MSD MANUAL
Professional Version

Orthostatic Hypotension

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Orthostatic (postural) hypotension is an excessive fall in blood pressure (BP) when an upright position is assumed. The consensus definition is a drop of > 20 mm Hg systolic, > 10 mm Hg diastolic, or both (1). Symptoms of faintness, light-headedness, dizziness, confusion, or blurred vision occur within seconds to a few minutes of standing and resolve rapidly on lying down. Some patients experience falls, syncope, or even rarely generalized seizures. Exercise or a heavy meal may exacerbate symptoms. Most other associated symptoms and signs relate to the cause.

Orthostatic hypotension is a manifestation of abnormal BP regulation due to various conditions, not a specific disorder. Evidence suggests that disorders of postural hemodynamic control increase risk of cardiovascular disease and all-cause mortality (2, 3).

Postural orthostatic tachycardia syndrome (POTS)

POTS (also called postural autonomic tachycardia, or chronic or idiopathic orthostatic intolerance) is a syndrome of orthostatic intolerance in younger patients. POTS is defined by a heart rate of ≥ 120 beats/minute or an increase of ≥ 30 beats/minute when a patient moves from supine to a standing position. Various symptoms (eg, fatigue, light-headedness, exercise intolerance, cognitive impairment) and tachycardia occur upon standing; however, there is little or no fall in BP. The reason for symptoms is unclear.

General references

1. Freeman R, Wieling W, Axelrod FB, et al: Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. *Clin Auton Res* 21(2):69–72, 2011. doi:10.1007/s10286-011-0119-5
2. Fedorowski A, Stavenow L, Hedblad B, et al: Orthostatic hypotension predicts all-cause mortality and coronary events in middle-aged individuals (The Malmo Preventive Project). *Eur Heart J* 31(1):85–91, 2010. doi:10.1093/eurheartj/ehp329
3. Verwoert GC, Mattace-Raso FU, Hofman A, et al: Orthostatic hypotension and risk of cardiovascular disease in elderly people: the Rotterdam study. *J Am Geriatr Soc* 56(10):1816–1820, 2008. doi:10.1111/j.1532-5415.2008.01946.x

Pathophysiology of Orthostatic Hypotension

Normally, the gravitational stress of suddenly standing causes blood (½ to 1 L) to pool in the veins of the legs and trunk. The subsequent transient decrease in venous return reduces cardiac output and thus BP. In response, baroreceptors in the aortic arch and carotid sinus activate autonomic reflexes to rapidly return BP to normal. The sympathetic nervous system increases heart rate and contractility and increases vasomotor tone of the capacitance vessels. Simultaneous parasympathetic (vagal) inhibition also increases heart rate. In most people, changes in BP and heart rate upon standing are minimal and transient, and symptoms do not occur.

With continued standing, activation of the renin-angiotensin-aldosterone system and vasopressin (antidiuretic hormone [ADH]) secretion cause sodium and water retention and increase circulating blood volume.

Etiology of Orthostatic Hypotension

Homeostatic mechanisms may be inadequate to restore low BP if afferent, central, or efferent portions of the autonomic reflex arc are impaired by disorders or medications, if myocardial contractility or vascular responsiveness is depressed, if hypovolemia is present, or if hormonal responses are faulty (see table Causes of Orthostatic Hypotension).

Causes differ depending on whether symptoms are acute or chronic.

The most common causes of acute orthostatic hypotension include

- Hypovolemia
- Medications
- Prolonged bed rest

The most common causes of chronic orthostatic hypotension include

- Age-related changes in BP regulation
- Medications
- Autonomic dysfunction

Postprandial orthostatic hypotension is also common. It may be caused by the insulin response to high-carbohydrate meals and blood pooling in the gastrointestinal tract; this condition is worsened by alcohol intake.

TABLE

Causes of Orthostatic Hypotension

Cause	Examples
Neurologic (involving autonomic dysfunction)	
Central	Multiple system atrophy Parkinson disease Strokes (multiple)
Spinal cord	Tabes dorsalis Transverse myelitis Tumors
Peripheral	Amyloidosis Diabetic, alcohol-related, or nutritional neuropathy Familial dysautonomia (Riley-Day syndrome) Guillain-Barré syndrome Paraneoplastic syndromes Pure autonomic failure Surgical sympathectomy
Cardiovascular	
Hypovolemia	Dehydration Hemorrhage Hyperthyroidism Adrenal insufficiency
Impaired vasomotor tone	Bed rest (prolonged) Hypokalemia
Impaired cardiac output	Aortic stenosis Left ventricular outflow obstruction (ie, hypertrophic obstructive cardiomyopathy) Constrictive pericarditis, pericardial effusion with tamponade Heart failure Myocardial infarction

	Tachyarrhythmias or bradyarrhythmias
Other	Hyperaldosteronism* Peripheral venous insufficiency Pheochromocytoma*
Medications and drugs	
Vasodilators	Calcium channel blockers Nitrates
Autonomically active	Alpha-blockers (eg, terazosin, doxazosin, phenoxybenzamine) Antihypertensives (eg, clonidine, methyldopa, [rarely] beta-blockers)† Antipsychotics (particularly phenothiazines) Monoamine oxidase inhibitors (MAOIs) Tricyclic or tetracyclic antidepressants
Other	Alcohol Barbiturates Diuretics (eg, loop diuretics such as furosemide, thiazide diuretics such as hydrochlorothiazide) Levodopa (in Parkinson disease [rarely]) Quinidine Vincristine (neurotoxic)

* Disorder causes supine hypertension.

† Symptoms are more common when treatment is begun.

Evaluation of Orthostatic Hypotension

Orthostatic hypotension is diagnosed when systolic BP drops by ≥ 20 mm Hg or diastolic BP drops by ≥ 10 mm Hg within 3 minutes of standing. Once orthostatic hypotension is diagnosed, a cause must be sought.

History

History of present illness should identify the duration and severity (eg, whether associated with syncope or falls) of symptoms. The patient is asked about known triggers (eg, medications, bed rest,

fluid loss) and the relationship of symptoms to meals.

Review of symptoms seeks symptoms of causative disorders, particularly symptoms of autonomic insufficiency such as visual impairment (due to mydriasis and loss of accommodation), incontinence or urinary retention, constipation, heat intolerance (due to impaired sweating), and erectile dysfunction. Other important symptoms include tremor, rigidity, and difficulty walking (Parkinson disease, multiple system atrophy); weakness and fatigue (adrenal insufficiency, anemia); and black, tarry stool (gastrointestinal hemorrhage). Other symptoms of neurologic and cardiovascular disorders and cancer are noted.

Past medical history should identify known potential causes, including diabetes, Parkinson disease, and cancer (ie, causing a paraneoplastic syndrome). The medication profile should be reviewed for offending prescription medications (see table Causes of Orthostatic Hypotension), particularly antihypertensives and nitrates. A family history of orthostatic symptoms suggests possible familial dysautonomia.

Physical examination

BP and heart rate are measured after 5 minutes supine and at 1 and 3 minutes after standing; patients unable to stand may be assessed while sitting upright. Hypotension without a compensatory increase in heart rate (< 10 beats/minute) suggests autonomic impairment. Marked increase (to > 100 beats/minute or by > 30 beats/minute) suggests hypovolemia or, if symptoms develop without hypotension, POTS.

The skin and mucosae are inspected for signs of dehydration and for pigment changes suggestive of Addison disease (eg, hyperpigmented areas, vitiligo).

The extent of physical examination is guided by clinical suspicion. A rectal examination is done to evaluate for gastrointestinal bleeding.

A detailed neurologic examination should include evaluation for peripheral neuropathy (eg, abnormalities of strength, sensation, and deep tendon reflexes).

Red flags

Certain findings suggest a more serious etiology:

- Bloody or heme-positive stool
- Abnormal neurologic examination

Interpretation of findings

In patients with acute symptoms, the most common causes—medications, bed rest, and volume depletion—are often apparent clinically.

In patients with chronic symptoms, an important goal is to detect any neurologic disorder causing autonomic dysfunction. Patients with movement abnormalities may have Parkinson disease or multiple system atrophy. Patients with findings of peripheral neuropathy may have an apparent cause (eg, diabetes, alcohol use disorder), but a paraneoplastic syndrome due to an occult cancer and amyloidosis

must be considered. Patients who have only peripheral autonomic symptoms may have pure autonomic failure.

Testing

ECG, serum electrolytes, creatinine, and glucose are routinely checked. However, these and other tests are usually of little benefit unless suggested by specific symptoms.

The dose of a suspected medication may be reduced or the medication stopped to confirm the medication as the cause.

Tilt table testing may be done when autonomic dysfunction is suspected; it gives more consistent results than supine and upright BP assessment and eliminates augmentation of venous return by leg muscle contraction. The patient may remain upright for 30 to 45 minutes of BP assessment.

Patients with autonomic symptoms or signs require further evaluation for diabetes, Parkinson disease, and possibly multiple system atrophy and pure autonomic failure. Testing for pure autonomic failure may require plasma norepinephrine or vasopressin (ADH) measurements with the patient supine and upright.

Autonomic function can also be evaluated with bedside cardiac monitoring, although this test is not often done. When the autonomic system is intact, heart rate increases in response to inspiration. The heart is monitored as the patient breathes slowly and deeply (about a 5-second inspiration and a 7-second expiration) for 1 minute. The longest inter-beat (R-R) interval during expiration is normally at least 1.15 times the minimum R-R interval during inspiration; a shorter interval suggests autonomic dysfunction, but this response to inspiration may decrease with aging. A similar variation in R-R interval should exist between rest and a 10- to 15-second Valsalva maneuver.

Treatment of Orthostatic Hypotension

Nonpharmacologic interventions

Patients requiring prolonged bed rest should sit up each day and exercise in bed when possible. Patients should rise slowly from a recumbent or sitting position, consume adequate fluids, limit or avoid alcohol, and exercise regularly when feasible. Regular modest-intensity exercise promotes overall vascular tone and reduces venous pooling. Older patients should avoid prolonged standing. Sleeping with the head of the bed raised may relieve symptoms by promoting sodium retention and reducing nocturnal diuresis.

Postprandial hypotension can often be prevented by reducing the size and carbohydrate content of meals, minimizing alcohol intake, and avoiding sudden standing after meals.

Waist-high fitted elastic hose may increase venous return, cardiac output, and BP after standing. In severe cases, inflatable aviator-type antigravity suits, although often poorly tolerated, may be needed to produce adequate leg and abdominal counterpressure.

Increasing sodium and water intake may expand intravascular volume and lessen symptoms. In the absence of heart failure or hypertension, sodium intake can be increased to 6 to 10 g daily by liberally salting food or taking sodium chloride tablets. This approach risks heart failure, particularly in older patients and in patients with impaired myocardial function; development of dependent edema without heart failure does not contraindicate continuing this approach.

Pharmacotherapy

Fludrocortisone, a mineralocorticoid, causes sodium retention, which expands plasma volume, and often lessens symptoms but is effective only when sodium intake is adequate. Dosage is 0.1 mg orally at bedtime, increased weekly to 1 mg or until peripheral edema occurs. This medication may also improve the peripheral vasoconstrictor response to sympathetic stimulation. Supine hypertension, heart failure, and hypokalemia may occur; potassium supplements may be needed.

Midodrine, a peripheral alpha-agonist that is both an arterial and a venous constrictor, is often effective. Dosage is 2.5 mg to 10 mg orally 3 times a day. Adverse effects include paresthesias and itching (probably secondary to piloerection). This medication is not recommended for patients with coronary artery or peripheral arterial disease.

Nonsteroidal anti-inflammatory drugs (NSAIDs—eg, indomethacin 25 to 50 mg orally 3 times a day) may inhibit prostaglandin-induced vasodilation, increasing peripheral vascular resistance. However, NSAIDs may cause gastrointestinal symptoms and unwanted vasopressor reactions (reported with concurrent use of indomethacin and sympathomimetic medications).

Droxidopa, a norepinephrine precursor, may be beneficial for autonomic dysfunction.

Propranolol or other beta-blockers may enhance the beneficial effects of sodium and mineralocorticoid therapy. Beta-blockade with propranolol leads to unopposed alpha-adrenergic peripheral vascular vasoconstriction, preventing the vasodilation that occurs when some patients stand.

Pyridostigmine (1) and octreotide (2) have been effective in small clinical studies.

Treatment references

1. Singer W, Sandroni P, Opfer-Gehrking TL, et al: Pyridostigmine treatment trial in neurogenic orthostatic hypotension. *Arch Neurol* 63(4):513–518, 2006. doi:10.1001/archneur.63.4.noc50340
2. Hoeldtke RD, Israel BC: Treatment of orthostatic hypotension with octreotide. *J Clin Endocrinol Metab* 68(6):1051–1059, 1989. doi:10.1210/jcem-68-6-1051

Geriatrics Essentials: Orthostatic Hypotension

Orthostatic hypotension occurs in approximately 15 to 20% of older adults (1). It is more common among people with coexisting disorders, especially hypertension, and among residents of long-term care facilities. Many falls may result from unrecognized orthostatic hypotension.

The increased incidence in older adults is due to decreased baroreceptor responsiveness plus decreased arterial compliance. Decreased baroreceptor responsiveness delays cardioacceleration and peripheral vasoconstriction in response to standing. Paradoxically, hypertension may contribute to poor baroreceptor sensitivity, increasing vulnerability to orthostatic hypotension. Older adults also have decreased resting parasympathetic tone, so that cardioacceleration due to reflex vagal withdrawal is lessened.

Geriatrics essentials reference

1. Rutan GH, Hermanson B, Bild DE, Kittner SJ, LaBaw F, Tell GS: Orthostatic hypotension in older adults. The Cardiovascular Health Study. CHS Collaborative Research Group. *Hypertension* 19(6 Pt 1):508–519, 1992. doi:10.1161/01.hyp.19.6.508

Key Points

- **Orthostatic hypotension typically involves volume depletion or autonomic dysfunction.**
- **Some degree of autonomic dysfunction is common in older adults, but neurologic disorders must be ruled out.**
- **Tilt table testing is sometimes done.**
- **Treatment involves physical measures to reduce venous pooling, increased sodium intake, and sometimes fludrocortisone or midodrine.**

More Information

The following English-language resource may be useful. Please note that THE MANUAL is not responsible for the content of this resource.

Freeman R, Abuzinadah AR, Gibbons C, et al: Orthostatic hypotension: JACC state-of-the-art review. *J Am Coll Cardiol* 72(11):1294–1309, 2018. doi: 10.1016/j.jacc.2018.05.079