

Review Article

Seated postural hypotension



Oleg Gorelik, MD* and Natan Cohen, MD

Department of Internal Medicine "F", Assaf Harofeh Medical Center (affiliated to the Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel), Zerifin, Israel

Manuscript received August 31, 2015 and accepted October 4, 2015

Abstract

Most studies of postural hypotension (PH) have focused on standing PH. Less is known about PH after transition from a supine to sitting position. Moreover, seated PH has not been previously reviewed in the English literature. The aim of this review was to provide current information regarding seating-induced PH. Seventeen studies were reviewed regarding prevalence, methods of evaluation, manifestations, predisposing factors, prognosis, and management of seated PH. Prevalence ranged from 8% among community-dwelling persons to 56% in elderly hospitalized patients. Dizziness and palpitations were the most frequent symptoms. Of a variety of factors that have been identified as predisposing and contributing to seated PH, aging, bed rest, and hypertension were most important. Because seated PH is a common, easily diagnosable and frequently symptomatic condition, especially in elderly inpatients, this disorder warrants attention. Moreover, seating-induced falls in blood pressure and the associated symptoms, may be largely prevented by nonpharmacologic interventions. *J Am Soc Hypertens* 2015;9(12):985–992. © 2015 American Society of Hypertension. All rights reserved.

Keywords: Orthostatic hypotension; seating; sitting.

Introduction

When a healthy person stands up, approximately 500 to 1000 mL of blood is pooled into the lower extremities and splanchnic circulation.^{1–6} As a consequence, venous return to the heart, cardiac filling pressure, stroke volume, and cardiac output are decreased. In response, blood pressure (BP) is stabilized by a compensatory sympathetic activation.^{1–6} Postural hypotension (PH), also called orthostatic hypotension, is caused by an excessive fall of cardiac output and/or by impaired compensatory vasoconstrictor mechanisms such as autonomic failure and age-related physiologic changes.^{1–6} PH is traditionally defined as a fall of at least 20 mm Hg in systolic BP (SBP) and/or at least 10 mm Hg in diastolic BP (DBP) within 3 minutes of active standing.^{1–6} Alternatively, PH can be determined by head-up tilt-table testing at an angle of at least 60°. ³ Moreover, in patients with hypertension, it is more appropriate to define PH as a reduction of SBP of ≥ 30 mm

Hg because the magnitude of the BP fall depends on the baseline BP value.³

The prevalence of PH increases with age and comorbidities.^{4–8} Reported prevalence rates of standing PH range from 5% to 30% among community-dwelling subjects,^{7–11} and from 24% to 68% among patients hospitalized in short-term care facilities.^{12–14} PH may be a disabling disorder and is a risk factor for falls and syncope, as well as for cardiovascular and cerebrovascular disease.^{4–7,11,15} In addition, PH has been reported as a strong predictor of all-cause mortality.^{7,9–11,15}

Traditionally, PH is classified as neurogenic and non-neurogenic.^{1,2,4–6} Neurogenic PH is less common and is caused by multiple system atrophy, Parkinson's disease, pure autonomic failure, and neuropathies.^{1,2,4–6} Nonneurogenic PH is more common and is associated with aging, postprandial state, medications, prolonged bed rest, blood volume depletion, hypertension, heart failure, and other nonneurogenic disorders.^{1,2,4–7,14}

PH may present as acute or chronic.^{4,6} Acute PH generally resolves with treatment of the underlying cause or discontinuation of medication that contributed to the development of PH.⁶ In patients with chronic PH, nonpharmacologic and pharmacologic interventions may be beneficial.^{1,2,4–6,16} Nonpharmacologic measures include avoiding

Conflict of interest: The authors declare no conflict of interest.

*Corresponding author: Oleg Gorelik, MD, Department of Internal Medicine "F", Assaf Harofeh Medical Center, Zerifin 70300, Israel. Tel: +972-8-9779994/1; fax: +972-8-9779976.

E-mail: internal6@asaf.health.gov.il

of prolonged standing and of exposure to a hot environment, increased fluid and salt intake, raising the head of the bed during nighttime, and leg crossing while standing.^{1,2,4–6,16} Performing gradual staged movements during postural change and the application of a variety of compression devices on the abdomen and lower extremities may also be beneficial for PH prevention.^{1,2,4–6,16,17} In patients who do not respond adequately to nonpharmacologic interventions for PH, treatment with fludrocortisone, midodrine, pyridostigmine, droxidopa, and other medications is recommended.^{1,2,4–6}

In patients who are unable to stand, evaluation of PH may be performed in the sitting position only. Moreover, in persons at high risk for symptomatic standing PH, such as patients with chronic standing PH and hospitalized acutely ill patients, assessment of PH should be performed in a sitting position before standing.^{1,2,14,18,19}

Investigation of PH is generally focused on standing PH. Less information is available concerning seating-induced PH. To the best of our knowledge, a review article on seated PH has not been previously published. The aim of this review is to emphasize the clinical importance and to provide the current information about seating-induced PH.

Methods

A systematic search of PubMed, MEDLINE, Embase, Cochrane, and Google Scholar databases was performed using the following keywords: postural hypotension, orthostatic hypotension or orthostatic intolerance, with the combination of the following: seating, seated or sitting. The search was performed for potentially eligible articles, available until August 2015. Articles were included for the review, if they met the following criteria: published as a full-length article, English language, and including data regarding adult individuals (≥ 18 years).

Results

A total of 17 studies were found eligible to be included in this review (Table 1).^{18–34} The selected studies were reviewed regarding prevalence, methods of evaluation, manifestations, predisposing factors, prognosis, and management of seated PH.

Prevalence of Seated PH

Prevalence of seating-induced PH has been studied in different patient populations consisting of healthy^{23,30} and community-dwelling persons,^{22,32} residents of long-term geriatric facilities,²⁰ and hospitalized patients with acute stroke,²⁴ spinal cord injury,^{28,29} exacerbated heart failure,³¹ and a variety of acute disorders.^{25,26} Kuchel et al studied seated PH among 996 healthy blood donors, aged 52–78 years, after withdrawal of 500 mL of blood.²³ In the entire

cohort, 11% of participants exhibited systolic PH. Further group subdivision revealed that in the middle-age group (65 years old and younger) PH was recorded in 6.9% of subjects, whereas in the group older than 65 years, PH was diagnosed in 15.2%.²³ Madhavan et al evaluated seated PH among 33 healthy women aged 30–65 years.³⁰ None demonstrated PH within 3 minutes of sitting, whereas delayed PH (after 15 minutes of sitting) was observed in 27%. In the largest available investigation that included 8574 noninstitutionalized community-dwelling white nondiabetic persons, aged 25–74 years, who participated in the second National Health and Nutrition Examination Survey in the USA, seated PH was diagnosed in 8.3% of the subjects.²² The prevalence of PH was higher in patients medicated for hypertension than in those not taking medication for hypertension (13.0% and 6.6%, respectively).²² In the study of Aronow et al, involving of 289 chair-bound patients, aged 62–101 years, from a long-term health care facility, seated PH occurred in 22 (8%).²⁰ Interestingly, among 187 counterparts who were able to assume upright standing from the supine position, the prevalence of standing PH (9%) was similar to seated PH.

The prevalence of seated PH is increased among patients with acute or chronic neurologic disorders.^{28,29,32} Seated PH was observed among 36% of 25 middle-aged patients with traumatic spinal cord injury compared with none of 18 healthy subjects who served as controls.²⁸ Among 100 patients, aged 15–74 years, who were in the initial phase of rehabilitation after spinal cord trauma, the rate of PH on assuming of a wheelchair position was 68%.²⁹ Panayiotou et al compared 40 hospitalized patients aged ≥ 65 years with mild or moderate severity acute stroke to a control group that included 40 nonstroke age- and sex-matched inpatients.²⁴ The prevalence of seating-induced systolic PH was low (8%) and similar in both groups. The low prevalence of PH observed among the stroke patients could be due to the exclusion of patients with coexisting illnesses associated with higher risk to develop PH, such as severe stroke, diabetes, Parkinson's disease, severe ischemic heart disease or heart failure, infection, anemia, and renal failure. In another study, consisting of 49 ambulatory patients aged 51–80 years with a history of stroke, seated PH was determined in 14.2% of the participants.³²

Seated PH is shown to be common among patients admitted to internal medicine wards.^{25,26,31} In a cohort of 108 patients, aged at least 60 years, hospitalized with acutely decompensated heart failure, PH occurred in 49.1% of subjects, and diastolic versus systolic PH prevailed (36.1% vs. 23.1%, respectively, $P = .05$).³¹ Moreover, seating-induced PH was detected in 54% of 98 aged ≥ 65 years patients admitted for various acute conditions and suffering from a variety of chronic disorders.²⁵ Finally, seated PH was observed in 56% of 61 elderly inpatients (mean age, 78 years), admitted for a variety of acute disorders and confined to bed for at least 36 hours.²⁶

Download English Version:

<https://daneshyari.com/en/article/2956166>

Download Persian Version:

<https://daneshyari.com/article/2956166>

[Daneshyari.com](https://daneshyari.com)