Postural changes in diastolic blood pressure and the risk of myocardial infarction: The Normative Aging Study

DAVID SPARROW, M.S., CHARLES P. TIFFT, M.D., BERNARD ROSNER, PH.D., and SCOTT T. WEISS, M.D.

ABSTRACT To assess the relationship of postural changes in blood pressure to risk of myocardial infarction, 1359 men were followed for an average of 8.7 years. The men were participants in the Normative Aging Study, a longitudinal study of aging initiated in 1963 at the Veterans Administration Outpatient Clinic in Boston. It was found that the relationship of sitting blood pressure to the subsequent incidence of myocardial infarction was modified by a variable formed by subtracting supine from standing diastolic blood pressure (ΔDBP). The effect of sitting diastolic blood pressure on risk of myocardial infarction was confined primarily to men with a ΔDBP of 10 mm Hg or more. The effect of sitting systolic blood pressure on risk of myocardial infarction was apparent in all categories of ΔDBP (<1, 1 to 9, ≥10 mm Hg), but the gradient of risk became stronger with increasing levels of ΔDBP. The modifying influence of ΔDBP remained even when standard coronary risk factors were included in multivariate analyses. These findings suggest a relationship of vascular responsiveness to risk of subsequent myocardial infarction and may have clinical utility.

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BLOOD PRESSURE has been identified as an important risk factor for coronary heart disease in epidemiologic studies, which have demonstrated that the higher the level of systolic blood pressure (SBP) or diastolic blood pressure (DBP), the greater the risk of subsequent coronary heart disease. With the growing recognition of blood pressure as a major risk factor, there has been increasing emphasis on detecting and treating individuals with elevated pressures. Yet there is considerable controversy regarding the exact level of blood pressure that warrants drug treatment. Furthermore, there is concern about using only casual measurements of blood pressure taken in the physician’s office as a basis for treatment decisions. There may be other considerations in assessing a person’s blood pressure status.

Although epidemiologic data have shown that casual measurements of blood pressure are predictive of subsequent coronary heart disease, predictability may be improved by considering pressure responses to certain stressful stimuli encountered during the day. One standardized stress stimulus is postural change, particularly the assumption of the standing position. The purpose of this study was to provide initial data concerning the relationship between postural changes in blood pressure and the incidence of myocardial infarction in a large cohort of men.

Methods

The population for this study was 2144 white men 21 to 80 years of age at entry to the Normative Aging Study. The Normative Aging Study is a longitudinal study of aging initiated in 1963 and located at the Veterans Administration Outpatient Clinic in Boston. Volunteers were screened at entry according to health criteria and were free of known chronic medical conditions at the outset. In particular, individuals with an SBP greater than 140 mm Hg or DBP greater than 90 mm Hg were excluded. The study protocol was approved by the Human Studies Committee of the Veterans Administration Outpatient Clinic and informed consent was obtained from all subjects.

Multiple positional blood pressure readings were first taken during the second cycle of data collection (1969 to 1974), hereafter referred to as the baseline examination for this investigation. (All subjects were normotensive in 1963 to 1968 when entered into the original cohort.) Participants reported to the study center at 8:00 a.m. after an overnight fast and abstinence from smoking. A complete history was conducted by the physi-
cian with the subject seated. Immediately thereafter a comprehensive physical examination was begun with the subject still seated. SBP and fifth-phase DBP were measured to the nearest 2 mm Hg in the left arm, then in the right arm. Heart rate was also measured. The subject then sat on the examination table for examination of head, neck, and lungs. Height and weight were measured and the subject was placed in a supine position for cardiac, abdominal, neurologic, and peripheral pulse examination. A blood pressure reading in the right arm and heart rate were then taken again with the subject in this position. The subject was assisted to a standing position, and approximately 30 sec later a blood pressure reading in the right arm and heart rate were taken for a third time. Hernia and rectal examinations were then performed.

In addition to the physical examination, an electrocardiographic examination and a variety of laboratory tests were performed. Total serum cholesterol was measured with the colorimetric method of Sperry.6 Smoking history was recorded and the current number of cigarettes smoked per day was noted. Weight and height were measured with the subjects wearing only stockings and undershorts.

Similar examinations were repeated every 3 to 5 years. The data were supplemented by information on cardiovascular illness obtained from hospital records.

Criteria for the diagnosis of myocardial infarction were those used in the Framingham Heart Study.7 The records of all cases suggestive of myocardial infarction were reviewed by a cardiologist (H. Emerson Thomas, Jr., Boston University Medical Center). Myocardial infarction was diagnosed only when documented by the occurrence of unequivocal electrocardiographic changes (i.e., pathologic Q waves), by a diagnostic elevation of serum enzyme levels (serum glutamic oxalacetic transaminase, lactic dehydrogenase) accompanying chest discomfort consistent with myocardial infarction, or postmortem examination.

The subjects for the current study were a subgroup of the Normative Aging Study population. These 1359 subjects underwent at least one examination after the baseline examination or a subsequent diagnosis of myocardial infarction. Those excluded were 37 men who had either a history of myocardial infarction or who were given antihypertensive medication at baseline, an additional 628 who had incomplete baseline information on one or more of the independent variables of interest, and 120 additional men who did not undergo a follow-up examination. Subjects excluded because of missing follow-up examinations and those excluded because of lacking baseline information were comparable to the subjects who participated with respect to baseline age, serum cholesterol levels, blood pressure, and body mass index, but excluded subjects were more likely to be current smokers (table 1). The subjects excluded because of missing baseline information had a similar occurrence of myocardial infarction (1.8%) as included subjects (2.5%).

The analyses presented here relate the development of myocardial infarction to baseline variables by Cox’s life table regression method, which assumes a proportional-hazards model.8 A total of 11 baseline variables were considered, including age, body mass index (weight/height2), serum cholesterol level, number of cigarettes smoked per day, sitting heart rate, sitting SBP, sitting DBP, standing DBP, supine DBP, standing minus supine DBP (ΔDBP), and the interaction between ΔDBP and sitting DBP. The latter interaction variable was shown by preliminary analysis to be of substantive importance. The ΔDBP component of this interaction variable was mean-centered (i.e., the overall mean ΔDBP was subtracted from each subject’s ΔDBP). This was done to make the interaction variable correlate less precisely with sitting DBP.

Calculations for Cox’s method were performed with a step-up procedure by means of the PHGLM program of the Statistical Analysis System.9 The sitting DBP was forced into the model first and then other variables were entered in a stepwise fashion with the variable entered on a given step that most improved the prediction of myocardial infarction. The stepwise process ended when no variables improved the model at a significance level of p = .05. The Cox procedure allows for the differing length of follow-up of the subjects. These subjects underwent their baseline examinations between 1969 and 1974 and remained under study until their last examination, the occurrence of myocardial infarction, or the end of observation (December 31, 1982). The mean length of follow-up was 8.7 years. A total of 11,829 man-years of follow-up was compiled for the 1359 study subjects. A similar analysis was performed forcing sitting DBP into the model first and then considering the other variables mentioned above.

To assess whether ΔDBP was related to standard coronary risk factors, one-way analyses of variance and covariance were performed with the BMDP2V program.10 Analysis of variance was done with age as a dependent variable and ΔDBP as the grouping variable. Separate analyses of covariance were done with each of the other coronary risk factors (sitting SBP, sitting DBP, serum cholesterol level, number of cigarettes smoked per day, body mass index, and sitting heart rate) as dependent variables, ΔDBP as the grouping variable, and age as the covariate.

Results

Of the 1359 men, 34 developed myocardial infarction over the follow-up period. The incidence of myocardial infarction increased with increasing baseline blood pressure. For sitting DBP, incidence rates went from 2.4 (per 1000 man-years) for those with DBP under 80 mm Hg to 2.8 for those with DBP from 80 to 89 mm Hg to 6.8 for those with DBP of 90 mm Hg or greater. For sitting SBP, incidence rates went from 1.8 (per 1000 man-years) for men with SBP under 130 mm Hg to 3.8 for men with SBP from 130 to 139 mm Hg to 6.5 for men with SBP from 140 to 149 mm Hg to 10.9 for men with SBP of 150 mm Hg or greater.

After adjustment for age, stratified analyses suggested that the relationship of both sitting DBP and sitting SBP to the subsequent incidence of myocardial infarction was modified by the ΔDBP. The effect of

TABLE 1
Baseline characteristics for participants in the Normative Aging Study (mean ± SD)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Included subjects</th>
<th>Subjects missing baseline data</th>
<th>Subjects missing follow-up exam</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>47.3 ± 8.7</td>
<td>46.1 ± 9.0</td>
<td>46.7 ± 10.1</td>
</tr>
<tr>
<td>Serum cholesterol (mg/dl)</td>
<td>222.8 ± 47.6</td>
<td>221.6 ± 48.2</td>
<td>221.6 ± 47.9</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>122.6 ± 13.9</td>
<td>123.8 ± 15.6</td>
<td>123.0 ± 16.3</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>76.2 ± 8.7</td>
<td>76.6 ± 9.8</td>
<td>75.6 ± 9.5</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>25.8 ± 2.8</td>
<td>25.6 ± 3.0</td>
<td>25.5 ± 2.6</td>
</tr>
<tr>
<td>Cigarette smokers (%)</td>
<td>29.7</td>
<td>42.4</td>
<td>45.8</td>
</tr>
</tbody>
</table>

Analysis System.9 The sitting DBP was forced into the model first and then other variables were entered in a stepwise fashion with the variable entered on a given step that most improved the prediction of myocardial infarction. The stepwise process ended when no variables improved the model at a significance level of p = .05. The Cox procedure allows for the differing length of follow-up of the subjects. These subjects underwent their baseline examinations between 1969 and 1974 and remained under study until their last examination, the occurrence of myocardial infarction, or the end of observation (December 31, 1982). The mean length of follow-up was 8.7 years. A total of 11,829 man-years of follow-up was compiled for the 1359 study subjects. A similar analysis was performed forcing sitting DBP into the model first and then considering the other variables mentioned above.

To assess whether ΔDBP was related to standard coronary risk factors, one-way analyses of variance and covariance were performed with the BMDP2V program.10 Analysis of variance was done with age as a dependent variable and ΔDBP as the grouping variable. Separate analyses of covariance were done with each of the other coronary risk factors (sitting SBP, sitting DBP, serum cholesterol level, number of cigarettes smoked per day, body mass index, and sitting heart rate) as dependent variables, ΔDBP as the grouping variable, and age as the covariate.

Results

Of the 1359 men, 34 developed myocardial infarction over the follow-up period. The incidence of myocardial infarction increased with increasing baseline blood pressure. For sitting DBP, incidence rates went from 2.4 (per 1000 man-years) for those with DBP under 80 mm Hg to 2.8 for those with DBP from 80 to 89 mm Hg to 6.8 for those with DBP of 90 mm Hg or greater. For sitting SBP, incidence rates went from 1.8 (per 1000 man-years) for men with SBP under 130 mm Hg to 3.8 for men with SBP from 130 to 139 mm Hg to 6.5 for men with SBP from 140 to 149 mm Hg to 10.9 for men with SBP of 150 mm Hg or greater.

After adjustment for age, stratified analyses suggested that the relationship of both sitting DBP and sitting SBP to the subsequent incidence of myocardial infarction was modified by the ΔDBP. The effect of
sitting DBP on risk was confined primarily to men with a ΔDBP of 10 mm Hg or more (table 2, top). In this group, incidence rates rose sharply with each succeeding level of sitting DBP, reaching 25.2 (per 1000 man-years) in those with a sitting DBP of 90 mm Hg or more. The effect of sitting SBP on risk of myocardial infarction was primarily apparent in men who showed a positive ΔDBP, and the gradient of risk became stronger as ΔDBP increased (table 2, bottom). In contrast, there was no evidence that the relationship of sitting blood pressure (DBP or SBP) to the subsequent incidence of myocardial infarction was modified by standing minus supine SBP. Thus this relationship was not examined further with multivariate techniques.

We considered whether ΔDBP was related to standard coronary risk factors (table 3). Significant differences in coronary risk factors between categories of ΔDBP were observed for age (p < .001, analysis of variance) and sitting DBP (p = .015, analysis of covariance). Men with the highest ΔDBP (≥10 mm Hg) had the youngest mean age and the lowest age-adjusted mean sitting ΔDBP, but no significant association was found with the other coronary risk factors.

Cox regression was used to assess the relationship of ΔDBP to risk of myocardial infarction after controlling for sitting DBP and other baseline factors. After forcing sitting DBP into the model first, 10 factors were considered and four (serum cholesterol, interaction between sitting DBP and ΔDBP, age, and log of number of cigarettes) were accepted at the 5% level of significance (table 4). The interaction variable was a highly significant (p = .001) independent predictor, indicating that the effect of sitting DBP on myocardial infarction depended on the level of ΔDBP. This is consistent with the stratified results shown in table 2.

When sitting SBP was forced into the model first instead of sitting DBP, the same four additional factors (serum cholesterol, interaction between sitting SBP and ΔDBP, log of number of cigarettes, and age) were accepted at the 5% level of significance (table 5). The

### TABLE 2
Age-adjusted incidence of myocardial infarction in 1359 men according to baseline data (1969 to 1974)

<table>
<thead>
<tr>
<th>Sitting DBP (mm Hg)</th>
<th>ΔDBP (mm Hg)</th>
<th>No. of men</th>
<th>Man-yr (×10³)</th>
<th>No. of MIs</th>
<th>1000 man-yr</th>
<th>No. of men</th>
<th>Man-yr (×10³)</th>
<th>No. of MIs</th>
<th>1000 man-yr</th>
<th>No. of men</th>
<th>Man-yr (×10³)</th>
<th>No. of MIs</th>
<th>1000 man-yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;80</td>
<td>&lt;1</td>
<td>347</td>
<td>3.1</td>
<td>6</td>
<td>2.0</td>
<td>264</td>
<td>2.3</td>
<td>9</td>
<td>3.8</td>
<td>187</td>
<td>1.7</td>
<td>2</td>
<td>4.2</td>
</tr>
<tr>
<td>80–89</td>
<td>1–9</td>
<td>217</td>
<td>1.9</td>
<td>2</td>
<td>1.0</td>
<td>161</td>
<td>1.4</td>
<td>3</td>
<td>2.2</td>
<td>80</td>
<td>0.7</td>
<td>6</td>
<td>9.6</td>
</tr>
<tr>
<td>≥90</td>
<td>≥10</td>
<td>49</td>
<td>0.4</td>
<td>1</td>
<td>1.5</td>
<td>39</td>
<td>0.3</td>
<td>2</td>
<td>5.5</td>
<td>15</td>
<td>0.1</td>
<td>3</td>
<td>25.2</td>
</tr>
<tr>
<td>&lt;130</td>
<td></td>
<td>436</td>
<td>3.8</td>
<td>5</td>
<td>1.3</td>
<td>309</td>
<td>2.7</td>
<td>7</td>
<td>2.9</td>
<td>205</td>
<td>1.8</td>
<td>3</td>
<td>1.6</td>
</tr>
<tr>
<td>130–139</td>
<td></td>
<td>108</td>
<td>0.9</td>
<td>3</td>
<td>2.9</td>
<td>96</td>
<td>0.8</td>
<td>2</td>
<td>2.3</td>
<td>45</td>
<td>0.4</td>
<td>3</td>
<td>8.9</td>
</tr>
<tr>
<td>140–149</td>
<td></td>
<td>45</td>
<td>0.4</td>
<td>0</td>
<td>—</td>
<td>35</td>
<td>0.3</td>
<td>3</td>
<td>5.6</td>
<td>24</td>
<td>0.2</td>
<td>3</td>
<td>9.4</td>
</tr>
<tr>
<td>≥150</td>
<td></td>
<td>24</td>
<td>0.2</td>
<td>1</td>
<td>2.5</td>
<td>24</td>
<td>0.2</td>
<td>2</td>
<td>11.1</td>
<td>8</td>
<td>0.07</td>
<td>2</td>
<td>18.4</td>
</tr>
</tbody>
</table>

*Adjusted with the direct method by 10 year age classes, taking the cohort of 1359 men as standard.

### TABLE 3
Age-adjusted values for coronary risk factors (mean ± SD) in 1359 men according to ΔDBP category

<table>
<thead>
<tr>
<th>ΔDBP (mm Hg)</th>
<th>n</th>
<th>Age (yr)</th>
<th>Sitting SBP (mm Hg)</th>
<th>Sitting DBP (mm Hg)</th>
<th>Total serum cholesterol (mg/dl)</th>
<th>Log (1 + cigs./day)</th>
<th>Body mass index (kg/m²)</th>
<th>Sitting heart rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1</td>
<td>613</td>
<td>48.4 ± 9.0</td>
<td>121.8 ± 13.7</td>
<td>76.4 ± 8.8</td>
<td>221.4 ± 45.5</td>
<td>0.41 ± 0.62</td>
<td>25.7 ± 2.8</td>
<td>74.0 ± 9.0</td>
</tr>
<tr>
<td>1–9</td>
<td>464</td>
<td>47.2 ± 8.3</td>
<td>123.5 ± 14.4</td>
<td>76.8 ± 9.0</td>
<td>225.2 ± 51.2</td>
<td>0.42 ± 0.65</td>
<td>25.9 ± 2.7</td>
<td>74.2 ± 9.2</td>
</tr>
<tr>
<td>≥10</td>
<td>282</td>
<td>45.0 ± 8.2</td>
<td>122.7 ± 13.4</td>
<td>74.9 ± 8.2</td>
<td>221.6 ± 46.0</td>
<td>0.32 ± 0.62</td>
<td>25.8 ± 2.7</td>
<td>74.4 ± 10.0</td>
</tr>
<tr>
<td>p values</td>
<td>&lt;.001</td>
<td>.001</td>
<td>.015</td>
<td>.379</td>
<td>.095</td>
<td>.359</td>
<td>.809</td>
<td></td>
</tr>
</tbody>
</table>

*Means for age are unadjusted.

* Differences between ΔDBP categories were tested for equality of group means by analysis of covariance (equality of mean ages was tested by analysis of variance).
interaction variable was a highly significant (p = .004) predictor, indicating that the effect of sitting SBP on myocardial infarction depended on the level of ΔDBP.

Further analysis revealed that with increasing levels of ΔDBP (<1, 1 to 9, ≥10 mm Hg), there was a corresponding increase in the standing minus supine heart rate (5.6, 7.6, and 8.5 beats/min; p < .001, analysis of variance). Consequently, the above regressions were repeated, forcing the standing minus supine heart rate into the models along with sitting blood pressure and considering the same risk factors as in tables 4 and 5. Standing minus supine heart rate was not a statistically significant predictor of myocardial infarction, and its inclusion in either model did not materially affect the regression coefficient of the interaction variable or the coefficients of the other factors.

Discussion

Blood pressure has been shown to be an important risk factor for myocardial infarction in several longitudinal studies such as the Framingham Study and Pooling Project Study. The present study is the first to indicate that risk associated with a given level of DBP or SBP is modified by ΔDBP. The data suggest that both sustained elevation of blood pressure and periodic "bursts" of DBP elevation are important in risk assessment. The modifying influence of ΔDBP remained even when standard coronary risk factors were included in a multivariate analysis.

The cardiovascular changes associated with the stress of standing are well known. The assumption of the upright position causes the pooling of blood in the venous system. This pooling is associated with a decrease in venous return and subsequent decrease in cardiac output, which causes a transient drop in systemic blood pressure. The drop in blood pressure results in baroreceptor relaxation and sympathetic stimulation, which causes venoconstriction and cardioacceleration, serving to stabilize cardiac output at a level lower than in the supine position. Total peripheral resistance increases sufficiently to maintain mean blood pressure in the face of reduced cardiac output. In general, diastolic blood pressure remains the same or is elevated slightly and systolic blood pressure is slightly reduced in the standing position relative to the supine position.

At least two possibilities exist to explain an exaggerated increase in DBP with the assumption of the upright posture. It is well known that sympathetic nervous system activity mediates the initial defense of arterial pressure. Plasma catecholamine levels measured in standing subjects are generally nearly double the basal levels in subjects in the supine position, and thus excessive sympathetic nervous system activity may be playing a role in the observed postural response. In further support of this hypothesis, Sapru et al. have used β-adrenergic blockade to abolish this response in one subject. Alternatively, changes in vascular volume may be responsible for this increased adrenergic response. Streten et al. have reported on 181 of 1800 subjects with supine DBP less than 90 mm Hg who had standing DBP greater than 90 mm Hg. The results of their studies suggest an excessive orthostatic pooling, which caused greater-than-control decreases in cardiac output; this might explain the excessive reflex arteriolar constriction in these subjects.

The subjects in this study who showed exaggerated increases in DBP on standing may have had a physiologic response compatible with excessive adrenergic activity or excessive sensitivity to adrenergic stimulation. Basal sitting or supine blood pressure determinations may be an incomplete reflection of the individual's risk of cardiovascular disease, and further studies including ambulatory blood pressure monitoring may

TABLE 4
Cox regression analysis of incidence of myocardial infarction (34 cases) among 1359 men in relation to independent variables that entered significantly (5% level) at succeeding steps

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Beta</th>
<th>SE</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting DBP</td>
<td>.0369</td>
<td>.0195</td>
<td>.050</td>
</tr>
<tr>
<td>Serum cholesterol</td>
<td>.0090</td>
<td>.0032</td>
<td>.005</td>
</tr>
<tr>
<td>(Sitting DBP) (ΔDBP)</td>
<td>.00089</td>
<td>.00028</td>
<td>.001</td>
</tr>
<tr>
<td>Age</td>
<td>.0611</td>
<td>.0202</td>
<td>.002</td>
</tr>
<tr>
<td>Log (1 + cigs./day)</td>
<td>.7033</td>
<td>.2606</td>
<td>.007</td>
</tr>
</tbody>
</table>

*Sitting DBP was forced into equation first.

ΔDBP component of this interaction variable was mean centered (i.e., the overall mean ΔDBP [3.0 mm Hg] was subtracted from each subject’s ΔDBP).

TABLE 5
Cox regression analysis of incidence of myocardial infarction (34 cases) among 1359 men in relation to independent variables that entered significantly (5% level) at succeeding steps

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Beta</th>
<th>SE</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting SBP</td>
<td>.0266</td>
<td>.0107</td>
<td>.013</td>
</tr>
<tr>
<td>Serum cholesterol</td>
<td>.0095</td>
<td>.0031</td>
<td>.002</td>
</tr>
<tr>
<td>(Sitting SBP) (ΔDBP)</td>
<td>.00049</td>
<td>.00017</td>
<td>.004</td>
</tr>
<tr>
<td>Log (1 + cigs./day)</td>
<td>.6664</td>
<td>.2600</td>
<td>.010</td>
</tr>
<tr>
<td>Age</td>
<td>.0448</td>
<td>.0200</td>
<td>.025</td>
</tr>
</tbody>
</table>

*Sitting SBP was forced into equation first.
show significantly higher readings throughout the day in subjects with isolated upright hypertension.

Certain aspects of our analysis deserve emphasis. Our results were obtained in a male population that was normotensive at the prebaseline examination (first cycle); thus, the implications of these findings for women and persons with preexisting hypertension are unknown. The uniqueness of our observations and the above-mentioned constraints on generalizing them suggest the need for confirmation of our results in other populations. Finally, blood pressure measurements in individuals are known to have significant between-visit variability. Our blood pressure measurements were obtained at a single visit and lack the precision obtained by averaging over several sittings. This lack of precision (large variability) would tend to obscure any relationship between postural blood pressure changes and risk of myocardial infarction; indeed, a more precise measure might reveal a stronger association than that observed in our data.

Our findings may have relevance in identifying which patients may benefit from therapy for hypertension. Data from recent clinical intervention trials have resulted in controversy regarding the level of blood pressure that requires treatment with antihypertensive agents. The benefits of treating all patients with mild hypertension remains to be conclusively established, and thus the clinician needs to be able to select those who are at higher risk of subsequent heart disease for consideration of earlier treatment. The postural change in DBP deserves further study as a potential risk factor that could be easily measured in clinical practice.

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