Journal of Clinical Images and Case Reports

Research Article

A SCITECHNOL JOURNAL

Postural and Prandial Tolerance in Severely Frail Older People A Study in Long-term Geriatric Care

Jochanan E Naschitz*, Irena Kozel, Anatoli Nemoi, Gregory Leibovitz, Yigal Avital and Peri Ofri

Abstract

Objective: To assess frail older peoples' tolerance to every-day postural and prandial challenges.

Design: Prospective cohort study.

Subjects: Older persons, bed and chair confined, hospitalized in long-term geriatric or hospice care, unfit to undergo customary postural and prandial testing.

Methods: Blood pressure (BP) and heart rate (HR) were measured with an automatic device in three situations: supine at 7 am; sitting before lunch at 12 pm., and continuing sitting 30 minutes after lunch. The patients' alertness and symptoms were assessed at 7 am., 12.00 and 12.40. BP changes were calculated: supine to sitting, and sitting before lunch to sitting after lunch. BP changes were related to incident symptoms.

Results: The meal was discontinued in two patients when a vasovagal reaction and dumping syndrome occurred. Sixty three tests in 48 patients were completed. The average supine systolic BP (SBP) was 121.2 ± 16.8 mmHg and the diastolic BP (DBP) 67.7 ± 10.5 mmHg. The average difference between supine SBP and sitting before lunch SBP was 3.2 ±19.2 mmHg, the DBP difference was 4.7 ± 10.8 mmHg (p=0.0003). The average difference between SBP sitting before lunch and SBP sitting after lunch was 0.4 ±12 mmHg, the DBP difference was 0.7 ± 7.5 mmHg. Orthostatic hypotension (OH) and/or postprandial hypotension (PPH) was present in 31/63 (47.6%) of tests: all patients remained free of symptoms during testing and the mean BP in all measurements was >60 mmHg.

Conclusions: A large proportion of severely frail older patients tolerated every-day postural and prandial challenges, even when asymptomatic OH and/or PPH occurred.

Keywords

Frailty; Orthostatic hypotension; Postprandial hypotension

Key Points

We assessed by simple, generally applicable means the postural and prandial tolerance of patients hospitalized in long-term geriatric or palliative care.

Severely frail older persons tolerated habitual postural and prandial challenges, even though asymptomatic orthostatic hypotension (OH) and/or postprandial hypotension (PPH) were present on many occasions.

It appears that routine screening of residents in palliative or skilled nursing institutions for OH and PPH is unnecessary and avoidable.

Introduction

Orthostatic hypotension (OH) and postprandial hypotension (PPH) are common disorders which accumulate with age. OH is defined as a sustained reduction of either systolic blood pressure (SBP) of at least 20 mmHg or diastolic blood pressure (DBP) of at least 10 mmHg within 3 minutes of standing or passive head-up tilt to at least 60°C [1]. Some patients have 'delayed OH' that occurs beyond 3 minutes of standing. The prevalence of OH may be as high as 70% among residents in long-term geriatric care [1-4]. OH may concur with dizziness, falls and frailty, and has been regarded as a major cause of morbidity. In older persons OH has been considered an omen of death [5], but adjusted for frailty this association has not been found to be significant [6]. PPH is defined as a decrease in SBP of at least 20 mmHg [7,8]. Nearly all older persons living in nursing homes experience some postprandial decrease in BP. PPH, like OH, is thought to be a major cause of morbidity in older people [9]. The possibility of PPH should be considered when evaluating syncope, falls and dizziness occurring within two hours after a meal. Experts recommend for the diagnosing of PPH that the patient have both postprandial symptoms and postprandial BP decrease. It is good practice that symptomatic patients undergo ambulatory BP monitoring with analysis of breakfast and lunch hemodynamics [7]. Alternatively, precisely timed small numbers of measurements may be valuable with monitoring the BP and symptoms for 2 hours after a meal, since the nadir in BP can occur as late as 2 hours postprandially [8].

There is a plea that BP measurements for diagnosing hypotensive syndromes should be a component of comprehensive geriatric assessment [10-12]. Yet, screening frail older people for OH and PPH has not been systematically explored. In the present study we assessed BP changes related to posture and meals in frail older patients who were unfit to undergo customary postural and prandial testing. The defining outcome of the study was postural fitness under real life conditions rather than results of postural and prandial 'laboratory tests'. This we presume to be the significance of our study.

Methods

Study population

The patient population comprised 50 older people, resident in long-term geriatric or hospice care, who were severely frail, ADL dependent, bed and chair confined, feeding orally, unfit to undergo customary postural and prandial challenges, and typically unable to comply with ambulatory 24 hour BP or beat-to-beat BP monitoring. The CSHA Clinical Frailty Scale [13] was used to estimate frailty severity, in which score 6 is the label for moderately frail persons

***Corresponding author:** Jochanan E Naschitz, Department of Internal Medicine , Bnai-Zion Medical Center, Haifa 31048, PO Box 4940, Haifa 31048, Israel, Tel: 972-50-8800860; Fax: 972-73-2377361; E-mail: naschitz@technion.ac.il

Received: August 02, 2017 **Accepted:** August 21, 2017 **Published:** August 28, 2017

needing help with both ADL and IADL and 7 indicates complete dependence. The average CSFA in the study population was 6.6 ± 0.32. Excluded were patients not fully alert and those affected by an intercurrent illness such as febrile state, diarrhea, severe acute pain, exacerbation of dyspnea, and acute renal failure.

Measurements

The brachial BP and HR were measured at heart level with a Spot Vital Signs® validated [13,14] automatic oscillometric device (**Welch Allyn Inc. Corporate**, NY 13153-0220 USA). Supine BP and HR were recorded by a nurse at bedside at 7 am.; for analysis, measurements taken over the previous 10 days were used, including the measurement on the test day. Sitting BP and HR before lunch at 12 am., were measured on test day by a physician after the patients had been sitting in the dining room for 30-120 minutes. Three to five measurements were acquired, scrutinized for artifacts in real time, and discarded as appropriate. Sitting BP and HR after lunch, in the dining room, at 12.40-13.00 am., were determined by the same physician. The medians of 3-5 measurements - supine, sitting before lunch and sitting after lunch-were chosen for analysis.

Patient alertness and incident symptoms were assessed shortly before lunch and shortly after lunch. Incident symptoms were recorded, including dizziness, fatigue, lightheadedness, visual impairment, headache, chest pain, and pain in the shoulders or neck. Shortly after lunch the patients were returned to their beds. Incident symptoms during the subsequent two hours were followed by nurses.

The caloric content and composition of the food consumed on the current lunch were calculated by a dietician.

Primary outcome measures of our study were the number of tests discontinued and incident symptoms during tests. Secondary outcome measures were incident OH (OH equivalent), incident PPH (PPH equivalent), and mean BP <60 mmHg at any time during the test. The differences between supine BP and sitting before lunch BP were used to diagnose OH. A BP drop to a magnitude, which on standard testing is diagnosed as OH [1], by the present protocol was called 'OH equivalent'. Differences between sitting before lunch SBP and sitting after lunch SBP were used to diagnose PPH. We used the label 'PPH equivalent' to indicate a BP drop that under standard conditions [8] would be called PPH. The latter was correlated with the caloric content of the lunch consumed.

Statistical analysis

Paired Student's test and Pearson's correlation coefficient were applied to compute statistical significance, as appropriate.

Results

During a 4 month period, 50 consecutive patients fitting the inclusion criteria were evaluated. Their average age was 79.4 ± 10 years, with 22 males and 28 females. Seventeen patients had advanced stage malignancy (12 in hospice status). The remainders were admitted for treatment of pressure sores stages III and IV. Forty-one patients were hypertensive and 34 among them received BP lowering medications. Twenty-two patients were diagnosed with type 2 diabetes mellitus, 17 with stable ischemic heart disease, 16 with stable congestive heart failure, and 8 with advanced chronic renal failure (GFR <30 mL/min). Thirty five patients went through the study once and 15 patients underwent the protocol twice, 1-3 weeks apart. Sixty three patients completed the test while in full alertness and free of symptoms. In two instances the test was aborted when presyncope occurred during the meal: the causes where other than OH and PPH. In one patient, a 72 year old woman, the obvious cause was dumping syndrome as a result of Roux-on-Y gastric bypass surgery for morbid obesity that was complicated by sepsis and development of pressure sores. After ingesting as little as 50 mL of soup the patient became dizzy, pale, was sweating, and only found relief ten minutes after being taken to bed. Another patient developed a typical vasovagal reaction during the meal (nausea, bradycardia, and hypotension); she was in hospice care having metastatic carcinoma involvement of the gastric antrum. Two previous syncopal episodes occurred under different circumstances. The latter two cases were excluded from analysis.

The BP and HR measurements in 48 patients (63 tests carried out) were evaluated. The average supine SBP was 121.2 ± 6.8 mmHg, DBP 67.7 ± 10.5 mmHg, HR 80.8 ± 13.7 bpm. The average sitting before lunch SBP was 118.2 ± 19.1 mmHg, DBP 62.5 ± 10 mmHg, HR 76.8 ± 12.8 bpm. The average sitting after lunch SBP was 117.2 ± 20.9 mmHg, DBP 61.5 ± 9.9 mmHg, HR 79.6 ± 13.9 bpm. The supine to sitting SBP difference was 3.2 ± 9.2 mmHg, DBP difference 4.7 ± 10.8 mmHg (p 0.0003), and HR difference -3.8 ± 14 bpm. The sitting before lunch to sitting after lunch SBP difference was 0.4 ± 12 mmHg, DBP difference 0.7 ± 7.5 mmHg, HR difference -2.9 ± 11.5 bpm. The supine to postprandial sitting differences were SBP 4.1 ± 19.9 mmHg, DBP 6.2 ± 10.3 mmHg (p 0.00001), HR -1 ± 14.8 bpm. Reproducibility of test results was assessed in 15 patients, the interval between the pair of tests being 7-21 days. No significant differences were found in either parameter.

In 31 of 63 completed tests (47.6%) the BP changes were consistent with OH (equivalent) and/or PPH (equivalent) (Table 1). During the tests all patients remained alert and free of symptoms, including patients having low supine BP, hypertensives receiving two or more antihypertensive medications, and patients with stage D heart failure. A patient with advanced Parkinson's disease and a paraplegic patient, both thought to be at high risk of OH, had no significant BP drop during the test. The mean BP in 63 tests were supine 85.5 ± 11 mmHg, sitting before lunch 80.8 ± 10.8 mmHg, and sitting after lunch 80.0 ± 11.5 mmHg. In no instance were the mean BP less than 60 mmHg. During a 2-16 months hospitalization, there were neither falls, syncope, stroke, nor acute coronary events in the study population.

The lunch caloric content on study day was calculated in 20 patients: the average caloric content was 378 ± 166 Kcal and the average carbohydrate content was 166 ± 92 Kcal. Correlations between lunch caloric content and the BP change following lunch were RR 0.123 for SBP and RR 0.127 for DBP

Table 1: Number of tests exhibiting OH and PPH (equivalents).

BP change	Supine to sitting before lunch	Sitting before lunch to sitting after lunch
SBP decrease by ≥ 20 mmHg	9	8
DBP decrease by ≥ 10 mmHg	16	4
A and/or B	23	8

Discussion

The literature offers a plethora of data about OH and PPH, in a variety of patient populations and under different clinical circumstances. We did not aspire to expand on the epidemiology of OH and PPH; rather we intended to observe whether OH and PPH do encumber frail older people and hinder them in sharing common social place and social activities. We found that a large proportion of frail older people, living in skilled nursing or palliative care, are able to withstand the postural and prandial challenges of daily life, even though a degree of asymptomatic OH and/or PPH may be present. Under probably similar conditions, Vaitkevicius found that only 2 among 113 patients became acutely symptomatic, while 36% had PPH [15].

In interpreting this data, several questions arise. Were the methods used in this study adequate to diagnose clinically significant OH and PPH? Does a mean BP ≥ 60 mmHg (63/63 tests) indicate that cerebral perfusion is adequate during OH and/or PPH, particularly in frail older people? Could asymptomatic OH and PPH (31/63 tests) impact on fall risk, cognition and life expectancy in frail older people? Are data of this study in line with the prevailing knowledge? Finally, can these observational figures impact on clinical practice?

An average of 3 office BP readings taken within 5 minutes of rest before measurement, the patients seated with their back supported, using proper cuff size, with no conversation during the rest period and BP determinations is considered to be an adequate routine [16]. The latter standard was adopted in our study. Three orthostatic tests are widely used for the diagnosis of OH: the supine-to-standing, supine-to-sitting and the head-up tilt test. Yet, the usual OH testing may be inappropriate or inaccessible in older people in hospice or skilled nursing care. Therefore we adopted a variant examination, with key features resembling the standard protocol. So, 3-5 measurements were taken at each posture; a longer than 30 minutes of sitting before the second round of measurements assured that neither delayed OH nor progressive OH is missed. Indeed, on tilt testing for 30 minutes most cases of delayed OH in older patients were identified [17]. Based on the rate of BP drop and the degree of BP recovery, Cooke described three distinct BP profiles: small drop-fast recovery; medium drop - slow recovery; large drop non-recovery [18]. In our study, a period longer than 30 minutes sitting before the second round of measurements assured that progressive OH had not been missed.

The consequences of OH and PPH occurring in frail older persons are controversial [19]. Residents in long-term geriatric care often have a propensity to falls, but the causality of the association between OH and falls has not been clearly demonstrated [20-23]. Numerous studies have implicated hypotension in causing cognitive impairment [24,25] but whether OH contributes to cognitive decline and dementia is uncertain [24,26]. Moreover, OH does not influence mortality in frail older persons [27]. In our study, a mild degree of asymptomatic OH and/or PPH was present in 31/63 tests, apparently without negative consequences during a hospitalization of 2-16 months since no falls, syncope, stroke or acute coronary events occurred.

In general, a brachial mean BP ≥ 60 mmHg (63/63 tests in the present study) is considered sufficient to provide an adequate cerebral blood flow. However, under certain acute disorders the situation may differ. So, cerebral blood flow autoregulation may be compromised early after head injury or during an acute ischemic stroke and the brain may remain unprotected against potentially harmful effects of BP changes. Also, cerebral blood flow autoregulation may be compromised during sepsis, potentially resulting in brain damage [28]. No patient in our study belonged to either category mentioned above. As a rule, a mean BP ≥ 60 mmHg is a precondition of an adequate cerebral blood flow, but research which established that a mean BP ≥ 60 mmHg is protective against hypotensive damage to the brain did not comprise subjects resembling our patient population.

Limitations

Brief episodes of hypotension may go unobserved under the method we used. Yet, any persistent, steady or incremental OH would not been missed. Second, when PPH occurs in the sitting patient there is no clear separation between a postural and a prandial trigger. This issue, which is of theoretical interest, does not cause a difficulty in the present study: two patients who experienced a decline of BP shortly after the meal were diagnosed with vasovagal reaction and dumping syndrome and not with PPH. We might have missed the diagnosis of PPH when occurring later on after lunch, since recumbence does not prevent PPH and a severe PPH may disguise an innocent nap after the meal. There is ambiguity also concerning the safety of a mean BP ≥ 60 mmHg with regard to cerebral ischemia.

In observing that severely frail older people well tolerated the postural and prandial challenges to which they had been habitually exposed, the message could be that systematic screening residents for OH and PPH might be unnecessary and avoidable. There is no proof that increasing diagnostic rates of asymptomatic OH and PPH in patients with severe frailty will improve the clinical outcomes [3,24]. On the other hand, when symptoms of low cerebral perfusion are manifest an appropriately elaborate work-up should be implemented. The routine of residents sitting and eating in the dining room is always preferred to isolation and being bed-bound.

Conclusions

A large number of frail older people residing in skilled nursing or palliative care tolerate every-day postural and prandial challenges, even though asymptomatic OH and/or PPH may be present. Based on observational data, we suggest that systematic screening residents in palliative or skilled nursing care for OH and PPH is unnecessary and should not be undertaken.

References

1. Freeman R, Wieling W, Axelrod FB, Benditt DG, Benarroch E, et al. (2011) Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. Clin Auton Res 21: 69-72.
2. Rutan GH, Hermanson B, Bild DE, Kittner SJ, LaBaw F, et al. (1992) Orthostatic hypotension in older adults. The Cardiovascular Health Study. CHS Collaborative Research Group. Hypertension 19: 508-519.
3. Frith J, Newton JL, Parry SW (2014) Measuring and defining orthostatic hypotension in the older person. Age Ageing 43: 168-170.
4. Scuteri A, Modestino A, Frattari A, Di Daniele N, Tesauro M (2012) Occurrence of hypotension in older participants. Which 24-hour ABPM parameter better correlate with? J Geront A Biol Sci Med Sci 67: 804-810
5. Oishi E, Sakata S, Tsuchihashi T, Tominaga M, Fujii K (2016) Orthostatic hypotension predicts a poor prognosis in elderly people with dementia. Intern Med 55: 1947-1952.
6. Rockwood MR, Howlett SE, Rockwood K (2012) Orthostatic hypotension (OH) and mortality in relation to age, blood pressure and frailty. Arch Gerontol Geriatr 54: e255-260.
7. Jansen RW, Lipsitz LA (1995) Postprandial hypotension; epidemiology, pathophysiology, and clinical management. Ann Intern Med 37: 438-443.

8. Barochiner J, Alfie J, Aparicio LS, Cuffaro PE, Rada MA, et al. (2014) Postprandial hypotension detected through home blood pressure monitoring: a frequent phenomenon in elderly hypertensive patients. Hypertens Res 37: 438-443

9. Trahair LG, Horowitz M, Jones KL (2014) Postprandial hypotension: a systematic review. J Am Med Dir Assoc 15: 394-409.

10. Vloet LC, Pel-Little RE, Jansen PA, Jansen RW (2005) High prevalence of postprandial and orthostatic hypotension among geriatric patients admitted to Dutch hospitals. J Gerontol A Biol Sci Med Sci 60: 1271-1277.

11. Lampela P, Lavikainen P, Huupponen R, Leskinen E, Hartikainen S (2013) Comprehensive geriatric assessment decreases prevalence of orthostatic hypotension in older persons. Scand J Public Health 41: 351-358.

12. Punchick B, Freud T, Press Y (2016) The association between orthostatic hypotension and cognitive state among adults 65 years and older who underwent a comprehensive geriatric assessment. Medicine (Baltimore) 95: e4264.

13. Rockwood K, Song X, MacKnight C, Bergman H, Hogan DB, et al. (2005) A global clinical measure of fitness and frailty in elderly people. CMAJ 173: 489-495.

14. Alpert BS (2007) Validation of the Welch Allyn Spot Vital Signs blood pressure device according to the ANSI/AAMI SP10: 2002. Accuracy and cost-efficiency successfully combined. Blood Press Monit 12: 345-347.

15. Vaitkevicius PV, Esserwein DM, Maynard AK, O'Connor FC, Fleg JL (1991) Frequency and importance of postprandial blood pressure reduction in elderly nursing-home patients. Ann Intern Med 115: 865-870.

16. Myers MG, Godwin M, Dawes M, Kiss A, Tobe SW, et al. (2010) Measurement of blood pressure in the office: recognizing the problem and proposing the solution. Hypertension 55: 195-200.

17. Gurevich T, Machmid H, Klepikov D, Ezra A, Giladi N, et al. (2014) Head-up tilt testing for detecting orthostatic hypotension: how long do we need to wait? Neuroepidemiology 43: 239-243.

18. Cooke J, Carew S, Quinn C, O'Connor M, Curtin J, et al. (2013) The prevalence and pathological correlates of orthostatic hypotension and its subtypes when measured using beat-to-beat technology in a sample of older adults living in the community. Age Ageing 42: 709-714.

19. Angelousi A, Girerd N, Benetos A, Frimat L, Gautier S, et al. (2014) Association between orthostatic hypotension and cardiovascular risk, cerebrovascular risk, cognitive decline and falls as well as overall mortalilty: a systematic review and meta-analysis. J Hypertens 32: 1562-1571.

20. Ooi WL, Hossain M, Lipsitz LA (2000) The association between orthostatic hypotension and recurrent falls in nursing home residents. Am J Med 108: 106-111

21. Hartog LC, Cimzar-Sweelssen M, Knipscheer A, Groenier KH, Kleefstra N, et al. (2016) Orthostatic hypotension does not predict recurrent falling in a nursing home population. Arch Gerontol Geriatr 68: 39-43.

22. Maurer MS, Cohen S, Cheng H (2004) The degree and timing of orthostatic blood pressure changes in relation to falls in nursing home residents. J Am Med Dir Assoc 5: 233-238.

23. Juraschek SP, Daya N, Appel LJ, Miller ER, Windham BG, et al. (2017) Orthostatic Hypotension in Middle-Age and Risk of Falls. Am J Hypertens 30: 188-195.

24. O'Callaghan S, Kenny RA (2016) Neurocardiovascular Instability and Cognition. Yale J Biol Med 89: 59-71.

25. Curreri C, Giantin V, Veronese N, Trevisan C, Sartori L, et al. (2016) Orthostatic Changes in Blood Pressure and Cognitive Status in the Elderly: The Progetto Veneto Anziani Study. Hypertension 68: 427-435.

26. Frewen J, Savva GM, Boyle G, Finucane C, Kenny RA (2014) Cognitive performance in orthostatic hypotension: Findings from a nationally representative sample. J Am Geriatr Soc 62: 117-122.

27. Freud T, Punchik B, Press Y (2015) Orthostatic hypotension and mortality in elderly frail patients: a retrospective cross-sectional study. Medicine (Baltimore) 94: e977.

28. Goodson CM, Rosenblatt K, Rivera-Lara L, Nyquist P, Hogue CW (2016) Cerebral Blood Flow Autoregulation in Sepsis for the Intensivist: Why Its Monitoring May Be the Future of Individualized Care. J Intensive Care Med.

Author Affiliations

Top

Department of Internal Medicine A, Bnai-Zion Medical Center, Israel