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Apogeotropic variant of posterior canal benign paroxysmal positional vertigo

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Abstract. Sometimes debris can be located inside the posterior canal close to the common crus, resulting in apogeotropic posterior canal-paroxysmal positional vertigo (APC-PPV), which is characterized by torsional down-beating nystagmus (TDBNy) in the Dix-Hallpike position. The aim of this study was to investigate a differential diagnosis of the anterior canal variant, which is characterized by the same nystagmus direction. We selected 28 patients from among those referred for positional vertigo with TDBNy in Dix-Hallpike position to two Italian Balance Centers from January to August 2014. All of the patients underwent specific physical therapies aimed to cure APC-PPV: liberatory maneuver (LM, n=23) or forced prolonged position (FPP, n=5). All patients were checked within 3 days. The LM was effective in 20 patients: 13 recovered and 7 showed a typical posterior canal torsional up-beating nystagmus (TUBNy). The FPP had a positive outcome in 3 patients: 1 was symptom and sign-free and 2 had typical TUBNy. Thus, the described therapeutic techniques were successful in 82.14% of cases, either recovering the APC-PPV or transforming it into a typical posterior canal form. Both eventualities could allow us to distinguish the apogeotropic posterior canal variant from anterior canal-paroxysmal positional vertigo.

Introduction

Benign paroxysmal positional vertigo (BPPV) is the most frequent peripheral vestibular disorder.[1]

The pathophysiological mechanisms underlying BPPV are still a matter of debate, but it is generally related to otolith detachment from the utricular macula. Akkuzu et al.[2] and Gacek et al.[3] hypothesized a degenerative process of the saccular macula.[4] This vestibulopathy is generally accepted, with few exceptions (e.g., central vestibular dysfunction), to be a labyrinthine mechanical disorder due mainly to otoconia dislodged from the utricular macula either attaching to the cupula surface, which becomes gravity-sensitive (cupulolithiasis),[5] or conglomerating with other debris and free-floating inside a semicircular canal, usually the posterior canal (canalolithiasis).[6,7]

The posterior semicircular canal (PSC) variant is the most frequent, followed by the lateral semicircular canal (LSC) variant and, more rarely, the anterior semicircular canal (ASC) variant.[7-10]

Paroxysmal positional nystagmus (PPN) generally exhibits very typical features, which usually allow deduction of where the otoconial debris are and towards which direction they are moving. However, proper interpretation of PPN can sometimes be very difficult and misleading because it can mimic the involvement of another semicircular canal. The typical PSC-BPPV, with debris moving inside the ampullary arm, evokes a characteristic torsional up-beating nystagmus (TUB-PPN) in Dix-Hallpike[11-14] with two components: vertical up-beating, with the fast phase beating upward (to the forehead), and torsional beating towards the undermost ear, referring to the upper corneal pole (Figure 1). The typical PSC PPN is geotropic, which means that the torsional component beats to the right or left when performing right or left Dix-Hallpike, respectively, and always towards the earth. Nevertheless, we have observed a PSC-BPPV variant with torsional down-beating nystagmus (TDB-PPN) in Dix-Hallpike with two components: vertical down-beating, with the fast phase directed

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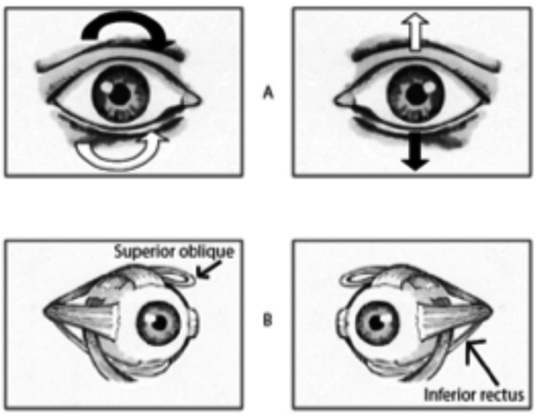

Figure 1

Torsional up-beating paroxysmal positional nystagmus (TUB-PPN) due to right geotropic posterior semicircular canal (PSC) benign paroxysmal positional vertigo (BPPV). (A) Black arrows indicate the direction of the nystagmus slow phase in the two eyes. White arrows indicate the direction of the nystagmus fast phase in the two eyes. (B) Arrows indicate the ocular muscles involved in nystagmus generation.

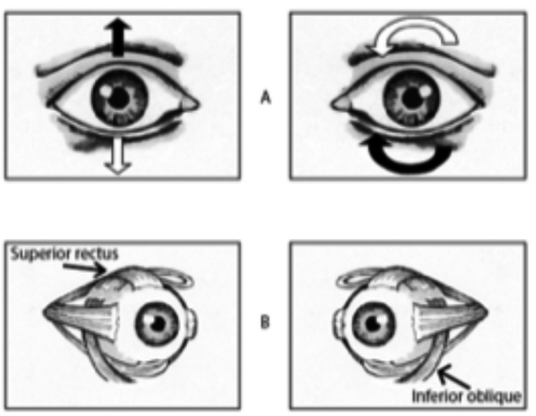

Figure 2

Torsional down-beating paroxysmal positional nystagmus (TDB-PPN) due to left apogeotropic posterior semicircular canal (PSC) benign paroxysmal positional vertigo (BPPV). (A) Black arrows indicate the direction of the nystagmus slow phase in the two eyes. White arrows indicate the direction of the nystagmus fast phase in the two eyes. (B) Arrows indicate the ocular muscles involved in nystagmus generation.

downward (towards the chin), and torsional beating towards the uppermost ear, referring to the upper corneal pole (Figure 2). This last nystagmus is apogeotropic, which means directing away from the earth in the provoking Dix-Hallpike positions.

To explain TDB-PPN of PSC-BPPV, we hypothesize that free-floating particles are located inside the distal portion of the non-ampullary arm of the PSC close to the common crus.[15] In such a case, when the patient is brought back into Dix-Hallpike positions, the debris move towards the ampulla, producing an ampullopetal endolymphatic flow. The resulting ampullopetal cupula deflection generates an inhibitory discharge of the posterior ampullary nerve and TDB-PPN due to contraction of the ipsilateral inferior oblique and contralateral superior rectus muscles. Thus, the linear component fast phase of the nystagmus is directed downwards; the torsional component referring to the corneal upper pole beats to the left or right shoulder in right or left PSC-BPPV, respectively (Figure 2).

However, an identical nystagmus could be generated by excitation of the anterior ampullary nerve of the opposite side, which evokes contraction of the same extraocular muscles, the inferior oblique and superior rectus, in this case contralateral and ipsilateral to the involved ASC, respectively.[16] Therefore, in this last condition, the vertical nystagmus fast phase is directing downwards and the torsional component referring to the corneal upper pole beats to the right or left shoulder for right or left ASC-BPPV, respectively, which is apogeotropic (Figure 3). Therefore, an apogeotropic variant of PSC-BPPV can mimic ASC-BPPV of the opposite side, evoking an identical nystagmic response.

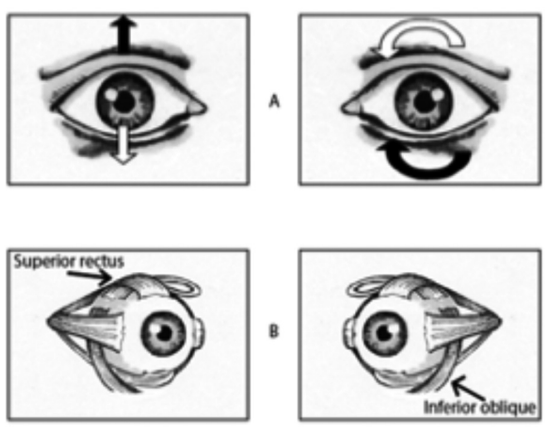

Figure 3

Torsional down-beating paroxysmal positional nystagmus (TDB-PPN) due to right anterior semicircular canal (ASC) benign paroxysmal positional vertigo (BPPV). (A) Black arrows indicate the direction of the nystagmus slow phase in the two eyes. White arrows indicate the direction of the nystagmus fast phase in the two eyes. (B) Arrows indicate the ocular muscles involved in nystagmus generation.

The aim of this study was to verify that debris located close to the common crus inside the distal portion of the non-ampullary arm of the PSC can move towards the ampulla when assuming the Dix-Hallpike position, eliciting an inhibitory torsional down-beating paroxysmal positional nystagmus identical to the nystagmic response due to ASC-BPPV of the opposite side. This study also aimed to suggest how to differentiate the PSC apogeotropic variant from ASC-BPPV of the opposite side. For this purpose, we compared two different specific techniques for apogeotropic PSC-BPPV and evaluated the effectiveness of these maneuvers to verify their success in resolving TDB-PPN by moving the debris towards the utricle or in transforming the TDB-PPN into TUB-PPN typical for geotropic PSC-BPPV by moving the debris towards the ampulla.

Material and methods

At the Careggi Hospital Unit of Audiology in Florence and the "Giovanni Paolo II" Hospital Unit of Vestibology and Otorhinolaryngology in Policoro (Matera), we enrolled 28 patients suffering from positional vertigo with TDB-PPN in the Dix-Hallpike position from January to August 2014.

The clinical features of all of our cases were consistent with the diagnosis of apogeotropic PSC-BPPV of one side or ASC-BPPV of the opposite side. Specifically, 18 patients had a TDB-PPN, in which the torsional fast phase component was directed with the upper corneal pole towards the left shoulder in right Dix-Hallpike, suggesting either right apogeotropic PSC-BPPV or left ASC-BPPV. In the remaining 10 patients, the torsional fast phase component of TDB-PPN was directed with the upper corneal pole towards the right shoulder in left Dix-Hallpike, suggesting either left apogeotropic PSC-BPPV or right ASC-BPPV.

All of our patients underwent specific physical therapies aimed and designed to move debris out of the canal only in the case of apogeotropic variant PSC-BPPV[17]; such maneuvers, though effective in moving debris, would have no effectiveness in pushing them outside the contralateral anterior canal.

In 23 patients, we used a liberatory maneuver called Demi Semont; in 5 patients, we used a 45° forced prolonged position (FPP). The term "Demi Semont" has been adopted because it represents the

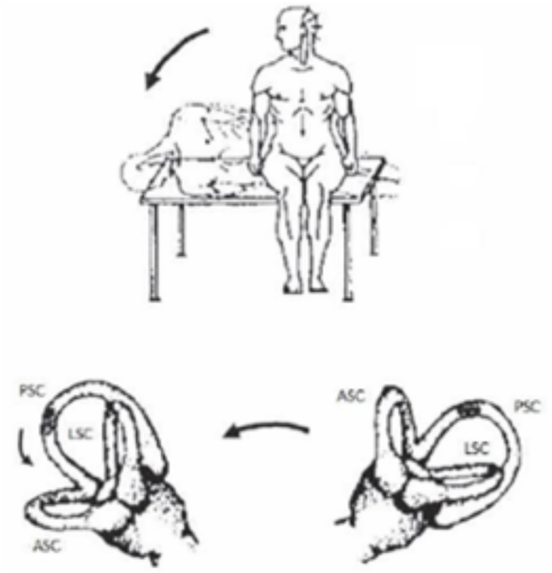

Figure 4

Schematic representation of the Demi Semont maneuver for left apogeotropic posterior semicircular canal (PSC) benign paroxysmal positional vertigo (BPPV). PSC: posterior semicircular canal; ASC: anterior semicircular canal; LSC: lateral semicircular canal.

second part of the well-known simplified Semont's maneuver.[18] The following sequence of positions describes how to perform such a maneuver in the case of left apogeotropic PSC-BPPV (Figure 4)[17]:

1) The patient sits in front of the examiner with his/her legs out of the bed and his/her head turned 45° to the healthy side (the right side in this case). The left PSC lies in the frontal plane in such a position.

2) The patient is brought down to the right, the healthy side. We suggest not moving the patient too briskly, to avoid ampullopetal endolymphatic flow, and applying final abrupt head deceleration to take advantage of the otoconia inertia in moving them towards the common crus and utricle. The examiner evaluates whether any liberatory nystagmus appears, which should be in the opposite direction with respect to the nystagmus previously detected in Dix-Hallpike (in this case, vertical up-beating and torsional with the upper corneal pole beating towards the left shoulder). On the other hand, in this same position, a torsional down-beating nystagmus can be detected due to debris coming through the last portion of the common crus, generating ampullofugal flow in the ASC of the same side.

3) After keeping the head in the same position for 20-30 seconds, the patient is brought back to

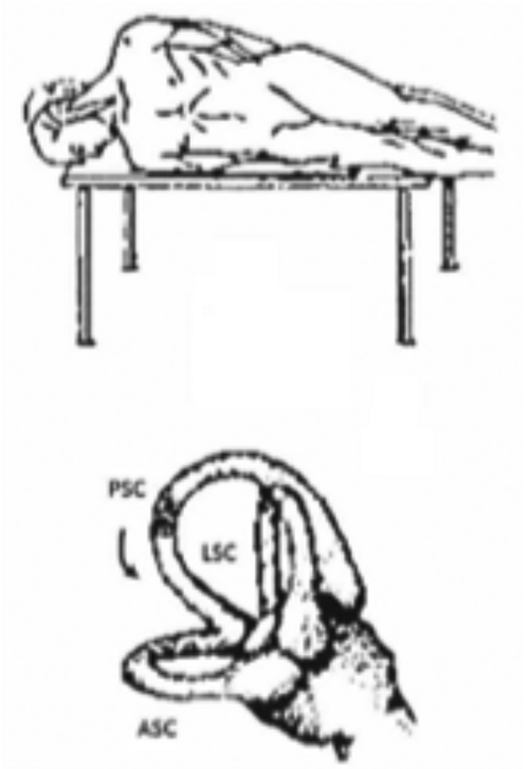

Figure 5

Schematic representation of the 45° forced prolonged position (FPP) technique for left apogeotropic posterior semicircular canal (PSC) benign paroxysmal positional vertigo (BPPV). PSC: posterior semicircular canal; ASC: anterior semicircular canal; LSC: lateral semicircular canal.

sit, with fast movement to move residual debris inside the canal towards the utricle due to the endolymphatic pressure. The examiner then bends the patient's head backwards while sitting to cause any remaining debris to fall into the utricle.

4) The sequence of movements is repeated at least 5 times.

5) The patient is asked to respect the same restrictions as for typical (geotropic) PSC-BPPV.

The 45° FPP is similar to the FPP procedure already described for LSC-BPPV.[19] This technique uses gravity to move the particles towards the utricle by simply arranging the PSC in a high position orienting the not-ampullary arm opening downwards. A schematic representation of this technique for left-sided apogeotropic PSC-BPPV is shown in Figure 5.[17] First, the patient is asked to lay on the their healthy side, the right in this case, with their head turned downwards 45°. The patient should remain in this position for at least 8 hours. Such a prolonged position could be completed only at home. We chose this technique to treat patients who have difficulties quickly moving due to their physical impediments.

All of the patients were checked within 3 days. The therapy outcome was considered successful in the following cases: patients were symptom and sign-free, or they transformed apogeotropic TDB-PPN into geotropic TUB-PPN.

Results

A total of 28 patients with TDB-PPN observed in Dix-Hallpike position were enrolled in this study. Eighteen of the patients were treated as right apogeotropic PSC-BPPV, with the torsional component of the nystagmus fast phase beating towards the patient's left shoulder in right Dix-Hallpike. The remaining 10 patients were treated as left apogeotropic PSC-BPPV, with the torsional component of the nystagmus fast phase beating towards the patient's right shoulder in left Dix-Hallpike.

Treatment outcomes are given in Table 1. Thirteen patients (65%) treated with the Demi Semont maneuver were symptom and signs-free, achieving complete resolution of BPPV. Another 7 patients (35%) presented with typical TUB-PPN, suggestive of a geotropic PSC-BPPV, with their apogeotropic PPN transformed into geotropic PPN. All of these patients subsequently resolved their geotropic PSC-BPPV by performing the Epley canalith repositioning procedure (CRP).[20] With 45°

Table 1

Results of physical therapy for apogeotropic PSC-BPPV

	Treated patients	Successful therapy	Cured	Transformed
Total	28	23 (82.14%)	14 (60.87%)	9 (39.13%)
Demi Semont	23	20 (86.96%)	13 (65%)	7 (35%)
45° FPP	5	3 (60%)	1 (33.33%)	2 (66.67%)

FPP: forced prolonged position. Data are given as n or n (%).

FPP, 1 patient (33.33%) was symptom and sign-free and 2 (66.67%) transformed into typical TUB-PPN. Both of these patients were subsequently treated successfully by the Epley CRP.

Discussion

Our patients were enrolled after a short delay from symptom onset, 3-7 days, to avoid the otoconial mass moving from the original position or the debris spreading out inside the involved canal. For the same reason, we did not use other techniques, such as habituation exercises. In addition, monitoring the evoked nystagmus during the course of treatment is crucial to properly understanding where the debris is and in which direction it is moving.

The morphological features of PPN in our select patients could suggest apogeotropic PSC-BPPV on one side, with otoconial particles floating inside the distal portion of the non-ampullary arm close to its common crus,[15] or ASC-BPPV of the opposite side, with debris floating inside the ampullary arm.[8,13,14] In both cases, patients would present with TDB-PPN in Dix-Hallpike positions, with the linear component fast phase beating downwards and the torsional component referring to the upper corneal pole beating towards the right shoulder for left apogeotropic PSC-BPPV or right ASC-BPPV, and towards the left shoulder for right apogeotropic PSC-BPPV or left ASC-BPPV. Unfortunately, it is impossible to distinguish the two pathologies on the basis of the evoked nystagmus. The TDB-PPN evoked by an inhibitory stimulation of PSC of one side has the same direction as the one generated by excitatory stimulation of ASC of the opposite side[21] because TDB-PPN results from contraction of the same extraocular muscles, the inferior oblique and superior rectus, which would be ipsilateral and contralateral to the affected PSC of one side, or contralateral and ipsilateral to the affected ASC of the opposite side, respectively. The PSC of each side and the opposite side ASC are coplanar and work in a push-pull mechanism to obtain the same compensating ocular movement, activating the same extraocular muscles.

Comparing the amplitudes of both linear and torsional nystagmus components observed in each eye is not effective in distinguishing whether the evoked TDB-PPN originates from the apogeotropic PSC-BPPV of one side or ASC-BPPV of the opposite side. When a TDB-PPN is evoked, the linear component in each eye is more evident in the case of contralateral PSC and ipsilateral ASC involvement, and the torsional component is more evident in the case of ipsilateral PSC and contralateral ASC involvement.

TDB-PPN of different origin cannot be argued even when observing the linear and torsional nystagmus component enhancement due to eccentric lateral gaze eye positions. Observing the TDB-PPN in each lateral gaze position, the nystagmus linear component increases in the case of contralateral PSC and ipsilateral ASC involvement, and the nystagmus torsional component increases in the case of ipsilateral PSC and contralateral ASC involvement.[21]

In most cases, it is difficult to distinguish whether the TDB-PPN is due to apogeotropic PSC- or ASC-BPPV of the opposite side, even considering the provoking positions. In many patients, each respective nystagmus may occur, bringing the patient down in any of the known head-hanging positions: right and left Dix-Hallpike, head-hanging position, and extra-extended head-hanging position.

Comparing the Dix-Hallpike evoked nystagmus intensity and the reverse nystagmus observed when coming back to sit may be theoretically useful for differentiating the TDB-PPN origin. As it is induced by an inhibitory stimulus, the Dix-Hallpike evoked TDB-PPN due to apogeotropic PSC-BPPV of one side should be less intense than the nystagmus evoked by the excitatory stimulus generated when coming back to sit, and vice versa for the TDB-PPN caused by ASC-BPPV of the other side. Unfortunately, in almost all of our patients, the nystagmus did not reverse its direction when bringing the patient back to sit, but persisted with the same direction for a short time or immediately disappeared.

It may be of interest in future studies to compare the slow phase angular velocities[22] in order to search for any difference in its intensity. Video-oculographic recording of a large number of TDB-PPN could be useful for distinguishing the PPN induced by PSC inhibitory stimulation, which are less intense, from the PPN induced by excitatory stimulation of the opposite side ASC, which are more intense.

In order to distinguish between the two different origins of TDB-PPN, our experience suggests attempting two options: try to resolve the TDB-

PPN or transform it into a typical PSC-BPPV with TUB-PPN, showing linear and torsional fast phase components with opposite directions with respect to the previous TDB-PPN. Both should be pursued by means of therapeutic maneuvers aimed to cure apogeotropic PSC-BPPV of one side and that would be ineffective in the case of opposite side ASC involvement.[17] Specific therapeutic maneuvers could either move the otoconial mass towards the common crus and utricle, resolving the TDB-PPN with disappearance of both positional nystagmus and positional vertigo, or move the debris from the non-ampullary arm towards the PSC ampulla, transforming the apogeotropic variant into the typical geotropic PSC-BPPV. Both of these conditions provide evidence that the first observed TDB-PPN was due to the involvement of PSC of one side, rather than ASC of the opposite side.

We used two different techniques, the Demi Semont, a fast maneuver, and 45° FPP, a static technique, both of which require previous identification of the affected side to suggest proper positioning, as both procedures specifically work on only one side of the posterior canal, unlike some maneuvers aimed to work simultaneously on the anterior canal of both sides, such as Yacovino's maneuver. Both techniques are specific for apogeotropic PSC-BPPV of one side[17]; such maneuvers would have no effectiveness in the case of ASC-BPPV of the opposite side. In the case of apogeotropic PSC-BPPV of one side, the debris would be pushed out of the canal when the patient is moved to the healthy side, whereas in the case of ASC-BPPV of the other side, our maneuvers would move the debris away from the ampullary arm of the anterior canal and not reach the common crus because of the very vertical position taken by the non-ampullary arm of the anterior canal when the patient is moved to the healthy side.

In our study group, 23 of the 28 patients with TDB-PPN surely had apogeotropic PSC-BPPV of one side, not ASC-BPPV of the opposite side, as TDB-PPN was complete resolved or transformed into typical PSC-BPPC by specific therapeutic maneuvers. Specifically, TDB-PPN was resolved in 14 patients, 13 of whom achieved resolution of TDB-PPN by means of the Demi Semont maneuver and 1 patient by means of the 45° FPP. On the other hand, TDB-PPN was transformed into typical PSC-BPPV in the remaining nine patients, seven by the Demi Semont maneuver and two by 45° FPP.

Conclusions

Canalolithiasis and cupulolithiasis are the most commonly accepted theories to explain the typical nystagmic patterns of BPPV. In addition, these theories can be used to understand some atypical forms of PPN, such as some BPPV variants mimicking other semicircular canal involvement, which could be wrongly interpreted as the involvement of the opposite site coplanar semicircular canal, such as in the case of apogeotropic PSC-BPPV. Such a PSC-BPPV variant demonstrates TDB-PPN in performing the Dix-Hallpike, mimicking ASC-BPPV of the opposite side.

We have developed two physical therapeutic techniques specifically designed to exclusively solve only the apogeotropic variant of PSC-BPPV,[17] as they act to move the otoliths eventually floating close to the PSC common crus. These techniques are effective in either resolving the TDB-PPN, or transforming it into TUB-PPN typical of geotropic PSC-BPPV. Both of the above conditions suggest that the TDB-PPN is most likely generated by debris gravitating through the distal portion of the non-ampullary arm of the PSC, close to the common crus, rather than through the ampullary arm of the ASC of the opposite side.

The preliminary results of these techniques appear to be encouraging and suggest that they differentiate between apogeotropic PSC-BPPV of one side and ASC-BPPV of the opposite side, showing success in 82.14% of treated patients. Based on these results, we propose using these two techniques in a first-line approach to positioning vertigo presenting as TDB-PPN in Dix-Hallpike positions. This may lead to finding that ASC-BPPV is less frequent than previously thought because ASC-BPPV is often misdiagnosed instead of an apogeotropic PSC-BPPV variant of the opposite side.

Similar to lateral canal canalolithiasis,[8,9] PSC canalolithiasis can exhibit two different nystagmic patterns: the most frequent is TUB-PPN, which is directed towards the earth in the provoking positions (i.e., geotropic), and the least frequent is TDB-PPN, and is directed away from the earth in provoking positions (i.e., apogeotropic). The two PSC canalolithiasis variants differ in regards to the otoconial mass location inside the canal; the geotropic PSC-BPPV variant is due to debris floating through the ampullary arm of the canal, and

the apogeotropic PSC-BPPV variant is provoked by debris floating through the non-ampullary arm of the canal, close to the common crus.

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