



**FIGURE 110-1** Orthostatic blood pressure profiles. **A**, The normal response to standing or head-up tilt is no change or a small decrease in blood pressure that recovers within one-half minute and a small increase in heart rate. **B**, Dehydration causing intravascular hypovolemia may cause a fall in blood pressure accompanied by reflex tachycardia. **C**, Neurogenic orthostatic hypotension may cause a more profound drop in blood pressure. Hypotension occurs immediately and is sustained without recovery during standing and often without adequate compensatory tachycardia. **D**, Postural tachycardia syndrome and other forms of orthostatic intolerance are characterized by an abnormal increase in heart rate without orthostatic hypotension. **E**, Neurally mediated syncope develops after standing for some time, may be preceded by oscillations in blood pressure, and may be accompanied by bradycardia with loss of consciousness in about 7 seconds if cerebral perfusion is not restored.

of the heart, or exhibit lower extremity rubor if cutaneous vasomotor function is impaired.

Orthostatic hypotension is a reduction in systolic blood pressure of at least 20 mm Hg or a reduction in diastolic blood pressure of at least 10 mm Hg, with or without symptoms, within 1 to 3 minutes of assuming an erect posture. Neurogenic orthostatic hypotension is typically sustained with continued standing and lacks the reflex tachycardia that may be seen if hypotension is caused by blood loss, dehydration, or excessive venous pooling.

Orthostatic intolerance is a sustained increase in postural heart rate of more than 30 beats per minute in adults (40 in adolescents). In postural tachycardia syndrome, the standing heart rate consistently exceeds 120 beats per minute.

Laboratory testing of autonomic responses under controlled conditions is useful to determine the presence, severity, and distribution of autonomic failure. Clinical autonomic testing typically evaluates beat-to-beat blood pressure and heart rate responses to the Valsalva maneuver, upright tilt, and periodic deep breathing, along with quantitative assessment of sweating responses. Ambulatory blood pressure testing is useful for the assessment of episodic or postprandial hypotension, nocturnal hypertension, and the volatile hypertension of autonomic storms.

## TREATMENT

Treatment options for orthostatic hypotension are outlined in Table 110-1. The goal is to enable the patient to stand long enough to engage in daily activities without symptoms. Medication is not always needed and can potentially exacerbate recumbent hypertension. Orthostatic intolerance was shown in a randomized controlled trial to improve after endurance exercise training (level A evidence).

Generalized hyperhidrosis may be reduced by oral anticholinergic agents such as 1 to 2 mg of glycopyrrolate taken one to three times daily (level B evidence). Topical glycopyrrolate reduces regional gustatory sweating (level A). Subdermal botulinum toxin injections are helpful for some forms of focal hyperhidrosis (level A), and palmar hyperhidrosis may respond to tap water iontophoresis (level B) or, in severe cases, to endoscopic thoracic sympathectomy (level A).

*For a deeper discussion of these topics, please see Chapter 418, "Autonomic Disorders and Their Management," in Goldman-Cecil Medicine, 25th Edition.*

## PROGNOSIS

Orthostatic intolerance and neurally mediated syncope are frequently benign, manageable, and improve or recover with time. Autonomic failure, in contrast, can signify a more serious prognosis, depending on the nature and extent of its pathophysiology. Persistent or severe orthostatic hypotension carries a worse prognosis.

Diabetic cardiovascular autonomic neuropathy doubles the risk for silent myocardial ischemia and overall mortality. Amyloid autonomic neuropathy is especially grave, with a median survival of less than 1 year if the patient has orthostatic hypotension. Pure autonomic failure may remain stable for many years, although some patients with this phenotype eventually develop signs of multiple system atrophy, which denotes a life expectancy of 7 to 9 years after diagnosis.

Regular physical exercise can reverse the autonomic deconditioning that comes from inactivity. In the elderly, it may compensate for some age-associated decline in autonomic function (level B evidence).

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