

Orthostatic hypotension

A primary care primer for assessment and treatment

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Orthostatic hypotension (OH), defined as a decline in blood pressure when a person moves from a supine to sitting or standing position, is a common physical finding in the primary care setting. It is associated with several medical conditions and its prevalence increases with age. Treatment is specific to cause. Drug-induced OH often can be alleviated by reducing dosage or completely changing medications. OH secondary to autonomic insufficiency or neurogenic causes remains a challenge to manage, and a combination of non-pharmacologic and pharmacologic measures are needed. Recommendations are made for preventive measures, patient and caregiver education, and non-pharmacologic and pharmacologic approaches to treatment. Approaches to managing OH in conjunction with hypertension are also discussed.

Sclater A, Alagiakrishnan K. Orthostatic hypotension: A primary care primer for assessment and treatment. *Geriatrics* 2004; 59(Aug):22-27.

Key words: Postural hypotension • Bradbury-Eggleston Syndrome
Shy Drager Syndrome • hypertension • prevention

Orthostatic hypotension (OH) is defined by the Consensus Committee of the American Autonomic Society and the American Academy of Neurology as a decline of >20 mm Hg in systolic blood pressure (SBP) or a decline of >10 mm Hg in diastolic blood pressure (DBP) that occurs when a person moves from a supine to a sit-

ting or standing position. The decrease must be present within 3 minutes after the postural change.¹

Prevalence of OH increases with age. In the Cardiovascular Health Study, prevalence of OH was 14.8% for those age 65 to 69 and 26% for those age 85 and older.² In individuals whose SBP is greater than 160 mm Hg, prevalence of OH is high regardless of age. OH occurs in 14.6% of community-dwelling older adults³ and in 52% of nursing home residents.⁴

Postural changes in BP

The normal BP response that occurs when an individual moves from a supine to a standing position is a small reduction (<10 mm Hg) in SBP and a small increase in DBP (approximately 2.5 mm Hg). When a person stands up from a supine position, approximately 500 to 700 mL of blood is pooled in the lower extremities and in the splanchnic and pulmonary circulations. In response to

the decrease in venous return to the heart, there is a transient reduction in cardiac output and stimulation of the baroreceptors, which reflexively increases sympathetic tone and vascular resistance and inhibits parasympathetic activity resulting in an increased heart rate.

Orthostatic hypotension can be:

- Asymptomatic, where BP changes occur without any symptoms.
- Symptomatic, in which symptoms such as dizziness and faintness occur with BP changes.
- Acute or reversible, typically caused by fluid volume loss or medication use.
- Chronic or irreversible, caused by endocrine and neurogenic factors.

Associated risk factors

Patients with medical conditions such as diabetes mellitus, hypertension, alcoholism, cardiac diseases, central nervous system disorders, dementia, venous diseases, and amyloidosis are at increased risk for OH.

Pathogenesis

Aging is associated with impairment of the baroreflex. Thus, OH in older patients results from an excessive reduction in blood volume when patients are upright or from inadequate cardiovascular compensation. Changes in baroreceptor sensitivity, heart rate response, vascular compliance, vasopressin, renin, angiotensin, and renal concentrating abilities all contribute to increased risk for OH.

OH also can result from diseases

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Disclosures: The authors have no real or apparent conflicts of interest related to the subject under discussion.

where portions of the autonomic reflex arc (afferent, central, or efferent) are impaired. Autonomic insufficiency results in depressed myocardial contractility and vascular responsiveness resulting in lowered BP due to inadequate homeostatic mechanisms.

Causes of OH

Multiple conditions can cause OH, including acute and chronic causes as well as non-neurogenic and neurogenic conditions. Non-neurogenic conditions that can predispose to OH include cardiovascular and endocrine conditions, volume depletion, and medications. Key neurogenic causes include primary or secondary autonomic insufficiency (table 1).

Assessment

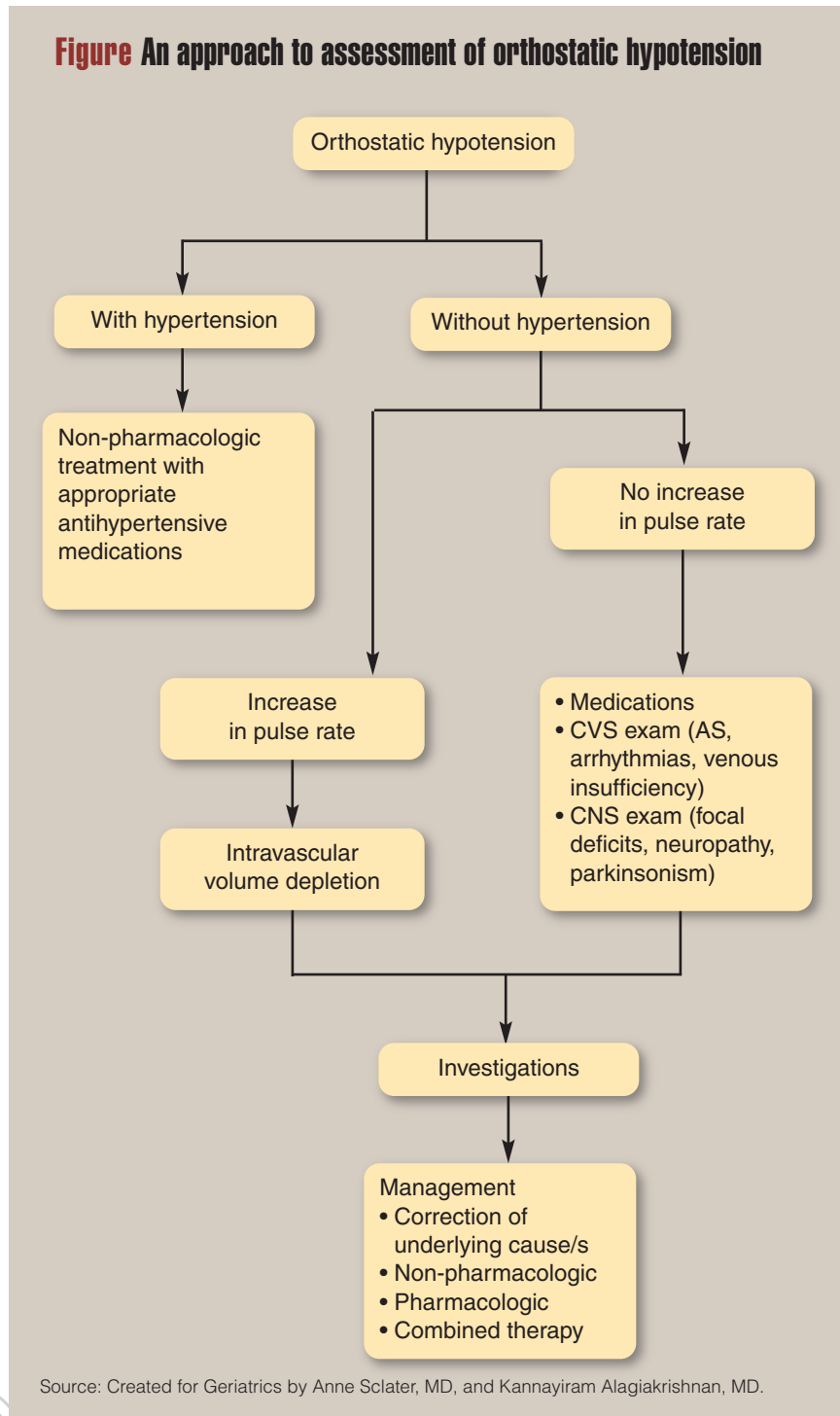
The initial evaluation should investigate reversible or treatable causes (especially medication effects, reduced blood volume, and dehydration) (figure).

History. Patients may complain of fatigue, lightheadedness, falls, and visual blurring when there is a mild reduction in cerebral blood flow; syncope, transient ischemic attacks, or generalized seizures may occur in more severe cerebral hypoperfusion. Muscle hypoperfusion can lead to neck pain, lower back pain, and calf claudication; cardiac hypoperfusion leads to angina pectoris.

The history should also focus on medications, alcohol use, and a review of autonomic, neurologic, cardiovascular, and endocrine systems. In the autonomic review, the physician should look for decreased sweating, symptoms related to gastroparesis, incontinence, and impotence. In older patients with autonomic failure, symptoms of OH often arise after excessive nocturia or after a meal, and may worsen during exercise. However, most patients are asymptomatic.

Physical examination. The standard technique for measuring orthostatic blood pressure and pulse rate includes

- measuring blood pressure and pulse rate after 5 minutes of supine rest, and
- repeating the measurement at 1 and 3 minutes after standing.



3 minutes after standing. A decrease in SBP of ≥ 20 mm Hg, or a decrease in DBP of ≥ 10 mm Hg with or without an increase in pulse rate is considered an abnormal response. The heart rate response to postural change can provide important information about the cause of OH. Minimal change in heart rate (< 10 bpm) following supine to

standing posture in the presence of hypotension indicates baroreceptor reflex impairment, whereas tachycardia (> 20 bpm) indicates volume depletion.

Baroreceptor sensitivity is impaired with old age, so the absence of cardioacceleration does not rule out volume depletion in an older patient. If the patient is unable to stand or is

Orthostatic hypotension

Table 1 Causes of orthostatic hypotension

Acute or reversible

Dehydration
Drugs
Deconditioning

Chronic or non-reversible

Cardiac failure
Diabetes mellitus
Adrenal insufficiency
Parkinsonism
Pure autonomic failure
Multiple system atrophy

Non-neurogenic

- Cardiovascular: myocardial infarction, aortic stenosis, constrictive pericarditis, advanced cardiac failure, hypertrophic obstructive cardiomyopathy (HOCM), arrhythmias (tachy and brady), large varicose veins
- Endocrine and renal: adrenal insufficiency, diabetes insipidus, hypoaldosteronism, renal concentrating defect
- Venous pooling: alcohol, postprandial dilation of splanchnic blood vessels, hot environment, fever, prolonged standing
- Reduced intravascular volume: dehydration, hemorrhage, burns, salt-losing nephropathy, adrenal insufficiency, diabetes insipidus

Neurogenic

- Primary causes: pure autonomic failure, multiple system atrophy
- Common causes: secondary autonomic failure due to stroke, diabetes, alcoholic polyneuropathy, idiopathic parkinsonism, amyloid neuropathy, pernicious anemia

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bedridden, a sitting measurement can be taken, but the sensitivity of the test may be reduced. OH is more likely to occur before breakfast than at any other time of day, but the risk increases immediately following any meal in patients who may have both orthostatic and postprandial hypotension occurring together. OH is more likely to be reproducible in the morning than in the afternoon so it is preferable to evaluate for OH in the morning.

A single reading is not sufficient to diagnose OH. The reproducibility of OH measurements is more likely to occur if autonomic function is abnormal. Only two-thirds of patients have reproducible postural blood pressure responses on consecutive mornings, due to day-to-day variation in intravascular volume and autonomic tone.

Cardiovascular. Assess fluid volume status clinically by an examination of jugular venous pressure. Heart auscultation should be performed to identify the presence of aortic stenosis, murmurs, and arrhythmias.

Neurologic. The neurologic examination should include mental status tests to evaluate for dementia, cranial nerve involvement in stroke, motor examination for stroke, and sensory evaluation for neuropathies and Parkinsonism features such as bradykinesia, rigidity, and tremor.

Laboratory and clinical testing. The following diagnostic tests are recommended based on need:

- CBC for anemia and when stool guaiac is positive
- BUN and serum creatinine for dehydration
- fasting and 2-hour postprandial blood glucose test for diabetes
- ECG for irregular rhythms
- CT or MRI to exclude CNS disorders.

Autonomic function tests that can be performed in select cases include:

- measuring the heart rate response to a change from supine to a standing position and valsalva maneuver
- spectral estimation of heart rate variability

- cold pressor test
- measurement of plasma norepinephrine levels in response to a postural change.

These tests are non-invasive and performed in a specialized testing laboratory. Tilt-table testing is also helpful in patients with syncope of unknown cause.

Treatment

Treatment of OH is individualized. Often, treatment is implemented to improve the functional capacity and sense of well-being rather than to reach a fixed value of arterial pressure. The goal of treatment is to make the patient symptom-free and as ambulatory as possible.

Nonpharmacologic treatments

Advise patient and caregiver that:

- Patients need to maintain adequate fluid intake, limit or avoid alcohol, and exercise regularly in horizontal position (eg, swimming and bed exercises such as moving feet up and down to activate calf muscle pump) when feasible.
- Patients need to change posture slowly and avoid standing still. When standing for a prolonged time, they should cross and uncross the legs a few times to increase the venous return.
- Patients on prolonged bed rest need to increase the amount of time they spend sitting up each day.
- Fitted elastic hose or compression stockings may enhance cardiac output and BP on standing, thereby reducing venous pooling in the legs. Hose or stockings must be worn all day to be useful. If patients have difficulty putting them on and taking them off, then they may not be practical. Patients may find these uncomfortable during hot weather.
- Patients need to eat small meals frequently and avoid standing up suddenly after eating.
- Patients need to avoid hot showers or excessive heat.
- Patients need to avoid straining during micturition and defecation.
- Patients without hypertension need to increase salt intake.

Pharmacologic treatments

Midodrine. This sympathomimetic vasoconstrictor acts directly on resistance vessels. After oral administration, pro-drug midodrine undergoes enzymatic hydrolysis in the liver to the selective α_1 -adrenoceptor agonist desglymidodrine. The drug significantly increases 1-minute standing SBP compared with placebo and improves clinical symptoms of OH including dizziness, lightheadedness, and syncope.⁵ Midodrine is well-tolerated; the most commonly reported side effects include piloerection, paresthesias, urinary retention, pruritus, and chills. Up to 25% of patients taking this medication experience supine hypertension, which can be reduced by taking the final daily dose at least 4 hours before bedtime.

A trial dose of 2.5 to 5 mg is given and the effects on blood pressure in supine and standing positions are determined after 1 hour. In patients with gastroparesis due to autonomic insufficiency, the peak effect may occur later, so blood pressure monitoring is conducted after 2 to 3 hours. The dose is titrated according to the patient's response. The starting dose is 2.5 mg taken at breakfast and lunch or three times daily; the dose is increased in 2.5 mg steps daily until satisfactory response occurs or a dosage of 30 to 40 mg/d is achieved. In a study on neurogenic hypotension, improvement was noted in 47% of patients receiving midodrine compared with 28% of patients taking placebo. The 10 mg dose of midodrine increased SBP by a mean of 18 mm Hg.⁶

Midodrine is contraindicated in patients with severe heart disease such as heartblocks, acute renal disease, urinary retention, pheochromocytoma, or thyrotoxicosis.

Fludrocortisone. This mineralocorticoid reduces salt loss and expands plasma volume. The initial dose is 0.1 mg/d and it is increased at weekly intervals up to 0.4 to 1 mg/d. The main side effects include edema, hypokalemia, and congestive heart failure. A weight gain of 2 to 5 pounds is expected, and some degree of pedal

edema is desirable. One study of older patients (mean age 80) showed poor tolerance to fludrocortisone.⁷ During follow-up, 20% of participants died of unrelated causes. Of the remainder, 33% discontinued fludrocortisone after a mean of 5 months due to hypertension, heart failure, or depression.

Older patients in particular need to be monitored for signs and symptoms of fluid overload.

Treatment of hypertension suggests that lowering blood pressure adequately decreases the prevalence of OH.

Dihydroergotamine. This sympathomimetic vasoconstrictor may increase venous return without increasing arterial pressure. Long-term use of this medication in older patients should be avoided because of the potential serious adverse effects associated with ergot alkaloids, such as severe vasospasm, gangrene, and convulsions.

Erythropoietin. This hematopoietic agent increases hemoglobin and blood volume. It has been studied in younger patients with OH caused by autonomic failure and accompanied by documented deficits in red blood cell mass, but there is only one case report showing improvement in older patients.⁸ Usefulness of erythropoietin therapy in older patients has yet to be determined.

Octreotide. This somatostatin analogue and peptide release inhibitor produces direct vasoconstriction and increases cardiac output. It has been used in patients with diabetes, pure autonomic failure, and multiple system atrophy, as well as OH. The dose is 5 to 50 mcg subcutaneously. Side effects include nausea and abdominal cramps. Therapy with octreotide is expensive and re-

quires frequent subcutaneous injections.

Combination therapy. A combination of non-drug and drug therapies can decrease OH. In severe cases, a combination of midodrine and octreotide has been used with encouraging results.⁹

Pacemaker therapy. Atrial tachypacing is a treatment option for patients with OH and bradycardia. Severe OH can be treated successfully with dual chamber pacemakers.

Managing OH and hypertension

The Epicardian Study (an examination of 2,700 patients age >65) found that adequate control of blood pressure itself reduces the incidence of OH associated with hypertension.¹⁰ The changes in frequency of OH before and after treatment of hypertension suggests that lowering blood pressure adequately with treatment decreases the prevalence of OH.¹¹

Antihypertensives that can cause OH, such as diuretics, alpha blockers and central alpha agonists, may be replaced by agents that are associated with a lower frequency of OH. These include beta-adrenergic agonists, angiotensin converting enzyme (ACE) inhibitors, and selected calcium channel blockers.

OH may be seen with beta blockers that have some alpha-blocking properties, such as labetalol. The reported prevalence of OH with labetalol is 1.4%.¹² A comparison of enalapril (5 to 20 mg/d) with long-acting nifedipine (30 to 90 mg/d) found enalapril reduces the number of OH episodes, whereas long-acting nifedipine increases the phenomenon.¹³ Enalapril and nifedipine were equipotent in reducing supine blood pressure levels. In this study, the crossover of enalapril and nifedipine reproduced the hypotensive effect and reversed the postural effect.

Common co-morbidities

Immobility and prolonged bed rest. Complete bed rest for as little as a few weeks may impair the ability to adjust to an upright position even in healthy older persons. During the initial weeks of immobilization, antidiuretic hormone

Table 2 Consequences of orthostatic hypotension

- Dizziness
- Syncope
- Falls
- Fractures
- Loss of independence
- Fear of falling
- Impaired quality of life
- Increased mortality

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release diminishes, leading to increased diuresis and a significant decrease of plasma volume, which can be up to 10% in the first 2 weeks and more than 20% thereafter. In older individuals experiencing prolonged immobilization, the increase in cardiac output rather than vasoconstriction protects them from OH.

Postural blood pressure and pulse rate should be monitored in bedridden older patients when patients are mobilized (eg, leave the bed for the first time). Strengthening exercises for abdominal and leg muscles, performed in the bed prior to mobilizations, promote venous blood return most efficiently, so early mobilization is recommended.

Falls. Among nursing home residents with previous falls, OH is associated with a two-fold increase in subsequent falls.¹⁴ In one study, falling was the presenting feature in 64% of subjects with OH.¹⁵ A study of the cause of falls revealed OH as the primary cause in 16% and as a possible contributing cause in an additional 26%.¹⁶ All patients who experience a fall should be routinely evaluated for OH.

Syncope. In patients with syncope 20% to 30% have OH.¹⁷ Syncope is seen in 8% of OH patients. Diagnostic assessment on all syncopal patients should include postural blood pressure measurement. This practice is rapid and cost-effective.

Diabetes mellitus. OH may be the first sign of autonomic nervous system dis-

ease in diabetic patients, but autonomic involvement is usually accompanied by distal symmetrical polyneuropathy. OH appears to indicate poor prognosis in diabetic patients with hypertension. Along with OH, these patients may also have gastroparesis, diabeticorum, diabetic cystopathy or detrusor hyporeflexia, and impotence.

Parkinson's Disease. Possible causes of OH in patients with Parkinsonian features are Parkinson's Disease with autonomic failure, side effects of antiparkinson medications (levodopa and bromocriptine), multiple system atrophy (OH with Parkinsonism, cerebellar dysfunction, or pyramidal signs), concomitant diseases with Parkinsonism causing OH (eg, diabetes mellitus), and drugs such as diuretics or alpha blockers for benign prostatic hyperplasia.

Stroke. Highly variable postural blood pressure may directly affect cerebral blood flow and may be an indicator of future stroke. The Atherosclerosis Risk in Communities (ARIC) longitudinal study found that OH (systolic, diastolic, or both) is a risk factor for stroke in a subset of middle-aged adults.¹⁸

Cognitive impairment. The effect of OH on cognition is variable. A 5-year follow-up study of healthy older women described OH as a risk factor for cognitive decline.¹⁹ During the course of Alzheimer's dementia, OH can contribute to frontal brain changes and may exacerbate the disease.

Passant et al found OH/low blood pressure with dementia in 39% to 52% of the study group; in 38%, the decrease in SBP was more than 40 mm Hg.²⁰ However, Viramo et al found that OH was not associated with cognitive decline, nor did it predict cognitive deterioration in older adults during a 2-year follow-up.²¹

Hypertension. OH is associated with isolated systolic hypertension.² The presence of severe OH with untreated hypertension is a common finding in pheochromocytoma.

All antihypertensive drugs have the potential for producing OH. Its development is most commonly associated

with diuretics, vasodilators, and alpha blockers; there is less association with calcium channel blockers, ACE inhibitors, and beta-adrenergic blockers.

Kario et al described an association of OH with non dippers (nocturnal hypertension).²² A marked orthostatic decrease in pretreatment blood pressure was seen in hypertensive patients with target organ damage and indicated a poor prognosis.

Successful therapy for hypertension may enhance baroreceptor sensitivity, increase vascular compliance, and reduce the threshold for cerebral autoregulation. The appropriate treatment of hypertension may be the best method for treating OH with hypertension.

Prognosis

The prognosis for individuals with OH depends on the underlying cause. OH is an independent predictor of frailty and cardiovascular and all-cause mortality.^{2,23} The presence of diastolic OH at 1 minute and systolic OH at 3 minutes after standing predicts vascular death in older patients.²⁴ OH appears to indicate poor prognosis in diabetic patients with hypertension in whom the risk of death is increased.

Prevention of OH

Patient and family education about causes, consequences (table 2), and management of symptoms is important. Preventive strategies include:

Diet

- Increase daily intake of sodium (5 to 10 gm/d) and water (not in patients with hypertension and heart failure).

- Eat small, frequent low-carbohydrate meals (6 small meals instead of 3 large meals) to prevent postprandial worsening of OH.

- Increase dietary fiber to prevent constipation.

- Increase caffeine intake, as an adenosine receptor blocker (adenosine causes splanchnic postprandial hypotension); drink coffee after meals to help prevent the postprandial OH.

- Drink at least 6 to 8 glasses of water to prevent dehydration; avoid alco-

hol (because it produces significant splanchnic vasodilation).

- Increase intake of salt and fluids during times of extreme heat and febrile illness.

Physical activity

- Exercise the calf muscles before sitting up, and sit on the edge of the bed for a few minutes before standing to give the body time to adjust to the postural change and help the blood flow back to the heart.

- Avoid bending at the waist to pick up items from the floor or to reach for something on a lower shelf. If possible, squat at the knees and keep the head above the level of the heart.

- Consider wearing waist-length elastic stockings to prevent venous pooling in the legs.

- Use a urinal or bedside commode to reduce the need to get up quickly or rely on assistance in order to use the bathroom.²⁵

Conclusion

Orthostatic hypotension is commonly seen in older adults, and the condition increases with age. Assessing patients for OH requires measuring blood pressure and pulse rate after supine rest and repeating the measurement at 1 and 3 minutes after standing. Chronic OH can be a severe, debilitating disorder and early detection can improve quality of life. Evaluation of postural changes in blood pressure in the clinical assessment of older patients should be routine. ☐

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Acknowledgements: We would like to thank Dr. Keith Sclater and Dr. Duncan Robertson for reviewing this manuscript.

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