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Research Article

Detecting initial orthostatic hypotension: a novel approach

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Abstract

Our purpose, by modification of standard bedside tilt–testing, was to search for lesser known but important initial orthostatic hypotension (IOH), occurring transiently within the first 30 seconds of standing, heretofore only detectable with sophisticated continuous photoplethysmographic monitoring systems, not readily available in most medical facilities. In screened outpatients over 60 years of age, supine blood pressure (BP) parameters were recorded. To achieve readiness for immediate BP after standing, the cuff was re–inflated prior to standing, rather than after. Immediate, 1–, and 3–minute standing BPs were recorded. One hundred fifteen patients were studied (mean age, 71.1 years; 50.5% male). Eighteen (15.6%) had OH, of whom 14 (12.1%) had classical OH, and four (3.5%) had IOH. Early standing BP detection time was 20.1 ± 5.3 seconds. Immediate transient physiologic systolic BP decline was detected in non–OH (−8.8 ± 9.9 mm Hg; $P < .0001$). In contrast to classical OH (with lesser but persistent orthostatic BP decrements), IOH patients had immediate mean orthostatic systolic/diastolic BP change of −32.8 (±13.8) mm Hg/−14.0 (±8.5) mm Hg ($P < .02$), with recovery back to baseline by 1 minute. Two of the four IOH patients had pre–syncopal symptoms. For the first time, using standard inflation–deflation BP equipment, immediate transient standing physiologic BP decrement and IOH were demonstrated. This preliminary study confirms proof of principle that manual BP cuff inflation prior to standing may be useful and practical in diagnosing IOH, and may stimulate direct comparative studies with continuous monitoring systems. J Am Soc Hypertens 2015;9(5):365–369.

Keywords: Falls; orthostatic hypotension; pre-syncope; syncope.

Introduction

Orthostatic hypotension (OH) may be associated with falls, injury, and sometimes death in the elderly and in patients with multiple co–morbidities.[1–3] Prevalence appears to range from 5% beyond age 65 to over 20% in the very elderly.[3–5] Blood pressure (BP) homeostasis upon standing depends on baseline peripheral vascular resistance and sympathetic reflex response, cardiac pump status and cardiac reflex response, volume status, and skeletal muscle pump (lower body muscles compressing veins). Underlying causes of OH thus may include baseline vasodilation (as may be seen with medications), autonomic dysfunction, pump failure, excessive venous pooling with deconditioning, or true volume depletion.[6–8] By consensus guideline criteria, classical orthostatic hypotension (COH) is defined as a sustained reduction in systolic BP (SBP) of ≥20 mm Hg and/or diastolic BP (DBP) fall of ≥10 mm Hg within 3 minutes of standing, and is clinically important if there are associated symptoms of cerebral hypoperfusion.[8]

Initial orthostatic hypotension (IOH) is a lesser known form of consensus OH, described in adolescent patients and the elderly, which represents an aberration of normal physiologic events occurring upon active standing.[9–13] It is defined as a reduction in SBP of >40 mm Hg and/or decrease in DBP of >20 mm Hg, presenting within the first

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15 seconds of standing, and correcting within 30–60 seconds.[8] As background, all humans may experience an immediate and occasionally symptomatic transient decrease in BP just after standing (nadir at 5–15 seconds), with recovery by 30 seconds. This normal physiologic phenomenon is not completely clarified but involves lower body muscle contraction and increased right atrial pressure (with initial neurovascular reflex changes) during the act of standing, immediate blood pooling below the diaphragm induced by gravity after completion of stand, and subsequent rapid sympathetic responses further affecting cardiac output and peripheral resistance. Thus, rarely does one experience pre–syncopal symptoms immediately after standing. However, if there is development of transient dramatic enhancement of early volume shifts associated with a temporal mismatch of cardiovascular compensation on standing, symptomatic IOH may occur consistently.[9–11] Elderly patients with underlying IOH physiology may be particularly susceptible to pre–syncopal symptoms, since separate and unrelated volume and distributive issues are common.[13,14]

IOH has been previously discernible and defined only by use of continuous non–invasive beat–to–beat finger photoplethysmographic BP monitoring systems (Finometer/Finapres).[12] The sophistication and expense of these devices has limited testing for IOH and awareness of this entity. The standard bedside tilt–test, using inflation–deflation methods, has been incapable of detecting immediate standing BP changes (IOH) due to time required to inflate the cuff after completion of standing.[15] In addition, routine formal tilt–table testing does not detect IOH since the gradual rise of the table does not replicate the physiology of the acute act of standing.[12] Hence, detection of IOH is problematic in most clinical settings. We sought to detect IOH by novel use of standard BP equipment involving supine cuff inflation prior to standing. Detection of an early transient fall in BP within physiologic parameters would serve as validation of this method. A dramatic early fall in BP upon standing, which corrects within 30–60 seconds, with or without symptoms, would be consistent with IOH.[8] Symptomatic patients may respond dramatically to readily performed counter–pressure maneuvers (see below).

Methods

Study approval was obtained from the Charleston Area Medical Center/West Virginia University HSC, Charleston Division Institutional Review Board. This was a prospective study of outpatients, who were recruited at clinic sign–in to participate in screening for BP changes upon standing. They were not initially told about what kind of BP changes might be expected, and possible orthostatic symptoms were not discussed prior to the procedure to avoid patient bias in reporting. Patients were excluded who were under 60 years of age, non–ambulatory (ie, those not able to walk or stand on their own), or who had history of orthostatic hypotension. Records were reviewed regarding basic demographics. The study was conducted during clinic hours, duplicating real–world conditions. Wall–mounted WelchAllyn Tycos aneroid sphygmomanometry was utilized. Large cuffs were used appropriately in obese patients. Investigators were trained in basic uniform BP recording technique.[15,16] Following informed consent, patients were maintained in the supine position for between 5 and 10 minutes. Baseline lying and standing BP and heart rate (HR) were recorded in the right arm with midline of cuff located in line with the brachial artery. A pulse oximeter device was used to provide HR throughout the standing process. To achieve readiness for BP measurement just after standing, the cuff was re–inflated in the supine position to 180 mm Hg. Patients were then asked to stand, with some assistance if necessary since right arm was extended. Immediately following completion of stand, BP, HR, and time to completion were recorded. Subsequent BP and HR were recorded at approximately 1 and 3 minutes. Patients were then queried for any symptoms such as ‘‘dizziness, wooziness, or faintness’’ occurring upon standing. Patients were grouped as non–OH (normal), COH, or IOH based on BP changes by the above criteria. Patients meeting COH criteria were evaluated for underlying causes and managed appropriately. Those meeting IOH criteria were provided with information on physical counter–pressure maneuvers to minimize orthostatic symptoms.

Statistical Analysis

To evaluate supine cuff inflation for detecting early standing BP changes, statistical differences were assessed assuming zero change in initial BP after standing. Basic descriptive statistics such as means and standard deviations for continuous variables and proportions and frequencies for categorical variables were used to analyze the data. The paired samples *t*–test was conducted to compare BPs and HRs from baseline to immediate, 1–minute, and 3–minute intervals. Comparisons were made for differences in baseline BP and HR in non–OH versus COH and non–OH versus IOH; for standing BP changes from baseline in all groups; and for differences in symptoms in non–OH versus OH (COH plus IOH) upon standing. Data are reported as mean (standard deviation [SD]). All data were analyzed using SAS 9.3 software. A P value of $< .05$ was used to determine statistical significance.

Results

One hundred fifteen patients were studied (mean age, 71.1 years; 50.5% male). Thirty–one percent had type 2 diabetes mellitus, 80% had hypertension, and the mean number of medications prescribed per patient was nine. Table 1 summarizes baseline BP data. Ninety–seven