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THE PRESENT AND FUTURE: *JACC* STATE-OF-THE-ART REVIEW

Orthostatic Hypotension

JACC State-of-the-Art Review

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ABSTRACT

Neurogenic orthostatic hypotension is a highly prevalent and disabling feature of autonomic failure due to both peripheral and central neurodegenerative diseases. Community-based epidemiological studies have demonstrated a high morbidity and mortality associated with neurogenic orthostatic hypotension. It is due to impairment of baroreflex-mediated vasoconstriction of the skeletal muscle and splanchnic circulation and is caused by damage or dysfunction at central and/or peripheral sites in the baroreflex efferent pathway. Nonpharmacological and pharmacological interventions may be implemented to ameliorate the symptoms of orthostatic intolerance and improve quality of life. Many patients will be adequately treated by education, counseling, removal of hypotensive medications, and other nonpharmacological interventions, whereas more severely afflicted patients require pharmacological interventions. The first stage of pharmacological treatment involves repletion of central blood volume. If unsuccessful, this should be followed by treatment with sympathomimetic agents. (J Am Coll Cardiol 2018;72:1294–309) © 2018 Published by Elsevier on behalf of the American College of Cardiology Foundation.

Orthostatic hypotension (OH) is arguably the most incapacitating feature of autonomic failure. The disorder is highly prevalent, but due to diverse clinical presentations, many of which are nonspecific, it is frequently unrecognized until late in the clinical course. Three common variants exist: OH (also known as classical OH), delayed orthostatic hypotension (DOH), and initial OH.

Classical OH is defined as a sustained reduction of at least 20 mm Hg of systolic blood pressure (SBP) or 10 mm Hg of diastolic blood pressure (BP) *within 3 min* of standing or head-up tilt-table testing (**Figure 1**) (1). Patients may have associated symptoms of orthostatic intolerance, although symptoms are not necessary for the diagnosis. Some patients with baroreflex failure and other forms of autonomic impairment may also exhibit supine hypertension, in which case a reduction in SBP of at least 30 mm Hg may be a more appropriate definition (1). Compensatory tachycardia can be seen; however, in more severe autonomic failure, this response may be blunted or even absent.

DOH is a sustained reduction in BP that occurs *after 3 min* of standing or upright tilt (**Figure 2**). In 1 study, 15% of 108 patients with OH on tilt-table testing demonstrated a BP drop between 3 and 10 min, and 39% had a BP drop after 10 min or more (2,3). When compared with patients with OH, patients with DOH have less severe impairment of other measures of sympathetic adrenergic function, suggesting a milder or earlier manifestation of the disease state. A 10-year follow-up study in a cohort of patients with DOH lends support to this notion (2). Data were available for 90 of the original 108 patients; of those with DOH, 54% progressed to OH.

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Initial OH is a transient reduction in BP (defined as a reduction ≥40 mm Hg SBP and/or ≥20 mm Hg diastolic BP) *within 15 s* of standing (**Figure 3**) (1). It tends to occur more often in younger patients, but it may also occur in elderly patients (4), and is more pronounced on active standing than on passive tilt. It likely represents a mismatch between cardiac output and peripheral vascular resistance (5), although the pathophysiology is not definitively established. It is not associated with a disease state, nor does it appear to predict an increased morbidity or mortality risk (6).

PREVALENCE AND NATURAL HISTORY

The prevalence of OH depends on the age, comorbidities, and medication profiles of the study group, along with the definition of OH used in the study. The prevalence of OH increases with advancing age, partly due to normal physiological decline in baroreceptor sensitivity and partly due to the age-associated increased prevalence of autonomic neurodegenerative disease. As a result, OH is relatively uncommon in patients younger than 65 years of age. In the ARIC (Atherosclerosis Risk In Communities) study, a community-based prospective cohort of 15,792 middle-aged adults 45 to 64 years of age, OH was present in 5% of individuals (7). OH is common in patients with diabetes, affecting an estimated 30% of individuals with type 1 diabetes and 25% to 30% of individuals with type 2 diabetes (8,9). Prevalence in the inpatient setting can be as high as 64% (10).

The natural history of OH largely depends on the underlying associated disorders (2,11). Individuals with diabetes or neurodegenerative disease as the cause of OH will experience progressive worsening over time. The rate of progression will depend on the subtype of degenerative disorder (pure autonomic failure [PAF] progresses most slowly, Parkinson disease [PD] and dementia with Lewy bodies [DLB] progress more rapidly, and multiple system atrophy [MSA] progresses most rapidly) (11–13). In contrast, in individuals with diabetes, the rate of disease progression may depend on the underlying glycemic control and associated risk factors (14). Solid epidemiological data on the natural history of OH across other disease states is not as well-established.

CLINICAL FEATURES

Patients with OH may use a variety of terms to describe their symptoms (**Table 1**). The central feature of these symptoms is that they are correlated with postural change, exacerbated by standing and relieved by sitting or lying flat. Symptom descriptors include, but are not limited to, "dizziness," "vertigo," "lightheadedness," or a "woozy" sensation. Patients may also note shortness of breath due to ventilation-perfusion mismatch in the lung apices (15), chest pain due to myocardial hypoperfusion, headache, fatigue, confusion, or difficulty concentrating (16). Some patients describe neck cramping on standing, otherwise known as "coat-hanger headache," due to hypoperfusion of the trapezius and shoulder girdle muscles (17). Visual blurring or dimming may occur, likely due to retinal or occipital lobe ischemia (18).

Symptoms are often exacerbated by warm environments, due to peripheral vasodilation, and prolonged standing, due to lower extremity venous pooling. Standing quickly after prolonged sitting, such as after long car rides, results in a sudden redistribution of 500 to 1,000 ml of blood to the lower extremities and may precipitate severe orthostatic intolerance or even syncope (19).

In patients with autonomic failure, symptoms tend to be worse in the early morning hours, especially in those with supine hypertension due to diurnal fluid shifts, nocturnal diuresis that leads to early morning hypotension—a reversal of the normal circadian variation in BP (20). As a result, many patients are at increased risk of falling when walking to the bathroom upon awakening from sleep or in the early morning hours, particularly if they take medication to treat supine hypertension. **Table 2** lists common exacerbating factors.

Loss of consciousness may occur, although usually more gradually than in other disorders that cause syncope, such as neurally-mediated or cardiogenic syncope. Some patients, however, are surprisingly asymptomatic, despite SBPs <90 mm Hg. This may be due, in part, to cerebral autoregulation, in which patients are able to maintain cerebral perfusion pressures despite critically low peripheral arterial BPs (21,22). Some studies using transcranial Doppler ultrasound in patients with OH have demonstrated minimal reductions in middle cerebral artery velocity, despite significant falls in BP on tilt-table testing (21–23). However, it is also apparent that some asymptomatic patients may have changes in cognitive capacity that they do not recognize (16,24).

MORBIDITY AND MORTALITY

OH is associated with increased morbidity and mortality, independent of the underlying etiology. In the ARIC study, a cohort of 674 middle-aged individuals with OH (mean age 54 years) had a 13-year

ABBREVIATIONS AND ACRONYMS

BP = blood pressure

DLB = dementia with Lewy bodies

DOH = delayed orthostatic hypotension

MSA = multiple system atrophy

MSA-C = multiple system atrophy-cerebellar form

MSA-P = multiple system atrophy-Parkinsonian form

OH = orthostatic hypotension

PAF = pure autonomic failure

PD = Parkinson disease

SBP = systolic blood pressure

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