quickly in one single movement step and so, if the patient is frail, old or overweight, an assistant can help the therapist achieve this from behind the patient. In order to memorise this manoeuvre, it is useful to think that the plane of the posterior semicircular canal lies vertically in the head at 45 degrees, midway between the sagittal and coronal planes. In order to move the head diagonally at 45

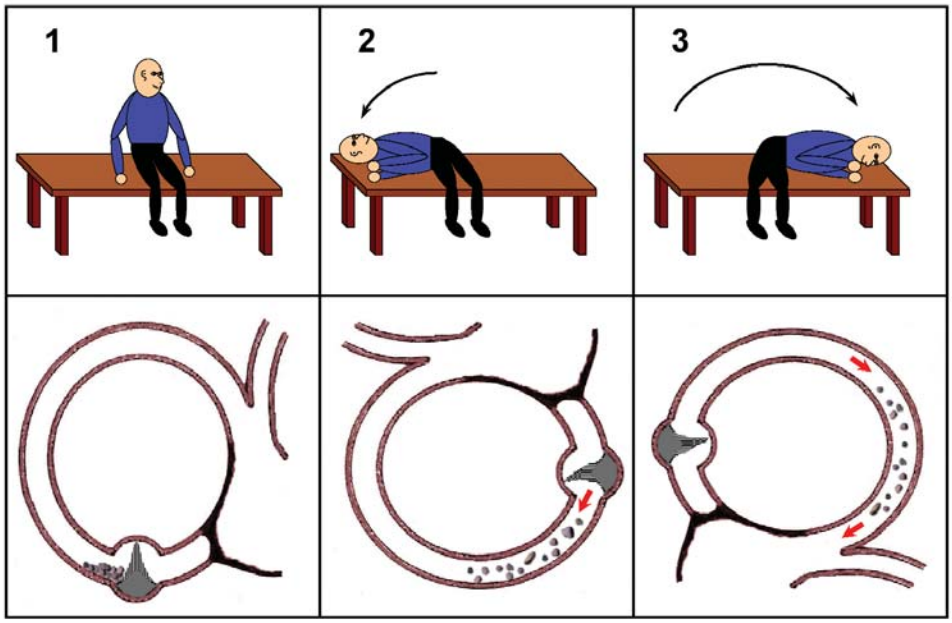


Figure 3: Pathophysiology and treatment of right sided BPPV. The cartoon illustrates canalolithiasis of the right posterior canal. On the left, the debris move down during the diagnostic positional manoeuvre (as illustrated in Figure 1B). On the right, the particles are swung out of the posterior canal back into the utricle by way of the fast head acceleration produced during the repositioning treatment (Semont manoeuvre).

degrees all you have to remember is to "go from one ear down to the opposite eye down". That is, to do a Semont manoeuvre on a patient with right BPPV (i.e. vertigo/nystagmus evoked by right ear down positional manoeuvre) swing the patient from right ear down all the way to a left eye down position (Figure 3).

Vibration of the mastoid during these repositioning treatments has been recommended but does not improve treatment outcome. Similarly, keeping upright for 48 hours after treatment has proven unnecessary. In a few patients with unusual anxiety, vertigo or nausea medication with sedatives or antiemetics before the repositioning treatment is required.

Both the Epley and Semont manoeuvres are highly effective when performed properly. After a single application complete recovery is achieved in approximately 70% of patients and 90% after a second session.[6] Randomised, controlled trials have shown that repositioning manoeuvres are clearly more effective than a sham procedure or no treatment.[7,8] For patients who do not respond to these manoeuvres or suffer from frequent recurrences, several useful procedures for self-treatment at home are available, eg Brandt-Daroff exercises or a modified Epley procedure.[9] It is advisable to visit the original publications or visit WebPages illustrating these self-treatments and prepare handouts for patients who require them.

Surgery of the posterior canal can be considered in those rare patients with longstanding BPPV who have not responded to appropriate and repeated therapeutic positionings, but this is very rarely required nowadays.

Differential diagnosis

Posterior canal BPPV must be differentiated from other forms of BPPV (horizontal and rarely anterior canal) and from central positional vertigo due to a lesion of the vestibular nuclei or caudal cerebellum. The distinction is mainly based on nystagmus features and a patient with atypical nystagmus should always be imaged. A purely vertical (either downbeat or upbeat) nystagmus strongly suggests a central positional nystagmus whereas a history of recurrences and remissions is in favour of BPPV and against a central lesion. Migrainous vertigo is often aggravated by changes of head position and may occasionally present with pure positional vertigo. The following factors help to distinguish migrainous positional vertigo from BPPV: short-duration symptomatic episodes and frequent recurrences, manifestation early in life, migrainous symptoms during episodes with positional vertigo.[10] A trial with antimigraine prophylactic agents is often required in a patient with migraine and recurrent vertigo, whether positional or not.

Conclusion

BPPV is one of the most common causes of vertigo. Diagnosis is straightforward if clinicians develop the healthy habit of doing positional manoeuvres (Hallpike or equivalent) as the single most important step in the diagnosis of patients with positional and recurrent vertigo. Treatment with repositioning procedures is effective and clinicians should get familiar with at least one of these manoeuvres (either Epley or Semont). The Semont manoeuvre is easily remembered: take your patient quickly in a big swing from the symptomatic ear down to the opposite eye down. You can see videos of these manoeuvres in www.imperial.ac.uk/medicine/dizziness

References

1. Pavlou M, Shumway-Cook A, Horak F, Yardley L, Bronstein A. *Rehabilitation of balance disorders in the patient with vestibular pathology.* In: Bronstein A, Brandt T, Woolacott M, Nutt JG, eds. *Clinical Disorders of Balance and Gait Disorders.* London: Edward Arnold; 2004:317-43.
2. Yardley L, Donovan-Hall M, Smith HE, Walsh BM, Mullee M, Bronstein AM. *Effectiveness of primary care-based vestibular rehabilitation for chronic dizziness.* Ann Intern Med. 2004;141(8):598-605.
3. Lempert T, Gresty MA, Bronstein AM. *Benign positional vertigo: recognition and treatment.* BMJ. 1995;311(7003):489-91.
4. Furman JM, Cass SP. *Benign paroxysmal positional vertigo.* N Engl J Med. 1999;341(21):1590-6.
5. Brandt Th, Bronstein AM. *Controversial Topics in Neurology: Cervical Vertigo.* J of Neurol Neurosurg Psychiatry. 2001;71:8-12.
6. Bronstein AM. *Benign paroxysmal positional vertigo: some recent advances.* Curr Opin Neurol. 2003;16(1):1-3.
7. Lempert T, Wolsley C, Davies R, Gresty MA, Bronstein AM. *Three hundred sixty-degree rotation of the posterior semicircular canal for treatment of benign positional vertigo: a placebo-controlled trial.* Neurology. 1997;49(3):729-33.
8. Woodworth BA, Gillespie MB, Lambert PR. *The canalith repositioning procedure for benign positional vertigo: a meta-analysis.* Laryngoscope. 2004;114(7):1143-6.
9. Radtke A, Neuhauser H, von Brevern M, Lempert T. *A modified Epley's procedure for self-treatment of benign paroxysmal positional vertigo.* Neurology 1999;53(6):1358-60.
10. von Brevern M, Radtke A, Clarke AH, Lempert T. Migrainous vertigo presenting as episodic positional vertigo. Neurology 2004;62(3):469-72.