Hands-on Course 3

Bedside examination of the vestibular and ocular motor system - Level 2

How to diagnose and treat BPPV

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Introduction

Benign paroxysmal positional vertigo (BPPV) is the most frequent and treatable vestibular disorder. Every neuro/otologist should become familiar with the positioning manoeuvres for diagnosis and treatment of this common condition. Diagnostic criteria and treatment for BPPV represent a key point due to the incidence of the disorders and its possible effects on quality of life in patients. The diagnostic criteria are well established for posterior and lateral canal BPPV. The last revision of these criteria that are part of the International Classification of Vestibular Disorders were published in 2015. Other recently proposed variants, like anterior canal BPPV, peripheral positional downbeat nystagmus and apogeotropic posterior canal BPPV still represent controversial entities.

BPPV is a common syndrome caused in most cases by displaced otoconia in the semicircular canals, making them gravity sensitive. It is characterised by brief episodes of vertigo, precipitated by rapid change in head position. Because of its anatomical position, the posterior canal (PC) is by far the most frequently involved; less often, otoconial debris enters the horizontal/lateral canal (HC) or move from one canal to another. Anterior canal (AC) BPPV is exceptional. Bilateral labyrinthine involvement is possible, especially after head trauma.
In most cases BPPV is idiopathic, but may follow head trauma or whiplash injury. Other predisposing factors include vascular inner ear disorders, acute unilateral vestibulopathy, prolonged inactivity/bed rest, chronic infection of the middle ear, inner ear surgery, Menière’s Disease, Migraine and vitamin D deficiency.

The management of BPPV relies on physical therapy, designed to free the affected canals of otoconial debris by displacing them into the utricle. Medical therapy is quite useless and vestibular suppressants help only to attenuate the nausea that often accompanies the vertigo. Surgical procedures are only used in persistent and incapacitating cases.

**Epidemiology**

BPPV can appear at any time from childhood to senility, but the idiopathic form is typically a disease of old age, peaking in the sixth to seventh decades. More than 95% of all patients are classified as degenerative or idiopathic (women:men = 2:1), whereas the symptomatic patients (women:men = 1:1) are most frequently caused by head injury (17%) or vestibular neuritis (15%). BPPV occurs often after extensive bed rest in connection with other illnesses or after operations. About 5% of the spontaneous patients and 10% of the trauma patients show a bilateral, generally asymmetrical BPPV. The right posterior canal is affected about twice as often as the left, which might be related to the fact that more people sleep on their right side.

**Patient History**

Benign paroxysmal positional vertigo is the most common cause of vertigo, not only in the elderly, with a lifetime-prevalence around 3%. The
symptoms are often stereotyped and a reliable diagnosis is often possible based on patient history: recurrent second-long attacks of rotatory vertigo triggered by certain head movements. “A few days ago, just seconds after I got out of bed, I felt the whole room spinning. It was so strong that I had to sit down again on the bed. I also had severe nausea, and thought that I must have eaten spoiled food. Lying back in bed, I had another episode of vertigo. In the following hours, I was only well when I kept my head still, but if I tried to turn in the bed or to get up, the spinning sensation returned again. The next day, I got up very slowly but I was again dizzy when I went to tie my shoes...I called my GP and he suggested that I have a brain MRI and X-rays of the cervical spine. Now I am better, but every time I get up or lie down in the bed, I see the room spinning. The spinning sensation lasts for a few seconds and stops if I’m still. Doctor, do you think I have a brain tumor?”. Often even the affected ear can be identified in this way. In most cases, when PC is involved, the episodes of vertigo occur in the morning, when the patient gets up, and in the evening, when he goes to bed. It may occur after a delay, when the patient is already on his feet, and this may cause him to fall over. This is common in elderly patients, who may suffer fractures because of the fall. Other triggering movements include hyperextension of the head, looking upward and bending forward. In patients with HC involvement the vertigo is triggered by rolling onto a side while lying down. Vertigo may be so intense and triggered by the slightest movement, that patients report a “spontaneous” vertigo and not a “positional” one. The vertigo is often accompanied by nausea and sometimes vomiting. Autonomic symptoms are more intense when the HC is involved. Some patients complain also unsteadiness and a feeling of walking on pillows.
Symptoms are recurrent, occurring every time the critical movement is performed, but generally over a limited period which may be a matter of days, weeks or exceptionally months. There are also borderline cases in which the vertigo occurs once or twice, and others in which it continues for years, if not treated. By definition, the syndrome is not associated with neurological symptoms. BPPV may determine anxiety and psychopathological mechanisms that produce non-organic symptoms. Many patients conserve a fear of vertigo and avoid certain head movements for a long time.

The diagnosis of BPPV requires positioning manoeuvres that result in a canal-specific positional nystagmus of PC, HC, AC, which are affected in this order of frequency.

**Diagnosis of Benign Paroxysmal Positional Vertigo of the Posterior Canal (PC-BPPV)**

**Diagnostic criteria:**

A. Recurrent attacks of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position

B. Duration of attacks < 1 min

C. Positional nystagmus elicited after a latency of one or few seconds by the Dix-Hallpike maneuver or side-lying maneuver (Semont diagnostic maneuver). The nystagmus is a combination of torsional nystagmus with the upper pole of the eyes beating toward the lower ear combined with vertical nystagmus beating upward (toward the forehead) typically lasting < 1 minute (Figure 1).

D. Not attributable to another disorder
It can be accompanied by nausea. BPPV is elicited positioning the head or body toward the affected ear (Fig. 1). Rotatory vertigo and nystagmus occur after such positioning with a short latency of seconds in the form of a crescendo/decrescendo course of maximally 30–60 seconds. The beating direction of the nystagmus is vertical and rotatory; it also depends on the direction of gaze, is primarily rotatory when gaze is directed to the undermost ear and mostly vertical (to the forehead) during gaze to the uppermost ear. In PC-BPPV the nystagmus corresponds to an (ampullofugal) excitation of the posterior canal of the undermost ear. Generally, the left ear should be tested before since the right side PC-BPPV is more frequent, in order to not miss bilateral cases. In patients with bilateral PC-BPPV, PN is upbeating and torsional toward the lower ear in both Dix-Hallpike positions.

Fig. 1: Dix-Hallpike manoeuvre for identification of posterior canal BPPV on the left. The manoeuvre starts with the patient-sitting upright with the head turned 45 degrees towards the examiner. The patient is moved to a lateral head hanging position. The lower part of the figure illustrates the canalolithiasis mechanism (UT=Utriculus, CU=Cupula, OT=Otoconia).
Pathophysiology and Therapeutic Principles

The canalolithiasis hypothesis can explain all symptoms of PC positional nystagmus. According to this hypothesis, the attacks are induced by otoconia that move freely in the semicircular canal. The movement of the conglomerate causes an ampullofugal or ampullopetal deflection of the endolymph depending on the direction of sedimentation and thus leads to a stimulation or inhibition of the vestibular hair cells. This model of the pathomechanism of BPPV can predict the direction, latency, duration and fatigability of the typical nystagmus, as well as changes in these parameters after head manoeuvres.

Latency. There is a latent period between reaching the diagnostic Dix-Hallpike position and the onset of nystagmus, in most patients in the range of 2-10 s. Generally shorter in the early stages of the disease, it is partially explained by the delay in setting the debris in motion.

Direction and plane. These are crucial for the diagnosis. When the patient is moved to the diagnostic Dix-Hallpike position, otoconial debris falls from its starting position in the canal toward the ground and away from the ampulla, so causing an excitatory stimulus and a mixed torsional-vertical paroxysmal nystagmus consistent with the excitatory connections of the PC to the vertical extraocular muscles. The fast phase of the vertical component beats towards the forehead (up) and the fast phase of the torsional component is directed such that the upper pole of the eyes beats towards the affected lower ear.
The torsional component may appear more prominent if or when the patient looks toward the lowermost ear, and the vertical component more prominent if the patient looks toward the uppermost ear.

**Intensity and Duration.** Paroxysmal means that PN is rapidly increasing in intensity and then begins to decay slowly. PN is typically transitory, that is, it dissipates in 10-40 s because, once the debris reaches the lowest point in the canal, the cupula returns to the primary position with its time constant, primarily due to (its) elasticity.

**Static Reversal of Nystagmus.** In some patients, when PN is particularly intense, a spontaneous reversal of its direction may occur, without any change in head position.

**Dynamic reversal of Nystagmus.** When the patient is returned to the sitting position, the particles fall back in the opposite direction and cause an ampullopetal flow, which produces an inhibitory response and a less intense nystagmus in the opposite direction, i.e. downbeating with the torsional component directed such that the upper pole of the eyes beats away from the affected ear.

**Fatigability.** The particles that form a plug or clump are loosely held together. During changes in the head position they tend to fall apart. Small particles cannot cause suction or pressure on the cupula independently of each other, as does a single clump with a diameter almost filling the canal. If the patient holds his head still for several hours (e.g., during sleep), the particles, which had fallen apart before, coalesce into a clump in the lowest place within the canal and again induce vertigo when the head position is changed.
TREATMENT OF PC-BPPV

Liberatory Manoeuvres. The efficacy of liberatory manoeuvres can only be explained by the canalolithiasis hypothesis, i.e., a clot that moves freely within the canal. By quickly turning the patient’s head to the opposite side, the plug is washed out of the canal and then can no longer cause any positioning vertigo. Brandt and Daroff in 1980 first devised an exercise programme, which, by means of simple head positioning, loosens the heavy degenerative otolithic material and distributes it into other areas of the labyrinth, where it no longer impairs canal function. The Semont’s liberatory maneuver is a simplified version of the original treatment suggested by Semont et al. (1988): from a side-lying position by a tilt of 180° to the opposite side. The liberatory maneuver provokes acceleration in the plane of the PC and should provoke the exit of debris from the canal into the utricle by centrifugal inertia. In 1992, Epley proposed another liberatory manoeuvre that involved turning the patient from a supine position into a head-hanging position.

Evidence-based reviews conclude that all manoeuvres are effective and can be explained by the mechanism of canalolithiasis. The success rate of the Semont as well as the Epley manoeuvre is around 90% after several applications (meta-analyses). There is evidence that these manoeuvre lead to a repositioning of the otoconia to the utricle.

Semont Manoeuvre (Figure 2). According to the liberatory manoeuvre developed by Semont - even before the mechanism of canalolithiasis was known. The examiner stands in front of the patient, who is seated on the examining bed and rotates his head 45° away from the pathological ear. Then, with a fast and continuous but not violent movement, the patient is
brought to a lying position on the side of the pathological ear, with the head turned 45° up. This position is similar to the Dix-Hallpike diagnostic position and triggers an episode of vertigo with typical paroxysmal nystagmus. The patient is kept in this position for two minutes and then is quickly turned onto the opposite side, maintaining the head in the same position in space. At the end of the manoeuvre the patient is lying on top of his shoulder with the cheekbone in contact with the bed (head 45° down). This is the liberatory position. The typical response to the liberatory manoeuvre is, after a variable latency, another episode of vertigo and a paroxysmal nystagmus with the same direction of rotation as in the diagnostic position. This is called liberatory nystagmus which is nearly always a good prognostic sign (10).

The nystagmus pattern is well explained by the movement of otoconial debris in the PC. When the patient is moved from the sitting to the diagnostic position, the debris fall away from the ampulla provoking an excitatory nystagmus (ampullofugal stimulus). When the patient is brought to the contralateral, liberatory, position the nystagmus in the same direction can only be due to a similar ampullofugal stimulus: the debris continue to move in the canal and are expelled into the utricle.

After 2 minutes the patient is brought to the sitting position with the head bent slightly forward. In this final position there is not usually any vertigo or nystagmus. If the liberatory manoeuvre doesn’t cause vertigo and nystagmus or causes a paroxysmal nystagmus beating in the opposite direction to that seen in the diagnostic position, the manoeuvre was probably unsuccessful. In these cases it is probable that a new bout of vertigo and nystagmus will occur in the sitting position.
The patient can be checked again with Dix-Hallpike test in an hour and this manoeuvre should be repeated until the patient is symptom-free. The success rate of the Semont manoeuvre is around over 80-90% with one treatment and 90-98% after several manoeuvres.

![Fig. 2: Semont manoeuvre for treatment of posterior canal BPPV on the left. The procedure is hand-guided by a therapist (not depicted here). All movements are performed swiftly.](image)

*Epley manoeuvre (Figure 3).* Epley’s repositioning manoeuvre consists of a five-position cycle, which has the aim to cause free canaliths to migrate by gravitation out of the PC through the common crus. The first position consists in bringing the patient into the Dix-Hallpike provoking position. Then the patient’s head is slowly rotated 45° towards the healthy ear. The head and body are then rotated so that the patient is prone with his head rotated at 180° with respect to the first position. With the fourth movement the patient is brought up to the sitting position and finally the head is turned forward with the chin down at 20°.
The manoeuvre provokes a nystagmus that reflects the direction in which the canaliths move, that is ampullofugal. Every position is held until the nystagmus is over and the five position cycle is repeated until no nystagmus is observed in the last cycle.

Major differences in the efficacy of the manoeuvres proposed by Semont and Epley do not emerge from the literature. Both manoeuvres are classified as class A treatment (highly recommended) according to the evidence based medicine.

Fig. 3:
Epley’s manoeuvre for treatment of posterior canal BPPV on the left.
The occurrence of nystagmus (so-called liberatory nystagmus) in the second step of the Epley manoeuvre indicates that the treatment will be successful.

The choice of the manoeuvre to be used should depend on which manoeuvre the therapist has experience with or if there are any individual contraindications. Very obese patients are easier to treat with the Epley method, while the Semont manoeuvre is suitable for patients with shoulder-neck problems.

Transient nausea can occur as an adverse effect, above all during repeated positionings within one sitting (prophylaxis with, e.g., 100 mg dimenhydrinate or another antivertiginous substance is indicated). About 20-40% of the successfully treated patients experience 1-3 days of light-headedness or postural vertigo with gait instability (most likely otolith vertigo) due to the partial repositioning of the otoconia toward the utricle. Occasionally a positional vertigo of the posterior canal converts into the horizontal or anterior canal variants during treatment.
Diagnosis of Benign Paroxysmal Positional Vertigo of the Horizontal Canal (HC-BPPV)

BPPV of the horizontal canal is less frequent than posterior canal BPPV but is still diagnosed too seldom. The diagnostic criteria for canalolithiasis of the HC (geotropic variant) are:

A. Recurrent attacks of positional vertigo or positional dizziness provoked by lying down or turning over in the supine position.

B. Duration of attacks < 1 min.

C. Positional nystagmus elicited after a brief latency or no latency by the supine roll test (Figure 4), beating horizontally toward the undermost ear with the head turned to either side (geotropic direction changing nystagmus) and lasting <1 min.

D. Not attributable to another disorder.

**Figure 4. The supine roll test.** When the head of the patient is turned on right side (right HC-BPPV) the geotropic nystagmus is more intense (ampullopetal stimulus) than when the head is turned to the left.
Its key features differ from those of posterior BPPV:

- It can be induced by turning the head along the longitudinal axis of the supine body (either to the right or to the left). This results in vertigo with horizontal geotropic nystagmus, i.e. nystagmus beating toward the ground in either head lateral position. Nystagmus is more intense when the head is turned to the side of the affected ear since for the lateral canal, the stronger response is due to an ampullopetal movement of the cupula.

- The duration of the attacks and the nystagmus is longer than in pc-BPPV (20-60 s) because of the horizontal canal’s so-called central storage mechanism of velocity. Positional nystagmus frequently shows a reversal of direction during the attacks; this corresponds to post-rotatory nystagmus.

- The positional nystagmus seems less susceptible to habituation with repeating the diagnostic maneuvers, even though this characteristic is often difficult to assess, due to nausea or vomiting. The latency of geotropic paroxysmal nystagmus is usually shorter than that of PC-BPPV, sometimes with no appreciable latency.

- The supine roll test is quite indispensable for the diagnosis of HC-BPPV and the affected ear is revealed by the direction toward which the most intense nystagmus beats. Sometimes, however, it may be difficult to appreciate a difference between the intensity of the two sides. Other diagnostic maneuvers, which should be performed before the supine roll test: the “pseudo spontaneous nystagmus” and the “bow and lean test” first described by Choung et al., in 2006.
If geotropic positional nystagmus is paroxysmal and transitory, diagnosis of HC-BPPV due to canalolithiasis is virtually certain and no differential diagnosis is required.

Typical HC-BPPV can also be explained by canalolithiasis, although occasionally the mechanism switches from canalolithiasis to cupulolithiasis.

In the rare form of HC-BPPV due to cupulolithiasis (the so called apogeotropic variant) nystagmus beats horizontally to the uppermost ear with weaker nystagmus when the head is turned to the affected side. (the “zero point” of positional nystagmus (beyond which direction changes) can be determined by turning the patient’s head 10-20° around the longitudinal axis while in the supine position; this is possible because the cupula of the ipsilateral horizontal canal is then parallel to the gravity vector. In this way one can also determine which side is affected by horizontal BPPV.

**Therapy for horizontal BPPV**

The first treatment described, the Barbecue rotation, involves rotations around the patient’s longitudinal axis while recumbent. In essence this is an altered version of the Epley manoeuvre. For canalolithiasis the supine patient is rotated in three 90°-steps around the longitudinal axis toward the healthy ear. The patient holds each position for 30.

The Force Prolonged Position or Vannucchi manoeuvre (Figure 5) is an alternative and very simple method: the patient has merely to lie on the healthy side for as long as possible (12 hours suggested). The patient is usually instructed to lie down, then to roll onto the side of the healthy ear and to stay in that position all night, if possible. This should cause the
otoconial debris to come out of the canal, by gravitation. If possible we check the result the next day. This method is particularly helpful in obese or very symptomatic subjects.

![Diagram](image)

**Fig. 5.** The force prolonged position for right side HC-BPPV canalolithiasis. Debris is forced to exit by gravity lying on the left side.

Alternatively one can perform the so-called Gufoni manoeuvre ([Figure. 6](#)), with which patients with either a canalolithiasis or a cupulolithiasis can be successfully treated. The advantage of this manoeuvre is that it is quick and it clears the labyrinth immediately. From a sitting position, the patient is simply laid down on the side exhibiting less nystagmus. It is advisable to create a good deceleration as the head makes contact with the bed. Afterwards the head is turned 45° downwards. The treatment should allow the particles to exit the canal under the centrifugal force created by the rapid deceleration, and by gravitation, when the head is maintained with the nose down.

The manoeuvre should be repeated two or three times sequentially. Gufoni’s treatment is a good option when the patient is moderately
tolerant to vertigo. Its effectiveness was recently validated in randomized double blind studies.

Fig. 6: Gufoni manoeuvre for treatment of horizontal canal BPPV (canalolithiasis on the left or cupulolithiasis on the right). Positioning is guided by a therapist. Each position is maintained for 30 seconds.

Additional treatments for BPPV

Self-treatment. The Epley and Semont manoeuvres as well as the Force Prolonged Position can be successfully applied by the patient himself. The treatments can be repeated daily until symptoms have disappeared. A thorough guidance by personal demonstration and an illustrated instruction is necessary. The success rates are not as high as when a physician performs the manoeuvre. Thus the self-therapy can be used in a complementary way, for example in patients with remaining complaints or frequent recurrences.
Recurrences after successful liberatory manoeuvres. According to follow-up observations over an average of 10 years, the recurrence rate in treated patients totals about 50%. Of these patients 80% have recurrences in the first year independently of the type of liberatory manoeuvre applied. Women have a rate of 58% and thus are more often affected than men who have a rate of 39%.

Additional Medication. Patients who develop severe nausea after a single manoeuvre can take an antiemetic, e.g. dimenhydrinate (100 mg) half an hour before undergoing the liberatory manoeuvre. Patients with excessive anxiety may require premedication with sublingual lorazepam or another benzodiazepine.

An association between osteoporosis, vitamin D deficiency and BPPV was described; this can probably also explain the predominance of women (2:1) in the idiopathic type of BPPV. Therefore, we determine in every patient vitamin D and if necessary substitute it.

BPPV of the anterior canal (AC-BPPV)

The existence and pathogenesis of canalolithiasis of the AC is still debated. It was suggested that a positional nystagmus from the AC can be elicited with both Dix-Hallpike maneuvers and even better in the supine straight-head hanging position, by bringing the patient to the supine position with the head 30° (or even more) below the earth-horizontal. For example, in the case of left AC canalolithiasis, the right Dix-Hallpike maneuver or the supine straight-head hanging positioning provokes a backward rotation of the left AC and the fall of otoconial debris away from the ampulla. The resulting positional nystagmus is mixed down-
beating and torsional with the top pole of the eyes beating towards the left pathologic ear and with the vertical component prevailing over the torsional component. The relative frequency of AC-BPPV is low.

Javocino et al. described a liberatory manoeuvre: from a midline head-hanging position, the patient must bow his head 30 degrees toward the chest and sit up after 1 minute. This study reported a success rate of 85% after one single manoeuvre. This high success rate does not correspond to our experience.

**Differential Diagnosis and Clinical Problems**

The diagnosis of BPPV can be made on the basis of a typical patient history (brief rotatory vertigo when turning over or sitting up/lying down in bed) and the clinical findings. Especially in patients with therapy-refractory positional vertigo (despite correct positioning exercises), the following syndromes should be considered in the differential diagnosis: central positional nystagmus (infrequent), vestibular migraine, bilateral BPPV, particularly post-traumatic (ca. 10%), BPPV of the horizontal canal (too rarely diagnosed, see above), and vestibular paroxysmia. Successful treatment represents the best differential diagnosis with central vestibular disorders.

Since HC-BPPV may present with spontaneous or “pseudo-spontaneous” nystagmus, differentiation from acute unilateral vestibular loss is mandatory. Apogeotropic variant of HC-BPPV needs differential diagnosis with central vestibular disorders.
**Central Positional Vertigo/Nystagmus**

Central positional vertigo and central positional nystagmus are caused by infratentorial lesions that affect connections between the vestibular nuclei in the medulla oblongata and cerebellar structures close to the midline (vermis). It is important to distinguish between peripheral and central vestibular disorders, as the Four characteristic forms of central positional vertigo/ nystagmus can be distinguished, although the symptoms overlap and combinations occur: central downbeat nystagmus, in head-hanging position (with or without accompanying vertigo), typically in lesions of the nodulus; central positional nystagmus (without vertigo); central paroxysmal positional vertigo with nystagmus, typically in nodulus lesions, and “central position vomiting”.

These central vestibular disorders occur much more seldom than typical BPPV. However, it can be difficult to distinguish peripheral and central disorders in an individual patient. The following clinical rules are important for diagnosing a central positional vertigo/nystagmus: persisting positional nystagmus (slow-phase velocity >5°/s) without associated vertigo; positioning-induced vomiting after single head movements without any substantial vertigo or nystagmus; positional vertigo with nystagmus of purely torsional or vertical character (downbeat or upbeat directions); a purely horizontal direction of nystagmus is typical for HC-BPPV; and positional nystagmus that does not correspond to the plane of the semicircular canal stimulated by the head positioning (e.g., torsional nystagmus after stimulation of the horizontal canal). In clinical practice the latter seems to be the most important feature by which a central positional nystagmus can be identified. According to traditional, positional nystagmus beating toward the uppermost ear or lasting longer than 1 minute indicated a
central pathology; this is no longer considered a reliable differentiating feature, as both features occur with the cupulolithiasis variant of BPPV.
Reference List


44. McClure JA. Horizontal canal BPV. J Otolaryngol 1985;14:30-35.


