Prevalence of Postural Hypotension at Baseline in the Systolic Hypertension in the Elderly Program (SHEP) Cohort

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Objective: The objective of this study was to examine the prevalence and correlates of postural hypotension (defined as a drop in systolic blood pressure of ≥20 mm Hg) in a cohort of elderly persons with isolated systolic hypertension (ISH).
Design: Baseline cross-sectional analysis of the 4,736 persons randomized in the Systolic Hypertension in the Elderly Program (SHEP).
Setting: A randomized multi-center double-blind outpatient clinical trial of the impact of treating ISH.
Participants: Men and women age ≥ 60 years with the systolic blood pressure (SBP) ≥ 160 mm Hg and diastolic blood pressure (DBP) < 90 mm Hg.
Measures: Medical histories were obtained using interviewer-administered, standardized clinical history forms. At entry into the study, seated and standing BP was measured by certified BP technicians using a random zero sphygmomanometer. Postural hypotension (PH) was assessed at 1 and 3 minutes after the participant arose from a seated position.
Main results: PH was found in 10.4% of participants at 1 minute and in 12.0% of participants at 3 minutes. 5.3% of participants demonstrated PH at both time intervals while 17.3% demonstrated PH at either or both of the time intervals. Factors significantly ($P < 0.05$) associated with the presence of PH were higher mean SBP and a lower mean body mass index.
Conclusions: Somewhat different persons were defined as having PH based upon the 1 minute and 3 minute standing measures of BP, and prevalence estimates of PH can vary depending on whether one or more intervals of measurement are used. Cross-sectional data analysis indicated that PH, in healthy community-dwelling older persons with ISH, may not be associated with a history of disorders or problems usually thought to be related to PH. However, prospective data are needed to determine the prognostic significance of PH, and whether one or multiple measurements carry more significance. **J Am Geriatr Soc 1057–1064, 1991**

In the healthy elderly, postural hypotension occurs infrequently and is usually asymptomatic.[1–3] In older persons who are ill or frail, postural hypotension can be a symptomatic disorder associated with increased falls and syncope.[4,5] Age and systolic blood pressure (SBP) have been reported to be risk factors for postural hypotension.[2] Analysis of the magnitude of postural hypotension in subgroups of persons with isolated systolic hypertension (ISH) may clarify approaches to screening for postural hypotension by possibly identifying persons at higher risk. Data on the frequency of postural hypotension among persons with ISH should also serve to guide treatment of ISH at a time when increasing numbers of physicians report they are using drug therapy for this condition.[6]

The Systolic Hypertension in the Elderly Program (SHEP) has enrolled 4,736 persons, ages 60 and greater, with a SBP ≥ 160 mmHg and diastolic blood pressure (DBP) <90 mmHg.[7] This paper describes the baseline prevalence of postural hypotension in the SHEP cohort and factors associated with orthostatic decreases in blood pressure.

PARTICIPANTS AND METHODS

Design SHEP is a multicenter clinical trial. Men and women age 60 years and above with isolated systolic hypertension (n = 4,736) were recruited into this double-blind, placebo-controlled, stepped-care treatment program and followed for an average of five years. The primary end point of the trial is fatal plus non-fatal stroke. Eligibility was determined at two baseline visits (BV). Persons not on antihypertensive medications at initial contact immediately began baseline visits to determine eligibility. Persons on antihypertensive medication at the initial contact were eligible for a drug withdrawal procedure monitored carefully at multiple drug evaluation visits (DEV) over an 8–10-week period to determine if they might have isolated systolic hypertension. If so, these persons were then eligible for the baseline visits. If the average of four blood pressure readings off all antihypertensive medications at the first and second baseline visits was SBP ≥160 mmHg and DBP <90 mmHg, then the participant was eligible for randomization if other inclusion criteria were met, no exclusion criterion was present, and written informed consent was given.

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Other inclusion criteria were age of 60 years or older, willingness to comply with study protocol, and no anticipated change in residence. Exclusion criteria were evidence of atrial flutter or fibrillation; second-or-third-degree atrioventricular block; multifocal or frequent ventricular premature beats; heart rate of less than 50 beats per minute; permanent pacemaker; history of stroke with residual paresis or other neurologic disability; significant renal dysfunction; presumed alcohol abuse; history of coronary artery bypass surgery or myocardial infarction within the past 6 months; current treatment with insulin, anticoagulants, or drugs having antihypertensive activity; uncontrolled congestive heart failure, malignant neoplasm or other life threatening disorder; contraindication to any antihypertensive medications potentially to be used in SHEP; peripheral arterial disease with evidence of tissue injury; dementia (based on clinical judgement); residence in a nursing home; history of a transient ischemic attack (TIA) and carotid bruit in the appropriate location, history of 2 previous TIA's in the same distribution; malignant hypertension past or present; or treatment for known diastolic hypertension.

Blood Pressure Measurement All blood pressures were taken by trained and certified staff using random zero sphygmomanometers. The participant was seated for a 5-minute rest period, and then three seated pressures were taken at each visit. The first pressure was discarded (as has been done in many randomized hypertension treatment trials to reduce variability in baseline blood pressure), and the second and third readings were averaged for the baseline blood pressure calculation. Immediately after these blood pressures were taken, the participant stood and blood pressure and pulse measurements were repeated after 1 and 3 minutes.

For the purposes of this paper, blood pressure and pulse measured at the second baseline visit are used in this analysis since most of the data used in the following analysis were also collected at this visit. Since the blood pressure entry criterion for the study was based on an average of the readings taken at *both* baseline visits, some of the participants had a systolic blood pressure somewhat below 160 mmHg at the second baseline visit.

Other Data Collection At the first clinic visit prior to randomization, participants completed a medical history questionnaire. At the second visit an in-depth medical history, physical examination, and electrocardiogram were performed. Body mass index was assessed as weight (kilograms) divided by height (meters) squared. The Rose Questionnaire for angina and the Center for Epidemiologic Studies Depression (CES-D) inventory were also administered.[8,9] For eligible patients, randomization occurred at the end of the second baseline visit.

Analysis A 20 mmHg or greater decrease in SBP at baseline visit 2 (BV2) was defined as postural hypotension.[1] In addition, since the absolute drop in systolic pressure could be correlated with the level of the SBP, analyses were also performed using a 10% or greater drop in SBP as an alternative definition of postural hypotension.[5]

T-tests and chi-square tests were used to compare means and proportions of selected variables in the groups of individuals with or without postural hypotension. Quartile analysis and logistic regression were also used to examine the relation of these variables to the presence of postural hypotension.

RESULTS

The distribution of postural changes in SBP at 1-minute standing among SHEP participants is shown in Figure 1. At baseline a decrease in SBP of 20 mmHg or greater was found in 10.4% of the participants at 1 minute. Of these, 3.4% had reported a recent history of troublesome or intolerable dizziness, while 3.0% of persons without postural hypotension had the same complaints. The distribution of change in SBP after 1 minute of standing among the remaining participants was as follows: −19 to −10 mmHg, 23.3%, −9 to 0 mmHg, 35.0%, +1 to +19 mmHg, 29.3%, and ≥ +20 mmHg, 2.0%. The largest drop in SBP at 1 minute was 66 mmHg and the largest increase 78 mmHg.

Postural changes in SBP after 3 minutes of standing were similar to those after 1 minute. A decrease in SBP of 20 mmHg or more after 3 minutes was found in 12.1% of participants. At this time interval, 2.6% of those with postural hypotension versus 3.1% of those without had a recent history of troublesome or intolerable dizziness. The distribution of orthostatic change in SBP at the 3-minute interval was nearly identical to that at the 1-minute interval. The distribution of change in SBP after 3 minutes of standing among the remaining participants was as follows: −19 to −10 mmHg, 23.4%, −9 to 0 mmHg, 34.7%, +1 to +19 mmHg, 27.9%, and ≥ +20 mmHg, 1.9%. The largest drop in SBP at 3 minutes was 102 mmHg and the largest increase 60 mmHg.

The percentage of persons who had a decrease in SBP of 20 mmHg or greater at both the 1-minute and the 3-minute time intervals was 5.3%. Therefore, although the percentage of the cohort having a drop of 20 mmHg or greater at 1 or 3 minutes was similar, this criterion often identified different individuals at the two time intervals; hence a smaller percentage manifested this response at both 1 and 3 minutes. Of those included in this definition of postural hypotension, 3.6% had a history of troublesome or intolerable dizziness, while 3.0% of persons without postural hypotension had the same complaints. The percentage having such a drop at either or both of the time intervals was 17.3%. Of those included in this definition of postural hypotension, 2.8% had reported a recent history of troublesome or intolerable dizziness, while 3.1% of persons without postural hypotension had the same complaints.

With use of a 10% or more drop in SBP at 1 minute to define postural hypotension, 14.1% of SHEP participants met this criterion; 3.3% with postural hypotension versus 3.0% without complained of troublesome or intolerable dizziness.

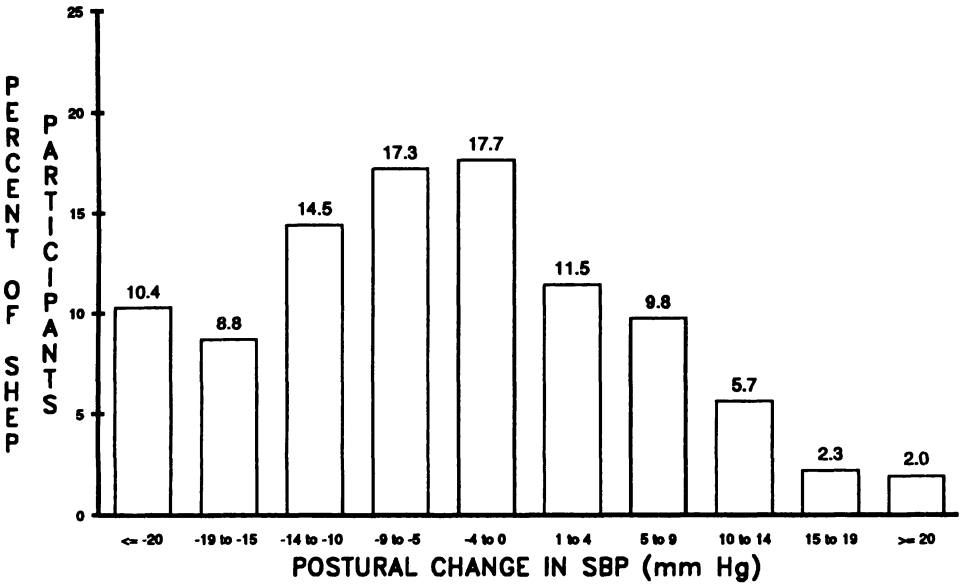

FIGURE 1. Distribution of postural change (sitting minus standing at 1 minute) in systolic blood pressure (SBP) at baseline in the Systolic Hypertension in the Elderly Program cohort.

TABLE 1. PERSONS WITH STANDING SBP DROPS OF 20 mmHg OR MORE AT BASELINE BY INTERVAL OF MEASUREMENT, STRATIFIED BY SELECTED CHARACTERISTICS

Characteristic	n	1-Minute		3-Minute		Both		Either or Both	
		n	%	n	%	n	%	n	%
Total	4732	493	10.4	573	12.1	249	5.3	817	17.3
Age 60-69 years	1963	193	9.8	215	11.0	102	5.2	306	15.6
70-79	2120	232	10.9	273	12.9	112	5.3	393	18.5
80+	649	68	10.5	85	13.1	35	5.4	118	18.2
BV2 SBP<160 mmHg	785	47	6.0	59	7.5	25	3.2	81	10.3
160-169	1917	189	9.9	185	9.7	85	4.4	289	15.1
170-179	1169	131	11.2	167	14.3	73	6.2	225	19.2
180+	861	126	14.6	162	18.8	66	7.7	222	25.8
White	3748	385	10.3	455	12.1	196	5.2	644	17.2
Black	984	108	11.0	118	12.0	53	5.4	173	17.6
Men	2044	199	9.7	230	11.3	100	4.9	329	16.1
Women	2688	294	10.9	343	12.8	149	5.5	488	18.2
Prior Meds*	1575	165	10.5	211	13.4	89	5.7	287	18.2
No Prior Meds	3157	328	10.4	362	11.5	160	5.1	530	16.8

SBP = systolic blood pressure; based on the mean of 4 readings at BV2.
BV2 = second baseline visit (prior to randomization).
** Prior Meds = receiving antihypertensive medication at initial contact visit.*

Table 1 shows baseline blood pressure and demographic characteristics of the entire study cohort: those with postural hypotension (20 mmHg or more orthostatic drop) at 1 minute, 3 minutes, both, or either and both. The percent with postural hypotension was slightly higher with greater age. It was also higher with higher levels of SBP. For example, of participants with sitting SBP <160 mmHg, 6.0% demonstrated postural hypotension at 1 minute, while 14.6% of persons with SBP ≥ 180 mmHg had a 20 mmHg or greater drop in SBP. Participants taking antihypertensive drugs prior to entering SHEP had a slightly higher prevalence of postural hypotension than those who had not taken such drugs. As shown in Table 1, the relationships between baseline characteristics and postural hypotension were essentially the same at the 1- and 3-minute measurement intervals. Similar results were observed when postural hypotension was defined as an orthostatic change of 10% or more of SBP (data not shown).

Table 2 further examines factors possibly associated with postural hypotension. Mean body mass index (BMI) and mean level of SBP were significantly ($P < 0.05$) different for subgroups with and without postural hypotension for all four definitions (based on measurement at 1 minute, 3 minutes, both, or either or both as shown in Table 2). For those with compared to those without postural hypotension at the various time intervals, mean SBP was about 3.3–4.3 mmHg higher, and mean BMI was about 0.8–1.1 units lower. There were no significant or substantial differences in age, sex, race, pulse change (standing minus sitting), or history of smoking, previous myocardial infarction, diabetes

TABLE 2a. FACTORS ASSOCIATED WITH POSTURAL HYPOTENSION (SBP DROP OF 20 mm Hg OR MORE) AT 1-MINUTE, 3-MINUTE, EITHER, BOTH OR NEITHER INTERVALS

Factor	Total Cohort (n = 4732)	With Postural Hypotension 1 Minute (n = 493)	With Postural Hypotension 3-Minute (n = 573)
Age 60–74, %	67.4	65.3	64.4
75+, %	32.6	34.7	35.6
Mean (SD)	71.6 (6.7)	72.0 (6.8)	72.1 (6.7)
Sex: Male, %	43.2	40.4	40.1
Race: White, %	79.2	78.1	79.4
On Meds, %	33.3	33.5	36.8
Body Mass Index,			
Mean (SD)	27.5 (5.0)	26.8* (4.6)	26.7* (4.5)
Serum Glucose,			
Mean (SD)	108.5 (33.6)	106.8 (33.4)	106.2 (27.0)
History of:			
Smoking, %	12.7	13.8	11.3
MI, %	3.8	4.3	3.5
Angina, By Rose Questionnaire, %	5.5	5.3	6.8
Diabetes, %	10.1	9.1	8.4
Falls, %	3.1	3.7	2.3
Troublesome or Intolerable Dizziness, %	3.0	3.4	2.6
MI by Baseline ECG, %	2.0	1.2	1.7
CESD Depression Score > 7, %	7.5	7.3	8.2
BV2 Pulse Rate (Seated)			
Mean (SD)	70.8 (10.5)	70.0 (10.0)	69.5* (10.2)
Pulse Change,			
Mean (SD)	5.1 (7.3)	5.2 (7.3)	5.3 (7.7)
BV2 SBP			
Mean (SD)	169.8 (11.7)	172.8* (12.4)	173.7* (13.1)
<160, %	16.6	9.5	10.3
160–169, %	40.5	38.3	32.3
170-179, %	24.7	26.6	29.1
180+, %	18.2	25.6	28.3
Hematocrit,			
Mean, SD	42.3 (4.5)	41.9 (4.1)	42.1 (4.3)

*$P < 0.05$ = Mean (percentage) is significantly different from mean (percentage) in group without postural hypotension.
BV2 = Second baseline visit (prior to randomization).
MI = Myocardial Infarction.
ECG = Electrocardiogram.

mellitus, falls, troublesome or intolerable dizziness, myocardial infarction by baseline electrocardiogram, or CES-D depression score for subgroups with and without postural hypotension for all four definitions (Table 2). The mean levels of age, serum glucose, baseline pulse, and hematocrit showed some differences for certain definitions of postural hypotension, but the differences shown were not consistent across all four definitions and were small.

With use of quartile analysis, the only two factors that were significantly ($P < 0.01$) and strongly associated with all definitions of postural hypotension were SBP and BMI. Logistic regressions were performed with the dependent variable postural hypotension (present or absent) and the independent variables, (1) either SBP or BMI, (2) SBP and BMI, and (3) SBP and BMI plus age, sex, race, pulse, hematocrit, serum glucose, and antihypertensive medication status at the initial contact (ie, whether an individual had been on antihypertensive medication prior to entry into the SHEP trial). For SBP, with the several definitions of postural hypotension, the logistic regression coefficients ranged from 0.021 to 0.029 ($P < 0.0001$), indicating prevalence of postural hypotension was about 1.2 to 1.3 times more likely for each 10 mmHg higher SBP. For BMI, coefficients ranged from −0.037 to −0.053 ($P < 0.002$), indicating prevalence of postural hypotension was about 1.2 to 1.3 times more likely for each 5 unit lower BMI.

DISCUSSION

Based on the criterion of a 20-mmHg or greater fall in SBP at 1 minute after standing, postural hypotension in the large SHEP cohort of individuals with isolated systolic hypertension was found among approximately 10% of the participants. Prevalence was similar at 3 minutes after standing. With prevalence estimate based on a 20-mmHg or greater drop at both of these intervals, prevalence was only 5%; with the criterion of a 20-mmHg drop at either or both of these intervals, prevalence was 17%. Prevalence was positively correlated with level of SBP and negatively correlated with body mass index. An orthostatic decrease of SBP of 20 mmHg or more was not significantly associated with

TABLE 2b. FACTORS ASSOCIATED WITH POSTURAL HYPOTENSION (SBP DROP OF 20 mm Hg OR MORE) AT 1-MINUTE, 3 MINUTE, EITHER, BOTH OR NEITHER INTERVALS

Factor	With Postural Hypotension Both (n = 249)	With Postural Hypotension Either (n = 817)	No Postural Hypotension (n = 3915)
Age 60–74, %	66.3	64.4	68.0
75+, %	33.7	35.6	32.0
Mean (SD)	71.9 (6.6)	72.1* (6.7)	71.5 (6.7)
Sex: Male, %	40.2	40.3	43.8
Race: White	78.7	78.8	82.0
On Meds, %	35.7	35.1	32.9
Body Mass Index, Mean (SD)	26.5* (4.4)	26.8* (4.6)	27.7 (5.1)
Serum Glucose, Mean (SD)	105.0* (25.7)	107.0 (31.3)	108.8 (34.0)
History of:			
Smoking, %	11.2	12.9	12.7
MI, %	2.8	4.2	3.8
Angina, by Rose Questionnaire, %	6.0	6.1	5.3
Diabetes, %	9.2	8.6	10.4
Fall, %	2.4	3.1	3.1
Troublesome or Intolerable Dizziness, %	3.6	2.8	3.1
MI by Baseline ECG, %	1.2	1.6	2.0
CESD Depression Score > 7, %	7.6	7.8	7.4
BV2 Pulse Rate (Seated) Mean (SD)	69.8 (10.2)	69.7* (10.1)	71.0 (10.6)
Pulse Change, Mean (SD)			
BV2 SBP			
Mean (SD)	173.4* (12.8)	173.2* (12.8)	169.1 (11.3)
<160, %	10.0	9.9	16.7
160-169, %	34.1	35.4	41.5
170-179, %	29.3	27.5	24.6
180+, %	26.5	27.2	17.2
Hematocrit, Mean (SD)	41.7* (4.1)	42.1 (4.3)	42.3 (4.6)

*$P < 0.05$ = Mean (percentage) is significantly different from mean (percentage) in group without postural hypotension.
BV2 = second baseline visit (prior to randomization).
MI = myocardial infarction.
ECG = electrocardiogram.

age, race, sex, prior antihypertensive medication status, pulse rate, change in pulse rate on standing, serum glucose, hematocrit, history of smoking, previous myocardial infarction, angina pectoris, diabetes mellitus, falls, troublesome or intolerable dizziness, myocardial infarction by electrocardiogram, or CES-D depression score. Therefore, it appears that in relatively healthy community-dwelling older persons with ISH, the presence of an orthostatic decrease of SBP of 20 mmHg or more is not associated with serious underlying disorders.

Appropriate timing for the measurement of an orthostatic drop in SBP is not clear from the literature.[1,10,11] Some studies have used a 1-minute interval to test for postural hypotension,[1] while others have used a 3-minute interval.[11] The SHEP data indicate that neither the prevalence nor the correlates of an orthostatic drop of SBP differ significantly when the blood pressure is measured at either 1 or 3 minutes after standing. However, different individuals were often characterized as having postural hypotension at the 1-minute measurement compared to the 3-minute measurement. Also, if prevalence is defined as having postural hypotension at either or both of these intervals, prevalence is considerably higher. The significance of these findings will have to await prospective follow-up of this cohort to determine if having postural hypotension at the 1- or 3-minute interval or both has prognostic significance.

In this study we used the difference between seated and standing blood pressure to define postural hypotension. Although the prevalence of postural hypotension would be somewhat higher if supine versus standing blood pressures were used, seated blood pressure is the standard recommended format for comparisons in clinical settings.[12] Previous studies have varied with regard to whether supine or seated measures were used to establish the baseline.[1,11]

A limitation of this study is that the cohort consists of volunteers who met screening criteria for a multicenter clinical trial on ISH. Although the cohort is large, SHEP findings are not necessarily generalizable to community populations of persons age 60 and over. However, SHEP findings on the prevalence of postural

hypotension after 1 minute of standing are similar to those reported in a smaller study by Mader.[1] The overall prevalence of postural hypotension in that study (defined as a decrease in SBP of 20 mmHg or greater after 1 minute of standing) was 10.7% in 304 consecutive community-dwelling elderly individuals who came to a center for a routine physical examination. In a subgroup in that study without known risk factors for systolic hypotension, such as use of medications influencing blood pressure, abnormal heart rate, arrhythmia, or abnormal neurological findings, prevalence was 6.4%. In addition, data on blood pressure measured in the supine and sitting position in over 8,000 participants in the National Health and Nutrition Examination Survey demonstrated a greater prevalence of postural hypotension with older age, but the data indicated that this was due primarily to an age associated greater SBP in the supine position.[2,12] In Mader's study and in SHEP, level of SBP was associated with postural hypotension. The reason for the inverse association between body mass index and the prevalence of postural hypotension is unknown, although this finding has been reported previously.[11] Both of these cross-sectional studies indicate that the prevalence of postural hypotension may be less than the 24% figure previously reported.[13]

In conclusion, in the SHEP cohort at baseline, prevalence of postural hypotension, defined as a decrease in SBP of 20 mmHg or more upon standing at 1 or 3 minutes, was 10%–12% when based on one time interval of measurement, 5% when based on both, and 17% based on either or both measurements. Because postural hypotension was not associated with a history of other possibly related disorders, its presence in healthy community-dwelling persons may not have serious implications. However, both the appropriate interval for measurement of postural hypotension and its prognostic significance can be better determined from prospective data.

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REFERENCES

1. Mader SL, Josephson KR, Rubenstein LZ. Low prevalence of postural hypotension among community-dwelling elderly. JAMA 1987;258:1511–1514.
2. Harris T, Kleinman J, Lipsitz L, Cornoni-Huntley J, Garrison R. Is age or level of systolic blood pressure related to positional blood pressure change? Gerontologist 1986;26:Suppl:59A.
3. Myers MG, Kearns PM, Kennedy DS, Fisher RH. Postural hypotension and diuretic therapy in the elderly. Can Med Assoc J 1978;119:581–584.
4. Tinetti ME, Williams TF, Mayewski R. Fall risk index for elderly patients based on number of chronic disabilities. Am J Med 1988;119:581–584.
5. Lipsitz LA, Pluchino FC, Wei JY, Rowe JW. Syncope in institutionalized elderly: the impact of multiple pathological conditions and situational stress. J Chronic Dis 1986;39:619–630.
6. Breckenridge MB, Kostis JB. Isolated systolic hypertension in the elderly: Results of a statewide survey of clinical practice in New Jersey. Am J Med 1989;86:370–375.
7. The Systolic Hypertension in the Elderly Program (SHEP) Cooperative Research Group. Rationale and design of a randomized clinical trial on prevention of stroke in isolated systolic hypertension. J Clin Epidemiol 1988;41:1197–1208.
8. Rose GA, Blackburn H. Cardiovascular Survey Methods. Geneva: World Health Organization, 1968.
9. Radloff LS. The CES-D Scale. Appl Psychol Meas 1977;1:385–401.
10. Mader SL, Palmer RM, Rubenstein LZ. Effect of timing and number of baseline blood pressure determinations on postural blood pressure response. J Am Geriatr Soc 1989;37:444–446.
11. Davis BR, Langford HG, Blaufox MD et al. The association of postural changes in systolic blood pressure and mortality in persons with hypertension: the Hypertension Detection and Follow-up Program experience. Circulation 1987;75:340–346.
12. Frolich ED, Grim C, Labarthe DR et al. Report of a special task force appointed by the Steering Committee. American Heart Association recommendations for human blood pressure determination by sphygmomanometer. Hypertension 1988;11:209A–222A.
13. Caird FI, Andrews GR, Kennedy RD. Effect of posture on blood pressure in the elderly. Br Heart J 1973;35:527–530.