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Hemodynamic orthostatic dizziness/vertigo: Diagnostic criteria

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Abstract. This paper presents the diagnostic criteria for hemodynamic orthostatic dizziness/vertigo to be included in the International Classification of Vestibular Disorders (ICVD). The aim of defining diagnostic criteria of hemodynamic orthostatic dizziness/vertigo is to help clinicians to understand the terminology related to orthostatic dizziness/vertigo and to distinguish orthostatic dizziness/vertigo due to global brain hypoperfusion from that caused by other etiologies. Diagnosis of hemodynamic orthostatic dizziness/vertigo requires: A) five or more episodes of dizziness, unsteadiness or vertigo triggered by arising or present during upright position, which subsides by sitting or lying down; B) orthostatic hypotension, postural tachycardia syndrome or syncope documented on standing or during head-up tilt test; and C) not better accounted for by another disease or disorder. Probable hemodynamic orthostatic dizziness/vertigo is defined as follows: A) five or more episodes of dizziness, unsteadiness or vertigo triggered by arising or present during upright position, which subsides by sitting or lying down; B) at least one of the following accompanying symptoms: generalized weakness/tiredness, difficulty in thinking/concentrating, blurred vision, and tachycardia/palpitations; and C) not better accounted for by another disease or disorder. These diagnostic criteria have been derived by expert consensus from an extensive review of 90 years of research on hemodynamic orthostatic dizziness/vertigo, postural hypotension or tachycardia, and autonomic dizziness. Measurements

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of orthostatic blood pressure and heart rate are important for the screening and documentation of orthostatic hypotension or postural tachycardia syndrome to establish the diagnosis of hemodynamic orthostatic dizziness/vertigo.

Keywords: Orthostatic vertigo, classification, orthostatic dizziness, hemodynamic, autonomic dysfunction, orthostatic hypotension, postural tachycardia syndrome, Bárány Society

1. Introduction

The Greek term "orthostasis" means upright posture [1]. Dizziness, unsteadiness or vertigo on standing up is a common symptom [2]. Orthostatic dizziness, unsteadiness or vertigo is also one of the symptoms of dysautonomia [3]. Orthostatic dizziness commonly occurs in the context of orthostatic hypotension (OH) or postural tachycardia syndrome (POTS) [4]. It has been nearly a hundred years since orthostatic dizziness was recognized. Bradbury and Eggleston first described the clinical syndrome of "postural hypotension" in 1925 [5]. They reported presyncopal dizziness on standing along with OH in patients with autonomic failure. Later, it was defined as a sustained fall in blood pressure on standing up, frequently accompanied by dizziness, fainting and other symptoms [6]. Even though orthostatic dizziness is a common condition, diagnosis of orthostatic dizziness due to global cerebral hypoperfusion (hemodynamic) may be demanding since the presentation is diverse and the sensitivity and specificity of the diagnostic tests are still unsatisfactory. The differential diagnosis of hemodynamic orthostatic dizziness/vertigo is important because it may have serious causes such as hypovolemia due to bleeding or autonomic disorders. The aim of defining diagnostic criteria for hemodynamic orthostatic dizziness/vertigo is to help clinicians to understand the related terminology and to distinguish it from other causes. Since orthostatic dizziness literally refers to dizziness in the upright (orthostatic) position, dizziness while upright due to bilateral vestibulopathy, orthostatic tremor, peripheral neuropathy or other clinical or subclinical gait disorders also can be termed, by definition, as 'orthostatic dizziness'[7, 8] (Table 1). In this regard, we emphasize that the diagnostic criteria developed in this paper are only for the orthostatic dizziness/vertigo of hemodynamic origin.

It should be noted that hemodynamic orthostatic dizziness/vertigo is distinct from head motion-induced or positional dizziness/vertigo, usually due to peripheral or central vestibular disorders. Hemodynamic orthostatic dizziness/vertigo has received relatively little attention among neuro-otologists compared to other causes of dizziness (e.g. vestibular) because the symptoms include various systemic features and its pathophysiology and treatments differ from those of vestibular disorders.

Table 1
Differential diagnosis of orthostatic dizziness/vertigo

Hemodynamic orthostatic dizziness
Orthostatic hypotension
Postural tachycardia syndrome
Syncope
Benign paroxysmal positional vertigo
Persistent postural-perceptual dizziness
Anxiety and depressive disorders
Bilateral vestibulopathy
Primary orthostatic tremor
Sensory neuropathy
Gait disorders
Dizziness/vertigo due to cardiac problems

The diagnostic criteria for hemodynamic orthostatic dizziness/vertigo have mostly required symptoms such as non-spinning dizziness, lightheadedness, and feeling of impending blackout or faint in the absence of spinning and positional vertigo. Oscillopsia and spontaneous episodic imbalance have been excluded from the symptoms of orthostatic dizziness [9]. However, OH can also induce orthostatic vertigo (i.e., spinning or other kinds of self-motion sensations) and, indeed, this has been reported in patients with poor autonomic regulation [3, 10].

2. Terminology

Orthostatic dizziness/vertigo refers to dizziness, unsteadiness or vertigo that is present in the upright position only or, more specifically, that develops on rising from a sitting to a standing, or from lying to a sitting or standing position [11]. If the symptoms are initiated while supine, then the term orthostatic dizziness/vertigo is not appropriate. The term hemodynamic orthostatic dizziness/vertigo is thus limited to orthostatic dizziness, unsteadiness or vertigo that occurs due to hemodynamic changes on arising to sitting or standing.

Autonomic dizziness/vertigo in contrast implies a more specific etiology and should be restricted

to dizziness/vertigo due to autonomic causes even though it usually occurs during orthostasis.

Orthostatic hypotension (OH) is defined by a significant reduction in systolic (>20 mmHg) and/or diastolic (>10 mmHg) blood pressure within 3 minutes upon standing from sitting or during head-up tilt test [12]. It may cause orthostatic dizziness/vertigo or not. Although the most common cause of orthostatic dizziness/vertigo is probably OH, it is not the only cause. Thus, the nomenclature, orthostatic dizziness/vertigo and OH, should be used distinctively. Orthostatic dizziness/vertigo is a symptom while OH is a disorder, a mechanism or an etiology; for instance, POTS can cause orthostatic dizziness without OH.

In contrast, *orthostatic intolerance* is a generic term and has a wider meaning. This is loosely used to describe symptoms occurring upon standing and relieved by recumbence [13–16]. Orthostatic intolerance may also be applied to the symptoms other than dizziness, such as headache, visual blurring, impending fainting sensation, palpitations, or shortness of breath that may be present during sitting or standing.

Patients with *presyncopal dizziness* may have a feeling similar to orthostatic dizziness, [2] but, the term "presyncope" implies a prodromal symptom of syncope and may occur in any position.

Exertional and postprandial dizziness are specific types of autonomic dizziness that develop in those special circumstances [17, 18]. Patients with exertional or postprandial dizziness frequently present with orthostatic dizziness.

Positional vertigo indicates dizziness/vertigo that appears when the head position is changed with respect to gravity, as in benign paroxysmal positional vertigo (BPPV) and central positional nystagmus [19, 20].

Postural dizziness refers to the dizziness that occurs during postural changes and does not necessarily indicate orthostatic dizziness; it is a non-specific term because it can be equally applied to orthostatic or positional dizziness/vertigo. Therefore, use of this term without additional discrimination is not recommended.

3. Methods

A principal aim of defining hemodynamic orthostatic dizziness/vertigo is to introduce and establish standardized criteria for the diagnosis and to avoid the confusing use of similar terms among neuro-otologists. At the XXIX Bárány Society Meeting 2016 in Seoul, South Korea, the General Assembly decided to launch an initiative to elaborate a classification of orthostatic dizziness/vertigo. A small working group of clinicians formed a Classification Subcommittee and began to draft the concept of the approach, reviewing and analyzing preexisting references. The Classification of Vestibular Symptoms provided the terminological foundation of the classification process [11]. Even if hemodynamic orthostatic dizziness/vertigo does not primarily arise from disturbances of the vestibular system, it was agreed that the classifications should cover all principal dizziness symptoms regardless of their etiology and topography. The classification project of the Bárány Society gradually involved the members and opinion leaders worldwide, mainly through electronic communications as well as several in-person meetings and phone conferences. The task was to make the best compromise among traditional use of terms, modern developments, and practical application both in research and clinical settings. The diagnostic criteria for hemodynamic orthostatic dizziness/vertigo have been developed iteratively over a two-year period (2016–2018) through discussion, presentation, and refinement.

4. Diagnostic criteria for hemodynamic orthostatic dizziness/vertigo

4.1. Hemodynamic orthostatic dizziness/vertigo

Criteria A-C should be fulfilled to make the diagnosis of hemodynamic orthostatic dizziness/vertigo.

A. Five or more episodes of dizziness, unsteadiness or vertigo triggered by arising (i.e. a change of body posture from lying to sitting/standing or sitting to standing), or present during upright position, which subsides by sitting or lying down[1),2)]
B. OH, POTS or syncope documented on standing or during head-up tilt test[3)]
C. Not better accounted for by another disease or disorder

4.2. Probable hemodynamic orthostatic dizziness/vertigo

Criteria A-C should be fulfilled to make a diagnosis of probable hemodynamic orthostatic dizziness/vertigo.

A. Five or more episodes of dizziness, unsteadiness or vertigo triggered by arising (i.e. a change of body posture from lying to sitting/standing or sitting to standing), or present during upright position, which subsides by sitting or lying down
B. At least one of the following accompanying symptoms[4)]
   – generalized weakness or tiredness
   – difficulty in thinking or concentrating
   – blurred vision
   – tachycardia or palpitations
C. Not better accounted for by another disease or disorder

4.3. Previously used terms

Orthostatic dizziness/vertigo, postural dizziness/vertigo, exertional dizziness/vertigo, presyncopal dizziness.

4.4. Notes

1) According to the Classification of the Vestibular symptoms of the ICVD, [11] orthostatic dizziness/vertigo is defined as dizziness, unsteadiness or vertigo triggered by and occurring on arising (i.e. a change of body posture from lying to sitting or sitting to standing). Orthostatic dizziness/vertigo should be distinguished from positional dizziness/vertigo (triggered by a change in head position relative to gravity) and head motion-induced dizziness/vertigo since positional symptoms may be triggered by the head motion that occurs during arising. The distinction between positional and orthostatic dizziness/vertigo can be accomplished by asking the patient with dizziness, unsteadiness or vertigo on arising whether the symptoms also occur on lying down or turning over in bed; if so, the symptoms are more likely positional rather than orthostatic.

Unsteadiness may be an autonomic symptom after sitting or standing, so it should be included in the symptoms of hemodynamic orthostatic dizziness/vertigo even though it is classified as a postural symptom while upright (e.g., standing), rather than the one linked to changing body posture with respect to gravity (e.g., standing up) in the Classification of Vestibular Symptoms.

2) The duration of episodes is variable. In a previous proposal for criteria of orthostatic dizziness, the duration of dizzy spells was defined as seconds to several minutes [9]. Patients with neurogenic OH usually can stand for only a few minutes and have to sit or lie back to avoid syncope [21]. Patients with initial orthostatic dizziness become dizzy right after standing for seconds [22]. However, patients with POTS usually report orthostatic symptoms lasting as long as they are upright.

3) Criteria for OH, POTS, and vasovagal syncope

Measurements of orthostatic blood pressure and heart rate are most important in screening for autonomic dysfunction. However, the results of head-up tilt test usually do not show a good correlation with orthostatic symptoms. According to prior studies [23, 24], negative results are much more reproducible than positive ones (about 95% and 50% respectively). The reproducibility of head-up tilt test depends strongly on population selection as positive results increase in patients with severe and frequent orthostatic symptoms [25].

OH is defined as a sustained reduction of systolic blood pressure of at least 20 mmHg or diastolic blood pressure of 10 mmHg within 3 minutes of standing or during head-up tilt test [12]. Neurogenic OH results from sympathetic adrenergic failure and usually shows a drop of systolic blood pressure of at least 30 mmHg or diastolic blood pressure of at least 15 mmHg within 3 minutes of standing or during head-up tilt test [21]. Sometimes, patients may develop delayed OH, which is defined as a sustained fall of blood pressure (systolic $\geq$ 20 mmHg or diastolic $\geq$ 10 mmHg) occurring later than 3 minutes after standing or head-up tilt test [26]. Delayed OH is associated with milder abnormalities of sympathetic adrenergic function and also is a frequent cause of orthostatic dizziness [26]. In contrast, initial OH is defined as a transient blood pressure decrease (systolic $>$ 40 mmHg or diastolic $>$ 20 mmHg) within 15 seconds of standing, which may be a common but under-recognized cause of syncope [12].

POTS is characterized by a sustained heart rate increase of at least 30 beats per minute or a heart rate of 120 beats per minute or more within 10 minutes of standing or during head-up tilt test in the absence of OH [12]. For individuals aged 12–19 years, the minimum increment required for diagnosis is 40 beats per minute [12].

Vasovagal syncope (neurocardiogenic syncope) is caused by an autonomic reflex which involves cessation of sympathetic vascular tone and vagal activation resulting in a drop of blood pressure and/or heart rate. It is provoked by prolonged standing or specific situational stimuli such as venipuncture or the sight

of blood. Typically vasovagal syncope is preceded by prodromal symptoms and signs such as pallor, diaphoresis, nausea, abdominal discomfort, yawning, sighing, and hyperventilation that may occur up to 60 seconds prior to loss of consciousness [12].

4) Other orthostatic symptoms accompanied by orthostatic dizziness/vertigo

The criteria for probable hemodynamic orthostatic dizziness/vertigo can be applied to patients with dizziness that occurs during orthostasis but without evidence of OH or POTS. Even in a well-defined group of patients with orthostatic symptoms and documented OH, reproducibility of OH with head-up tilt test is relatively low [27]. Thus, other symptoms of OH and POTS will be helpful to diagnose orthostatic dizziness/vertigo of hemodynamic causes if OH or POTS cannot be documented. Apart from dizziness, unsteadiness or vertigo, the most common orthostatic symptoms are weakness, cognitive impairment, and blurred vision [28]. Weakness generally affects the legs or has a diffuse pattern of involvement. Cognitive difficulties, such as interference with thinking and concentrating, are prominent in older patients. Patients sometimes use phrases such as "I feel goofy or silly". Blurred vision and occasionally tunnel vision are also well-recognized complaints. Patients with autonomic failure report head and neck discomfort (coat-hanger headache) more frequently than controls. The discomfort is usually localized to the occiput, the nape of the neck and shoulders [29].

The most common aggravating factors of orthostatic dizziness/vertigo are physical exertion or exercise (53%) and environmental warming (32%) [3]. The types of physical activity that provoke symptoms are rather ordinary, for example, climbing stairs and doing housework. Environmental warming includes activities on a hot day, taking a hot shower, or immersion in a hot tub. Postprandial aggravation of symptoms is well recognized and occurs in 24% of patients [3]. In contrast, only 6% of patients report aggravation of symptoms during specific periods of their menstrual cycle [3].

5. Comments

5.1. *Epidemiology of hemodynamic orthostatic dizziness/vertigo*

Although orthostatic dizziness is a common symptom, its epidemiology has received little attention. In studies based on patients' history, the prevalence of orthostatic dizziness varied according to the age group investigated. It was estimated at 41% in healthy medical students and 57% in young females [30, 31]. In several larger community-based studies on subjects aged over 60 years, orthostatic dizziness was found in 2–30% [15, 32, 33]. In a population-based study [9] across a wide range of ages, the one-year and lifetime prevalence of orthostatic dizziness was 10.9 and 12.5%, respectively. Only a few studies have measured OH in association with orthostatic dizziness while standing in older individuals (over 65 years), and found orthostatic dizziness in 2 to 20% [32, 34–37]. A large population-based study on orthostatic dizziness during standing test in adults aged more than 20 years showed an overall prevalence of orthostatic dizziness of 4.8% [35]. However, in these studies, the quality of symptoms (dizziness versus vertigo) was not determined or vertigo was excluded from the symptoms of orthostatic dizziness.

In a study on 90 patients with OH confirmed with head-up tilt test, 88% of the patients experienced orthostatic dizziness during the testing, but 37% also experienced orthostatic vertigo [3]. Two studies have focused on the presence of vertigo during head-up tilt test in patients with orthostatic dizziness or related symptoms. These studies found orthostatic vertigo in 47% of patients with vasovagal (neurocardiogenic) syncope and in 29% of patients with orthostatic dizziness [10, 38]. Another study also found rotatory vertigo and nystagmus in approximately 30% of the patients with profound OH during the orthostatic challenging tests [39].

5.2. *Two common causes of hemodynamic orthostatic dizziness/vertigo: OH versus POTS*

Two common findings observed in patients with orthostatic dizziness/vertigo are decreased blood pressure (OH) or increased heart rate (POTS) on standing or during head-up tilt test [40]. OH may result from a sympathetic adrenergic failure mediating peripheral vasomotor responses due to an inadequate norepinephrine release from the sympathetic nerves (neurogenic OH) [12, 40, 41]. It is usually associated with diabetic or non-diabetic autonomic neuropathy, neurodegenerative diseases such as Parkinson's disease or multiple system atrophy, and primary autonomic failure [42]. However, there also are non-neurogenic causes such as drugs, hypovolemia, deconditioning, or systemic infection (non-neurogenic OH).

POTS is also a common cause of orthostatic intolerance and is defined by development of orthostatic symptoms in association with a heart rate increment of 30 or more beats per minute on assuming an upright posture [12]. The age of presentation of POTS is mostly between 15 and 50 years [43, 44]. Females predominate over males by 5:1 [28]. The pathophysiology of POTS is complex and heterogeneous. POTS may be ascribed to a partially denervated circulatory system, a hyperadrenergic state, hypovolemia, peripheral pooling of the blood, or chronic bed rest [40, 45]. Some patients with POTS have anti-ganglionic (α3) acetylcholine receptor antibody, suggesting a limited form of autoimmune autonomic neuropathy [46]. Hyperventilation and psychological factors may contribute to the pathophysiology of POTS [47, 48].

5.3. *Pathophysiology of hemodynamic orthostatic dizziness/vertigo*

Orthostatic dizziness/vertigo occurs in patients with OH when cerebral perfusion is critically impaired. Cerebral hypoperfusion develops when cerebral autoregulation fails in the face of a severe reduction in blood pressure [49]. In the autoregulated range of systolic blood pressure, which is typically between 80 and 150 mmHg, cerebral blood flow remains constant in spite of changes in blood pressure [21]. The symptoms of POTS are due to reduced cerebral perfusion or sympathetic activation. Since orthostatic dizziness in patients with POTS is similar to that observed in the OH group, orthostatic dizziness in POTS is presumably due to reduced cerebral perfusion [50]. Although orthostatic dizziness is believed to occur as a result of an acute decrease in cerebral blood flow, the mechanism of orthostatic vertigo is poorly understood. Vertigo is generally the result of a pathological asymmetry within the vestibular system. If the vestibular structures in the cerebellum or inner ear suffer from an asymmetric drop in perfusion pressure due to variations of the local vasculature, such a left–right asymmetry may produce a sense of rotation [51]. 'Global' hypotension may cause 'focal' transient ischemic attacks in the presence of flow-limiting vascular stenosis [52]. Especially in patients with a profound stenosis of the vertebral or proximal basilar artery, a smaller drop in BP may cause dizziness or vertigo. A recent study found rotatory vertigo and downbeat nystagmus during the orthostatic challenging in 30% of the patients with profound OH [39]. These results imply that orthostatic vertigo may result from cerebellar dysfunction due to hypoperfusion [53]. However, some patients exhibited mixed downbeat and horizontal nystagmus with or without a torsional component, which may be attributed to asymmetrical excitation of the vestibular system [54, 55] or by floccular disinhibition [56–58], both induced by transient hypoperfusion.

5.4. *Differential diagnosis of hemodynamic orthostatic dizziness/vertigo*

1) Benign paroxysmal positional vertigo (BPPV)

BPPV should be distinguished from orthostatic dizziness/vertigo. Vertical canal BPPV will produce symptoms not only on sitting up from the supine position but also on lying down from sitting. Patients with orthostatic dizziness/vertigo instead present symptoms only on arising but not during other positional changes [20]. Positional tests for BPPV should be performed in patients with orthostatic dizziness/vertigo even when their dizziness is not positional [59].

2) Persistent postural-perceptual dizziness (PPPD)

This recently defined condition is a very common form of functional (non-structural) dizziness [60]. PPPD manifests with one or more symptoms of dizziness, unsteadiness, or non-spinning vertigo that are present on most days for three months or more. Upright posture, active or passive movements, and exposure to moving or complex visual stimuli may exacerbate symptoms [61]. Because most patients with PPPD report more severe symptoms when standing or walking than sitting or lying down, dizziness in patients with PPPD may be confused with orthostatic dizziness [62, 63]. Differential diagnosis depends on changes in heart rate or blood pressure on standing from the supine position. Patients with hemodynamic orthostatic dizziness/vertigo tend to have more pronounced orthostatic and exertional dizziness than those with PPPD [64]. Patients with PPPD have dizziness elicited by complex or moving visual stimuli even in the supine or sitting position [61]. Patients with PPPD may not show a complete resolution of symptoms even when lying down. Somatosensory inputs such as touching fixed objects (furniture or walls), using gait aids, or holding onto other people alleviate symptoms in patients with PPPD but not in those with orthostatic dizziness [61]. However, orthostatic dizziness can trigger or co-exist with PPPD [64].

3) Chronic anxiety and depressive disorders

Lightheadedness with near fainting is suggestive of a diffuse decrease in cerebral blood flow that

may occur in cardiac arrhythmia or OH. However, lightheadedness also occurs with chronic anxiety due to generalized anxiety disorder, agoraphobia, social phobia, obsessive compulsive disorders, depression and traumatic stress disorders [65, 66]. Changes of blood pressure and heart rate indicative of OH or POTS during the position change are the key for differential diagnosis, but simple self-report questionnaires can offer a valid and efficient means of detecting a psychiatric morbidity [63, 67]. However, a positive screen for anxiety or depression does not rule out a hemodynamic or other causes of symptoms as psychiatric disorders often co-exist with medical morbidity.

4) Bilateral vestibulopathy

Bilateral vestibulopathy is a chronic vestibular syndrome characterized by postural imbalance and/or unsteadiness of gait secondary to vestibular hypofunction. Typically there are no symptoms when sitting or lying down under static conditions because patients do not rely very much on the vestibular system under these circumstances. Walking or quick head or body movements can induce blurred vision or oscillopsia in these patients [8]. Bilaterally reduced or absent angular VOR function is essential for diagnosis of bilateral vestibulopathy. Dizziness or unsteadiness worsens in darkness or on uneven ground in patients with bilateral vestibulopathy, but not in patients with orthostatic dizziness/vertigo [8].

5) Primary orthostatic tremor

Primary orthostatic tremor is characterized by unsteadiness on standing due to a high-frequency (14–18 Hz) tremor involving the legs or the arms on weight-bearing [68, 69]. Based on the functional imaging data, pathological ponto-cerebello-thalamo-primary motor cortical activations is believed to be the pathomechanism of primary orthostatic tremor [70]. Because hemodynamic orthostatic dizziness/vertigo can be combined with tremor during upright posture, orthostatic tremor needs to be included in the differential diagnosis of hemodynamic orthostatic dizziness/vertigo [7]. Both disorders can cause a feeling of imbalance while standing. However, the symptoms of orthostatic tremor rapidly improve on sitting or walking, and the need to sit down or to move maybe so strong that patients with orthostatic tremor even avoid the situations where they have to stand still [68]. Orthostatic tremor can be diagnosed in a few minutes with Fourier (frequency) analysis of the signals from a posturography platform [71] or superficial electromyography.

6) Sensory neuropathy

Although loss of balance may be out of proportion to the signs of peripheral neuropathy, most patients with large fiber peripheral neuropathy that is severe enough to cause unsteadiness will have distal paresthesia, sensory impairment and loss of ankle jerks. Nerve conduction and Romberg tests in addition to measurements of blood pressure and heart rate during position changes can discriminate between sensory neuropathy and hemodynamic orthostatic dizziness/vertigo. Dizziness due to postural imbalance and OH may coexist in neuropathic patients when sympathetic fibers of the vasculature are involved.

7) Gait disorders

Hemodynamic orthostatic dizziness/vertigo is a common cause of gait and balance problems and a major cause of falls, especially in the elderly. Patients with hemodynamic orthostatic dizziness/vertigo may report sensations of veering from side-to-side when walking. On examination, they may exhibit a mildly slow or cautious gait. These changes are correlated with reduced balance confidence. Lightheadedness, evoked on suddenly rising from a sitting or supine position, and relieved with sitting, can be helpful in identifying hemodynamic orthostatic dizziness/vertigo. However, parkinsonian gait or cerebellar ataxia can co-exist in patients with hemodynamic orthostatic dizziness/vertigo because Parkinson's disease and multiple system atrophy are the main causes of neurogenic OH. Small vessel white matter disease is also a common cause of gait disorder and orthostatic dizziness in the elderly [72].

8) Dizziness/vertigo due to cardiac problems

Vertigo may be present in more than a half of the patients with dizziness due to cardiovascular problems, and may be isolated [74]. About 10% of patients with acute myocardial infarction experience dizziness as a dominant or presenting symptom [73]. In this instance, dizziness and vertigo are hemodynamic but not necessarily orthostatic. Dizziness/vertigo due to cardiac problems may occur during exertion or when supine. Palpitations, chest discomfort or dyspnea may accompany the dizziness/vertigo. Patients may have a family history of unexplained sudden death at a young age, structural heart diseases, coronary artery diseases or arrhythmias [75].

5.5. Syncope

Hemodynamic orthostatic dizziness/vertigo may be followed by a loss of consciousness (i.e., syncope)

or not. If dizziness/vertigo is followed quickly by syncope, the differential diagnosis is narrowed down to hemodynamic causes. Three main types of syncope are reflex syncope, syncope due to OH and cardiogenic syncope [75]. Hemodynamic orthostatic dizziness/vertigo can be a prodromal symptom of reflex syncope or syncope due to OH. Dizziness or vertigo due to hemodynamic changes also can be a prodromal symptom of cardiogenic syncope, but this is not necessarily related to a position change. Syncope may happen without prodromal symptoms, especially in the presence of long-standing OH. Therefore, hemodynamic causes cannot be excluded from the etiology of syncope even when the patients have no preceding dizziness/vertigo.

5.6. *Cardiovascular autonomic regulation during orthostasis*

Standing up from a sitting or supine position causes a gravitational redistribution of the blood volume and a pooling of 300–800 ml of the blood in the lower extremities and splanchnic venous capacitance system. This fluid shift can lead to decreases in the venous return, stroke volume and cardiac output. In response to these changes, sympathetic outflow to the heart and blood vessels increases and cardiac vagal nerve activity decreases. These autonomic adjustments increase vascular tone, heart rate and cardiac contractility, and stabilize arterial pressure. During standing, contraction of the lower body skeletal muscles prevents excessive pooling and augments venous return to the heart. Thus, orthostasis can ultimately lead to lightheadedness, dizziness, vertigo or even syncope if rapid autonomic adjustments do not occur. The most recognized autonomic reflex engaged during orthostasis is the baroreflex [76]. Unloading either the arterial or cardiopulmonary baroreceptors leads to an increase in heart rate, muscular sympathetic nerve activity, and vasoconstriction in the extremities [77, 78].

5.7. *Roles of the vestibular system in autonomic regulation*

The vestibular system is an important regulator of the autonomic nervous system activity, and is involved in postural-related adjustments of blood pressure. Stimulation of the labyrinthine receptors alters the firing of sympathetic efferents for vasoconstriction [79], and modifies blood flow through the arterial vascular beds [80]. Accordingly, bilateral labyrinthectomy leads to a drop of blood pressure at the onset of head-up tilts in conscious animal models. However, this impairment is transient [81, 82]. In contrast, central vestibular lesions can produce a prolonged impairment in posture-related cardiovascular responses [82]. Although ablation of the posterior cerebellar vermis did not affect regulation of blood pressure, combination of damage to the cerebellar uvula along with bilateral labyrinthectomy resulted in hypotension during head-up rotations in cats [83]. The deficits in adjusting blood pressure remained one month after the removal of vestibular inputs when the experiment was terminated [83]. These findings led to the conclusion that the plasticity within the central nervous system is responsible for recovery of cardiovascular responses after damage to the peripheral vestibular system, and that occurrence of this adaptation depends upon the integrity of the cerebellar uvula. The "autonomic region" of the vestibular nuclear complex is comprised of the portions of the medial and inferior vestibular nuclei located caudal to the lateral vestibular nucleus [84, 85]. Lesions involving these vestibular nuclei produce a permanent loss of the capacity to rapidly adjust blood pressure during head-up tilts in cats [86]. The rostral ventrolateral medulla plays a predominant role in controlling blood pressure [87, 88]. Stimulation of the rostral ventrolateral medulla produces a large increase in blood pressure [87, 89], and the activity of rostral ventrolateral medulla neurons is inhibited by activation of the baroreceptors [90, 91]. Furthermore, bilateral destruction or inhibition of this region produces a profound drop in blood pressure, similar to that observed after transection of the cervical spinal cord [92, 93], and also eliminates baroreceptor reflexes [94, 95].

5.8. *Vestibular disorders can cause hemodynamic orthostatic dizziness/vertigo*

Patients with absent cervical vestibular evoked myogenic potentials (cVEMP) show a higher incidence of OH upon active standing compared to those with normal cVEMP [96]. This suggests a role of the otolithic organs in cardiovascular autonomic function. Acute unilateral peripheral vestibulopathy (vestibular neuritis) also impairs cardiovascular autonomic responses during postural changes [97–99]. These patients may show symptomatic POTS [98] and OH [99], and no increase of low-to-high frequency ratio in the heart rate variability test while standing [97]. These findings are, however, prominent only during the acute period.

Tumarkin attacks may be complicated by syncope. A false otolith input appears to activate an erroneous vestibular sympathetic reflex, leading to paradoxical inactivation of the baroreflex, and resulting in syncope that mimics a vasovagal attack [100]. Patients with BPPV occasionally experience non-specific dizziness with postural lightheadedness, especially when rising from sitting, despite successful removal of detached otolith particles with appropriate canal repositioning procedures [101, 102]. This residual dizziness is similar to orthostatic dizziness reported by patients with OH [103]. In a recent study [104], the incidence of OH was significantly higher in patients with residual dizziness than in those without residual dizziness. Furthermore, patients with residual dizziness showed a larger fall in systolic blood pressure during the Valsalva and head-up tilt test than those without residual dizziness. The authors suggested that residual dizziness after successful treatments of BPPV may be partly associated with sympathetic neural autonomic dysfunction. Another study showed that a dysfunctional otolith system causes a blood pressure drop while standing up in astronauts who just returned from space. The authors concluded that an intact otolith system plays an important role in preventing blood pressure instability during orthostatic challenges. These findings indicate a link between the otolith system and blood pressure control (vestibulosympathetic reflex) [105] although the vestibular system also seems involved in the control of heart rate [106].

Several studies have described OH in association with isolated cerebellar lesions in humans [107–109]. OH was found in 31% of the patients (9/29) with isolated cerebellar lesions and the most common pattern of OH was the transient OH right after tilting (7/9, 80%). The medial part of the superior semilunar lobule and the tonsil were more commonly involved in the OH group [109]. Cardiovascular responses during orthostasis in cerebellar lesions require further elucidation.

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