Pulse Pressure Compared With Other Blood Pressure Indexes in the Prediction of 25-Year Cardiovascular and All-Cause Mortality Rates

The Chicago Heart Association Detection Project in Industry Study

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Abstract—We compared the relations of 4 blood pressure (BP) indexes (pulse pressure [PP], systolic BP [SBP], diastolic BP [DBP], and mean arterial pressure [MAP]) with 25-year mortality rates for coronary heart disease (CHD), cardiovascular disease (CVD), and all causes in younger, middle-aged, and older men and women by using data from a long-term prospective epidemiological study of employed persons who were screened between 1967 and 1973. A single supine BP measurement was obtained at baseline. Vital status was determined through 1995. We report on 5 groups (total, 28,360 participants) consisting of men age 18 to 39, 40 to 59, and 60 to 74 years and of women age 40 to 59 and 60 to 74 years who were not receiving antihypertensive treatment, had no history of CHD, and did not have diabetes. Cox proportional hazards analyses were used to determine multivariate-adjusted hazard ratios with a 1-SD higher value for each BP index; Wald $\chi^2$ tests were used to compare the strength of relations. Relations of PP were less strong than were those of SBP for all end points in all age/gender groups. SBP or MAP showed the strongest relations to all end points in all age/gender groups (hazard ratio, 1.17 to 1.36). The relations of SBP to death were stronger than were those of DBP, except for middle-aged men and for CVD in women. DBP showed significant positive associations with death, after control for SBP, in middle-aged participants. In conclusion, these data indicate that the long-term risk of high BP should be assessed mainly on the basis of SBP or of SBP and DBP together, not on the basis of PP, in apparently healthy adults. (Hypertension. 2001;38:232-237.)

Key Words: blood pressure ■ mortality ■ coronary disease ■ cardiovascular diseases ■ epidemiology

Blood pressure (BP) is an established major risk factor for coronary heart disease (CHD) and stroke.1–6 Risk relations for both systolic BP (SBP) and diastolic BP (DBP) are generally regarded as continuous, graded, strong, independent of other risk factors, and etiologically significant. Some data indicate that SBP is a stronger predictor of cardiovascular disease (CVD) than is DBP.7–10 Several recent epidemiological studies reported that pulse pressure (PP), the difference between SBP and DBP, is a useful predictor for CHD or total CVD, especially in middle-aged or older persons.11–18 These reports emphasized the importance of PP as a CHD or CVD risk factor, especially because PP is often higher after age 50, apparently because of increased arterial stiffness.19–22

In regard to prior reports on PP, many did not compare the significance of PP with that of SBP or DBP, and some reports were studies on hypertensive persons only. Only 2 reports, from the Framingham Heart Study, compared the relations to CHD and CVD incidence of various BP indexes,10,11 and such comparisons for total mortality in general populations have not been reported. Therefore, it is uncertain whether PP is superior to SBP or DBP in the prediction of future CHD, CVD, and all-cause mortality in various age/gender groups of the general (ie, apparently healthy) population.

Because recent discussions have emphasized the importance of SBP compared with DBP7–10 and because these are strongly correlated, it is also important to assess whether DBP has any additional role in the prediction of risk independent of SBP. Such analyses are relevant to understand the meaning of PP as a cardiovascular risk factor.

The present report compares the relations of 4 BP indexes (PP, SBP, DBP, and mean arterial pressure [MAP]) to 25-year mortality risks from CHD, CVD, and all causes in 5 population cohorts (total, 28,360 men and women) classified by age and gender from the Chicago Heart Association (CHA) Detection Project in Industry Study. The specific goals of this research are to assess, in each of the 5 age/gender...
cohorts of this general population sample, for each of the 3 mortality end points (1) whether PP is the best predictor among the 4 indexes, (2) whether the relation of SBP is stronger than that of DBP, (3) the relation of MAP to the 3 end points, and (4) the relations of SBP and DBP, after adjustment for each other, to death.

**Methods**

**Population**

The methods of the CHA study have been described previously.\(^{23,24}\) Briefly, 39,573 men and women age ≥18 years were screened between November 1967 and January 1973. All employees at 84 cooperating Chicago-area companies and organizations, with a labor force of ∼75,000 people, were invited to participate; the volunteer rate was 53%. The study received periodic institutional review board approval at Northwestern University (Chicago, Ill.).

**Survey Methods**

Screening was conducted by 2 trained and standardized 4-person field teams. Baseline screening included an assessment of age, gender, ethnicity, level of education, BP, total serum cholesterol level, smoking status, height and weight used to calculate body mass index (BMI), resting ECG findings, medical history, and current treatment for chronic diseases, including hypertension and diabetes. A single casual supine BP measurement was obtained by trained staff using a standard mercury sphygmomanometer. DBP was recorded as Korotkoff phase V. BP was taken to the nearest 2 mm Hg. Standardized, high-quality methods were used for total serum cholesterol level determination.\(^{25}\) The criteria of the National Cooperative Pooling Project and the Hypertension Detection and Follow-up Program were used to code ECG abnormalities.\(^{26}\)

**Mortality End Points**

Vital status was ascertained through 1995, with an average follow-up of 25 years. Deaths were determined through several methods: before 1979, by direct mail, telephone, contact with employer, and matching of cohort records with Social Security Administration files; and after 1979, by the matching of study records with National Death Index records. Multiple causes of death listed on the death certificate were coded by trained research staff according to the eighth revision of the International Classification of Diseases (ICD-8).\(^{27}\) Coding decisions were based on the underlying cause of death and were cross-checked by study team members. All coders were blinded to baseline data. CHD mortality was defined as ICD-8 codes 410.0 to 414.9, and CVD mortality was defined as ICD-8 codes 390.0 to 445.9.

**Inclusions and Exclusions**

Men age 18 to 74 years and women age 40 to 74 years at baseline were included in these analyses. Women age 18 to 39 years at baseline were excluded because too few deaths from CHD and CVD occurred during the follow-up period. Participants with ECG evidence of prior myocardial infarction at baseline (0.4%), past history of myocardial infarction or other CHD (1.5%), antihypertensive drug treatment at baseline (5.5%), previously diagnosed diabetes mellitus (2.3%), or missing data at baseline or on follow-up (1.8%) were excluded. Thus, this report is based on a total of 28,360 persons with the following age/gender breakdown: 10,874 men age 18 to 39 years, 8307 men age 40 to 59 years, 1257 men age 60 to 74 years, 6909 women age 40 to 59 years, and 1013 women age 60 to 74 years.

**Statistical Analysis**

Pearson product-moment correlation coefficients were calculated for each pair of indexes at baseline (ie, SBP, DBP, MAP [calculated as SBP/3 + 2DBP]/3),\(^{28}\) and PP [calculated as SBP — DBP]). For each BP index considered separately as a continuous variable, Cox proportional hazards regression was used to determine multivariate-adjusted hazard ratios (HRs) for a level greater by 1 SD. Wald \(\chi^2\) tests were also used to compare the strength of relations. One Cox model also included SBP and DBP simultaneously to assess relations with adjustment for each other. HRs were adjusted for other major risk factors and for potential confounders (age [in years], race [black or not], education [in years], serum total cholesterol [mg/dL], cigarette smoking [cigarettes/d], BMI [calculated as weight in kilograms divided by the square of the height in meters, or kg/m\(^2\)], and any ECG abnormality).

**Results**

**Baseline Findings**

Mean ages by age/gender group were 29.7, 48.5, and 63.0 years for the 3 age groups of the men and 49.3 and 63.1 years for the 2 age groups of the women. Mean values of 4 BP indexes are shown in Table 1 for men and in Table 2 for women. Mean values of SBP, DBP, and MAP were higher with age in both men and women. Mean PP was higher in older men and women than in the younger groups. Mean SBP, DBP, and MAP were higher in men than in women for those age 40 to 59 and 60 to 74 years. Mean PP was similar between genders. The SD for each index increased with age in both men and women.

For product-moment (Pearson) simple correlations among the 4 BP indexes by age/gender groups, all indexes were positively and significantly correlated with each other (\(P < 0.001\)), except for an inverse correlation between DBP and PP in men age 18 to 39 years. PP was strongly correlated with SBP in each age/gender group (coefficients 0.73 to 0.84) and weakly correlated with DBP (−0.12 to 0.22). MAP was strongly correlated with both SBP (0.85 to 0.92) and DBP (0.92 to 0.94), as expected for an index that is the weighted average of SBP and DBP. Correlations between SBP and DBP ranged from 0.59 to 0.73. Similar correlations were observed when Spearman’s rank order correlations were calculated.

**Baseline BP Indexes and 25-Year Mortality Rates in Men**

The numbers of deaths during the follow-up period were 197 from CHD, 257 from CVD, and 759 from all causes in men age 18 to 39 years; 802, 1123, and 2647 in men age 40 to 59 years; and 286, 427, and 883 in men age 60 to 74 years, respectively. Table 1 shows multivariate-adjusted HRs for CHD, CVD, and all-cause mortality for each BP index higher by 1 SD from the continuous variable Cox models for each age group of men. PP was positively and significantly related to the end points in 8 of 9 analyses (HR 1.09 to 1.20). Wald \(\chi^2\) tests indicated that the relations were less strong for PP than for SBP in all age groups for all end points and less strong than for DBP in 7 of 9 analyses. Among the 4 indexes, either SBP or MAP, with generally similar Wald \(\chi^2\) tests, had the strongest relations (HR 1.17 to 1.30). In men age 18 to 39 and 60 to 74 years, the relation of SBP was stronger than that of DBP for all end points. Consistent with these results, a Cox model that included both SBP and DBP showed significantly higher HRs for SBP (1.10 to 1.26) for all end points in these 2 age groups. For all end points in men age 40 to 59 years, DBP had a significant positive and stronger relation than did SBP; after adjustment for SBP, HRs ranged from 1.12 to 1.23 for the relations of DBP to the mortality end points.
Baseline BP Indexes and 25-Year Mortality Rates in Women

The numbers of deaths during the follow-up period were 310 from CHD, 491 from CVD, and 1412 from all causes in women age 40 to 59 years, and 163, 269, and 536 in women age 60 to 74 years, respectively. Table 2 shows multivariate-adjusted HRs for CHD, CVD, and all-cause mortality for each BP index higher by 1 SD from continuous variable Cox models for both age groups of women. PP was positively and significantly related to 3 end points in 5 of 6 analyses (HR 1.13 to 1.21). Wald $x^2$ tests indicated that the relations were less strong for PP than for SBP and DBP in both age groups for all end points. Among the 4 indexes, either SBP or MAP, with generally similar $x^2$ tests, had the strongest relations (HR 1.21 to 1.36). The relation of SBP was stronger than that of DBP for CHD and all causes; however, the relation of DBP was slightly stronger for CVD. A Cox model that included both SBP and DBP yielded significant HRs (1.21 to 1.36) for SBP in 3 of 6 analyses, with $x^2$ tests higher for SBP than for DBP in 4 analyses. In the models that included both SBP and DBP, DBP had a significant positive relation to CVD and all-cause mortality in middle-aged women (HR 1.13 to 1.20).

Overall, with the exceptions of PP and CVD in women age 60 to 74 years and of PP and all-cause mortality in men age 18 to 39 years, each BP index was significantly related to each end point (most $P<0.01$), independent of other confounding factors. Either SBP or MAP, generally with similar $x^2$ tests, had the strongest relation in each age/gender group (HR 1.17 to 1.36).

Similar results were obtained when the 1625 participants on antihypertensive drug treatment at baseline were included in the analyses (data not shown).

Three Cox Models That Include Dual BP Indexes
For the purpose of illustration, Table 3 shows the results for 3 Cox models that include 2 BP indexes: SBP and DBP (model 1), PP and SBP (model 2), and PP and DBP (model 3). The results are demonstrated for CHD risk in men age 60 to 74 years at baseline, because recent discussions regarding PP have been focused mainly on CHD in older persons. The regression coefficient for PP in model 3 is equal to the regression coefficient for SBP in model 1, and the regression coefficient for PP in model 2 is equal to minus the regression coefficient for DBP in model 1. Also, the regression coefficients for SBP and DBP are identical when PP is the second
These results show the functional equivalence of models 2 and 3 to model 1.

**Discussion**

The main findings from this 25-year follow-up study on CHD, CVD, and all-cause mortality for 5 age/gender–specific cohorts from an employed population are that (1) relations of PP were less strong than those of SBP for all end points in all age/gender groups studied, (2) relations of PP were less strong than those of DBP for all end points in middle-aged men and women and in older women, (3) among the 4 BP indexes, the strongest relation was observed for either SBP or MAP in all age/gender groups, (4) relations of SBP to death tended to be stronger than or similar to those of DBP, and (5) with control for SBP, DBP was positively and significantly related to death in middle-age men and women but not in younger men and older men and women.

Several recent studies have suggested that PP may be a useful predictor of CHD and CVD in middle-aged or older persons, especially in those with hypertension or prevalent CVD.11–18 PP has been reported to be a good indicator of arterial stiffness; it generally is higher after age 50, due in part to the tendency of DBP to be lower and SBP to be higher with age, which is attributed to arterial stiffness.19–22 However, in our population-based study of men and women age 18 to 74 years, PP was a weaker predictor of CHD, CVD, and all-cause mortality than were both SBP and MAP in all age/gender groups, including men and women age 60 to 74 years at baseline. Further, in 13 of 15 analyses, PP was a weaker predictor than DBP. Most previous epidemiological studies conducted in general populations, emphasizing the importance of PP, have not compared PP and SBP as predictors.12,13,15–18 As far as we know, only the Framingham Heart Study made such a comparison. In 20-year follow-up of 1924 men and women age 50 to 79 years at baseline, PP was reported to be more strongly related to CHD incidence than SBP or DBP in multivariate-adjusted Cox models.11 However, in contrast, in another Framingham report, SBP was a stronger predictor of 30-year CVD incidence than PP, MAP, and DBP in men and women age 65 to 94 years.10 The Framingham results suggest SBP may be superior to PP in predicting long-term CVD morbidity but do not address CHD or CVD mortality. In contrast, our data relate to CHD, CVD, and all-cause mortality in younger, middle-aged, and older men and women but do not address the role of PP in the prediction of CHD or CVD morbidity.

### TABLE 2. Adjusted Hazard Ratios for 4 BP Indexes for 25-Year Mortality Rates for CHD, CVD, and All Causes by Age Group in Women

<table>
<thead>
<tr>
<th>Age of Women, y</th>
<th>Mean (SD), mm Hg</th>
<th>CHD</th>
<th>CVD</th>
<th>All Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hazard Ratio†</td>
<td>95% CI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40 to 59 (n=6909)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PP</td>
<td>55.4 (13.7)</td>
<td>12.2</td>
<td>1.20</td>
<td>1.08 1.33</td>
</tr>
<tr>
<td>DBP</td>
<td>79.6 (11.3)</td>
<td>13.2</td>
<td>1.24</td>
<td>1.10 1.39</td>
</tr>
<tr>
<td>SBP</td>
<td>135.0 (19.5)</td>
<td>21.3</td>
<td>1.28</td>
<td>1.15 1.41</td>
</tr>
<tr>
<td>MAP</td>
<td>98.1 (13.0)</td>
<td>19.9</td>
<td>1.28</td>
<td>1.15 1.43</td>
</tr>
<tr>
<td>SBP†</td>
<td>7.6</td>
<td>1.23</td>
<td>1.06</td>
<td>1.43</td>
</tr>
<tr>
<td>DBP</td>
<td>0.4</td>
<td>1.05</td>
<td>0.90</td>
<td>1.24</td>
</tr>
<tr>
<td>60 to 74 (n=1013)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PP</td>
<td>64.3 (16.2)</td>
<td>5.7</td>
<td>1.21</td>
<td>1.03 1.41</td>
</tr>
<tr>
<td>DBP</td>
<td>146.6 (21.3)</td>
<td>12.1</td>
<td>1.31</td>
<td>1.12 1.52</td>
</tr>
<tr>
<td>SBP</td>
<td>82.3 (11.7)</td>
<td>9.1</td>
<td>1.28</td>
<td>1.09 1.50</td>
</tr>
<tr>
<td>MAP</td>
<td>103.8 (13.6)</td>
<td>12.9</td>
<td>1.32</td>
<td>1.14 1.54</td>
</tr>
<tr>
<td>SBP†</td>
<td>3.7</td>
<td>1.22</td>
<td>1.00</td>
<td>1.50</td>
</tr>
<tr>
<td>DBP</td>
<td>0.9</td>
<td>1.11</td>
<td>0.90</td>
<td>1.38</td>
</tr>
</tbody>
</table>

*χ2 of 3.8 corresponds to P value of 0.05, 6.6 to 0.01, and 10.8 to 0.001.
†Hazard ratios are adjusted for age, cholesterol, cigarettes per day, BMI, BMI², ECG abnormality, race, and education. Hazard ratios are calculated for each BP index higher by 1 SD.
‡SBP and DBP are included in the same model.

### TABLE 3. Three Cox Models That Include 2 Indexes of SBP, DBP, and PP for 25-Year CHD Mortality Rates in Men Age 60 to 74 Years at Baseline

<table>
<thead>
<tr>
<th>Model</th>
<th>Coefficient</th>
<th>χ²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>SBP 0.0091</td>
<td>5.2</td>
<td>0.022</td>
</tr>
<tr>
<td></td>
<td>DBP 0.0069</td>
<td>0.9</td>
<td>0.333</td>
</tr>
<tr>
<td>2</td>
<td>PP −0.0069</td>
<td>0.9</td>
<td>0.333</td>
</tr>
<tr>
<td></td>
<td>SBP 0.0160</td>
<td>10.1</td>
<td>0.001</td>
</tr>
<tr>
<td>3</td>
<td>PP 0.0091</td>
<td>5.2</td>
<td>0.022</td>
</tr>
<tr>
<td></td>
<td>DBP 0.0160</td>
<td>10.1</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*Coefficients are adjusted for age, cholesterol, cigarettes per day, BMI, BMI², ECG abnormality, race, and education.
Some previous reports on PP included other BP indexes in the same multivariate model. However, in our primary analyses, we showed results only for a model that included both SBP and DBP. We also reported results of analyses with both SBP and PP and with both DBP and PP in the same multivariate model to illustrate the problems encountered in attempting to interpret results regarding PP and death with adjustment for SBP or DBP or for DBP or SBP regarding death with adjustment for PP (Table 3). Those analyses show that for the PP-SBP model, the regression coefficient for PP is identical to the negative of the coefficient for DBP from the SBP-DBP model. Similarly, for the PP-DBP model, the regression coefficient for PP is equal to the regression coefficient for SBP from the SBP-DBP model. Furthermore, the regression coefficient for SBP from the PP-SBP model and the regression coefficient for DBP from the PP-DBP model are identical. It can be shown mathematically that both coefficients measure the association of DBP adjusted for PP. Hence, one cannot by regression assess the association of SBP with death adjusted for PP, because the coefficient for SBP from that model is actually the association of DBP adjusted for PP. Because of the difficulty of interpreting results of PP-SBP or PP-DBP models and the functional overlap of PP with SBP and DBP (ie, \( PP = SBP - DBP \)), our judgment is that the appropriate regression model with 2 BP indexes is the model that includes SBP and DBP. The relative hazard for SBP in that model has a PP interpretation, because the relative hazard for SBP represents the increased risk associated with higher SBP when DBP is held constant. The relative hazard for DBP in such a model also has a PP interpretation, because the relative hazard for DBP represents the difference in risk when SBP is held constant. With SBP held constant, PP can be related to increased risk only if DBP has an inverse association with death in the SBP-DBP model, because PP is higher as DBP is lower with SBP held constant.

Recent discussions on the diagnosis and treatment of high BP have focused more on SBP than on DBP, especially in older persons. In our analyses, SBP was generally more strongly related to death than was DBP. The relations of DBP, however, were stronger than were those of SBP in middle-age men. Moreover, with adjustment for SBP, DBP also was positively and significantly related to death in middle-aged men and women. These results indicate a role for DBP in the long-term risk evaluation in men and women, especially in this age range. In fact, in middle-aged persons, previous epidemiological studies have shown that CHD risk generally increased with DBP level in each stratum of SBP level, as demonstrated in cross-classified analyses of SBP and DBP. Our results also support a recent advisory by the Coordinating Committee of the National High Blood Pressure Education Program that urged SBP become the major criterion for diagnosis, staging, and therapeutic management of hypertension in persons age \( \geq 60 \) years, and our results suggest that this advisory may be appropriate for men age 18 to 39 years.

Although MAP showed the strongest relation with death in 10 of 15 analyses, apparently reflecting modest additive effects of SBP and DBP in some age groups, \( \chi^2 \) tests and HRs were generally similar to those of SBP. MAP could be a slightly nonsignificantly better predictor of long-term risk than SBP in middle-aged men and women.

A limitation of the present study is that results were based on a single measurement of BP and thus probably are underestimates of true associations due to regression dilution bias. Nevertheless, as shown here and in many prospective studies, a single BP reading is strongly predictive of future CVD events. Data from other studies indicate that SBP is more reproducibly measured than is DBP. In INTERSALT, estimates of the reliability of BP, based on repeat measurement at an average 14-day interval, were 0.74 for SBP and 0.67 for DBP. These same data also produce reliability estimates of 0.52 for PP and 0.74 for MAP. The lower reliability for PP reflects the fact that PP is the difference between 2 measurements that have substantial day-to-day variability. Given the different estimates of reliability for the 4 BP indexes, it is not unexpected that the 2 most reliable measures (ie, SBP and MAP) are stronger predictors in the present study. Further, given the substantially lower reliability of PP compared with the other 3 indexes, hence the greater misclassification that is likely to result, its use in clinical practice as a primary measure of BP appears unwarranted.

Because this cohort was identified at employment sites, the role of the “healthy worker effect” should be considered. Because working populations tend to be healthier than general populations, the mortality rate of the CHA cohort was lower (ie, \( \approx 70\% \) of that expected for a similar sample of the general population). However, this phenomenon has little or no bearing qualitatively on the relation of baseline risk factors (including BP) to long-term mortality rates, as shown by many prospective studies with similar qualitative results on this matter for workplace- and community-based population samples. Thus, it is a reasonable inference that these findings are generalizable to other “healthy” general population samples.

A further consideration is the age ranges that were under study here. Although we included both men and women spanning ages 18 to 74, the mean baseline ages of the oldest men and women in our study were 63.0 and 63.1 years, respectively. Previous reports have emphasized that PP may become important prognostically and physiologically only at ages of \( \geq 60 \) years. Given that most of our oldest participants were in their early 60s at entry, our findings may have limited implications for persons beyond the age of \( \approx 63 \) years.

Therefore, regarding the implications of our results for public health policy and clinical practice, first, the results affirm a continued emphasis on SBP, particularly for younger men and older persons of each gender. For middle-aged persons age 40 to 59 years, DBP should be given concomitant careful consideration because of its strong independent relation to death. As reported previously, the majority of hypertensives can be diagnosed based only on SBP. However, it also is true that the majority of hypertensives (in our study, 59\% of middle-aged hypertensives) have diastolic hypertension, isolated or combined with systolic. The severity of diastolic hypertension should be carefully assessed in middle-aged persons. Second, in younger and middle-aged persons, an emphasis on PP should be avoided. There is no evidence, in a general population younger than age 60, that...
PP is superior to SBP in the prediction of CVD or all-cause mortality. Our data also do not place an emphasis on PP in men and women between the ages of 60 and 74 (mean baseline age, 63 years in these working men and women). An emphasis on the risks associated with PP is likely to underestimate the true risks. Third, relations of MAP to risk were generally as strong as or slightly stronger than those of SBP. Use of this index, however, may not be practical in daily clinical and public health practice, because there are no guidelines for hypertension diagnosis and management using MAP. Detection and evaluation of hypertension based mainly on SBP remain the most practical and easy approach in the general population for young adult, middle-aged, and older men and women (at least up to age ~63 years). Regarding the role of PP for prognosis in considerably older “healthy” populations, other studies may provide pertinent information.

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References


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