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Review Article

Minimum Stimulus Strategy: A step-by-step diagnostic approach to BPPV

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ABSTRACT

Benign Paroxysmal Positional Vertigo (BPPV) is among the most common vestibular disorders, characterized by brief vertigo spells triggered by head position changes with abrupt onset and rapid decrease. BPPV is ascribed to otoconial matter dislodged from utricular macula and attached to the cupula of the affected semicircular canal (cupulolithiasis) or free-floating within its lumen (canalolithiasis).

According to the vestibulo-ocular reflex pathophysiology, each cupular deflection, either exciting or inhibiting the corresponding ampullary afferents, generates the contraction of specific extraocular muscles couples leading to pathognomonic nystagmus.

The Upright BPPV Protocol (UBP) is a diagnostic approach to BPPV conducted in the sitting position slowly bending the patient's head along the spatial axes, aiming to move canaliths by gravity within the involved semicircular canal, under continuous nystagmus monitoring by video-Frenzel goggles.

UBP starts with the evaluation of pseudo-spontaneous nystagmus in the primary gaze position and continues with the upright Head Pitch Test (uHPT) by forward and backward head bendings along the pitch plane. The uHPT can indicate whether horizontal or vertical semicircular canal is involved. If horizontal canal is suspected, the upright Head Roll Test (uHRT) usually provides the diagnosis of the involved side and arm by tilting the patient's head rightward and leftward along the roll plane. Conversely, canalolithiasis involving the posterior semicircular canal can be diagnosed with the uHPT alone. Nevertheless, if necessary, the diagnostic sensitivity can be increased by head movements along the right anterior – left posterior (RALP) and left anterior – right posterior (LARP) canal planes (uRALP/uLARP test).

Following the UBP, most BPPV form can be diagnosed in upright position, allowing clinicians to proceed immediately with proper physical treatment and avoiding unpleasant maneuvers to patients.

1. Background

Vestibular lithiasis is a mechanical disorder affecting the human posterior labyrinth: according to the most widely accepted theory it could be defined as a "maculo-canalopathy" where otoconial debris dislodged from the utricular macula and fall into one or more semicircular canals (SCs) resulting in an inappropriate activation of the involved ampullary receptors by linear accelerations such as gravity [1,2].

Benign Paroxysmal Positional Vertigo (BPPV) is the most common form of vestibular lithiasis and is among the most common vestibular disorders, presenting with a 10% cumulative lifetime incidence. Despite the BPPV peak of incidence occurs between 50 and 60 years of age, it might occur at any age, even in infants, with higher incidence among women (2:1–3:1) [3–5].

It is characterized by brief vertigo attacks lasting 15–60 s, triggered by head position changes (hence the term "positional") with abrupt onset and rapid decrease (hence the term "paroxysmal") [1–7]. The natural course of BPPV includes both spontaneous remissions, occurring within days or weeks, and a recurrence rate of 50% [2,8–11]. Although the favourable course (hence the term "benign"), BPPV might include disabling variants characterized by high recurrence rates and inadequate responses to physical therapy, resulting in a considerable personal and socio-economic burden [12–20].

Despite BPPV is usually an idiopathic disorder, it might be a consequence of either triggering events or conditions inducing otoconial

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detachment, such as head trauma, ear surgery, dental implant, and inner ear diseases [21–27]. In recent years, several studies suggested that calcium metabolism disorders related to vitamin D deficiency might increase BPPV recurrence [28–31].

The pathophysiological mechanism of BPPV is commonly ascribed either to free-floating particles within the SC (canalolithiasis) or to debris attached to the cupula of the affected SC (cupulolithiasis) [1–4]. According to the canalolithiasis theory, otoconia move back and forth within the canal due to head position changes and, by doing that, they displace the endolymph and the cupula. In this condition, otoconia, acting like a piston, push on the endolymphatic column provoking cupular deflections, thus generating either excitatory or inhibitory stimuli and short-lasting paroxysmal positional nystagmus and vertigo [32]. Conversely, in cupulolithiasis theory, otoconial debris adhering to the cupula rendering it heavy and gravity sensitive (heavy cupula). In this condition, both positional nystagmus and vertigo triggered by head movements present longer duration [1–4,32]. In both, canalolithiasis and cupulolithiasis, the cupula becomes sensitive to linear accelerations, including gravity, and to any acceleration provoked by brisk head movements along the same plane of the involved SC.

BPPV can be classified, in clinical practice, according to the affected SC and the involved arm, as follows [6,7]:

- Posterior Semicircular Canal (PSC) BPPV: Geotropic and Apogeotropic variants
- Horizontal Semicircular Canal (HSC) BPPV: Geotropic and Apogeotropic variants
- Anterior Semicircular Canal (ASC) BPPV
- Multicanal BPPV

The involvement of a single SC represents the most frequent condition, although BPPV might affect simultaneously more SCs on one or both sides. PSC-BPPV is the most frequent variant (80–90%), whereas cases involving the HSC and ASC account for 10–20% and 3% of all BPPV, respectively [6].

In addition, Canalith Jam (CJ) is an uncommon variant of vestibular lithiasis. In fact, it might occur whenever canaliths aggregate in a clot plugging the canal lumen and consequently leading to endolymphatic flow's blockage within the SC [33–36]. In CJ, the hydrostatic pressure between the otolithic clot and the cupula of the affected canal results in cupula persistent deflection, evoking a direction-fixed nystagmus regardless of head position. Therefore, in clinical practice, when a direction-changing nystagmus develops in a direction-fixed one, it suggests that CJ complicated a typical canalolithiasis [37–40].

The diagnosis of the affected canal in BPPV should always be based on the step-by-step observation of the evoked nystagmus rather than on the kind of maneuver used to provoke positional vertigo.

According to the vestibulo-ocular reflex (VOR) pathophysiology, each cupular deflection, either exciting or inhibiting the corresponding ampullary afferents, generates the contraction of specific extraocular muscles couples leading to pathognomonic nystagmus [41].

The BPPV diagnosis concerning the affected ear and the involved canal and arm is commonly made performing positioning tests, also known as diagnostic maneuvers. Diagnostic maneuvers move free-floating debris by using both gravity and inertial/centrifugal forces bending the affected SC cupula [3–7]. Several maneuvers have been proposed to properly diagnose each BPPV variant moving the patient's head along the plane of the examined SC. However, a diagnostic maneuver recommended for a specific semicircular canal investigation could evoke a nystagmus which features suggest another canal's involvement. Therefore, diagnosis should always be based on the evoked nystagmus rather than on the positioning test performed [1,2].

The typical paroxysmal nystagmus is more easily evoked by examining the patient after a short delay from the onset of symptoms, within the so-called “active period”, i.e., 24/72 h. Nevertheless, in this “active period”, the diagnostic maneuvers might hardly be conducted in patients suffering from intense autonomic and anxiety-related symptoms. These last conditions might prevent from detecting the affected side and the involved canal/arm, mainly in HSC-BPPV where repeated positionings are usually needed. In these cases, it is hard to rapidly make a certain diagnosis, especially at the first positioning examination. The “Minimum Stimulus Strategy” (MSS) represents a nystagmus-based approach aimed to reduce the number of diagnostic and therapeutic maneuvers required for BPPV management and patient's discomfort [42–46]. The pathophysiological principles and the steps of the MSS are discussed below browsing clinical features of each BPPV variant.

2. Minimum Stimulus Strategy: an overview

MSS represents a diagnostic and therapeutic approach to BPPV run with continue monitoring of the evoked nystagmus by video-Frenzel goggles according to the head position changes. A key point of MSS is the “Upright BPPV Protocol” (UBP), consisting of diagnostic tests performed by keeping the patient in sitting position bending his head slowly along the spatial axes in order to move canaliths by gravity along the involved SC (Fig. 1). In UBP each test has been properly named according to the spatial plane (roll, pitch and yaw plane) identified by the axis around which the head is rotated (X, Y and Z axis, respectively) (Fig. 2a). Moreover, considering the spatial orientation of the vertical SCs, two additional planes aligned with each couple of vertical SCs can be described: left anterior – right posterior (LARP) and right anterior – left posterior (RALP) (Fig. 2b,c).

The first step of the UBP is the evaluation of pseudo-spontaneous nystagmus (PSN) in the primary gaze eyes position, looking straight ahead during forward erect-head posture. Any purely horizontal persistent nystagmus observed in patients with a consistent history of BPPV, should be checked performing the upright head pitch test while sitting (uHPT). PSN is a direction-changing nystagmus, which changes its beating direction according to the head bending angle. Over 50% of HSC-BPPV cases presents with PSN [44–52].

Therefore, the uHPT (also called “Bow and Lean test”) represents the second step of the UBP (Fig. 3a). It is performed by slowly bending the patient's head 60° forward and then 30° backward from the horizontal plane, turning the head around the X axis along the pitch plane. The head should be held still in both positions until nystagmus appears (usually within 30 s). The uHPT modifies the direction of the previous detected PSN whereas if no PSN is observed uHPT can elicit horizontal direction-changing nystagmus reversing according to the head bending angle. [44–52]. Conversely, the uHPT might evoke vertical nystagmus with variable torsional components in BPPV affecting vertical SCs. Therefore, the uHPT represents the pivot point in the UBP algorithm [57,58].

The upright Head Roll Test (uHRT) is the UBP second step since it is performed after positive HPT (Fig. 3b) [47,48]. Accordingly, the patient's head is slowly bent about 30° laterally towards each side, turning around the Z axis along the roll plane, and, in so doing, bringing the patient's ear close to the shoulder of the same side while sitting. Even in this case, the head should be held still in each position until nystagmus appears (usually within 30 s). Once horizontal nystagmus (either geotropic or apogeotropic) is elicited, the same maneuver is performed towards the contralateral side to check if the nystagmus reverses its direction.

According to the literature, the HSC-BPPV affected side and its variant (geotropic or apogeotropic) can be identified in over 90% of cases matching data obtained from UBP test battery, even if the above-mentioned upright tests give incomplete results [47,48]. When UBP allows a reliable diagnosis of HSC-BPPV, we suggest proceeding with the appropriate therapeutic maneuvers. Otherwise, it is mandatory to continue evaluating the nystagmus evoked by bringing the patient supine (Supine Positioning Test - SSPT) [59,60] and performing the other positioning tests while supine (Supine Head Yaw Test - sHYT, also known as Supine Head Roll Test or Pagnini-McLure maneuver).

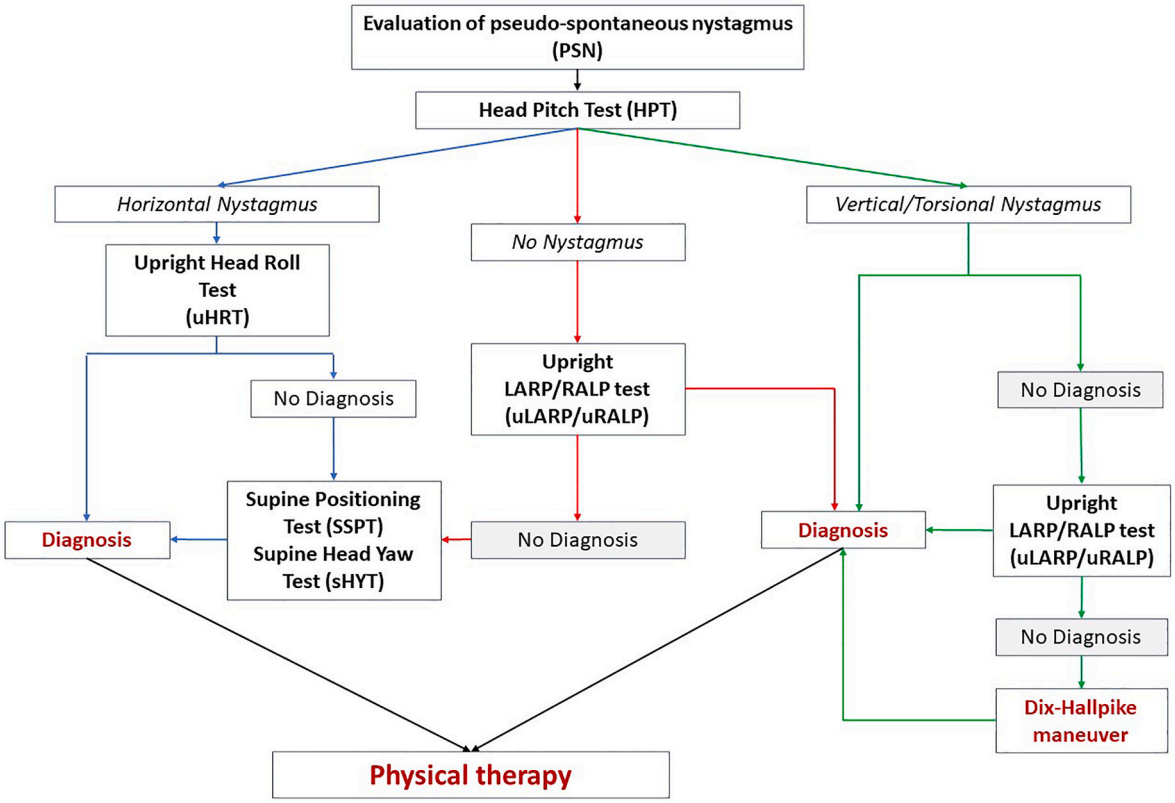

Fig. 1. Diagnostic algorithm for BPPV according to the Minimum Stimulus Strategy (MSS).

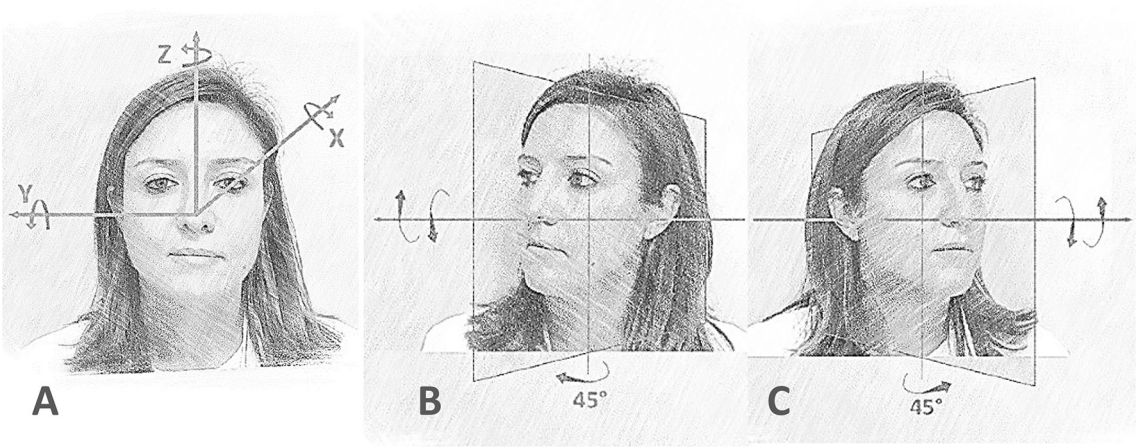

Fig. 2. a) Schematic overview of head rotations along the X, Y and Z axes. Left anterior – right posterior (LARP) (b) and right anterior – left posterior (RALP) canal planes (c) with corresponding head rotations.

In BPPV affecting vertical SCs, the uHPT might evoke vertical nystagmus, either up-beating or down-beating, with torsional components [57,58]. The uHPT in typical PSC-BPPV (also called geotropic variant) usually evokes a direction-changing nystagmus. Namely, down-beating nystagmus with torsional components towards the unaffected ear might be observed bending the patient's head forward, whereas opposite nystagmus (up-beating with rotational components towards the affected side) might be detected in backward head bending.

Conversely, in both apogeotropic form of PSC-BPPV and ASC-BPPV, both head bending along the pitch plane, forwards and backward, might result in down-beating nystagmus, whereas torsional components might be less easily detectable [61,64]. In these cases, the identification of the affected SC might be challenging, because none or very poor torsional components might be evoked also by performing the usual positioning tests such as the Dix-Hallpike maneuver. Moreover, the differential diagnosis between ASC-BPPV and contralateral apogeotropic PSC-BPPV might be sometimes doubtful even if any torsional component is detectable. In these conditions, the video Head Impulse Test (vHIT) is a valid key tool in identifying the affected SC [65,66].

The uHPT diagnostic sensitivity increases aligning the patient's head with the plane of the examined vertical SCs couple. We adopted the terms upright LARP test (uLARP) and upright RALP test (uRALP) to describe the positioning maneuvers conducted, while sitting, along the left anterior – right posterior and right anterior – left posterior canal planes, respectively, in accordance with the terminology commonly used for the vHIT (Fig. 3c). Right PSC-BPPV can be diagnosed

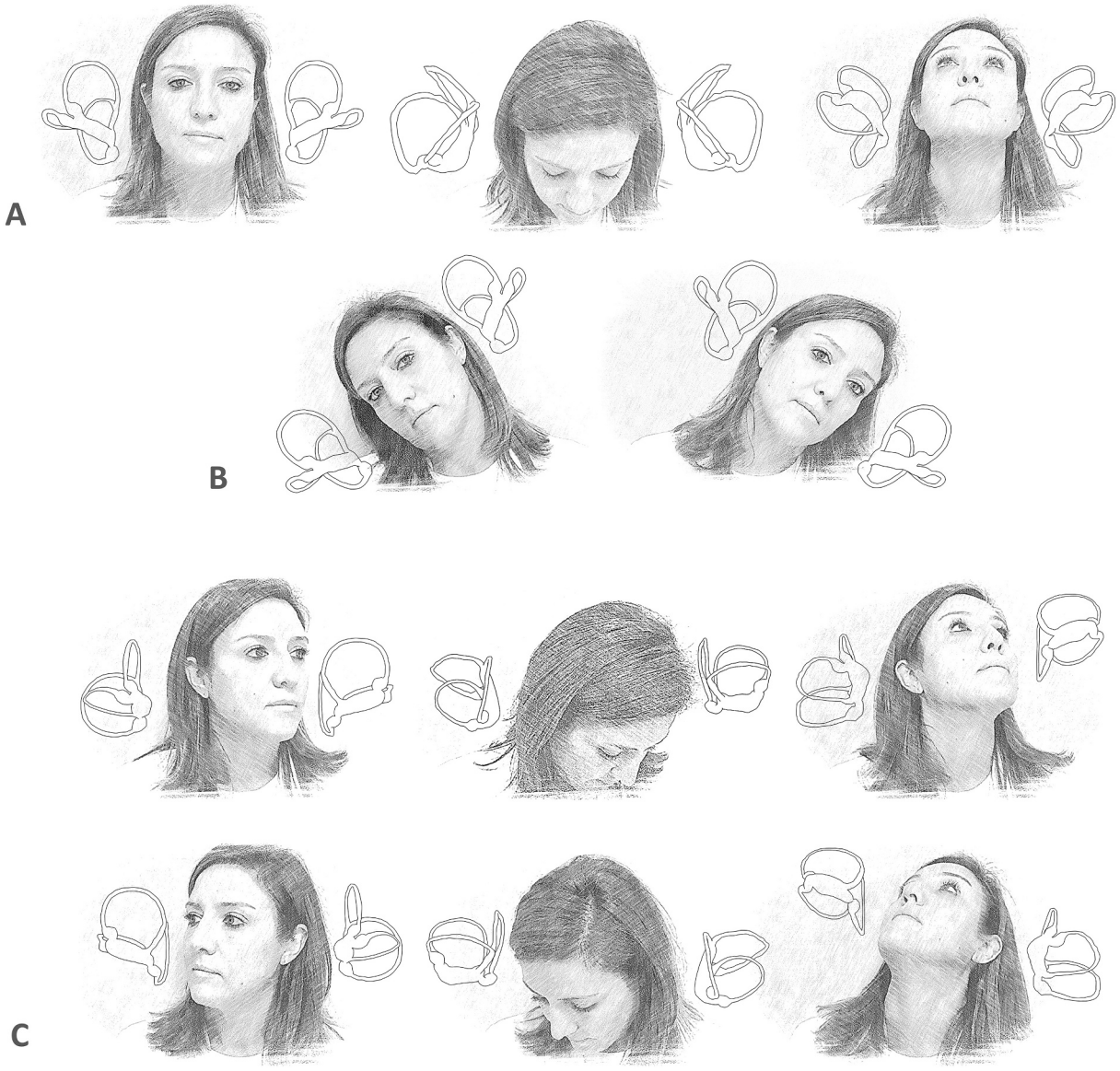

Fig. 3. Upright BPPV protocol (UBP). a) Upright Head Pitch Test (uHPT): the patient's head is bent forward and backward around the Y (interaural) axis and along the pitch plane. b) Upright Head Toll Test (uHRT): the patient's head is tilted rightward and leftward around the X (naso-occipital) axis and along the roll plane. c) uRALP/uLARP test: the patient's head is bent forward and backward along the RALP and LARP planes, respectively.

performing the uLARP test by slowly bending the patient's head backward and forward along the LARP plane, similarly, the involvement of the left PSC can be easily detected performing the uRALP test. Once the affected SC has been identified, it is possible to confirm the right diagnosis performing the ipsilateral Dix-Hallpike or Semont maneuver, and then, finally, performing the therapeutic maneuvers [67].

3. MSS in PSC-BPPV

The typical form of PSC-BPPV, also known as geotropic variant, accounts for around 80% of all BPPV diagnosis. Although most patients report the first positional vertigo while getting up from bed, positional symptoms recur frequently, by simply moving the head along the vertical planes. Therefore, the most of patients experience brief and recurrent short-lasting vertigo attacks (from 15 to 30 s), triggered by flexing or extending the head while upright, lying down on the bed, or turning the head towards the affected side while supine, and usually associated with any autonomic symptom, such as nausea and/or vomit. Geotropic PSC-BPPV is due to an otolithic mass freely floating within the ampullary arm of the posterior canal, thus modifying the acceleration sensitivity of the cupula, according to the canalolithiasis theory.

PSC-BPPV might follow an acute peripheral unilateral loss of vestibular function, which is sometimes responsible for massive otoconial detachment. In this case, the typical paroxysmal up-beating/torsional nystagmus evoked by ipsilateral positioning test (e.g., Dix-Hallpike) should likely overlap the spontaneous horizontal nystagmus due to the acute vestibular loss. Nevertheless, the association of these conditions, named “Lindsay-Hemenway syndrome” [68], might be missed as positioning tests are usually avoided in patients presenting with acute vertigo spontaneous nystagmus, because of the intense autonomic symptoms.

The diagnosis of PSC-BPPV is traditionally achieved by performing the Dix-Hallpike or the Semont diagnostic maneuver and detecting the typical positional nystagmus. This latter is generated by otoconial shift away from the cupula, thus resulting in an ampullofugal excitatory endolymphatic flow and typically lasts less than 1 min presenting with up-beating and ipsilesional torsional components. The eyes' position and gaze-direction are crucial points in evaluating positional nystagmus, as

torsional components could be more easily detected with the gaze towards the affected ear, while vertical components could be easily perceived looking to the opposite side.

The UBP algorithm allows the proper diagnosis of PSC-BPPV in most cases, particularly in recent-onset cases. The typical excitatory nystagmus (up-beating with torsional components towards the affected ear) is commonly observed bending the patient's head backward during uHPT, whereas opposite inhibitory nystagmus (down-beating with torsional components towards the healthy side) can be observed in forward head bending (Video 1).

When the uHPT is ineffective to detect any clear torsional components, the UBP diagnostic sensitivity is significantly increased moving the patients' head along the RALP and LARP planes in performing the uRALP and uLARP tests.

Since the RALP plane approximately aligns with the left PSC, in the uRALP test the patient's head is rotated 45° to the left side from the neutral position and then bent 60° forward and 30° backward.

In case of left PSC-BPPV, the uRALP test more easily induce either inhibitory or excitatory endolymphatic flows compared to the uHPT, since it moves the head along the plane of the affected canal resulting more effective in evoking either inhibitory (down-beating/right-torsional) or excitatory (up-beating/left-torsional) nystagmus (Fig. 4a). Contrarily, uRALP should not detect any eye movements in case of right PSC-BPPV, as both head movements and the gravity vector axis align with a plane orthogonal to the right PSC plane, preventing any otoconial movements.

Similarly, the uLARP test is conducted with the patient's head rotated 45° to the right side to align head bending and gravity with the right PSC plane. In case of right PSC-BPPV, forward head bending elicits inhibitory down-beating/left-torsional nystagmus whereas backward head bending results in up-beating/right-torsional nystagmus (Fig. 4b, Video 2). Accordingly, the uLARP is unable to detect any eye movements in case of left PSC-BPPV as both head movements and gravity axis align with a plane orthogonal to left PSC plane.

Our data showed that in typical PSC-BPPV correct diagnosis was achieved in 75.23% with the sole HPT and in 87.16% of cases adding uRALP/uLARP test. The diagnostic delay appeared closely related to the sensitivity of the UBP: the correct diagnosis was achieved in 100% of patients with symptoms occurred less than 7 days (64/64), reducing to 68.89% in patients with symptoms for more than 7 days (31/45) ($p < .001$) [67].

Once the affected side has been identified, it should be confirmed by proceeding with one of the traditional diagnostic tests (Dix-Hallpike or Semont maneuver). When performing the Dix-Hallpike, the patient's head is turned 45° towards the examined side to align the ipsilateral PSC with the sagittal plane, then the patient is rapidly brought supine with his head hyperextended approximately 20° off the bed. According to Semont diagnostic maneuver, the patient's head is rotated 45° towards the unaffected side to align the examined PSC with the frontal plane, then the patient is rapidly brought down face-up towards the tested side. In both these maneuvers, the typical paroxysmal nystagmus consequent to a transient excitation of the PSC appears after 3–15 s delay (Video 3). Once the PSC-BPPV diagnosis has been confirmed we suggest to immediately continue in performing the treatment. The most used therapeutic procedures for PSC-BPPV are the canalith repositioning procedure according to Epley (CRP) and the Semont liberatory maneuver.

4. MSS in HSC-BPPV

HSC-BPPV is the second most common type of BPPV, accounting for 15–25% of all BPPV diagnosis. In this case, positional vertigo can be triggered by head movements along the HSC plane and are usually more severe than PSC-BPPV, as they are typically associated with stronger autonomic symptoms and last longer than 30 s. The patient usually experiences his/her first acute vertigo attack when turning sideways in the bed. Nevertheless, subjects can experience mild transient dizziness whenever head movements force otoliths to float within the affected HSC, e.g., bending or turning the head while sitting, or lying down supine and turning head on one side [42,43].

The diagnosis is usually obtained by identifying the typical horizontal, direction-changing positional nystagmus evoked by the diagnostic maneuver known as “Supine Head Roll Test” or “McClure-Pagnini test” [2,5–7]. Since it is conducted turning the head 180° to either side along the yaw plane while supine, the name “Supine Head Yaw Test” (sHYT) seems more correct.

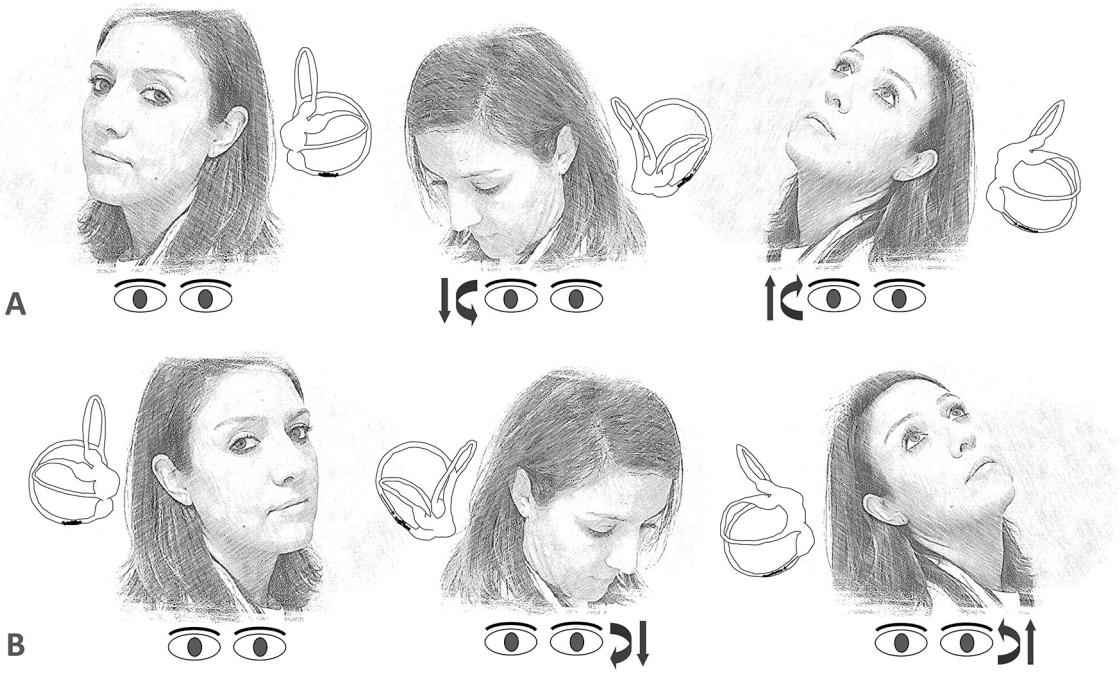

Fig. 4. a) uRALP test for left PSC-BPPV. b) uLARP test for right PSC-BPPV. The arrows within the canal represent the direction of endolymphatic flows, whereas the arrows beneath the eyes represent the direction of the fast phase of nystagmus.

In HSC-BPPV, nystagmus is purely horizontal, with variable directions according to the otoliths position within the affected HSC. Two forms of HSC-BPPV can be identified, depending on the direction of the nystagmus evoked by the sHYT [2–7]:

1. The geotropic form accounts for about 75% of all HSC-BPPV and it is due to debris floating along the non-ampullary (posterior) arm of the HSC ("geotropic canalolithiasis"). The otoliths rapidly shift towards the ampulla, shortly after rotating the patient's head towards the impaired side while supine, so generating a transient ampullopetal (excitatory) endolymphatic flow and paroxysmal geotropic nystagmus beating towards the downmost ear, which is the affected one. When the patient's head is turned on the opposite side, canaliths float back along the posterior arm of the HSC towards the utricle, thus resulting in a transient ampullofugal (inhibitory) endolymphatic flow and in a paroxysmal geotropic nystagmus beating towards the healthy side, the downmost ear one again. The evoked nystagmus always beats towards the ground direction, consequently, it is called "geotropic".
2. The apogeotropic form accounts for 25% of all BPPV involving the HSC, and it is due either to debris attached to the cupula ("cupulolithiasis" or "heavy cupula") or to free-floating particles within the ampullary (anterior) arm of the canal ("apogeotropic canalolithiasis"). When the patient's head is turned towards the affected side while supine, in case of cupulolithiasis the heavy cupula deflects towards the canal, due to gravity, in case of canalolithiasis canaliths gravitate away from the cupula. Both these conditions result in a ampullofugal (inhibitory) endolymphatic flow so evoking an apogeotropic nystagmus beating towards the healthy ear, the uppermost one. Conversely, when the head is turned to the healthy side, the cupula deflects towards the utricle or canaliths float towards the cupula. This results in an ampullopetal (excitatory) endolymphatic flow generating an apogeotropic nystagmus beating towards the affected ear, the uppermost one once again. The evoked nystagmus always beats away from the ground direction, consequently it is called "apogeotropic". Positional nystagmus usually persists longer than 1 min in cupulolithiasis, while it exhibits a paroxysmal course in canalolithiasis.

The diagnosis of the affected side by the sHYT is based on comparing the nystagmus intensity evoked on each side in accordance with Ewald's second law, postulating that the response to an excitatory stimulus, due to ampullopetal HSC cupula deflection, is always more intense than the one following an inhibitory stimulus, due to ampullofugal HSC cupula deflection. The sHYT always evokes the strongest nystagmus beating towards the impaired ear. Therefore, the affected side is the one where the nystagmus is more intense in geotropic variant, whereas the involved ear is the side where the nystagmus is weaker in apogeotropic form.

The nystagmus intensity difference in performing the sHYT is sometimes hardly detected [42–49]. In these cases, checking the patients more times by repeated repositioning tests could result in significant discomfort due to severe autonomic symptoms, especially in phobic and anxious subjects or in patients with recent onset of BPPV, thus preventing the proper diagnosis and treatment. The UBP protocol allows to avoid such complications [47,48].

The evaluation of PSN represents the first UBP step. Horizontal PSN can be detected in 50–75% of patients with HSC-BPPV. In fact, the HSC works as an inclined surface drawing a 30° front-open-angle with the horizontal plane, either allowing free-floating particles to gravitate along the canal (in canalolithiasis) or resulting in a persistent displacement of the heavy cupula (in cupulolithiasis). Therefore, the PSN, if detected in upright erected-head position, is generally directed to the healthy side in geotropic forms, as debris float away from the ampulla, and to the affected side in apogeotropic variants, as particles move towards the ampulla [42–52].

Bending the head backward during the uHPT, the angle between the HSC and the horizontal plane increases either enhancing PSN amplitude, if detectable, or eliciting nystagmus beating towards the healthy side in geotropic forms and towards the affected side in apogeotropic variants. Otherwise, PSN disappears when the head is bent 30° forward (neutral position) as the HSC aligns with the horizontal plane, while it reverses when the head is further bent forward. In case PSN is not detectable in head-erect position, forward head bending should elicit nystagmus beating to the affected side in geotropic variant and towards the healthy side in apogeotropic form [42–48,53–56].

Therefore, the uHPT allows clinicians to restrict the diagnostic hypotheses to only two options: the geotropic variant of one side or the apogeotropic variant involving the opposite HSC, however it does not allow to distinguish between these two. In this situation, the correct diagnosis can be achieved with the uHRT [47,48]. In fact, head bending towards each side allow to move debris due to gravity within the involved HSC eliciting positional nystagmus. This latter could be directed on both sides either towards the undermost ear in the geotropic form or towards the uppermost ear in the apogeotropic one. Therefore, the correct detection of the affected side and the canal arm in HSC-BPPV can be simply achieved combing the data provided by both upright tests: uHPT and uHRT.

For example, in case of canalolithiasis of the non-ampullary arm of the right HSC (i.e. right geotropic HSC-BPPV), the PSN, if detectable, should beat to the left side, whereas the uHPT should result in right-beating nystagmus in forward head bendings and left-beating nystagmus in head extensions. Finally, the uHRT should likely result in left-beating (geotropic) nystagmus in leftward head tilts and in right-beating (still geotropic) nystagmus in rightward head tilts (Video 4).

Conversely, in case of right apogeotropic HSC-BPPV either due to cupulolithiasis or to ampullary arm canalolithiasis, PSN, if present, should be directed rightward similarly to the nystagmus induced in backward head bending at the uHPT, whereas forward head flexions should result in left-beating nystagmus. Finally, the uHRT should provide the correct diagnosis leading to apogeotropic nystagmus tilting the head on both sides along the roll plane (Video 5).

Figs. 5–8 summarize all possible scenarios in HSC-BPPV.

In case of incomplete responses, we suggest to apply some slight inertial accelerations to the patient's head, if the nystagmuses observed in upright protocol have been poor and ineffective to achieve a convincing diagnosis, and in so doing we should start the otoconial mass to gravitate along the HSC performing both uHPT and uHRT.

According to our experience, correct diagnosis for HSC-BPPV was achieved in 95.5% of cases using exclusively the UBP, regardless of the time elapsed from symptom onset to diagnosis while the sole sHYT diagnosis in 85.1% of cases [47,48].

In case the UBP has been ineffective, other secondary signs of lateralization, such as the evoked nystagmus at the SSPT, should be sought. This nystagmus can be evoked bringing the patient supine from the sitting position along the pitch plane [43]. In this position, the HSC aligns with the gravity vector and otoliths gravitate along the involved HSC arm. Therefore, in geotropic variants, debris float away from the ampulla along the non-ampullary arm leading to an inhibitory endolymphatic flow resulting in contralesional nystagmus. Otherwise, in apogeotropic variants, either particles float towards the cupula along the ampullary arm, in canalolithiasis, or the heavy cupula bends ampullopetally, in cupulolithiasis, both resulting in excitatory stimulus and in ipsilesional nystagmus.

Once the nystagmus evoked at the SSPT has been detected, the sHYT is performed turning the patient's head 180° to either side.

Several therapeutic maneuvers have been proposed for HSC-BPPV, aiming to achieve the ampullofugal progression of the debris within the affected canal. These therapeutic techniques can be classified as it follows:

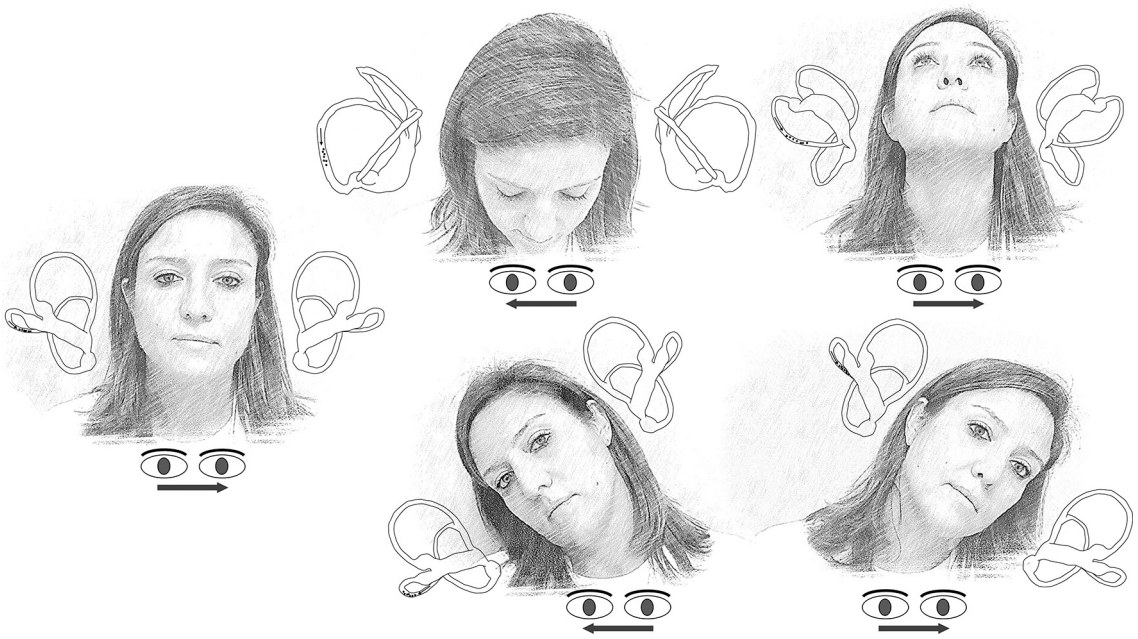

Fig. 5. UBP for right geotropic HSC-BPPV. Arrows within the canal represent the direction of endolymphatic flows, whereas arrows beneath the eyes represent the direction of the fast phase of nystagmus.

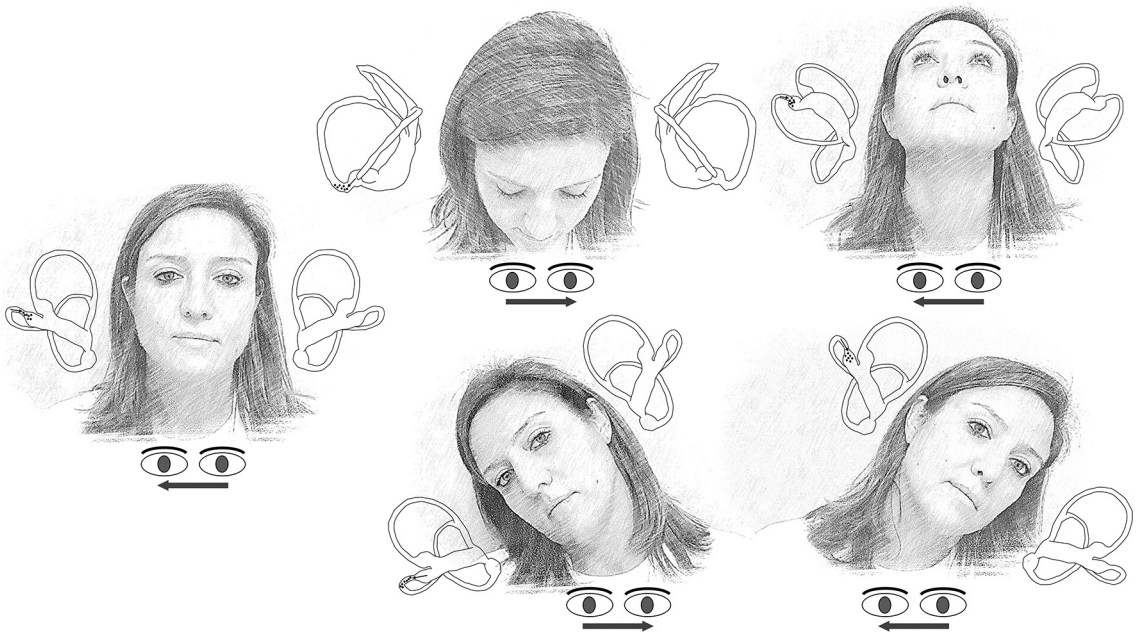

Fig. 6. UBP for right apogeotropic HSC-BPPV. Arrows within the canal represent the direction of endolymphatic flows, whereas arrows beneath the eyes represent the direction of the fast phase of nystagmus.

1. Techniques working by angular accelerations plus inertia (barbecue rotation techniques, Vannucchi-Asprella maneuver) [69,70].
2. Techniques working by gravity (forced prolonged position proposed by Vannucchi) [71].
3. Techniques working by linear accelerations plus inertia (Gufoni maneuver and its variants) [72,73].
4. Mixed Techniques (Asprella maneuver; Zuma maneuver) [2,71,74,75].

The description of each therapeutic technique goes beyond the aim of this review. However, no technique has shown to achieve better results compared the other according to clinical evidence. The choice of the therapeutic strategy is therefore decided by the examiner depending on his preference and experience. Nevertheless, in case the UBP provides unequivocal results allowing to establish a confident diagnosis, it should be recommended to proceed with a therapeutic procedure starting from the sitting position, such as the Gufoni maneuver.

5. BPPV presenting with positional down-beating nystagmus: a diagnostic dilemma

Positional downbeat nystagmus (pDBN) represents one of the most common findings related to central nervous system disorders involving the brainstem and cerebellum and usually present purely vertical

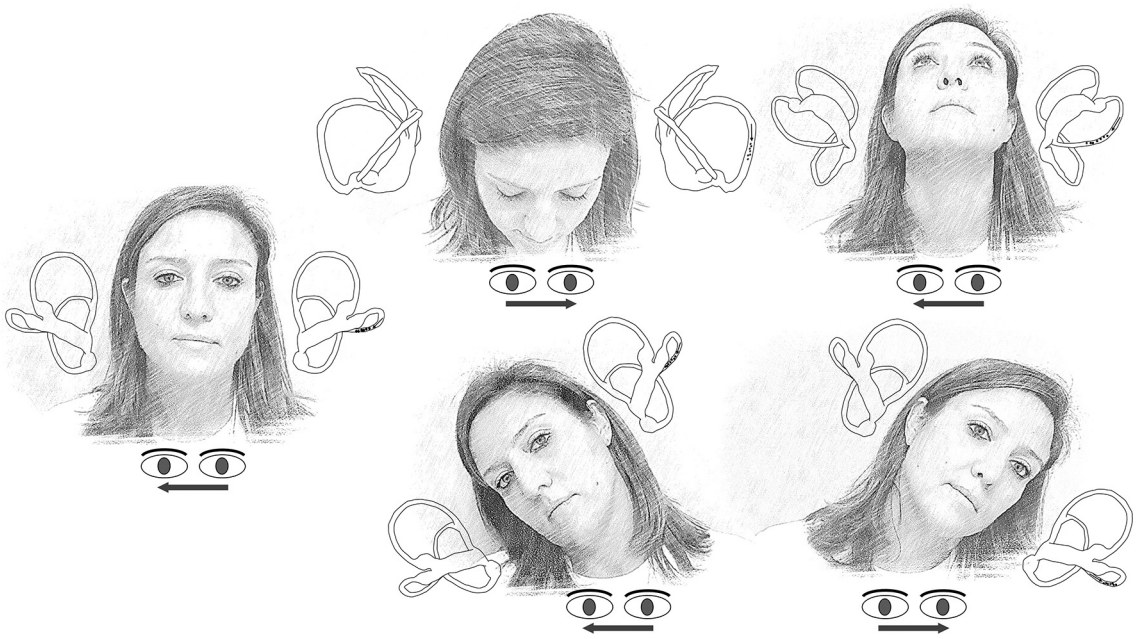

Fig. 7. UBP for left geotropic HSC-BPPV. Arrows within the canal represent the direction of endolymphatic flows, whereas arrows beneath the eyes represent the direction of the fast phase of nystagmus.

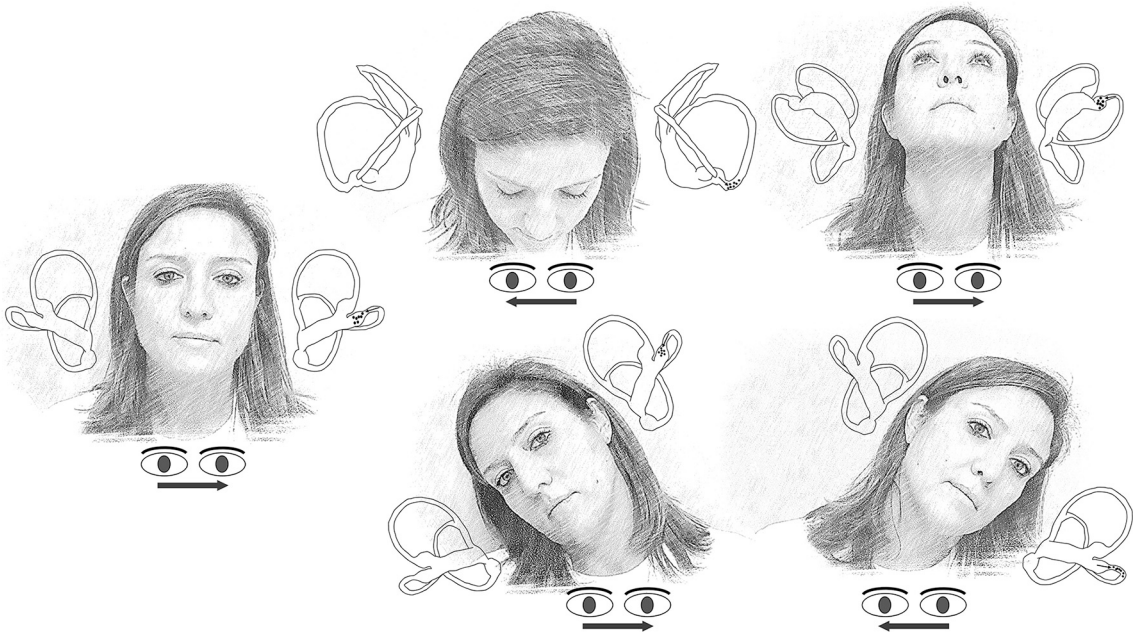

Fig. 8. UBP for left apogeotropic HSC-BPPV. Arrows within the canal represent the direction of endolymphatic flows, whereas arrows beneath the eyes represent the direction of the fast phase of nystagmus.

direction, long duration, lack of latency, fatigability, and no suppression with visual fixation.

Nevertheless, it has been widely demonstrated how pDBN may also occur in peripheral pathologies such as inferior vestibular neuritis and rarer form of vestibular lithiasis usually referred to as "atypical BPPV" [61–65].

The two forms of canalolithiasis associated with pDBN are ASC-BPPV, the most uncommon subtype of BPPV, and a recently described variant of PSC-BPPV known as "apogeotropic PSC-BPPV" (aPSC-BPPV). In this latter form, it has been hypothesized that otoliths settle in the distal portion of the non-ampullary tract of the posterior semicircular canal so that this variant has been named "apogeotropic variant" [62,63] as nystagmus evoked in provoking positions beats away from the ground and in the opposite direction to positional paroxysmal upbeat nystagmus due to classical PSC-BPPV.

An intensity-variable vertical down-beating can be elicited in both atypical forms when the patient is brought into the straight head hanging (SHH) position and by Dix-Hallpike test. Positional DBN associated with atypical BPPV is usually not paroxysmal with a torsional component less intense than the vertical one and not always clear. Furthermore, nystagmus does not invert its direction in returning to the sitting position [61–65]. The same nystagmus features can also be observed using the UBP: the down-beating nystagmus can be elicited during the HPT by bending the patient's head forward or backward,

while uRALP/uLARP test can sometimes facilitate the recognition of torsional components.

However, even in patients with a typical history of BPPV, finding a pDBN poses a diagnostic challenge since it is hard to identify the affected semicircular canal due to the possible missing torsional components of nystagmus. Furthermore, also in the presence of torsional components, ASC-BPPV is generally hardly distinguishable from a contralateral aPSC-BPPV as in both cases resulting nystagmus is generated by the contraction of the same ocular muscles (i.e., a patient presenting with pDBN with left beating torsional nystagmus could suffer from right aPSC-BPPV or left ASC-BPPV).

In a recent multicentric study, vHIT sensitivity in detecting the affected SC in patients showing pDBN was over 70%, suggesting that it should be routinely used to enhance diagnosis of these BPPV forms [65].

Demi-Semont (DS) maneuver, 45°-forced prolonged position (FPP) and quick liberatory rotation represent physical treatments proposed for aPSC-BPPV [62,63] while liberatory maneuver proposed by Yacovino (64) is the most used procedure for ASC-BPPV.

6. Conclusions

Most of BPPV diagnosis can be obtained in the sitting position following the UBP. In line with MMS principles, UBP can likely spare unpleasant maneuvers to the patients, allowing clinicians to proceed immediately with proper physical treatment.

Since nystagmus resulting from HPT and uHRT are produced by a low acceleration such as gravity, they are often weak. The major limitation of UBP is differentiating BPPV subtypes with paroxysmal nystagmus due to canalolithiasis from variants with persistent nystagmus due to a gravity sensitive cupula. This aspect may also be relevant in cases with direction-changing positional nystagmus due to central disorders mimicking BPPV, such as vestibular migraine. Therefore, carrying out diagnostic tests in the supine position is strongly recommended in patients with an atypical clinical history or if the therapeutic maneuvers are not effective.

Further investigation and instrumental tests such as vHIT are recommended in the framing of less common forms of BPPV such as those associated with pDBN.

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Disclosure

This research has never been previously presented and this manuscript is unpublished.

Conflicts of interest

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