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Orthostatic Hypotension in Hypertensive Adults

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Abstract

Orthostatic hypotension affects roughly 10% of individuals with hypertension and is associated with a number of adverse health outcomes, including dementia, cardiovascular disease, stroke, and death. Among adults with hypertension, orthostatic hypotension has also been shown to predict patterns of blood pressure dysregulation that may not be appreciated in the office setting, including nocturnal non-dipping. Individuals with uncontrolled hypertension are at particular risk of orthostatic hypotension and may meet diagnostic criteria for the condition with a smaller relative reduction in blood pressure compared with normotensive individuals. Antihypertensive medications are commonly de-prescribed to address orthostatic hypotension; however, this approach may worsen supine or seated hypertension, which may be an important driver of adverse events in this population. There is significant variability between guidelines for the diagnosis of orthostatic hypotension with regards to timing and position of blood pressure measurements. Clinically relevant orthostatic hypotension may be missed when standing measurements are delayed or when taken after a seated rather than supine position. The treatment of orthostatic hypotension in patients with hypertension poses a significant management challenge for clinicians; however, recent evidence suggests that intensive blood pressure control may reduce the risk of orthostatic hypotension. A detailed characterization of blood pressure variability is essential to tailoring a treatment plan and can be accomplished using both in-office and out-of-office monitoring.

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I. Introduction

Orthostatic hypotension (OH) is relatively common among individuals with hypertension, affecting about 10% of hypertensive adults.[1–3] Individuals with OH and hypertension face a number of important health concerns related to both conditions, including increased risk of falls, syncope, dementia, cardiovascular disease (CVD), stroke, and mortality.[4–6] The treatment of patients with concurrent OH and hypertension is a particular challenge as treatments for OH may exacerbate hypertension. In this review, we discuss the physiology of OH in hypertensive patients, subtypes and causes of OH, and management approaches to treat OH in the hypertensive patient.

II. Hypertension and Blood Pressure Dysregulation

Blood pressure (BP) fluctuates in response to environmental (i.e., altitude, ambient temperature), physical (i.e., posture, volume status, diet), and emotional (i.e., stress) factors.[7] BP variability (BPV) is a term used to describe the magnitude and patterns of BP changes over time, from the short-term (between beats, minutes, hours, or day-to-night changes) to the long-term (over days, weeks, clinic visits, or years).[7,8] Greater long-term variability in systolic BP (SBP) is associated with higher risk of all-cause mortality, fatal and nonfatal CVD, and stroke independent of mean BP.[9] BPV is greater in patients with hypertension than those without hypertension and increases with higher BP.[10] Orthostatic hypotension, also called postural hypotension, may be considered a form of BP dysregulation and a manifestation of BPV.[11] Along these lines, OH has been shown to be useful in predicting hypotensive events[12] as well as patterns of BPV outside of the clinic among adults with hypertension.[13–16]

III. Orthostatic Hypotension as a Clinical Assessment Predicting Hypotensive Events and Ambulatory Blood Pressure Phenotypes

OH is defined by various guidelines as a decrease in SBP by ≥20 mm Hg or a decrease in diastolic BP (DBP) by ≥10 mm Hg when an individual stands from a seated or supine position.[17–19] During the normal physiological response to standing, baroreceptors in the carotid sinus and the aortic arch sense the sudden decrease in BP due to venous pooling and respond by increasing sympathetic tone and decreasing parasympathetic tone. The influx of sympathetic tone results in increased peripheral vascular resistance leading to an increase in venous return and BP augmentation.[20] The withdrawal of parasympathetic tone results in augmentation of heart rate.[20] OH results from an insufficient physiologic response to the decreased venous return upon standing, and can be broadly classified as neurogenic or non-neurogenic. Non-neurogenic OH is far more common and may be caused by various etiologies, including intravascular volume depletion, medications, CVD, and prolonged recumbency or deconditioning.[20] While rare in the general population, neurogenic OH is relatively common among patients with neurodegenerative disorders (i.e., Parkinson disease, multiple system atrophy, Lewy body dementia, and pure autonomic failure) or small-fiber neuropathies, including diabetes mellitus or B12 deficiency.[21] A less common etiology of OH is baroreceptor failure which occurs due to bilateral disruption of baroreceptor function and can result from neck radiation, surgery, or trauma.[22]

OH is a predictor of hypotensive events among adults with treated hypertension. In a secondary analysis of the Systolic Blood Pressure Intervention Trial (SPRINT), adults with OH had nearly a two-fold higher risk of hypotension-related hospitalizations or emergency department visits, which was independent of assigned treatment goal (i.e. intensive BP control [< 120 mm Hg] versus standard BP control [<140 mm Hg]).[12]

OH also associates with distinct ambulatory BP phenotypes.[14,16] In a subgroup analysis of 849 adults from SPRINT, OH was found to be associated with white coat hypertension (a state where BP measured in the clinic are higher than BP measured at home) and nocturnal elevations in BP.[13]

IV. Orthostatic Hypotension and Hypertension

The term OH may itself be a misnomer, implying hypotension upon standing. However, given that OH is derived from two BP measurements, a supine/seated measurement and a standing measurement, having an elevated supine/seated BP can meet criterion for OH even in absence of standing hypotension.

It should thus come as little surprise that OH is more frequently observed among those with hypertension.[23,24] In observational studies, approximately 1 in 10 individuals with hypertension have OH[1–3] and those with uncontrolled hypertension are at higher risk for OH.[2,25] There may be multiple reasons for this observation. First, altered BP regulatory mechanisms and autonomic dysfunction underpin both OH and hypertension.[20,26] Second, hypertension itself may blunt sympathetic nervous system regulation of BP.[27] Third, certain antihypertensive drug classes are associated with OH.[28] Fourth, increased age and clinical comorbidities such as heart failure, diabetes mellitus, and chronic kidney disease are associated with both conditions. Fifth, those with hypertension may meet diagnostic criteria for OH based on absolute reductions in BP (i.e. mm Hg) with smaller relative reduction (i.e. percent decrease) in BP. Sixth, cerebral autoregulation which normally protects the brain from transient hypotension is impaired by hypertension and associated cerebrovascular disease.[29] As such, hypertensive individuals may develop symptoms of cerebral hypoperfusion during standing at blood pressure values typically considered normal.[30] Last, vascular remodeling, CVD, and stroke are all adverse complications of hypertension that are related to OH.[31]

Individuals with both hypertension and OH pose a particular treatment challenge, as OH carries an increased risk of falls, syncope, and dementia while under-treatment of hypertension is associated with CVD, stroke, and all-cause mortality.[4–6] A common approach to treating OH is to recommend increased salt and fluid consumption, deprescribe antihypertensives, and initiate mineralocorticoids for BP augmentation. These approaches operate under the assumption that the adverse events caused by OH are secondary to organ hypoperfusion and injury. However, it is possible that the impetus of adverse events in hypertensive patients with OH is supine/seated hypertension, not the standing BP or even the change between the two.

Supine Hypertension

Supine hypertension is defined by the American Autonomic Society and the European Federation of Autonomic Societies as an SBP of 140 mm Hg or greater and DBP of 90 mm Hg or greater while in the supine position, and is physiologically linked to OH.[32,33] Orthostatic episodes during the day can activate the renin-angiotensin-aldosterone pathway and exacerbate supine hypertension.[34] Meanwhile, supine hypertension causes significant nocturnal diuresis, which can worsen daytime orthostasis.[32] Non-pharmacologic strategies to decrease supine hypertension include avoiding the supine position during the day and elevating the head of the bed at night;[35] although evidence in support of these maneuvers is limited. Pharmacologic treatment of supine hypertension is challenging and should be balanced against the potential exacerbation of morning hypotensive episodes, which can have immediate consequences such as orthostatic symptoms and falls.[36] Certain antihypertensives including captopril, clonidine, overnight nitroglycerine patches, and short-acting nifedipine have been trialed to reduce nocturnal hypertension.[37–39]

While there is uncertainty whether supine hypertension without seated hypertension requires treatment, particularly in cases with OH in which treatment can exacerbate orthostasis, supine hypertension is associated with adverse outcomes. The Irish Longitudinal Study on Ageing (TILDA) of 1500 community-dwelling adults found that orthostasis with co-existing hypertension either in the seated/supine position was significantly associated with a higher risk of falls and syncope, though the effect size was notably larger for seated compared to supine hypertension.[40] Studies of patients with autonomic dysfunction have found that those with supine hypertension had worse renal function, higher rates of left ventricular hypertrophy, and earlier incidence of CVD and all-cause mortality than those without supine hypertension.[41,42]

V. Measurement Considerations

Measurement Timing

While guidelines generally recommend measuring BP in the seated or recumbent position after a period of 5 minutes,[17,43] there is substantial heterogeneity with respect to the timing of standing OH measurements across guidelines with the majority focused on delayed measurements within 1–3 minutes after standing (Table).[17,18,43–48] Despite these recommendations, hypotension is more frequently detected when measured at earlier intervals upon standing. One study of 8,908 individuals found a prevalence of OH immediately after standing of 12.3% and at 2 minutes of standing of 2.9%.[49] ‘Initial OH,’ i.e., a transient decrease in SBP of >40 mm Hg within 15 s of standing regardless of symptoms of orthostatic intolerance,[50,51] is often missed by traditional automated or auscultatory BP measurements and may be more readily captured by self-reported symptoms of light-headedness, visual disturbances, and presyncope.[52] ‘Delayed recovery OH’, OH that resolves by 2–3 minutes after standing, is yet another OH phenotype often missed with delayed assessments.[35] One observational study found that OH measurements performed within 1 minute of standing were associated with dizziness and adverse outcomes including fall, fracture, syncope, mortality, and motor vehicle accidents,[53] suggesting that delayed OH assessments may miss important clinical information with respect to concurrent symptoms

and long-term risks. Furthermore, an analysis of data from the African American Study of Kidney Disease and Hypertension (AASK) trial found that consensus definitions of OH yielded high specificity for OH symptoms but low sensitivity.[54]

Starting Position: Supine Versus Seated

Guidelines have conflicted with regards to the starting position for OH assessments (Table). The American College of Cardiology /American Heart Association (ACC/AHA) guidelines describe OH being performed from the seated to standing position.[17] In contrast, the autonomic society consensus recommends recumbent to standing or tilt table testing with respect to OH assessments.[45] Utilization of seated versus supine initial positions can alter the detection of OH. In a study of older adults age 70 years and older, where nearly two thirds had hypertension, OH was diagnosed more frequently when using supine assessments (14.8%) compared to seated assessments (2.2%) and the two protocols were not interchangeable based on both their observed physiologic responses and their associations with clinical symptoms and falls.[55] Similarly, in study of 831 patients presenting to a neurology clinic, Shaw et al found that using a lower cutoff point (i.e., SBP drop of >15 mm Hg or a DBP of >7 mm Hg) to define OH from a seated position yielded a comparable sensitivity relative to standard cutoff points (i.e. SBP drop ≥20 mmHg or DBP drop ≥10 mmHg) to define OH from a supine position.[56] These findings have important clinical implications, as detection of OH using seated assessments can result in an underestimation of the prevalence of clinically relevant OH compared to supine assessments.

VI. Management

Traditional Approaches

Management of OH generally begins with an appraisal of potential precipitants or exacerbators of OH. Patients should be screened for etiologies of OH such as neurodegenerative diseases, diabetes mellitus, B12 deficiency, renal failure, rheumatologic or autoimmune conditions, dehydration, prolonged recumbency, deconditioning, and medications.[19,57] Evaluation for whether medications that contribute to orthostasis can be withdrawn or replaced is warranted. The medications most strongly associated with OH include alpha blockers, beta blockers, tricyclic antidepressants, nitrates, phosphodiesterase-5 inhibitors, and antipsychotics.[19,23,58,59] Several non-pharmacologic interventions may improve orthostatic symptoms, including advising patients to change position slowly, maintaining adequate hydration, and avoiding alcohol and large meals.[19] Countermeasure maneuvers such as crossing legs while standing have evidence for improving orthostasis in a placebo-controlled crossover study.[60] Abdominal compression binders have also been shown to increase upright blood pressure in small studies.[61,62]

When non-pharmacologic therapies are ineffective, pharmacologic therapies such as midodrine, droxidopa, or fludrocortisone may be trialed, with the goal being to relieve symptoms rather than focusing on a specific blood pressure target. Midodrine is a prodrug of a sympathomimetic agent and is approved by the Food & Drug Administration (FDA) for symptomatic OH.[63] Midodrine should be used with caution in patients with heart failure or renal disease and should also be avoided within 3–4 hours of lying down to prevent

supine hypertension.[32] Droxidopa, a prodrug of noradrenaline, is approved by the FDA for patients with neurogenic OH. While droxidopa appears to be less likely to cause supine hypertension than midodrine, the use of droxidopa in patients with non-neurogenic OH is less well-established.[64] Fludrocortisone is a synthetic adrenocortical steroid that is often used off-label for OH but its effects on positional blood pressure changes and orthostatic symptoms is uncertain.[65] Fludrocortisone poses particular risk to patients with CVD and is generally avoided in patients with heart failure or supine hypertension.[66] The addition of pressor agents in patients with concomitant OH and hypertension should be avoided to prevent exacerbating hypertensive episodes; however, if pharmacologic therapy is necessary to improve hypotensive symptoms, midodrine is preferred due to its short half-life as well as the stronger evidence base for symptomatic improvement compared with the other available medications.

Pharmacologic Treatment of Hypertension and OH

While some observational data support the traditional view that antihypertensive therapies increase the risk of the OH,[23,67] a number of clinical studies have demonstrated that targeting lower BP goals does not increase risk of OH. In a study of 702 adults aged 70 and older, OH prevalence was found to be higher in adults with uncontrolled rather than controlled hypertension (19% vs 5%; P <0.001).[25] In 4266 patients of the Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial, the incidence of OH was unrelated to randomization into the intensive (<120 mm Hg) vs standard (<140 mm Hg) blood pressure arm.[68] Furthermore, in a follow-up study of ACCORD, the prevalence of OH did not differ between either arm at baseline, 12 months, or at 48 months.[69] Similarly, the SPRINT trial found comparable rates of OH in patients randomized to intensive blood pressure (<120 mmHg) and standard blood pressure (<140 mmHg) goals.[70] Finally, an individual participant level meta-analysis of 9 randomized controlled trials found that among 18,466 adults, more aggressive antihypertensive treatment lowered OH risk.[71]

A common critique of the above trials (e.g., SPRINT) is the exclusion of adults with more severe forms of OH thereby excluding a proportion of patients who could develop more severe orthostasis from antihypertensive treatment. However, in subgroups by low standing BP or pre-randomization OH in the meta-analysis above, there was no evidence of greater risk of OH with treatment. It should be noted that the antihypertensive agents used in hypertension trials may differ from those used in clinical practice, which may account for the discrepancy between hypertension trials and some observational studies. For example, in the AASK trial, initiating hypertension therapy with metoprolol was associated with a greater risk of OH compared with ramipril or amlodipine.[72] Nevertheless, evidence does not support the routine down-titration or discontinuation of antihypertensives in asytablemptomatic patients with hypertension and orthostasis. Rather, a more nuanced approach with selective discontinuation of medications associated with OH, while maintaining appropriate hypertension treatment goals is warranted.

Antihypertensive Classes Selection and OH

Antihypertensive classes that interfere with sympathetic compensatory responses to positional changes are most strongly associated with OH.[26] These classes include alpha

blockers, beta blockers, and central sympatholytics.[73] In contrast, numerous studies suggest that dihydropyridine calcium channel blockers, ACE inhibitors, and angiotensin receptor blockers (ARBs) have either a neutral or protective effect on OH.[26,73] A recent meta-analysis of randomized trials provides further evidence that drugs causing sympathetic inhibition are associated with a significantly increased odds of OH, whereas medications with a predominantly vasodilator mechanism of action are not associated with OH.[28] Observational studies have found that diuretics and, in particular, loop diuretics, are associated with OH.[2,23] Chlorthalidone, however, has not clearly been found to increase OH risk compared to other drug classes as seen in a secondary analysis of the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) trial.[74]

While most available data suggest that calcium channel blockers are not associated with OH, there are notable exceptions. In a secondary analysis of ALLHAT there was a short-term increased risk of falls among patients taking amlodipine compared with either lisinopril or chlorthalidone.[74] A cross-sectional analysis of the SPRINT trial examining patterns of orthostatic SBP changes found that greater postural reductions in SBP were associated with calcium channel blockers as well as alpha and beta blockers.[75] However, this study did not differentiate between dihydropyridine and non-dihydropyridine calcium channel blockers.

Short-term Risks of Antihypertensive Changes

Several studies suggest that the risk of adverse events related to OH is highest in the short-term period surrounding BP medication changes, particularly among older adults. A case cross-over study of over 90,000 adults aged 65 or older found a significantly higher risk of serious fall injuries within the 15 days after antihypertensive medication initiation or intensification.[76] A similar finding in relationship to antihypertensive withdrawal was observed in a secondary analysis of the Trial of Nonpharmacologic Interventions in the Elderly (TONE), in which 975 older adults were followed for 36 months after randomization to lifestyle interventions or usual care in the context of antihypertensive medication withdrawal. This study found that the majority of symptomatic adverse events (i.e., dizziness, blacking out, or presyncope) occurred in the 3 months after drug withdrawal.[77] Together, these data suggest increased vulnerability to orthostatic symptoms and falls in the short-term period following either the addition or withdrawal of antihypertensive medications, likely related to adaptations in BP regulation. As such, vigilance is warranted during the short-term period after antihypertensive medication changes.

Dietary sodium modulation

Volume expansion through increased dietary sodium intake, in some cases as high as 10 g daily, is recommended in clinical practice guidelines to improve orthostatic tolerance.[78] There is evidence from small observational studies to suggest sodium restriction worsens OH in patients with neurogenic OH[79,80] and other forms of orthostatic intolerance;[81] however, the quality of the evidence for improvement in symptoms is lacking.[82] Moreover, it is unclear that the above recommendations are applicable to hypertensive adults with OH, given the effects of sodium on supine hypertension. On the contrary, reduced dietary sodium intake of less than 1.5 g/day is recommended for the prevention and treatment of hypertension.[17] There is a paucity of data on the effects of increased sodium intake

in older adults with OH and cardiovascular comorbidities both with respect to clinical outcomes as well as symptoms. In fact, in a secondary analysis of the Dietary Approaches to Stop Hypertension (DASH) trial, higher sodium intake increased symptoms of postural light-headedness among participants assigned the DASH diet.[83]

Meanwhile contrary to traditional recommendations, there is physiologic evidence to support sodium restriction as a therapeutic approach for adults with nocturnal/supine hypertension and OH. Sodium homeostasis and BP regulation are linked: dietary sodium intake is a determinant of urinary sodium excretion and increased renal perfusion in the presence of hypertension induces a pressure natriuresis to lower total body sodium and reduce BP. The pressure natriuresis associated with nocturnal hypertension may be as high as 2 liters, which can exacerbate early morning symptoms of OH.[26] Dietary sodium restriction has been shown to reduce BP with sustained effects over a 24-hour period.[84] This raises the hypothesis that improved nocturnal BP through sodium restriction could mitigate nocturnal pressure diuresis, therefore preserving intravascular volume while optimizing cardiovascular risk in patients with hypertension.[85] Nevertheless, this would require formal testing and may be specifically relevant for adults with hypertensive forms of OH.

Special Considerations in Older Adults

Both OH and hypertension are particularly prevalent and morbid in older adults due to age-related dysfunction in BP regulation mechanisms. Physiologic changes which predispose older adults to OH include vascular stiffness and endothelial dysfunction,[86] impaired diastolic ventricular filling which causes pre-load dependent cardiac output reductions,[87] impaired salt and water conservation with reduced renin, angiotensin, and aldosterone levels, and reduced thirst.[88] Older adults also have a decline in baroreflex sensitivity characterized by a beta-receptor defect which manifests as a reduction in the chronotropic cardiac response to sympathetic stimulation.[89] One consequence of this age-related baroreflex impairment is an increased sensitivity to alpha blockers which effectively diminish the remaining compensatory mechanism against OH. Another consequence is that postural heart rate change is not a reliable measure in the diagnosis of OH in older adults, as the typical beta receptor mediated tachycardia response is blunted.

Clinical Approach to OH in Hypertensive Adults

Clinical practice guidelines for OH in hypertensive adults are not well-established. A suggested diagnostic approach is presented in the Figure. The initial assessment should begin with a detailed characterization of OH symptoms. This should be followed by an in-office evaluation of OH through physical exam and BP assessments with repeat measurements over serial visits. Out-of-office monitoring of OH should be performed in conjunction with in-office assessments and may include a symptom diary to elucidate the context and triggers of OH and 24-hr ABPM to identify BP phenotypes. Patients who present with symptoms of OH that are not corroborated by BP measurements should prompt an investigation of mimics of OH or other BP-related conditions that can provoke similar symptoms. For patients with confirmed OH, further work-up and treatment should be tailored to the suspected subgroup of OH (i.e., neurogenic, cardiovascular, pharmacologic,

or other) and should emphasize improving orthostatic symptoms as well as controlling BP to avoid supine/seated hypertension.

VII. Conclusions

OH is common among adults with hypertension and is associated with numerous adverse outcomes, including hypotensive events, falls, dementia, CVD, and death. The diagnostic approach for detecting OH can miss pathologic phenotypes when standing assessments are delayed or occur after a seated (versus supine) position. Since OH may be observed in the setting of hypertension without standing hypotension, thorough characterization of patterns of BP variability is critical for tailoring a treatment plan. For hypertensive adults, there is strong evidence that more aggressive BP lowering, may reduce risk of OH. Nevertheless, further research on optimal approaches to screening, diagnosis, and treatment of OH in hypertensive adults is needed to inform evidence-based guidelines for this heterogeneous and complex condition.

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Abbreviations used:

AASK	African American Study of Kidney Disease and Hypertension
ABPM	Ambulatory blood pressure monitoring
ACC/AHA	American College of Cardiology/ American Heart Association
ACCORD	Action to Control Cardiovascular Risk in Diabetes
ALLHAT	Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial
BPV	Blood pressure variability
CI	Confidence Interval
CVD	Cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	Diastolic blood pressure
FDA	Food & Drug Administration
OH	Orthostatic Hypotension
SBP	Systolic blood pressure
SPRINT	Systolic Blood Pressure Intervention Trial
STURDY	Study To Understand Fall Reduction and Vitamin D in You

TONE Trial of Nonpharmacologic Interventions in the Elderly

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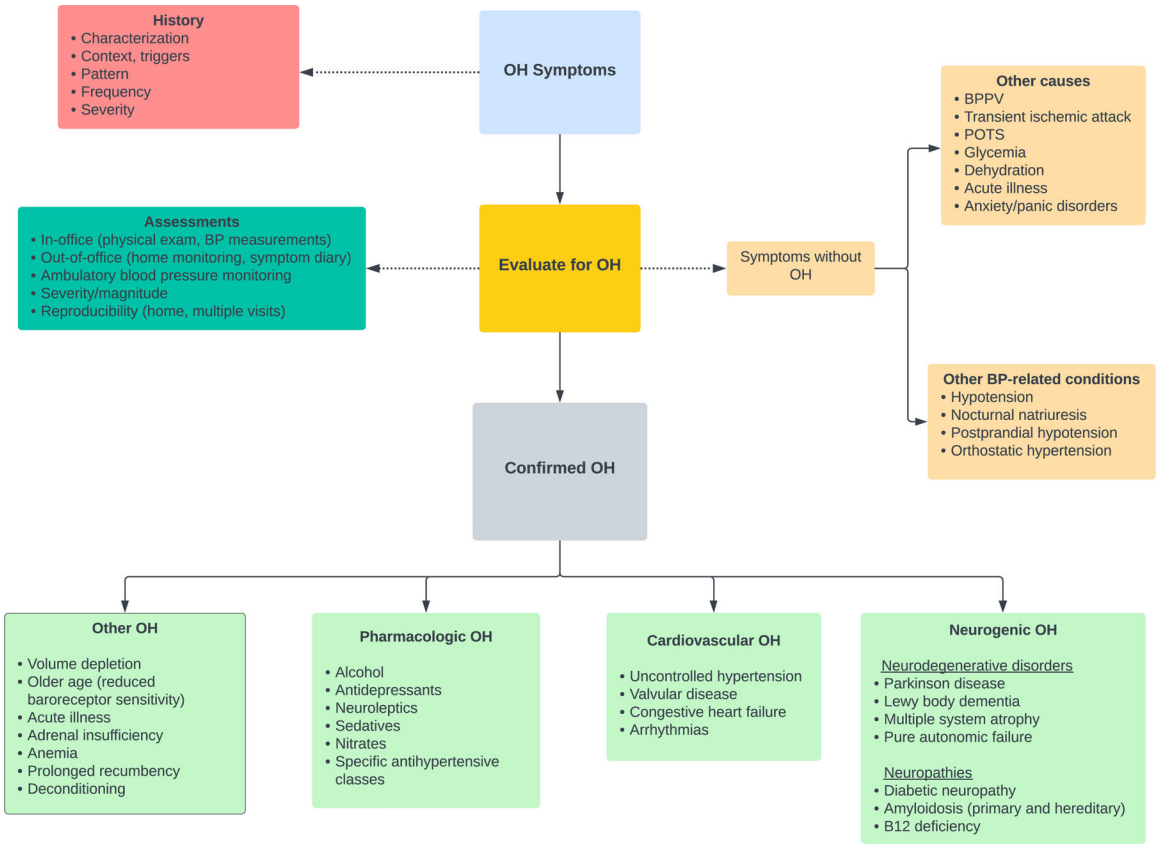

Figure. Diagnostic Approach for Patients with Hypertension and Orthostatic Hypotension

Abbreviations: BPPV, benign paroxysmal positional vertigo; BP, blood pressure; OH, orthostatic hypotension; POTS, postural orthostatic tachycardia syndrome.

Table.

Guideline Definitions of Orthostatic Hypotension

Guideline	Blood Pressure Change Cutoffs	Time Cutoffs	Position
American Academy of Family Physicians 2022	Decrease in SBP ≥20 mm Hg or DBP ≥10 mmHg	3 minutes	Supine to standing or tilt-table testing
American Autonomic Society Consensus Definition 1996 and 2011	Sustained decrease in SBP ≥20 mm Hg or DBP ≥10mmHg. Consider SBP ≥30 mm Hg for adults with supine hypertension	Two measurements <3 minutes	Recumbent to standing or tilt-table testing.
American College of Cardiology / American Heart Association 2017	Decrease in SBP >20 mm Hg or DBP >10 mmHg	1 minute	Seated to standing
American Diabetes Association 2017	Decrease in SBP >20 mm Hg or DBP >10 mmHg	3 minutes	Seated or supine to standing
Centers for Disease Control and Prevention 2017	Decrease in SBP ≥20 mm Hg or DBP ≥10 mm Hg	1 minute and 3 minutes	Supine to standing
European Society of Hypertension/ European Society of Cardiology 2018	Decrease in SBP ≥20 mm Hg or DBP ≥10 mmHg	1 minute and 3 minutes	Seated to standing as initial screen, can consider supine to standing in subsequent visits for high risk patients
National Heart Foundation of Australia 2016	Not specified	2 minutes	Seated to standing
National Institute for Health and Care Excellence 2019 (United Kingdom)	Decrease in SBP ≥20 mmHg	1 minute	Seated or supine to standing