The response of blood pressure to change in body position has been used in epidemiological studies as a measure of cardiovascular reactivity. Sparrow et al reported that a 10-mm Hg or greater increase in diastolic blood pressure (DBP) from the supine to standing position significantly modified the effect of seated systolic blood pressure (SBP) and DBP on the incidence of myocardial infarction during 8.7 years of follow up in a cohort of middle-aged white men. On further investigation of this population, the investigators concluded that the difference between the supine and seated blood pressures is positively associated with subsequent development of systemic hypertension independent of supine blood pressure.

The response of blood pressure to change in body position is well suited as a measure of cardiovascular reactivity for epidemiological studies. Several experimental studies have suggested a differential response of blood pressure to standing due to ethnicity and gender. However, other studies found no gender differences. Little is known about the descriptive epidemiology of this measure of reactivity, especially among women and blacks. The purpose of this investigation was to focus on the descriptive epidemiology of the response of blood pressure to change in posture in a biracial, population-based sample, the Atherosclerosis Risk in Communities (ARIC) Study.

The ARIC Study

Key Words: blood pressure ■ cardiovascular reactivity ■ epidemiology

The response of blood pressure to change in body position, described in a cohort of 13,340 men and women aged 45 to 65 years enrolled in the Atherosclerosis Risk in Communities (ARIC) Study. The distribution of ΔSBP was found to be symmetrical and unimodal, with a mean value near zero (±0.45 mm Hg). The range of ΔSBP was from −63.2 to 54.3 mm Hg, and the standard deviation was 10.8. Stratification of ΔSBP by race and gender shows a slight shift in distribution toward higher values for black men and women. ΔSBP was categorized into deciles. Participants in the top 30% and bottom 30% of the distribution were compared with individuals in the middle 40% of the distribution, who had little or no change in SBP on standing. Participants in the bottom 30% (ie, SBP decreased on standing) were significantly older, had a greater prevalence of hypertension and peripheral vascular disease, had higher values of SBP, and had more cigarette-years of smoking. Among participants in the top 30% (ie, SBP increased on standing), a significantly larger proportion were black, mean seated SBP was higher, and the predicted risk of developing coronary heart disease after 8 years was greater. The response of SBP to change in posture showed considerable variability in a population sample of middle-aged adults. Cardiovascular morbidity, sociodemographic factors, and cigarette smoking were associated with the magnitude and direction of the postural change. (Hypertension. 1999;33:1123-1129.)

Study Population

The study population was drawn from the cohort component of the ARIC Study, which is sponsored by the National Heart, Lung, and Blood Institute. The ARIC cohort was selected as a probability sample of 15,792 men and women between 45 and 64 years of age from 4 US communities: Forsyth County, NC; Jackson, Miss; suburban Minneapolis, Minn; and Washington County, Md. The ARIC Study protocol was approved by the institutional review boards of participating institutions. All ARIC participants agreed voluntarily to participate in the study and gave written informed consent. Details of the sampling frames and methods and cohort examination procedures were described previously.

Data from ARIC participants examined at the initial clinic visit were used in these analyses (1987–1989). During the baseline examination, cardiovascular conditions were determined and risk factors for cardiovascular disease were collected.
factors were measured. Three measurements of blood pressure were obtained with a random-zero sphygmomanometer while the participant was seated. The mean of the second and third DBP and SBP measurements were used for analysis. Height (cm), weight (kg), waist circumference (measured at the umbilicus), and hip circumference (measured at the widest point) were measured by trained technicians. Blood was drawn for assays by the ARIC Central Lipid, Hemostasis, and Chemistry Laboratories. Seated blood pressure measurement, anthropometry, and venipuncture were performed while the participant was fasting, and then a caffeine-free snack was provided. Ankle and brachial blood pressures were measured by a Dinamap 1846 SX automated blood pressure recorder. The ankle SBP was divided by the brachial SBP to calculate the ankle-brachial index (ABI). Medical history was recorded and years of smoking exposure were determined during a home interview. Medications taken by each participant were brought to the baseline examination and coded by a trained interviewer. The presence of coronary heart disease (CHD) was determined by reported physician diagnosis, evidence from the electrocardiographic examination, or history of coronary revascularization. Current hypertension was defined as a seated SBP of \( \geq 140 \) mm Hg, a seated DBP of \( \geq 90 \) mm Hg, or the use of antihypertensive medications in the past 2 weeks. Diabetes mellitus was defined as a fasting glucose level of \( >7.77 \) mmol/L, a nonfasting glucose level of \( >11.1 \) mmol/L, or use of hypoglycemic medications in the 2 weeks before examination.

Participants were excluded from the sample if (1) they reported their race as other than black or white (n=48), (2) the postural change examination was not administered (n=1856), or (3) the postural change examination data were not fully recorded or missing (n=548). After exclusions, the sample size was 13,340. The postural change examination has been shown to be an accurate marker of autonomic dysfunction in diabetic individuals.\(^9\) Therefore, participants with diabetes (n=1410) were excluded from all postural change analyses to avoid confounding due to any potential relationship between autonomic dysfunction and diabetes.\(^10\)

### Measurement of Blood Pressure Response to Change in Posture

The measurement of postural change in blood pressure was conducted by a trained and certified technician. Supine and standing blood pressures were measured by using a Dinamap 1846 SX oscillometric device and a dedicated microcomputer. Details of the measurement of blood pressure can be found in ARIC Manual 11: Sitting Blood Pressure and Postural Changes in Blood Pressure and Heart Rate.\(^11\) Supine measurements of blood pressure and heart rate were taken after the participant had lain on the examination table during the ultrasound examination for a minimum of 25 minutes. Heart rate was measured on a beat-to-beat basis, and blood pressure was determined approximately every 30 seconds. The computer collected these data for 2 minutes. After the supine data were collected, the participant immediately stood and additional blood pressure measurements were taken for 2 minutes at \( \approx 30\)-second intervals to assess the cardiovascular response to change in posture. Participants were instructed to bend their elbows and to hold their hands over the midriff in a comfortable position to prevent the cuff from sliding, to distract the participant, and to place him or her in a standard and comfortable position.

The ARIC investigators tested the validity of the Dinamap early in the study and found the device to be accurate and to provide highly repeatable blood pressure measurements (within-person SD, 2.5 mm Hg; reliability coefficient, 0.96).\(^12\)

### Statistical Methods

The response variable, postural change in SBP (ΔSBP), was defined as the average of all available supine SBP readings minus the average of SBP readings on standing (excluding the first blood pressure reading after standing). Although all analyses were also performed on postural change in DBP (ΔDBP), only results for ΔSBP are presented here. The response of ΔDBP was quite similar to that of ΔSBP, although it was \( \approx 4 \) mm Hg greater on average.

Figure 1 shows a positive linear association (\( \alpha=-3.9, \beta=1.2, \) and \( r^2=0.62 \)) between ΔSBP and ΔDBP.

Frequency histograms of ΔSBP based on all participants (and on all race and gender groups) were prepared, accompanied by descriptive statistics. A categorical measure of ΔSBP was created by stratifying the variable into deciles. We then examined the distribution of various demographic variables across deciles of ΔSBP. These results were obtained in a logistic regression model with probability of demographic characteristics or positive prevalent disease status as outcome variables and with indicators of deciles of ΔSBP as predictor variables, controlling for age and seated SBP. These models were then expanded with the addition of race and gender as predictors plus interaction terms of race and gender with the indicators of deciles of ΔSBP. We then created 3 ΔSBP strata based on the decile cutoffs: decrease in SBP on standing (deciles 1 through 3), little or no change in SBP on standing (deciles 4 through 7), and increase in SBP on standing (deciles 8 through 10). The cutoffs for these groups were \( <-4.83 \), \(-4.83\) to 4.80, and \( >4.80 \) mm Hg, respectively. The relative percentage difference in study variables, such as serum lipids and disease prevalence, between the value in the increase in SBP group (or decrease in SBP group) and the value in the no change in SBP group was calculated (\( \pm 95\% \) confidence interval), adjusting the difference for age and seated SBP. All analyses were performed using SAS version 6.09 (SAS Institute).

### Results

Table 1 provides baseline characteristics of the ARIC Study sample for whom ΔSBP measurements were available (n=13,340). This sample was not significantly different in age, race, and gender distributions, anthropometric variables, or seated SBP from participants who had missing ΔSBP data. Figure 2 shows a frequency histogram of ΔSBP for the entire sample. A normal distribution curve was superimposed on the histogram using the baseline mean and SD. Although the average response of ΔSBP was near zero (mean, \(-0.45\); median, 0.08), the range was large (–63.2 to 54.3 mm Hg; SD, 10.8). Table 2 lists related descriptive statistics for the entire sample and each race/gender group. Although the shape of the distribution was similar for each group, there was a slight positive shift of the distribution for black participants; this shift was more pronounced among men than women.

Estimates of the percentages of demographic characteristics and disease prevalence by deciles of ΔSBP after adjustments for age and seated SBP are shown in Table 3. The race...
Table 1. Baseline Characteristics of Study Participants (n=13,340)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;60 y, %</td>
<td>18.1</td>
</tr>
<tr>
<td>Gender, %</td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>44.8</td>
</tr>
<tr>
<td>Women</td>
<td>55.2</td>
</tr>
<tr>
<td>Race, %</td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>27.1</td>
</tr>
<tr>
<td>White</td>
<td>72.9</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>34.5</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>9.8</td>
</tr>
<tr>
<td>CHD, %</td>
<td>4.9</td>
</tr>
<tr>
<td>ABI &lt;0.9, %</td>
<td>3.1</td>
</tr>
<tr>
<td>Age, y</td>
<td>54.1 (5.75)</td>
</tr>
<tr>
<td>Smoking, cigarette-years</td>
<td>323 (439)</td>
</tr>
<tr>
<td>Seated SBP, mm Hg</td>
<td>121 (19.0)</td>
</tr>
<tr>
<td>Seated DBP, mm Hg</td>
<td>73.6 (11.2)</td>
</tr>
<tr>
<td>Height, cm</td>
<td>169 (9.30)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.7 (5.34)</td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.93 (0.08)</td>
</tr>
<tr>
<td>Cholesterol, mmol/L</td>
<td>5.53 (1.08)</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>3.54 (1.01)</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.33 (0.44)</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>3.39 (2.30)</td>
</tr>
<tr>
<td>Insulin, μU/mL</td>
<td>14.1 (29.1)</td>
</tr>
<tr>
<td>Glucose, mmol/L</td>
<td>2.82 (1.05)</td>
</tr>
</tbody>
</table>

*Values in parentheses are standard deviations.

An ABI of <0.9 across the deciles of ΔSBP. The distribution curve for ABI of <0.9 across ΔSBP deciles was U- or J-shaped for blacks, whereas a generally decreasing trend across deciles of ΔSBP was observed for whites. The distribution curve for prevalence of hypertension was U-shaped across the deciles of ΔSBP.

Figure 3 shows the relative percentage difference (±95% confidence interval) in the prevalence or level of selected cardiovascular risk factors between nondiabetic participants who showed a change (increase or decrease) in SBP on standing and those with little or no change in SBP on standing. The relative percentage difference was adjusted for age and seated SBP with 2 exceptions. The percentage difference in the probability of age >60 years was not adjusted for age, and hypertension and 8-year risk of developing CHD were not adjusted for seated SBP. The black bars represent comparisons for risk factors between participants with a ΔSBP of <−4.83 (deciles 1 through 3) and those with a ΔSBP between −4.83 and 4.80 (deciles 4 through 7); white bars represent comparisons for risk factors between participants with a ΔSBP of >4.80 (deciles 8 through 10) and those in deciles 4 through 7. Error bars represent 95% confidence intervals for point estimates. For example, the decrease in SBP group had 10.1% more mean cigarette-years of exposure than the no change group. The 95% confidence interval for the point estimate was 4.4% to 15.7%. The increase in SBP group had 5.9% fewer mean cigarette-years of exposure than the no change group (point estimate, −5.9%; 95% confidence interval, −12.10 to 0.19).

Results from Figure 3 are similar to those in Table 3. Study participants who exhibited a decrease in SBP on standing were older and had more age-adjusted concomitant disease than their counterparts who had a relatively small change in SBP on standing. There were 35% more individuals older than 60 years of age among the decrease in SBP group (21.9% versus 15.4%). Also, there were 15% more hypertensives (36.7% versus 29.2%), 47% more participants with an ABI of <0.9 (3.9% versus 2.4%), and a 22% difference in prevalence of CHD (5.0% versus 4.0%) in the decrease in SBP group. There were significantly more (21%) blacks among those who had an increase in SBP on standing (28.1% versus 22.7%). All of these differences were statistically significant. Gender differences between groups were small.

Differences in anthropometric variables between groups were virtually zero, except for the 2.6% difference in body mass index (BMI) between men and women. Black participants also had a different pattern of peripheral arterial disease prevalence (estimated by the proportion with an ABI of <0.9) across the deciles of ΔSBP. The distribution curve for ABI of <0.9 across ΔSBP deciles was U- or J-shaped for blacks, whereas a generally decreasing trend across deciles of ΔSBP was observed for whites. The distribution curve for prevalence of hypertension was U-shaped across the deciles of ΔSBP.

Figure 2. Frequency histogram of ΔSBP (mm Hg) with fitted normal distribution curve.
mass index (BMI, 27.8 versus 27.1 kg/m²) for the increase in SBP group, which was statistically significant. The percentage difference for serum lipids between change and no change groups was also quite small. Participants in the decrease in SBP group had a 1.9% greater mean total cholesterol level (5.59 versus 5.48 mmol/L), a 2.8% higher mean LDL cholesterol level (3.59 versus 3.49 mmol/L), and a 4.3% greater mean triglycerides level (3.33 versus 3.18 mmol/L). Participants in the increase in SBP group had a 0.8% greater mean total cholesterol level (5.53 versus 5.48 mmol/L) and a 1.3% higher mean LDL cholesterol level (3.54 versus 3.49 mmol/L). All of these differences were statistically significant. There was a near-zero difference in fasting serum glucose level and a small but statistically significant difference in mean serum insulin level (5.6%) within the increase in SBP group (11.3 versus 10.7 μU/mL) and a small but statistically significant difference (3.6%) within the decrease in SBP group (11.1 versus 10.7 μU/mL).

Although there was virtually no difference in seated DBP between the groups, there was a significant 4% greater seated SBP within the increase (122 versus 117 mm Hg) and decrease (122 versus 117 mm Hg) in SBP groups. Smoking, measured in cigarette-years of exposure, was also significantly greater (10.1%) among the decrease in SBP group (351 versus 317 cigarette-years). Both the increase (4.9% versus 4.7%) and decrease (5.0% versus 4.7%) in SBP groups had a 5% greater predicted risk of developing CHD after 8 years, which was statistically significant. The 8-year CHD risk equation was based on an analysis of Framingham Study data.

### Discussion

The usual cardiovascular changes that follow postural change from the supine to standing position are well established. Blood begins to pool in the venous system on standing. This causes a decrease in venous return and a resultant drop in cardiac output. Systemic blood pressure falls with cardiac output, so the baroreceptors in the carotid arch relax and induce sympathetic stimulation. This promotes vasoconstriction and increases heart rate, which stabilizes cardiac output at a level lower than when supine. Mean blood pressure is maintained by an increase in total peripheral resistance. Overall, the response to standing is usually a very slight reduction of SBP (<4 mm Hg) and slight elevation of SBP (<10 mm Hg). Our results indicate that the response of SBP to standing is normally distributed around a mean near zero. However, the range of the distribution is quite wide (±60 mm Hg). Furthermore, it appears that an individual’s ΔSBP value (ie, their position within the distribution) is associated with a number of disease states and common cardiovascular risk factors.

Large fluctuations in heart rate and blood pressure occur in the first 20 to 30 seconds of standing upright. In the ARIC protocol, the first standing blood pressure is measured after 30 seconds in the upright position. Therefore, this early response was not included in the data analysis. Readings obtained after 1 minute indicated a very slight overall mean reduction in SBP (~0.45), yet we also saw that some participants had large (>20 mm Hg) decreases or increases in SBP on standing. Age, hypertension, CHD, smoking, systolic blood pressure, and an ABI of <0.9 were associated with a fall in SBP on standing.

β-Blockade blunts the response of the cardiovascular system to stress and therefore may lead to misclassification of ΔSBP if the sickest individuals have suppressed responses. However, Mills and Dimsdale performed a thorough meta-analysis of 59 studies examining the effects of β-blockade on cardiovascular reactivity to a variety of stressors, including postural change. They found that even though β-blockade does diminish the response of heart rate to stress, response of blood pressure is unaffected. A more strenuous stressor (ie, exercise) is required before β-blockade affects the response of blood pressure. As a precautionary measure, we reran our models for Figure 3 excluding all participants on β-blockers. These exclusions did not materially change the point estimates or the width of confidence intervals.

The association of postural hypotension with increasing age is well documented. The initial stroke volume and cardiac output reductions usually seen in response to postural change are more marked with increasing age, and the rise in

### TABLE 3. Distribution of Demographic and Morbidity Variables by Deciles of Postural Change in SBP Adjusted for Age and Seated SBP

<table>
<thead>
<tr>
<th>Attribute</th>
<th>D1</th>
<th>D2</th>
<th>D3</th>
<th>D4</th>
<th>D5</th>
<th>D6</th>
<th>D7</th>
<th>D8</th>
<th>D9</th>
<th>D10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blacks, %</td>
<td>29.2</td>
<td>25.6</td>
<td>23.1</td>
<td>23.7</td>
<td>24.1</td>
<td>26.6</td>
<td>24.2</td>
<td>26.5</td>
<td>30.6</td>
<td>38.0</td>
</tr>
<tr>
<td>Men, %</td>
<td>40.7</td>
<td>46.8</td>
<td>46.5</td>
<td>48.4</td>
<td>47.4</td>
<td>44.0</td>
<td>44.1</td>
<td>46.0</td>
<td>46.8</td>
<td>39.7</td>
</tr>
<tr>
<td>Age &gt;60 y, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>38.1</td>
<td>21.5</td>
<td>19.5</td>
<td>21.7</td>
<td>16.4</td>
<td>17.6</td>
<td>18.3</td>
<td>15.9</td>
<td>17.6</td>
<td>18.1</td>
</tr>
<tr>
<td>Women</td>
<td>22.9</td>
<td>18.9</td>
<td>17.8</td>
<td>14.6</td>
<td>14.7</td>
<td>15.1</td>
<td>12.7</td>
<td>15.0</td>
<td>14.7</td>
<td>16.1</td>
</tr>
<tr>
<td>CHD, %</td>
<td>6.5</td>
<td>5.3</td>
<td>5.5</td>
<td>4.9</td>
<td>4.8</td>
<td>5.0</td>
<td>3.8</td>
<td>4.4</td>
<td>4.8</td>
<td>4.7</td>
</tr>
<tr>
<td>Diabetes, %</td>
<td>14.5</td>
<td>10.3</td>
<td>10.5</td>
<td>10.0</td>
<td>7.4</td>
<td>8.4</td>
<td>7.6</td>
<td>8.1</td>
<td>9.1</td>
<td>12.7</td>
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<tr>
<td>Hypertension, %</td>
<td>42.6</td>
<td>37.0</td>
<td>35.5</td>
<td>34.1</td>
<td>31.0</td>
<td>34.0</td>
<td>31.4</td>
<td>31.5</td>
<td>34.0</td>
<td>35.4</td>
</tr>
<tr>
<td>ABI &lt;0.9, %</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>6.2</td>
<td>5.2</td>
<td>2.6</td>
<td>5.2</td>
<td>1.1</td>
<td>3.2</td>
<td>1.5</td>
<td>4.8</td>
<td>3.6</td>
<td>3.2</td>
</tr>
<tr>
<td>Whites</td>
<td>5.2</td>
<td>4.1</td>
<td>3.1</td>
<td>2.7</td>
<td>3.2</td>
<td>2.2</td>
<td>2.7</td>
<td>1.7</td>
<td>2.1</td>
<td>2.2</td>
</tr>
</tbody>
</table>

*Excluding angina pectoris.
†Blood pressure >140/90 mm Hg or taking anti hypertensive medication.
Total peripheral resistance is much less than seen in younger adults. Possible explanations for this response in older individuals include a decline in the sensitivity of cardiac \( \beta \)-adrenergic responses,\textsuperscript{20} reduced baroreceptor sensitivity,\textsuperscript{21,22} and elevated circulating plasma noradrenaline levels that attempt to compensate for reduced receptor sensitivity but may blunt the normal response of an increase in noradrenaline release on standing.\textsuperscript{23}

A fall in SBP on standing was associated with hypertension, smoking, SBP, and an ABI of <0.9. The prevalence of CHD was greater among those with a decrease in SBP as well and was statistically significant. Results of laboratory investigations by Abelmann and Fareeduddin\textsuperscript{24} and Zambrano and Spodick\textsuperscript{25} indicate that individuals with cardiovascular disease have a blunted heart rate and ejection time response to orthostatic stress. Structural changes in the circulatory system resulting in less compliant vessels would contribute to a decrease in their ability to vasoconstrict as well as a reduction in the sensitivity of the baroreflex mechanism. Both conditions would inhibit the return of blood pressure to normal levels on standing. However, the role of autonomic dysfunction, which was not measured in this study, cannot be ruled out.

The response to postural change was similar in men and women, as seen in a previous study.\textsuperscript{7} These findings are also supported by MacLennan et al.,\textsuperscript{6} who found no significant difference in the prevalence of postural hypotension between men and women. However, other studies reported a gender

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**Figure 3.** Percentage difference (±95% confidence interval) in selected cardiovascular risk factors between participants with a change in SBP and those with little or no change in SBP on standing. Adjusted for age and seated SBP. Nondiabetic participants only (n=11 930).
difference in the blood pressure response to standing.4,5 Our results are the first reported from a large community-based, biracial sample of adults.

Although many variables were associated with a decrease in blood pressure on standing, only race, SBP, insulin level, BMI, and 8-year predicted risk of CHD were significantly associated with an increase in blood pressure in our study. The data indicate that individuals who had an increase in blood pressure on standing may be younger, smoke less, have less CHD, and have a greater prevalence of ABI <0.9 than their counterparts, but none of these differences were significant. It appears that different mechanisms should be postulated for an increase versus a decrease in SBP on standing.

That blacks had a greater SBP response to standing than whites is consistent with findings from reactivity studies.26 Light and Sherwood27 and more recently Sherwood and Hinderliter28 investigated the underlying causes of this association and proposed 3 possible mechanisms. First, blacks may have greater a-adrenergic activity or a-receptor sensitivity than whites. This would induce a larger vasoconstrictor response in the vascular beds. Second, blacks may have less D-adrenergic activity or D-receptor sensitivity than whites. This would induce less vasodilation in skeletal muscle and other tissues. Third, early structural changes in the vasculature may potentiate vasoconstrictive responses without altered D-receptor activity or sensitivity. Fourth, these effects may occur together and jointly contribute to enhanced vasoconstriction.

The U-shaped association of seated SBP with ΔSBP, although small (4% greater in both the SBP increase and SBP decrease group), is interesting. It is probable that different mechanisms are responsible for the association in the 2 groups. There is some indication in the literature that postural hypotension and decreases in blood pressure on standing may be greater in individuals with elevated average blood pressure.6,29 The most plausible explanation for this is that the SBP level contributes to decreased baroreflex capacity.22 However, several recent studies report an association between elevated blood pressure and increased blood pressure reactivity.30,31 Although we have no information on previous blood pressure for our study participants, it is possible that individuals with a negative ΔSBP may have had elevated blood pressure for some time, resulting in decreases in baroreceptor sensitivity. In contrast, individuals with a positive ΔSBP may have only recently had increases in blood pressure, possibly influenced by their high stress reactivity.

Both decreases and increases in SBP in response to posture change were associated with similar, statistically significant increases in the 8-year predicted risk of CHD. Although this remains tentative until confirmed by incidence data, these results suggest that changes in SBP, regardless of direction, may be useful indices of cardiovascular risk in this age group.

Certain limitations apply to this study. First, the timing and number of blood pressure measurements deserve some discussion. The Dinamap blood pressure recorder was in an automatic recording mode that measured SBP and DBP as frequently as possible in a 2-minute period. Although this maximizes the number of available blood pressure measurements, it has the drawback of increasing the variability in timing and number of measurements. Therefore, individual blood pressures are not directly comparable with respect to length of time since standing. Because different mechanisms control blood pressure response at specific time points in the first several seconds after standing,14 the varied measurement times prevent us from examining the specific mechanisms involved in the blood pressure response to change in posture. Instead, a measure of reactivity integrated over nearly 2 minutes of standing was calculated as the difference of the mean supine and mean standing blood pressure measurements.

Although ARIC is a population-based study, the overall participation rate was 65% for the field center examination. As in most epidemiological studies, individuals who agree to participate in the study are likely to select themselves into the cohort according to a variety of attributes. Participants may be more likely to be healthier and have a higher socioeconomic status than individuals in the communities from which they were sampled. If that is the case, the estimates of disease prevalence may be conservative and not necessarily reflective of the true rates in these 4 communities. The magnitude of this possible bias is unknown. Because most of the black participants were inducted at the Jackson, Miss, field center, the ability to make generalizations on the basis of our findings in blacks is limited. Finally, these are cross-sectional data. Therefore, the associations reported neither suggest causality nor establish a temporal relationship between any of the variables studied and postural change in blood pressure.

However, this study is the first to examine blood pressure response to postural change in a large community-based sample of black and white adults. Previous studies have been conducted with smaller samples that have not been community based, or the study participants were adolescents,52 or the investigators chose to report the prevalence of orthostatic hypotension instead of absolute change in blood pressure.33 The quality control procedures established in the ARIC Study have been described previously.8 These rigorous measures are designed to ensure that data are collected uniformly at each center and over time and are also applied to the measurement of postural change in blood pressure. Examinations were performed by trained and certified technicians. Each participant had at least 20 minutes of comfortable rest before the start of the postural change examination. A clinic setting can be a stressor itself and may artificially increase blood pressure and blood pressure reactivity, but experiments have shown that catecholamines return to basal levels after 20 minutes of supine rest.34

Conclusion
Our findings indicate the practicality and usefulness of the postural change in blood pressure examination in an epidemiological study. The response of blood pressure to change in posture may serve as a tool at the population level for measuring a variety of mechanisms related to cardiovascular morbidity. Of particular interest is the difference in ΔSBP between black and white participants. This may partially explain the high prevalence of hypertension among US blacks, which is nearly twice that of whites.35 Analysis of the
association of this pressor response with incident cardiovascular diseases should be performed.

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