Benign Paroxysmal Positional Vertigo: A Lesson in Clinical Reasoning

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Key Clinical Message

Repositioning maneuvers for benign paroxysmal positional vertigo (BPPV) designed to induce otoconial movement in one canal can trigger or treat BPPV in other canals. We suggest for standardized testing protocols to ensure correct diagnosis of BPPV.

MeSH keywords : BPPV; positional vertigo; horizontal canal; positional nystagmus; case report

Background:

Benign paroxysmal positional vertigo (BPPV) is a common cause of episodic vertigo in which otoconia (calcium carbonate crystals) become detached from their normal location on the macula of the otolith organs.[1] They can then find their way into the semicircular canals (SCC), become free-floating or attached to the cupula, and create abnormal sensations of motion when the position of the head is changed with respect to the pull of gravity. BPPV of the horizontal canal (hcBPPV) is less commonly diagnosed than BPPV of the posterior canal BPPV (pcBPPV), in part because most emergency department and primary care physicians do not attempt to distinguish the canal variants.[2, 3] The supine roll test (SRT) is primarily used to diagnose hcBPPV, while the Dix-Hallpike test is primarily used to diagnose pcBPPV.[4, 5]

BPPV is usually treated with particle repositioning maneuvers that move the otoconia out of the SCC: the Epley or Semont maneuvers for pcBPPV and, for example, the Gufoni, Zuma, or Lempert (barbeque roll) maneuvers for hcBPPV.[4-7] We report here a woman with hcBPPV who came to the emergency department with acute positional vertigo and positional nystagmus and experienced a favorable outcome despite the wrong side and the wrong canal being diagnosed and consequently receiving the wrong treatment maneuver. To explain this seeming paradox, we simulated the movement of otoconia within the labyrinth in a right-sided hcBPPV in response to an Epley treatment maneuver for left-sided pcBPPV. Our simulations suggest that an extended Epley maneuver (i.e., the patient's head being turned beyond the conventional 45° angle to 60° in the second step) caused the otoconia to leave the horizontal SCC, resulting in this patient's successful outcome.

Case Report:

A 19-year-old woman came to the emergency department around noon because of acute vertigo with unsteadiness and light-headedness triggered by changing her head position. She woke up around 3:00 am with

vertigo on the day of the emergency department visit. Her symptoms partially subsided when lying in bed and not moving. She denied headaches, photophobia, migraines, vomiting, diarrhea, recent viral illness, head injury, loss of hearing, or trauma. She previously had a similar episode of vertigo that resolved on its own. She had no other relevant medical history.

With the present episode, the emergency department physician performed a Dix-Hallpike maneuver and diagnosed a *left-sided BPPV,*presumably of the *posterior canal* , though the details of any elicited nystagmus were not reported. The patient was treated with a*left-sided Epley* maneuver, and upon a repeat Epley maneuver, her symptoms and nystagmus were documented as resolved by the emergency department physician. During the first Epley maneuver, the patient developed vertigo when lying with her head turned to the left side. After her head was moved to point to the floor for two minutes more, her vertigo stopped. No imaging was obtained, and the patient was discharged directly from the emergency department with a diagnosis of a*left-sided BPPV* , presumably *pcBPPV.*

While the patient was in the emergency department, she was also enrolled in a clinical trial to assess her vertigo symptoms. A video-oculography (VOG) study was obtained by our research staff before undergoing any treatment maneuvers. The VOG study was not repeated after the emergency department physicians' treatment, as the patient had been discharged following the resolution of symptoms. The details of the recording and analysis are described in the legend of Supplemental Video 1. Hearing was normal.

Video Head Impulse Testing (vHIT) was performed for the horizontal canals, and gains were within normal limits without corrective saccades (head left, 0.83; head right, 0.97). With the VOG, with the patient sitting upright in the dark at center gaze, there was a right-beating spontaneous nystagmus of 1-2 °/s. In the supine position, there was a right-beating spontaneous nystagmus of 3 °/s. The cause of the weak spontaneous nystagmus on center gaze and in the supine position could have been from the residual effects of prior testing maneuvers, the presence of pseudo-spontaneous nystagmus, or be within normal limits.[8-12] In the chin-down (bow position), there was a right-beating sustained horizontal nystagmus (maximum slow-phase velocity [SPV] of 7°/s). In the right Dix-Hallpike maneuver, no nystagmus occurred. In the left Dix-Hallpike maneuver, there was a right-beating horizontal nystagmus that was sustained (maximum SPV of 12 °/s). In the SRT, in the right ear-down position, there was a crescendo-decrescendo right-beating nystagmus, lasting around 40 seconds (maximum SPV of 12 °/s), as shown in Supplemental Video 1. In the left ear-down position, there was a briefer decrescendo left-beating nystagmus, lasting around 15 seconds (maximum SPV of 13°/s), as shown in Supplemental Video 2. Since the chin-down position elicited right-beating nystagmus, a diagnosis of *right-sided hcBPPV* , with *geotropic* horizontal positional nystagmus was thought most likely.[13]

In summary, the diagnosis by the emergency department clinicians was*left-sided BPPV* , presumably of the *posterior canal* , for which they applied an "appropriate"*left-sided Epley* maneuver. However, after the emergency department visit, the expert review of the research VOG recordings found the overall picture was most consistent with *hcBPPV, geotropic variant, on the right side* . The BPPV was likely*right-sided* , based on the intense right-beating nystagmus induced in the bow position.[13] Thus, in the emergency department, the patient was diagnosed with the incorrect side, the incorrect canal, and applied the incorrect treatment. Nevertheless, the patient's symptoms and nystagmus resolved. Despite several diagnostic and treatment errors in this patient with BPPV, we wondered why the treatment worked, so we performed simulations to test the hypothesis.

Simulation Findings

Simulations of possible treatment maneuvers for this patient were created using a 3D model of the inner ear. This generic model (i.e., not specific to the patient described in this case report) was based on reconstructed images from high-resolution computed tomography of the temporal bone (Digital Imaging and Communication in Medicine files). This methodology has been described by Bhandari and colleagues.[14] The orientation of the canals and the angles between the canals were as previously reported.[15] The simulations allowed the placement of the debris in any of the canals and at variable positions within the canal. Given the uncertainty of whether the emergency department physician correctly performed the Epley maneuver on this patient, we

simulated both possibilities: a correctly performed Epley maneuver and an extended Epley maneuver.

Simulation 1 (Supplemental Video 3) shows the effect of a correctly performed left ear Epley maneuver on a right ear hcBPPV. When the subject is brought to the head-hanging position in the second step of the maneuver, the debris moves through but not out of the horizontal canal away from the ampulla, which could generate a left-beating horizontal nystagmus. When the subject's head is turned by 90° to the right side, the debris moves back through the horizontal canal toward the ampulla and could generate a right-beating horizontal nystagmus. Thus, performing the correct Epley maneuver with a hcBPPV of the opposite side causes free-floating otoconia to move within but not out of the offending horizontal SCC. The patient's hcBPPV symptoms would likely not be resolved.

Simulation 2 (Supplemental Video 4) showed the effect of a left Epley maneuver when the head is turned by 15° more than the usual 45° in the second step of the maneuver, in a right hcBPPV. In this case, when the subject is brought to the head-hanging position, the contralateral horizontal SCC becomes nearly vertical, allowing the debris to move not only within but also out of the right horizontal SCC under the effect of gravity, thus successfully treating a hcBPPV on the contralateral side before the patient is rolled over. Note this simulation also applies to the second stage of an extended Dix-Hallpike maneuver.

Discussion:

In sum, despite diagnosing the wrong side, the wrong canal, and applying the wrong treatment maneuver, the emergency department clinicians cured the patient's BPPV.

Among patients experiencing acute vertigo symptoms, hcBPPV is often underdiagnosed in favor of pcBBPV.[2, 3] While the Dix-Hallpike Test (DHT) is the preferred test to diagnose pcBPPV, other diagnostic tests, such as the supine roll test (SRT), are often needed so that a diagnosis of hcBPPV is not missed.[4-7] Our simulations emphasize the importance of performing these diagnostic tests using standardized maneuvers in a specific sequence. We have shown that the Dix-Hallpike maneuver can cause debris to move within the horizontal SCC, producing symptoms and potentially leading to a misdiagnosis of pcBPPV instead of hcBPPV. However, if the SRT is performed first, there will be little to no movement of particles within the posterior SCC, and even if there was movement in the posterior SCC, it would produce a vertical / torsional nystagmus pointing to the posterior SCC being the affected canal. This is consistent with the findings of Bhandari et al. (2022) and Li and Yang (2023), who showed that the sequence of performing the SRT steps can alter the position of otoconial debris within the canal as well as the resultant nystagmus patterns, which could lead to ambiguity of the type and side of a hcBPPV.[16, 17] Thus, following a protocol in which the SRT is performed first in patients with the triggered (positional) episodic vestibular syndrome should provide an optimal window to observe Ewald's 1st law in action ("the plane of nystagmus parallels the anatomic plane of the semicircular canal that generated it"), with pcBPPV producing vertical / torsional nystagmus and hcBPPV producing horizontal nystagmus.[18]

These issues raise the question of whether the approach to BPPV should change to clinicians first performing the SRT to optimally assess for hcBPPV. If the SRT is negative, clinicians should then perform the DHT to optimally assess for pcBPPV. Alternatively, clinicians could be trained to start with the DHT but, more specifically, observe nystagmus patterns of pcBPPV or hcBPPV since the DHT triggers nearly all pcBPPV and at least half of hcBPPV.[19] Lastly, this case also raises the possibility that the approach to BPPV could be simplified for frontline doctors since it is possible that single maneuvers can treat multiple variants. Future clinical trials about training physicians in the approach to BPPV could better address this issue.

In our patient, in view of the inaccurate classification of BPPV in the emergency department, the "curative" effect of the Epley treatment was initially surprising. The Epley maneuver, if correctly performed, should be specific for resolving pcBPPV.[6] There have been anecdotal reports, however, that hcBPPV may resolve with the Epley maneuver, but it is unclear how the Epley maneuver was applied in those cases. Our simulation of a left Epley maneuver with extended (i.e., head turned too far) angulation of the head successfully resolving a right hcBPPV is like a proposed treatment maneuver for hcBPPV that involves starting in the supine position, turning the head towards the healthy side (left in our patient's case) for 45°, then another 45°

towards the healthy side, and then sitting up.[20] The optimal repositioning maneuvers for hcBPPV and pcBPPV are well-documented in prior studies and should be encouraged in clinical practice.

Our patient illustrates that a successful outcome does not always depend on the correct classification of BPPV and correct treatment. While the adage, "It's better to be lucky than good," may have been at play in this case, benefitting this patient, our results should not be interpreted to suggest that clinicians need not strictly follow testing or treatment protocols. Rather, our results suggest the *opposite*– that placing the head in the correct position for a canal-plane-specific BPPV is critical to interpreting nystagmus and accurate clinical diagnosis.

Limitations of our study include the lack of precise knowledge of how the testing and treatment maneuvers were performed by the clinical staff in the emergency department, the lack of precise knowledge of the nystagmus patterns observed by the treating clinician, the unavailability of the patient for evaluation by our research team after treatment, and the lack of follow-up data on the long-term response of the patient. It remains possible that the patient initially had pcBPPV, and the treating clinician's repositioning moved the particles out of the posterior canal and into the horizontal canal – known as 'canal switch.' Furthermore, since there was only a small difference in the intensity of the horizontal nystagmus elicited in the two ear-down positions during the SRT, the localization of the offending labyrinth by the research team was primarily based on the response to the bow test. The lack of significant asymmetry between the two sides during the SRT could be related to the relatively low peak slow phase velocity of nystagmus induced in the SRT due to the relatively low velocity of the movements of the head to the ear down position.[21, 22] In this case, the head movements were not fast enough for Ewald's 2nd law – excitation produces a stronger response than inhibition – to take effect. The asymmetry between right and left ear-down positions may be better brought out by moving the head quickly to the ear-down positions. Additionally, our simulations assume an average anatomical orientation of the canals, the membranous labyrinth, and the cupula.[15] The variability of the orientation of the canals in the head, the membranous labyrinth within them, and the cupula among individual subjects; the presumed differences among subjects in the location and constitution (otoconia and sluffed otolith membrane) of the debris within the SCCs; and the speed at which the head is brought to a new position in the diagnostic and treatment maneuvers, can all contribute to the uncertainty of the classification of the canal subtype of BPPV and of the effects of different treatment maneuvers.[1, 21-24] Despite these limitations, our analysis and simulations are most compatible with diagnosing a right-sided hcBPPV, treated with an extended left Epley maneuver for a misdiagnosed left-sided pcBPPV.

Conclusion:

This case and our simulations emphasize that maneuvers that are designed to induce the movement of displaced otoconia in one specific canal can trigger or treat BPPV in other canals. Our study also raises the question of whether the diagnosis and treatment of BPPV could be improved if clinicians perform the SRT first and then if no nystagmus is elicited, perform the Dix-Hallpike maneuver. Finally, we recommend that clinicians place the head in the correct position for all diagnostic and therapeutic maneuvers, carefully observe and report the direction of the nystagmus, and always consider the location of otoconia within the canals and the possibility of other canals being involved when rendering a canal-specific diagnosis and treatment for BPPV.

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