Dizziness in Orthopaedic Physical Therapy Practice: Classification and Pathophysiology

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Abstract: Dizziness is a frequent complaint in patients presenting to orthopaedic physical therapists. Differential diagnosis of dizziness is complex but essential and requires knowledge of musculoskeletal, vestibular, cardiovascular, neurological, metabolic, and psychiatric conditions, thus transcending the musculoskeletal boundaries of orthopaedic physical therapy clinical practice. Physical therapy intervention is not indicated for many causes of dizziness. Some types of dizziness present contra-indications to certain orthopaedic physical therapy interventions. This article presents a diagnostic classification system and relevant pathophysiology that may facilitate orthopaedic physical therapy diagnosis, screening, and subsequent appropriate physical therapy management or medical referral.

Key Words: Dizziness, Classification, Vertigo, Presyncope, Dysequilibrium, Other Dizziness

Health care providers are frequently confronted with patients complaining of dizziness. Dizziness accounts for 7% of physician visits for patients over the age of 45[1]; for adults over 65, it is the number one reason to visit a physician[2]. Dizziness is more common in women than men[3] and the prevalence of dizziness increases with increasing age[4]. Approximately 15 to 30% of people experiencing dizziness will seek medical attention[4].

Differential diagnosis of dizziness can be quite challenging: A wide range of benign and serious conditions can cause dizziness[3]. To further complicate matters, patients use the word "dizziness" to mean, for example, lightheadedness, blurry vision, loss of balance, or a feeling of weakness in the legs. The term "dizziness" is also used for various sensations of body orientation and position that are frequently difficult for patients to describe[5].

Dizziness may result in loss of balance and falls. Falls occur each year in 32% of people aged 65 to 74; this increases to 35% in people aged 75 to 84 and to 51% in people over 85[6]. Falls are directly and indirectly responsible for 12% of all deaths in the geriatric population. In addition, approximately 5% of falls in the elderly result in fractures; another 5 to 10% result in serious injuries requiring medical care[6]. The need for knowledge regarding correct diagnosis and subsequent appropriate management of complaints of dizziness is evident.

The consistent use of a classification system may serve to minimize confusion regarding a patient's dizziness symptoms. Patients with complaints of dizziness can be classified into four subtypes [5, 7]:

1. Vertigo
2. Presyncope
3. Dysequilibrium
4. Other dizziness

Vertigo is a false sensation of movement of either the body or the environment, usually described as spinning, which suggests vestibular system dysfunction[3,4,8,9].

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It is usually episodic with an abrupt onset and often associated with nausea or vomiting[7]. This dysfunction can be located in the peripheral or central vestibular system[8,10]. Peripheral vestibulopathies account for about 35-55% of all cases of dizziness[4]. Central vestibular disorders are less frequent and are responsible for only about 5% of cases of dizziness[4].

Presyncope is described as a sensation of an impending faint or loss of consciousness and is not associated with an illusion of movement[3,7,9]. It may begin with diminished vision or a roaring sensation in the ears[7]. This subtype of dizziness results from conditions that compromise the brain's supply of blood, oxygen, or glucose[9]. The frequency reported for presyncopal dizziness varied from 2% in a dizziness clinic to 16% in an emergency room[11,12]. This type of dizziness may be accompanied by transient neurological signs, e.g., dysarthria, visual disturbances, and extremity weakness[13,14].

Dysequilibrium is a sense of imbalance without vertigo that is generally attributed to neuromuscular problems[3]. It is also described as the feeling that a fall is imminent[7]. The unsteadiness or imbalance occurs only when erect and disappears when lying or sitting[7]. This subtype of dizziness may result from visual impairment, peripheral neuropathy, and musculoskeletal disturbances, and may include ataxia. Sloan et al[3] cited a prevalence of 1-15% for dysequilibrium in patients complaining of dizziness.

Other dizziness is dizziness described as a vague or floating sensation with the patient having difficulty relating the specific feeling to the clinician[3]. It includes descriptions of vague lightheadedness, heavy-headedness, or wooziness and cannot be classified as any of the three previous subtypes[7]. Psychiatric disorders are the main cause for this subtype and account for about 10 to 25% of dizziness cases[3,5]. Anxiety, depression, and hyperventilation are often at the root of this dizziness[3,15]. Changes in vision and tilting of the environment are included in the subtype of other dizziness, as is psychogenic or psychosomatic dizziness due to panic disorder[3,16].

The classification of dizziness into these four subtypes attempts to differentiate complaints of dizziness by symptoms and pathophysiology. This classification system is challenged when an individual complains about more than one subtype of dizziness. Dizziness may result from disorders in the musculoskeletal, vestibular, cardiovascular, neurologic, and metabolic systems as well as from psychiatric disorders[4]. The term "geriatric syndrome" was proposed to describe dizziness in older adults occurring as a result of multi-system impairment[17]. The problem with this term, though, is that it suggests that dizziness is due to old age; however, recent studies have demonstrated that dizziness is prevalent in all adult populations[3,5]. The system is also challenged by symptoms of ataxia, a dyscoordination or clumsiness of movement not associated with muscular weakness, which can be the result of

neuromuscular, i.e., proprioceptive, disorders but also of cerebellar and vestibular disorders occurring with or without symptoms of vertigo[9].

Dizziness is a frequent patient complaint in the orthopaedic physical therapy practice and can result from dysfunctions in multiple body systems. Certain types of dizziness are amenable to physical therapy (PT) interventions; others produce contra-indications to certain PT interventions, while still other causes of dizziness require medical referral. Differential diagnosis is complex but essential and requires knowledge that transcends the musculoskeletal boundaries of typical orthopaedic physical therapy clinical practice. Therefore, this article will extend its discussion of the pathophysiology of dizziness to include not only musculoskeletal but also vestibular, cardiovascular, neurologic, metabolic, and psychiatric causes for dizziness within the framework of the classification system presented above. This article is part one of a two-part series. The second article discusses the history, tests, and measures based on the classification system and pathophysiology introduced here, thus providing the practical information for the orthopaedic physical therapist on diagnosis and screening of patients with complaints of dizziness. Discussion of the anatomy and physiology of the balance control systems is not part of this series and we refer the reader to relevant texts[18-26]. Discussion of PT interventions is also outside the scope of these articles.

Vertigo

As discussed earlier, vertigo is the illusion of movement of the body or of the environment[9]. It is often accompanied by other signs and symptoms[9]:

- Impulsion, i.e., the sensation that the body is being hurled or pulled in space[9]
- Oscillopsia, i.e., the visual illusion of moving back and forth or up and down[1,9]
- Nystagmus, i.e., the rhythmic oscillation of the eyeballs[9]
- Gait ataxia, i.e., dyscoordinated gait not resulting from muscle weakness[9]
- Nausea
- Vomiting

Together, these symptoms are highly indicative of a peripheral or central vestibular dysfunction as discussed below. Table 1 summarizes the general differential diagnostic criteria for central and peripheral vestibular lesions.

Peripheral Vestibular Disorders

Benign Paroxysmal Positional Vertigo

Benign paroxysmal positional vertigo (BPPV) is considered the most common peripheral vestibular disorder[19,27,28]. Annual incidence in the general US population

Table 1. Differential diagnostic characteristics of central versus peripheral vertigo. Brainstem signs may include motor and sensory deficits, hyperreflexia, positive Babinski sign, dysarthria, and limb ataxia[9].

	Central lesions	Peripheral lesions
Vertigo	• Often constant • Less severe	• Often intermittent • Severe
Nystagmus	• Sometimes absent • Uni- or multi-directional • May be vertical	• Always present • Unidirectional • Never vertical
Hearing loss or tinnitus	• Rarely present	• Often present
Brain stem signs	• Typically present	• Never present

has been estimated at 64 per 100,000[28]. BPPV accounts for 17 to 30% of all new patients presenting to vestibular clinics[28]. BPPV is generally seen in people over the age of 40 and it is rare in people under 20[28]. Idiopathic BPPV has a peak incidence of onset in the sixth decade of life[29]. Non-idiopathic BPPV has been associated with head trauma, insult to the labyrinth, surgical stapedectomy, chronic suppurative otitis media, and degeneration of the inner ear[27,28,30].

Two pathophysiological theories have been proposed to explain the etiology of BPPV: cupulolithiasis and canalithiasis[28]. The theory of cupulolithiasis holds that sedimentous material, possibly macular otoconia, is released into the endolymphatic fluid in the semicircular canals (SCCs). This release of sedimentous material is hypothesized to result from trauma or degenerative changes (see non-idiopathic BPPV above). When the head is upright, this material will settle on the SSC cupula. Fixed deposits on the cupula increase the density of this structure making the cupula, which previously had the same density as the surrounding endolymphatic fluid, now sensitive to gravity and, therefore, head position[28]. The theory of cupulolithiasis is based on dissection studies: cupular deposits appear to be relatively common, but a correlation with clinical symptomatology is lacking[28].

The canalithiasis theory proposes that BBPV is the result of free-floating endolymphatic densities in the SCCs[28]. Changes in head position are hypothesized to produce movement of these particles creating a current in the SCCs; the resultant hydrodynamic drag could then displace the cupula, stimulating the SCC hair cells. The canalithiasis theory provides a better explanation for the latency of vertigo observed in BPPV than does the cupulolithiasis theory: One could assume that it takes a few seconds for the endolymphatic densities to overcome the inertia of the endolymphatic fluid in the SCC before they are able to create sufficient hydrodynamic drag to move the cupula. Fatigability or habituation of the vertigo response with repeated provocative head

movement could be explained by dissolution of the clumped endolymphatic material. Free-floating particulate matter has been observed in SCC endolymphatic fluid during surgery[28].

Canalithiasis and cupulolithiasis may well represent two stages of the same pathological process: All etiological factors for BPPV may result in endolymphatic densities, which may be free-floating or adhered to the cupula[28]. Infections, e.g., acute labyrinthitis and chronic suppurative otitis media, result in white blood cells, phagocytes, and endothelial fragments floating in the endolymphatic fluid. Head trauma or stapedectomy may result in red blood cells in the endolymphatic fluid, and age-related degeneration may release cellular debris and macular otoconia[28].

The name BPPV implies that this type of vertigo is positional in nature. However, it may be more correct to call BPPV a positioning-type vertigo. In posterior SCC BPPV, vertiginous symptoms occur when a patient transfers quickly into a supine position, especially when the head is turned to the affected side, extending the head on the neck[27]. Symptom onset occurs within one to five seconds upon a change in position[27,28]. Symptom duration is brief: 30-60 seconds; hence, it is a positioning-type vertigo rather than positional-type vertigo as occurs in vertebrobasilar insufficiency. A horizontal-rotary nystagmus is the hallmark of BPPV originating in the posterior SCC[27,30]. Excitation of neurons innervating the ipsilateral superior oblique muscle and the contralateral inferior rectus muscle are responsible for the horizontal-rotary direction[27]. Symptoms of dysequilibrium and nausea may also occur, with a rare occurrence of vomiting[27,30]. Most commonly the posterior SCC is involved in BPPV[19,27,30]. However, BPPV may also involve the horizontal SCC[31]. Rolling the head in the plane of the horizontal SCC while in a supine position usually induces the horizontal SCC variant of BPPV. Other provocations include flexion and extension of the head or shifting from supine to upright[31].

Meniere's Disease

Meniere's disease is characterized by paroxysmal vertigo lasting minutes to days accompanied by tinnitus, fluctuating low-frequency hearing loss, and a sensation of fullness in the ear[7,9,25]. Sensorineural hearing loss (a hearing loss caused by lesions in the cochlea or the cochlear nerve[9]) is progressive over multiple episodes, whereas the vertigo tends to become less severe[9,25]. Before the first acute attack, patients frequently note an insidious onset of tinnitus, hearing loss, and the sensation of fullness in the ear[9]. Subsequent attacks typically occur suddenly with incapacitating vertigo, roaring unilateral tinnitus, and ipsilateral hearing loss[7]. Attacks are often associated with nausea and vomiting[7,9]. Age of onset is usually between the ages of 20 and 50, and men are more often affected than women[9]. Up to 20% of patients have a family history of Meniere's disease[7]. During an acute episode, spontaneous horizontal or rotary nystagmus may be present, which may change direction[9]. The underlying cause is thought to be an increase in the volume of the endolymphatic fluid in the membranous labyrinth[9]. This endolymphatic hydrops results in excessive amounts of endolymphatic fluid, which displaces the inner ear structures with resultant signs and symptoms[7].

Acute Peripheral Vestibulopathy

Sudden onset vertigo, nausea, and vomiting lasting up to 2 weeks characterize acute peripheral vestibulopathy. This diagnosis includes diagnoses of acute labyrinthitis and vestibular neuronitis. Simon, Aminoff, and Greenberg[9] noted that these two diagnoses are based on unverifiable inferences regarding pathophysiology and location of disease. Eaton and Roland[7] mentioned acute labyrinthitis as a separate diagnosis characterized by acute onset severe vertigo, nausea, vomiting, and diaphoresis lasting 1-5 days with subsequent resolution of complaints over a 2-3 week period. A viral origin is likely: Approximately 50% of patients have an associated upper respiratory infection[7]. Simon et al[9] noted that acute vestibular neuropathy is not accompanied by hearing loss, whereas Souza[25] used the presence of some hearing deficit in labyrinthitis as a differential diagnostic criterion to distinguish this disease from vestibular neuronitis. The disease may be recurrent and some degree of vestibulopathy may be permanent[9]. Differential diagnosis from central disorders characterized by acute vertigo is imperative[9].

Otosclerosis

The pathophysiology mechanism behind otosclerosis is immobility of the stapes. The stapes normally transmits sound-induced vibration of the tympanic membrane to the inner ear. Otosclerosis is characterized by conductive hearing loss (a hearing loss resulting from disorders in the external or middle ear[9]) but sensorineural hearing loss and vertigo also occur. Vertigo is recurrent and episodic, with or without positional vertigo, but may also be more constant. Nystagmus can be spontaneous or positional. Hearing loss has an age of onset before 30, and a positive family history is common[9].

Head Trauma

We discussed head trauma in the etiology of BPPV. Head trauma may result in labyrinthine damage and subsequent vertigo. Undiagnosed skull fractures (petrosal bone) may damage the vestibulo-cochlear nerve with resultant vertigo and hearing loss. Otorrhea (discharge of CSF from the ear) may be present[9].

Cerebellopontine Angle Tumor

A benign acoustic neuroma, a tumor of the Schwann cells covering the vestibular portion of the vestibulo-cochlear nerve in the internal auditory canal, and meningiomas of the cerebellopontine angle can produce insidious unilateral sensorineural hearing loss[9,32]. Vertigo, tinnitus, and a sensation of fullness in the ear are less common. Due to their anatomic relationship to the vestibulo-cochlear nerve, the trigeminal (CN V) and facial nerves (CN VII) are often affected: Patients may complain of headache, facial pain, or facial weakness[9]. Vertigo develops in 20 to 30% of patients, but a non-specific unsteadiness is more common[9]. Age of onset is usually between 30 and 60. Acoustic neuromas are more common in neurofibromatosis patients. Café-au-lait spots on the skin and axillary or inguinal freckles may be external indicators to suspect neurofibromatosis 1 or Von Recklinghausen disease[9].

Toxic Vestibulopathies

Ingested alcohol differentially distributes between the cupula and the endolymphatic fluid: It initially diffuses preferentially into the cupula decreasing its density relative to that of the endolymphatic fluid, thus rendering the peripheral vestibular apparatus unusually sensitive to gravity. With time, alcohol also diffuses the endolymphatic fluid. As blood levels decrease, alcohol leaves the cupula before leaving the endolymphatic fluid again causing temporary differences in sensitivity to gravity. Alcohol-induced vertigo, therefore, consists of two symptomatic phases separated by one to two hours. The whole episode lasts up to 12 hours. Vertigo and nystagmus are evident in the lateral recumbent position and are accentuated when the eyes are closed[9].

Aminoglycosides are antibiotics that can cause both vestibular and auditory symptoms[9,32]. Streptomycin, gentamicin, and tobramycin are likely to cause vestibular toxicity by destroying hair cells in the membranous

labyrinth. The use of amikacin, kanamycin, and tobramycin is associated with hearing loss. Vertigo, nausea, vomiting, and a spontaneous nystagmus may have an acute onset[9].

Salicylates (aspirin and derivatives) can damage the vestibular and cochlear end organ resulting in vertigo, tinnitus, and sensorineural hearing loss. Headache, nausea, vomiting, thirst, hyperventilation, and sometimes confusion may all indicate chronic salicylate overdosage[9].

Quinine and quinidine can cause tinnitus, hearing loss, vertigo, vision deficits (including disorders of color vision), nausea, vomiting, abdominal pain, hot flushed skin, and diaphoresis. Fever, encephalopathy, coma, and death can occur in severe cases. Symptoms can be the result of overdosage or may result from a single dose[9].

The chemotherapeutic drug *Cis*-platinum causes ototoxicity in about 50% of patients with resultant reversible tinnitus, hearing loss, and vestibular dysfunction[9].

Acoustic Neuropathy

Basilar meningitis from a bacterial, syphilitic, or tuberculous infection or from sarcoidosis can lead to compression of the vestibulo-cochlear (acoustic) nerve. Hypothyroidism, diabetes mellitus, and Paget's disease can also affect the vestibulo-cochlear nerve. Vertigo can occur, but hearing loss is more common. Other cranial nerves may also be affected[9].

Perilymphatic Fistula

A perilymphatic fistula is a rare cause of vertigo. An opening develops between the middle and inner ear (oval or round window rupture)[25]. Head injury, barotrauma due to diving or flying, or a very forceful Valsalva maneuver are thought to induce the fistula[7,25]. With rapid loss of perilymphatic fluid, severe vertigo and hearing loss may develop. Low-volume leaks may only produce episodic vertigo or dysequilibrium and hearing loss may be absent[7].

Autoimmune Disease of the Inner Ear

This diagnosis is associated with fluctuating deafness and recurrent vertigo. Other autoimmune diseases, e.g., rheumatoid arthritis, Crohn's disease, or polyarteritis, are often concurrently present[32].

Central Vestibular Disorders

We discussed above the differences between vertigo of central and peripheral origin (Table 1). Some central vestibular disorders may occur without vertigo. All cerebellar disorders produce gait ataxia, inviting a misdiagnosis of the dizziness subtype of dysequilibrium (Table 2). Central vestibular disorders can also be the result of ischaemic processes. We will discuss vascular causes of

central vestibular dysfunction (vertebrobasilar ischaemia and infarction) under presyncope.

Drug Intoxication

Many drug intoxication syndromes produce global cerebellar dysfunction. Agents include[9,32]:
- Alcohol
- Sedative hypnotics: barbiturates, benzodiazepines, meprobamate, ethchlorvynol, methaqualone
- Anti-convulsants: phenytoin
- Hallucinogens: phencyclidine
- Street drugs: heroin
- Mercuric and organophosphoric compounds

Drug intoxication often also produces a confused state. Alcohol and sedatives tend to produce somnolence, and hallucinogens tend to cause agitation. The importance of taking a good history including medication or drug use is evident.

Wernicke's Encephalopathy

This acute disorder is comprised of the diagnostic triad of ataxia, ophthalmoplegia (lateral rectus palsy), and confusion. It is caused by thiamine (vitamin B_1) deficiency and is common in alcoholics but may also be caused by general malnutrition. Ataxia affects the arm in 10% of patients; the legs are involved in 20%. A horizontal or combined horizontal-vertical nystagmus is classically present[9].

Inflammatory Disorders

Viral cerebellar infections can occur in patients with St. Louis encephalitis, AIDS dementia complex, and meningoencephalitis associated with varicella, mumps, poliomyelitis, infectious mononucleosis, and lymphocytic choriomeningitis. Bacterial infection of the cerebellum is rare: Only 10 to 20% of brain abscesses are located in the cerebellum. The cerebellum may be infected by bacteriae in *Haemophilus Influenzae* meningitis and Legionnaire's disease[9]. Some diseases are hypothesized to have an autoimmune origin. Acute cerebellar ataxia of childhood usually follows a viral infection or inoculation. Acute disseminated encephalomyelitis and a variant of Guillain-Barre syndrome also fall in this category[9].

Multiple Sclerosis

Vertigo is rarely the first symptom of multiple sclerosis (MS) but is common during the course of the disease. It may have an acute onset and can be positional. Gait ataxia is the presenting complaint in 10 to 15% of patients with MS. Nystagmus is also a common sign in MS. A history of remitting and relapsing neurologic dysfunctions affecting multiple sites in the central neu-

Table 2. Signs and symptoms of cerebellar disorders (modified from Simon et al[9]).

Location of Cerebellar Involvement	Signs	Diagnoses
Midline	• Nystagmus • Head and trunk oscillation (titubation) • Gait ataxia	• Tumor • Multiple sclerosis
Superior vermis	• Gait ataxia	• Wernicke's encephalopathy • Alcoholic cerebellar degeneration • Tumor • Multiple sclerosis
Cerebellar hemisphere	• Nystagmus • Ipsilateral gaze paralysis • Dysarthria • Ipsilateral hypotonia • Ipsilateral limb ataxia • Gait ataxia • Falling to the side of the lesion	• Infarction • Hemorrhage • Tumor • Multiple sclerosis
Global cerebellar	• Nystagmus • Bilateral gaze paralysis • Dysarthria • Bilateral hypotonia • Bilateral limb ataxia • Gait ataxia	• Drug intoxications • Hypothyroidism • Hereditary cerebellar degeneration • Paraneoplastic cerebellar degeneration • Wilson's disease • (Para) infectious encephalomyelitis • Creutzfeldt-Jakob disease • Multiple sclerosis

rologic system may be indicative of MS in undiagnosed patients[9].

Alcoholic Cerebellar Degeneration

Alcoholic cerebellar degeneration usually occurs in patients with a history of 10 or more years of binge drinking. It usually has its onset between the ages of 40 and 60 and is more common in men. The onset is insidious and progression is gradual. As in Wernicke's encephalopathy, this syndrome affects mainly the superior cerebellar vermis. Gait ataxia is the most common finding; nystagmus is a less frequent finding. Distal sensory deficits in the feet and absent ankle reflexes from diabetic polyneuropathy and signs of malnutrition may clue the clinician in to the diagnosis[9].

Phenytoin-Induced Cerebellar Degeneration

Phenytoin is an anti-epileptic medication. Long-term treatment with phenytoin may produce a global cerebellar degeneration[9].

Hypothyroidism

Hypothyroidism produces a global cerebellar dysfunc-tion. The cerebellar syndrome associated with hypothyroidism is usually subacute or chronically progressive in its onset. It is most common in middle-aged or elderly women. Gait ataxia is the prominent finding; nystagmus and dysarthria are less common. Other neurologic disorders associated with hypothyroidism include sensorineural hearing loss, carpal tunnel syndrome, neuropathy, and myopathy; these signs and symptoms may raise suspicion of hypothyroidism in undiagnosed patients[9].

Paraneoplastic Cerebellar Degeneration

The pathophysiological mechanism in paraneoplastic cerebellar degeneration appears to involve antibodies to tumor cell antigens cross-reacting with cerebellar Purkinje cells. Patients with lung cancer, ovarian cancer, Hodgkin's disease, and breast cancer are most at risk of developing this type of global cerebellar degeneration. Onset can be before or after the diagnosis of cancer; progression occurs over the course of months. Gait and limb ataxia and dysarthria occur in most cases; nystagmus is rare[9].

Hereditary Spinocerebellar Degenerations

There are seven autosomal dominant spinocerebellar ataxias characterized by adult-onset, slowly progres-

sive cerebellar ataxia that affects gait early and severely. Friedreich's ataxia is an autosomal recessive spinocerebellar disease with an onset in childhood. Progressive gait ataxia is the first symptom, followed by ataxia of all limbs within two years. Decreased tendon reflexes in the legs, dysarthria, impairments of proprioceptive and vibration sense, and weakness in the legs are common, as are nystagmus, vertigo, and hearing loss[9].

Ataxia-Telangiectasia

This syndrome is an autosomal recessive disorder with an onset before age 4 and global cerebellar involvement producing nystagmus, dysarthria, and gait, limb, and trunk ataxia. Loss of vibration and position sense in the legs further adds to gait ataxia. Vascular lesions are present on the skin and the eyes, especially on the ears, nose, face, and antecubital and popliteal fossae[9].

Wilson's Disease

Wilson's disease is a disorder of the copper metabolism with copper deposition in multiple body tissues. A rusty-brown ring around the cornea is indicative of this disease and may indicate the origin of the cerebellar symptoms that are associated with this disorder[9].

Creutzfeldt-Jakob Disease

This disease is characterized by dementia, cerebellar signs in 60% of patients, and gait ataxia in 10%. The ataxia is usually accompanied in 50% of patients thus affected with nystagmus, dysarthria, and trunk and limb ataxia. The disease involves progressive dementia, (extra) pyramidal dysfunction, and myoclonus, with death within a year of onset[9].

Posterior Fossa Tumors

Cerebellar tumors frequently present with headache due to increased intracranial pressure or with ataxia. Nausea, vomiting, vertigo, and cranial nerve palsies are also common. Most common in adults are secondary tumors metastasized from primary tumors in the breast or lung. Primary tumors are more frequent in children and include astrocytomas and medullablastomas. Headache and vomiting are frequently presenting symptoms; ataxia and visual dysfunction is also a common first symptom with medullablastomas in children[9].

Posterior Fossa Malformations

Congenital anomalies may cause vestibular or cerebellar symptoms in adulthood[9,32]. Type I Arnold-Chiari malformation involves downward displacement of the cerebellar tonsils through the foramen magnum causing symptoms of cerebellar involvement and brainstem compression. Ataxia in this malformation affects gait and is bilateral. Hydrocephalus may cause headache and vomiting. Brainstem compression can be associated with vertigo, nystagmus, and cranial nerve palsies[9].

Familial Paroxysmal Ataxia

This hereditary recurrent ataxia is associated with nystagmus and dysarthria. Other symptoms may include vertigo, tinnitus, diplopia, oscillopsia, facial palsy, headache, and fever. Attacks last from 15 minutes to several hours and may be triggered by physical exercise, caffeine, alcohol, or sudden movements[32].

Presyncope

As discussed earlier, presyncope is a sensation of an impending faint or loss of consciousness that is not associated with an illusion of movement. Presyncope results from conditions that compromise the brain's supply of blood, oxygen, or glucose. This can compromise the function of the cerebral hemispheres or the brainstem. Different conditions can cause either a pancerebral hypoperfusion or a selective hypoperfusion of the brainstem[9].

Pancerebral Hypoperfusion

Causes for presyncope due to pancerebral hypoperfusion can be classified into four categories[9]:
- Vasovagal presyncope
- Cardiovascular presyncope
- Cerebrovascular presyncope: migraine, Takayasu's disease, carotid sinus syncope
- Miscellaneous causes of presyncope: orthostatic hypotension, hyperventilation syncope, cough syncope, micturition syncope, glossopharyngeal neuralgia

Insufficient supply of glucose and subsequent compromised pancerebral function can also result in presyncope[25].

Vasovagal Presyncope

With a vasovagal presyncope, parasympathetic hyperactivity causes a decrease in cardiac output with a subsequent decrease in cerebral blood flow[7]. Precipitating factors include emotional stimulation, pain, the sight of blood, fatigue, medical instrumentation, blood loss, or prolonged motionless standing. Vasovagal presyncope occurs in all age groups and affects men and women equally. Short prodromes (10 seconds to a few minutes) will precede syncope and include lightheadedness, nausea, pallor, salivation, blurred vision, and tachycardia[7,9]. Vasovagal presyncope mainly occurs with the patient in a sitting or standing position; it is rare with a patient in the recumbent position[9,25].

Cardiovascular Presyncope

A cardiovascular syncope should be suspected when syncope occurs with the patient in a recumbent position, during or after physical activity, or in a patient with a known medical history of heart disease. Table 3 provides an overview of cardiac causes of syncope. Associated symptoms may include chest pain or discomfort, neck and/or arm pain and discomfort, palpitation, dyspnea, fatigue, cough, cyanosis, edema, and claudication[33].

Migraine

Migraine is characterized by headache that is usually unilateral and of a pulsatile quality. Nausea, photophobia, phonophobia, vomiting, and lassitude are frequently associated with migraine. Visual or other neurological auras occur in 10% of patients. Also in about 10% of patients, migraine may be associated with presyncope. Syncope will occur during the migraine attack, often when the patent quickly rises to a standing position, suggesting a component of orthostatic hypotension. A family history of migraine is usually present. Migraine is more common in women with an onset early in life[9].

Takayasu's disease

Takayasu's disease is most common in Asian-descent women. Presyncope can occur after exercise, standing, or head movement and is associated with impaired vision and confusion. The disease is also known as pulseless disease: Brachial artery pulsations are absent or decreased[9].

Carotid Sinus Syndrome

Carotid sinus syndrome is relatively rare. It occurs

Table 3. Causes of cardiovascular syncope (modified from Simon et al[9]).

Cardiac Arrest
Cardiac inflow obstruction • Left atrial myxoma or thrombus • Tight mitral stenosis • Constrictive pericarditis • Cardiac tamponade • Restrictive cardiomyopathies • Tension pneumothorax
Cardiac outflow obstruction • Aortic stenosis • Pulmonary stenosis • Hypertrophic obstructive cardiomyopathy
Dissecting aortic aneurysm
Severe pulmonary-vascular disease • Pulmonary hypertension • Acute pulmonary embolus
Cardiac dysrhythmias • Tachyarrhythmias: paroxysmal atrial tachycardia, atrial filter, atrial fibrillation, accelerated junctional tachycardia, ventricular tachycardia, ventricular fibrillation • Bradyarrhythmias: sinus bradycardia, sinus arrest, second or third degree heart block, implanted pacemaker failure • Mitral valve prolapse • Prolonged Q-T interval syndromes • Sick sinus syndrome • Drug toxicity: digitalis, quinidine, procainamide, propranolol, phenothiazines, tricyclic antidepressants, potassium

in men twice as frequently as in women. Patients are usually over the age of 60. Propranolol, digitalis, and methyldopa seem to predispose patients to this syndrome[9]. Pressure on the carotid sinus due to a tight collar or local neck tumor will cause vagal stimulation leading to bradycardia and subsequent syncope[9,25].

Orthostatic Hypotension

Orthostatic hypotension may occur in teenagers, but it is most common in the sixth and seventh decades of life. It occurs more often in men than in women. Syncope and presyncope happen when rapidly rising to a standing position, standing motionless for prolonged periods, and standing after prolonged recumbency. The pathophysiology of orthostatic hypotension usually involves reduced blood volume or autonomic nervous system dysfunction[7,9]. Table 4 lists causes of orthostatic hypotension.

Table 4. Causes of orthostatic hypotension (modified from Simon et al[9]).

Hypovolemia or hemorrhage
Addison's disease
Drug-induced hypotension • Antidepressants • Antihypertensives • Bromocriptine • Diuretics • Levodopa • Monoamine oxidase (MOA) inhibitors • Nitroglycerin • Phenothiazines
Polyneuropathies • Myeloid neuropathy • Diabetic neuropathy • Guillain-Barre syndrome • Porphyric neuropathy • Vincristine neuropathy
Other neurologic disorders • Idiopathic orthostatic hypotension • Multiple sclerosis • Parkinsonism • Posterior fossa tumor • Shy-Drager syndrome • Spinal cord injury with paraplegia • Surgical sympathectomy • Syringomyelia • Syringobulbia • Tabes dorsales (syphilis) • Wernicke's encephalopathy
Cardiovascular disorders
Prolonged bed rest or immobilization

Hyperventilation

Hypocapnia is the pathophysiological reason for presyncope in patients with hyperventilation: It induces cerebral vasoconstriction resulting in central nervous system hypoperfusion. Patients are usually women between the ages of 20 and 40. Other symptoms include lightheadedness, shortness of breath, peri-oral numbness and paraesthesiae, and muscular twitching. Hyperventilation-induced presyncope commonly occurs when supine and can be brought on by asking the patient to hyperventilate[9].

Cough-Related Syncope

Cough syncope is immediately preceded by coughing, which need not be prolonged. It occurs mainly in middle-aged men with a history of chronic obstructive pulmonary disease. There are no prodromal symptoms. Syncope (and presyncope) may be caused by an increase in intracranial pressure due to coughing with resultant cerebral hypoperfusion[9]. Cough-induced presyncope may occur when the patient is supine and may be reproduced by asking the patient to cough[9,25].

Micturition Syncope

Micturition-induced presyncope occurs almost exclusively in men; episodes occur mainly at night before, during, or after micturition. Peripheral blood pooling, vagus-induced bradycardia, and prolonged recumbency are likely responsible[9].

Glossopharyngeal Neuralgia

Syncope in glossopharyngeal neuralgia is the result of a glossopharyngeal-vagal reflex circuit causing transient brady-arrhythmia that results in cerebral hypoperfusion. Glossopharyngeal neuralgia is rare; symptoms include paroxysmal pain in the tonsillar pillar or the external ear canal during swallowing, talking, or coughing[9,25].

Hypoglycaemia

Presyncopal dizziness may also be the result of a hypoglycaemic reaction. Asking the patient whether the dizziness occurs mainly when he or she has not eaten may clue in the clinician to hypoglycaemia as a cause for dizziness[25]. Polyuria (excessive urination), polydipsia (excessive thirst), polyphagia (excessive hunger), weight loss, and fatigue may be diagnostic indicators for patients with undiagnosed diabetes[34].

Brainstem Hypoperfusion

Selective hypoperfusion of the brainstem is the re-

sult of compromise or disease processes in the vertebrobasilar system.

Vertebrobasilar Insufficiency

Vertebrobasilar insufficiency (VBI) is characterized by dizziness symptoms associated with focal neurologic abnormalities of sudden onset and brief duration (seconds to minutes) that relate to the specific areas supplied by the vertebrobasilar vessels[10,28,35]. Terrett[36] provided us with a useful mnemonic for signs and symptoms associated with VBI (Table 5). Van der Velde[28] suggested classifying dizziness caused by VBI as a positional-type dizziness to distinguish it from the positioning-type

Table 5. Five D's And three N's (modified from Terrett[36]).

- Dizziness
- Drop attacks
- Diplopia
- Dysarthria
- Dysphagia
- Ataxia of gait
- Nausea
- Numbness
- Nystagmus

dizziness produced by BPPV and cervicogenic dizziness: Dizziness (and other symptoms) will increase in patients with VBI when maintaining the head in the provocative position in contrast to the latter two pathologies where symptoms are provoked by positioning but adapt when the head is maintained in the provocative position.

VBI may be caused by intrinsic and/or extrinsic mechanical disorders[35]. Atherosclerosis, thrombo-embolic events, and arterial dissection are examples of intrinsic mechanical disorders[35-37]. Anomalous soft tissue structures, such as bands of the deep cervical fascia, or compression of the vertebral artery between the longus colli and anterior scalene muscles are examples of extrinsic mechanical disorders[35]. Osteophytes laterally projecting from the uncinate processes and backward bending of the upper cervical spine in the forward head posture may also cause mechanical compression[24,37]. Additionally, vertebral artery occlusion may result following acute cervical spine trauma, causing fracture and dislocation[38].

The nystagmus in patients with VBI may be vertical, implicating the central vestibular structures affected by this ischaemic process[39]. When syncope occurs, recovery is frequently prolonged (30-60 minutes or longer)[9]. Patients may report symptoms following cervical manipu-

lation, but symptoms have also been reported after ADL activities such as a shampoo treatment at the hair salon, yoga exercises, painting a ceiling or wall, changing a light bulb, or turning the head when backing up the car, childbirth, surgery, hanging the wash, archery, swimming, Tai Chi, sexual intercourse, and wrestling[36,40]. Terrett[36] provided an exhaustive overview of all reported non-manipulative causes of VBI. A position of rotation or extension is frequently implicated in causing VBI. However, even in a patient with mechanical disorders of the vertebral artery, this position need not result in symptoms due to compensation by the collateral circulation, namely the opposite vertebral artery and the carotid arteries[35]. Symptoms of VBI are more likely to occur when the collateral circulation is concurrently compromised[35].

Vertebrobasilar Infarction

Recurrent vertebrobasilar ischaemic attacks lead to stroke in 20% of patients[9]. Stroke can develop acutely or subacutely after arterial wall damage due to a combination of vasospasm, thrombus formation, thrombo-embolization, and the formation of a dissecting aneurysm[36]. Due to its association with cervical manipulation and other (non) traumatic causes but also because of the contraindication to (further) manipulative treatment and the need for medical referral, knowledge of signs and symptoms of a dissecting aneurysm is important. A sudden onset of severe neck and occipital pain is the hallmark of dissection of the vertebral artery[41,42]. Vertigo, unilateral facial paraesthesiae, cerebellar signs, and visual field defects may also be present[41]. It is sobering to realize that the sudden onset of neck and occipital pain may, in fact, be the complaint for which a patient with arterial dissection may seek PT intervention.

Vertebrobasilar infarction will produce signs and symptoms based on the area cut off from its vascular supply. Presyncopal dizziness in an acute cerebellar infarction is accompanied by unilateral sensorineural hearing loss and nystagmus with occlusion of a branch from the basilar artery or anterior inferior cerebellar artery (AICA), the internal auditory artery, which supplies the vestibulo-cochlear nerve[9]. Acute proximal vertebral artery occlusion may result in Wallenberg's syndrome, combining presyncopal dizziness with vertigo, vomiting, nausea, dysphagia, hoarseness, nystagmus, ipsilateral Horner's syndrome, sensation loss in the face, limb ataxia, and loss of light touch and position sense in the limbs. Cerebellar infarction due to occlusion of the AICA, posterior inferior cerebellar artery (PICA), or superior cerebellar artery all result in ipsilateral limb ataxia and hypotonia. Other symptoms and signs include headache, nausea, vomiting, vertigo, nystagmus, dysarthria, ocular or gaze palsies, facial weakness or sensory loss, contralateral hemiparesis, and hemisensory deficit[9].

Vertebrobasilar Migraine

This pathology usually affects young women with prolonged attacks (up to 72 hours) consisting of intense vertigo, vomiting, dysarthria, and limb and peri-oral paraesthesiae. Transient quadriplegia, fainting, confusion, and stupor for a period of hours can occur during an attack but are rare. A visual aura may occur preceding the attack, and an attack may be followed by an occipital region headache. A family history of migraine and a correlation of attacks with the menstrual period are diagnostic indicators[32].

Subclavian Steal Syndrome

This syndrome results from retrograde flow in the vertebral artery and subsequent brainstem hypoperfusion due to subclavian or innominate artery stenosis[9]. In the subclavian steal syndrome, blood passes from the vertebral artery into the distal subclavian artery with physical activity of the ipsilateral arm[43]. Symptoms include vertigo, diplopia, limb paresis, arm fatigue, paraesthesiae, and ataxia, all of which are more common than presyncope[9]. Symptoms are brought on by arm exertion, not head or neck movements allowing for differential diagnosis with, for example, vertebrobasilar insufficiency[43].

Dysequilibrium

As discussed earlier, dysequilibrium is a sense of imbalance without associated vertigo.

Visual Impairment

We already discussed the gaze-related palsies and decreased gaze fixation related to vestibular, cerebellar, and brain stem lesions. More germane changes, such as decreased visual field, visual acuity, and depth perception, may also produce dysequilibrium and subsequent complaints of dizziness[44].

Somatosensory Impairment

Somatosensory deficits result from pathologies that cause sensory ataxia. These pathologies can be classified as polyneuropathies, myelopathies, or a combination of both. Table 6 reviews causes of sensory ataxia. Myelopathy and cervicogenic dizziness are most relevant to orthopaedic physical therapy practice.

Myelopathy

Table 6 also provides an overview of pathologies associated with spinal cord compression. Cord compression can occur in the cervical, thoracic, and high lumbar regions of the spine. With obvious variations in pre-

Table 6: Causes of sensory ataxia (modified from Simon et al[9]).

Polyneuropathy or Myelopathy
• Friedreich's ataxia
• Neurosyphilis (tabes dorsalis)
• Nitrous oxide
• Vitamin B_{12} deficiency
• Vitamin E deficiency
Polyneuropathy
• Autosomal dominant sensory ataxic neuropathy
• *Cis*-platinum use
• Dejerine-Sottas disease
• Diabetes
• Diphtheria
• Hypothyroidism
• Immune-mediated neuropathies
• Isoniazid
• Paraneoplastic sensory neuronopathy
• Pyridoxine
• Refsum's disease
• Taxol
Myelopathy
• Acute transverse myelitis
• AIDS (vacuolar myelopathy)
• Multiple sclerosis
• Tumor or cord compression
• Vascular malformations

sentation depending on the segmental level of spinal cord compromise and the amount of compromise, spinal cord compression may result in[45-47]:

- Variable upper limb, lower limb, neck, and trunk pain
- Sensory impairment of the upper and lower limbs not limited to a single dermatome
- Non-myotomal arm and/or leg weakness
- Velocity-dependent limb hypertonia
- Upper motor neuron signs in the extremities: hyperreflexia, positive Babinski sign and Hoffman's reflex, gait ataxia, and spastic bladder

Atrophy of the intrinsic hand muscles is the result of segmental necrosis of anterior horn cells in patients with cervical myelopathy[45,47]. Anterior horn cell necrosis usually occurs at the level of compression: "Myelopathic hands" result from compression of the C8-T1 nerve levels at the C6-C7 spinal level because of the oblique course of the cervical nerve roots. Cord compression can also result from interactions with the musculoskeletal system. This indicates a possible need for range of motion and provocative (i.e., spinal arthrogenic instability) testing; the cervical spine has especially been implicated due to a combination

of degenerative stenosis, instability, and a physiological narrowing of canal diameters in backward bending[48].

Cervicogenic Dizziness

Cervicogenic dizziness is defined as a non-specific sensation of altered orientation in space and dysequilibrium originating from abnormal afferent activity from proprioceptors in the neck[49]. Initially, "cervical vertigo" was the term used to describe dizziness symptoms stemming from the cervical spine, but in more recent literature, "cervicogenic dizziness" has become the preferred term, as vertigo usually suggests vestibular involvement[3,50].

Animal and human studies have implicated the upper cervical spine, C1-C3, in symptoms of dizziness originating from the neck[22]. However, the existence of cervicogenic dizziness is controversial, and the proposed pathophysiology is not well understood[51]. The upper cervical spine contains a great density of mechanoreceptors with an influence on postural control[52,53]. Input from cervical mechanoreceptors allows the body to interpret afferent vestibular input despite changes in head position[51]. The cervical mechanoreceptors are hypothesized to become irritated due to trauma and immobilization, which may lead to an incorrect interpretation of the head's position in relation to the trunk[23]. Additionally, poor postural awareness may induce upper cervical hypomobility causing a decreased stimulation of postural mechanoreceptors[24]. Aberrant afferent information stemming from the upper cervical mechanoreceptors is hypothesized to produce a sensory mismatch with the visual and vestibular systems at the level of the vestibular nuclei and cerebellum resulting in a conscious awareness of balance and dysequilibrium[23,54].

Associated symptoms in patients with cervicogenic dizziness may include unsteady gait and postural imbalance associated with neck pain and/or injury. Limited neck range of motion and headache may also be reported[50]. There is no "gold standard" test for cervicogenic dizziness, making this condition a diagnosis of exclusion[50,51]. Vestibular function tests and a thorough neurological examination are performed to rule out vestibular dysfunction and central nervous system involvement[50]. Cervicogenic dizziness is a more likely diagnosis when the clinician can relate the patient's dizziness symptoms to neck pain and a history of neck injury[50]. A pattern of symptoms that may suggest that cervicogenic dizziness is likely may be present when the patient describes the dizziness as worsening when the pain worsens or dizziness occurring only when neck pain is present[23]. Cervical spondylosis has been hypothesized as a possible cause for this type of dizziness[55]. Spasms of the upper trapezius and sternocleidomastoid muscles have also been cited as a trigger for cervicogenic dizziness[50]. Approximately 20-58% of individuals who sustain a cervical acceleration-deceleration injury will experience dizziness[56]. This type of trauma can also produce dizziness based on BPPV and VBI. Table 7 provides information helpful for differential diagnosis[28,36,57].

Musculoskeletal Impairment

The musculoskeletal system is the effector organ of the balance control systems. Decreased muscle strength and endurance, decreased joint range of motion and stability, increased through-range resistance of joints, and posture negatively affecting the location of the center of gravity

Table 7. Differential diagnostic characteristics of cervicogenic dizziness, BPPV, and VBI[28,36,57].

	Vertigo Type	Nystagmus Characteristics	Associated Signs and Symptoms
Cervicogenic dizziness	Positioning-type	• No latency period • Brief duration • Fatigable with repeated motion	• Nystagmus • Neck pain • Suboccipital headaches • Cervical motion abnormality
BPPV	Positioning-type	• Short latency: 1-5 seconds • Brief duration: <30 seconds • Fatigable with repeated motion	• Nystagmus
VBI	Positional-type	• Long latency: 55+/-18 seconds[57] • Increasing symptomatology with maintained head position • Not fatigable with repeated motion	• Dizziness • Drop attacks • Diplopia • Dysarthria • Dysphagia • Ataxia of gait • Nausea • Numbness • Nystagmus

in relation to the base of support can all have a negative effect on balance and may contribute to a sense of dysequilibrium for which the patient will seek our assistance. This provides the indication for a comprehensive evaluation of musculoskeletal impairment in the differential diagnostic process of patients complaining of dizziness.

Basal Ganglia Impairment

Parkinsonism is common especially in older adults[7,9]. It occurs in all ethnic groups with an approximately equal sex distribution[9]. Clinical findings include tremor, rigidity, hypokinesia, and gait and postural abnormalities[9]. The characteristic forward bent posture negatively affects the location of the center of gravity in relation to the base of support. The characteristic festinating gait with short, shuffling steps that become successively more rapid can further contribute to a sense of imbalance[7]. Phenothiazines, butyrophenones, metoclopramide, reserpine, and tetrabenazine are drugs associated with a reversible Parkinsonian syndrome. Manganese dust, carbon disulfide, and carbon dioxide poisoning can also result in Parkinsonism. Use of the illicit meperedine-analogue Angel Dust can also cause Parkinsonism[9]. We earlier mentioned Parkinsonism as a cause for presyncopal dizziness due to orthostatic hypotension (Table 4).

Other Dizziness

As discussed above, other dizziness is dizziness described as a vague or floating sensation with the patient having difficulty describing the sensation[5]. Other dizziness may be associated with anxiety and depression. In fact, psychiatric disorders are considered the primary cause of this subtype of dizziness accounting for about 10 to 25% of all dizziness cases[5,21]. Yardley et al[58] found a significantly higher rate of psychiatric disorders in primary care patients who complained of dizziness as compared to age-matched controls who did not complain of dizziness. In older adults, anxiety, depression, and adjustment reactions were factors contributing to dizziness[59]. Tilting of the environment also falls under the *other dizziness* subtype.

Psychogenic Dizziness

We will discuss two types of psychiatric disorders frequently associated with dizziness: panic disorder and phobic postural vertigo.

Panic Disorder

Panic disorder is a chronic illness characterized by somatic and psychological complaints (Table 8)[16,60]. This disorder occurs in one of 75 persons worldwide and can be either inherited (incomplete autosomal dominance) or acquired[16]. It accounts for 15% of all medical visits and has been reported to average 10 different physician evaluations before it is correctly diagnosed[16]. Patients with panic disorder are thus seven times more likely to be high users of health care[16]. Work disability is also common in panic disorder[16]. Additionally, panic disorder is associated with significantly increased risk of suicide, increased cardiovascular morbidity, and if left untreated, increased occurrence of stroke[61]. Patients who suffer from panic disorder usually have an abrupt onset of fear or discomfort that peaks in approximately 10 minutes[16]. It is accompanied by at least four of the signs and symptoms listed in Table 8. Correct diagnosis also requires that panic attacks either recur unexpectedly every two weeks or that a single attack be followed by at least one month of the following symptoms[16]:

- Persistent concern about future attacks
- Worry that attacks will cause physical illness or insanity
- Significant changes in behavior related to the attacks

Table 8. Diagnosis of panic disorder[16,60].

Signs and symptoms: diagnosis requires four
• Sweating
• Rapid heart rate, palpitations, pounding heart
• Tremor
• Shortness of breath
• Feeling of choking
• Chest pain
• Nausea/abdominal distress
• Dizziness
• Lightheadedness
• Feeling of unreality
• Fear of losing control
• Fear of dying
• Paraesthesiae
• Hot flashes
Associated signs and symptoms
• Anxiety
• Depression
• Insomnia
• Chronic fatigue
• Gastroesophageal reflux

Phobic Postural Vertigo

Phobic postural vertigo may be the second most common cause of patient complaints of dizziness after BPPV, and it seems associated with somatization as well

as compulsive, depressive, and anxiety disorders[62]. The diagnostic criteria for phobic postural vertigo consist of[62]:

- Vertigo and subjective complaints of dysequilibrium in the absence of findings from neurologic or balance examination
- Description of fluctuating dysequilibrium with standing or walking and paroxysmal fear of falling without actual falls
- Description of feelings of anxiety and sympathetic symptoms during or shortly after attacks or vertigo without accompanying anxiety
- Provocation of dizziness in situations commonly implicated in other phobic syndromes (e.g., crossing bridges, driving a vehicle, being in empty spaces or among large numbers of people)
- Premorbid compulsive personality traits and depressive characteristics
- Initial onset of symptoms related to vestibulopathy or external stressors

Tilting of the Environment

Tilting of the environment is a rare form of dizziness attributed to otolith dysfunction. The function of the otoliths (utricle and saccule) is to provide sensory information on linear motion and acceleration in the horizontal and vertical directions, respectively. They also provide information on static head tilt due to the presence of the otoconia. Tilting of the environment is probably caused by an imbalance of otolith signals due to unilateral vestibular loss and is enhanced when rapid perturbations of posture make somatosensory cues difficult to interpret[63]. This asymmetry in otolith input to the vestibular nuclei causes the individual to sense a tilt of the environment to the side of the involved inner ear[63,64]. As the symptom is tilting of the environment and not spinning or rotating, this asymmetry of otolith input is not classified under vertigo, despite it being a vestibular disorder. The symptom of tilting of the environment may be rare due to central compensation and substitution from the visual and somatosensory systems[65]. Additionally, roll tilt illusion and linear acceleration perception deficit due to unilateral vestibular loss are generally experienced only within the first few hours or up to one week following onset[65].

Tilting of the environment is another form of dizziness that tends to challenge the diagnostic classification system introduced in this article. Ischaemia or infarction in the vertebrobasilar system and its branches unilaterally affecting the vestibular nuclei, the medial longitudinal fascicle, other nuclei involved in the vestibular mechanism, or the thalamus can also result in a patient reporting a subjective tilt of the visual vertical axis in a frontal plane[66]. We will discuss the differential diagnostic characteristics in the second article in this series.

Conclusion

Dizziness is a frequent complaint in patients presenting to orthopaedic physical therapists but may pose a diagnostic dilemma. Comprehensive diagnosis of the cause of dizziness requires that, as orthopaedic physical therapists, we leave our "comfort zone" of the musculoskeletal system and include vestibular, cardiovascular, neurological, metabolic, and psychiatric conditions in our diagnostic considerations. In this first article of the series, we have introduced a diagnostic classification system based on symptomatology and pathophysiology. It is obvious that the diagnosis of most causes of dizziness discussed here falls outside the scope of practice of physical therapy. However, medical screening to identify indications for medical referral or contra-indications to PT interventions is a required part of a PT patient evaluation[67]. This first article provides the knowledge required for PT diagnosis, screening, and subsequent appropriate PT management or medical referral. The second article in this series translates this knowledge into clinically useful information for the orthopaedic physical therapist by discussing history, tests, and measures for the patient presenting to PT with a complaint of dizziness. ■

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