The association of postural changes in systolic blood pressure and mortality in persons with hypertension: the Hypertension Detection and Follow-up Program experience

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ABSTRACT Participants in the Hypertension Detection and Follow-up Program (HDFP) were classified on the basis of baseline standing minus sitting systolic blood pressure into four groups (≤ −20 [group 1], −19 to 0 [group 2], 1 to 20 [group 3], and >20 mm Hg [group 4]) to study 5 year mortality. Group 1, 3.3% of the total, contained those participants who had postural hypotension. The 5 year total and age-adjusted mortality rates for these groups were significantly different (p < .04), with group 1 having the highest rates. To account for the possible confounding effects of certain baseline risk factors — age, sex, race, prior antihypertensive treatment, randomization group, diabetes, end-organ damage, sitting diastolic and systolic blood pressures, pulse, hematocrit, smoking status, and relative weight (percent of ideal weight) — in assessing group differences in mortality rates, a multiple logistic model was used. Relative weight proved to be a confounding factor for the association of drop in postural systolic blood pressure with mortality. However, there is an interaction between history of diabetes and postural change in systolic blood pressure. Thus, postural hypotension may indicate a poor prognosis in diabetic hypertensive patients.


MANY epidemiologic studies have identified blood pressure as an important risk factor for both cardiovascular disease and total mortality.1-12 Elevated levels of either diastolic blood pressure (DBP) or systolic blood pressure (SBP) are associated with an increased mortality risk. As a result of these findings, the importance of the detection and treatment of individuals with high blood pressure has been established.

Several pressure measures have been used to improve predictability of subsequent cardiovascular mortality. These include pulse and mean arterial pressure4 and postural changes in blood pressure.13

When a person assumes the upright posture, cardiovascular adjustments occur to maintain the blood pressure. These actions include reflex arteriolar and venous constriction, reflex acceleration of heart rate, increase in muscle tone, and an immediate increase in plasma catecholamines.14 The net result of these adjustments in the normal individual is that blood pressure remains largely unchanged, although a 10 to 15 mm Hg rise or fall in SBP is probably not an uncommon experience.15-17 A precise distribution of the normal response of blood pressure during orthostasis has not been well documented.18

If SBP drops more than 20 mm Hg 1 to 3 min after a person moves from the supine to the standing position, this person is said to have postural or orthostatic hypotension.19,20 Postural hypotension may be either symptomatic or asymptomatic. It may be idiopathic or may result from known causes; some of these causes are functional and reversible, whereas others have an anatomic basis and may be progressive.
The incidence of postural hypotension increases with advancing age and occurs in anywhere from 11% to 24% of the population over age 60.\textsuperscript{19, 20} Postural hypotension has been associated with primary hypertension.\textsuperscript{5, 16, 19, 21–23} However, few studies have examined the long-term follow-up of patients with hypertension in relation to postural changes.\textsuperscript{13, 24} This article presents an analysis of the association of baseline postural changes in SBP and mortality in a population-based study of persons with diastolic hypertension, the participants in the Hypertension Detection and Follow-up Program (HDFP).

**Methods**

The HDFP was a multicenter trial designed to investigate benefits of treatment of hypertension in a community-based population sample. Participants were recruited from 14 communities around the United States, generally by formal sampling or full screening from defined census tracts with various ethnic and socioeconomic characteristics. Within these communities, 158,906 men and women 30 to 69 years old were screened for hypertension, and ultimately 10,940 identified hypertensive individuals were enrolled in the treatment program. Half were randomly assigned to a group referred for care to existing community sources, the referred care (RC) group, and half were assigned to a special treatment group, the stepped care (SC) group. Those randomized to the SC were enrolled in special HDFP clinics designed to treat their hypertension vigorously toward a set normotensive goal, in accordance with a carefully structured stepped approach to drug therapy.

In the HDFP, three consecutive blood pressure readings (right arm, sitting) obtained with a standard manometer were taken for all persons 30 to 69 years old. If the mean of the second and third measurements of DBP (fifth phase) was 95 mm Hg or greater, the individual was invited to the HDFP clinical center regardless of current antihypertensive treatment status. At the center, if the mean of the second and fourth measurements of DBP — taken with a special mercury manometer, the Hawksley random-zero device — was 90 mm Hg or greater, the person was eligible for the study and was randomly assigned by blood pressure status (90 to 104, 105 to 114, and 115 + mm Hg) and center to the SC or RC group.

The findings contained in this article are based on data from 10,536 hypertensive participants in the HDFP clinical trial who had their standing pressures taken at a second clinic visit. At the end of this visit, participants were randomly assigned to the RC and SC groups. Further details of the methods of recruitment of the HDFP population have been given elsewhere.\textsuperscript{12}

During the second clinic visit, four measurements of sitting blood pressure were obtained, the second and fourth with the random-zero device. In this report the average of the second and fourth is considered to be the sitting blood pressure. The participant was then asked to stand, wait 90 sec, and then raise his arm to a position parallel to the floor for 30 sec (for some participants, it was necessary to support the arm in this position for the necessary 30 sec). A stethoscope was applied to the arm and the cuff was inflated, while the participant's arm was raised. Immediately thereafter the participant was asked to lower his arm and the fifth blood pressure reading was obtained with a standard sphygmomanometer. The arm was relaxed at the side during the reading. The participant remained standing and the connection was changed to the random-zero device. The participant was then asked to raise his arm for 30 sec (again, for some participants it was necessary to support the arm). At the end of the 30 sec, the stethoscope was applied to the arm; the participant was then asked to lower his arm, and the sixth reading was obtained with the random-zero device. This sixth reading, usually obtained at approximately 3 to 4 min after standing, is defined here as the standing blood pressure. Also at this time, the participant was asked if he was dizzy or faint after standing.

In this analysis, persons were classified by baseline standing minus sitting SBP (\(\Delta\) SBP) into four subgroups: (1) \(-20\) mm Hg or less, (2) \(-19\) to \(0\) mm Hg, (3) \(1\) to \(20\) mm Hg, and (4) greater than \(+20\) mm Hg) with various postural changes. Since the standard clinical definition of orthostatic postural hypotension is a fall of \(20\) mm Hg or more after assumption of the erect position from the supine one, intervals of \(20\) mm Hg were selected to divide the population into subgroups. In the HDFP, participants moved from the sitting (not the supine) to the standing position, but we have applied the same criterion (\(20 +\) mm Hg fall) in the selection of participants with orthostatic hypotension. It is reasonable to conclude that if the participants had moved from a supine to a standing position, the postural drop in pressure would likely have been even greater than that observed.

The analyses presented here include comparisons of the crude and age-adjusted 5 year mortality rates for participants by category of \(\Delta\) SBP. A logistic regression model is also presented to relate the 5 year total mortality to baseline variables. A total of 15 baseline variables were considered, including age, race, sex, treatment group, antihypertensive medication status at entry, history of diabetes, end-organ damage status, hematocrit, sitting DBP, age \(\times\) sitting DBP, sitting pulse, current smoking status, percent of ideal weight (relative weight),\textsuperscript{25} and \(\Delta\) SBP. Additional logistic models look at the interaction of \(\Delta\) SBP with all the above factors.

**Results**

The distribution of postural changes in SBP among HDFP participants is presented in figure 1. At entry a \(\Delta\) SBP of \(-20\) mm Hg or less was found in 3.3% of the participants and 14% of these complained of light-headedness or dizziness on standing. The distribution of \(\Delta\) SBPs among the remaining participants was as follows: \(-19\) to \(0\) mm Hg in 38.3%, \(+1\) to \(+20\) mm Hg in 52.9%, and greater than \(+20\) mm Hg in 5.5%.

Figure 2 shows the frequency of orthostatic hypotension in the HDFP within four age categories: 30 to 39, 40 to 49, 50 to 59, and 60 to 69 years. The percentage of participants with orthostatic hypotension increased with increasing age.

Selected baseline characteristics of subjects in each of the \(\Delta\) SBP subgroups are summarized in table 1. There was no pattern of a greater drop in SBP on standing in older participants; the average age moving from subgroup 1 to subgroup 4 varied from 53.8 to 51.0, to 50.2 to 52.8. White women did make up a larger percentage of subgroup 1 as compared with the other subgroups. For several variables, highest mean values were found in the subgroup with the largest fall in standing SBP and the next highest mean values were obtained in the subgroup with the largest rise in stand-
ing SBP. This was true for sitting SBP, sitting DBP, serum cholesterol, and postload glucose. The mean sitting pulse rate was slightly higher in subgroup 4 than in other subgroups, in which it was about the same. (A standing pulse was not obtained in the HDFP.) The mean percent of ideal weight steadily increased from 124.7 to 136.5 moving from subgroup 1 to subgroup 4. Comorbid conditions such as left ventricular hypertrophy, history of stroke, myocardial infarction, and diabetes were more prevalent in participants in subgroup 1, as was an overall index of end-organ damage. (End-organ damage includes one or more of the following: myocardial infarction by history or electrocardiography, major left ventricular hypertrophy by electrocardiography, history or evidence of stroke, intermittent claudication, or baseline creatinine > 2.6.) The mean hematocrit was slightly less in subgroup 1 than the other subgroups. Also, about 40.7% of those participants with a postural change in SBP of -20 mm Hg or less were on antihypertensive medication at baseline, compared with about 24.3% to 27.5% in the other subgroups.

The relation of postural changes in SBP to cumulative all-cause mortality is presented in figure 3. Here the participants in the HDFP are subdivided into deciles of Δ SBP and the corresponding crude mortality rates are noted. The highest mortality rate (9.7 per 100) was in the decile with the greatest drop in standing SBP (Δ SBP < -12 mm Hg).

The crude and age-adjusted mortality rates for the participants in the four previously defined Δ SBP subgroups are presented in table 2. The crude mortality rate of 12.07 per 100 for the participants in subgroup 1 was significantly greater than that for the participants in subgroups 2 (6.83) and 3 (6.35), but not greater than that for subgroup 4 (8.64). However, the crude mortality rate for the participants in subgroup 4, although higher than that for subgroups 2 and 3, was not significantly so.

The age-adjusted mortality rate of 10.21 per 100 for the participants in subgroup 1 was significantly greater than that for the participants in subgroups 2 (6.70) and 3 (6.56). Again, the age-adjusted mortality rate of 7.70 per 100 for the participants in subgroup 4 was not significantly different from that for the other subgroups.

To allow for the possible confounding effects of baseline risk factors, the predictive value of baseline postural changes in SBP was studied by multivariate analysis with a multiple logistic model. In addition to Δ SBP, the baseline risk factors included in this model were age, sex, race, randomization group, antihypertensive medication status, history of diabetes, end-organ damage status, hematocrit, sitting DBP, age × sitting DBP, sitting SBP, sitting pulse, smoking status, and relative weight (percent of ideal weight).

The results of the multiple logistic analysis are presented in table 3. A multiple logistic model that did not include relative weight as a factor showed Δ SBP to be a significant predictor of 5 year total mortality (p < .04) and all the other factors were accepted at the 5% level of significance. A second multiple logistic model that did include relative weight showed that Δ SBP was no longer significant for predicting 5 year total mortal-

![Figure 1](http://circ.ahajournals.org/)

**FIGURE 1.** Distribution of Δ SBPs among 10,536 participants in the HDFP.

![Figure 2](http://circ.ahajournals.org/)

**FIGURE 2.** Prevalence of orthostatic hypotension among the four age groups (30 to 39, 40 to 49, 50 to 59, 60 to 69) in the HDFP.
TABLE 1
Characteristics at entry of participants in the four ΔSBP subgroups

<table>
<thead>
<tr>
<th></th>
<th>Subgroup 1</th>
<th>Subgroup 2</th>
<th>Subgroup 3</th>
<th>Subgroup 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>349</td>
<td>4036</td>
<td>5572</td>
<td>579</td>
</tr>
<tr>
<td>Average age (yr)</td>
<td>53.8</td>
<td>51.0</td>
<td>50.2</td>
<td>52.8</td>
</tr>
<tr>
<td>White men (%)^</td>
<td>27.2</td>
<td>36.6</td>
<td>34.9</td>
<td>23.0</td>
</tr>
<tr>
<td>White women (%)^</td>
<td>30.4</td>
<td>23.4</td>
<td>19.5</td>
<td>18.0</td>
</tr>
<tr>
<td>Black men (%)</td>
<td>16.3</td>
<td>16.2</td>
<td>21.4</td>
<td>27.6</td>
</tr>
<tr>
<td>Black women (%)</td>
<td>26.1</td>
<td>23.9</td>
<td>24.2</td>
<td>31.4</td>
</tr>
<tr>
<td>Sitting SBP (mean mm Hg)</td>
<td>171.3</td>
<td>158.9</td>
<td>156.9</td>
<td>164.4</td>
</tr>
<tr>
<td>Sitting DBP (mean mm Hg)</td>
<td>103.7</td>
<td>101.1</td>
<td>100.8</td>
<td>102.4</td>
</tr>
<tr>
<td>Standing SBP (mean mm Hg)</td>
<td>141.7</td>
<td>148.9</td>
<td>158.3</td>
<td>179.6</td>
</tr>
<tr>
<td>Standing DBP (mean mm Hg)</td>
<td>97.7</td>
<td>102.5</td>
<td>106.4</td>
<td>113.1</td>
</tr>
<tr>
<td>Sitting pulse rate (mean beats/min)</td>
<td>81.9</td>
<td>81.9</td>
<td>81.6</td>
<td>82.7</td>
</tr>
<tr>
<td>Serum cholesterol (mean mg/dl)</td>
<td>239.2</td>
<td>236.1</td>
<td>234.3</td>
<td>237.8</td>
</tr>
<tr>
<td>Smoking more than 10 cigarettes per day (%)</td>
<td>24.4</td>
<td>25.2</td>
<td>26.0</td>
<td>24.5</td>
</tr>
<tr>
<td>Percent of ideal weight (mean)</td>
<td>124.7</td>
<td>127.1</td>
<td>129.0</td>
<td>136.5</td>
</tr>
<tr>
<td>Serum creatinine (mean mg/dl)</td>
<td>1.19</td>
<td>1.08</td>
<td>1.07</td>
<td>1.07</td>
</tr>
<tr>
<td>Plasma glucose (1 hr post-load; mean mg/dl)</td>
<td>187.8</td>
<td>179.9</td>
<td>177.1</td>
<td>183.3</td>
</tr>
<tr>
<td>Hematocrit (mean %)</td>
<td>42.6</td>
<td>43.4</td>
<td>43.7</td>
<td>43.2</td>
</tr>
<tr>
<td>Left ventricular hypertrophy on electrocardiogram (%)^</td>
<td>8.7</td>
<td>4.9</td>
<td>4.6</td>
<td>5.8</td>
</tr>
<tr>
<td>History of stroke (%)</td>
<td>5.4</td>
<td>2.4</td>
<td>2.3</td>
<td>3.1</td>
</tr>
<tr>
<td>History of myocardial infarction (%)</td>
<td>6.3</td>
<td>4.8</td>
<td>5.3</td>
<td>4.7</td>
</tr>
<tr>
<td>History of diabetes (%)</td>
<td>13.2</td>
<td>7.2</td>
<td>6.4</td>
<td>7.9</td>
</tr>
<tr>
<td>Receiving antihypertensive medication (%)</td>
<td>40.7</td>
<td>27.5</td>
<td>24.3</td>
<td>26.1</td>
</tr>
<tr>
<td>End-organ damage^</td>
<td>26.4</td>
<td>14.2</td>
<td>14.0</td>
<td>15.4</td>
</tr>
</tbody>
</table>

\^ Includes less than 1\% other, e.g., Asians.
\^ Based on combined R wave and ST-T segment changes: tail R wave (Minnesota code 3.1) and major ST segment depression (Minnesota code 4.1-4.3) or major T wave inversion (Minnesota code 5.1-5.3).
\^ End-organ damage includes one or more of: myocardial infarction by history or electrocardiography, major left ventricular hypertrophy by electrocardiography, history of evidence of stroke, intermittent claudication, or baseline creatinine >2.6.

ity (p = .08). Hematocrit was also no longer significant (p = .11). Thus, it appears that relative weight has an unexpected strong relationship to both ΔSBP and mortality; i.e., it is a confounding factor. As noted in Table 2, as ΔSBP increased from negative to positive the mean percent ideal weight tended to increase.

Further studies with the multiple logistic model that included relative weight addressed the question of possible effects of interaction of various factors with ΔSBP on the outcome of 5 year all-cause mortality. There was no significant indication that the risk of ΔSBP depended on whether a participant was on treatment at entry or on the level of sitting DBP or sitting SBP.

The only factor shown to significantly interact with ΔSBP with respect to 5 year total mortality was a history of diabetes (p < .01). The effect of this intervention on participants with ΔSBPs of -20 mm Hg was examined, although the model accounts for the influence of diabetes in all the subgroups since ΔSBP is a continuous variable. The results of this examination are presented in Table 4. The logistic model with interaction indicated an approximate 2.3 times increase in 5 year total mortality risk for persons with diabetes and a 20 mm Hg fall in SBP on standing. In comparison, the model without interaction indicated a 1.6 times increase in risk. Thus, the presence of both diabetes and a 20 mm Hg fall in SBP on standing resulted in a 5 year total mortality risk greater than the combined multiplicative risk of the two factors.

Finally, we considered whether ΔDBP (standing minus sitting DBP) was predictive of mortality. In the multiple logistic model that included relative weight, ΔSBP was replaced with ΔDBP. Here ΔDBP was not a significant predictor of 5 year total mortality. We focused on ΔSBP rather than ΔDBP because the former is a common measurement that has been examined and written about extensively.

**Discussion**

Elevated blood pressure has been shown to be a significant risk factor for all-cause mortality in the HDFC. We examined the risk associated with various levels of change in SBP in participants standing from a
sitting position. The findings suggest that the postural change in SBP is important in risk assessment; Δ SBP is a useful predictor of mortality within this population-based sample of hypertensives recruited by HDFP criteria. The influence of Δ SBP remained even when other baseline risk factors were included in a multivariate analysis and even when the effect of sitting SBP was already incorporated. Its influence diminished when relative weight was considered.

There was no evidence of any interaction between sitting blood pressures (DBP or SBP) and Δ SBP. This is consistent with the one previously reported analysis in which the risk of myocardial infarction in relation to postural changes in DBP was examined. However, there were several differences between that study sample (normotensive, male, white) and the HDFP sample (hypertensive, male and female, black and white).

There is no standard criteria for measurement of postural changes in blood pressure. Almost all studies have begun with the patients supine. However, the amount of time remaining supine was either not given or varied from 1 to 3 min. Studies also differed in how the measurements of change were obtained. Some participants were tilted, some sat for a while and then stood, and others stood for a time period ranging from 1 min to 1 hr. The groups studied also varied. Many were elderly and some were hospitalized; a few studies included young volunteers, and some included people with both hypertension (as defined by sitting criterion) and orthostatic hypotension.

At least two possible mechanisms exist to explain an exaggerated decrease in SBP with the assumption of the upright posture. Orthostatic hypotension has been classified as either primary (idiopathic) or secondary. Primary orthostatic hypotension is that not due to a known associated disorder. Secondary orthostatic hypotension is caused by a variety of factors. These include metabolic endocrinologic disorders such as diabetes and adrenal insufficiency; nervous system disorders such as tremors and cerebral infarcts; and miscellaneous disorders such as hypovolemia, anemia, and the use of psychotropic and antihypertensive drugs.

In the HDFP, the subgroup of participants with an orthostatic drop in SBP of 20 mm Hg or more had a higher prevalence of diabetes, history of stroke, and use of antihypertensive medications than those in the other subgroups. One or all of these factors could re-

<table>
<thead>
<tr>
<th>ΔSBP (mm Hg)</th>
<th>CRUDE MORTALITY RATE (PER 100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; -12</td>
<td>10.21</td>
</tr>
<tr>
<td>-12 to -6</td>
<td>6.86</td>
</tr>
<tr>
<td>-6 to -3</td>
<td>5.22</td>
</tr>
<tr>
<td>-3 to 0</td>
<td>7.16</td>
</tr>
<tr>
<td>1 to 2</td>
<td>7.64</td>
</tr>
<tr>
<td>2 to 5</td>
<td>6.66</td>
</tr>
<tr>
<td>5 to 8</td>
<td>6.25</td>
</tr>
<tr>
<td>8 to 12</td>
<td>5.98</td>
</tr>
<tr>
<td>12 to 17</td>
<td>6.16</td>
</tr>
<tr>
<td>&gt; 17</td>
<td>6.70</td>
</tr>
</tbody>
</table>

**FIGURE 3.** Life table 5 year mortality rate (per 100) by decile of Δ SBP.

**TABLE 2**

<table>
<thead>
<tr>
<th>Subgroups</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>349</td>
<td>4036</td>
<td>5572</td>
<td>579</td>
</tr>
<tr>
<td>Deaths</td>
<td>42</td>
<td>275</td>
<td>353</td>
<td>50</td>
</tr>
<tr>
<td>Rate</td>
<td>12.07</td>
<td>6.83</td>
<td>6.35</td>
<td>8.64^</td>
</tr>
<tr>
<td>Age-adjusted rate</td>
<td>10.21</td>
<td>6.70</td>
<td>6.56</td>
<td>7.70^</td>
</tr>
</tbody>
</table>

^Adjusted by age, using total HDFP population as the standard.
^Rate significantly different at p < .005.
^Rate significantly different at p < .04.
result in an increased mortality risk. However, in the logistic model (without relative weight), these factors were controlled for and \( \Delta \text{SBP} \) remained a significant prognostic factor. Only when relative weight was added did \( \Delta \text{SBP} \) lose its significance.

The further studies with interaction models suggested that \( \Delta \text{SBP} \) had a significant positive interaction with diabetes with respect to mortality outcome, even when calculations were controlled for relative weight. Angell-James et al.\(^{29} \) have shown that the baroreceptors of hypertensive rabbits are not as responsive as those of normal rabbits and that decreased arterial wall compliance is the mechanism of decreased baroreceptor responsiveness in arteriosclerosis and in hypertension. Therefore, it seems likely that postural hypotension is a manifestation of arterial vascular disease. A comparatively more negative \( \Delta \text{SBP} \) in participants with diabetes may be indicative of arteriosclerosis or a more severe case of diabetes. Either might increase the mortality risk.

Participants without diabetes may have some disorder resulting in secondary orthostatic hypotension. If this disorder is associated with low relative weight and an increased mortality risk it would explain why relative weight was a confounding factor. (Diabetes is a disorder associated with a high relative weight.) A previous analysis of the HDFP data has shown that there is an inverse relationship between relative weight

\[\begin{array}{|c|c|c|c|}
\hline
\text{Factor} & \text{Ratio} & \text{Without relative weight} & \text{With relative weight} \\
\hline
\text{Age} & \text{Continuous, per year increase} & 1.17^c & 1.18^c \\
\text{Sex} & \text{M/F} & 1.70^c & 1.55^c \\
\text{Race} & \text{W/B} & 0.65^c & 0.64^c \\
\text{Randomization} & \text{SC/RC} & 0.81^c & 0.81^c \\
\text{Treatment} & \text{Yes/no} & 1.25^c & 1.28^c \\
\text{Diabetes} & \text{Yes/no} & 1.34^c & 1.43^c \\
\text{End-organ damage} & \text{Yes/no} & 2.27^c & 2.29^c \\
\text{Hematocrit} & \text{Continuous, \%} & 0.98^c & 0.98^d \\
\text{Sitting DBP} & \text{Continuous, per mm Hg increase} & 1.05^c & 1.05^c \\
\text{Sitting SBP} & \text{Continuous, per mm Hg increase} & 1.01^c & 1.01^c \\
\text{Sitting pulse} & \text{Continuous, beats/min} & 1.01^c & 1.01^c \\
\text{Age \times sitting DBP} & \text{Continuous, per 10 years \times mm Hg increase} & 0.99^c & 0.99^c \\
\text{Smoking} & \text{Yes/no} & 1.72^c & 1.61^c \\
\text{Relative weight} & \text{Continuous, per \% increase over ideal weight} & — & 0.99^c \\
\text{\( \Delta \text{SBP}^b \)} & \text{Continuous, per mm Hg decrease} & 1.02^c & 1.01^e \\
\hline
\end{array}\]

\(^a\)Data from 9890 participants were used in the logistic analysis because some information for all the selected variables was missing for 646 participants.

\(^b\)\(\Delta \text{SBP} \) was mean centered. (The mean of the \( \Delta \text{SBP} \) variable was subtracted from the \( \Delta \text{SBP} \) value for all participants.)

\(^c\)\(p < .05\); \(^d\)\(p = .11\); \(^e\)\(p = .08\).

\[\begin{array}{|c|c|c|c|}
\hline
\text{\( \Delta \text{SBP} \)} & \text{No-interaction model} & \text{Interaction model} \\
(\text{mm Hg}) & \text{No diabetes} & \text{Diabetes} & \text{No diabetes} & \text{Diabetes} \\
\hline
0 & 1.00^b & 1.54 (1.09, 1.89) & 1.00^b & 1.30 (0.97, 1.75) \\
-20 & 1.12 (0.99, 1.28) & 1.61 (1.19, 2.17) & 1.05 (0.92, 1.21) & 2.28 (1.27, 4.11) \\
\hline
\end{array}\]

Values in parentheses are 95% confidence intervals.

\(^a\)Adjusted for age, sex, race, prior antihypertensive treatment, randomization group, diabetes, end-organ damage, hematocrit, sitting DBP and SBP, sitting pulse, smoking status, and relative weight.

\(^b\)Reference group.
and mortality. Nonobese hypertensive individuals have a poorer prognosis than obese ones. It has been hypothesized that the cause or causes of hypertension in obese persons may differ from those in nonobese persons. If the cause(s) of hypertension in nonobese persons also results in a more negative Δ SBP, this would explain the confounding effect of relative weight.

The results of the present study were obtained in persons with diastolic hypertension (DBP ≥ 90 mm Hg) at baseline; thus the implications of these findings for persons who are normotensive are not known. Also, the HDFP was not designed to determine whether mortality is different for treated and nontreated orthostatic hypertensive individuals.

Postural change in blood pressure is an easily obtainable measurement that may have clinical significance in both normotensive and hypertensive individuals. Such a measurement deserves further study as a potential risk factor.

References
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