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Complex nystagmus in traumatic benign paroxysmal positional vertigo: A case study on the critical value of knowing semicircular canal excitation and inhibition patterns

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ABSTRACT

A 73-year-old female presented to the emergency department with chief complaint of dizziness after sustaining a fall one month prior to dizziness onset. Although careful examination of eye movement patterns during positional testing was attempted at varying stages of her inpatient admission, her complex nystagmus patterns as a result of traumatic benign paroxysmal vertigo were difficult to manage. In particular, the nystagmus pattern from this case suggests the BPPV was variable and affecting either 1) left posterior semicircular canal (pSCC) exclusively 2) left pSCC and right anterior semicircular canal, 3) left and right pSCC canal. This case illustrates the importance of two critical details; positional testing should include observing nystagmus with fixation removed and an experienced clinician should be involved as early as possible.

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1. Introduction

Benign paroxysmal positional vertigo (BPPV) is a peripheral vestibular condition that causes room-spinning vertigo with changes in head position (Von Brevern et al., 2017; Hornibrook, 2011). Approximately 4% of all emergency department (ED) visits report a chief complaint of vertigo or dizziness (Newman-Toker et al., 2007), yet BPPV continues to be difficult for physicians in the ED to successfully diagnose and treat (Kerber et al., 2017). Traumatic Benign Paroxysmal Positional Vertigo (T-BPPV) occurs when symptoms of dizziness with position change or head movement begin within three days to three months after head or neck injury, typically caused by motor vehicle accident, fall, direct or indirect blow to the head, concussion or whiplash injuries. It is estimated to account for 8.5–20% of all BPPV cases (Haripriya et al., 2018; Bashir et al., 2017; Ahn, 2011). T-BPPV often involves a more complex clinical presentation of BPPV with an increased incidence of horizontal, anterior, and multiple semicircular canal (SCC)

involvement (Soto-Varela, 2013). Balatsouras et al. suggests that T-BPPV resulted in 24.2% of BPPV cases with multiple canal involvement compared to 7.5% in patients with idiopathic BPPV (Balastorus et al., 2017). Lui et al. suggested that 55% of patients with T-BPPV had involvement of two or more SCCs canals compared to 6.5% of patients with idiopathic BPPV (Lui et al., 2012). Females have higher incident rates of T-BPPV, with a greater incidence of multi-canal involvement (Kim, 2018; Soto-Valero, 2013). Additionally, T-BPPV tends to require multiple treatments prior to obtaining therapeutic success (Soto-Valerio, 2013) (Kim, 2018).

This case report details the plan of care for a 73-year-old female who presented to the ED with a chief complaint of dizziness. Initially, the patient was diagnosed with BPPV in the ED; however, given the list of possible differential diagnoses, she was admitted to the hospital for vertigo workup. The patient remained in the hospital for 6 days to complete clinical workup and to receive treatment for BPPV. While in the hospital, clinical examination was completed by multiple providers, who failed to document a comprehensive vestibular bedside exam (including testing with fixation removed). The absence of performing positional testing during fixation blocked likely led to increased healthcare costs, contributed to the patient receiving multiple yet unnecessary repositioning maneuvers, given their inability to appreciate

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nystagmus patterns within the unique testing positions. Only when a specialty trained clinician was consulted, did this complex presentation of BPPV resolve.

CONTEXT: Setting and population in which the innovation occurs.

This case occurred within a level 1 trauma center in Baltimore, MD.

2. Case details

A 73-year-old female presented to the emergency department (ED) with chief complaints of vertigo, dizziness and nausea. She denied current headache and/or current change in vision. Her symptoms started at 4:30 a.m. when she changed position to get out of bed, and persisted throughout the morning. She noted that rolling over in bed exacerbated her symptoms. As her symptoms did not resolve, she sought guidance from the ED. In the ED, she reported a pre-existing, constant weakness and painful dysesthesia of bilateral upper extremities, and a new onset of tingling in both of her lower extremities. These symptoms were presumed to be related to a recent cervical surgery. Her past medical history was significant for a mechanical fall (i.e. tripping over a root) approximately one month prior to the onset of her severe dizziness. The fall resulted in cervical hyperextension with immediate loss of strength and sensation in all extremities. Imaging from the time of her fall demonstrated a C3–C5 cord contusion that was treated with discectomy and decompression surgery. Her past medical history was significant for ocular migraine, migraine headache during menopause, and motion sensitivity.

The patient's bedside oculomotor examination in the ED identified equal and reactive pupils, intact extraocular range of motion, normal smooth pursuit, absent ptosis, and absent nystagmus (room light). Her horizontal and vertical saccades were within normal limits. No skew deviation was observed on cover/uncover test; of note, head impulse testing was not completed due to patient's history of cervical fusion and spinal precautions. Hearing was normal to finger rub, and the patient denied symptoms of tinnitus or aural roaring sensation. Side lying positional testing was completed at bedside, which identified left beat and 'rotational' nystagmus (we presume the clinician intended this to imply torsion, the direction was not specified) when the patient was positioned on her right and left side. The patient had subjective report of dizziness in the left side-lying only. The note further reported that the patient described a room-spinning vertigo and had one episode of emesis. Positional testing was limited due to her recent surgical history, as she had spinal precautions and was required to wear a Miami J Cervical Collar, thereby limiting her ability to participate in conventional positional testing. A head CT and MRI was ordered to rule out a posterior circulation cerebrovascular accident and cervical fusion complications. The imaging studies in the ED were interpreted as unremarkable with no acute intracranial abnormalities or significant arterial stenosis. In summary, the patient's ED examination revealed a direction fixed left beat nystagmus with a presumed torsional component during positional testing. The patient was diagnosed with left horizontal canal BPPV and treated unsuccessfully using a left Epley maneuver, despite the nystagmus pattern. She was admitted for further management of her dizziness.

2.1. Initial intervention

In the hospital, the neurology team consulted on this patient. Their exam included positional testing in room light, during which the patient reported dizziness and a left beat nystagmus with left torsion observed when transitioning from supine to sit. Next, she

was tested in the Dix-Hallpike position and treated using the left Epley maneuver although there was no notation to suggest the patient had nystagmus from a posterior semicircular canal excitation. A second left Epley maneuver with vibration applied to left mastoid process was performed because her symptoms did not "resolve or change"; a subsequent left Dix-Hallpike test was reported as "negative". After this bedside treatment, she continued to report 2–3 episodes of dizziness per day when rolling on either side and when transitioning from supine to sit. A consult for physical therapy (PT) was placed.

2.2. Physical therapy intervention

The PT attempted an additional left Epley maneuver despite no reported nystagmus during positional testing in room light. The patient continued to report positional dizziness; thus, the PT elected to try the Semont maneuver for the right ear twice followed by a single Semont maneuver for the left ear. Given the continued intermittent positional vertigo symptoms and that this 1st PT had less than one-year experience treating BPPV, a second PT with extensive experience treating BPPV was consulted.

2.3. Treatment session 1

Upon transfer of care, the patient received bedside testing with fixation removed; eye movements were recorded using video oculography (Micromedical Visual Eyes, Middlefart, Denmark). The Miami J collar was donned throughout assessment, and the hospital bed was placed in varying positions to complete positional testing with respect to cervical spine precautions (Fig. 1). During Dix-Hallpike testing, the patient was initially positioned in approximately 45 degrees of side-lying with the bed tilted approximately 15° in Trendelenburg, as she was unable to turn her head laterally or complete cervical extension. Right Dix-Hallpike induced down beating and right torsion nystagmus with duration less than 60 s (Video Fig. 1). No reversal pattern of nystagmus was noted when the patient returned to sitting. Next, a left Dix-Hallpike position was positive for dizziness with brief up beating and left torsional nystagmus with dizziness diminishing within 25 s, (Video Fig. 1). Upon return from left Dix-Hallpike testing she had down-beat and right torsional nystagmus. Bilateral roll test for the horizontal canal induced a persistent down-beating nystagmus accompanied with symptoms of dizziness and vertigo that was more severe in right side-lying. Repeat right Dix-Hallpike was negative for nystagmus, albeit she felt mild dizziness. Repeat left Dix-Hallpike revealed up beat with left torsion lasting 15 s accompanied with vertigo Fig. 2.

She was treated with a modified left Epley Maneuver (Fig. 1). The maneuver was modified by fitting her with a Miami J collar and positioning her hospital bed in Trendelenburg position - in order eliminate cervical extension. Repeat Dix-Hallpike test to each ear was negative after a single maneuver, the patient denied vertigo.

2.4. Treatment session #2

At the follow up session (approximately 18 h from Session 1), the patient reported improved symptoms of positional vertigo. Positional testing was again completed with Frenzel goggles. Right Dix-Hallpike was positive for upbeat and right torsional nystagmus lasting 15 s with room-spinning vertigo. Left Dix-Hallpike was negative for nystagmus and symptoms. Roll test bilaterally demonstrated an asymptomatic down-beat nystagmus. She was treated with a modified right Epley Maneuver (bed was placed in Trendelenburg and the patient was using a cervical collar); subsequent Dix-Hallpike testing was negative.

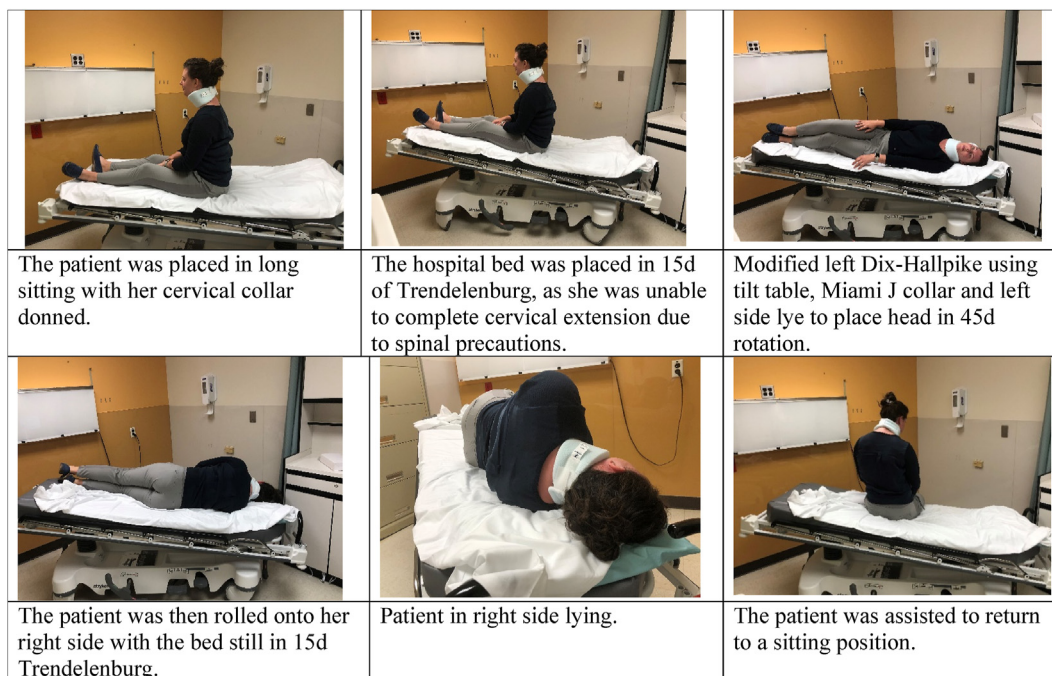


Fig. 1. Example of modified left Dix Hallpike and Modified Left Epley Repositioning Maneuver.

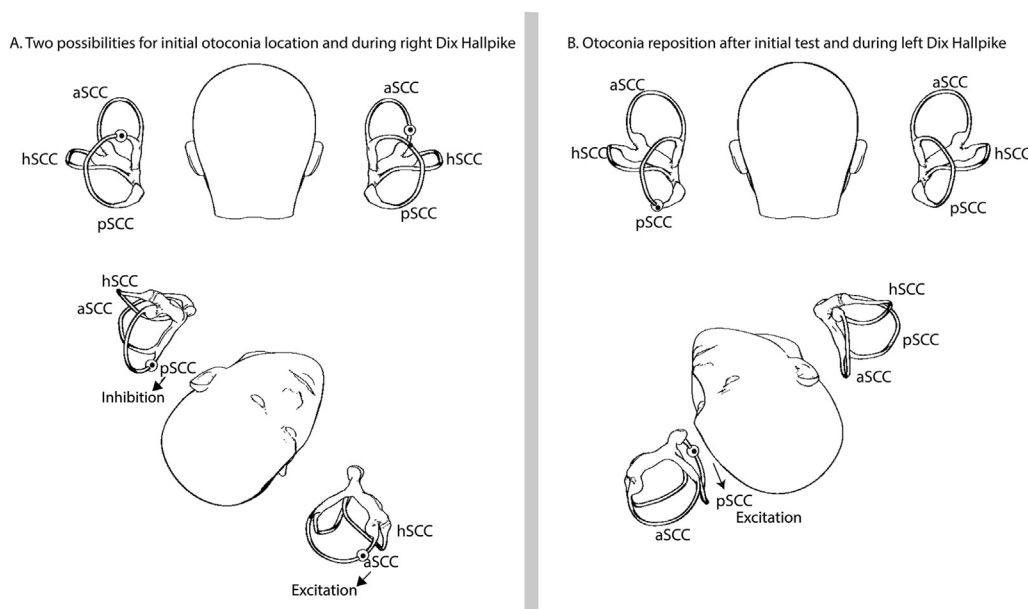


Fig. 2. Semicircular canals with otolith describing the complex nystagmus pattern during the clinical exam with video Frenzel lens. A. The right Dix Hallpike was tested first and caused a downbeat nystagmus with right torsion from otoconia motion that excited the right anterior semicircular canal afferents or inhibited the left posterior semicircular canal afferents. B. The left Dix Hallpike was tested next, and the otoconia from the left first posterior semicircular canal had repositioned to the dependent, cupula segment, generating an upbeat and left torsional nystagmus. aSCC - anterior semicircular canal; hSCC – horizontal semicircular canal; pSCC – posterior semicircular canal; Circle with black dot represents the displaced otoconia. Figure created using software developed by (Taboulsi and Teixido, 2020).

2.5. Treatment session #3

Twenty-four hours from Session #2, the patient completed positional testing with fixation removed. She denied symptoms of vertigo in all testing positions, however right Dix-Hallpike demonstrated up beating nystagmus with right torsion lasting less than 5 s. In the left Dix-Hallpike position she has no nystagmus or vertigo symptoms. Roll test also did not produce any symptoms

of dizziness, although she had a persistent down-beat nystagmus in both positions. The patient was not treated for BPPV as she denied symptoms of vertigo within testing positions. Instead, she was educated on BPPV and scheduled for outpatient vestibular physical therapy to include management of her BPPV, balance training, and dynamic gait training before being discharged from the hospital. Outpatient PT therapy was started 12 days after discharge, and she reported significant improvement regarding vertigo, though

continued to report mild residual dizziness.

3. Discussion

Our case highlights three clinical pearls managing BPPV: 1) the importance of conducting positional testing with fixation blocked, 2) the need to repeat positional testing as long as the vertigo symptoms persist, and 3) the importance of consulting clinicians experienced in treating BPPV. In this case, the patient sustained a fall that presumably disrupted the utricular otolith membrane and subsequently lead to the displacement of otoconia to settle within each posterior semicircular canal (pSCC) and/or other canals. At the 1st treatment session, the nystagmus pattern suggested one of three possible BPPV scenarios (Table 1): left ear only (posterior semicircular canal canalithiasis that caused excitation in left Dix-Hallpike and inhibition in right Dix-Hallpike); 2) left pSCC canalithiasis and right anterior SCC canalithiasis; and 3) bilateral pSCC (left posterior canal canalithiasis and right apogeotropic posterior canal canalithiasis). Given the reversal of nystagmus (downbeat with right torsion) upon return to sitting from the left Dix Hallpike coupled with the excitatory nystagmus in left Dix-Hallpike (upbeat with left torsion), it seems that most likely explanation was a severely affected left pSCC (Imai et al., 2008). However, at treatment session 2 positional testing revealed a right pSCC excitation, thus it remains possible that bilateral pSCC (apogeotropic right pSCC and typical canalithiasis of the left pSCC) were initially affected with a recurrence occurring from treatment session 1 and 2.

DH-Dix Hallpike; aSCC- Anterior Semicircular Canal; pSCC-Posterior Semicircular Canal; nystagmus named by the direction of the quick phase.

Down-beat nystagmus is typically a marker of central pathology and often associated with lesions to the caudal vermis of the cerebellum (Ye, 2010). In this case study, the patient had extensive imaging that ruled out cerebellar pathology. Therefore, it is likely that her persistent downbeat nystagmus in bilateral roll test was related to her history of migraine. As many as 28% of individuals with vestibular migraine present with central positional nystagmus without vertigo (Beh et al., 2019; Huang et al., 2020). Nystagmus observed in individuals with vestibular migraine, typically is persistent and characterized by an absence of latency with a low velocity and may only be visible during the fixation removed component of the examination (Polensek et al., 2010.). Nystagmus presentation is thought to occur due to altered neuronal activity

within the trigeminovascular system, causing imbalance between neuronal excitation and inhibition (Elsherif, 2020; Huang, 2020).

It is generally regarded that nearly 80% of BPPV cases involve the posterior semicircular canal (Nutti, 2020), which is also very prominent after T-BPPV (Pisani, 2015). Conversely, BPPV affecting the anterior SCC (aSCC) has a much smaller (1–2%) frequency (Yang et al., 2019; Cambi, 2013; Imbaud-Genieyes, 2018), unless trauma is the cause – in which case the incidence rates climb and vary between 12.2 and 27.3% (Balatsouras, 2017; Dlugaiczyk, 2011). Given that aSCC BPPV has lower incidence than other canals, it is often overlooked (Nutti, 2020; Kim, 2018). In our case, aSCC BPPV cannot be excluded, as marked hyperextension of the head may have enabled the mechanical disposition of otoconia into this canal (Imbaud-Genieyes, 2013). Apogeotropic pSCC BPPV is considered a rare variant and causes a positional down beat nystagmus with vertigo via inhibition of the ipsilesional vestibular afferents - when positioned in the Dix-Hallpike test (Helminski, 2019). Incidence rates are reported to be 2.5% (Califano, 2014).

3.1. Repeat testing

Repeat testing during the initial session was completed soon after the treatment, which may have produced a false negative. False negative positional testing may be related to the fatigue phenomena of BPPV, presumed to result from otoconia relocating into different locations within a SCC that are less provocative upon repeat testing, or otoconial clusters breaking into smaller particles thus causing different trajectories of otoconial debris (Boselli, 2014; Parnes, 1992, 1993, 2003). Some authors recommend waiting 30 min prior to retest in order to prevent fatigue effects (Imai, 2018). BPPV fatigue may explain why repeat testing on treatment session 1 in this case did not reveal suggestion of bilateral semicircular canal involvement.

3.2. Experienced clinician

The difficulty with clinical interpretation of the nystagmus pattern in the ED that was perpetuated during the inpatient setting contributed to over-utilization of resources and increased health-care cost. In our case, although imaging in the ED was critical to rule out a central pathology, the patient was nonetheless admitted for management of a peripheral vestibular pathology. We believe the inpatient admission was conservative, and her treatment could have been effectively managed by an ED visit from an experienced

Table 1
Positional testing during treatment session 1 with fixation removed.

Positional Test	Nystagmus, duration	Symptom	Affected SCC Stimulation
Right DH	Down beat with right torsion,	Vertigo	1. Right aSCC excitation 2. Left pSCC inhibition, common crus (apogeotropic variant)
Return from Right DH	<60 s		
Repeat Right DH	Absent	Vertigo	
	Absent	Vertigo	
Left DH	Up beat with left torsion, 25 s duration	Vertigo	Left pSCC excitation
Return from Left DH	Down beating with right torsion	Vertigo	Left pSCC inhibition
Repeat Left DH	Up beat with left torsion, 15 s duration		Left pSCC excitation
Right Roll Test	Persistent down beat nystagmus	Vertigo, patient reported severe symptoms within right roll test compared to left.	1. Vestibular migraine 2. Cerebellar pathology
Repeat Roll	N/A		
Left Roll Test	Persistent down beat nystagmus	Vertigo, patient reported less severe than in right roll.	1. Vestibular migraine 2. Cerebellar pathology
Repeat Roll	N/A		

clinician or referral to be seen urgently in an outpatient setting. Barriers for ED physicians to appropriately recognize and treat BPPV is thought to be related to past negative experiences with performing Dix-Hallpike testing and inappropriately relying on subjective history, with minimal consideration to patterns of nystagmus (Kerber, 2017). Moreover, this patient's hospital stay was poorly managed, as clinician specialists were not contacted until 3 days into her 6-day hospitalization. This exemplifies how individuals with dizziness often pay more for healthcare (Neuhauser et al., 2008; Wang et al., 2019; Grill et al., 2014; To-alemanji et al., 2016). The present case epitomizes the need for standardized care for individuals with vestibular dysfunction, as this patient had multiple providers performing numerous BPPV treatments without following the currently accepted clinical practice guidelines for management of BPPV (Bhattacharyya, 2017). This case further exemplifies that despite clinical practice guidelines from neurology and otolaryngology societies for BPPV, a disconnect among ED providers remains, possibly due to lack of ability to consult clinicians with vestibular experience (Kerber et al., 2013, 2017). Finally, this case mirrors the recent report that despite evidence suggesting the benefits of physical therapy for treating BPPV, referrals for PT to treat individuals with BPPV continue to be low within the United States (Dunlap et al., 2020).

4. Conclusion

A comprehensive and careful bedside exam delivered from experienced providers that includes fixation removed positional testing is essential in the accurate diagnosis and management of BPPV. As the present case demonstrates, timely and efficient care is dependent on the availability of knowledgeable vestibular clinicians and the availability of relevant technology. Efforts to adequately train healthcare providers along the continuum of care (urgent, inpatient, outpatient) is necessary to reduce patient burden and optimize healthcare resources (Wang, 2019; Grill et al., 2014). With lack of uniformity, individuals with vestibular dysfunction may be misdiagnosed or mismanaged. Incorrect diagnosis of vestibular dysfunction may lead to prolonged symptom duration, increased fall risk, increased health service utilization, increased disability, and secondary dizziness including disuse and somatoform dizziness (Grill et al., 2014).

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Declaration of competing interest

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.joto.2021.01.004>.

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