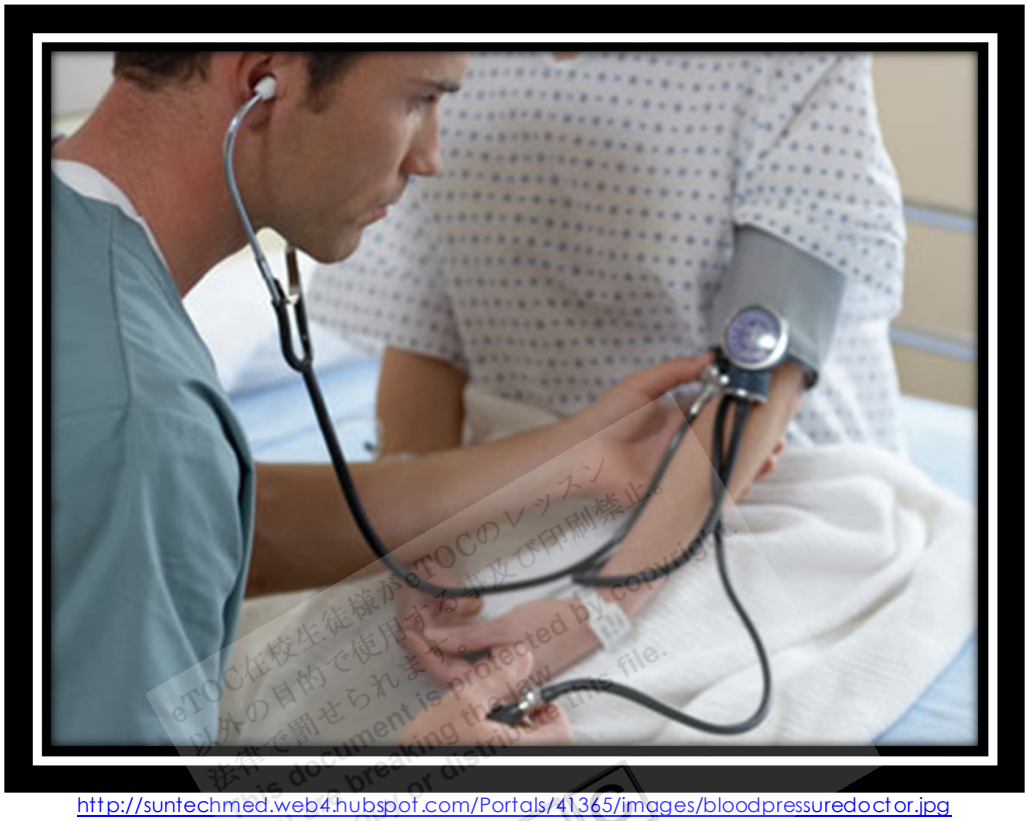
Orthostatic Hypotension



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Orthostatic (postural) hypotension is an excessive fall in BP when an upright position is assumed. The consensus definition is a drop of > 20 mm Hg systolic, 10 mm Hg diastolic, or both. 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 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.

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. Various symptoms (eg, fatigue, light-headedness, exercise intolerance, cognitive impairment) and tachycardia occur with standing; however, there is little or no fall in BP. The reason for symptoms is unclear.

Pathophysiology

Normally, the **gravitational stress** of suddenly standing causes blood (½ to 1 L) to pool in the capacitance 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 bodies activate autonomic reflexes to rapidly return BP to normal. The sympathetic 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 ADH secretion cause Na and water retention and increase circulating blood volume.

Etiology

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 drugs, if myocardial contractility or vascular responsiveness is depressed, if **hypovolemia** is present, or if hormonal responses are faulty.

Causes differ depending on whether symptoms are acute or chronic.

The most common causes of acute orthostatic hypotension include

- Hypovolemia
- Drugs
- Prolonged bed rest
- Adrenal insufficiency

The most common causes of chronic orthostatic hypotension include

- Age-related changes in BP regulation
- Drugs
- 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 GI tract; this condition is worsened by alcohol intake.

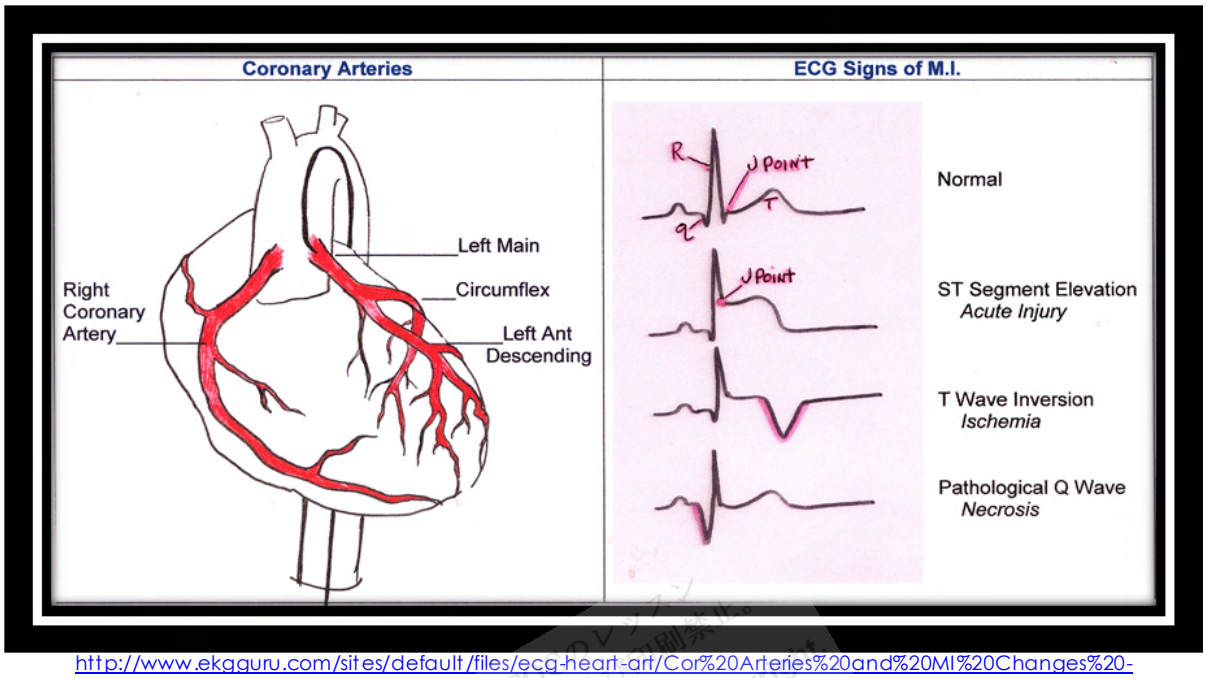

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Table 4

Causes of Orthostatic Hypotension

Cause	Examples
Neurologic (involving autonomic dysfunction)	
Central	Multiple system atrophy (previously Shy-Drager syndrome) **Parkinson disease** Strokes (multiple)
Spinal cord	**Tabes dorsalis** **Transverse myelitis** Tumors
Peripheral	Amyloidosis Diabetic, alcoholic, or nutritional neuropathy Familial dysautonomia (Riley-Day syndrome) Guillain-Barré syndrome **Paraneoplastic syndromes**

	Pure autonomic failure (formerly called idiopathic orthostatic hypotension) Surgical **sympathectomy**
Cardiovascular	
Hypovolemia	Adrenal insufficiency Dehydration Hemorrhage
Impaired vasomotor tone	Bed rest (prolonged) Hypokalemia
Impaired cardiac output	**Aortic stenosis** **Constrictive pericarditis** Heart failure MI **Tachyarrhythmias** or **bradyarrhythmias**
Other	**Hyperaldosteronism*** Peripheral venous insufficiency **Pheochromocytoma***
Drugs	
Vasodilators	Ca channel blockers Nitrates
Autonomically active	α-Blockers (prazosin, phenoxybenzamine) Antihypertensives (clonidine, methyldopa, reserpine, [rarely] β-blockers)† Antipsychotics (particularly phenothiazines) Monoamine oxidase inhibitors (MAOIs) Tricyclic or tetracyclic antidepressants

Other	Alcohol Barbiturates Levodopa (in Parkinson disease [rarely]) Loop diuretics (eg, furosemide) Quinidine Vincristine (neurotoxic)

*Disorder causes supine hypertension.

†Symptoms are more common when treatment is begun.

Evaluation

Orthostatic hypotension is diagnosed when a marked fall in measured BP and symptoms suggesting hypotension are provoked by standing and relieved by lying down. 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, drugs, 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** (GI 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 drug profile should be reviewed for offending prescription drugs, particularly **antihypertensives** and **nitrates**. A family history of orthostatic symptoms suggests possible familial **dysautonomia**.

Physical examination: BP and heart rate are measured after 5 min supine and at 1 and 3 min after standing; patients unable to stand may be assessed while sitting upright. Hypotension without a compensatory increase in heart rate (< 10 beats/min) suggests autonomic impairment. Marked increase (to > 100

beats/min or by > 30 beats/min) 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**). A rectal examination is done to detect GI bleeding.

During the neurologic examination, GU and rectal reflexes are tested to evaluate autonomic function; assessment includes the cremasteric reflex (normally, stroking the thigh results in retraction of the testes), anal wink reflex (normally, stroking perianal skin results in contraction of the anal sphincter), and bulbocavernosus reflex (normally, squeezing the glans penis or clitoris results in contraction of the anal sphincter). Signs of peripheral neuropathy (eg, abnormalities of strength, sensation, and deep tendon reflexes) are assessed.

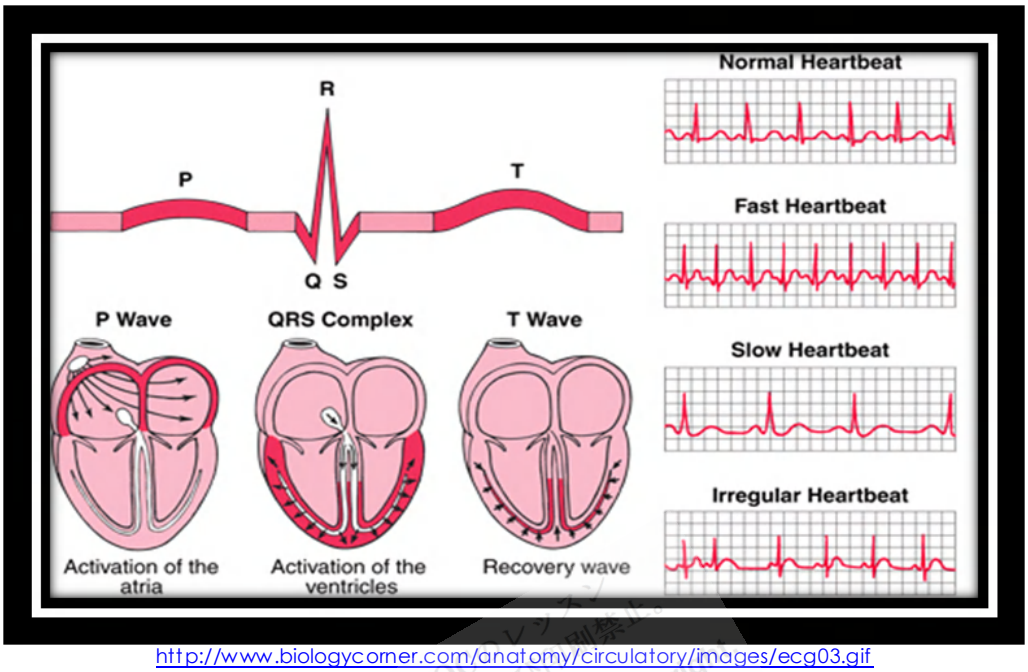
Red flags: Certain findings suggest a more serious etiology:

- Bloody or heme-positive stool
- Abnormal neurologic examination

Interpretation of findings: In patients with acute symptoms, the most common causes—drugs, 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, alcoholism), but a **paraneoplastic syndrome** from an occult cancer and amyloidosis must be considered. Patients who have only peripheral autonomic symptoms may have pure autonomic failure.

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



http://www.biologycorner.com/anatomy/circulatory/images/ecg03.gif

Autonomic function can be evaluated with bedside cardiac monitoring. 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-sec inspiration and a 7-sec expiration) for 1 min. The longest interbeat (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. A similar variation in R-R interval should exist between rest and a 10- to 15-sec Valsalva maneuver. Patients with abnormal R-R intervals or with autonomic symptoms or signs require further evaluation for diabetes, Parkinson disease, and possibly multiple system atrophy and pure autonomic failure; the last may require plasma norepinephrine or ADH (vasopressin) measurements with the patient supine and upright.

The dose of a suspected drug may be reduced or the drug stopped to confirm the drug 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 min of BP assessment.

Treatment

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**. Elderly patients should avoid prolonged standing. Sleeping with the head of the bed raised may relieve symptoms by promoting Na 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 Na intake may expand intravascular volume and lessen symptoms. In the absence of heart failure or hypertension, Na intake can be increased 5 to 10 g above the usual dietary level by liberally salting food or taking NaCl tablets. This approach risks heart failure, particularly in elderly patients and patients with impaired myocardial function; development of dependent edema without heart failure does not contraindicate continuing this approach.

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

Midodrine, a peripheral α-agonist that is both an arterial and venous constrictor, is often effective. Dosage is 2.5 mg to 10 mg po tid. Adverse effects include **paresthesias** and itching (probably secondary to piloerection). This drug is not recommended for patients with coronary artery or peripheral arterial disease.

NSAIDs (eg, indomethacin 25 to 50 mg po tid) may inhibit prostaglandin-induced vasodilation, increasing peripheral vascular resistance. However,

NSAIDs may cause GI symptoms and unwanted vasopressor reactions (reported with concurrent use ofindomethacin and sympathomimetic drugs).

L-Dihydroxyphenylserine, a norepinephrine precursor, may be beneficial for autonomic dysfunction (reported in limited trials).

Propranolol or other β-blockers may enhance the beneficial effects of Na and mineralocorticoid therapy. β-Blockade with propranolol leads to unopposed a-adrenergic peripheral vascular vasoconstriction, preventing the vasodilation that occurs when some patients stand.

Geriatrics Essentials

Orthostatic hypotension occurs in about 20% of the elderly; 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 the elderly 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. The elderly also have decreased resting parasympathetic tone, so that cardioacceleration due to reflex vagal withdrawal is lessened.

Key Points

- Orthostatic hypotension typically involves volume depletion or autonomic dysfunction.
- Some degree of autonomic dysfunction is common in the elderly, but neurologic disorders must be ruled out.
- Bedside tests of autonomic function and often tilt table testing are done.
- Treatment involves physical measures to reduce venous pooling, increased Na intake, and sometimes fludrocortisone or midodrine

Reference: http://www.merckmanuals.com