The Journal of International Advanced Otology

J Int Adv Otol 2022; 18(2): 177–182 • DOI: 10.5152/iao.2022.20087

Review

Benign Paroxysmal Positional Vertigo in Children: A Narrative Review

Francesca Galluzzi[1], **Werner Garavello**[1,2]

[1]Department of Otorhinolaryngology, San Gerardo Hospital, Monza, Italy
[2]Department of Otorhinolaryngology, University of Milano-Bicocca Faculty of Medicine and Surgery, Monza, Italy

ORCID IDs of the authors: F.G. 0000-0001-9796-1964, W.G. 0000-0003-2339-2281.

Cite this article as: Galluzzi F, Garavello W. Benign paroxysmal positional vertigo in children: A narrative review. *J Int Adv Otol.* 2022;18(2):177-182.

Benign paroxysmal positional vertigo is a rare vestibular disorder in the pediatric population. It is a vestibulopathy characterized by brief attacks of vertigo, which occur after specific movements. This review aims to provide the current evidence regarding benign paroxysmal positional vertigo in children. This is a narrative review of the available literature on benign paroxysmal positional vertigo in children. The studies were retrieved from systematic searches on PubMed and by cross referencing. Few studies have focused on pediatric benign paroxysmal positional vertigo, and most are retrospective non-controlled studies that include a small number of children. The vast majority of cases of benign paroxysmal positional vertigo in children have been reported to be secondary. The most frequent forms involve the posterior canal and the horizontal canal. The diagnosis is based on positional maneuvers, respectively the Dix–Hallpike maneuver, which reveals a torsional upbeating nystagmus; and the supine roll test, which reveals a geotropic, horizontal nystagmus. The treatment consists of physical repositioning maneuvers: the Semont or the modified Epley maneuver for benign paroxysmal positional vertigo involving the posterior canal and the Gufoni or the Barbecue maneuver in case of the horizontal canal. Benign paroxysmal positional vertigo in children can be resistant to treatment and repetitive positional maneuvers may be necessary, particularly for children with vestibular migraine or benign paroxysmal vertigo of childhood, who have a statistically significant major risk of having recurrences compared to patients who do not. Benign paroxysmal positional vertigo in children is a rare but well-recognized clinical entity. It is diagnosed by positional testing and treated by repositioning maneuvers. Wide awareness and education among pediatric providers and otolaryngologists are needed in order to avoid a delay in identification and treatment.

KEYWORDS: Benign paroxysmal positional vertigo, children, positional nystagmus, vertigo

INTRODUCTION

Epidemiology

Vestibular disorder in the pediatric population is a rare condition, with a prevalence that ranges between 0.4% and 8%.[1,2] Epidemiologic studies among pediatric patients with dizziness reported that the prevalence of benign paroxysmal positional vertigo (BPPV) ranges from 1-1.6% to 5%, and that it is prevalent among elementary school children and adolescents.[2-4] However, Brodsky et al[5] in a retrospective study regarding pediatric dizziness, have found that out of 605 children in their program for dizziness, 120 patients (19.8%) were diagnosed with BPPV. This high percentage of BPPV occurrence may be related to various factors: (1) the affiliation of their hospital with a concussion clinic (in fact, 80.7% of BPPV cases were secondary to trauma); (2) the inclusion of children with other comorbid diagnoses such as vestibular neuritis and vestibular migraine, which usually are mutually exclusive; (3) the use of positional testing in routine examination; and (4) the use of videonystagmography (VNG), which improves the identification of nystagmus.

Considering this study, which is the largest single report of pediatric BPPV cases, the authors reported a female-to-male ratio of approximately 3:2.[5] In addition, they observed the long delay in diagnosis (mean time of diagnosis was 172.2 days) that reflects a lack of awareness about BPPV among pediatric healthcare providers.

In this perspective, we performed a narrative review, considering the available evidence in the literature on BPPV in children.

Corresponding author: Francesca Galluzzi, e-mail: f.galluzzi@asst-monza.it
Recieved: November 10, 2020 • **Accepted:** July 6, 2021
Available online at www.advancedotology.org

Evidences of Pediatric Benign Paroxysmal Positional Vertigo

Reports of pediatric BPPV are very limited in medical literature. Indeed, it has been studied less extensively than the other predominant forms of vertigo such as benign paroxysmal vertigo of childhood (BPVC) or vestibular migraine. The first case of pediatric posterior canal BPPV (PC-BBPV) was documented in 1987[6] and a case of horizontal canal BPPV (HC-BBPV) in 2003.[7] During the past decades, various cases of BPPV in children have been reported and different causes were noted such as cochlear implant surgery,[8] head trauma,[9] and the association with the enlarged vestibular aqueduct.[10] Considering the studies including children with BPPV, Steenerson et al[11] presented a retrospective study counting a large number of cases with BPPV (923 patients) ranging in age from 12 to 94 years, but they did not report selected data of the pediatric patients. Few studies are focused on pediatric BPPV, and they are mostly retrospective and non-controlled, including only a small number of children. Overall, they report a prevalence among females,[5,12-17] and the mean age ranges from 6.1[14] to 13.4 years.[5]

Pediatric Benign Paroxysmal Positional Vertigo and Migraine

In the pediatric age, the most common cause of vertigo is BPVC, which is considered an early manifestation of migraine called "migraine precursor and/or equivalent."[18-20] Recently, the Bárány Society and the International Headache Society had decided to change the terminology from BPVC to vestibular migraine of childhood (VMC), probable VMC, and recurrent vertigo of childhood (RVC), 3 disorders of a spectrum in which the migraine component varies from definite to possible absent.[21]

Although the evidence is scanty, some authors have suggested a relationship between migraine and BPPV in children.[5,16,22]

Baloh et al[22] in 1998 first described a childhood onset of benign positional vertigo in 3 members of a family, each of whom also had migraine, thus meeting the criteria of the International Headache Society. They hypothesized that in this family, the benign positional vertigo was probably part of the migraine syndrome. After this report, the association between migraine and BPPV in children was reported by Balatsouras et al[16] who described 4 children, with a mean age of 9.2 years, with a family history of migraine. Most recently, Brodsky et al[5] suggested that there is an increased risk of BPPV in pediatric patients with migraine, though it had been previously reported in adult patients.[23-27] They reported that out of 110 children with BPPV, 33 included cases who were also diagnosed with vestibular migraine or BPCV; and they demonstrated that these patients had 5-fold higher odds of recurrence of BPPV, $P = .003$, 95% [1.735, 15.342] than those who did not have any.[5] Table 1 shows the main differences between BPPV and BPVC.

MATERIALS AND METHODS

Two authors (G.F. and G.W.) independently searched the electronic databases (PubMed, Embase, and the Cochrane Library) to find articles regarding BPPV in children. The following search terms were used: "benign paroxysmal positional vertigo," "vertigo," "positional maneuvers," "positional nystagmus," "children," and "pediatric." The terms were adjoined with adjuncts of "and" as well as "or." The exclusion criteria were non-English articles and those focused on adults. All resulting titles, abstract, and full text, whenever available, were read and kept for reference, and the findings were critically summarized. A comprehensive narrative review regarding BPPV in children was conducted.

RESULTS AND DISCUSSION

Etiology and Pathophysiology

Benign paroxysmal positional vertigo is a disorder of the vestibular labyrinth, characterized by episodes of transient attacks of vertigo in particular head positions or after specific movements. As it has been previously descibed by Nuti et al[28], this condition is associated with positional nystagmus, provoked by changes position

Table 1. Differential Diagnosis Between BPPV and BPVC

	Benign Paroxysmal Positional Vertigo (BPPV)	Benign Paroxysmal Vertigo of Childhood (BPVC)
Age (years)	6-13	Two peaks 2-4 and 7-11
Gender	Female	Female
Symptoms	Transient attacks of vertigo in particular head positions or after specific movements Duration < 30s	Recurrent attacks of severe vertigo resolving spontaneously after minutes to hours, occurring without warning 2-10/episodes/month
Physiopathology	Cupulolithiasis and canalithiasis	Migraine precursor or equivalent
Diagnosis	Positional maneuvers: Dix–Hallpike for PC-BPPV Pagnini–McClure for HC-BPPV	International Classification of Headaches (ICHD-2) criteria: A. at least 5 attacks fulfilling criterion B B. Multiple episodes of severe vertigo, occurring without warning and resolving spontaneously after minutes to hours C. Normal neurological examination; audiometric and vestibular functions between attacks D. Normal electroencephalogram
Treatment	Semont or modified Epley maneuver for PC-BPPV Gufoni or Barbecue maneuver for HC-BPPV	Spontaneous recovery before adolescence
Recurrences	14.5%	16-19% evolved to migraine 15-27% developed abdominal pain or cyclic vomiting syndrome

PC-BPPV. posterior canal-benign paroxysmal positional vertigo; HC-BPPV. horizontal canal-benign paroxysmal positional vertigo; BPVC. paroxysmal positional vertigo of childhood.

of the head with respect to gravity. Two major theories have been proposed for the underlying mechanism of BPPV, namely, cupulolithiasis and canalithiasis. Both hypotheses were initially formulated with regard to adult patients. Cupulolithiasis was first described by Schuknecht et al.[29] In this mechanism, the cupula of the semicircular canal becomes gravity-sensitive when detached otoconial debris adheres to it. Canalithiasis was identified surgically by Parnes et al[30] It occurs when otoconial debris is displaced in the semicircular canal following head movements. Therefore, endolymph with otoconia flows in the semicircular canal and the displaced cupula, leading to an attack of rotational vertigo. Later, the same 2 mechanisms have been recognized in pediatric BPPV, although the most accepted hypothesis in children is cupulolithiasis (heavy cupula). Baloh et al[6] speculated that otoconia are firmly attached to the macula and not dislocated in childhood. Later, a histopathological study of the temporal bones revealed cupular deposits that were most often observed on the lateral cupula, especially in case of very young children.[31] However, data indicate a lower incidence of cupular deposits in pediatric temporal bones compared with those of adults, suggesting that cupulolithiasis could be a phenomenon primarily associated with aging of the vestibular system.[31] This is confirmed by epidemiological studies which report BPPV as the most common cause of vertigo in adults, with a rate of incidence that increases at over 60 years of age and decreases exponentially below 40, being very rare in children.[32] Interestingly, Brodsky et al[5] have found a progressive incidence of BPPV with age in a large study on the pediatric population.

The cause of the detachment of otoconia from the utricular macula in children is unknown. It has been suggested that ischemic damage has a role in the etiopathogenesis of BPPV. Baloh et al[6] hypothesized that a vasospasm of the labyrinth arteries resulted in ischemic damage to the utricular macule, leading to the development of BPPV, similar to the vascular pathogenesis of migraine. In this regard, Uneri et al[33] concluded that there might be a causal connection between migraine and BPPV because of the incidence of BPPV in children with a history or a positive family history of migraine. The other recognized conditions that could have a role in the etiopathogenesis of BPPV are head trauma that mechanically could determine the dislodgement of the otoconia, surgery, or vestibular neuritis.[5,9,15,17] Finally, a potential contributing factor for BPPV is vitamin D deficiency. Although this association has been well described for adults,[34] no conclusions have been drawn in the pediatric population. Brodsky et al[5] reported that they routinely test for and treat vitamin D deficiency in children with treatment-resistant or recurrent BPPV. Overall, as Brodsky et al[5] confirmed, the vast majority of BPPV (80.7%) in children may be considered secondary.

Clinical Features and Diagnosis

Typical symptoms of BPPV are elicited when the head is positioned so that gravity causes sustained movement of the endolymph within the affected semicircular canal. The presence of the otoliths in the canal or on the cupula results in nystagmus in the plane of the affected canal and causes brief and intense subjective vertigo. A prolonged instability may persist even following the disappearance of the acute vertigo.[14] However, the clinical features of BPPV in the pediatric population are still to be well-characterized. Indeed, vertigo is not easy to study in children.[12] In most cases, the children could not narrate their symptoms (such as true vertigo, unsteadiness, or imbalance) and the parents were not able to understand and describe what vertigo is to their children. In addition, as in the case of BPVC and migraine,[35] children with BPPV may experience emotional and behavioral symptoms such as anxiety and depressive and hyperactivity symptoms, which is extremely troublesome for parents; therefore, a prompt differential diagnosis is important.[14] Casani et al[36] suggested that this condition may also generate a sense of anxiety, both in parents and physicians, leading to an excessive number of prescriptions for functional testing and imaging examinations that are often unsuitable for proper therapeutic decision-making. Before the visit, it is necessary to explain to the parents the modality of execution of the vestibular bedside examination and gather their consent. Indeed, children cannot tolerate the Frenzel goggles or video-oculography, and the positional maneuvers that induce vertigo could scare them. Obtaining the children's compliance with their parents' agreement allows a proper vestibular evaluation.

As mentioned above, the diagnosis of BPPV is based on the presence of positional nystagmus evoked by positional maneuvers, according to the Bárány Society's criteria.[37] To date, the Dix–Hallpike maneuver for diagnosis of posterior semicircular canal BPPV and the supine roll test (Pagnini–McClure maneuver) for HC-BPPV have been reported.[5,14,16,17] The Dix–Hallpike maneuver is conducted by moving the patient rapidly from the sitting position, with the head rotated at 45° toward the pathological ear, to a position with the head hanging 45° below the horizontal plane. According to the canalolithiasis mechanism, when the debris is located in the ampullary arm, the movement produces an utriculofugal deflection of the cupula by the shift of the otoliths away from the ampulla, and the onset, after a latency of 2-10 seconds, of the typical mixed torsional, paroxysmal, and upbeating nystagmus.[5,14,16,17] A rotational vertigo is associated with the nystagmus and lasts for 10-20 seconds. The return to the sitting position causes an utriculopetal deflection with a nystagmus in the opposite direction. In case of cupulolithiasis, the nystagmus pattern could be different: identical in direction but gradual in onset, not paroxysmal, and persisting as long as the provocative position is maintained.[28] The supine head roll test is the preferred maneuver for the diagnosis of HC-BPPV.[5,17] It is performed by initially positioning the patient in a supine position, with the head in a neutral position and bent about 30° forward to bring the lateral canal into the vertical plane. After checking for any positional nystagmus, the patient's head is quickly rotated 90° to one side and 180° to the other side. This test acts in a plane parallel to the lateral canal, and therefore, depending on the different locations of the calcium carbonate debris, it could elicit 2 potential types of direction-change positional nystagmus. The most common is the geotropic variant, when the otoconia are located in the long non-ampullary arm. The rotation of the head to the pathologic side causes an intense horizontal nystagmus beating toward the lower ear or the ground (geotropic form), and the rotation to the opposite site provokes a less intense nystagmus, again geotropic, according to Ewald's second law.[28,38] The apogeotropic HC-BPPV variant is extremely rare, and only Yao et al[17] in a retrospective study conducted on 6 children with age ranging from 7 to 14, described 1 case of a 14-year-old girl. In this form, the calcium carbonate debrides seem to be located in the ampullary arm or adherent/close to the cupula of the lateral semicircular canal. The roll test results in a horizontal nystagmus beating toward the uppermost ear (apogeotropic nystagmus). Similarly, on rolling to the opposite

side, the nystagmus change direction will have the fast phase toward the uppermost ear.

Another form is the BPPV of the anterior canal (AC-BPPV). The existence of this type of canalolithiasis is still debated and the cupulolithiasis has not been documented.[37] It is extremely rare in adults, and only Brodsky et al[5] reported 21 cases of AC-BPPV in children. Indeed, this is the largest single report of pediatric BPPV in which positional maneuvers were routinely performed in all children. The anterior canal is located in the higher portion of the labyrinth and it is unlikely that otoconia enter it unless the patient is upside-down. Moreover, since its posterior arm descends directly into the common crus, they should easily leave the canal when the patient is upright. The diagnostic maneuver is the supine straight-head-hanging position. It provokes a positional torsional downbeating nystagmus that should reverse when the patient returns to the sitting position.

Finally, debris might be also temporarily in the common crus area, which is the shared canal between the anterior and posterior canals. However, to date, there are no data regarding common crus BPPV in the pediatric population. Further studies are needed in order to study atypical forms of BPPV in children.[39]

Overall, considering the different forms of pediatric BPPV, the posterior canal is the most frequently affected, followed by the horizontal canal. Brodsky et al[5] noticed a higher proportion of patients with multiple and/or bilateral canal involvement and/or canal conversion. They supposed that children are generally more active than adults, routinely participating in activities such as swimming, gymnastics, or dancing that could increase the displacing of otoconia into a less gravity-dependent canal or into multiple canals. However, if children are more active, then the spontaneous resolution of BPPV may be an equal possibility.

Instrumental Diagnosis and Differential Diagnosis

Although the diagnosis of pediatric BPPV is based on positional maneuvers, positional nystagmus (mainly downbeating nystagmus) is sometimes caused by structural lesions of the central vestibular system. A careful vestibulo-ocular examination, including vestibulo-ocular-provocative maneuvers and eye movement assessment, could reveal "atypical features" that do not conform to the Bárány Society's criteria for BPPV,[37] prompting further investigation. Usually, these cases are associated with neurological signs and symptoms. Therefore, complete vestibular, neurological, and ophthalmological assessments, including electroencephalography and brain imaging, are mandatory. To date, in these clinically atypical cases, a standardized protocol is missing. Magnetic resonance imaging or computed tomography are routinely performed,[5,14,17] or in selected cases, depending on the presence of risk factors: a history of headaches or loss of consciousness, head trauma, otitis media, or central pattern electronystagmography (ENG) findings.[12,15,16] The central disorders to rule out comprise congenital anomalies (such as the Arnold–Chiari malformation that may be associated with vertigo), vestibular epilepsy, familial diseases (mainly the benign paroxysmal torticollis of infancy, vestibular migraine, and familial periodic ataxia-vertigo) or acquired diseases (essentially infratentorial tumors, trauma, encephalitis, degenerative diseases, vascular events, toxins, and psychogenic vertigo).[40] Furthermore, all of these conditions may coexist with BPPV.[5] Thus, both vestibular and neurological assessments are equally required.

Children with BPPV need to have a full vestibular function test battery as BPPV is usually secondary.[5] Vestibular testing is particularly indicated in patients suggesting a comorbid diagnosis in addition to BPPV or to confirm BPPV in cases where diagnosis is uncertain.[38] Brodsky et al[5] analyzed a large number of BPPV children (94 subjects) using vestibular instrumental exams. They reported abnormal results on VNG, both for oculomotor (20 cases) and positional VNG (82 cases); the rotary chair test was pathological in 10 cases; the video head impulse test in 25, and bithermal caloric testing in 2 patients. Overall, they concluded that VNG could be useful in order to confirm positional nystagmus and determining which canal(s) is affected, considering that the observation of the video itself is essential to see the nystagmus torsional component that the VNG tracing does not demonstrate. Other vestibular exam findings in BPPV children are described by Balatsouras et al[16] who reported in 2 cases a peripheral pattern recorded on ENG,[16] and Choung et al[12] testing with rotatory chair, who reported 2 patients who obtained abnormal values (gain and asymmetry) of the vestibulo-ocular reflex and the visual vestibulo-ocular reflex. Erbek et al[15] and Marcelli et al[14] analyzed respectively 6 and 8 subjects, finding the vestibular examination normal except for the positional maneuvers. Finally, Yao et al[17] also retrospectively studied 6 children with BPPV who had undergone otoneurological and neurological assessments, but they did not specify the performed tests. Overall, vestibular testing does not diagnose BPPV but is pivotal for diagnosing other coexisting vestibular lesions.

The audiological assessment (pure-tone audiometry, impedance test with stapedial reflex search, auditory evoked potentials, and otoacoustic emissions) is required for a proper differential diagnosis. Indeed, BPPV is not normally associated with auditory symptoms. In case vertigo is accompanied by otological symptoms, some peripheral pathologies should be suspected. These include congenital anomalies (i.e., enlarged vestibular aqueduct), familial causes (i.e., familial vestibular areflexia), and acquired causes (viral or bacterial neuro-labyrinthitis, trauma, delayed endolymphatic hydrops, otitis media- or middle-ear-effusion-related dizziness, cholesteatoma, ototoxic drugs, or autoimmune disease, and rarely, Meniere's disease).[14,36,40,41]

Treatment and Recurrence

The therapy for BPPV is based on physical repositioning maneuvers and positions. To date, the same types proposed for adult patients are used. The liberatory maneuvers involve head movements allowing the repositioning of the otoconial mass through the non-ampullar arm towards the utricle.

In the case of geotropic PC-BPPV, this is achieved by the Semont maneuver, the Epley maneuver, or the Modified Epley maneuver.[5,14,16,17] Geotropic HC-BPPV is treated with the Gufoni maneuver,[5,17] while the apogeotropic variant with the Barbecue maneuver, as it was reported in 1 child by Yao et al.[17] Finally, the approach in case of the involvement of the anterior canal (AC-BPPV) could involve the Yacovino maneuver, the Rahko maneuver, or the reverse Epley maneuver as it was performed by Brodsky et al.[5] There are no

standardized criteria for maneuver execution in terms of types and numbers. Yao et al[17] performed maneuvers weekly until no nystagmus occurred and/or vertigo disappeared. Brodsky et al[5] have considered 5 maneuvers as the cutoff to establish treatment resistance in a child. The duration of follow-up ranges from 12[14] to 22 months. Interestingly, Yao et al[17] reported the time of resolution according to the types of canal involvement and found that 2 patients with HC-BPPV were cured in 3-7 days, while 4 children affected by PC-BPPV needed almost from 1 month to 1 year. For resistant positional vertigo, though repeated maneuvers are described, risk factors of recurrences have been hypothesized. Yao et al[17] reported recurrences in 1 out of 6 patients with PC-BPPV, Brodsky et al[5] in 20 out of 138 cases, while Marcelli et al[14] reported no recurrences in 8 children with PC-BPPV. Table 2 shows the comorbid diagnoses in children with BPPV. Only Brodsky et al[5] demonstrated that children with vestibular migraine or benign paroxysmal vertigo have a statistically significant major risk of having recurrences than patients who do not.

Finally, the key findings of BPPV in children in terms of etiology, pathophysiology, diagnosis, and treatment are summarized in Table 3.

Table 2. Comorbidities in Children with BPPV

Comorbidities in BPPV
Vestibular migraine or benign paroxysmal vertigo of childhood[5,16]
Head trauma[5,17]
Family history of vertigo[17]
Vestibular neuritis or labyrinthitis[5]
Ototoxic drugs[17]
Otitis media[5]
Congenital sensorineural hearing loss[5,17]
Persistent postural-perceptual dizziness[5]
Idiopathic[5,14]

Table 3. Key Findings in BPPV in Children

Etiology	The cause of the detachment of otoconia from the utricular macula is still unknown. The vast majority were reported to be secondary.
Pathophysiology	Cupulolithiasis Canalithiasis
Forms	Posterior canal BPPV Horizontal canal BPPV Anterior canal BPPV (rarely)
Diagnosis	Positional maneuvers: PC-BPPV: Dix–Hallpike maneuver – upbeating, torsional ny HC-BPPV: Supine roll test – geotropic, horizontal ny AC-BPPV: Straight-head-hanging position --> downbeating, torsional ny
Treatment	Repositioning maneuvers: PC-BPPV: the Semont or the modified Epley maneuver HC-BPPV: the Gufoni or the Barbecue maneuver AC-BBPV: the Yacovino or the Rahko or the reverse Epley maneuver

BPPV, benign paroxysmal positional vertigo; ny, nystagmus; PC-BPPV, posterior canal BPPV; HC-BPPV, horizontal canal BPPV; AC-BPPV, anterior canal-BPPV.

CONCLUSION

Although benign paroxysmal positional vertigo in children is a rare condition, to date it is a well-recognized clinical entity. It can be accurately diagnosed by positional testing and appropriately treated by repositioning maneuvers. In case of atypical positional nystagmus, further investigations are needed. In order to avoid a delay in identification and treatment of BPPV, wide awareness and education among pediatric providers as well as referred otolaryngologists should be required.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – G.F., G.W.; Design – G.F., G.W.; Supervision – G.W.; Resources – G.F.; Materials – G.F., G.W.; Data collection and/or Processing – G.F.; Analysis and/or interpretation – G.F., G.W.; Literature Search – G.F., G.W.; Writing Manuscript – G.F.; Critical Review – G.W.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study has received no financial support.

REFERENCES

1. Niemensivu R, Pyykkö I, Wiener-Vacher SR, Kentala E. Vertigo and balance problems in children—an epidemiologic study in Finland. *Int J Pediatr Orl*. 2006;70(2):259-265. [CrossRef]
2. O'Reilly RC, Morlet T, Nicholas BD, et al. Prevalence of vestibular and balance disorders in children. *Otol Neurotol*. 2010;31(9):1441-1444. [CrossRef]
3. Lee JD, Kim CH, Hong SM, et al. Prevalence of vestibular and balance disorders in children and adolescents according to age: a multi-center study. *Int J Pediatr Otorhinolaryngol*. 2017;94:36-39. [CrossRef]
4. Wiener-Vacher SR, Quarez J, Priol AL. Epidemiology of vestibular impairments in a pediatric population. *Semin Hear*. 2018;39(3):229-242. [CrossRef]
5. 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(3):344-350. [CrossRef]
6. Baloh RW, Honrubia V, Jacobson K. Benign positional vertigo: clinical and oculographic features in 240 cases. *Neurology*. 1987;37(3):371-378. [CrossRef]
7. D'Agostino R, Melagrana A, Taborelli G. Benign positional paroxysmal vertigo of horizontal semicircular canal in the child: case report. *Int J Pediatr Orl*. 2003;67(5):549-551. [CrossRef]
8. Shetye A. Benign paroxysmal positional vertigo in a child: an infrequent complication following a fairground ride and post-cochlear implant surgery. *Cochlear Implants Int*. 2012;13(3):177-180. [CrossRef]
9. Nørgaard MS, Rokkjær MS, Berg J, Lüscher M. [Benign paroxysmal positional vertigo in children after head trauma]. *Ugeskr Laeger*. 2015 Jun 15;177(25):12140706.
10. Song JJ, Hong SK, Kim JS, Koo JW. Enlarged vestibular aqueduct may precipitate benign paroxysmal positional vertigo in children. *Acta Oto-Laryngol*. 2012;132(Suppl 1):S109-S117. [CrossRef]
11. Steenerson RL, Cronin GW, Marbach PM. Effectiveness of treatment techniques in 923 cases of benign paroxysmal positional vertigo. *Laryngoscope*. 2005;115(2):226-231. [CrossRef]
12. Choung YH, Park K, Moon SK, Kim CH, Ryu SJ. Various causes and clinical characteristics in vertigo in children with normal eardrums. *Int J Pediatr Otorhinolaryngol*. 2003;67(8):889-894. [CrossRef]
13. Riina N, Ilmari P, Kentala E. Vertigo and imbalance in children: a retrospective study in a Helsinki University Otorhinolaryngology Clinic. *Arch Otolaryngol Head Neck Surg*. 2005;131(11):996-1000. [CrossRef]

14. Marcelli V, Piazza F, Pisani F, Marciano E. Neuro-otological features of benign paroxysmal vertigo and benign paroxysmal positioning vertigo in children: a follow-up study. *Brain Dev*. 2006;28(2):80-84. [CrossRef]
15. Erbek SH, Erbek SS, Yilmaz I, et al. Vertigo in childhood: a clinical experience. *Int J Pediatr Orl*. 2006;70(9):1547-1554. [CrossRef]
16. Balatsouras DG, Kaberos A, Assimakopoulos D, Katotomichelakis M, Economou NC, Korres SG. Etiology of vertigo in children. *Int J Pediatr Otorhinolaryngol*. 2007;71(3):487-494. [CrossRef]
17. Yao Q, Song Q, Wang H, Shi H, Yu D. Benign paroxysmal positional vertigo in children. *Clin Otolaryngol*. 2019;44(1):21-25. [CrossRef]
18. Fenichel GM. Migraine as a cause of benign paroxysmal vertigo of childhood. *J Pediatr*. 1967;71(1):114-115. [CrossRef]
19. Ralli G, Atturo F, de Filippis C. Idiopathic benign paroxysmal vertigo in children, a migraine precursor. *Int J Pediatr Orl*. 2009;73(Suppl 1):S16-S18. [CrossRef]
20. Brodsky J, Kaur K, Shoshany T, Lipson S, Zhou G. Benign paroxysmal migraine variants of infancy and childhood: transitions and clinical features. *Eur J Paediatr Neurol*. 2018;22(4):667-673. [CrossRef]
21. van de Berg R, Widdershoven J, Bisdorff A, et al. Vestibular migraine of childhood and recurrent vertigo of childhood: diagnostic criteria consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society and the International Headache Society. *J Vestib Res*. 2021;31(1):1-9. [CrossRef]
22. Baloh RW, Honrubia V. Childhood onset of benign positional vertigo. *Neurology*. 1998;50(5):1494-1496. [CrossRef]
23. Faralli M, Cipriani L, Del Zompo MR, Panichi R, Calzolaro L, Ricci G. Benign paroxysmal positional vertigo and migraine: analysis of 186 cases. *B-ENT*. 2014;10(2):133-139.
24. Chu CH, Liu CJ, Lin LY, Chen TJ, Wang SJ. Migraine is associated with an increased risk for benign paroxysmal positional vertigo: a nationwide population-based study. *J Headache Pain*. 2015;16(1):62. [CrossRef]
25. Yetiser S, Gokmen MHA. Clinical aspects of benign paroxysmal positional vertigo associated with migraine. *Int Tinnitus J*. 2015;19(2):64-68. [CrossRef]
26. Teixido M, Baker A, Isildak H. Migraine and benign paroxysmal positional vertigo: a single-institution review. *J Laryngol Otol*. 2017;131(6):508-513. [CrossRef]
27. Kim SK, Hong SM, Park I-S, Choi HG. Association between migraine and benign paroxysmal positional vertigo among adults in South Korea. *JAMA Otolaryngol Head Neck Surg*. 2019;145(4):307.
28. Nuti D, Masini M, Mandalà M. Benign paroxysmal positional vertigo and its variants. In: *Handbook of Clinical Neurology [Internet]*. Elsevier; 2016:241-256. Available at: https://linkinghub.elsevier.com/retrieve/pii/B9780444634375000182. Citato 5 novembre 2020.
29. Schuknecht HF, Ruby RR. Cupulolithiasis. *Adv Otorhinolaryngol*. 1973;20:434-443. [CrossRef]
30. Parnes LS, McClure JA. Free-floating endolymph particles: a new operative finding during posterior semicircular canal occlusion. *Laryngoscope*. 1992;102(9):988-992. [CrossRef]
31. Bachor E, Wright CG, Karmody CS. The incidence and distribution of cupular deposits in the pediatric vestibular labyrinth. *Laryngoscope*. 2002;112(1):147-151. [CrossRef]
32. 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(7):710-715. [CrossRef]
33. Uneri A, Turkdogan D. Evaluation of vestibular functions in children with vertigo attacks. *Arch Dis Child*. 2003;88(6):510-511. [CrossRef]
34. Chen J, Zhang S, Cui K, Liu C. Risk factors for benign paroxysmal positional vertigo recurrence: a systematic review and meta-analysis. *J Neurol*. 2021;268(11):4117-4127. [CrossRef]
35. Reale L, Guarnera M, Grillo C, Maiolino L, Ruta L, Mazzone L. Psychological assessment in children and adolescents with benign paroxysmal vertigo. *Brain Dev*. 2011;33(2):125-130. [CrossRef]
36. Casani AP, Dallan I, Navari E, Sellari Franceschini S, Cerchiai N. Vertigo in childhood: proposal for a diagnostic algorithm based upon clinical experience. *Acta Otorhinolaryngol Ital*. 2015;35(3):180-185.
37. von Brevern M, Bertholon P, Brandt T, et al. Benign paroxysmal positional vertigo: diagnostic criteria: consensus document of the Committee for the Classification of Vestibular Disorders of the Bárány Society. *VES*. 2015;25(34):105-117. [CrossRef]
38. Bhattacharyya N, Gubbels SP, Schwartz SR, et al. Clinical practice guideline: benign paroxysmal positional vertigo (update). *Otolaryngol Head Neck Surg*. 2017;156(3_suppl):S1-S47. [CrossRef]
39. Yetiser S. A new variant of posterior canal benign paroxysmal positional vertigo: a nonampullary or common crus Canalolithiasis. *Case Rep Otolaryngol*. 2015;2015:816081. [CrossRef]
40. Gioacchini FM, Alicandri-Ciufelli M, Kaleci S, Magliulo G, Re M. Prevalence and diagnosis of vestibular disorders in children: a review. *Int J Pediatr Orl*. 2014;78(5):718-724. [CrossRef]
41. Gruber M, Cohen-Kerem R, Kaminer M, Shupak A. Vertigo in children and adolescents: characteristics and outcome. *Sci World J*. 2012;2012:1-6.