Prognostic Value of Systolic and Diastolic Blood Pressure in Treated Hypertensive Men

Athanase Benetos, MD, PhD; Frédérique Thomas, PhD; Kathryn Bean, MA, MPH; Sylvie Gautier, MD; Harold Smulyan, MD; Louis Guize, MD

Background: The aim of this study was to assess the cardiovascular risk in hypertensive subjects according to systolic blood pressure (SBP) and diastolic blood pressure (DBP) levels.

Methods: The study sample consisted of 4714 hypertensive men, treated by their physician, who had a standard health checkup at the d’Investigations Prévénitives et Cliniques Center, Paris, France, between 1972 and 1988. Cardiovascular disease (CVD) and coronary heart disease (CHD) mortality were assessed for a mean period of 14 years.

Results: Among treated subjects, 85.5% presented uncontrolled values for SBP (≥140 mm Hg) and/or DBP (≥90 mm Hg). After adjustment for age and associated risk factors, these subjects presented an increased risk for CVD mortality (risk ratio [RR], 1.66; 95% confidence interval [CI], 1.04-2.64) and for CHD mortality (RR, 2.35; 95% CI, 1.03-5.35) compared with controlled subjects. After adjustment for age, associated risk factors, and DBP, compared with subjects with SBP under 140 mm Hg, the RR for CVD mortality was 1.81 (95% CI, 1.04-3.13) in subjects with SBP between 140 and 160 mm Hg and 1.94 (95% CI, 1.10-3.43) in subjects with SBP over 160 mm Hg. By contrast, after adjustment for SBP levels, CVD risk was not associated with DBP. Compared with subjects with DBP under 90 mm Hg, RR for CVD mortality was 1.17 (95% CI, 0.80-1.70) in subjects with DBP between 90 and 99 mm Hg and 1.03 (95% CI, 0.67-1.56) in subjects with DBP over 100 mm Hg. Similar results were observed for CHD mortality.

Conclusions: In hypertensive men treated in clinical practice, SBP is a good predictor of CVD and CHD risk. Diastolic blood pressure, which remains the main criterion used by most physicians to determine drug efficacy, appears to be of little value in determining cardiovascular risk. Evaluation of risk in treated individuals should take SBP rather than DBP values into account.

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The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure1 and the recent report of the World Health Organization/International Society of Hypertension2 classified blood pressure (BP) into stages based on either systolic (SBP) or diastolic (DBP) levels. Both classifications define the lowest SBP and DBP levels as being optimal, and the highest levels of both as having the highest cardiovascular morbidity and mortality rates. In the past few years, the importance of SBP has been emphasized, especially in older subjects, and more recently, it has been proposed that DBP values could be the best predictor of cardiovascular risk in younger subjects and SBP or pulse pressure in older subjects.

It has been suggested that SBP should be under 140 mm Hg and that DBP be under 90 mm Hg for all treated hypertensive subjects, unless diabetes or target organ damage are present, in which case lower BP levels (<130/85 mm Hg) should be targeted.1,2 Observational studies from several countries have demonstrated that among treated hypertensive individuals, the proportion of those who are well controlled is less than 30%, and a recent survey in the United Kingdom indicated that only 6% of hypertensive subjects presented BP levels below the limit of 140/90 mm Hg.

One of the reasons for these results may be the relative complexity of the guidelines that include SBP and/or DBP in the various classifications. In addition, physicians may not be convinced that the results found among hypertensive subjects participating in controlled studies,
which show the benefits of low BP levels (<140/90 mm Hg), are applicable to hypertensive subjects treated in their everyday clinical practices.

In this observational study, the prognostic value of SBP and DBP levels on cardiovascular mortality, especially coronary heart disease (CHD) mortality in hypertensive subjects, treated in “everyday” clinical practice, was assessed. The study sample consisted of 4714 treated hypertensive men from the Centre d’Investigations Préventives et Cliniques (IPC) cohort.

In the study population, age (mean±SD) was 52±11 years; SBP, 152±18 mm Hg; and DBP, 94±12 mm Hg. Only 14.5% of the patients presented controlled values for both SBP and DBP, 10.8% presented uncontrolled values for SBP alone, 4.2% presented uncontrolled values for DBP alone, and 70.5% of the treated hypertensive men presented uncontrolled values for both. Compared with subjects with controlled BP values, those with high BP values had a statistically significant increase in multivariate-adjusted RR for CVD mortality (1.66; 95% CI, 1.04-2.64) and for CHD mortality (2.35; 95% CI, 1.03-5.35) (Table 1).

Table 2 shows CVD mortality rates and adjusted RRs according to the SBP and DBP categories. There was a 4-fold increase in unadjusted CVD mortality rates in subjects with SBP of 160 mm Hg or higher compared with subjects with SBP under 140 mm Hg. After adjustment for age, there was a 2.2-fold increase in CVD mortality (Figure A). As shown in the Figure, the relationship between SBP and CVD risk was linear. The group with SBP between 140 and 159 mm Hg showed a 63% (P<.01) increase in age-adjusted CVD mortality compared with the group with SBP under 140 mm Hg. After adjustment for age and associated risk factors, the risk for CVD mortality increased in the group with SBP between 140 and 160 mm Hg by almost 70% compared with the group with SBP under 140 mm Hg (Table 2). In the group with SBP
of 160 mm Hg or higher, RR for CVD mortality was 2.5 times greater than in the reference group. After complementary adjustment for DBP levels, the risk of CVD mortality was still significantly higher in the group with SBP over 140 mm Hg compared with the group with SBP under 140 mm Hg.

When subjects were classified according to DBP levels, those with DBP over 100 mm Hg showed less than a 2-fold increase in CVD mortality compared with subjects with DBP under 90 mm Hg (Table 2). After adjustment for age, the relationship between DBP levels and CVD mortality was not significant (Figure, B). After adjustment for age and associated risk factors, the risk for CVD mortality in the group with DBP between 90 and 99 mm Hg did not increase significantly, whereas in the group with DBP over 100 mm Hg, it increased by 60% (Table 2). However, after adjustment for SBP, there was no association between DBP levels and CVD mortality.

The role of SBP and DBP on cardiovascular mortality was not influenced by age (interaction term age × SBP, P = .44 and age × DBP, P = .80).

Table 3 shows CHD mortality rates and adjusted RRs according to the SBP and DBP categories (Figure). Based on SBP and DBP levels, very similar results to those observed for CVD mortality were found. Once again, the roles of SBP and DBP in CHD mortality were not influenced by age (interaction term age × SBP, P = .65 and age × DBP, P = .54).

The association between pulse pressure and CVD mortality and CHD mortality was also evaluated. The results from these analyses showed that pulse pressure had the same predictive value as SBP (data not shown).

Our data show that more than 85% of the treated hypertensive men had uncontrolled SBP or DBP levels. Among them, most (70%) had high levels of both SBP and DBP, followed by those with high SBP and controlled DBP. This clearly confirms that, as measured in a clinical setting, a controlled BP, especially SBP, is uncommon. The most important result of this study is that cardiovascular mortality, especially CHD mortality, is much higher in uncontrolled hypertensive men than in controlled hypertensive men, and that SBP levels, but not DBP levels, can predict CVD risk independent of age.

It is well documented that reducing BP is associated with a decrease in the risk of coronary and cerebrovascular complications. A meta-analysis of 14 controlled clinical trials demonstrated that lowering DBP by...
6 mm Hg reduced cerebrovascular morbidity and mortality by 42% and the risk of CHD by 14%. The Hypertension Optimal Treatment trial also confirmed that reduction in DBP with treatment decreased CVD risk by 30%. Although the benefits of reducing DBP were clearly shown in numerous controlled studies in younger and older patients with systolic-diastolic hypertension, recent findings suggest that normalization of SBP rather than DBP should be the main goal of antihypertensive treatment. The benefits of treating high SBP, especially in older subjects, were established by the Systolic Hypertension in the Elderly Program and the Systolic Hypertension Europe trials in older subjects with isolated systolic hypertension, but also in patients with an increase in both SBP and DBP. Our results were obtained from subjects who were not a part of a specific research program and were followed up by their own physicians. This study can therefore better evaluate the long-term CVD risk in hypertensive subjects followed up and treated by their physicians according to the standard clinical practice. Under these conditions that reflect the regular treatment of French hypertensive persons, we confirmed the results of several studies showing that getting SBP to goal levels is much more difficult than controlling DBP levels. The new element provided by our study is that lack of SBP control may be a major determinant of the increased morbidity and mortality in treated hypertensive persons reported by other population studies. Also, under these circumstances, the level of DBP in treated men does not seem to be of a significant prognostic value.

As shown in Table 2 and the Figure, CVD mortality was 60% higher in men with DBP values over 100 mm Hg compared with those with DBP values under 90 mm Hg. However, this was mainly because most subjects with high DBP also had high SBP; thus, after adjustment for SBP, DBP levels lost all predictive value. On the other hand, the RR for adjusted CVD and CHD mortality was approximately 2.5 times higher in treated hypertensive men with SBP values over 160 mm Hg compared with those with SBP values under 140 mm Hg. It is important to note that the predictive value of SBP remained significant even after adjustment for DBP levels. It is also interesting to point out that the presence of an SBP between 140 and 160 mm Hg (corresponding to a “mild” increase in SBP) under treatment is accompanied by a 2-fold increase in CVD and CHD age-adjusted mortality compared with treated subjects with well-controlled SBP levels. This result is important in terms of public health since a large proportion of treated hypertensive persons fall into this group (44.5% in our study).

The role of SBP and DBP in predicting CVD and CHD risk in our population was not influenced by age. However, it has previously been reported in a general population that DBP better reflects the CVD risk in younger subjects and that SBP better reflects the risk in older subjects. These differences may be explained by the fact that low DBP in treated subjects may not have the same significance as low DBP in untreated subjects. DBP levels are influenced by arterial or arteriolar alterations in opposite ways: an increase in peripheral vascular resistance leads to an elevation in DBP, whereas stiffening of large arteries can contribute to a decrease in DBP. Since stiffness is a major sign of arterial aging, it could also be suggested that subjects receiving treatment have a more advanced arterial age than chronological age. This could explain why what is found for older subjects in the general population is observed even in younger treated hypertensive subjects.

There were some study limitations. Although the age range of this population was very large (19-91 years), the large majority of subjects were middle-aged men (52±11 years), with fewer individuals at the extremes. Therefore, it is possible that the lack of interaction between age and the role of SBP or DBP may not be valid in younger (<40 years) or older (>65 years) subjects. Another limitation is that we included only men. In previous studies that included both normotensive and hypertensive sub-

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**Table 2:** Table showing the number of deaths per 10,000 person-years for different SBP and DBP groups.

<table>
<thead>
<tr>
<th>SBP Group</th>
<th>DBP Group</th>
<th>CVD Mortality Rate</th>
<th>CHD Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;140 mm Hg (n=881)</td>
<td>&lt;90 mm Hg (n=1195)</td>
<td>0.5</td>
<td>0.3</td>
</tr>
<tr>
<td>140-159 mm Hg (n=2097)</td>
<td>90-99 mm Hg (n=1679)</td>
<td>0.7</td>
<td>0.4</td>
</tr>
<tr>
<td>≥160 mm Hg (n=1736)</td>
<td>≥100 mm Hg (n=1840)</td>
<td>1.0</td>
<td>0.6</td>
</tr>
</tbody>
</table>

**Figure:** Graphs showing age-adjusted cardiovascular disease (CVD) and coronary heart disease (CHD) mortality rates according to systolic blood pressure (SBP) (A) and diastolic blood pressure (DBP) (B) levels in men treated for hypertension.
jcts of the IPC Center cohort, the predominant roles of SBP and pulse pressure were observed in men, but not in women. Due to the lower CVD mortality rates in women, especially CHD mortality rates, the lack of statistical power prevented us from carrying out the same analyses in women.

It is also important to note that single-visit BP measurements could overestimate the percentage of uncontrolled treated subjects. However, this study clearly shows that under these circumstances, similar to those faced by physicians in the follow-up of their treated hypertensive patients, an SBP value over 140 mm Hg is strongly associated with high CVD mortality in treated men.

The lack of predictive value of DBP may be because patients with high DBP levels were subsequently more likely to receive more aggressive treatment than those with high SBP. This was especially true in the 1980s when isolated systolic hypertension was not uniformly treated. Although this hypothesis could partially explain the observed lack of predictive value of DBP, all recent studies show that the high DBP values were not normalized in a majority of treated hypertensive subjects. Therefore, it cannot be assumed that subjects with high DBP levels were subsequently normalized and that this was the reason for the lack of association between DBP levels and CVD and CHD mortality.

In conclusion, in treated hypertensive men, SBP is a good predictor of CVD and CHD risk. Diastolic BP, which remains the main criterion used by most physicians in determining drug efficacy, appears to be of little value in determining CVD risk. Our results show that in clinical practice, a well-controlled SBP (<140 mm Hg) should be the goal of antihypertensive treatment.

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Corresponding author and reprints: Athanase Benetos, MD, PhD, Centre d’Investigations Préventives et Cliniques, 6/14 rue de la Pérouse, 75116 Paris, France (e-mail: benetos@ipc.asso.fr).

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