Vertigo and Dizziness in Childhood

李薰華

- 16.1 Clinical Aspects
- 16.2 Vestibular Migraine of Childhood and Recurrent Vertigo of Childhood
- 16.3 Episodic Ataxias
- 16.4 Motion Sickness
- 16.5 Visual Height Intolerance and Acrophobia
- 16.6 Therapy of Childhood Forms of Vertigo

The dizzy child

TJ Balkany, RS Finkel - Ear and hearing, 1986 - journals.lww.com

0196/0202/86/0703-0138$02.00/0
EAR AND HEARING
Copyright © 1986 by The Williams & Wilkins Co.
Vol. 7, No. 3
Printed in U.S.A.

The Dizzy Child

Thomas J. Balkany, and Richard S. Finkel

Colorado Ear Clinic, Denver, Colorado

"Dizziness" encompasses several subjective disorders of imbalance and has no strict definition. Patients with vertigo, ataxia, and light-headedness may present to the physician in similar ways, especially young children in whom an accurate description of the complaint is lacking and the observed response to dizziness is nonspecific. Although such complaints are most often benign and self-limiting, they may herald an important otologic or neurologic disorder and require careful evaluation. Evaluation of the young dizzy child is made more difficult by limited communication abilities.

Most diagnostic efforts in dizzy adults are focused on chronic diseases, degenerative diseases, or tumors. On the contrary, in children most processes are acute and self-limiting. Nonetheless, because of the importance of disease processes which may present with vertigo in children, it is essential to come to an appropriate diagnosis.

As in adults, vertigo in children is most often a symptom of lesions of the peripheral vestibular system. However, other less specific forms of dizziness such as "light-headedness," "tipsyness," "dysequilibrium," and "imbalance" are too often overlooked as important symptoms and may represent significant lesions of the eighth cranial nerve or central nervous system.

The adult with dizziness may be categorized into three subgroups: (1) vertigo, usually of labyrinthine or central nervous system origin; (2) light-headedness or presyncope, usually of cardiac origin or related to systemic disease; and (3) vague dysequilibrium which may be an organic or functional symptom (1). In children, the differential diagnosis is broader due to immaturity of the developing nervous system and the limited repertoire of prior like-experiences.

The vestibular system is among the first of the central nervous projections to function in infancy. It is clear that the vestibular system is responsive to positional change within the first 6 weeks of life and that full development is thought to occur by 16 weeks (2).

The evaluation of dizziness in children includes obtaining as complete a history as possible from both the parent and child, complete physical examination, tests of hearing and balance as well as evaluation of possible systemic or central nervous system etiologies.

PHYSIOLOGY

Maintenance of balance depends upon proper assimilation in the brain of afferent input from the optic, labyrinthine, and peripheral neuromuscular systems. This network usually works at a subliminal level and is adapting continuously to alterations in these stimuli. These neural pathways are incompletely developed at birth with myelination of the optic radiations (3), for example, not completely myelinated until age 4 months. Labyrinthine function, by comparison, is more advanced at birth with a tonic neck response usually elicitable by 1 month of age. Proprioceptor input from the muscles and joints ascends along sensory pathways in the spinal cord which are likewise incompletely myelinated at birth. These afferent stimuli connect with the cerebellum via the oculomotor, vestibular and red nuclei. Thus, diseases which affect the visual, labyrinthine, or proprioceptor symptoms, the brain stem connections, or the cerebellar pathways will result in a sense of imbalance.

The hallmark of vertigo is a sense of motion, usually spinning, although dropping, swaying, and less dramatic sensations are also described. Even the young child can describe vertigo in graphic terms, e.g., "the room is spinning"; "the walls are falling"; "my legs are running around." Vertigo usually reflects labyrinthine, VIIIth nerve, or brain stem disease. However, any posterior fossa lesion can, directly or through pressure effect, cause vertigo. Irritation of upper cervical sensory roots and spasm of cervical muscles can cause alteration in spinovestibular input and cause vertigo with nystagmus, as can lesions of the cortical projections in the temporal lobe.

Vague imbalance or dysequilibrium is often difficult to assess as it is not associated with a fine anatomic localization; rather, it may reflect a variety of systemic, neurologic, or functional problems. These symptom descriptors must be separated from actual ataxia. Ataxia is the loss of proper motor coordination and is more evident with executed activity than at rest. It usually reflects cerebellar disease with midline lesions causing truncal instability and lateral lesions producing more difficulties with limb movement.

DIFFERENTIAL DIAGNOSIS

Dizziness in children may be caused by a variety of peripheral vestibular, central, or systemic disease processes. Many of these disease processes will be identified by the child's primary care physician and are rarely seen by the otologist or neurologist. Among those more commonly seen on otologic consultation are: diseases of the middle ear including otitis media with effusion, purulent otitis

The evaluation of dizziness in children includes obtaining as complete a history as possible from both the parent and child, complete physical examination, tests of hearing and balance as well as evaluation of possible systemic or central nervous system etiologies.

The 'dizzy child': a 12-minute consultation

Syed, M.I.,* Rutka, J.A.,† Sharma, A.* & Cushing, S.L.‡

*Royal Hospital for Sick Children, Edinburgh, UK, †University of Toronto, and ‡The Hospital for Sick Children, Toronto, ON, Canada

Accepted for publication 8 June 2014
Clin. Otolaryngol. 2014, 39, 228–234

How does it feel?

- Is there an illusion of movement or is it just light-headedness?. It is pertinent to clarify whether the symptoms the child is describing are true vertigo (illusion of movement such as rotation, or to and fro in the vertical or horizontal plane), disequilibrium, light-headedness (feeling 'fuzzy') orpre-syncope (feeling faint). In children who present without a history of 'true vertigo', common medical conditions (anaemia, diabetes mellitus) should be excluded first. Those who give a history of pre-syncope, cardiac (arrhythmias etc.) and circulatory (postural hypotension) causes should be investigated. There is significant overlap between symptoms of peripheral and central dysfunction, and in all cases, children presenting with vertiginous complaints, central neurological causes should be excluded (hereditary ataxia, posterior fossa disease, hydrocephalus).

What you should cover on examination

- the milestones outlined in the history section above
- having the child stand on one foot (30 months – briefly, 36 months – 2 s, 4 years – 5 s, 5 years – 10 s)
- head thrust testing

Original Article

Epidemiology of Dizziness and Balance Problems in Children in the United States: A Population-Based Study

Portions of the study were presented at the meeting of the Association for Research in Otolaryngology, February 23, 2014, San Diego, CA.

被引用 161 次

	Vertigo, %	Light-headedness, %	Poor coordination, %	Poor balance, %	Frequent falls, %	Other dizziness and balance problems, %
Vertigo	7.2*	12.6	8.4	14.1	3.1	2.1
Light-headedness		14.8*	7.2	12.4	3.7	3.3
Poor coordination			17.8*	16.0	15.9	3.3
Poor balance				4.4*	7.4	3.8
Frequent falls					7.2*	1.1
Other dizziness and balance problems						3.0*
Total‡	29.0	35.1	46.0	30.9	25.0	8.5

The Journal of Pediatrics Volume 171, April 2016, Pages 240-247.e3

16.1 Clinical Aspects

Epidemiology

(Brodsky et al. 2019)

- Aged 3–17 years, dizziness or imbalance was reported in 3.5 million patients (5.6%) with a mean age of 11.5 years (dizziness: 1.5 million patients; balance impairment: 2.3 million patients)
- Without any preference of sex
- In this study, the most common diagnoses for dizziness were depression or child psychiatric disorders, side effects from medication, head/neck injury or concussion, and developmental motor coordination disorders.

Epidemiology

Davitt et al. 2017

- vestibular migraine 23.8%
- benign paroxysmal vertigo of childhood 13.7%
- Idiopathic or no identifed association 11.7%
- labyrinthitis/acute vestibular syndrome (8.47%)

Batu et al. 2015

- vestibular migraine accounted for 39%
- 21% with functional psychogenic vertigo

Hülse et al. 2019

- A population-based epidemiological survey revealed a prevalence of peripheral vestibular disorders of 15.16 per 100,000 individuals.
- The most frequent were benign paroxysmal positional vertigo (10.21)
- Acute vestibular syndrome (3.5)
- Menière's disease (1.54)
- Not report on vestibular migraine, which in other studies is the most frequent cause of vertigo in children.

- Potential independent risk factors: gender, stress, muscular pain in the neck and shoulder region, sleep duration, and migraine
- A functional vestibular impairment was found in 36.5% of 2528 children with the key symptom of balance or hearing disorders

Diagnoses of Dizziness and Balance Problems

- Among the 586 children with dizziness and balance problems, only 32.8% were reported to have had a diagnosis.
- For children with moderate/big/very big problems, this percentage increased to 59.6%.

Clinical Aspects

Table 16.1 Vertigo and disturbance of vestibular function in childhood

Labyrinth/nerve	Central vestibular origin
Hereditary/congenital	
Labyrinthine malformation	Familial episodic ataxias especially EA2
Syndrome of the third mobile windows/Perilymph fistulas	Cerebellar ataxias Downbeat nystagmus syndrome
Embryopathic malformations (rubella, cytomegalovirus)	
Toxic agents	
Various hereditary audiovestibular syndromes	
Familial bilateral vestibulopathy	
Syphilitic labyrinthitis	
Positive family history of migraine	
Recurrent vertigo of childhood	
Vestibular migraine of childhood Basilar-type migraine	

Acquired	
Labyrinthitis with vestibulopathy (viral, bacterial)	Infratentorial tumors (medulloblastoma, astrocytoma, epidermoid cysts, meningioma)
Syndrome of the third mobile windows/perilymph fistulas	Epileptic aura, vestibular epilepsy
Trauma (transverse petrous bone fracture, benign paroxysmal positioning vertigo)	Trauma (brainstem or vestibulocerebellar contusion)
Menière's disease	Encephalitis
Acute unilateral vestibulopathy/labyrintitis/otitis media	Toxic agents (e.g., upbeat/downbeat nystagmus when on anticonvulsants)
Herpes zoster oticus	
Cholesteatoma	
Ototoxic drugs	
Cogan's syndrome, other inner-ear autoimmune disorders	

16.1 Clinical Synopsis

- Episodic vertigo syndromes in childhood are associated with migraine in about 50% of the cases, now called “vestibular migraine of childhood”
- Benign paroxysmal positional vertigo
- acute peripheral vestibular syndrome due to viral or bacterial inflammations of the ear
- vestibular paroxysmia
- syndrome of the third mobile windows of a semicircular canal,

16.1 Clinical Synopsis

- episodic ataxias
- vestibular epilepsy occur more rarely.
- Predisposing factors for vertigo or dizziness in childhood are recurrent bouts of otitis, traumatic brain injury, and a positive family history of migraine

16.1
Vestibular Migraine of Childhood and Recurrent Vertigo of Childhood

- Recurrent spontaneous attacks of vertigo or dizziness with different duration and different accompanying symptoms such as vomiting, pallor, fearfulness, postural imbalance, ataxia, and/or nystagmus in otherwise healthy children.

- 補: recommend using the term benign paroxysmal vertigo only for children who fulfill the diagnostic criteria but lack a personal history of migraine headache.

Paediatr Child Health. 2004 Jan; 9(1): 31–34.

Benign Paroxysmal Positional Vertigo (BPPV)

- The most frequently affected semicircularcanal is the posterior
- children with a history of a vestibular migraine or recurrent paroxysmal vertigo of childhood have signifcantly higher rates of relapses

- 補: Benign paroxysmal positional vertigo (BPPV) accounts for 5% of dizziness amongst children and adolescents. CRM was performed in 97.9% of patients, whereby 80.5% recovered following a single attempt of CRM. Recurrence of symptoms was identified in 10% of patients with no reported major complications.

Outcome of canalith repositioning manoeuvre in benign paroxysmal positional vertigo in children and adolescents: A systematic review. Clinical OtolaryngologyVolume 48, Issue 3May 2023Pagesi-iii, 371-500

Vestibular Paroxysmia

- low-dose carbamazepine administration (2–4 mg/kg daily
- Operative decompression should only be considered as a last resort ratio since vascular, brain, and bone structures grow at different speeds in children, so that compression syndromes in our experience mostly resolve spontaneously

- 補: This disabling disorder accounts for about 4% of all diagnoses in vertiginous children presenting in the setting of a tertiary referral center for vertigo and balance disorders. This frequency is similar to that in adults

Developmental Medicine & Child Neurology 2015, 57: 393–396

Syndrome of the Third Mobile Windows

- There is also a unilateral or bilateral (Kanaan et al. 2011) bony dehiscence of the superior semicircular canal syndrome in childhood.
- It differs from that in adults in manifesting primarily with auditory symptoms (autophonia, tinnitus, and hearing disorders).
- Initial experience has shown that conservative treatment should be given first.

Acute Peripheral Vestibular Syndrome

- In childhood and adolescence, traumatic head/brain injuries may cause peripheral vestibular dysfunctions in about 25%, especially post-traumatic benign paroxysmal positional vertigo or a vestibular deficit by a fracture of the petrous bone
- In most cases, the type of dizziness is described as numbness or light-headedness
- In most cases, e.g., athletes after a head trauma, the complaints were not attributable to one main type of dysfunction, even after special tests of the vestibular and ocular motor system

Bilateral Vestibulopathy

- Swaying vertigo and postural imbalance worsening in darkness and on uneven ground as well as oscillopsia with active or passive head movements are typical for bilateral vestibulopathy also in childhood
- In children, it can be caused by malformation of the labyrinth, viral or bacterial inflammation, or ototoxic antibiotics.

Functional Dizziness

- comorbidity of postural imbalance with anxiety disorders
- The fraction of functional (“somatoform”) vertigo and dizziness in childhood was reported to be 5–21%

Central Vertigo/Dizziness Syndromes

- If dizziness occurs with slowly increasing central vestibular and ocular motor dysfunctions, an MRI investigation is mandatory because of a relative frequency of brainstem and cerebellar tumors in childhood

16.1.3 Differential Diagnoses of Vertigo, Dizziness, and Balance Disorders

- Attacks of Vertigo
- Sustained Vertigo
- Stance and Gait Imbalance With and Without Oscillopsia

Attacks of Vertigo

Episodic vertigo without pathological findings in the attack-free interval:

- vestibular migraine/recurrent paroxysmal vertigo of childhood, vestibular paroxysmia, epileptic aura or vestibular epilepsy, orthostatic dysregulation, functional or psychogenic vertigo

Episodic vertigo with inner-ear hypoacusis:

- syndrome of a third mobile window/superior canal dehiscence syndrome/perilymph fistula, Menière's disease, vestibular paroxysmia

Attacks of Vertigo

Episodic vertigo with ocular motor abnormalities in the attack-free interval:

- vestibular migraine, familial episodic ataxia type 2

Episodic vertigo followed in the course of the disease by oscillopsia during head movements and imbalance that worsens in darkness

- development of bilateral vestibular failure (also with familial vestibulopathy)

- Paroxysmal, short (<1 min) attacks of spinning vertigo during changes in head position relative to gravity: BPPV (post-traumatic)

Sustained Vertigo

(Lasting Days or a Few Weeks)

- Sustained vertigo without hearing loss: acute vestibular syndrome (former vestibular neuritis) or bacterial inflammation
- Sustained vertigo with hearing loss: labyrinthitis, inner-ear autoimmune disease
- Post-traumatic sustained vertigo: transverse petrous bone fracture, labyrinthine contusion

Stance and Gait Imbalance With and Without Oscillopsia

- **Delayed stance and gait development with or without hearing loss**: congenital bilateral vestibulopathy
- **Stance and gait instability with oscillopsia during walking and head movements**: congenital or early acquired bilateral vestibulopathy, perilymph fstula, post- traumatic otolithic vertigo
- **Slowly increasing stance and gait instability as well as oscillopsia during head movements**: various hereditary and congenital disorders causing progressive audio-vestibular loss
- **Progressive ataxia, balance, and ocular motor disorders**: infratentorial tumors with lesions of vestibulocerebellar and/or pontomedullary brainstem structures, spinocerebellar ataxias with or without downbeat nystagmus

16.2 Vestibular Migraine of Childhood and Recurrent Vertigo of Childhood

Vestibular migraine of childhood

A. At least five episodes with vestibular symptoms of moderate or severe intensity, lasting between 5 min and 72 h
B. A current or past history of migraine with or without aura
C. At least half of episodes are associated with at least one of the following three migraine features:
   1. Headache with at least two of the following four characteristics:
      (a) One-sided location
      (b) Pulsating quality
      (c) Moderate or severe pain intensity
      (d) Aggravation by routine physical activity
   2. Photophobia and phonophobia
   3. Visual aura
D. Age < 18 years
E. Not better accounted for by another headache disorder, vestibular disorder, or other condition

Probable vestibular migraine of childhood

A. At least three episodes with vestibular symptoms of moderate or severe intensity, lasting between 5 min and 72 h
B. Only one of the criteria B and C for vestibular migraine of childhood
C. Age < 18 years
D. Not better accounted for by another headache disorder, vestibular disorder, or other condition

Recurrent vertigo of childhood

A. At least three episodes with vestibular symptoms of moderate or severe intensity, lasting between 1 min and 72 h
B. None of the criteria B and C for vestibular migraine of childhood
C. Age < 18 years
D. Not better accounted for by another headache disorder, vestibular disorder, or other condition

About 50% of dizzy children also complain about headache

Episodic ataxia type 2 can clinically mimic vestibular migraine in childhood/recurrent paroxysmal vertigo

Adult: Headache attacks lasting 4-72 hr (untreated or unsuccessfully treated)

Treatment

- The therapy of choice for frequent attacks of vestibular migraine/recurrent paroxysmal vertigo of childhood is the analogous migraine prophylaxis administered to adults with migraine
- **Expert experience** : that children may benefit from magnesium 300–600 mg per day in particular.
- Non-pharmacologic prophylaxis is still generally recommended in accordance with the guidelines of the American Academy of Neurology: physical exercise, sleep hygiene, avoidance of triggering food items, and behavioral therapy

16.3 Episodic Ataxias

Episodic Ataxias

- 9 forms of episodic ataxia (EA), AD ion-channel diseases.
- recurrent attacks with ataxia in combination with cerebellar and/or vestibular disorders even in the attack-free interval.
- EA2 is clearly the most frequent form, whereas EA1 is very rare.
- PQ-calcium channel gene on chromosome 19p13; this is found in only 60–70% of all clinically confirmed diagnoses

Table 2. Cardinal features of the various subtypes of EA. Clinical features of childhood-onset EA related to FGF14 mutation are emphasized in bold characters.

EA type (gene)	EA1 (KCNA1)	EA2 (CACNA1A)	EA3 (12q42, no gene)	EA4 (No gene-	EA5 (CACNB4)	EA6 (SLC1A3)	EA7 (No gene)	EA8 (UBR4)	EA9 (FGF14)
Age at onset (years)	Childhood	Childhood or adolescence	1–40	20–50	20–60	From infancy to adulthood	<20	Infancy	From **childhood** to adulthood
Attacks duration	Seconds to minutes	Hours to days	Minutes to hours	Brief	Hours	Hours to days	Hours to days	Minutes to hours	Seconds to **several days**
Triggering factors	Movement, stress, fatigue, caffeine, alcohol	Exertion, fatigue, stress	Stress, fatigue, sudden changes in head position	Changes in head position, fatigue, environmental motion		Fever, stress, heat, smoking	Excitement, exercise	Anxiety, cold, stress	**Fever**, stress, physical activity
Interictal manifestations	Myokymia	Nystagmus	Myokymia	Nystag-mus, abnormal smooth pursuit	Nystag-mus, ataxia	Nystagmus, ataxia	None	Nystag-mus, ataxia, myoky-mia, tremor	Nystagmus, upper limb postural and action tremor
Additional features/atypical manifestations	Epileptic seizures, myokimia, prolonged attacks,rigidity and cataplexy, deafness, distal weakness	Other forms of episodic neurological syndroms, permanent ataxia, developmental delay,cerebellar atrophy	Epileptic seizures	Epileptic seizures	None	Cognitive deficit, hemiplegic migraine	None	None	**Develop-mental delay, Learning disabilitiesP Paroxysmal dyskinesia**
Follow-up	Improve-ment with age	Improvement after acetozo-lamide	Poor therapeutic response	Poor therapeutic response	Poor therapeutic response	Variable response to acetazo-lamide	Poor therapeutic response	Poor therapeutic response	Variable **Improvement with age**

NK, not known.

Annals of Clinical and TranslationalNeurology2020;7(4):565–572

EA2 is important differential diagnosis for vestibular migraine

TABLE 1. *Differentiation of Episodic Ataxia Type 2 and Vestibular Migraine*

	Picture of EA 2	Picture of VM
Clinical features	• Recurrent attacks of vertigo, imbalance and ataxia (minutes to hours) • Migraine headaches (>50%) • Familial history in the majority	• Spontaneous recurrent attacks of vertigo/dizziness (minutes to hours) • Associated headache or other migrainous symptoms (60%–70%) • Individual or familial history of migraine in the majority
Examination findings during attacks	• Pathological positional nystagmus (70%) • Ataxia/postural imbalance (>90%)	• Pathological spontaneous or positional nystagmus (70%) • Postural imbalance (90%)
During attack-free interval	• Central ocular motor signs (>90%)	• Central ocular motor signs (>60%) • Peripheral vestibular deficit (10%–20%)
Genetic background	• *CACNA1A* mutations (60%)	• *CACNA1A* mutations in FHM • None found to date in VM
Treatment of choice	• Acetazolamide, 4–aminopyridine	• Beta-blockers, valproate

EA 2 and VM cannot be differentiated solely by clinical presentation. Both disorders are characterized by a combination of episodic vertigo or ataxia as well as ocular motor disturbances in the spell-free interval. Monosymptomatic presentations of VM without headaches occur in up to 30% of patients. In a subgroup of patients with FHM, there is a genetic linkage to the *CACNA1A* gene, which is the locus most often mutated in EA 2. The frequency of *CACNA1A* gene mutations in ordinary vestibular migraine is not known. Data adapted from Jen et al., 2004,[14] and Brandt and Strupp, 2006.[15]
EA 2 = episodic ataxia type 2; FHM = familial hemiplegic migraine; VM = vestibular migraine.

Neurotherapeutics, Vol. 4, No. 2, 2007

EA2

- An important differential diagnosis for vestibular migraine
- EA2 manifests in late childhood or in early adulthood. The attacks generally last for hours or up to one day
- They can occur spontaneously but typical triggers are exercise, stress, coffee, or alcohol.
- Attacks are prevented by avoiding physical exertion, emotional stress, and alcohol.
- Similar to vestibular migraine, about 90% of patients with EA2 present with central cerebellar ocular motor disorders, such as saccadic pursuit, gaze-evoked nystagmus, impaired fxation suppression of the vestibulo-ocular reflex, and especially downbeat-nystagmus

- Some of these patients develop subtle and slowly progressive limb ataxia and postural imbalance later in life.
- Mental retardation has also been reported
- Several patient have complained of generalized(myasthenic) weakness; patients rarely suffer from dystonia,which maybe a late feature of the disease
- Cerebellar atrophy, especially of the anterior vermis, can be detected on MRI

Neurotherapeutics,Vol.4,No.2,2007

Video

- https://doi.org/10.1007/000-96f)
- https://doi.org/10.1007/000-96e)

Treatment

- **Acetazolamide** and increasingly the prolonged-release form of **4-aminopyridine** are used for prophylaxis.
- A placebo-controlled cross-over study revealed a significant effect of both acetazolamide in a dose of 750 mg daily and the prolonged-release form of 4-aminopyridine (ACTZ effectively prevents or attenuates the attacks in approximately 50%to75% of all patients.)
- **The effectiveness of acetazolamide frequently decreases after 1–2 years or the treatment must be discontinued because of adverse side effects such as nephrolithiasis.**

Open trial by Strupp

- 4-aminopyridine (fampridine)
- Potassium channel blocker 4-aminopyridine (Strupp et al. 2004).

A placebo-controlled double-blind cross-over study showed that 4-aminopyridine (3 $\times$ 5 mg daily) signifcantly reduced the attack frequency and improved quality of life (Strupp et al. 2011).

- Normalized the irregular depolarization of Purkinje cells in mutant mice models
- In adults is 4-aminopyridine in its sustained release form, fampridine, 2 $\times$ 10 mg per day

Episodic ataxia, EA1

- recurrent attacks with ataxia and interictal neuro-myokymia.
- The neuro-myokymia and in part even the attacks can be successfully treated with **potassium channel blockers like phenytoin or carbamazepine**. In addition, acetazolamide can be administered in daily doses of 62.5–1000 mg to avoid attacks
- The effect of acetazolamide is probably due to an alteration of the pH value, in terms of an acidosis, which leads, for example, to reduced potassium conductivity.

補充:

- Late onset EA2 : multiple-basepair insertions in CACNA1A

16.4 Motion Sickness

- Visual-vestibular conflicts
- Children below the age of 1 year are highly resistant to motion sickness probably because they only use the visual system to a limited extent for self-motion perception and, therefore, are less subject to visual-vestibular conflicts
- 6 months to 18 years : 9.2% were susceptible to motion sickness with a slight female preponderance(Huppert et al. 2019).
- 3 months to 5 years: 1.2% were susceptible.
- The frequency of susceptibility was highest in the range between the age of 4 and 13 years, and declined in postpubertal children and adolescents.

- This course of the frequency of motion sickness susceptibility from infancy to adolescence is characterized by an inverse U-shaped curve:
- Phase 1 has high resistance in the first year of life when infants may be less subject to visual-vestibular mismatch
- phase 2 has a prepubertal peak possibly due to an over-sensitivity to a visual-vestibular mismatch
- phase 3 has a postpubertal decline best explained by habituation through repetitive motion stimulation during various kinds of vehicle transportation
- In an earlier study on children between 9 and 18 years old, no differences were found with respect to susceptibility, gender, age, type of transportation, or physical activity

16.5 Visual Height Intolerance and Acrophobia

- Visual height intolerance, including the specific phobia fear of heights (acrophobia), has a life-time prevalence in adults of 28% (females = 32%, males = 25%)
- A study on the frequency and phenomenology of visual height intolerance in prepubertal children (8–10 years) reported that the prevalence rate was higher than in adults and there was no gender preponderance as in adults
- about 50% of the afflicted children reported a spontaneous improvement or remission by the age of 8–10 years, two types of courses must be assumed: a favorable, spontaneous course in childhood and a chronic course in adulthood

16.6 Therapy of Childhood Forms of Vertigo

- The treatment of these various forms of vertigo/dizziness corresponds to that in adults
- However, a pediatrician should be closely consulted. There are no specific studies available on the therapy of most forms of vertigo in childhood. Consequently, the treatment recommendations are similar to those made for adults but with adjusted dosages.

Vertigo and dizziness in children

Klaus Jahn[a,b], *Thyra Langhagen*[a,c], *and Florian Heinen*[a,c]

被引用 96 次

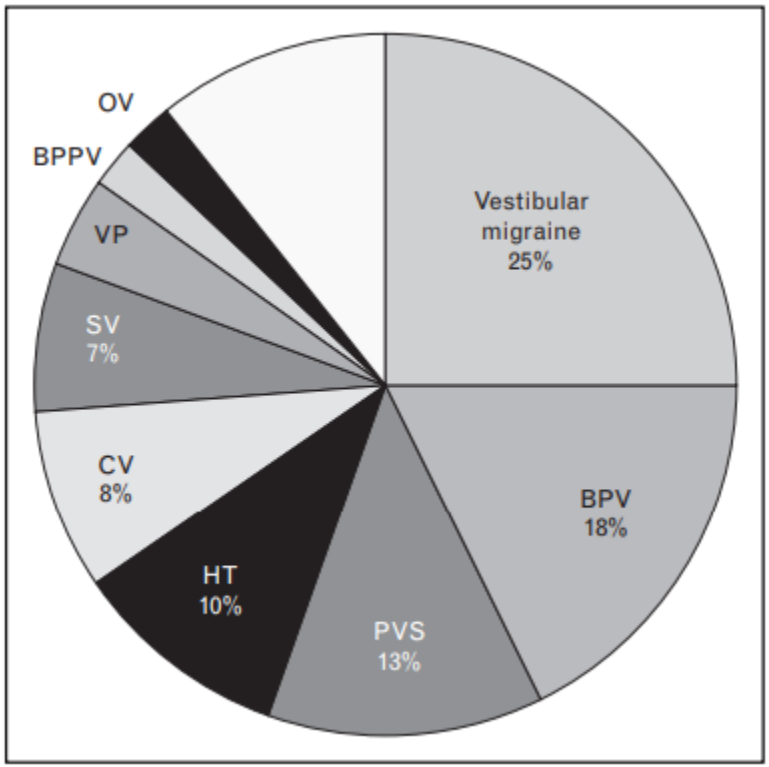

FIGURE 1. Diagnostic spectrum of the causes of dizziness in children. Data are pooled from Wiener-Vacher [7] ($n > 2000$), Lehnen *et al.* [6▪] ($n = 400$), and Gioacchini *et al.* [4▪▪] ($n = 724$). Not all diagnoses were included in all studies. If a diagnosis was only recognized in one study and did not overlap with the remaining groups (e.g., vestibular paroxysmia first described in children by Lehnen *et al.* [6▪], 4%), the group of unclear/other diagnoses (unlabeled, 11%) was reduced by an equivalent proportion to counterbalance the bias. BPPV and orthostatic vertigo each account for 2% of the diagnoses. About 40% of patients are consistently diagnosed with migraine-related syndromes (vestibular migraine and BPV). BPPV, benign paroxysmal positioning vertigo; BPV, benign paroxysmal vertigo; CV, central vertigo (includes cerebellar syndromes, central ocular motor disorders, and episodic ataxia); HT, head trauma; OV, orthostatic vertigo; PVS, peripheral vestibular syndrome (includes unilateral and bilateral vestibular loss, vestibular neuritis, labyrinthitis, Menière's disease, and vertigo in middle ear effusion/otitis media); SV, somatoform vertigo (includes phobic postural vertigo, chronic subjective dizziness, and vertigo in psychiatric disorders); VP, vestibular paroxysmia.

KEY POINTS

- Migraine-related vertigo (vestibular migraine) is the most common diagnosis in dizzy children.
- Vestibular paroxysmia is an important differential diagnosis for frequent, short spells of vertigo.
- About half of dizzy children presenting to specialized units have psychiatric comorbidity.
- Serious causes of vertigo in childhood, for example, a brain tumor, are rare.
- Vestibular testing can be reliably done with modern techniques even in small children.

Preliminary evidence shows that vHIT can be applied to children (>2 years) and is able to detect even mild semicircular canal function deficits

Table 1. Differential diagnosis of vertigo and dizziness in children

Monophasic vertigo	Episodic vertigo	Persistent vertigo
Vestibular neuritis/ labyrinthitis: rotatory vertigo, nausea, imbalance for days	Vestibular migraine: attacks lasting minutes to 72 hours, migraine headache, phonophobia/ photophobia; Benign paroxysmal vertigo: attacks lasting seconds to minutes (to hours) without migraine headache	Somatoform vertigo: subjective imbalance with normal findings on examination, aggravation in certain situations (in school, in department stores)
Head trauma: vertigo/ dizziness related to the trauma	Orthostatic vertigo: dizziness when getting up from a supine or sitting position	Bilateral vestibular loss: gait instability in darkness and on uneven ground, oscillopsia during walking
	Vestibular paroxysmia: attacks lasting seconds to minutes, with/without vestibulocochlear signs, neurovascular cross-compression on MRI, response to low-dose carbamazepine	Central vertigo: pathological ocular-motor findings such as gaze-evoked nystagmus, hypermetric saccades, skew deviation
	Benign paroxysmal positional vertigo: attacks (lasting seconds to minutes) provoked by change of head position relative to gravity; positional nystagmus	
	Perilymph fistula: attacks (seconds) provoked by coughing, sneezing, Valsalva	