Balance Disorders in Children

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Balance disorders in children may be difficult to recognize. Children often are unable to describe their symptoms and they may just seem clumsy. The episodes may be of short duration, autonomic symptoms may be prominent, or symptoms may be thought of as a behavioral disorder. Dizziness may indicate a problem in the vestibular system or may indicate a problem in other sensory systems or an abnormality of other organ systems. Management of these disorders depends on an accurate diagnosis. Disorders associated with dizziness in children can be divided into three broad categories: (1) acute nonrecurring spontaneous vertigo; (2) recurrent vertigo; and (3) nonvertiginous dizziness, disequilibrium, and ataxia [1].

Vertigo is defined in clinical practice as a subjective sensation of movement, such as spinning, turning, or whirling, of patients or the surroundings. Dizziness is a nonspecific term used by patients to describe sensations of altered orientation to the environment that may or may not include vertigo [2]. Vertigo and dizziness are symptoms, not diagnoses. Balance is maintained through visual, proprioceptive, and vestibular signals. Damage to any of these systems or an abnormality in the central nervous system (CNS) that coordinates impulses from these three sensory systems can cause symptoms. In children and in adults, a careful history, physical
examination, and laboratory testing can establish the cause of dizziness in most patients.

**Physiologic basis of balance**

When a hair cell is stimulated by rotation, translation, or change in orientation due to gravity, the firing rate in the VIIIth nerve fiber innervating that particular hair cell either increases or decreases. Movements that cause the stereocilia to bend toward the kinocilium result in a depolarization of the hair cell and cause the eighth nerve fiber to increase its firing rate, whereas movements that bend the stereocilia away from the kinocilium decrease the neural firing in the eighth nerve. The eighth nerve synapses in the vestibular nuclei, which consist of superior, medial, lateral, and inferior divisions. In addition to the input from the labyrinth, the vestibular nuclei receive input from other sensory systems, such as vision, somatic sensation, and audition. The sensory information is integrated and the output from the vestibular nuclei influences eye movements, truncal stability, and spatial orientation.

The vestibulo-ocular reflex (VOR) is a mechanism by which a head movement automatically results in an eye movement that is equal and opposite to the head movement so that the visual axis of the eye stays on target: a leftward head movement is associated with a rightward eye movement and vice versa. The VOR is mediated by a three-neuron arc that includes, for the horizontal system, the eighth cranial nerve, an interneuron from the vestibular nucleus to the abducens nucleus, and the motor neuron to the eye muscle. Even when the head is at rest, there are action potentials creating a resting discharge in each neuron in the vestibular portion of the eighth nerve. This resting discharge is unique in that it allows the neurons to sense motion in the excitatory and the inhibitory directions by increasing and decreasing their firing rates, respectively.

Another feature of the VOR is that the two vestibular nuclear complexes on each side of the brain stem cooperate with one another in such a way that, for the horizontal system, when one nucleus is excited, the other is inhibited. This reciprocal push-pull effect increases the sensitivity of the VOR (Fig. 1). The CNS responds to differences in neural activity between the two vestibular complexes. When there is no head movement, the neural activity (ie, the resting discharge) is symmetric in the two vestibular nuclei. The brain detects no differences in neural activity and concludes that the head is not moving. For example, when the head moves to the left, endolymph flow produces an excitatory response in the labyrinth on the side toward which the head moves (to the left), and an inhibitory response on the opposite side (on the right). Thus, neural activity in the vestibular nerve and nuclei (on the left and right) increases and decreases, respectively. The brain interprets this difference in neural activity between the two vestibular complexes as a head movement and generates appropriate vestibulo-ocular and postural responses. This
Fig. 1. Schematic illustrations of the push-pull effect of the VOR: (A) no head movement in healthy subject; (B) head movement to the left in healthy subject; (C) right acute peripheral vestibular injury. (From Furman JM, Cass SP. Evaluation of dizzy patients. Slide lecture series. Alexandria, VA: American Academy of Otolaryngology-Head and Neck Surgery; 1994; with permission.)
reciprocal push-pull balance between the two labyrinths is disrupted after labyrinthine injury.

An acute loss of peripheral vestibular function unilaterally causes a loss of resting neural discharge activity in that vestibular nerve and the ipsilateral nucleus. Because the brain responds to differences between the two labyrinths, loss of resting neural discharge is interpreted by the brain as a rapid continuous head movement toward the healthy labyrinth, as the brain responds to differences between the two labyrinths. Corrective eye movements are produced toward the opposite side, resulting in nystagmus, with the slow component moving toward the abnormal side, and with the quick components of nystagmus moving toward the healthy labyrinth. Through compensatory mechanisms, the CNS restores the resting discharge activity within the deafferented vestibular nucleus, which reduces the asymmetry of neural activity within the bilateral vestibular nuclei, partially restoring a functional VOR. Thus, during head movements with only one functional labyrinth, although neural activity within only one vestibular nerve is modulated up and down, this activity causes increases and decreases in vestibular nuclei activity. A unilateral loss of vestibular function thus results in a reduction of sensitivity to vestibular stimuli (ie, a reduced gain of the VOR) and an asymmetric response to high intensity stimulation, such as a rapid head movement.

**Disorders of balance in children**

*Acute nonrecurring spontaneous vertigo*

Acute nonrecurring spontaneous vertigo is unusual in children (Table 1). In an acute vestibular syndrome, the vertigo experienced is a result of the rapid loss of unilateral peripheral vestibular function, which disrupts the push-pull interaction of the two labyrinths. Patients experience vertigo and exhibit nystagmus, with the fast component beating toward the healthy ear. Additionally, children may experience autonomic symptoms, including nausea and vomiting. A process called vestibular compensation begins immediately and the CNS learns to use the signal from one labyrinth as a sole source of vestibular input. This process of compensation depends on several factors, including a normal CNS, especially brainstem and cerebellar function; a significant amount of active eye, head, and body movements; and abstinence from vestibular suppressant medications [3]. Typically, children recover from an acute loss of unilateral peripheral vestibular function within days; some children may recover so quickly that it scarcely is known that they had an acute vestibular episode.

*Head trauma*

It is estimated that more than 400,000 children under the age of 15 present to United States emergency departments with head trauma each year [4].
Head trauma, including mild traumatic brain injury, may result in abnormalities on balance testing [5]. Head trauma can cause an acute episode of vertigo by abruptly affecting the vestibular end organ directly as in a labyrinthine concussion. Although several theories are proposed, the mechanism of injury in labyrinthine concussion is poorly understood. Pressure waves transmitted directly to the labyrinth through the skull or intracranially via the cochlear aqueduct may cause rupture of the membranous labyrinth or damage to hair cells, hair bundles, or specialized structures in the ampulla or macula. In addition to dizziness, other symptoms indicating otologic trauma include a sudden hearing loss (conductive or sensorineural), tinnitus, or clear or bloody drainage from the ear. Children usually recover completely within a short period of time, but, infrequently, benign paroxysmal

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<td>Labyrinthine concussion</td>
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<td>Meniere’s disease</td>
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<td>Bilateral vestibular loss</td>
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positional vertigo or delayed endolymphatic hydrops may develop. Benign paroxysmal positional vertigo is characterized by nystagmus and associated vertigo elicited by rapid changes in head position from upright to head hanging. It is believed to be caused by canalithiasis (free-floating particles in the semicircular canals) and can be treated with the canalith repositioning maneuver (Epley maneuver) [6].

Other mechanisms of vertigo after head trauma include injury of the CNS, specifically a brainstem or cerebellar contusion, or a temporal bone fracture. A temporal bone fracture may be longitudinal (70% to 80%) or transverse (20% to 30%), with the latter the most common cause of vertigo caused by injury to the eighth nerve or the otic capsule. This type of fracture also causes a significant sensorineural hearing loss.

**Perilymphatic fistula**

Perilymphatic fistula is an anomalous connection between the inner ear and middle ear spaces and has been well documented in children [7]. Although it usually is associated with hearing loss, perilymphatic fistula can be associated with vertigo alone [8]. A perilymphatic fistula can be congenital or acquired. The congenital fistulas are associated with abnormalities in the temporal bone, particularly in the area of the stapes, but also in the round window area. Acquired perilymphatic fistulas generally are caused by trauma, including surgical procedures, barotrauma, penetrating trauma, or head trauma, which may or may not be associated with a temporal bone fracture. Barotrauma can be either implosive or explosive [9]: implosive barotrauma can occur during diving, flying, or violent sneezing or coughing and is the result of a sudden pressure change in the middle ear, whereas explosive barotrauma is caused by a sudden increase in the spinal fluid pressure and, in susceptible individuals, may be induced by straining or any type of excessive exertion, such as heavy lifting or sit-ups.

Although there are no symptoms pathognomonic for a perilymphatic fistula, several features of the history are suggestive: (1) a history of hearing loss or vertigo after physical strain or stress, exposure to sudden alterations in environmental pressure (eg, diving or flying), or marked alteration in middle ear pressures (eg, violent sneezing, laughing, or blowing a wind instrument); (2) a sensorineural hearing loss that is sudden, fluctuating, or both; (3) dizziness, which may be increased by postural change, or continuous poor balance, or ataxia; and (4) a sensation of a pop in the ear followed by hearing loss or dizziness.

No test is specific for perilymphatic fistula, and the diagnosis may be difficult. A fistula test, the application of positive and negative pressures to the external ear canal, may produce eye movements or nystagmus, which may indicate the presence of a fistula. A negative response, however, does not exclude a perilymphatic fistula. Fluctuating hearing loss also may be indicative of a perilymphatic fistula. An inner ear or middle ear anomaly, such as a Mondini malformation or ossicular deformities, demonstrated on
a high-resolution CT scan, should heighten the suspicion of a congenital perilymphatic fistula [10]. At the time of surgical exploration, fluid from the middle ear should be collected and sent for β2-transferrin testing, the presence of which is considered supportive of the diagnosis [11].

**Vestibular neuritis**

Vestibular neuritis rarely is seen in children younger than 10. It should be considered when a viral syndrome is followed by symptoms suggestive of an acute unilateral peripheral vestibular loss [12]. It presents with acute severe vertigo, nystagmus, nausea, and vomiting. The vertigo is worsened by head movements, and patients often prefer to lie down, usually with the affected ear up. There is no hearing loss or tinnitus. The symptoms resolve in children within a few days. Management is supportive and symptomatic with early ambulation.

**Labyrinthitis**

Acute labyrinthitis indicates an inflammatory condition that affects the labyrinth and generally leads to vestibular and auditory symptoms and signs. The cause of serous (toxic) labyrinthitis is unknown but bacterial toxins or other biochemical substances in middle ear fluid are believed absorbed into the inner ear, usually through the round and oval windows. Symptoms may be mild with little or no sensorineural hearing loss and resolve spontaneously. In bacterial or suppurative labyrinthitis, there is an invasion of a bacterial infection into the labyrinth from the middle ear through preformed pathways that may be caused by chronic otitis media with cholesteatoma, a prior temporal bone fracture, or a congenital bony abnormality. Alternatively, in patients who have bacterial meningitis, there may be invasion of bacteria via the internal auditory canal or the cochlear aqueduct. The symptoms of suppurative labyrinthitis are severe, and the condition often results in loss of vestibular and auditory function on the affected side. Bacterial or suppurative labyrinthitis is a serious complication that requires immediate intravenous antimicrobial therapy and surgical intervention. Labyrinthitis also can be viral and is associated with measles, mumps, and rubella.

**Recurrent vertigo**

Recurrent vertigo in children can be a result of disease of the peripheral or central vestibular system. Most recurrent vertigo in children is a result of a CNS disorder rather than a peripheral vestibular disorder.

**Meniere’s disease**

Meniere’s disease, a syndrome presumably caused by endolymphatic hydrops, can occur spontaneously or as a delayed sequela of previous insult
from trauma or viral infection. The disorder occurs rarely in children \[13–15\]. The disease is characterized by a complex of symptoms, including dizziness, unilateral fluctuating hearing loss, and unilateral tinnitus, which usually are preceded by a feeling of fullness in the affected ear. Symptoms vary among patients and may vary in the same patient over time. Some patients may have only hearing loss and tinnitus, whereas others may have only vestibular symptoms. The duration of the vertiginous episode may vary from a half hour to several hours and episodes frequently are accompanied by autonomic symptoms, such as pallor, perspiration, nausea, and vomiting. Between these acute episodes, adults and, rarely, children may have vague symptoms of disequilibrium. The hearing loss in Meniere’s disease usually is a low-frequency sensorineural loss that fluctuates (ie, returns to normal between attacks) during the early stages of the disease. Later, the hearing loss may progress to a flat sensorineural hearing loss that does not fluctuate. Children are more likely to recover auditory function than are adults. Meniere’s disease can be bilateral. Also, with time, a reduction in the responsiveness of the involved peripheral vestibular system occurs.

**Migraine**

Migraine probably is the most common cause of recurrent vertigo in children. Whereas migraine typically presents as headache in adults, other manifestations of migraine, including recurrent vertigo and disequilibrium, are more common in children. Benign paroxysmal vertigo of childhood, which is likely to be of migrainous origin, and paroxysmal torticollis of infancy and basilar artery migraine can present with recurrent vertigo in children. Nonvertiginous symptoms of vestibular dysfunction also can be related to migraine. Thus, the manifestations of migraine in childhood are varied and include episodic true vertigo, constant imbalance, movement-associated true vertigo, and space and motion discomfort. Because of this highly varying presentation of symptoms, the diagnosis of migraine-related vestibulopathy requires an awareness of the potential vestibular manifestations of migraine, a meticulous history with specific inquiry into the occurrence of headache and other migraine-associated symptoms, and a careful family history. Ultimately, migraine-related dizziness remains a diagnosis of exclusion. Benign paroxysmal vertigo \[16\] of childhood is a disorder that occurs mostly in children 3 to 8 years of age. Vertigo occurs in isolation, without cochlear symptoms, such as tinnitus and hearing loss. Episodes are brief, usually less than 1 minute, but may range from seconds to a few minutes. During a severe attack, children usually remain still and unable to move, and children frequently become limp. During a less severe attack, children may clutch something. There are no known precipitating factors and the attacks can occur while sitting, standing, or lying. Pallor, nausea, sweating, and occasionally vomiting occur. Consciousness is not impaired and children can recall the episode. There is no pain or headache associated with the attacks, and immediately after, children resume normal
activities. The interval between attacks varies from weekly to every 6 months, with monthly to bimonthly episodes most common. The attacks usually cease spontaneously after a few years. Physical examination, including a neurologic evaluation, is normal, as is imaging of the skull and temporal bones. Often, there is a positive family history of migraine, and migraine headaches may develop in later years [17,18].

Paroxysmal torticollis of infancy consists of episodes of head tilt, which may be associated with nausea, vomiting, pallor, and agitation. The torticollis may alternate from side to side. Episodes are brief and self-limiting and may recur for several months but usually resolve by age 2 to 3. Physical examination is normal aside from the torticollis, and nystagmus has not been reported. Paroxysmal torticollis of infancy and benign paroxysmal vertigo of childhood have been reported to occur in the same patient [19,20].

Bickerstaff [21] was the first to associate vertiginous symptoms preceding migraine with dysfunction of the brainstem and areas within the distribution of the basilar artery. The majority of his patients were adolescent girls in whom the symptoms often occurred premenstrually, but symptoms also are described in preadolescent children [22,23] and adults [24,25]. Basilar artery migraine typically begins with visual symptoms, consisting of either a total loss of vision or visual aberrations through both visual fields. Other symptoms, such as vertigo, ataxia of gait, dysarthria, and motor weakness (often hemiparetic), usually follow. The headache usually is occipital rather than hemicranial and lasts between 5 minutes and 1 hour. There also may be hearing loss, tinnitus, restlessness, or impairment of consciousness, which is occasionally complete.

**Seizure disorders**

Although not frequently associated with true vertigo, seizure disorders often may be accompanied by some sense of dizziness and disequilibrium. Tornado epilepsy, however, is used to describe seizures that are associated with a sense of spinning that can mimic the symptoms of a peripheral vestibular problem [26]. Treatment for seizures accompanied by dizziness is similar to treatment for other seizures that have an aura.

**Familial periodic ataxia**

Familial periodic ataxia is a rare syndrome with autosomal dominant inheritance and is characterized by episodes of dizziness, disequilibrium, and gait instability that may last for several hours. At least two types of the syndrome have been identified [27,28] and genetic testing is available. These syndromes differ in the duration of the episodes of ataxia. Auditory symptoms usually are absent in these patients. Often, these patients experience migraine headaches. In adults, such patients may have a cerebellar syndrome comparable to that seen in patients who have cerebellar degeneration.
Nonvertiginous dysequilibrium

Patients who have peripheral and central vestibular disorders can have nonvertiginous disequilibrium, imbalance, and ataxia. Peripheral vestibular disorders that occur typically without vertigo and, therefore, may mimic a central disorder include bilateral peripheral vestibular loss and otitis media. Many CNS abnormalities can be associated with nonvertiginous dizziness. Many of these abnormalities involve the cerebellum and include cerebellar hypoplasia, posterior fossa tumors, and Chiari malformations. Also, medication side effects should not be overlooked when evaluating children who have dizziness and disequilibrium.

Bilateral peripheral vestibular loss

Bilateral peripheral vestibular loss is a rare disorder in children and can be either congenital or acquired. The most common causes of acquired bilateral vestibular loss include meningitis, exposure to ototoxic medications (such as aminoglycoside antibiotics), and autoimmune disease of the inner ear. Congenital bilateral vestibular loss often is the result of an inner ear malformation. Some of these malformations affect only hearing, some affect only balance, and some affect hearing and balance function. The most common inner ear malformations include Mondini’s dysplasia (incomplete partition of the cochlea), enlarged vestibular aqueduct syndrome, and Scheibe’s dysplasia (cochleosaccular dysplasia). Regardless of cause, bilateral vestibular loss, if severe, causes Dandy syndrome, characterized by two specific symptoms, oscillopsia (jumbling of the visual surround during head motion) and severe gait instability in darkness [29]. Many children who have bilateral vestibular loss have delay in gross motor functions and balance but not fine motor functions. By using alternative sensory inputs, such as vision and proprioception, they catch up later in childhood [30]; they also modify strategies of eye movements. Motions in environments that require vestibular function, such as ambulating in dimly lit spaces or trying to maintain stable vision during walking, are extremely challenging for individuals who have bilateral vestibular loss.

Otitis media

Eustachian tube dysfunction with and without middle ear effusion is considered the most common cause of vestibular disturbances in children [31–36]. Often, parents report that children have started to walk or are less clumsy after tympanostomy tube insertion. Only recently, however, have studies been performed in children that confirm the anecdotal evidence that vestibular balance and motor function may deteriorate during an episode of middle ear effusion [37–44]. Also, children who have otitis media may be more visually dependent [45], as a result of the deterioration of vestibular function causing excessive reliance on other nonvestibular sensory cues to maintain balance. Placement of tympanostomy tubes in children who have
otitis media is shown to improve balance [38–42,44]. In addition to postural control abnormalities, some studies indicate that children who have otitis media can have spontaneous and positional nystagmus [40], which resolves after tympanostomy tube insertion. The pathophysiologic basis for the balance disturbance seen in children who have otitis media is unknown and further studies are required to determine the cause.

**Motion sickness**

Motion sickness refers to pallor, diaphoresis, dizziness, nausea, and vomiting induced by passive motion, such as riding in a car, or by visual motion while standing still. The cause of motion sickness is believed to be a sensory mismatch between vision and vestibular cues [46]. Motion sickness is more common in patients who have migraine. In a study by Bille [47], severe motion sickness was present in 49% of children who had migraine compared with only 10% of control children; a later study by Barabas et al [48] shows similar results, with motion sickness occurring in 45% of children who had migraine compared with 5% to 7% of control children.

**Central nervous system disorders**

Many CNS disorders cause dizziness, disequilibrium, imbalance, and ataxia. In childhood, cerebellar abnormalities, such as cerebellar vermian hypoplasia, posterior fossa tumors, and Chiari malformation, are the most common disorders encountered. The clinical presentation of such patients may be confusing, because they are unlikely to have vertigo and may not display evidence of limb ataxia if their abnormalities affect solely midline cerebellar structures. Multiple sclerosis, although rare in children less than 10 years of age, frequently presents with ataxia.

Chiari malformation consists of four (I to IV) congenital anomalies of the cerebellum and brainstem. Despite their common anatomic location, the four anomalies have distinct features. In type I, there is caudal displacement of the cerebellar tonsils into the upper cervical spinal canal, but there usually is no hydrocephalus. Type I previously was diagnosed in older children or young adults based on late onset of symptoms, but since the advent of MRI, the diagnosis is made at an earlier age. The caudal midline cerebellum typically is affected, resulting in gait instability and vestibulo-ocular abnormalities in addition to other neurologic signs and symptoms, such as recurrent headache, neck pain, urinary frequency, and progressive lower extremity spasticity [49]. Type II is the more common lesion and manifests usually in the first few months of life. In type II malformation, the structures herniating through the foramen magnum include the cerebellar vermis, brainstem, and fourth ventricle. Type II malformation often is associated with progressive hydrocephalus, myelomeningocele, and multiple brain anomalies. Approximately 10% present in infancy with stridor, weak cry, and apnea; others may present with gait abnormalities, spasticity, and increasing incoordination during childhood [49]. Types III and IV are rare.
The most common posterior fossa tumors in children are astrocytoma, medulloblastoma, ependymoma, and glioma. The clinical manifestations largely are those of increased intracranial pressure, except for the gliomas, because the majority of the tumors are in the midline structures where a mass lesion causes obstruction of cerebrospinal fluid circulation. Tumors also may present with signs of unilateral cerebellar dysfunction, with symptoms such as hypotonia, intention tremor on the side of the lesion, nystagmus, and gait ataxia.

Acoustic neuroma is a benign schwannoma arising from the eighth nerve and presents usually with unilateral sensorineural hearing loss and tinnitus. When large, these tumors can cause ataxia and dizziness. This tumor usually does not occur in children except as part of neurofibromatosis type II. Neurofibromatosis is inherited as an autosomal dominant trait. Type I (von Recklinghausen’s disease) is common, with the neurinomas occurring throughout the body, whereas neurofibromatosis type II, the central form with bilateral acoustic neurinoma occurring in 96% of patients, is less common [50].

**Drug-induced dizziness**

Many drugs can cause dizziness in addition to the ototoxicity caused by aminoglycosides, especially gentamicin, which may cause oscillopsia. In the pediatric age group, phenytoin is used in the treatment of epilepsy but may produce dizziness and nystagmus as signs of intoxication. Other drugs that may cause vestibular symptoms include chemotherapeutic drugs, loop diuretics, quinine, thalidomide, and nonsteroidal anti-inflammatory drugs. Dizziness developing in children who are taking a medication should be viewed as possible cases of iatrogenic dizziness.

**Non-neurotologic disorders**

Psychiatric dizziness may be associated with depression, adjustment reaction of adolescence, and behavior problems. Such children usually are school age and have normal vestibular and auditory testing, electroencephalogram, and imaging studies. When evaluating children who have dizziness, it is essential to determine if patients have an associated anxiety disorder either as the sole cause for their vertiginous complaints, as an accompaniment to an underlying balance system abnormality, or indirectly related to the dizziness (eg, through a common brainstem ailment causing disequilibrium and an anxiety disorder).

Systemic disorders tend to cause vague lightheadedness and disequilibrium rather than vertigo or ataxia. Anemic states and hypoperfusion states, such as vasovagal instability, typically produce lightheadedness but not syncope. Congenital heart diseases and arrhythmia also should be considered a cause of dizziness. Autonomic nervous system dysfunction may produce orthostatic hypotension. Thyroid disease, hypoglycemia, and
Addison’s disease are other disorders that may produce lightheadedness. A recent study of children and young adults who had type I diabetes found evidence of vestibular involvement even in patients who did not have vestibular symptoms [51].

Ocular disorders have also been found responsible for vertigo or dizziness in children. Anoh-Tanon et al [52] reported that 7 of 523 (1.3%) of pediatric patients in their series who were referred for vestibular testing had normal neurologic and vestibular examinations but were found to have vergence or refractive problems. Correction of these problems resulted in alleviation of the symptoms.

Hearing loss

Several clinical studies address the issue of balance and vestibular function in children who have hearing loss. Although balance function often is impaired in children who have otitis media, which is associated with a conductive hearing loss, most studies of balance and vestibular function in children who have hearing loss concern children whose hearing loss is sensorineural. These studies typically report children whose hearing loss could be attributed to a specific causal factor. Taken together, these studies suggest that vestibular function is impaired in children who have sensorineural loss. Horak et al [53] studied vestibular function in children who had unilateral or bilateral mild-to-moderate sensorineural hearing loss that could not be attributed to a specific cause. The study suggests that many children who have sensorineural hearing loss of uncertain origin have an associated vestibular system abnormality, based on laboratory testing, whether or not the children complain of dizziness. It is important to assess vestibular function in infants and young children who have sensorineural hearing loss and delayed motor development because of the possibility of underlying vestibular problems as the cause for the delay.

Vestibular evaluation

History

It is important that children explain the symptoms using their own vocabulary and describe associated sensations, such as headache, nausea, vomiting, or motion sickness. It may be helpful to relate patients’ symptoms to experiences, such as being on a merry-go-round. The onset, duration, and frequency of dizziness episodes should be established and an attempt should be made to associate the episodes with certain activities (see Table 2).

Clinicians should inquire about the presence of hearing loss (sensorineural or conductive); its onset, evolution, progression, fluctuation, and worsening; and improving or stable status. It should be established whether patients have tinnitus or a feeling of fullness, if the hearing loss is
bilateral or unilateral, if there is a history of otitis media, and if otorrhea or otalgia is present. Previous audiograms, if available, should be reviewed.

To establish the presence of neurologic symptoms, clinicians should determine whether there have been convulsions, altered mental status, weakness, numbness, disturbances of swallowing or taste, coughing, facial paralysis, or blurring and loss of vision.

In establishing past medical history, clinicians should acquire information regarding pregnancy and delivery (ie, history of birth trauma, anoxia at delivery, presence of infectious diseases [measles, mumps, or syphilis] and presence of CNS infections [including meningitis]). It should be established whether any ototoxic medications were administered, especially in the neonatal period, and whether patients have had diabetes, hypothyroidism, or other endocrine disease; renal disease; eye disorders; epilepsy; noise exposure; head trauma; or relevant previous surgeries. Clinicians should examine family history, particularly in regard to migraine, epilepsy, hearing loss or deafness, endocrine or renal disease, and neurofibromatosis.

### Physical examination

A general physical examination should be done, including obtaining blood pressure (sitting, standing, and lying down). Examination of the ears should be performed using pneumatic otoscopy or an operating microscope. Perilymph fistula testing is performed by pressing the tragus and creating a positive pressure in the external auditory canal or by applying positive and negative pressure in the external ear canal using a Politzer’s bag or tympanometer while observing the patient’s eyes behind Frenzel glasses. Frenzel glasses are +20 lenses that have internal illumination shining into the eyes. The glasses reduce visual fixation and magnify the size of the eyes, aiding in observation of the subjects’ eye movements (Fig. 2). The fistula test response is positive if nystagmus and a sensation of dizziness occur.

### Table 2

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<th>History</th>
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Because vertigo in childhood can be a symptom of a neurologic abnormality, a complete neurologic evaluation with special attention to examination of the cranial nerves should be obtained. The optic nerve is tested by standard acuity, and both eyes are tested separately. Visual field defects should be ascertained and the fundi should be visualized. The presence or absence of nystagmus should be noted using Frenzel glasses. If present, nystagmus should be characterized as spontaneous or gaze evoked and whether or not it is positional. Children also should be observed when walking or running for incoordination of movements (ie, ataxia). Dysmetria may be demonstrated by the finger-to-nose and heel-to-shin tests. Additional abnormalities associated with cerebellar lesions can be assessed by evaluating patients for dysdiadochokinesia, hypotonia, and decreased tendon reflexes.

**Motor function testing**

Because children’s motor performance changes with age, the tools used to evaluate motor performance change. Among the more commonly used validated measures are the Peabody Developmental Motor Scales (PDMS) [54], the Bruininks-Oseretsky Test of Motor Proficiency (BOTMP) [55], and the Pediatric Clinical Test of Sensory Interaction for Balance (P-CTSIB) [56].

The PDMS was developed to identify gross and fine motor skills that are delayed or abnormal and can assess developmental change over time. One component, the gross motor scale, is used to assess motor performance. The gross motor scale includes assessment of reflexes, balance, nonlocomotor skills, locomotion, and the ability to grasp and move objects in the test environment. Developed for use in children from birth to age 7, it provides much information but is time and energy intensive and requires a large,
quiet space and equipment that typically is not found in otolaryngologists’ or neurologists’ offices.

The BOTMP was developed for use in children from 4.5 to 14.5 years of age. It includes gross motor and fine motor components and has more difficult balance items than the PDMS. It generally is used after children are able to complete the PDMS gross motor items.

The P-CTSIB is an inexpensive way to replicate dynamic posturography in a clinical setting and is shown to have good to fair reliability in children. Children stand on medium-density foam or with a visual conflict dome for 30 seconds (Fig. 3). Body sway under six sensory conditions is determined.

**Audiologic evaluation**

Age-appropriate behavioral testing is performed to determine if there is a concomitant hearing loss and to help define the side of the lesion. If possible, the audiologic evaluation should include, in addition to pure tone thresholds, speech reception threshold and word recognition scores, acoustic reflex, and tone decay. Masking is mandatory when there is a difference of hearing sensitivity between ears. Tympanometry is performed to assess middle ear status, particularly in regard to middle ear pressure, presence of effusion, and if the tympanic membrane is intact.

In cases of unilateral hearing loss and asymmetric hearing loss, the auditory brainstem response is useful in the diagnostic evaluation process to

![Fig. 3. A child standing on medium-density foam with her eyes open (condition 4) (A) and standing on the foam with the visual conflict dome (condition 5) (B).](image)
determine the site of the lesion. The procedure is noninvasive and excellent for testing small children and children who cannot cooperate with behavioral testing. Small children may require sedation for auditory brainstem response testing. During this testing, clicks are delivered through earphones and monitored by signal averaging while patients are relaxed or asleep. The waveform and latency are studied, and the waves are compared in both ears and with those of normal subjects. The latencies are the most sensitive indicator of disease. Another objective method to assess cochlear function in children is otoacoustic emissions. Transducers in the ear canal are used to pick up cochlear emissions in response to clicks, tone bursts, or spontaneous emissions. Fluid in the middle ear can abolish otoacoustic emissions.

**Vestibular laboratory evaluation**

Vestibular laboratory testing may be helpful in distinguishing a peripheral vestibular abnormality from a central one. Also, vestibular laboratory testing may identify the side of the lesion in a peripheral vestibular abnormality. In addition, it provides permanent documentation and changes can be followed up by repeat testing.

Vestibular laboratory testing includes vestibulo-ocular and vestibulospinal tests. Both types of tests provide only an indirect measure of the function of the vestibular end organs, in that they rely on measures of motor response (ie, eye movements or postural sway) resulting from vestibular sensory input.

**Electronystagmography**

Electronystagmography is the laboratory tool most commonly used to study children who have complaints of dizziness, vertigo, or imbalance. Eye movements are recorded with electro-oculography or infrared video goggles. Electronystagmography includes ocular motor testing, positional testing, and caloric testing and provides a permanent record of the spontaneous nystagmus or induced nystagmus with objective measurement of the response.

Spontaneous nystagmus that is present in darkness without fixation and decreases or resolves with fixation suggests a peripheral vestibular disorder. Spontaneous nystagmus that is present with fixation and does not significantly decrease with loss of fixation, however, most likely is a CNS abnormality. Sinusoidal pursuit tracking (following a moving target back and forth along a slow pendular path) may be abnormal with CNS lesions. Asymmetry or absence of optokinetic nystagmus (elicited by moving a visual pattern, usually vertical stripes, across the visual field) may suggest a CNS abnormality. Positional testing, in which the patients are moved into various positions, may be useful in elucidating various problems. Visual fixation normally suppresses static positional nystagmus; failure to suppress may suggest a CNS lesion. Paroxysmal positional nystagmus is associated with
vertigo, has a brief latency of 5 to 10 seconds, fatigues on repeat provocation, and is suggestive of a peripheral lesion. Caloric testing produces nystagmus by thermal stimulation of each horizontal canal. Cold irrigation results in the fast component away from the stimulated ear, whereas warm irrigation produces the fast component toward the stimulated ear. In bilateral vestibular loss, caloric responses are reduced or absent in both ears. Caloric responses, however, can be reduced or even absent with normal rotational responses in the same patient; this is explained by the fact that caloric stimulation is nonphysiologic, whereas rotational stimulation is the natural stimulus to the labyrinth. Caloric stimulus intensity can vary as a result of alterations in blood flow, middle ear fluid, and thickness of the temporal bone. The most commonly used parameter of the caloric response is the peak slow-component velocity, whose magnitude reflects the intensity of the vestibular response. A reduced vestibular response as computed by Jongkee’s formula (a standard formula often incorporated into the software in vestibular laboratories) of more than 24% is considered abnormal. Rotational testing using a rotary chair stimulates both labyrinths at the same time (Fig. 4). Three parameters are obtained on rotational testing: gain (ratio of eye velocity to head velocity), phase shift (offset in the timing of eye

Fig. 4. A child sitting in the rotational chair.
movement relative to head motion), and symmetry. A gain of 1.0 and a phase shift of 180° indicate perfect VOR function, in that the eyes move synchronously with head movement but in the opposite direction. Reduced gain indicates decreased vestibular sensitivity and usually indicates bilateral vestibular loss. Phase changes with peripheral vestibular injury and is a nonspecific measure of vestibular system abnormalities. Asymmetry of response also is a nonspecific sign. Although changes in these measures do not indicate the site or the side of the lesion, rotational testing measures change in response to vestibular disease and can be used to monitor patients’ progress.

**Dynamic platform posturography**

Platform posturography also uses a physiologic stimulus and can be used to assess patients’ reliance on sensory inputs and the motor responses generated to maintain proper equilibrium when the floor is moved (Fig. 5). Computerized dynamic posturography is marketed commercially under the trade name EquiTest (NeuroCom International, Clackamas, Oregon). The platform and the background move while the anteroposterior sway of

Fig. 5. The EquiTest system with the child standing on the platform surrounded by a visual scene. A safety harness is attached to the child to prevent falls in case of loss of balance. The platform surface and visual surround are capable of moving independently or simultaneously. Pressure-sensing strain gauges beneath the platform surface detect the patient’s sway by measuring vertical and horizontal forces applied to the surface.
patients standing on the platform is monitored. The testing software allows two broad categories of tests: (1) recording of responses to small, brief movements of the support surface, either translations or rotations (motor control tests) and (2) recording of postural sway during various combinations of sensory inputs (sensory organization tests). The sensory organization test is the most useful part of the test in the assessment of patients who have suspected vestibular disorders. There are six conditions under which patients are tested (Fig. 6). During conditions I to III, the support is fixed while the visual input varies: eyes open, eyes closed, and sway referenced. During conditions IV to VI, the support is sway referenced (ie, the support is moving and the visual conditions are repeated). Conditions V and VI are the most difficult, as patients have to rely on the vestibular input alone. By providing reduced or distorted sensory information from the visual system and somatosensory system, the sensory organization test forces children to rely on their vestibular sensations to maintain upright balance.

**Imaging**

Imaging studies include CT with or without contrast enhancement for the evaluation of bony structures of the temporal bone and middle ear. CT
scans are performed to rule out any congenital malformations or bony abnormalities caused by infectious processes or a cholesteatoma eroding the bone or a temporal bone fracture. MRI with gadolinium injection is the most important test for ruling out a CNS lesion, cerebellopontine angle mass, posterior fossa disease, and craniovertebral abnormalities.

Additional laboratory testing

Additional testing is indicated when a nonvestibular condition, such as metabolic abnormalities or blood dyscrasia, is suspected of causing dizziness. Such testing may include a complete blood count; serum glucose, thyroid function, triglyceride, and cholesterol determinations; fluorescent treponemal antibody absorption test; erythrocyte sedimentation rate; and rheumatoid factor, antinuclear antibody, and autoimmune studies when appropriate.

Summary

There are many causes of balance disturbances and dizziness in children. It is important to obtain a thorough history and understand what parents and children mean by being dizzy. Evaluating physicians need to be aware of all the many possible causes that may be involved.

References


