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Atypical PC-BPPV – Cupulolithiasis and Short-Arm Canalithiasis: A Retrospective Observational Study

Janet O. Helminski, PhD

Background and Purpose: Atypical posterior canal (pc) benign paroxysmal positional vertigo (BPPV) may be caused by cupulolithiasis (cu), short arm canalithiasis (ca), or jam. The purpose of this study was to describe the clinical presentation and differential diagnosis of pc-BPPV-cu and short arm canalithiasis.

Methods: This retrospective observation study identified persons with atypical pc-BPPV based on history and findings from four positional tests. Patterns of nystagmus suggested canal involved and mechanism of BPPV. Interventions included canalith repositioning procedures (CRP).

Results: Fifteen persons, 17 episodes of care, met inclusion criteria, 65% referred following unsuccessful CRPs. Symptoms included persistent, non-positional unsteadiness, "floating" sensation, with half experiencing nausea/vomiting. Downbeat nystagmus with/without torsion in Dix-Hallpike (DH) and Straight Head Hang (SHH) position and no nystagmus upon sitting up, occurred in 76% of persons attributed to pc-BPPV-cu. Upbeat nystagmus with/without torsion and vertigo/retropulsion upon sitting up, occurred in 24% attributed to pc-BPPV-ca short arm. During SHH, canal conversion from pc-BPPV-cu to long arm canalithiasis occurred in 31%. The Half-Hallpike position identified pc-BPPV-cu in 71%. The Inverted Release position identified pc-BPPV-cu adjacent short arm and pc-BPPV-ca short arm.

Discussion and Conclusion: Persistent, peripheral nystagmus that is downbeat or downbeat/torsion away from involved ear in provoking positions and no nystagmus sitting up, may be attributed to pc-BPPV-cu, and nystagmus that is upbeat or upbeat/torsion towards involved ear upon sitting up may be attributed to pc-BPPV-ca short arm. Both are effectively treated with canal- and mechanism-specific CRPs.

Video Abstract available for more insights from the authors (see the Video, Supplemental Digital Content 1 available at: (http://links.lww.com/JNPT/A487).

Key words: *apogeotropic variant, benign paroxysmal positional vertigo, positional downbeat nystagmus, posterior semicircular canal, sitting up vertigo*

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INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is the most common cause of vertigo in the adult general population with an overall prevalence of 2.4%.[1] The posterior semicircular canal (pc) is most commonly involved with a frequency of occurrence of 85%[2] but more recently reported to be as low as 39%.[3] Positional downbeat (DB) nystagmus of peripheral origin is more common than was previously described with a frequency of occurrence of 15%[4] compared to 2%.[5,6] This suggests a possible increase in atypical BPPV variants and a need to identify and differentiate the pathophysiology of variants to successfully resolve BPPV.

BPPV is a mechanical disorder caused by otoconia from the utricular maculae becoming detached and displaced into one of the semicircular canals, rendering the semicircular canal sensitive to gravity. With changes in position of the head in the plane of the involved canal, debris within the lumen settles to the lowest portion of the canal causing motion of the endolymph and deflection of the cupula referred to as canalithiasis (ca).[7] Debris may be located within the long arm (la) or within the short arm (sa).[8] With changes in position of the head in the plane of the involved canal, debris adherent to the cupula deflects the cupula referred to as cupulolithiasis (cu) (Figure 1A).[9] It has been suggested that depending on the location of the debris within the short arm, debris could cause atypical patterns of nystagmus associated with vertigo or vertigo with no nystagmus referred to as Type II BPPV.[10] If debris is located inferiorly at the base of the pc cupula, or if it is attached to the pc cupula on the side near the utricle the cupula could be weighted.[10] If debris is in the right location, during/after

College of Health Professions, Rosalind Franklin University, North Chicago, Illinois

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Correspondence: Janet O. Helminski, PhD, College of Health Professions, Rosalind Franklin University, 333 Green Bay, North Chicago, IL 60064 (Janet.Helminski@RosalindFranklin.edu).

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sitting up from the ipsilateral DH position, the debris may cause a small deflection of the cupula resulting in complaints of positional vertigo but putatively small enough to preclude nystagmus. In addition, large fragments of particulate matter may move from a wide to narrow area of the canal lumen, or a constriction of the lumen may change the flow of endolymph and cause a "jam." The jam creates a positive or negative pressure between the obstruction and cupula leading to continuous deflection of the cupula and uni-directional, spontaneous peripheral nystagmus with intensity modulated by position of the head.[11] The changes in canal dynamics cause temporary canal hypofunction.[12] The hypofunction resolves with dispersion of the debris. The pc jam is reported to occur either in the lumen of the non-ampullary segment of the pc referred to as apogeotropic PC-BPPV[13,14] or in the lumen of the peri-ampullary segment of the long arm referred to as sitting-up vertigo.[15] It has been suggested that both types of pc jams present with inhibition of the afferent in either right or left DH positions or both, and the supine head hang (SHH) position, with either no nystagmus[13,14] or reversal of nystagmus upon sitting up.[15]

Deflection of the pc cupula away from the utricle causes excitation of the afferent generating a nystagmus that is upbeat (UB)/torsion toward the involved ear. Deflection toward the ampulla causes inhibition generating a nystagmus that is DB/torsion away from involved ear. The response is dependent on the location of the debris and the position of the head relative to gravity.

The diagnosis of BPPV is based on history and findings on positional testing.[16,17] To hypothesize the canal involved and location of debris, canal- and mechanism-specific positional tests should be administered and location of debris

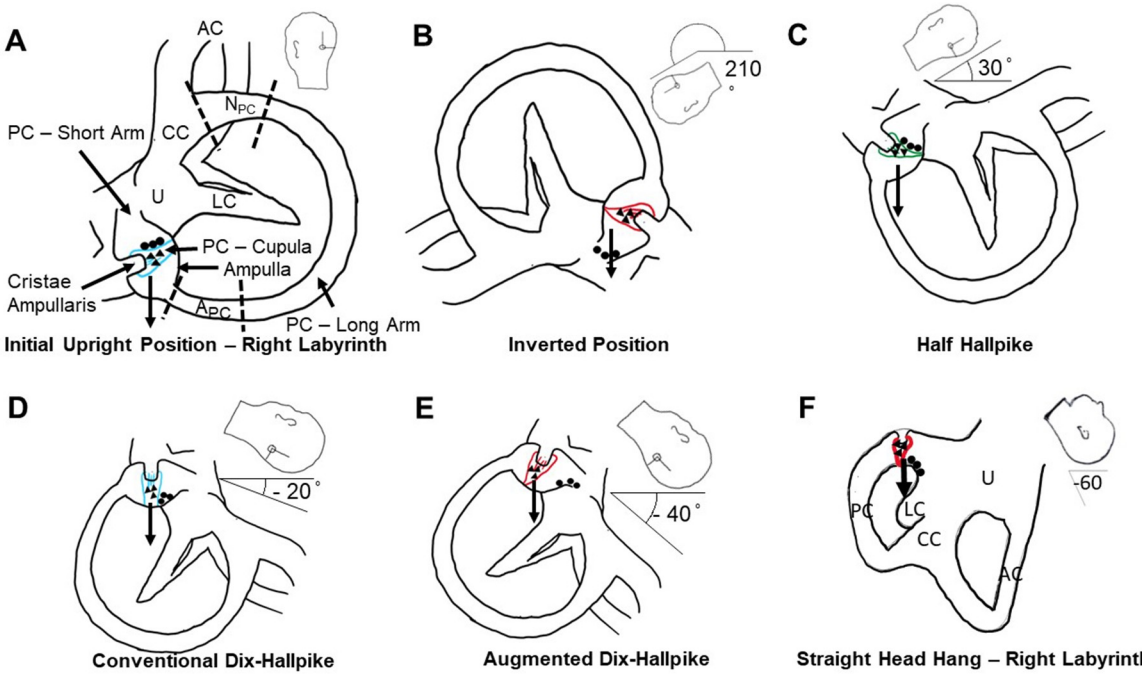

Figure 1. Useful test positions to identify atypical pc-BPPV-cu and pc-BPPV-ca of short arm. Illustrated for the right membranous labyrinth. Location of debris relative to cupula is adherent (▲) – cupulolithiasis and adjacent to short arm side (●) – short arm canalithiasis. The arrow indicates the line of gravity. (A) Initial position of the Dix-Hallpike Test (DH) and half-Hallpike position (HH) in sitting. To align the plane of right pc in the plane of gravity, rotate the head 45˚ toward the right – the side to be evaluated. The area of the pc between the common crus (CC) and ampulla is the long arm and between the ampulla and the utricle is the short arm. The pc long arm has two regions, A_{PC} – proximal segment near the ampulla and N_{PC} – distal segment near the CC. (B) Contralateral inverted release (IRc) position for the right cupula. In sitting, rotate the head 45˚ away from the involved side and position the person side lying on the uninvolved side with the neck flexed 20˚. If pc-BPPV-cu adherent short or long arm, or pc-BPPV-cu adjacent long arm, in the IRc the longitudinal axis of the cupula is in the earth horizontal plane and maximally deflects towards the ampulla inhibiting (red) the right pc afferent. (C) Ipsilateral Half Hallpike (HHi) position for the right cupula. In the supine recumbent position, the head is rotated 45˚ toward the side to be evaluated and positioned 30˚ above earth horizontal plane. (D) Conventional Dix-Hallpike (DH) position for the right cupula and no nystagmus due to absent cupular deflection. In the supine recumbent position, the head is rotated 45˚ toward the side to be evaluated and extended 20˚ below earth horizontal plane.[16] (E) Augmented Dix-Hallpike (DH) position for the right cupula. In the supine recumbent position, the head is rotated 45˚ towards the side to be evaluated and extended 40˚ below the earth horizontal plane.[23] (F) Straight Head Hanging (SHH) position for the right cupula. In the supine recumbent position, the head is in neutral rotation and extended 60˚ below the earth horizontal position. AC, anterior canal; LC, lateral canal; PC, posterior canal; CC, common crus; U, utricle; N_{PC}, non-ampullary or distal segment of pc; A_{PC}, periampullary or proximal segment of pc. This figure is available in color online (www.jnpt.org).

hypothesized based on the direction and characteristics of nystagmus in the initial and provoking position of positional tests, and knowledge of biomechanics of canals. Several factors influence the position of the longitudinal axis of the pc cupula relative to the gravitational forces which include the position and orientation of the head in provoking positions of positional tests, variability in location of pc attachment to the utriculus,[18] variability in position of the cupula within the ampulla,[18] location of debris on the cupula,[10] and debris adherent or adjacent to cupula.[10,18] Intervention success is dependent on identifying the involved canal and administration of canal-specific canalith repositioning procedure (CRP). An understanding of the pathophysiology of atypical BPPV is evolving.

The clinical presentation of pc-BPPV-cu was first described as a persistent, low amplitude excitatory response in the ipsilateral Dix-Hallpike (DH) provoking position and inhibitory response upon return to sitting.[18] Taking into account the longitudinal plane of the pc cupula and its alignment with the gravitational force during positional testing, Imai et al[19] first suggested that the clinical presentation of pc-BPPV-cu may actually be a low intensity, persistent, inhibitory response in either or both DH positions and SHH position[20,21] with no nystagmus upon return to sitting. In 2015, the operational diagnostic criteria of BPPV were published and described the clinical presentation of pc-BPPV-cu as a persistent excitatory response, UB with torsion towards the involved ear, brief or no latency of onset, lasting > 1 minute in the half Dix-Hallpike test (HH) (Figure 1C), no nystagmus in the DH position (Figure 1D), and an inhibitory response in the inverted release position (IR) (Figure 1B).[17] The ipsilateral half-Hallpike position (HH) – neck in 30° of flexion, putatively aligning the longitudinal axis of the weighted pc cupula in the earth horizontal plane resulting in excitation of the afferent (Figure 1C).[17,22] In a standard ipsilateral DH – neck in 20° of extension, the weighted pc cupula may be aligned with the gravitational force (Figure 1D),[16] resulting in no deflection of the cupula and no nystagmus.[17,22] If the head is moved further within the vertical plane of the involved pc into an ipsilateral augmented DH – neck in 40° of extension (Figure 1E),[23] the longitudinal axis of the weighted pc cupula may invert relative to gravity resulting in inhibition of the afferent.[24] The longitudinal plane of the weighted pc cupula may be aligned in the earth horizontal plane perpendicular to forces of gravity in 1 of 2 opposing positions, in the ipsilateral HH resulting in excitation of the afferent (Figure 1C),[17,18,22] and IR resulting in inhibition (Figure 1B).[17,18,22]

In the provoking position, the pattern of nystagmus observed due to deflecting the pc cupula in an inhibitory direction caused by pc-BPPV-cu is similar to that described of anterior canal (ac) BPPV-ca.[17] According to the operational diagnostic criteria for ac-BPPV-ca, the clinical presentation is a predominately DB nystagmus with immediate or brief latency of onset lasting < 1 minute in either DH position and the SHH position.[17] If a small degree of torsion is present, the torsion is always towards the involved ear.[23]

In this study, to determine the vertical canal involved and identify the pathophysiology, a series of positional tests were performed based on the diagnostic criteria of pc-BPPV-cu[17] (Figure 2). First, to identify vertical canal involvement, the plane of the pc is positioned in the plane of gravity.[16] In this study, the augmented DH was administered to assess vertical canal involvement as suggested by Aw et al.[23] In the supine recumbent position, the head is rotated 45° toward the side to be tested with the neck in 40° of extension.[23] With the standard DH position, the neck is positioned in 20° of extension.[16] If typical pc-BPPV, the pc cupula is deflected in an excitatory direction in the DH position resulting in UB nystagmus with torsion towards involved ear. Upon sitting upright, the cupula is deflected in an inhibitory direction resulting in DB nystagmus with torsion towards the uninvolved ear. If atypical pc-BPPV, inhibition in either DH positions would result in DB nystagmus with torsion towards uninvolved ear, or no deflection of cupula and no nystagmus. Upon return to sitting no nystagmus, deflection of the pc cupula in an excitatory direction results in UB nystagmus with torsion towards the involved ear, or inhibitory direction resulting in DB nystagmus with torsion towards the uninvolved ear may be observed.

If DB or no nystagmus is observed in the DH, the SHH position (Figure 1F) is performed to attempt to differentiate between ac-BPPV-ca and atypical pc-BPPV. In the supine recumbent position, the head is positioned in 0° of rotation and 60° of extension.[4] The SHH is sensitive in identifying ac-BPPV-ca. In the SHH, a DB nystagmus has been reportedly observed 100% of the time with ac-BPPV-ca and 75% of the time with atypical pc-BPPV variants.[4]

If findings suggest vertical canal BPPV – DB nystagmus in the DH or SHH position, then the second step is to determine the proposed pathophysiology by aligning the plane of the longitudinal axis of the cupula relative to the earth horizontal plane with the HH and IR. Findings from the HH and IR are used to predict the location of the debris relative to the side of the cupula and to determine if the debris is adherent or adjacent to the cupula.

If pc-BPPV-cu, the ipsilateral HH results in excitation of the afferent and the contralateral IR position results in inhibition, then the debris may be non-adherent to the cupula on the side of the long arm or adherent on the side of the long or short arm. (Figure 1C). If the ipsilateral HH results in excitation of the afferent and contralateral IR no nystagmus, then the debris may be non-adherent to the cupula on the side of the utriculus or within the short arm (Figure 1C).

With pc-BPPV-ca short arm, debris likely is adjacent to the cupula and moves within the short arm.[18] In DH and SHH, debris moves within the short arm or from the short arm into the utriculus, from a narrow to a wider area, resulting in no nystagmus (Figure 1E).[25] Upon returning to sitting, the debris moves back onto the cupula, the cupula deflects away from the ampulla, and the afferent is excited resulting in a nystagmus that is UB/torsion towards the involved ear. If during/after sitting up from the ipsilateral DH position, the findings of positional vertigo with truncal retropulsion and no nystagmus may suggest Type II BPPV.[10,26]

Step 3 is to determine the diagnosis based on findings on positional testing. The differential diagnosis of atypical vertical canal BPPV is central paroxysmal positional vertigo most frequently involving lesions to midline cerebellar structures,[27,28] posterior fossa mass lesions,[27] and vestibular

migraine.[29,30] The differential is based on timing and triggers of symptoms[31]; direction, characteristics, and temporal pattern of intensity of nystagmus found on positional testing[27]; associated neurological[27] and migraine[29] symptoms; and no response to therapeutic interventions within 2–3 sessions[16] (Table 2).

The pathophysiology of atypical pc-BPPV is uncertain therefore there is insufficient evidence to determine the preferred intervention. A theoretical review of typical and atypical BPPV,[18] two reviews on the current state of management of atypical pc-BPPV,[32,33] three-dimensional simulation models of ac-BPPV-cu,[34] and several cohort and cases studies on management of atypical pc-BPPV[10,13-15,22,24,35,36] exist. No comparison studies exist. Depending on the pathophysiology, CRP may be performed to intentionally cause an ipsilateral canal switch from pc-BPPV-cu to pc-BPPV-ca, or canal switch from ac-BPPV-cu to pc-BPPV-ca; to remove debris from the side of the short or long arm; or to reverse particles out of the jam.

Emerging evidence suggests that atypical BPPV variants may be due to pc-BPPV-cu or pc-BPPV-ca short arm.[18] If peripheral in origin, atypical vertical nystagmus may be identified using the augmented DH and/or SHH. The observed nystagmus may be categorized as persistent peripheral DB nystagmus in the provoking positions of one or both augmented DH and SHH positions, and no nystagmus upon return to sitting due to pc-BPPV-cu, or DB or no nystagmus in the provoking positions and UB nystagmus (UBN) upon return to sitting due to pc-BPPV-ca short arm[10,18,24,26,36] or pc-BPPV-cu short arm. Additional positional testing such as the HH and IR may be used to determine whether the debris is adjacent or adherent to the cupula and if adjacent on the side of the short arm. The purpose of this retrospective observational study was to describe the clinical presentation of two possible categories of atypical pc BPPV, pc-BPPV-cu and pc-BPPV-ca short arm, and to illustrate the clinical decision process to differentiate atypical vertical canal BPPV variants by determining if debris is adjacent or

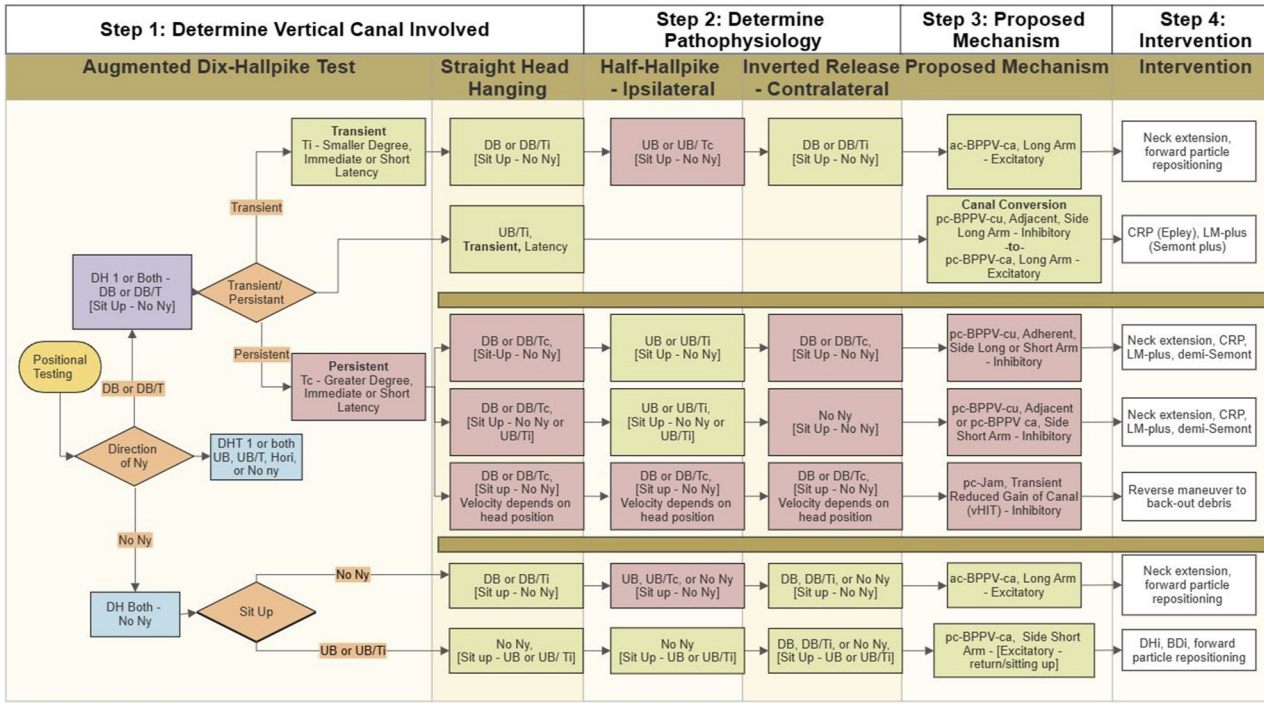

Figure 2. Clinical algorithm to assist in differential diagnosis and management of variant BPPV affecting the pc. Clinical findings that would suggest atypical vertical canal BPPV, potential diagnosis, and proposed intervention. Step 1 – Determine vertical canal involvement with findings on augmented Dix-Hallpike (DH) and straight head hang (SHH) position. Examine the vertical canals with the augmented DH.[23] Examine both sides to determine presence of nystagmus and direction and characteristics of nystagmus. The SHH position is always positive in persons with ac-BPPV-ca and positive in 75% with atypical pc-BPPV variant.[4] The SHH may also cause ipsilateral canal switch from pc-BPPV-cu to pc-BPPV-ca or canal switch from ac-BPPV-ca to pc-BPPV-ca. Step 2 – Use findings from half Hallpike ipsilateral (HHi) and inverted release position contralateral (IRc). Both sides are examined. Step 3 – Determine canal involved, side involved, and location of debris. Step 4 – Progression of suggested Canalith Repositioning Procedure(s) (CRP) for given diagnosis. c, contralateral; i, ipsilateral; ac, anterior canal; ca, canalithiasis; cu, cupulolithiasis; DBN, DB nystagmus; DCPN, direction changing positional nystagmus; DH, Dix-Hallpike Test; HH, Half-Hallpike Test; IR, Inverted Release Position; ny, nystagmus; NT, not tested; pc, posterior canal; SHH, Straight Head Hang Position; T, torsion nystagmus; UBN, UB nystagmus; vHIT – video head impulse test. Afferent response color of box: green – excitatory; blue – null; red – inhibitory. This figure is available in color online (www.jnpt.org).

adherent to the cupula and if debris is on the side of the long or short arm.

METHODS

This retrospective observational study conducted at Midwestern University's Multispecialty Clinic, outpatient physical therapy, between June 2018 and January 2022, met all institutional Health Insurance, Portability, and Accountability Act requirements, and was determined to be exempt by Midwestern University's Institutional Review Board. The sample was of convenience. Inclusion criteria were persons 18 years of age and older with a history of episodic positional vertigo and findings on positional testing of DB with/without torsion, or no nystagmus in the DH or SHH, and/or UB with/without torsion, or no nystagmus upon return to sitting. Exclusion criterion was central nervous system involvement. All participants underwent a neurologic screen,[37] comprehensive oculomotor examination,[38] and positional testing with the use of video-oculography (RealEyes xDVR Binocular Video Goggles, Micromedical Technologies, Chatham, IL). Positional testing included the augmented DH,[23] SHH,[20,21] HH,[17,22] and IR[17] (Figure 2), towards both sides. Positional testing towards the involved side was ipsilateral (i) and uninvolved was contralateral (c). The direction of the peripheral positional nystagmus may be influenced by the individual's position of gaze. For example, with right typical pc-BPPV in the right DH position, torsion is enhanced with rightward gaze and upbeat enhanced with leftward gaze. Therefore, individuals were asked to look towards the right and left to assist with identifying canal involved. For each position, documentation of subjective and objective findings occurred.

Based on the findings/symptoms on positional testing, two main categories of atypical BPPV emerged. The first clinical presentation, attributed to pc-BPPV-cu, was a low intensity, persistent nystagmus that was pure DB or DB/torsion towards uninvolved side in 1 or both augmented DH and SHH, and no nystagmus upon return to sitting from each position. A nystagmus that was pure UB or UB/torsion towards involved side in the HHi confirmed pc-BPPV-cu and side involved, and a nystagmus that was pure DB or DB/torsion towards uninvolved side in the IRc position suggested pc-BPPV-cu adherent or pc-BPPV-ca long arm. No nystagmus in IRc suggested PC-BPPV-cu adjacent to short arm or short arm canalithiasis, being unable to differentiate between the two. The second clinical presentation, attributed to pc-BPPV-ca short arm, was a nystagmus that was transient, pure UB or UB/torsion towards involved side associated with vertigo/retropulsion upon return to sitting from each augmented DH and SHH. With pc-BPPV ca short arm, in the augmented DH and SHH, either no nystagmus or a nystagmus that was DB/DB with torsion towards uninvolved side occurred with mild or no symptoms. The clinical presentation of ac-BPPV-ca was a transient pure DB/DB with slight torsion towards involved side in 1 or both positions of the DH and SHH and no nystagmus upon return to sitting.[17] If no nystagmus in both augmented DH positions, in the SHH, a pure DB/DB with torsion towards involved side suggested ac-BPPV-ca.

Intervention Protocol

Once the mechanism of BPPV was hypothesized, a canal- and mechanism-specific series of CRPs were administered based on knowledge of canal biomechanics over 1 or several sessions until nystagmus/symptoms resolved or no change in symptoms over 2-3 sessions (Figure 2). During each session, 2-3 repetitions of each maneuver, and 1-2 maneuvers were administered depending on response to treatment or neurovegetative response, usually nausea or vomiting. If a strong response occurred, the session terminated. If appropriate, self-administered interventions augmented the in-person treatment sessions. There is limited evidence to support the use of the maneuvers for treatment of atypical pc-BPPV. Maneuvers used were based on proposed pathophysiology and three-dimensional visual simulations of maneuvers. Some maneuvers were used for reasons other than their typical indication. For example, deep neck extension[39] was used to facilitate an ipsilateral canal switch from pc-BPPV-cu to pc-BPPV-ca long arm,[32] canal switch from ac-BPPV-ca to pc-BPPV-ca,[34] and to move debris from short arm into utriculus in pc-BPPV-ca short arm. If switched to typical pc-BPPV-ca, an intervention to remove debris from the long arm of the pc was selected such as a modified CRP[25] or liberatory maneuver-plus.[40] If diagnosed ac-BPPV-ca and side involved was known, then the forward particle repositioning maneuver was performed.[41] If a switch did not occur, and pc-BPPV-ca long arm or pc-BPPV-cu adherent was suspected, a CRP[25,40] was performed to remove debris from both sides of the cupula the positions of the head similar to the quick liberatory rotational maneuver.[32] If a person had pc-BPPV-cu adherent, with a strong neurovegetative response, to minimize response, only one side of the cupula was treated, usually the side of the short arm, with a demi-Semont[14] or 45º forced prolonged position,[14] both developed to treat a jam to non-ampullary segment of pc. To clear the long arm side, a reverse CRP was performed designed to treat a jam to peri-ampullary segment of pc.[15] With pc-BPPV-cu adjacent or pc-BPPV-ca short arm, debris was moved from the short arm to the utriculus with deep neck extension,[39] DHi,[10] Brandt-Daroff exercises ipsilateral,[42] modified forward particle repositioning maneuver,[41] or 45° forced prolonged position.[14] The forward particle repositioning was originally designed to treat ac-BPPV-ca.[41] If pc-jam, then maneuvers to reverse particles out of the jam were performed such as demi-Semont[14] or 45° forced prolonged position,[14] both developed to treat a jam to non-ampullary segment of pc and reverse CRP to treat pc jam to peri-ampullary segment.[15]

Activity restrictions were provided due to the complexity of BPPV[16] and were consistent with those provided to patients with intractable BPPV.[43] Activity restrictions consisted of sleeping elevated 30° on back or uninvolved side and avoiding vertical movements of the head for 1 week. A follow-up clinic visit occurred within 1 week. Positional testing occurred at the start of each session and findings were documented. Following each episode of care, symptoms and/or nystagmus were classified as "resolved" – absent symptoms/nystagmus, "improved" – reduced symptoms and nystagmus but still present, and "worse/no change" – increase or

no change in symptoms or no change or new vector of nystagmus.

Statistical Analysis

Descriptive statistics were used to present the demographic data, frequency counts, and proportions of reported symptoms and clinical features. Missing data were described as not tested and were included in calculations. All statistical calculations were performed using Microsoft 365 – Excel (Seattle, Washington).

RESULTS

Fifteen persons (13 female, 2 male) and 17 episodes of care (15 female, 2 male) met the inclusion criteria for atypical vertical canal BPPV (Table 1). They tended to be women (87%) with a mean age of 62 ± 16 years. The frequency of women affected compared to men was higher than the reported female:male ratio of 2 to 1.[44] Individuals with pc-BPPV-ca short arm were older than those with pc-BPPV-cu. Two had a history of migraine that did not meet the diagnostic criteria of vestibular migraine and one mild traumatic brain injury. The length of time of symptoms prior to treatment was 1.8 + 3.7 months. Prior to referral to vestibular specialty clinic, 11 (65%) persons received unsuccessful CRP. All data sets had results of DH and SHH, except for one to avoid canal switch. In 2019, included in the clinical protocol was HH and IR. Data were available for 12 episodes of care. The length of time across intervention was 0.49 ± 0.75 months. Eight episodes of care resolved within 1 session and 6 within 2 sessions. Two individuals discharged with partial relief were followed by the referring therapist. No patients lost to follow-up.

Distribution Incidence of Atypical pc-BPPV-cu and Short Arm Canalithiasis

Most persons (76%) presented with pc-BPPV-cu while a small group of persons (24%) presented with pc-BPPV-ca short arm. Most had right ear involvement (60%) (Table 1) (see Data, Supplemental Digital Content 1 (SDC: http://links.lww.com/JNPT/A486), which contains clinical description of each episode of care)

Table 1. Demographics, Symptoms, and Outcomes Associated with Posterior Canal Benign Paroxysmal Positional Vertigo (pc-BPPV) Cupulolithiasis (cu) and Short Arm Canalithisis (ca)

	pc-BPPV-cu	pc-BPPV-ca Short Arm
Number of cases	13(76%)	4(24%)
Female/male	12(92%):1(8%)	3(75%):1(25%)
Affected side right:left	9(69%):4(31%)	0:4(100%)
Age in years, AVE(SD)	59(±17.4)	71(±4.8)
Symptoms		
Floating	7(54%)	3(75%)
Nausea/vomiting	5(38%)	3(75%)
Imbalance	10(77%)	3(75%)
Vertigo transition supine to sit	5(38%)	4(100%)
Vertigo transition sit to supine	2(15%)	0
Vertigo with rolling	0(0%)	2(50%)
Fell during daily routine	0(0%)	2(15%)
Successful treatment		
Complete	9(69%)	2(50%)
Partial	4(31%)	2(50%)
No change or worse	0(0%)	0(0%)

Symptoms Associated with Atypical pc-BPPV

In both groups, the primary symptoms were severe, persistent postural unsteadiness and sense of "floating" (Table 1). Prior to referral, two people fell with routine activities, both diagnosed with pc-BPPV-ca short arm. The difference between groups was the symptom of vertigo. Vertigo was a primary symptom of people with pc-BPPV-ca short arm. All persons reported vertigo/retropulsion with transitions from supine to sitting, and half reported vertigo with rolling. Vertigo was described as a secondary concern for people with pc-BPPV-cu. Half complained of vertigo associated with transitions between supine and sitting. Severe nausea/vomiting occurred in over half of the persons within both groups. Two (12%) had recurrence of an atypical BPPV variant.

Clinical Features of Nystagmus

The primary vector of nystagmus of pc-BPPV-cu was either pure DB (50%) or DB/torsion towards uninvolved side (50%) in the provoking positions (Figure 3). Nystagmus was immediate or short latency to onset and persistent. Nystagmus occurred in both DH positions in 7 (54%) persons, in only the DHc in 3 (23%) persons, in only the DHi in 2 (15%) persons, and no nystagmus in both positions in 1 (8%) person. Upon sitting up, most persons had no nystagmus. In the SHH, nystagmus was either DB/DB with torsion (69%), or an ipsilateral canal switch occurred from pc-BPPV-cu to pc-BPPV-ca of long arm (31%). With a switch, there was a long latency of onset and an intense transient nystagmus that was UB/torsion towards involved side that suggested excitation of the afferent. Upon sitting up, a reversal of nystagmus suggested inhibition.

Data from HH and IR were collected from 7 of 14 possible persons. Seven persons had findings suggestive of pc-BPPV-cu, 4 had an ipsilateral canal switch, 2 presented with pc-BPPV-ca short arm, and 1 had minimal testing to avoid complications. Of the 7 persons with DB nystagmus in provoking positions, five had UB/torsion towards involved side in the HHi suggesting an excitatory response of the involved canal primary afferent. In the DHi, of those 3 had no nystagmus, two had pure DB, and 1 had DB/torsion away from involved side. Of the two remaining episodes, one had no nystagmus in HHi and the other had an UB/torsion towards involved side in both positions of HH, but only a DB nystagmus in SHH.

In all persons diagnosed with suspected short arm canalithiasis, an excitatory response occurred, nystagmus that was UB or UB/torsion towards involved side upon sitting up from all provoking positions. In the DH and SHH, the direction of nystagmus was not consistent. No ipsilateral canal switch occurred.

Intervention Response

Of persons with pc-BPPV-cu, 13(69%) had complete resolution and 4(31%) had partial resolution following

a CRP. This success rate is lower than previously reported success rates of 80-85% following CRP for typical pc-BPPV.[45] Two of the individuals with partial resolution were referred to the referring therapist for follow-up. Of the persons with short arm canalithiasis, 2(50%) had complete resolution of symptoms and 2(50%) had partial resolution (Table 1).

DISCUSSION

The mechanisms of atypical vertical canal BPPV proposed here are hypothetical, and the clinical findings between variants are similar with subtle differences making the differential diagnosis difficult. A series of positional tests is necessary to identify the pathophysiology of atypical vertical canal BPPV. During positional testing, several factors influence the position of the longitudinal axis of the cupula relative to the gravitational forces. First, is the position and orientation of the head in the DH. Modifications of the DH position may be unique to a person's limited spine mobility or pain. Second, variability in location of pc attachment to the utriculus is important.[18] If the short arm is attached more superiorly, the longitudinal plane of the cupula is aligned in the earth gravitational plane and no nystagmus is observed. If attached more inferiorly, the plane of the cupula is aligned beyond the earth gravitational plane and DB nystagmus is observed. Other factors include variability in position of the cupula within the ampulla,[18] location of debris on the cupula, and debris adherent or adjacent to cupula.[10,18]

The clinical presentations of two major categories of atypical pc-BPPV were described – one the direction of nystagmus DB with/without torsion towards the uninvolved side in provoking positions and no nystagmus upon return to upright, attributed to pc-BPPV-cu, and the other UB with/without torsion towards involved side upon return to upright, attributed to short arm canalithiasis. Once classified as atypical vertical canal BPPV, findings from the HHi and IRc were used to confirm the canal involved and determine location of the debris.

The differential diagnosis is critical. The origin of positional DB nystagmus may be due to central nervous system disorders, mainly of the cerebellum or posterior fossa[16] or vestibular migraine,[29,30] or peripheral disorders (Table 2).[18,32,33] Positional DB nystagmus of peripheral origin is more common than was previously described with a frequency of occurrence of 15%[4] compared to 2%.[5,6] Peripheral

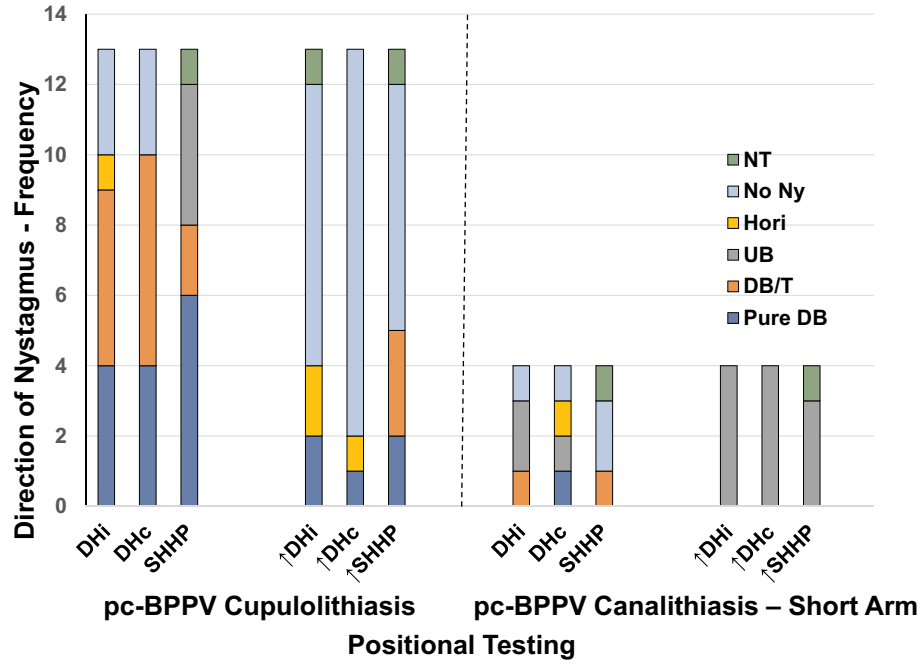

Figure 3. Frequency of primary vector of nystagmus for provoking position and return to sitting for each positional test – Dix-Hallpike (DH) ipsilateral (i) and contralateral (c), and Straight Head Hang Position (SHH). Two clinical presentations of atypical pc-BPPV were identified – one the primary vector of nystagmus was pure DB or DB/torsion towards the uninvolved side in provoking positions and no nystagmus upon return to sitting, attributed to pc-BPPV-cu, and the other, no, or variable nystagmus in the provoking position and pure UB or UB/torsion towards involved side upon return to upright, attributed to short arm canalithiasis. In the SHH, either a DB nystagmus with/without torsion towards uninvolved side was observed (69%), or an intense transient nystagmus that was UB with torsion towards involved side (31%) suggesting an ipsilateral canal switch from pc-BPPV-cu to pc-BPPV-ca of long arm. Once converted, upon return to upright, a DB with torsion towards uninvolved side was observed suggesting inhibition. This figure is available in color online (www.jnpt.org).

Table 2. Positional Nystagmus Classified According to Location of Pathology, Direction of Nystagmus, and Temporal Profile of Nystagmus

	CPPN[24,26]	Migraine[25,27]	Atypical BPPV – Controversial				
			ac-BPPV-ca[14,15,20]	pc-BPPV-cu[14-16,19]	pc-BPPV-ca short arm[9,15]	Apogeotropic pc-BPPV or pc jam non-ampullary segment[10,11]	Sitting-up Vertigo or pc jam peri-ampullary segment[12]
Pathology	**Central** – Damage brainstem or cerebellum. Posterior circulation stroke (acute) or posterior fossa mass lesions (chronic)	**Central** –	**Peripheral** Endolymph flow in direction particles falling (transient stimuli)	**Peripheral** – Deflection of the cupula due to gravity (sustained stimuli)	**Peripheral** – Endolymph flow in direction particles falling	**Peripheral** – Negative or positive pressure between obstruction and cupula results in deflection of the cupula (sustained stimuli)	**Peripheral** – Negative or positive pressure between obstruction and cupula results in deflection of the cupula (sustained stimuli)
Timing	Acute or Chronic Vestibular Syndrome	Episodic	Episodic	Episodic	Episodic	Episodic	Episodic
Triggers	Spontaneous	Spontaneous	Trigger	Trigger	Trigger	Trigger	Trigger
Positional Testing							
DHT (left/right)	Both positions	Both positions	Both positions	1 or both positions	1 or both positions	1 or both positions	1 or both positions
SHH	Always	Always	Always	Sometimes	Sometimes	Sometimes	Sometimes
Direction of Nystagmus	Variable – aligned with vector sum of rotational axes of canals normally inhibited	Variable – horizontal, vertical, or torsional.	Weak torsion towards involved ear-or-no torsion Predominantly downbeat	**HHi** Torsion towards involved ear-or-no torsion Upbeat **DHT** Torsion towards uninvolved ear-or-no torsion Downbeat **IR** Torsion towards uninvolved ear-or-No torsion Downbeat	No nystagmus-or-Mild downbeat	Torsion towards uninvolved ear-or-no torsion Downbeat	Torsion towards uninvolved ear-or-no torsion Downbeat
Response	Variable – aligned with vector sum of rotational axes of canals normally inhibited	Variable – horizontal, vertical, or torsional.	Excitatory	**HHi** Excitatory **DHTi** No response-or-Inhibitory **IR** Inhibitory	No Response	Inhibitory	Inhibitory
Return to Sitting							
Direction of Nystagmus	Reversal-or-No reversal	Variable – horizontal, vertical, or torsional.	Absent	Absent	Torsion towards involved side Upbeat-or-Symptoms no nystagmus (Type 2 BPPV)	Absent-or-present but does not reverse direction	Absent-or-present but does not reverse direction-or-reverses direction with reversal > intensity
Response	Normally inhibited by positional test	Variable – horizontal, vertical, or torsional.			Excitatory	Excitatory	Excitatory

(continues)

Table 2. Positional Nystagmus Classified According to Location of Pathology, Direction of Nystagmus, and Temporal Profile of Nystagmus (*Continued*)

	CPPN[24,26]	Migraine[25,27]	Atypical BPPV – Controversial			
Temporal Pattern of Nystagmus Intensity	Peak initially – decreases exponentially over time	Persistent, low velocity. Dissipates when patient is free of symptoms.	Crescendo (gradual build over 10-20s) – decrescendo		Persistent, intensity of nystagmus modulated by position changes that affect magnitude of cupular deflection	Persistent, intensity of nystagmus modulated by position changes that affect magnitude of cupular deflection
Duration of Episode		Lasts seconds and occurs repeatedly during head motion, visual stimulation, or after changes of head position. Total period Typically <72 hours but up to 4 weeks.	Transient, <60s	Persistent, >60s if provoking position maintained	Persistent, >60s	Persistent, >60s
Additional Associated Neurologic Findings	Yes deficits smooth pursuit and others	Yes spontaneous vertigo, visually induced vertigo, head motion induced dizziness with nausea, phonophobia, visual auras, somatosensory or dysphasic aura, with/without headache	No	No	No transient dysfunction of ipsilateral PC during vHIT	No transient dysfunction of ipsilateral PC during vHIT

Abbreviations: AC, anterior canal; BPPV, benign paroxysmal positional vertigo; CA, canalithiasis; CU, cupulolithiasis; DH, dix-hallpike test; HH, half-hallpike test; I, ipsilateral; IR, inverted release position; (L), left; PC, posterior canal; (R), right; SHH, straight head hang position; vHIT, video head impulse test.

positional DB nystagmus originally was attributed to ac-BPPV-ca but ac-BPPV-ca is rare[20,23] due to the location of its anatomical attachment to the superior portion of the utriculus.[46] Emerging evidence suggests that positional DB nystagmus may be due to pc-BPPV-cu.[17] The clinical presentation attributed to pc-BPPV-cu is a low intensity, persistent nystagmus that is purely DB or DB/torsion towards the uninvolved side, observed in 1 or both DH and SHH, suggesting deflection of the pc cupula in an inhibitory direction that resolves upon return to sit.

The data suggests translocation of debris within the pc following canal-specific CRP. In this study, the findings of 65% is higher than reported by others and may be due to referral to vestibular specialty clinic or small sample size. In a series of persons identified with peripheral positional DB nystagmus, 42% (n = 46) had typical pc-BPPV 1 week prior or after observation.[4] Following a successful CRP for pc-BPPV-ca long arm, findings on DH suggested translocation of debris to pc-BPPV-cu short arm in 19% (n = 100) of persons presenting with persistent DB nystagmus in DH,[10] and pc-BPPV-ca of short arm in 20% (n = 18) of persons presenting with an UBN upon sitting up from provoking positions.[36]

The clinical features of pc-BPPV-cu are similar to ac-BPPV-ca and pc jams. Both pc-BPPV-cu and ac-BPPV-ca generate a nystagmus, the direction being DB or DB/torsion in 1 or both positions of the DH and SHH. The most sensitive test for ac-BPPV is the SHH.[4,47] The differences are the direction and degree of the torsional component in the provoking positions. The velocity of the torsional component may help indicate the affected canal with smaller velocities indicative of ac-BPPV and larger velocities indicative of pc-BPPV-cu.[17,23] The direction of the torsion suggests the affected semicircular canal – torsion towards involved side indicates ac-BPPV-ca[17,23] and towards the uninvolved side indicates pc-BPPV-cu.[17,18] Translocation of otoconia occurs during canal-specific CRPs. Findings from the HH and ipsilateral canal switch during SHH support pc-BPPV-cu as the mechanism for peripheral position DB nystagmus. Of the 4 persons with ipsilateral canal switch in SHH, 2 presented with pure DB and 2 with DB/torsion. As a result of ipsilateral canal switch, the direction of vertical and torsional nystagmus reversed, suggesting pc-BPPV-cu. If a canal switch had occurred, from ac-BPPV-ca to pc-BPPV cu, the direction of nystagmus would continue to be DB with torsion initially towards the involved ear and then away from the involved ear. In this study, half of the people had pure DB nystagmus, the other half DB/torsion. These findings are similar to previous reports.[4] Of the eight persons with HHi data, the direction of torsion confirmed canal involved in 7 and no nystagmus in 1. In this study, the clinical presentation suggested pc-BPPV-cu and not a pc jam.

Intervention success is dependent on identifying the involved canal and mechanism, and administration of canal- and mechanism-specific CRP. Reducing time to resolution of episodic positional vertigo will improve quality of life by reducing disruption in daily activities, time off from work, and risks of falls, reducing costs to society and the person. Modifications to intervention strategies need to be identified to

reduce risk of translocation of otoconia causing atypical BPPV and to develop appropriate interventions for each mechanism. Biomechanistic modeling including three-dimensional visualization could assist in the development of positional testing and interventions that are canal- and mechanism-specific.

This is a retrospective observational study with a small sample of convenience, from an outpatient vestibular specialty physical therapy clinic. Therefore, findings may be biased and not be generalized to the general population. The reported ratio of females to male was higher than previously reported. Diagnostic tests not performed were the video head impulse test to identify transient function of the involved canal associated with canal jam and neuroimaging to rule out CNS involvement. Limited time prevented consistent use of the IR. The mechanisms of atypical vertical canal BPPV are hypothetical, and the clinical findings between variants are similar with subtle differences making the differential diagnosis difficult.

CONCLUSION

Persistent, peripheral, positional nystagmus that is DB or DB/torsion towards the uninvolved side in the DH and SHH, and when nystagmus is absent upon sitting up, may be attributed to pc-BPPV-cu. Nystagmus that is DB or no nystagmus in the DH and SHH, and UB or UB/torsion towards the involved side upon sitting up may be attributed to pc-BPPV-ca short arm. The HH can be effective in identifying pc-BPPV-cu. The inverted release position is effective in determining adherence of debris either attached or adjacent to cupula. Treatment of both variants may be effective with canal-specific CRPs.

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