TABLE 454-3 SYMPTOMS OF ORTHOSTATIC INTOLERANCE

Lightheadedness (dizziness)	88%
Weakness or tiredness	72%
Cognitive difficulty (thinking/ concentrating)	47%
Blurred vision	47%
Tremulousness	38%
Vertigo	37%
Pallor	31%
Anxiety	29%
Palpitations	26%
Clammy feeling	19%
Nausea	18%

Source: PA Low et al: Mayo Clin Proc 70:617, 1995.

nausea, vomiting of old food, diarrhea), or bladder disorders (urinary frequency, hesitancy, or incontinence). Symptoms may be widespread or regional in distribution. An autonomic history focuses on systemic functions (BP, heart rate, sleep, fever, sweating) and involvement of individual organ systems (pupils, bowel, bladder, sexual function). The autonomic symptom profile is a self-report questionnaire that can be used for formal assessment. It is also important to recognize the modulating effects of age. For example, OH typically produces lightheadedness in the young, whereas cognitive slowing is more common in the elderly. Specific symptoms of orthostatic intolerance are diverse (Table 454-3). Autonomic symptoms may vary dramatically, reflecting the dynamic nature of autonomic control over homeostatic function. For example, OH might be manifest only in the early morning, following a meal, with exercise, or with raised ambient temperature, depending on the regional vascular bed affected by the dysautonomia.

Early symptoms may be overlooked. Impotence, although not specific for autonomic failure, often heralds autonomic failure in men and may precede other symptoms by years (Chap. 67). A decrease in the frequency of spontaneous early morning erections may occur months before loss of nocturnal penile tumescence and development of total impotence. Bladder dysfunction may appear early in men and women, particularly in those with a CNS etiology. Cold feet may indicate increased peripheral vasomotor constriction. Brain and spinal cord disease above the level of the lumbar spine results first in urinary frequency and small bladder volumes and eventually in incontinence (upper motor neuron or spastic bladder). By contrast, PNS disease of autonomic nerve fibers results in large bladder volumes, urinary frequency, and overflow incontinence (lower motor neuron flaccid bladder). Measurement of bladder volume (postvoid residual) is a useful bedside test for distinguishing between upper and lower motor neuron bladder dysfunction in the early stages of dysautonomia. Gastrointestinal autonomic dysfunction typically presents as severe constipation. Diarrhea may develop (typically in diabetes mellitus) due to rapid transit of contents or uncoordinated small-bowel motor activity, or on an osmotic basis from bacterial overgrowth associated with small-bowel stasis. Impaired glandular secretory function may cause difficulty with food intake due to decreased salivation or eye irritation due to decreased lacrimation. Occasionally, temperature elevation and vasodilation can result from anhidrosis because sweating is normally important for heat dissipation (Chap. 23). Lack of sweating after a hot bath, during exercise, or on a hot day can suggest sudomotor failure.

OH (also called *orthostatic or postural hypotension*) is perhaps the most disabling feature of autonomic dysfunction. The prevalence of OH is relatively high, especially when OH associated with aging and diabetes mellitus is included (Table 454-4). OH can cause a variety of symptoms, including dimming or loss of vision, lightheadedness, diaphoresis, diminished hearing, pallor, and weakness. Syncope results when the drop in BP impairs cerebral perfusion. Other manifestations of impaired baroreflexes are supine hypertension, a heart rate that is fixed regardless of posture, postprandial hypotension, and an excessively high nocturnal BP. Many patients with OH have a preceding

TABLE 454-4 PREVALENCE OF ORTHOSTATIC HYPOTENSION IN DIFFERENT DISORDERS

Disorder	Prevalence
Aging	14–20%
Diabetic neuropathy	10%
Other autonomic neuropathies	10–50 per 100,000
Multiple system atrophy	5–15 per 100,000
Pure autonomic failure	10–30 per 100,000

diagnosis of hypertension or have concomitant supine hypertension, reflecting the great importance of baroreflexes in maintaining postural and supine normotension. The appearance of OH in patients receiving antihypertensive treatment may indicate overtreatment or the onset of an autonomic disorder. The most common causes of OH are not neurologic in origin; these must be distinguished from the neurogenic causes (Table 454-5). **Neurocardiogenic and cardiac causes of syncope are considered in Chap. 27.**

APPROACH TO THE PATIENT: Orthostatic Hypotension and Other ANS Disorders

The first step in the evaluation of symptomatic OH is the exclusion of treatable causes. The history should include a review of medications that may affect the ANS (Table 454-6). The main classes of drugs that may cause OH are diuretics, antihypertensives, antidepressants, ethanol, narcotics, insulin, dopamine agonists, barbiturates, and calcium channel-blocking agents. However, the precipitation of OH by medications may also be the first sign of an underlying autonomic disorder. The history may reveal an underlying cause for symptoms (e.g., diabetes, Parkinson's disease) or specific underlying mechanisms (e.g., cardiac pump failure, reduced intravascular volume). The relationship of symptoms to meals (splanchnic pooling), standing on awakening in the morning (intravascular volume depletion), ambient warming (vasodilatation), or exercise (muscle arteriolar vasodilatation)

TABLE 454-5 NONNEUROGENIC CAUSES OF ORTHOSTATIC HYPOTENSION

Cardiac pump failure
- Myocardial infarction
- Myocarditis
- Constrictive pericarditis
- Aortic stenosis
- Tachyarrhythmias
- Bradyarrhythmias
- Salt-losing nephropathy
- Adrenal insufficiency
- Diabetes insipidus
- Venous obstruction

Reduced intravascular volume
- Straining or heavy lifting, urination, defecation
- Dehydration
- Diarrhea, emesis
- Hemorrhage
- Burns

Metabolic
- Adrenocortical insufficiency
- Hypoaldosteronism
- Pheochromocytoma
- Severe potassium depletion

Venous pooling
- Alcohol
- Postprandial dilation of splanchnic vessel beds
- Vigorous exercise with dilation of skeletal vessel beds
- Heat: hot environment, hot showers and baths, fever
- Prolonged recumbency or standing
- Sepsis

Medications
- Antihypertensives
- Diuretics
- Vasodilators: nitrates, hydralazine
- Alpha- and beta-blocking agents
- Central nervous system sedatives: barbiturates, opiates
- Tricyclic antidepressants
- Phenothiazines