Benign Paroxysmal Positional Vertigo in Children and Adolescents With Concussion

Alicia Wang, BSE,[†] Guangwei Zhou, ScD,[†‡] Kosuke Kawai, ScD,[†‡] Michael O'Brien, MD,[‡§] A. Eliot Shearer, MD, PhD,[†‡] and Jacob R. Brodsky, MD*[†‡]

Background: Dizziness after concussion is primarily attributed to effects on the brain, but traumatic inner ear disorders can also contribute. Benign paroxysmal positional vertigo (BPPV) is a common vestibular disorder that can result from minor head trauma and can be easily diagnosed and rapidly treated in an office setting. The role of BPPV in pediatric postconcussive dizziness has not been well-studied.

Purpose: To evaluate the prevalence and clinical features of BPPV in a group of pediatric patients with concussion and prolonged dizziness after concussion.

Study Design: Case-control study.

Level of Evidence: Level 3.

Methods: Retrospective review of 102 patients seen within the past 3 years in a pediatric multidisciplinary concussion clinic for evaluation of postconcussive dizziness.

Results: BPPV was diagnosed in 29.4% (30/102) of patients with postconcussion syndrome and dizziness. All patients with BPPV were treated with repositioning maneuvers, except for 5 patients who had spontaneous resolution of symptoms. Patients were evaluated at an average of 18.8 weeks (SD, 16.4 weeks) after the injury. BPPV was diagnosed at similar rates regardless of gender or age group (children vs adolescents). The mean Post-Concussion Symptom Scale (PCSS) score did not differ significantly between patients with (58.3 [SD, 22.5]) or without BPPV (55.8 [SD, 29.4]; $P = 0.39$). The PCSS "balance problems or dizziness" subscore also did not differ between patients with (3.3 [SD, 1.7]) or without BPPV (2.8 [SD, 1.6]; $P = 0.13$).

Conclusion: BPPV is fairly common in pediatric concussion, occurring in one-third of the patients studied. BPPV is often not diagnosed and treated until many weeks after the injury. Increased awareness of the evaluation and management of BPPV among pediatric concussion providers may help expedite resolution of dizziness and hasten overall recovery in affected patients.

Clinical Relevance: BPPV is a treatable cause of dizziness caused by minor head injuries and is more common than previously reported in pediatric patients with concussion. Improved awareness of BPPV by concussion providers may expedite recovery.

Keywords: concussion; dizziness; BPPV; pediatric; vestibular; vertigo; postconcussion syndrome

Dizziness is the second most common symptom of concussion after headache, both at the time of injury and in patients with prolonged symptoms.[7] In high school athletes with concussion, dizziness is the only on-field symptom that is independently predictive of a protracted recovery, leading to a 6-fold increased risk of recovery lasting longer than 3 weeks.[13] The World Health Organization defines symptoms that persist for greater than 4 weeks after a concussion as postconcussion syndrome (PCS).[19] Although dizziness after concussion and in PCS is often attributed to

From [†]Department of Otolaryngology and Communication Enhancement, Boston Children's Hospital, Boston, Massachusetts, [‡]Harvard Medical School, Boston, Massachusetts, and [§]Division of Sports Medicine, Boston Children's Hospital, Boston, Massachusetts

*Address correspondence to Jacob R. Brodsky, MD, Department of Otolaryngology and Communication Enhancement, Boston Children's Hospital, 300 Longwood Avenue, Boston, MA 02115 (email: jacob.brodsky@childrens.harvard.edu).

The authors report no potential conflicts of interest in the development and publication of this article.

DOI: 10.1177/1941738120970515

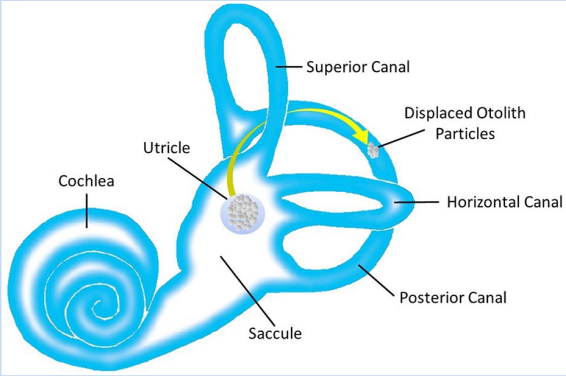

Figure 1. Diagram of the peripheral vestibular organs in the inner ear. Yellow arrow indicates displacement of otolith particles in benign paroxysmal positional vertigo.

effects on the brain,[1] dysfunction of the peripheral vestibular system in the inner ear may also contribute.[5,10,20]

The peripheral vestibular system mainly refers to the balance organs within the inner ear, which consist of 2 linear motion sensors (saccule and utricle) and 3 rotational motion sensors (superior, horizontal, and posterior semicircular canals) on each side of the head (Figure 1). These organs collectively send information to the brain (via the central vestibular system) and subsequently to the eyes and body about head position and movement in order to maintain balance, a sense of equilibrium, and stability of images on the retina. The most common peripheral vestibular disorder in adults is benign paroxysmal positional vertigo (BPPV),[3,18] which is symptomatically characterized by episodes of transient vertigo provoked by positional changes. The most accepted etiology for BPPV is displacement of calcium carbonate debris (otoliths) from the utricle into 1 or more of the semicircular canals. This leads to vertigo when changing head or body position in the plane of the affected canal(s). One of the most common causes of BPPV is head trauma.[2]

The presence of BPPV is determined by performing diagnostic maneuvers in which the examiner rotates the patient's head through the plane of each semicircular canal. Subjective vertigo and characteristic nystagmus during a maneuver for a given canal is consistent with a diagnosis of BPPV affecting that particular canal.[16] Treatment for BPPV consists of a repositioning maneuver, which involves moving the head through the planes of the affected canal in a manner that utilizes gravity to relocate the displaced otoliths back to the utricle. There are a variety of repositioning maneuvers that can be performed to treat BPPV involving each specific semicircular canal, and treatment is often curative after only 1 or 2 maneuvers in the office.

BPPV is a common cause of dizziness after concussion in adults. A study of military patients with concussion found that approximately one-third of those with dizziness had evidence of BPPV, and prompt treatment resulted in a more rapid return to duty compared with other patients with dizziness not treated for BPPV.[10] Although BPPV has historically been rarely reported in the pediatric population,[11] a recent study found that it may be the most common peripheral vestibular disorder in children and adolescents[4] and can often occur secondary to an incident such as a concussion or other brain injuries. Another recent study found that nearly 1 in 5 pediatric patients with prolonged dizziness after concussion had BPPV, and all of them were successfully treated with repositioning maneuvers.[5]

Training in the diagnosis and treatment of BPPV is often limited to specific specialties that regularly manage vestibular disorders, such as otolaryngology. In contrast, providers who routinely manage patients with concussion may have little to no experience with BPPV, which could lead to a significant delay in its management and even prolong overall concussion recovery.

In this study, our goal was to evaluate the prevalence of BPPV in children and adolescents with PCS and to compare clinical characteristics between patients with and without a diagnosis of BPPV. We also determined the effect of treatment for BPPV in these patients. The multidisciplinary concussion clinic (MDCC) at our institution involves evaluation by a number of specialties, including otolaryngology, which consistently includes a thorough assessment for BPPV. Patients who are referred to the MDCC have received a prior diagnosis of concussion and PCS by a clinician with expertise in the evaluation of traumatic brain injury—typically specializing in sports medicine or neurology. The protocol for MDCC admission and evaluation provides a unique opportunity to evaluate the prevalence and clinical features of BPPV in pediatric patients with PCS.

METHODS

We conducted a retrospective review of all patients aged <21 years who were seen at our MDCC between April 2016 and May 2019 for evaluation of PCS. This study was approved by the institutional review board at our hospital. All patients included in this study had reported symptoms of dizziness at the time of their clinic visit and had undergone a comprehensive otological and neurological evaluation by a pediatric otolaryngologist and/or nurse practitioner specializing in the evaluation and treatment of vestibular disorders in children and adolescents. No patients diagnosed with BPPV had been previously diagnosed by another provider, and nearly all patients demonstrated characteristic nystagmus and vertigo with at least 1 diagnostic positional maneuver (bilateral Dix-Hallpike maneuver, bilateral supine head roll maneuver, and/or midline head-hang maneuver [Supplemental Videos 1-3, available in the online version of this article]) with the aid of videonystagmography (VNG) goggles (Micromedical Technologies). A small number of patients with resolved symptoms did not have a positive diagnostic maneuver but had a clinical history highly suggestive of BPPV.

Study data were collected and managed using REDCap (research electronic data capture) tools.[8,9] Elements of data such

Table 1. Demographic and clinical features of the study sample

	BPPV (n = 30)	No BPPV (n = 72)	RR (95% CI)	*P*
Age, n (%)				
<14 y	8 (25.0)	24 (75.0)	0.80 (0.40, 1.59)	0.51
≥14 y	22 (31.4)	48 (68.6)		
Sex, n (%)				
Female	19 (28.4)	48 (71.6)	0.90 (0.49, 1.68)	0.75
Male	11 (31.4)	24 (68.6)		
History of concussion, n (%)				
Yes	16 (34.0)	31 (66.0)	1.36 (0.74, 2.53)	0.32
No	13 (25.0)	39 (75.0)		
Mode of injury, n (%)				
Sport	12 (31.6)	26 (68.4)		0.27
Fall	9 (33.3)	18 (66.7)		
Motor vehicle accident	1 (7.1)	13 (92.9)		
Other	8 (34.8)	15 (65.2)		

BPPV, benign paroxysmal positional vertigo; RR, risk ratio.

as date of concussion and symptom severity as measured by Post-Concussion Symptom Scale (PCSS)[6] score were self-reported by the patient through a standard day-of-visit questionnaire. The PCSS consists of a 22-item inventory of symptoms, rated on a 7-point Likert-type scale of severity. All data were stored on a protected server, and identifying information was removed prior to final analysis. Data analysis was performed using IBM SPSS Statistics for Windows, Version 24 (IBM Corp), and SAS Software (SAS Institute Inc). Differences in characteristics between patients with and without a diagnosis of BPPV, respectively, were assessed using chi-square test for categorical variables and a nonparametric Mann-Whitney *U* test for continuous variables.

RESULTS

A total of 102 patients aged <21 years with PCS and dizziness were seen in the MDCC during the 3-year study period. The mean age at the time of clinic visit was 14.4 ± 3.2 years (range 5-20 years), and 67 (65.7%) patients were women. The most common cause of injury among patients was sports (n = 38), followed by falls (n = 27). Prior additional concussions were reported in 47 patients. Patients were evaluated a mean of 18.8 weeks (range 2.9-116.9 weeks) after sustaining their most recent concussion.

Thirty patients (29.4%) were diagnosed with BPPV. Demographics and clinical features of patients with and without a diagnosis of BPPV are summarized in Table 1. The mean age for patients diagnosed with BPPV was 14.7 ± 3.4 years (range 7-20 years), and the mean age for patients without a diagnosis of BPPV was 14.3 ± 3.1 years (range 5-20 years). Adolescent patients (≥14 years of age at time of evaluation) were not significantly more likely to be diagnosed with BPPV than preadolescent patients (<14 years old at time of evaluation; 31.4% vs 25.0%; $P = 0.51$). The rate of BPPV in female patients (28.4%) compared with male patients (31.4%) was also not significantly different ($P = 0.75$). In addition, patients with prior additional concussion(s) and patients with no known prior concussion were not found to have significantly different rates of BPPV diagnosis (34.0% vs 25.0%; $P = 0.32$).

Out of the entire cohort, 100 patients completed a PCSS inventory with a median score of 49.5 (range 10-128). Among patients diagnosed with BPPV, the median PCSS score was 51.0 (range 14-116) while the median PCSS score among patients not diagnosed with BPPV was 49.5 (range 10-128), a difference that was not statistically significant ($P = 0.39$). Additionally, patients with BPPV did not report more severe dizziness symptoms than patients not diagnosed with BPPV based on PCSS vestibular subscore (Table 2).

Treatment maneuvers were performed on 25 out of the 30 patients that were diagnosed with BPPV (83.3%). The treatment maneuvers were performed during the same clinic visit that the BPPV was diagnosed in 23 patients. Two underwent vestibular

Table 2. Symptom severity between patients with and without BPPV

	Mean ± SD	Median (Range)	*P*
Total PCSS score			
BPPV	58.3 ± 22.5	51.0 (14-116)	0.39
No BPPV	55.8 ± 29.4	49.5 (10-128)	
Balance problems/dizziness subscore			
BPPV	3.3 ± 1.7	3.0 (1.0-6.0)	0.13
No BPPV	2.8 ± 1.6	3.0 (1.0-6.0)	

BPPV, benign paroxysmal positional vertigo; PCSS, Post-Concussion Symptom Scale.

Table 3. Affected semicircular canals in patients with BPPV (n = 30)

Semicircular Canal	No. of Patients (%)
Posterior	16 (53.3)
Horizontal	12 (40.0)
Superior	5 (16.7)

BPPV, beign paroxysmal positional vertigo.

testing on a separate day after the visit to confirm the affected semicircular canal, with treatment maneuvers at the subsequent follow-up visit. Of the remaining 5 patients, 3 experienced spontaneous symptom resolution by the time of their initial MDCC clinic visit. These patients were diagnosed with BPPV based on clinical history. An additional 2 patients underwent vestibular testing after the visit to confirm the affected semicircular canal and subsequently experienced symptom resolution without treatment maneuvers. The specific treatment maneuver performed was dependent on the affected semicircular canal. Table 3 summarizes affected semicircular canals among patients diagnosed with BPPV. In 4 patients, BPPV was found unilaterally in multiple canals, and 2 patients had bilateral BPPV.

DISCUSSION

Nearly 1 in 3 pediatric patients with dizziness in the setting of PCS was diagnosed with BPPV in this study, and the diagnosis was made several weeks after the injury in most cases. Given that dizziness is the second most common symptom of concussion and of PCS, and is highly predictive of a prolonged recovery,[13] these findings suggest that an increased awareness of the evaluation and management of BPPV among concussion providers could have a major positive impact on accelerating recovery in a large number of patients with concussion. At the time of diagnosis, most patients were treated with repositioning maneuvers, which are often immediately curative. Clinical features and demographics did not predict a diagnosis of BPPV, so providers are encouraged to perform diagnostic maneuvers to assess for BPPV on all patients with postconcussive dizziness.

The presence of BPPV is determined by a series of 5 diagnostic maneuvers in which the examiner rotates the head through the plane of each semicircular canal. A positive diagnostic maneuver is characterized by the presence of subjective vertigo and visible nystagmus around the axis of the affected canal(s) during the maneuver. See Figures 2 to 4 and Supplemental Videos 1 to 3 (available online) for specific details on performing each diagnostic maneuver. The typical characteristic nystagmus for each canal is described in Table 4. The nystagmus can sometimes be difficult to detect without the aid of VNG goggles. VNG goggles use a dual infrared cameras that record eye movement in real time, allowing the examiner to visualize the patient's eyes in darkness, which prevents visual fixation that suppresses nystagmus. While most concussion providers do not have access to VNG goggles, the nystagmus can often be seen on direct inspection of the eyes without goggles, and vertigo with a diagnostic maneuver alone may be enough to raise suspicion for the presence of BPPV, even if nystagmus cannot be seen directly. This should prompt the provider to perform a treatment maneuver or refer the patient to a specialist that can perform a treatment maneuver, such as a physical therapist, otolaryngologist, or neurologist with experience in treating vestibular disorders.

The posterior canal is the most commonly affected by BPPV[12,17] and is easiest to diagnose. More than half of the BPPV cases in the current study involved the posterior canal. If dizziness is only stimulated by a Dix-Hallpike maneuver on a single side and not with any of the other maneuvers (contralateral Dix-Hallpike maneuver, bilateral supine head roll maneuver, and midline head-hang maneuver) then BPPV of the posterior canal on that side should be suspected, and a treatment maneuver could be performed. The most commonly

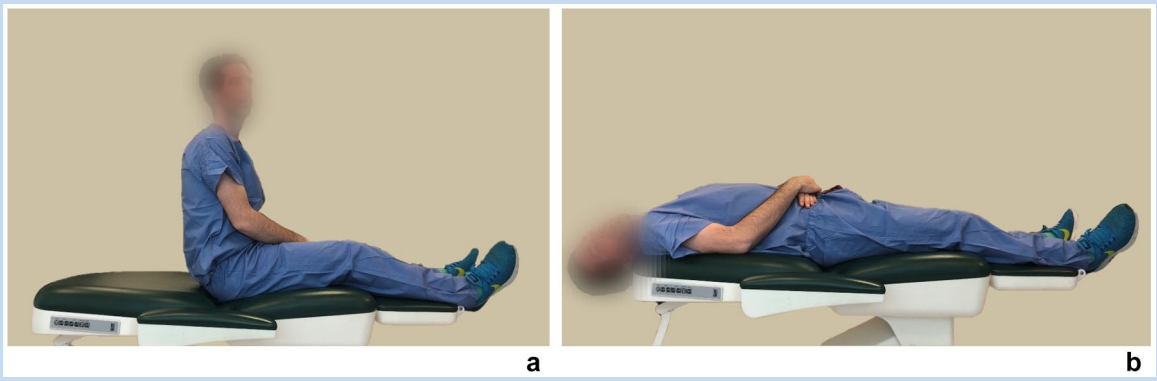

Figure 2. The right Dix-Hallpike maneuver to diagnose benign paroxysmal positional vertigo of the right posterior semicircular canal. The left-sided maneuver is identical, but with the head turned to the left side instead of to the right. (a) Patient is seated on examination table with head turned approximately 45° to the right side. (b) The head and torso are moved rapidly to the supine position with the head hanging approximately 45° below the horizontal plane with the head still turned to the right side.

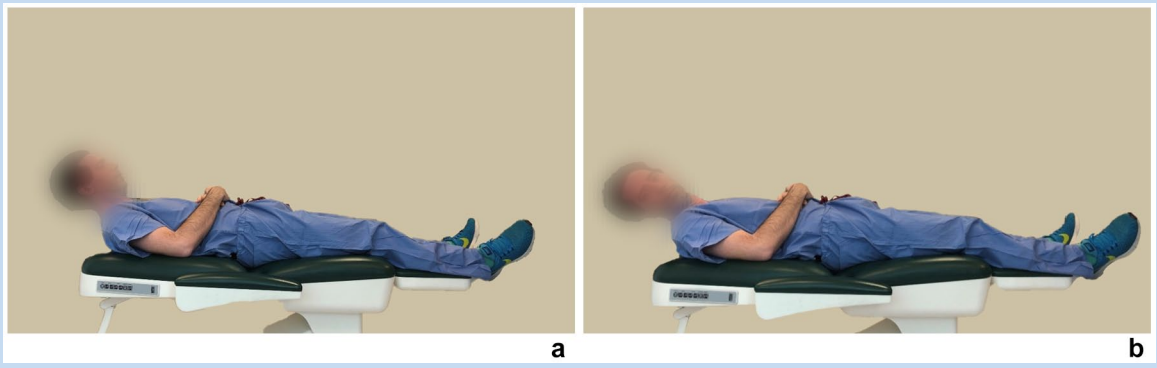

Figure 3. The right supine head roll maneuver to diagnose benign paroxysmal positional vertigo of the right horizontal semicircular canal. The left-sided maneuver is identical, but with the head turned to the left side instead of to the right. (a) The patient starts in the supine position on the examination table with head tilted up approximately 20° from the horizontal plane. (b) The head is rotated by the examiner approximately 90° to the right side.

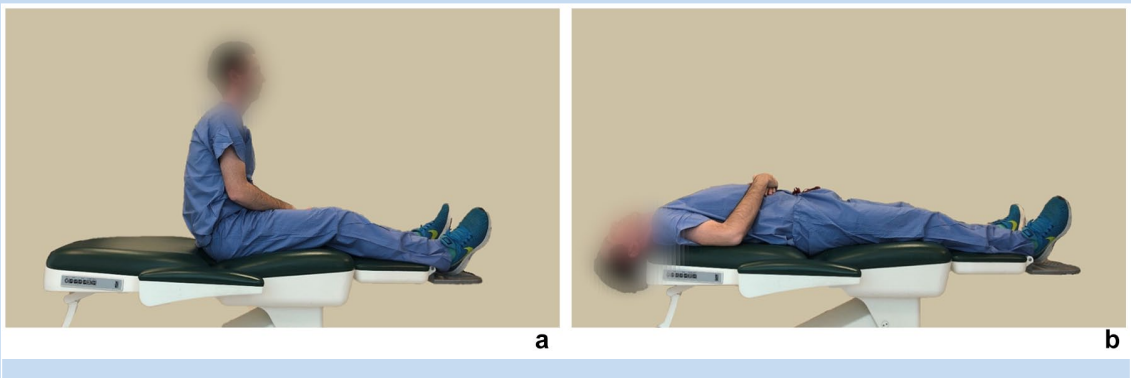

Figure 4. The midline head-hang maneuver to diagnose benign paroxysmal positional vertigo of the superior semicircular canal. (a) Patient is seated on examination table with head upright and in the midline. (b) The head and torso are moved rapidly to the supine position with the head hanging approximately 90° below the horizontal plane.

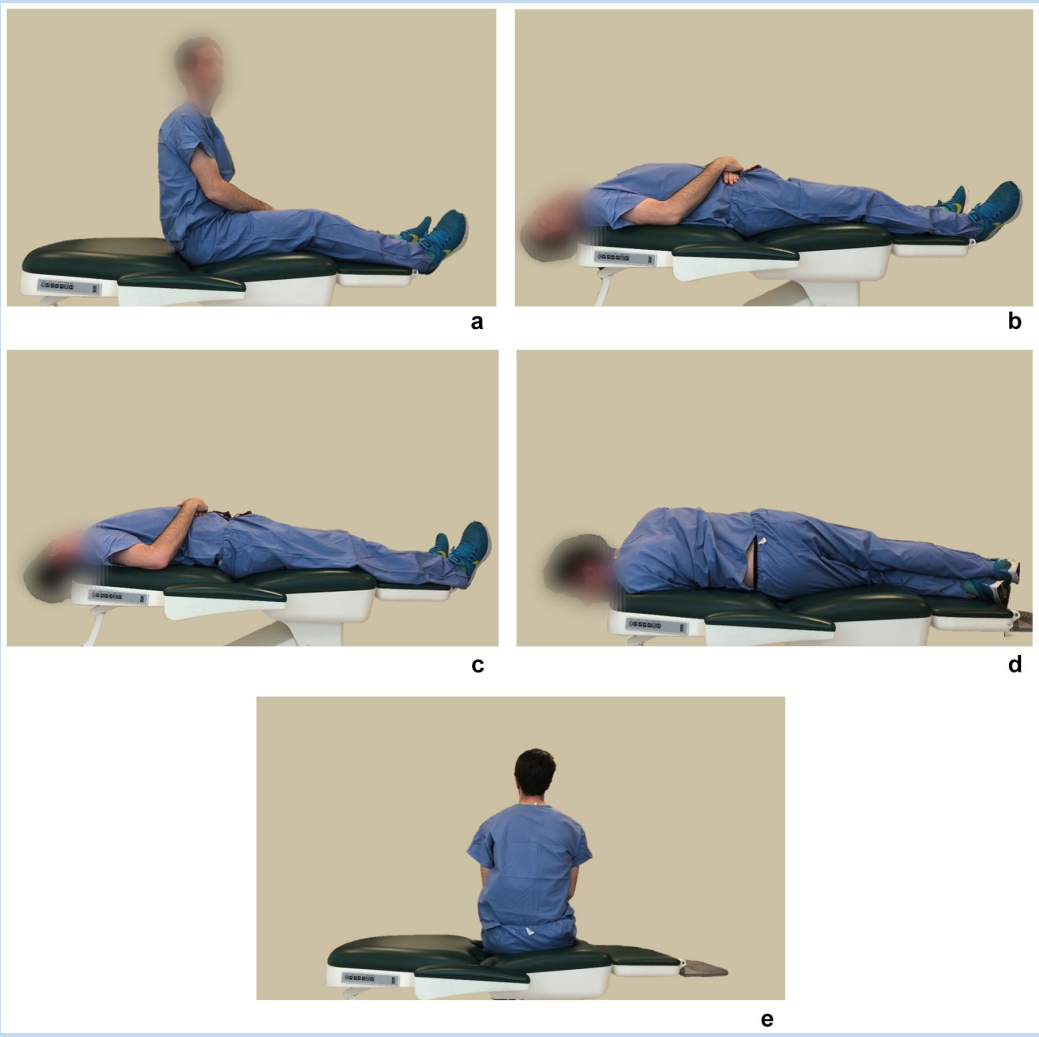

Figure 5. The Epley repositioning maneuver to treat benign paroxysmal positional vertigo (BPPV). The right Epley maneuver is pictured. The left Epley maneuver is the exact opposite of the right-sided maneuver, starting with the head turned to the left. Each position is maintained for approximately 30 seconds or until dizziness and nystagmus (if visible) resolves/improves for that position. (a) Patient is seated on examination table with head turned approximately 45° to the right side. (b) The head and torso are moved rapidly to the supine position with the head hanging approximately 45° below the horizontal plane with the head still turned to the right side. (c) The head is rotated 90° to the opposite side. (d) The head and torso are further rotated to bring the head an additional 90° toward the opposite side. (e) The patient is returned to the sitting position.

performed maneuver for posterior canal BPPV is the Epley maneuver (Figure 5 and Supplemental Video 4, available online). If superior or horizontal canal BPPV is suspected or if the affected canal is unclear or thought to be bilateral or multiple, then referral to a vestibular specialist should be pursued because determination of the appropriate treatment maneuver in these cases can be difficult without the use of VNG goggles. Patients will frequently experience resolution of symptoms after 1 or 2 maneuvers, but symptom improvement may take several hours to occur. It is also important to note that many patients may require several treatments over multiple visits for the BPPV to fully resolve. Additionally, many patients may have other contributing factors to their dizziness symptoms, so reexamination should be performed if symptoms persist. If the examination is then negative for persistent BPPV, then other causes should be considered. Other causes of dizziness should also be considered if a similar degree of subjective dizziness is reported in all diagnostic positions, particularly in the absence of visible nystagmus.

Prior studies in adults have shown that the risk of idiopathic BPPV increases with age and female gender.[12,15] However, our study did not find gender, age, or PCSS symptom score to be predictive factors for a diagnosis of BPPV in the setting of PCS; therefore, it may not be feasible to determine the risk of having

Table 4. Expected nystagmus patterns for benign paroxysmal positional vertigo of each semicircular canal[a]

Semicircular Canal	Vertical Component	Horizontal Component	Torsion
Posterior	Upbeating	Geotropic	Present
Horizontal	None	Geotropic or apogeotropic	Absent
Superior	Downbeating	Geotropic or none	May be either present or absent

[a]Direction refers to the fast phase of nystagmus, which is most easily seen by the examiner. Geotropic nystagmus refers to the fast phase moving toward the direction of the ground relative to the head. Apogeotropic nystagmus refers to the fast phase moving away from the direction of the ground relative to the head. Torsion refers to a component of the nystagmus where the eye appears to rotate around the axis of the pupil.

BPPV in the pediatric or adolescent population by evaluating risk factors alone. It is important to directly assess for BPPV on examination of the patient. It should also be noted that BPPV of the superior canals can cause dizziness when going from a supine to sitting position or from sitting to standing, so it can frequently mimic orthostatic dizziness, which is common after concussion. The brief vestibular-oculomotor screening examination has become a popular bedside examination tool to assess for vestibular dysfunction in patients with concussion.[14] This series of tests does not include assessment for BPPV. We propose that concussion specialists consider addition of BPPV assessment to their routine evaluation of all patients with concussion and ongoing dizziness.

Limitations of our study include a retrospective approach and a relatively small sample size. Additionally, our study sample was biased toward only patients with PCS because of the use of MDCC to identify affected patients. This program is primarily intended for patients with prolonged recovery and a diagnosis of PCS. Long-term follow-up to determine treatment impacts and overall recovery was limited by the retrospective study design. Involvement of primary concussion providers, including sports medicine specialists, neurologists, and primary care providers may help to better assess the role of earlier diagnosis and treatment of BPPV in patients with concussion before they reach the point of prolonged recovery and warrant evaluation in such a multidisciplinary clinic setting.

CONCLUSION

BPPV is common in children and adolescents with persistent dizziness after concussion. It can be diagnosed and treated relatively easily in an office setting. Increased awareness of the evaluation and management of BPPV among concussion providers may help expedite recovery for many patients.

REFERENCES

1. Alhilali LM, Yaeger K, Collins M, Fakhran S. Detection of central white matter injury underlying vestibulopathy after mild traumatic brain injury. *Radiology*. 2014;272:224-232.
2. Balatsouras DG, Koukoutsis G, Aspris A, et al. Benign paroxysmal positional vertigo secondary to mild head trauma. *Ann Otol Rhinol Laryngol*. 2017;126:54-60.
3. Bhattacharyya N, Baugh RF, Orvidas L, et al; American Academy of Otolaryngology–Head and Neck Surgery Foundation. Clinical practice guideline: benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2008;139(suppl 4):S47-S81.
4. Brodsky JR, Lipson S, Wilber J, Zhou G. Benign paroxysmal positional vertigo (BPPV) in children and adolescents: clinical features and response to therapy in 110 pediatric patients. *Otol Neurotol*. 2018;39:344-350.
5. Brodsky JR, Shoshany TN, Lipson S, Zhou G. Peripheral vestibular disorders in children and adolescents with concussion. *Otolaryngol Head Neck Surg*. 2018;159:365-370.
6. Chen JK, Johnston KM, Collie A, McCrory P, Ptito A. A validation of the post concussion symptom scale in the assessment of complex concussion using cognitive testing and functional MRI. *J Neurol Neurosurg Psychiatry*. 2007;78:1231-1238.
7. Eisenberg MA, Meehan WP 3rd, Mannix R. Duration and course of post-concussive symptoms. *Pediatrics*. 2014;133:999-1006.
8. Harris PA, Taylor R, Minor BL, et al; REDCap Consortium. The REDCap consortium: building an international community of software partners. *J Biomed Inform*. 2019;95:103208.
9. Harris PA, Taylor R, Thielke R, Payne J, Gonzalez N, Conde JG. Research electronic data capture (REDCap)—a metadata-driven methodology and workflow process for providing translational research informatics support. *J Biomed Inform*. 2009;42:377-381.
10. Hoffer ME, Gottshall KR, Moore R, Balough BJ, Wester D. Characterizing and treating dizziness after mild head trauma. *Otol Neurotol*. 2004;25:135-138.
11. Jahn K, Langhagen T, Heinen F. Vertigo and dizziness in children. *Curr Opin Neurol*. 2015;28:78-82.
12. Katsarkas A. Benign paroxysmal positional vertigo (BPPV): idiopathic versus post-traumatic. *Acta Otolaryngol*. 1999;119:745-749.
13. Lau BC, Kontos AP, Collins MW, Mucha A, Lovell MR. Which on-field signs/symptoms predict protracted recovery from sport-related concussion among high school football players? *Am J Sports Med*. 2011;39:2311-2318.
14. Mucha A, Collins MW, Elbin RJ, et al. A Brief Vestibular/Ocular Motor Screening (VOMS) assessment to evaluate concussions: preliminary findings. *Am J Sports Med*. 2014;42:2479-2486.
15. Neuhauser HK, Lempert T. Vertigo: epidemiologic aspects. *Semin Neurol*. 2009;29:473-481.
16. O'Reilly RC, Morlet T, Brodsky JR, Cushing SL. *Manual of Pediatric Balance Disorders*. 2nd ed. Plural; 2020.
17. Parnes LS, Agrawal SK, Atlas J. Diagnosis and management of benign paroxysmal positional vertigo (BPPV). *CMAJ*. 2003;169:681-693.
18. von Brevern M, Radtke A, Lezius F, et al. Epidemiology of benign paroxysmal positional vertigo: a population based study. *J Neurol Neurosurg Psychiatry*. 2007;78:710-715.
19. World Health Organization. *International Statistical Classification of Diseases and Related Health Problems*. World Health Organization; 2012.
20. Zhou G, Brodsky JR. Objective vestibular testing of children with dizziness and balance complaints following sports-related concussions. *Otolaryngol Head Neck Surg*. 2015;152:1133-1139.