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Orthostatic blood pressure disorders in older adults: clinical phenotypes, diagnostic challenges, and cognitive and functional implications

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ABSTRACT Orthostatic blood pressure disorders are common in older adults and represent an important cardiovascular and hemodynamic challenge associated with falls, cognitive impairment, and loss of independence. This Perspective highlights the clinical spectrum of orthostatic blood pressure disorders in older adults, emphasizing the interplay between age-related cardiovascular and autonomic changes, diagnostic challenges, and their implications for cerebral perfusion, cognition, and functional status. Orthostatic hypotension, supine hypertension, orthostatic hypertension, and postprandial hypotension frequently coexist and fluctuate over time, reflecting impaired physiological reserve rather than isolated hemodynamic abnormalities. Because even modest posture-related blood pressure changes may have important clinical consequences, careful orthostatic assessment, including standardized supine-to-standing measurements and interpretation of heart rate responses, is essential for recognizing overlapping phenotypes and guiding individualized management. Recognition of these disorders as markers of impaired cardiovascular–cerebral reserve may improve clinical decisions, reduce falls and cognitive complications, and help preserve function and independence in older adults.

Orthostatic blood pressure (BP) disorders are common in older adults and represent an important, yet often underrecognized cardiovascular and hemodynamic contributor to morbidity, functional decline, and reduced quality of life. Age-related changes in autonomic function, vascular compliance, and cardiovascular reserve increase susceptibility to inadequate BP responses during postural change, especially with multimorbidity and polypharmacy.[1–3] As a result, orthostatic BP disorders frequently coexist with core geriatric syndromes, including frailty and falls.[4–6]

Orthostatic hypotension (OH) is traditionally defined as a sustained reduction in systolic and/or diastolic BP upon standing,[7] but this classical definition captures only part of a broader spectrum of orthostatic abnormalities. In older adults, relevant phenotypes include neurogenic and non-neurogenic OH, delayed OH, OH with coexisting supine hypertension (SH), orthostatic hypertension (OHT), and postprandial hypotension (PPH).[2,8–9] These phenotypes often coexist within the same individual and may vary over time, reflecting heterogeneous impairments in autonomic regulation, vascular responsiveness, and cardiovascular compensation.[2,9–10]

Clinical manifestations extend beyond classical symptoms such as dizziness or syncope.[2] In older adults, orthostatic BP instability frequently presents with fatigue, gait disturbance, and falls, particularly in the context of frailty or multimorbidity. From a cardiovascular perspective, even without overt hypotensive episodes, recurrent or subclinical disturbances in cerebral and systemic perfusion may contribute to transient cognitive symptoms and increased brain vulnerability.[11–13]

Despite their relevance, orthostatic BP disorders remain underdiagnosed and undertreated.[4] Standardized diagnostic approaches are inconsistently applied, supine BP measurements are often omitted, and transient, delayed, or context-dependent phenotypes may be missed during routine assessment.[4,7] Inadequate recognition of coexisting phenotypes may lead to inappropriate antihypertensive intensification, aggravating upright hypoperfusion. Management is complicated by the need to balance upright perfusion against SH and treatment-related adverse effects, particularly in older adults with multimorbidity, polypharmacy, and reduced physiological reserve.[2,14–16]

Given these challenges, a comprehensive, phenotype-oriented approach is essential. Existing reviews have predominantly addressed pathophysiology or disease-specific management, with less emphasis on integrated cardiovascular–geriatric complexity and functional outcomes. In this review, we consider orthostatic BP disorders as dynamic clinical markers of reduced physiological reserve and impaired cardiovascular–cerebral reserve in older adults. The relationship between reduced physiological reserve, orthostatic BP phenotypes, and their associated outcomes is illustrated in Figure 1. By integrating cardiovascular, autonomic, and geriatric perspectives, this review aims to support phenotype-oriented assessment, individualized treatment decisions, and recognition of subthreshold impairment in older adults.

EPIDEMIOLOGY

OH is highly prevalent in older adults, with prevalence estimates rising from approximately 5% in middle-aged cohorts to about 30% in adults older than 70 years.[17] Population-based studies and meta-analyses indicate that OH affects approximately one in five community-dwelling older adults, with pooled estimates of about 22%.[18] Higher rates of OH are reported with multimorbidity, diabetes, neurodegenerative disease, hospitalization, and institutionalization, reflecting the combined effects of vascular aging, disease-related autonomic dysfunction, and treatment complexity.[2,17,19-20]

Hypertension is common in older adults, particularly among very old and multimorbid individuals, in whom orthostatic BP disorders frequently coexist.[6,21-23] Al-

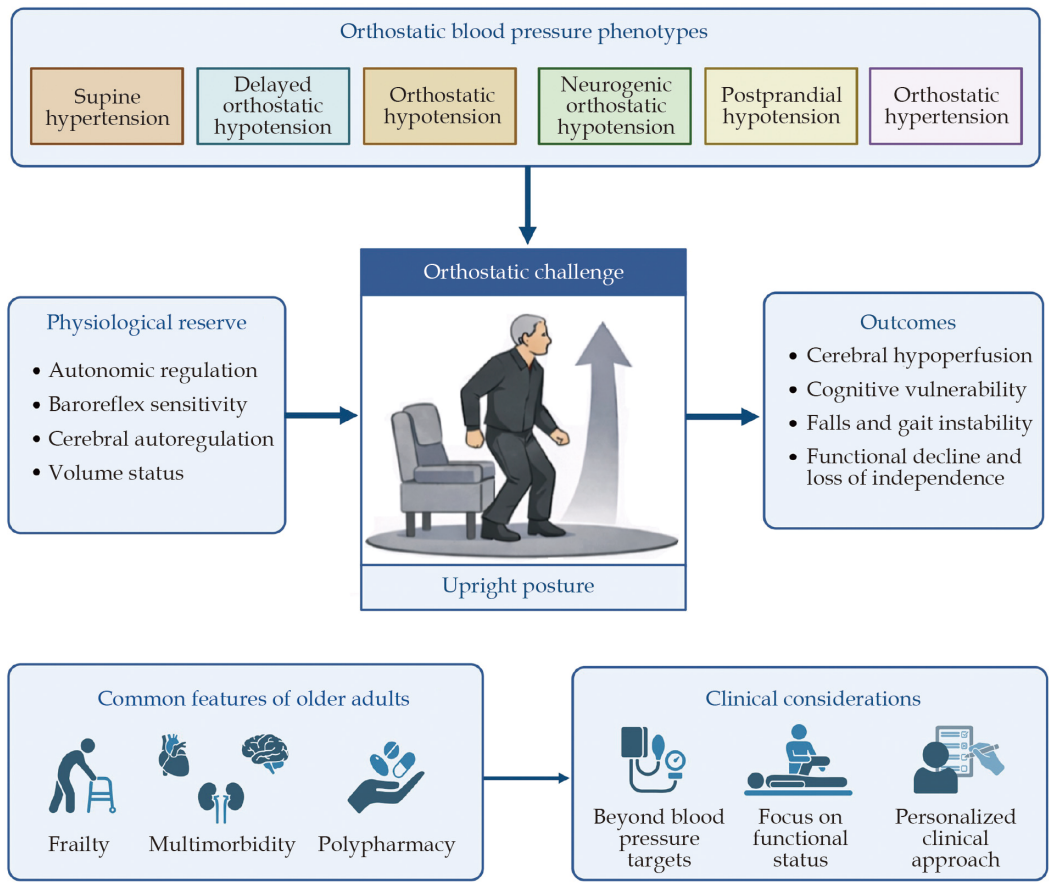

Figure 1 Orthostatic blood pressure phenotypes and functional vulnerability in older adults. This figure illustrates how age-related reductions in physiological reserve, including impaired autonomic regulation, reduced baroreflex sensitivity, altered cerebral autoregulation, and changes in volume status, contribute to heterogeneous orthostatic blood pressure phenotypes during standing in older adults. These phenotypes are associated with clinically relevant outcomes, including cerebral hypoperfusion, cognitive vulnerability, falls and gait instability, and functional decline with loss of independence. The figure highlights a phenotype-oriented, function-centered approach to the assessment and management of orthostatic blood pressure disorders in aging. Figure created in BioRender. Music, T. (2026) https://BioRender.com/hv8iz74.

though current international guidelines emphasize BP control to reduce cardiovascular risk, very old, frail, and multimorbid adults remain underrepresented in major clinical trials, precisely those in whom OH is common.[6,21–23] Consequently, clinicians caring for older adults with OH must balance the prevention of cardiovascular events against the risks of syncope, falls, and hypoperfusion-related complications.[19,24]

Despite its high prevalence and prognostic relevance, OH is frequently underrecognized in clinical practice, particularly when symptoms are nonspecific, intermittent, or overshadowed by comorbid disease.[4,20] Hospital-based studies demonstrate that OH is common yet often overlooked in acutely ill, multimorbid patients and is associated with both falls and in-hospital mortality.[20,25–26] In older adults, OH has been associated with an approximately 2.5-fold higher risk of recurrent falls.[2]

In community-based cohorts, OH has been linked with increased risks of incident heart failure (HF), stroke, and all-cause mortality, supporting its role as a marker of vascular-autonomic vulnerability rather than a benign physiological finding.[17,27–28]

Beyond cardiovascular outcomes, orthostatic BP dysregulation has also been associated with adverse brain outcomes. Epidemiologic studies suggest associations between OH, cerebrovascular disease, and brain vulnerability, underscoring the systemic consequences of disturbed BP regulation in aging.[11,29–30] Exaggerated BP increases on standing (orthostatic hypertension, OHT) have also been linked with silent cerebrovascular injury, suggesting that both hypotensive and hypertensive orthostatic responses may carry prognostic significance.[31–32]

Taken together, these findings characterize OH and related orthostatic BP disorders as highly prevalent, clinically important, and still insufficiently recognized conditions in older adults. They support systematic orthostatic BP assessment and individualized management strategies that account for frailty, multimorbidity, and competing clinical risks, integrating cardiovascular, autonomic, and geriatric perspectives.[2,19,23]

REVIEW APPROACH

This article is a narrative perspective review rather than a systematic review. Its aim is to synthesize clinically relevant evidence on orthostatic blood pressure disorders in older adults, with emphasis on cardiovascular mechanisms, diagnostic challenges, overlapping phenotypes, and cognitive and functional implications. Relevant literature was identified through targeted PubMed searches using combinations of terms related to orthostatic hypotension, neurogenic orthostatic hypotension, supine hypertension, orthostatic hypertension, postprandial hypotension, aging, frailty, cognition, cerebral perfusion, HF, and hypertension treatment. Additional references were identified through manual screening of relevant reviews, consensus statements, and key original studies. Because the aim was clinical synthesis rather than exhaustive evidence mapping, formal systematic-review methods and meta-analysis were not performed.

PATHOPHYSIOLOGY

Autonomic and Cardiovascular Mechanisms Underlying Orthostatic Blood Pressure Dysregulation

OH in older adults largely reflects age-related autonomic decline and impaired baroreflex sensitivity, both of which reduce the ability to maintain arterial pressure on standing.[2,33] Structural stiffening of the carotid sinus and aortic arch diminishes baroreceptor sensitivity and blunts afferent signaling to central autonomic centers, delaying and attenuating compensatory cardiovascular responses.[33]

These changes result in slower sympathetic activation and inadequate vasoconstriction on standing, often accompanied by delayed or blunted heart rate (HR) responses.[33–35] Older adults also exhibit reduced β-adrenergic responsiveness, limiting chronotropic and inotropic capacity during orthostatic stress.[24,33] Loss of muscle mass, with reduced efficiency of the skeletal-muscle pump, promotes venous pooling, reducing venous return and stroke volume.[1,36–37] Even in the absence of neurodegenerative disease, aging is associated with diminished sympathetic vasoconstrictor reserve and reduced norepinephrine release, reflecting limited autonomic reserve.[13,33,35] Together, these mechanisms reduce the physiological reserve required to maintain adequate blood pressure during orthostasis. Importantly, OH in older adults is not always neurogenic. In many cases, it may be reversible and related to hypovolemia, medication effects, venous pooling, or deconditioning.[1,9,36]

Neurogenic orthostatic hypotension

nOH is characterized by impaired autonomic regulation of BP, most commonly due to insufficient norepinephrine release from postganglionic sympathetic

neurons.[35,38] The consequent failure of appropriate sympathetic vasoconstriction leads to excessive orthostatic BP falls, often with relatively modest HR increases.[35,38]

Although classically associated with primary autonomic disorders, similar hemodynamic patterns are also observed in older adults with diabetes, multimorbidity, or medication-related interference with autonomic function.[1,38]

In clinical practice, nOH in older adults rarely appears as an isolated autonomic problem; instead, it coexists with increased vascular stiffness, impaired baroreflex buffering, and hypertension, producing complex, and sometimes fluctuating orthostatic responses.[2,9,35]

This interaction between neurogenic impairment and age-related cardiovascular changes helps explain why older adults with nOH often show delayed and incomplete maintenance of BP after standing, accompanied by blunted HR responses and prominent functional symptoms.[2,9,38]

Supine Hypertension in Orthostatic Hypotension

SH, defined as a BP of at least 140/90 mmHg after five minutes of supine rest, occurs in approximately one-half of patients with nOH, with reported prevalence varying across autonomic failure syndromes, and is often missed if supine measurements are not obtained.[16] Although frequently attributed to pressor treatment, SH may also occur in untreated patients, indicating that it is often an intrinsic feature of autonomic dysfunction rather than solely a treatment-related adverse effect.[39]

In conditions with relatively preserved sympathetic fibers, such as multiple system atrophy, SH may result from residual sympathetic vasoconstriction that is inadequately buffered by the baroreflex. Conversely, in disorders with postganglionic denervation, SH may occur despite low plasma norepinephrine, suggesting contributions of non-sympathetic pressor mechanisms.[16,38–39]

A blunted HR response to standing remains a characteristic feature of neurogenic OH and helps distinguish neurogenic from non-neurogenic causes.[7,38] Importantly, SH is not confined to patients with primary autonomic disorders. It may also occur in older adults with primary hypertension and age-related autonomic decline, where impaired baroreflex buffering and increased arterial stiffness permit higher supine BP, even in the absence of neurodegenerative disease.[24,33,36]

Orthostatic Hypertension

OHT is defined as an exaggerated orthostatic pressor response, characterized by a sustained increase in systolic BP of ≥ 20 mmHg upon standing, resulting in an upright systolic BP ≥ 140 mmHg.[8] It likely reflects impaired baroreflex buffering together with excessive sympathetic activation and increased peripheral vascular resistance. Earlier studies have linked exaggerated orthostatic BP rises with markers of silent cerebrovascular disease, supporting the view that OHT represents a pathological rather than compensatory response.[31]

Potential mechanisms include increased arterial stiffness, diminished baroreceptor sensitivity, and enhanced sympathetic vasoconstrictor responses, all of which intensify with aging.[19,32,40] OHT is commonly observed in patients with chronic hypertension, complicating treatment decisions and underscoring the importance of routine orthostatic BP assessment, particularly in older adults.[8,32]

Postprandial Hypotension

PPH is defined as a fall in systolic BP of at least 20 mmHg within two hours after a meal and reflects inadequate cardiovascular compensation for splanchnic vasodilation.[41] In older adults or those with autonomic impairment, insufficient sympathetic compensation for meal-induced vasodilation predisposes to postprandial BP falls.[41]

PPH is highly prevalent among institutionalized older adults. In a cohort of nursing home residents, approximately 38% of participants were affected, and PPH independently predicted all-cause mortality (RR 1.79, 95% CI 1.19–2.68). Moreover, a dose–response relationship was observed between the magnitude of postprandial systolic BP decline and mortality risk.[42] Mechanistically, PPH reflects impaired baroreflex sensitivity and reduced sympathetic compensation, and frequently coexists with OH in multimorbid older adults.[43–44]

Because orthostatic BP disorders often overlap and evolve over time, a phenotype-oriented approach may facilitate clinical assessment and management. Key phenotypes, definitions, and clinical clues are summarized in Table 1.

CLINICAL PRESENTATION IN OLDER ADULTS

In older adults, OH presents across a broad and frequently underrecognized spectrum of symptoms. Although classic manifestations such as dizziness, lightheadedness, and syncope are well recognized, many

Table 1 Orthostatic blood pressure phenotypes: definitions, clinical clues, and diagnostic priorities.

Phenotype	Definition	Typical clinical clues	Diagnostic priorities
OH	↓ SBP ≥ 20 mmHg or ↓ DBP ≥ 10 mmHg within 3 min of standing or tilt	Dizziness, presyncope, gait instability, cognitive clouding	Supine → standing BP/HR; repeat testing if equivocal
Delayed OH	OH criteria met after >3 min of standing	Symptoms with prolonged standing, rehabilitation activities	Prolonged standing or tilt-table testing
Neurogenic OH	OH with blunted HR rise < 15 beats/min	Autonomic failure, parkinsonism, neuropathy	Assess for SH; assess broader autonomic features
Non-neurogenic OH	OH with appropriate HR rise	Hypovolemia, medications, venous pooling	Identify reversible contributors (volume, drugs, deconditioning)
Supine hypertension (SH)	Supine BP ≥140/90 mmHg after ≥5 min rest	Morning OH, nocturia, headache	Intentional supine measurement (essential)
Orthostatic hypertension (OHT)	↑ SBP ≥20 mmHg on standing, with upright SBP ≥140 mmHg	Standing headache, discomfort	Repeat testing; assess orthostatic BP variability across repeated measurements
Postprandial hypotension (PPH)	↓ SBP ≥ 20 mmHg within 2 h after meals or SBP ≤ 90–100 mmHg	Fatigue, dizziness, "mental fading" after meals	BP measurement linked to meals
Orthostatic tachycardia	HR rise ≥ 30 beats/min with preserved BP*	Tremor, palpitations, cognitive clouding	Distinguish from POTS; consider deconditioning and reversible triggers

*Formal POTS criteria were developed primarily in younger populations and should be applied with caution in older adults. Key supporting references: (4, 9, 10, 17, 39, 42). DBP: diastolic blood pressure; HR: heart rate; OH: orthostatic hypotension; OHT: orthostatic hypertension; POTS: postural orthostatic tachycardia syndrome; PPH: postprandial hypotension; SBP: systolic blood pressure; SH: supine hypertension.

older adults instead report nonspecific symptoms, including fatigue, generalized weakness, gait unsteadiness, or visual disturbances, that are easily misattributed to aging or comorbidities.[2,9,26] In frail or multimorbid older adults, symptoms may be subtle, show daytime variability, or occur only in specific contexts. Hemodynamically significant orthostatic BP falls may therefore be present even when patients deny typical symptoms.[4] Medication burden further complicates recognition, as antihypertensives, psychoactive agents, and diuretics may blunt compensatory responses and increase susceptibility to orthostatic intolerance.[1]

The clinical presentation of orthostatic BP disorders in older adults is heterogeneous and often reflects the interplay of autonomic dysfunction, vascular aging, and common geriatric features such as frailty, multimorbidity and polypharmacy.[2,9,24]

Age-related arterial stiffening reduced chronotropic responsiveness, and impaired venous return on standing limit cardiovascular compensation, so that even modest orthostatic systolic BP declines, including those not meeting formal diagnostic thresholds, may precipitate clinically relevant symptoms. Variability related to hydration, time of day, medication timing, and meal-related BP changes often obscures the clinical picture and complicates diagnosis.[41–43]

Falls and gait instability are key clinical consequences of OH, which is strongly associated with impaired mobility and an increased risk of falls, particularly among frail older adults with reduced physiological reserve.[3–4] PPH further adds to vulnerability, especially in institutionalized settings, where post-meal syncope or collapse may occur when orthostatic phenotypes coexist.[41–42,44]

Several symptoms are characteristic of orthostatic hemodynamic compromise. One of the most typical is the "coat-hanger headache," a dull ache across the occipital and paraspinal muscles that reflects hypoperfusion of the trapezius and cervical extensors and is commonly reported in nOH.[2,9] Transient visual dimming and profound weakness may accompany orthostatic hypoperfusion and are often mistaken for fatigue, deconditioning, or medication side effects.[2,13]

In vulnerable older adults, recurrent orthostatic hypoperfusion, BP variability, and coexisting supine or nocturnal hypertension may contribute to end-organ vulnerability, including worsening renal function.[2,19] Patients with OH experience more than transient symptomatic episodes. Over time, OH may impair quality of life by promoting activity avoidance, fatigue, fear of falling, and progressive mobility limitation, particularly in the presence of multimorbidity, frailty, and polypharmacy.[1,4,6,36] In addition, OH may also be accompanied by posture-related cognitive complaints, including reduced attention, mental slowing, impaired executive function,

and transient confusion. Cognitive symptoms and posture-related cognitive impairment are addressed in a dedicated section later in this review.[11,13]

Cardiovascular Implications for Cardiology Practice

From a cardiology perspective, orthostatic BP disorders have implications beyond falls and functional decline.[2,17,28] OH is a frequent and often underrecognized cause of syncope and should therefore be considered alongside arrhythmic and structural cardiac causes during the evaluation of syncope or unexplained presyncope.[2,17] Orthostatic BP disorders may also influence myocardial perfusion, HF management, and hypertension treatment decisions, because cardiovascular drugs, volume status, and BP targets may affect upright BP stability and end-organ perfusion.[2,17,19,28] In older adults with coronary artery disease, OH may manifest as posture- or exertion-related chest discomfort or angina-like symptoms due to transient systemic and myocardial hypoperfusion, particularly when reduced coronary perfusion reserve and low upright diastolic pressure limit coronary blood flow (see Supplementary Vignette 1).[2,17,45] When SH coexists, nocturnal cardiovascular load and pressure natriuresis may contribute to nocturia, overnight volume loss, and worsening morning OH. This makes BP management particularly complex and creates a therapeutic dilemma in patients with both upright hypoperfusion and nocturnal or supine hypertension.[14,16]

HF adds further complexity. In decompensated HF, congestion and elevated filling pressures may mask orthostatic vulnerability, whereas decongestion, diuretic escalation, or vasodilator therapy may reduce effective circulating volume or vascular tone, thereby unmasking clinically relevant upright hypoperfusion.[2,19,24,46] In patients with HF with reduced ejection fraction (HFrEF), low BP, symptomatic hypotension, or orthostatic intolerance may limit optimization of guideline-directed medical therapy in routine practice, particularly therapies with BP-lowering or volume-modifying effects.[46] OH has also been associated with incident HF and broader cardiovascular risk, supporting its interpretation as a marker of vascular–autonomic vulnerability rather than an isolated bedside finding.[17,27-28] Orthostatic BP assessment should therefore be repeated after changes in volume status and when BP-lowering or volume-modifying HF therapy is initiated, withdrawn, or titrated, particularly in older or frail patients.[2,19,24,26]

Orthostatic BP phenotypes should be considered in hypertension treatment strategies in cardiology practice because seated clinic BP alone may miss clinically important upright hypotension, supine hypertension, or BP variability.[16,19,28] Coexisting or fluctuating phenotypes should not automatically preclude cardiovascular risk reduction. Rather, they should prompt individualized treatment decisions that account for standing and supine BP, symptom burden, medication timing, frailty status, and the competing risks of upright hypoperfusion and supine or nocturnal hypertension.[19,24,28,47] This approach may help avoid both unnecessary withdrawal of cardiovascular protection in asymptomatic OH and excessive BP lowering in patients with symptomatic upright hypoperfusion or limited perfusion reserve.[24,28,48] In older adults with atrial fibrillation, orthostatic symptoms, falls, or syncope may further complicate cardiovascular risk–benefit assessment, particularly when anticoagulant-related bleeding concerns must be balanced against the need for stroke prevention.[49]

Thus, orthostatic BP disorders should be integrated into cardiovascular decision-making as dynamic modifiers of treatment tolerance, perfusion reserve, and the risk–benefit balance of cardiovascular therapy.

DIAGNOSTIC EVALUATION OF ORTHOSTATIC BLOOD PRESSURE DISORDERS

Diagnosing orthostatic BP disorders in older adults requires a structured and context-sensitive approach. OH and related orthostatic phenotypes are often intermittent, influenced by circadian and physiological variation, and therefore easily overlooked.[2,9] Age-related autonomic decline, multimorbidity, frailty, and polypharmacy further complicate interpretation because they may modify or obscure typical symptom patterns.[1,24] As a consequence, orthostatic BP disorders are frequently underrecognized until functional decline or complications emerge.[2,9]

Accurate characterization relies on standardized supine-to-standing BP measurements with simultaneous HR assessment. Repeated measurements should be performed when initial findings are inconclusive or discordant with symptoms.[7,10] A standardized protocol begins with at least five minutes of quiet supine rest, followed by BP and HR measurement in the supine position and after standing, typically at one and three minutes. Supine measurements are essential, because

seated-to-standing testing may fail to detect OH and overlook coexisting SH.[1,9]

Orthostatic BP responses often vary over time, and normal findings at three minutes do not exclude clinically relevant hypotension. Some patients develop BP falls only with prolonged standing (delayed OH), a phenotype associated with adverse outcomes, including neurodegenerative disease and increased mortality.[9,17] In addition, circadian variation is common: morning OH frequently occurs due to nocturnal pressure natriuresis and relative volume depletion upon arising. Repeated measurements, particularly in the morning or with structured home monitoring, can therefore improve diagnostic sensitivity.[1,24]

Heart Rate Response in Orthostatic Hypotension: Diagnostic Implications

Interpretation of the HR response is a key element of diagnostic evaluation. In nOH, HR increase is typically blunted. Despite substantial BP falls, HR usually rises by fewer than 15 beats per minute, reflecting impaired sympathetic activation and reduced norepinephrine release.[35,38] This dissociation between the magnitude of the BP decline and the HR response represents an important diagnostic clue.[35,38] By contrast, in non-neurogenic OH, HR typically shows a more pronounced compensatory rise, reflecting an appropriate physiological attempt to preserve cardiac output.[2,7] When bedside testing is inconclusive, formal autonomic testing may help detect autonomic impairment, particularly when nOH is suspected.[9,34]

An excessive increase in HR on standing may suggest postural tachycardia syndrome (POTS), although this condition is uncommon in older adults. Pronounced orthostatic tachycardia usually reflects non-neurogenic factors such as hypovolemia, pain, anxiety, or medication effects rather than POTS.[2,9] (see Supplementary Vignette 2).

Recognition of Overlapping Orthostatic Blood Pressure Phenotypes and Symptom-guided Interpretation

Several orthostatic BP phenotypes frequently coexist in older adults and may fluctuate over time, making isolated interpretation of single measurements unreliable. Recognition of these overlapping patterns is essential to avoid misclassification and unsafe management decisions.[2,8]

Coexisting SH should be actively sought during diagnostic evaluation, particularly in patients with suspected neurogenic OH or morning orthostatic symptoms. Intentional supine BP measurement is essential, because SH is easily missed when assessment is limited to seated or standing BP.[7,16]

OHT is defined as an exaggerated orthostatic pressor response, characterized by a sustained increase in systolic BP of at least 20 mmHg upon standing.[8] Because OHT may be intermittent, repeated standardized measurements are often required. Some patients report headache, discomfort, or unsteadiness during BP rises, symptoms that may otherwise be misattributed to anxiety or measurement artifact.[8,19,32] (see Supplementary Vignette 3).

PPH is diagnosed when systolic BP falls by at least 20 mmHg within two hours after a meal, or when postprandial systolic BP reaches 90–100 mmHg or lower.[41–42] Targeted post-meal measurements are particularly important when symptoms such as fatigue, gait instability, presyncope, or unexplained falls occur after eating, as standard orthostatic testing alone may fail to detect this phenotype.[41–43] (see Supplementary Vignette 4).

Symptom-guided interpretation remains central to diagnosis, as many older adults do not report classical dizziness or presyncope. Instead, nonspecific symptoms such as fatigue, unsteadiness, tremulousness, or visual dimming are common.[2,9] Symptoms that occur within minutes of standing, improve rapidly with recumbency, cluster in the morning, or follow meals are strongly suggestive of orthostatic BP dysregulation.[1-2,43]

Diagnostic Pitfalls

Several pitfalls commonly undermine diagnostic accuracy. Omission of true supine BP measurements may result in missed diagnoses of OH, SH, OHT, or PPH.[7,9] Medications including antihypertensives, diuretics, psychoactive agents, and α-blockers may precipitate OH and mimic other causes of dizziness or falls.[1,15] In some cases, medication burden rather than disease progression is the dominant driver of symptoms, and deprescribing alone can normalize orthostatic responses (see Supplementary Vignette 5).[1,15]

Acute illness, dehydration, infection, and rapid diuresis can exaggerate BP drops, so abnormal results obtained during acute deterioration should be reassessed after stabilization.[1,24] Conversely, hypervolemic states may temporarily mask OH, particularly in decompensated HF, with orthostatic vulnerability becoming

evident after diuresis.[1–2]

Finally, neglecting HR interpretation may result in misclassification between neurogenic and non-neurogenic forms.[7,38] Overall, diagnostic evaluation of orthostatic BP disorders in older adults relies on standardized supine-to-standing measurements, careful interpretation of HR responses, and repeated assessment across clinical contexts. Recognition of overlapping phenotypes, including SH, OHT, and PPH, is essential to avoid misclassification and guide safe, individualized management.[8,16,41] Key diagnostic pearls and common pitfalls are summarized in Table 2, while clinical vignettes illustrate common real-world presentations and challenges.

Vignette 1: Severe orthostatic hypotension with marked supine hypertension

Presentation

An 82-year-old man with type 2 diabetes and long-standing hypertension presented with recurrent morning dizziness, unsteadiness, and several syncopal episodes shortly after rising from bed, including one fall with a head contusion. His antihypertensive regimen included a β-blocker and a calcium-channel blocker, both administered in the evening.

Assessment

After five minutes of supine rest, BP was 170/90 mmHg, consistent with SH. After one minute of standing, BP fell to 78/54 mmHg with only a minimal HR increase (from 62 to 68 beats/min). This combination of severe OH and marked SH was more suggestive of underlying autonomic dysfunction than simple antihypertensive overtreatment.

Management

Management focused on separating upright and supine BP goals. Evening antihypertensive doses were reduced, the head of the bed was elevated, morning hydration was encouraged, and low dose midodrine was initiated for use during daytime upright activities.

Table 2 Diagnostic pearls and pitfalls in orthostatic blood pressure disorders.

Domain	Diagnostic pearl	Common pitfall
Measurement technique	Obtain true supine BP after ≥ 5 min of rest, then measure BP and HR at 1 and 3 minutes of standing.	Seated-to-standing testing may miss OH and cannot detect SH.
Timing of assessment	Prefer morning testing when orthostatic symptoms are most likely to be expressed.	Afternoon testing may appear "normal" despite pronounced morning symptoms.
Repeated testing	Repeat orthostatic testing when symptoms persist but initial results are negative.	False reassurance from a single "normal" test.
Heart-rate interpretation	Blunted HR rise (< 15 beats/min) suggests nOH.	Assuming all OH is neurogenic and missing hypovolemia, anemia, or medication effects.
Supine hypertension	Measure supine BP intentionally; SH commonly coexists with OH.	Interpreting morning OH as dehydration rather than SH-related nocturnal natriuresis.
Orthostatic hypertension	A ≥ 20 mmHg systolic rise on standing is abnormal and clinically relevant.	Dismissing OHT as anxiety or measurement artifact.
Postprandial hypotension	Measure BP before and after meals when symptoms are meal-related.	Attributing post-meal dizziness to "normal aging."
Symptom correlation	Symptoms relieved by sitting or lying are highly suggestive of orthostatic dysregulation.	Over-reliance on rigid BP thresholds without symptom context.
Cognitive vulnerability	Inattention or slowing on standing suggests orthostatic cognitive vulnerability.	Mislabeling cognitive changes as dementia without orthostatic BP assessment.
Polypharmacy	Perform structured medication review.	Adding pressor therapy while offending medications remain unaddressed.
Frailty context	Small BP reductions may have large functional impact.	Requiring strict ≥ 20/10 mmHg thresholds to recognize clinical relevance.
Acute illness	Reassess after clinical stabilization; consider volume shifts.	Misclassifying reversible OH as nOH; hypervolemia may temporarily mask OH.

This table summarizes practical diagnostic considerations and common sources of misinterpretation or missed diagnosis. Key supporting references: (3,4,8,9,10,17,42). BP: blood pressure; HR: heart rate; OH: orthostatic hypotension; OHT: orthostatic hypertension; nOH: neurogenic orthostatic hypotension; SH: supine hypertension.

Outcome

Orthostatic symptoms improved, and SH did not worsen.

Key clinical takeaways

- Supine BP must be intentionally measured.
- A blunted HR response supports the diagnosis of nOH.
- Upright and supine BP often require separate management strategies.
- Antihypertensive overtreatment is not always the main cause; in older adults, autonomic failure and SH may coexist and require individualized management.

Vignette 2: Heart failure decompensation unmasking severe orthostatic hypotension

Presentation

An 83-year-old woman was admitted with acute decompensated heart failure. Prior to admission, she was able to manage daily activities on her own. On admission, she was markedly volume overloaded, with a BP of 168/98 mmHg despite taking four antihypertensive medications. Initial orthostatic testing was negative.

Assessment

She underwent guideline-directed decongestive therapy with intensive diuresis, resulting in a 14-kg weight loss and normalization of NT-proBNP levels. Antihypertensive therapy was progressively withdrawn. After euvolemia was achieved, she developed severe presyncope on standing. Supine BP normalized to 135/80 mmHg, but upright BP fell to 82/56 mmHg, leaving her unable to stand despite clear improvement in cardiac status. Severe OH, previously masked by congestion and elevated filling pressures, then became clinically evident.

Management

Orthostatic BP was reassessed after decongestion, antihypertensive therapy was adjusted, and management focused on safe upright tolerance before rehabilitation and discharge planning.

Outcome

Severe OH became clinically evident only after decongestion, underscoring the need for repeated orthostatic assessment after major changes in volume status.

Key clinical takeaways

- Hypervolemia and congestion can temporarily mask clinically relevant OH.
- Orthostatic testing should be repeated after major changes in volume status or medication burden.
- Functional recovery may lag behind cardiac stabilization if OH is not recognized and addressed.
- Improvement in cardiac biomarkers does not guarantee safe upright perfusion; reassessment of orthostatic BP is essential before rehabilitation and discharge.

MANAGEMENT OF ORTHOSTATIC BLOOD PRESSURE DISORDERS IN OLDER ADULTS

Management of orthostatic BP disorders in older adults requires a multimodal, phenotype-oriented strategy tailored to autonomic physiology, comorbid conditions, and functional goals.[1-2,9] In this population, the primary aim is not to normalize BP, but to improve upright tolerance, prevent falls, and preserve independence and quality of life.[2,9] Age-related autonomic decline, frailty, and polypharmacy frequently modify therapeutic responses, emphasizing the need for individualized, stepwise plans that are reviewed regularly.[1,3,24] Table 3 highlights practical considerations and common pitfalls that may help guide clinical decision-making in older adults.

Non-pharmacologic Management

Non-pharmacologic interventions are first-line therapy for all orthostatic BP disorders and often provide substantial benefit with very little risk.[2,10] Their goals are to optimize intravascular volume, improve venous return, support autonomic compensation, and reduce meal-related BP drops.[2,10]

Adequate hydration increases intravascular volume and attenuates orthostatic BP reductions, particularly in older adults with impaired thirst perception or those taking diuretics.[1-2] Salt supplementation may be considered when not contraindicated by HF or chronic kidney disease, with regular monitoring for edema, weight gain, and electrolyte disturbances.[1-2]

For example, rapid ingestion of approximately 300–500 mL of water induces a sympathetically mediated pressor response and can transiently improve up-

Table 3 Management pearls and pitfalls in orthostatic blood pressure disorders.

Domain	Management pearl	Common pitfall
Initial strategy	Start with non-pharmacologic strategies whenever possible.	Starting pressors too early, increasing BP variability, or worsening SH.
Volume optimization	Encourage fluids and salt when appropriate.	Liberal salt or fluid intake in HF or CKD, leading to decompensation.
Physical countermeasures	Teach active postural and isometric maneuvers.	Providing generic advice without practical coping strategies.
Compression	Prefer abdominal binders, with or without stockings.	Relying on stockings alone, with minimal benefit.
Medication timing	Adjust timing to reduce upright BP drops.	Abrupt withdrawal or excessive de-intensification leading to uncontrolled hypertension or SH.
Pressor therapy	Use short-acting agents and avoid doses close to recumbency.	Using pressors on fixed schedules without regard to SH or daily pattern.
Mineralocorticoids	Use fludrocortisone only with careful monitoring.	Edema, hypokalemia, and HF exacerbation.
Pyridostigmine	Consider selectively; modest effect.	Expecting large BP increases.
Meal-related symptoms	Modify meals and medication timing.	Misattributing post-meal symptoms to aging or nonspecific fatigue.
Supine hypertension	Head-of-bed elevation and, in selected cases, short-acting antihypertensives at bedtime.	Over-treating SH and worsening morning OH.
Orthostatic hypertension	Monitor variability and context before treating.	Managing OHT as ordinary essential hypertension.
Medication review	Structured deprescribing of culprit drugs.	Adding pressors while offending medications remain.
Frailty	Individualize BP targets and expectations.	Applying uniform guideline thresholds.
Cognitive vulnerability	Consider posture-related cognitive symptoms.	Mislabeling cognitive changes as dementia without orthostatic BP assessment.
Monitoring	Home BP monitoring combined with a symptom diary.	Relying only on clinic readings.

This table summarizes practical therapeutic considerations and common treatment-related pitfalls or unintended consequences. Key supporting references: (2,9,14,15,24,32,41). BP: blood pressure; CKD: chronic kidney disease; HF: heart failure; OH: orthostatic hypotension; OHT: orthostatic hypertension; PPH: postprandial hypotension; SH: supine hypertension.

right BP, an effect that is often preserved in nOH.[2,10,30] Physical counter-maneuvers, including leg crossing, contraction of the buttock and thigh muscles, or sustained handgrip, reduce venous pooling and improve venous return. Compression garments, particularly abdominal binders or waist-high stockings, may further increase venous return and peripheral resistance and can be implemented in routine clinical practice, especially in patients with impaired skeletal muscle pump function.[2,9]

Elevation of the head of the bed reduces nocturnal pressure natriuresis and associated volume loss, thereby alleviating morning OH, particularly in those with coexisting SH.[14,16] A structured medication review is essential. Antihypertensives, psychoactive agents, diuretics, and α-blockers commonly contribute to orthostatic intolerance; when appropriate, deprescribing, dose reduction, or redistribution of dosing may substantially improve symptoms.[1,15,47]

Education of patients and caregivers is a pivotal, yet often underrecognized, component of management. Clear explanations of symptom triggers, safe postural transitions, and practical strategies such as slow rising, pre-emptive hydration, and counter-maneuvers support proactive self-management, reduce fear-driven activity avoidance, and improve confidence.[1–2,9,10]

In older adults with frailty or cognitive vulnerability, involvement of family members or caregivers is essential to support implementation and safety, particularly when multiple orthostatic phenotypes coexist.[2,47] Education should be regarded as an ongoing process, reinforced during follow-up as symptoms and treatment evolve.[2,9]

Pharmacologic Management

Pharmacologic therapy is indicated for patients who remain symptomatic despite optimized non-pharmacologic strategies or when orthostatic intolerance substantially limits mobility, independence, and quality of life.[1–2] Because older adults have heightened drug sensitivity and multimorbidity, treatment should proceed gradually, with close monitoring for adverse effects and SH.[1,24]

Midodrine, an α1-adrenergic agonist, increases peripheral vascular resistance and improves standing BP and orthostatic tolerance.[2,35,38] Its short half-life allows dosing to be limited to upright hours. Evening doses should be avoided to reduce the risk of SH.[2,35] Monitoring for urinary retention is advised.[2,16]

Droxidopa, a synthetic precursor of norepinephrine, enhances sympathetic activity and is particularly useful in nOH characterized by reduced noradrenaline release.[2,35,38] Randomized studies report improvements in standing BP, dizziness, and walking capacity, although the risk of SH requires careful surveillance.[2,35,38]

Fludrocortisone expands intravascular volume by promoting renal sodium retention. Although useful in carefully selected patients, it carries important risks, including edema, hypokalemia, SH, and HF decompensation, necessitating judicious dosing and regular clinical and laboratory monitoring.[2,9,14,24]

Pyridostigmine augments cholinergic transmission at autonomic ganglia and may modestly improve standing BP without significantly worsening SH, particularly in milder forms of autonomic impairment. However, clinical benefit is often modest and careful patient selection is essential.[2,9] Management of coexisting SH aims to limit nocturnal BP surges, primarily by minimizing nighttime pressor effects.[14,16] When possible, evening doses of pressor agents should be avoided, the head of the bed elevated, and antihypertensive therapy redistributed toward nighttime when clinically appropriate. In carefully selected patients, short-acting agents at bedtime (for example, transdermal nitroglycerin or clonidine) may be considered, but the evidence base is limited and close monitoring is required.[14,16]

In older adults with OH or PPH, careful adjustment of antihypertensive therapy may improve orthostatic tolerance without compromising long-term cardiovascular protection.[15,47] Conversely, overly aggressive BP lowering may increase vulnerability to hypoperfusion-related adverse outcomes.[6,48]

Management of Specific Orthostatic BP Phenotypes

In nOH, management is stepwise and individualized.[2,16,38] Core measures include adequate hydration, compression garments, head-up sleeping, bolus water drinking, and, when indicated, pharmacologic therapy (most commonly midodrine, droxidopa, or pyridostigmine), with careful titration and active surveillance for SH.[2,16,38]

In patients with OH and coexisting SH, management requires clear separation of upright and supine BP goals. Strategies include careful timing and dosing of pressor agents, avoidance of long-acting antihypertensives, elevation of the head of the bed at night, and prioritizing mobility and symptom control while balancing long-term cardiovascular risk.[14,16]

Management of OHT is largely non-pharmacologic. The focus is on avoiding unnecessary escalation of antihypertensive therapy and on reducing triggers that may provoke excessive sympathetic activation, recognizing that evidence for specific treatment thresholds remains limited and that cerebrovascular risk must be considered.[8,32]

PPH is addressed through meal modification (smaller, more frequent meals with reduced simple carbohydrates), adequate hydration, and redistribution or timing of medications when feasible.[14,42–43] In selected refractory cases, agents such as acarbose or caffeine may be considered, although evidence in older adults remains limited and potential adverse effects should be weighed carefully.[41–43]

ORTHOSTATIC COGNITIVE VULNERABILITY IN OLDER ADULTS: THE BRAIN UNDER ORTHOSTATIC STRESS

Orthostatic BP disorders have implications that extend beyond cardiovascular health and mobility. Even modest orthostatic BP reductions may compromise cerebral perfusion in frail older adults, unmasking limited cerebrovascular reserve and increasing cognitive vulnerability.[4,11–12]

Orthostatic Challenge as a Cerebral Stress Test

Emerging evidence supports the concept that orthostatic BP instability may act as a clinical stressor for the aging brain, revealing limited cerebrovascular and autonomic reserve.[4,50] In older adults, autonomic decline (including impaired baroreflex sensitivity), vascular stiffening, and reduced cerebral autoregulation collectively constrain the ability to maintain stable cerebral perfusion during postural change.[9,24,30,33]

Consequently, even modest orthostatic BP reductions may lower cerebral perfusion below the levels sufficient for optimal cognitive functioning, particularly in very old or frail individuals and in those with existing cerebrovascular disease.[4,13,48] From this perspective, orthostatic BP

disorders represent not only systemic hemodynamic disturbances but also clinically important challenges to cerebral perfusion that may contribute to transient cognitive fluctuations and longer-term cognitive vulnerability.[11–12]

Acute Effects of Orthostatic BP Dysregulation on Cognition

Rapid orthostatic reductions in cerebral perfusion have been linked to transient impairments in attention, processing speed, executive function, and information integration.[12,30,50] Clinically, these episodes may present as slowed responses, cognitive clouding, inattention, word-finding difficulty, or brief situational disorientation.[12,30,50] In some individuals, these manifestations lie on a continuum with presyncope and, more rarely, syncope, reflecting progressively greater cerebral hypoperfusion.[12,30,50] Symptoms usually improve after sitting or lying down and may therefore be misattributed to fatigue, aging, or medication effects in routine clinical practice.[2,9]

In older adults with impaired cerebrovascular autoregulation, even relatively small orthostatic BP reductions may translate into disproportionate changes in cerebral blood flow, amplifying symptom burden despite only modest systemic BP changes.[9,50] These transient disturbances can interfere with daily activities, increase the risk of falls, and undermine confidence in mobility, particularly among frail or multimorbid individuals.[3,4]

Vignette 3: The J-curve of cerebral hypoperfusion

An 85-year-old man treated with multiple antihypertensive agents developed episodic confusion and cognitive slowing shortly after standing. Supine BP was 118/64 mmHg, falling to 102/60 mmHg on standing. Although values did not meet diagnostic criteria for OH, symptoms were reproducible and clearly posture-related. Transcranial Doppler revealed marked reductions in cerebral blood flow during orthostasis. After cautious modification of the antihypertensive regimen, cognitive symptoms markedly improved. This case illustrates that clinically significant cerebral hypoperfusion may occur below conventional OH thresholds, especially in very old adults with limited autoregulatory reserve.

Key clinical takeaways

- Orthostatic cognitive vulnerability may occur below diagnostic BP thresholds.
- Posture-related cognitive symptoms should raise suspicion for cerebral hypoperfusion.
- BP target values in very old or frail adults should be individualized and reassessed over time.

Chronic Consequences: Longitudinal Associations with Cognitive Decline and Dementia

Population-based longitudinal studies underscore the long-term implications of orthostatic BP dysregulation for brain health, demonstrating associations with cognitive decline, dementia, and cerebrovascular disease.[11,29] In the prospective, population-based Rotterdam Study, lower baseline cerebral perfusion was linked to an increased risk of incident dementia, supporting the hypothesis that recurrent hypoperfusion may contribute to progressive brain injury.[11]

Cohort studies have reported that OH is linked to higher risks of dementia, stroke, and accelerated cognitive decline, suggesting that orthostatic BP instability may have lasting effects on brain structure and function. In the ARIC cohort, OH was associated with incident dementia (HR = 1.54, 95% CI: 1.20–1.97) and ischemic stroke (HR = 2.08, 95% CI: 1.65–2.62) over approximately 25 years of follow-up.[12] Additional population-based studies show that OH is associated with markers of white matter injury and small-vessel disease, suggesting a role for cerebral hypoperfusion.[11,29] A recent meta-analysis of longitudinal studies further confirmed poorer cognitive performance and a higher risk of cognitive impairment (OR = 1.28, 95% CI: 1.16–1.41) and incident dementia (HR = 1.27, 95% CI: 1.16–1.39) among individuals with OH.[51]

In very old adults, lower treated BP has been associated with steeper cognitive decline and higher mortality, highlighting the narrow margin between cardiovascular protection and cerebral hypoperfusion in advanced age.[48] Overall, OH may function both as a marker of vascular–autonomic vulnerability and as a potential contributor to cognitive decline.

Cerebral Perfusion, Autoregulation, and Blood Pressure Variability

Impaired cerebrovascular autoregulation and reduced vasodilatory reserve limit the brain's ability to maintain stable perfusion during orthostatic stress, such that cerebral hypoperfusion may occur even with relatively modest systemic BP declines.[9,50] In patients with nOH, mechanistic studies suggest reduced cerebral

blood flow during standing, underscoring the vulnerability of cerebral perfusion in this context.[50]

Beyond acute orthostatic changes, BP variability represents an additional dimension of cerebrovascular risk. Greater long-term BP variability has been associated with faster cognitive decline and a higher incidence of dementia, consistent with cumulative microvascular injury from unstable perfusion pressures.[52] Orthostatic BP instability likely interacts with these broader BP patterns, further amplifying cerebral vulnerability in older adults.[51–52]

Clinical Implications: Delirium, Mood, Frailty, and Treatment Thresholds

Clinically, orthostatic cognitive vulnerability is highly relevant to the care of older adults. Transient cerebral hypoperfusion may increase susceptibility to delirium during acute illness, perioperative stress, or hospitalization.[1,4,48] Fluctuating attention, slowed processing, and "on-off" cognitive performance in the upright position may mimic neurodegenerative progression if orthostatic BP is not assessed.[9,12,51] In many cases, symptoms improve with sitting or lying down, supporting orthostatic cerebral hypoperfusion rather than primary cognitive decline as the underlying mechanism.[9,13,30] (see Supplementary Vignette 6).

Recurrent orthostatic symptoms may lead to activity avoidance, fear of falling, and reduced participation in daily activities. Behavioral adaptations are often accompanied by social withdrawal and low mood, which may gradually lead to functional decline.[3–5]

In routine practice, orthostatic cognitive vulnerability in older adults is often recognized through subtle functional changes rather than formal cognitive testing. Patients may become quieter, and less alert when upright, hold on to furniture or caregivers, struggle to follow physiotherapy instructions, or experience postprandial drowsiness. Such seemingly nonspecific behaviors may reflect orthostatic cerebral hypoperfusion and should prompt targeted orthostatic BP assessment.

In frail older adults, orthostatic BP reductions may accelerate loss of intrinsic capacity, reflecting reduced physiological reserve. This may result in worsening mobility and independence, particularly when other geriatric syndromes are present.[4–5]

Importantly, clinically significant cognitive and functional effects may occur at BP levels that do not meet diagnostic criteria for OH.[6,47–48] This highlights the need for individualized BP targets and symptom-guided management in older adults, particularly in those with cognitive impairment, cerebrovascular disease, or high BP variability.[6,47–48] Recognition of orthostatic cognitive vulnerability therefore supports a shift away from rigid hemodynamic thresholds toward a function-centered approach that prioritizes preservation and improvement of cerebral perfusion, mobility, participation in daily life, and cognitive stability as key therapeutic goals.[2,9]

FUTURE PERSPECTIVES

Future work should improve the recognition and longitudinal characterization of orthostatic BP disorders in older adults, which remain frequently underdiagnosed and inconsistently monitored.[4,26] Routine clinic assessments capture only brief hemodynamic "snapshots" and may underestimate the dynamic, context-dependent nature of orthostatic BP responses, especially in older adults with frailty or reduced autonomic reserve.[4,26]

Clinically relevant BP declines and related symptoms, including cognitive fluctuations, often occur in everyday situations rather than during formal testing.[4] Morning volume depletion, prolonged standing, rehabilitation activities, and postprandial states can provoke transient episodes of hypotension or cerebral hypoperfusion that are easily missed at routine visits.[4,13] As a result, the daily impact of orthostatic BP instability in older adults is likely underestimated.[4]

Methodological advances may help bridge this gap. Repeated or continuous BP measurements (such as beat-to-beat recordings and structured home BP monitoring) may capture rapid transitions and short-duration phenomena that would otherwise be missed.[2,9,30] These approaches are particularly useful for characterizing BP variability over time, which has been increasingly linked to cognitive decline and dementia through cumulative cerebrovascular injury.[30,48,52]

Digital tools may further extend monitoring beyond the clinic. Wearable sensors and home BP devices with posture-linked recordings, combined with simple symptom diaries, can reveal patterns during morning routines, meals, and periods of activity.[2,9] When integrated thoughtfully into clinical care, these tools may support earlier recognition, improve patient engagement, and more timely therapeutic adjustment.[2,26]

Data-driven analytic approaches applied to longitudinal datasets may help move the field from binary BP

thresholds toward trajectory-based risk stratification, helping to explain why individuals with similar BP values experience divergent outcomes and informing more personalized treatment targets.[11,14,53–54] Beyond improved detection, future work should clarify which patients are most likely to benefit from targeted interventions. Strategies for deprescribing antihypertensive medications and setting individualized BP targets should be carefully evaluated, particularly in frail adults who may be especially susceptible to harm from cerebral hypoperfusion.[15,24,47] Pragmatic trials incorporating cognitive outcomes, falls, daily function, and quality of life, rather than BP alone, are especially needed.[11,24,48]

Finally, integrating orthostatic BP assessment into falls prevention, rehabilitation programs, memory clinics, and post-hospital transitions may support earlier recognition of reduced autonomic resilience.[4,26,54] Approaches aligned with frailty and intrinsic-capacity frameworks may facilitate pragmatic, function-focused interventions aimed at preserving mobility, cognition, and independence.[3,5–6,55–56]

Looking ahead, key priorities include better capturing real-world BP patterns, clarifying their links to cognitive and functional outcomes, and developing practical, validated approaches for everyday clinical care in older adults.

CONCLUSION

Orthostatic blood pressure disorders in older adults represent more than isolated abnormalities of blood pressure regulation. They reflect reduced physiological reserve arising from the interaction of age-related autonomic dysfunction, vascular changes, multimorbidity, and medication effects. As a result, orthostatic stress may lead to clinically important consequences even in the absence of large blood pressure declines.

Across phenotypes, orthostatic BP disorders are characterized by impaired cardiovascular and cerebrovascular compensation, leading to falls, mobility limitation, transient cerebral hypoperfusion, and cognitive vulnerability. They may also affect myocardial perfusion, limit optimization of heart failure therapy, and complicate hypertension management.

In frail and very old adults, symptoms and functional impairment may occur below conventional diagnostic thresholds, underscoring the limitations of rigid hemodynamic cutoffs. Accurate clinical assessment therefore requires standardized yet flexible evaluation strategies that incorporate supine and upright measurements, heart-rate responses, repeated testing, and careful attention to real-world triggers. Recognition of overlapping orthostatic phenotypes is essential to avoid misclassification and unsafe management decisions.

Management in older adults should prioritize upright tolerance, systemic and cerebral perfusion, mobility, and cognitive stability rather than blood pressure normalization alone. A phenotype-oriented, function-centered approach, combining medication review, non-pharmacologic strategies, and cautious pharmacologic therapy, offers the greatest potential to improve outcomes while minimizing harm. Integrating orthostatic assessment into the routine care of older adults supports earlier identification of impaired hemodynamic reserve, informs individualized treatment decisions, and may help preserve upright perfusion, mobility, cognition, independence, and quality of life.

CONFLICT OF INTERESTS

None.

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