Increased Pulse Pressure and Risk of Heart Failure in the Elderly

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The prevalence of congestive heart failure (CHF), an important cause of morbidity and mortality, will increase with the aging of the US population. The incidence of CHF doubles in each decade after the age of 45 years, and CHF is the leading hospital diagnosis in those older than 65 years.1 Risk factors such as hypertension have been well described.2 However, data are sparse regarding other risk factors for CHF specifically in the elderly.

Pulse pressure (PP), measured as the difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP), rises markedly after the fifth decade due to arterial stiffening with age, which results in a progressive rise in SBP and fall in DBP as the elastic capacity of the aorta diminishes.3-6 Arterial stiffness increases afterload7,8 and myocardial oxygen demand,9 impairs ventricular relaxation,10 and causes subendocardial ischemia in the presence11,12 or absence13 of coronary artery stenoses. These effects may increase the susceptibility of the aging heart to ischemia and ventricular dysfunction.14

Arterial stiffness has been shown to be a risk factor for cardiovascular disease (CVD). In prospective cohorts, PP is associated with risk of myocardial infarction (MI)15-16 and CVD mortality17 in normotensive and hypertensive populations, as well as reinfarction and total mortality after MI in patients with left ventricular dysfunction.18 Measures of arterial stiffness are strongly correlated with left ventricular hypertrophy.19,20 However, none of these studies examined the possible relationship of arterial stiffness with CHF.

We addressed the hypothesis that increased arterial stiffness may contribute to CHF in the elderly, by prospectively examining whether PP is an independent predictor of CHF risk in a community-based elderly cohort.

METHODS

Study Population

The study population consisted of participants in the East Boston Senior Health Project, East Boston, Mass, 1 of the 4 centers of the National Institute on Aging’s Established Populations for Epidemiologic Studies of the Elderly. A community census, conducted in 1982-1983, identified 4497 East Boston residents aged 65 years or older, of whom 3809 (85%) participated in baseline interviews and in-home evaluations. Follow-up in-home examinations were done in 1985-1986 and were done in 1988-1989, and telephone interviews were done in 1997.

Context Arterial stiffness increases with age. Thus, pulse pressure, an index of arterial stiffening, may predict congestive heart failure (CHF) in the elderly.

Objective To study prospectively the association between pulse pressure and risk of CHF.

Design Prospective cohort study.

Setting The community-based East Boston Senior Health Project, East Boston, Mass.

Patients A total of 1621 men and women (mean [SD] age, 77.9 [5.0] years) free of CHF who had blood pressure measurements taken in 1988-1989 and were followed up for 3.8 years.

Main Outcome Measure Incidence of CHF as ascertained by hospital discharge diagnosis (n = 208) and death certificates (n = 13).

Results After controlling for age, sex, mean arterial pressure, history of coronary heart disease, diabetes mellitus, atrial fibrillation, valvular heart disease, and antihypertensive medication use, pulse pressure was an independent predictor of CHF. For each 10-mm Hg elevation in pulse pressure, there was a 14% increase in risk of CHF (95% confidence interval, 1.05-1.24; P = .003). Those in the highest tertile of pulse pressure (>67 mm Hg) had a 55% increased risk of CHF (P = .02) compared with those in the lowest (<54 mm Hg). Pulse pressure was more predictive than systolic blood pressure alone and was independent of diastolic blood pressure.

Conclusion Pulse pressure, an easily measurable correlate of pulsatile hemodynamic load, is an independent predictor of risk of CHF in this elderly cohort.
were completed in the other years. The Medicare Provider Analysis and Review files from the Health Care Financing Administration (HCFA) provided information on all hospital admissions and 5 discharge diagnoses per participant admission from 1985 to 1992. Subjects with unavailable HCFA data (n = 19) were excluded. Vital status through December 1992 was determined through local death notices, records of the East Boston Neighborhood Health Center, and yearly linkage with the National Death Index. For the entire cohort, mortality follow-up was complete, and 99.2% of death certificates were obtained and coded by a single nosologist using the International Classification of Diseases, Ninth Revision (ICD-9).

In 1988-1989, 2348 subjects provided follow-up data either in person or through a proxy, 208 refused or were lost to follow-up, and 1253 had died. Of these, 1810 had blood pressure (BP) measurements. To define a cohort free of evidence of prior CHF, we excluded those with a prior HCFA diagnosis of CHF (n = 138) and those without a prior HCFA diagnosis of CHF but who were taking combinations of digoxin and loop diuretics (n = 25) or digoxin, loop diuretics, and angiotensin-converting enzyme inhibitors (n = 6). We also excluded 20 subjects with missing medication data. This resulted in a cohort of 1621 subjects without known CHF who had complete BP measurements and were followed up prospectively for 3.8 years (median).

**Exposure Variables**

Using a standard mercury sphygmomanometer, trained interviewers took 3 BP measurements at 30-second intervals after the participant had been seated for at least 5 minutes, using the protocol from the Hypertension Detection and Follow-up Program. Details of quality control of BP measurements have been published previously. The averages of 3 SBP and DBP measurements were used. Mean arterial pressure (MAP) was defined as (SBP + (DBP x 2))/3. Pulse pressure was defined as SBP – DBP.

We assessed demographic characteristics, medical history, functional abilities, and health habits. Interviewers suspected all medication containers for prescription and nonprescription drugs taken in the prior 2 weeks. Drugs were coded using the Drug Product Information Coding System.

Hypertension was defined as any of the following: self-report of high BP, previous HCFA diagnosis, use of antihypertensive medications, or an SBP greater than 160 mm Hg or a DBP greater than 90 mm Hg on examination. Diabetes mellitus (DM) was defined by self-report, use of insulin or oral hypoglycemic agents, or previous HCFA diagnosis. Prevalent CHD (ICD-9 code 410-414, prior percutaneous transluminal coronary angioplasty or coronary artery bypass grafting), valvular heart disease, and atrial fibrillation (AF) were defined by HCFA diagnoses. Smoking status (current, past, never) and alcohol use (none in past year, none in past month, <15 g/d, ≥15 g/d) were categorized based on self-report.

**Outcome Measures**

In a median of 3.8 years of follow-up, 221 subjects reached the end point of first hospitalization with CHF, defined by ICD-9 code 428 in any of 5 fields for any hospitalization after the subject’s 1988-1989 evaluation (n = 208) or death with CHF on the death certificate but no prior HCFA diagnosis of CHF (n = 13).

**Statistical Methods**

We used analysis of variance for continuous variables and tested for linear trends for categorical variables to examine study population characteristics stratified by tertiles of PP. Linear regression models were used to determine predictors of PP. Participants contributed follow-up time from the date of their baseline examination in 1988-1989 to the date of first hospitalization with CHF, death, or end of follow-up, whichever came first. Hazard ratios (HRs) were calculated using Cox proportional hazards models, controlling for age, to evaluate the association of BP parameters with CHF risk. Continuous variables were examined in categories chosen a priori to determine if they had a linear relationship with the outcome. The categories for SBP (<120 [referent], 120-139, 140-159, or ≥160 mm Hg) and DBP (<70, 70-79 [referent], 80-89, or ≥90 mm Hg) were chosen to reflect clinically relevant values. Because clinically relevant values are unknown for PP, tertiles of less than 54 (referent), 54 to 67, and greater than 67 mm Hg were chosen. Tests for trend were performed by assigning an ordinal variable for each category of the BP parameter and modeling this as a continuous variable in separate Cox proportional hazards models. If a significant linear relationship was found, the variable was used as a continuous variable. Other clinical predictors of CHF risk and potential founders were tested individually for associations with CHF risk in separate Cox models, after controlling for age. The relationships between BP parameters and risk of CHF were examined in multivariate Cox proportional hazards model, controlling for age, sex, CHD, and significant univariate predictors of CHF risk. For each HR, 2-sided P values and 95% confidence intervals (CIs) were calculated.

In analyses assessing the relationship of PP with other CVD events and CHF, we excluded participants with prior CHD at baseline (n = 167), leaving a total of 1454 subjects of whom 190 developed CHF, 118 had nonfatal or fatal MIs (defined as the primary or underlying cause of death on the death certificate, without prior known CHD), and 68 died with CVD attributed as the immediate or underlying cause of death. In multivariate proportional hazards models, we examined the relationship of PP with MI and total CVD (259 first events of MI, CHF, or CVD death). To assess whether the association between PP and CHF risk is mediated through CHD, subjects with MI occurring before or after the incident CHF hospitalization were excluded, or MI was controlled for.

Effect modification by sex, DM, and CHF risk factor status (defined as having any of the following: DM, CHD, AF, valvular heart disease, or antihypertensive medication use) was also examined.

**RESULTS**

**Clinical Characteristics**

The study population (n = 1621) was elderly (mean [SD] age, 77.9 [5.0] years)
and predominantly female (66.1%). Prevalent conditions at baseline included hypertension (54.6%), DM (19.3%), CHD (10.3%), AF (2.8%), and valvular heart disease (1.8%). Nearly half (45.6%) were taking antihypertensive medications, and 12% were current smokers. Mean (SD) BP parameters included SBP of 137 (19), DBP of 75 (10), MAP of 96 (11), and PP of 62 (17) mm Hg.

Age, MAP, presence of DM, hypertension, vascular heart disease, and AF were not related to PP. In a multivariate regression model, independent predictors of PP included age, MAP, and DM (all P < .001), antihypertensive medication use (P = .002), and valvular heart disease (P = .008). Using Spearman rank order correlation coefficients (r), PP was correlated with MAP (r = 0.41; P < .001) and SBP (r = 0.83; P < .001); the correlation of PP with DBP was small although statistically significant (r = −0.07; P = .004). Systolic blood pressure and MAP were also correlated (r = 0.44; P < .001).

**BP and CHF Risk**

Age was a strong univariate predictor of CHF risk (HR, 1.44/5 y; 95% CI, 1.28-1.61; P < .001). After controlling for age (Figure), systolic blood pressure had a graded relationship with increasing risk of CHF (P for trend, < .001) with a 12% increase in risk for every 10-mm Hg rise in SBP (95% CI, 1.05-1.20). Diastolic blood pressure, however, tended to have a U-shaped relationship with CHF risk. Compared with subjects whose DBP ranged from 70 to 79 mm Hg, those with DBPs less than 70, 80 to 89, and greater than 90 mm Hg had HRs of 1.60 (95% CI, 1.17-2.20; P = .003), 1.10 (95% CI, 0.76-1.60; P = .60), and 1.69 (95% CI, 1.05-2.78; P = .04), respectively. Pulse pressure had a significant linear relationship with CHF risk (P for trend, < .001), with an age-adjusted 17% increase in risk for every 10-mm Hg rise in PP (95% CI, 1.09-1.26). Subjects in the highest tertile of PP had a 75% increase in risk of CHF (95% CI, 1.25-2.45; P = .001) compared with those in the lowest. The relationship of PP to CHF risk was not materially altered by the presence (to control for the steady component of BP)* or absence of MAP in the model. MAP had no association with CHF in these data.

After adjusting for age, use of antihypertensive medications, DM, valvular heart disease, and AF were significant predictors of CHF (Table 2). Smoking, and alcohol use were not associated with CHF risk. Coronary heart disease was included as a covariate in the multivariate models with the age-adjusted association of borderline significance (P = .07).

In a multivariate proportional hazards model controlling for age, sex, MAP, CHD, and significant univariate predictors of CHF risk (DM, AF, valvular disease, antihypertensive medication use), PP was a significant independent predictor of CHF (P for trend, .003) (Table 2). Every 10-mm Hg rise in PP was associated with a 14% increase in risk of CHF (95% CI, 1.05-1.24). Subjects in the highest tertile (PP > 67 mm Hg) had a 55% greater risk of CHF (P = .02) compared with those in the lowest (PP < 54 mm Hg).

We addressed the relative importance of PP, SBP, and DBP in predicting CHF risk in multivariate proportional hazards models examining BP parameters alone and in combination (Table 3). When PP and SBP were examined in the same model, PP remained a significant independent predictor of CHF risk (HR per 10-mm Hg increase in PP, 1.16; 95% CI, 1.00-1.35; P = .049), while SBP did not.

### Table 1. Clinical Characteristics Stratified by Pulse Pressure Tertile

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>&lt; 54 (n = 534)</th>
<th>54-67 (n = 547)</th>
<th>&gt; 67 (n = 540)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean ± SD, y</td>
<td>77.3 ± 4.8</td>
<td>77.9 ± 5.2</td>
<td>78.6 ± 4.9</td>
<td>&lt; .001</td>
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<tr>
<td>Mean arterial pressure, mean ± SD, mm Hg</td>
<td>90.9 ± 9.0</td>
<td>95.3 ± 10.1</td>
<td>101.5 ± 11.7</td>
<td>&lt; .001</td>
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<tr>
<td>Male sex</td>
<td>33.0</td>
<td>37.5</td>
<td>31.3</td>
<td>.51</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>13.3</td>
<td>17.2</td>
<td>27.2</td>
<td>.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>44.0</td>
<td>49.5</td>
<td>70.2</td>
<td>.001</td>
</tr>
<tr>
<td>Antihypertensive medication</td>
<td>38.8</td>
<td>44.6</td>
<td>53.3</td>
<td>.001</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>9.2</td>
<td>10.8</td>
<td>10.9</td>
<td>.34</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>2.4</td>
<td>2.2</td>
<td>3.7</td>
<td>.20</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>1.1</td>
<td>1.5</td>
<td>2.8</td>
<td>.04</td>
</tr>
</tbody>
</table>

*Data are presented as percentages unless otherwise indicated.

†P Values were obtained for analysis of variance of continuous variables and test for linear trend for categorical variables.

### Figure. Age-Adjusted Blood Pressure Parameters and Congestive Heart Failure Risk

Univariate relationships of age-adjusted systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse pressure (PP) to congestive heart failure risk.
(HR/10 mm Hg, 0.97; 95% CI, 0.84-1.11; P = .63), suggesting that PP provides additional predictive information independent of SBP alone. In a separate multivariate model of PP and DBP, the effect of PP (HR/10 mm Hg, 1.12; 95% CI, 1.04-1.20; P = .003) on CHF risk was independent of DBP. In this model, a DBP of less than 70 mm Hg (HR, 1.54; 95% CI, 1.12-2.12; P = .007), which is an additional correlate with large artery stiffness, was also independently associated with CHF risk. In these analyses, PP was independent of SBP and DBP, and was additive to DBP in predicting CHF risk.

TABLE 2. Proportional Hazards Models of Pulse Pressure and Congestive Heart Failure Risk

<table>
<thead>
<tr>
<th>Variable</th>
<th>Age (95% CI)</th>
<th>Multivariate Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, per 5 y</td>
<td>1.44 (1.28-1.61)</td>
<td>1.37 (1.22-1.54)</td>
</tr>
<tr>
<td>PP, per 10 mm Hg</td>
<td>1.17 (1.09-1.26)</td>
<td>1.14 (1.05-1.24)</td>
</tr>
<tr>
<td>MAP, per 1 mm Hg</td>
<td>1.01 (1.00-1.02)</td>
<td>1.00 (0.98-1.01)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.68 (1.40-2.50)</td>
<td>1.66 (1.24-2.23)</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>3.27 (1.73-6.16)</td>
<td>2.65 (1.34-5.26)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>3.02 (1.85-4.93)</td>
<td>2.84 (1.69-4.77)</td>
</tr>
<tr>
<td>Antihypertensive medication</td>
<td>1.60 (1.22-2.08)</td>
<td>1.45 (1.10-1.91)</td>
</tr>
<tr>
<td>Male sex</td>
<td>1.24 (0.94-1.63)</td>
<td>1.34 (1.01-1.76)</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>1.42 (0.97-2.08)</td>
<td>0.88 (0.57-1.36)</td>
</tr>
</tbody>
</table>

*HR indicates hazard ratio; CI, confidence interval; PP, pulse pressure; and MAP, mean arterial pressure.

Table 3. Multivariate Proportional Hazards Models of Blood Pressure Components and Risk of Congestive Heart Failure

<table>
<thead>
<tr>
<th>Variable</th>
<th>β Coefficient</th>
<th>HR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse pressure, per 10 mm Hg†</td>
<td>0.12846</td>
<td>1.14 (1.05-1.24)</td>
<td>.003</td>
</tr>
<tr>
<td>Systolic BP, per 10 mm Hg</td>
<td>0.0877</td>
<td>1.09 (1.02-1.16)</td>
<td>.01</td>
</tr>
<tr>
<td>Pulse pressure, per 10 mm Hg</td>
<td>0.15108</td>
<td>1.16 (1.00-1.35)</td>
<td>.049</td>
</tr>
<tr>
<td>Systolic BP, per 10 mm Hg</td>
<td>-0.03367</td>
<td>0.97 (0.84-1.11)</td>
<td>.63</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg‡&lt;70</td>
<td>0.46810</td>
<td>1.60 (1.16-2.19)</td>
<td>.004</td>
</tr>
<tr>
<td>80-89</td>
<td>0.13439</td>
<td>1.14 (0.79-1.66)</td>
<td>.48</td>
</tr>
<tr>
<td>≥90</td>
<td>0.51856</td>
<td>1.68 (1.02-2.76)</td>
<td>.04</td>
</tr>
<tr>
<td>Pulse pressure, per 10 mm Hg</td>
<td>0.11109</td>
<td>1.12 (1.04-1.20)</td>
<td>.003</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg‡&lt;70</td>
<td>0.43646</td>
<td>1.54 (1.12-2.12)</td>
<td>.007</td>
</tr>
<tr>
<td>80-89</td>
<td>0.13535</td>
<td>1.15 (0.79-1.66)</td>
<td>.48</td>
</tr>
<tr>
<td>≥90</td>
<td>0.50624</td>
<td>1.66 (1.01-2.73)</td>
<td>.046</td>
</tr>
</tbody>
</table>

*All models controlled for age, sex, diabetes mellitus, atrial fibrillation, coronary heart disease, valvular heart disease, and use of antihypertensive medications. HR indicates hazard ratio; CI, confidence interval; and BP, blood pressure.
†Controlled for mean arterial pressure and the above variables.
‡Models using diastolic blood pressure included 70 to 79 mm Hg as reference category.

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Pulse pressure reflects the complex interaction between intermittent cardiac ejection and the dynamic properties of the large arteries. The distensible, elastic aorta transiently stores part of the stroke volume during each systole and transmits this volume forward in diastole as its walls recoil. This limits end-systolic pressure and wall tension and maintains diastolic perfusion pressure. The progressive loss of aortic compliance with age, due to elastin fragmentation and increased collagen deposition, results in elevation in PP, from the associated rise in SBP and fall in DBP.

Several plausible mechanisms may explain an association between increased PP and CHF. Increased afterload and premature return of reflected waves in late systole impose a greater load on the ventricle, reducing ejection fraction and increasing myocardial oxygen demand. Diastolic relaxation is also impaired. Conduit vessel stiffness may increase vulnerability to ischemia as well. The increase in systolic load and myocardial work, coupled with the fall in diastolic coronary perfusion pressure, results in subendocardial ischemia with and without coronary artery stenoses.

Arterial stiffness is strongly correlated with left ventricular hypertrophy in normotensive and hypertensive subjects and with left ventricular mass in population-based cohorts. A plausible consequence of its excess hemodynamic load and a known risk factor for CHF and CVD events.

In addition to hemodynamic consequences, the association between elevated PP and CHF risk may in part be mediated by CHD. Arterial stiffness is correlated with atherosclerosis and may promote primary atheroma development, possibly mediated by greater cyclical wall stress. Several prospective studies in middle-aged populations support the hypothesis that higher PP is associated with increased CHD, but none addressed its relationship with CHF. In our data of the elderly, the association between PP and CHF was independent of CHD, suggesting that it may be mediated by other adverse effects of arterial stiffness on hemodynamics, systolic and diastolic function, and myocardial perfusion.

These findings have potential preventive and therapeutic implications regarding modifiability of arterial stiffness and perhaps risk of CHF. For example, angiotensin-converting enzyme inhibitors cause greater regression of left ventricular hypertrophy than other vasodilators despite lowering MAP to the same degree. This may be due to reduction in pulsatile load and premature wave reflection or modulation of renin-angiotensin system effects on arterial compliance. Nitrates and low-salt diets improve arterial distensibility. Adverse effects on coronary blood supply, including decreased diastolic perfusion pressure and a greater proportion of myocardial perfusion occurring in systole, may have implications for treatment of elderly patients with CHD, in whom hearts perfused by wide PP may be more sensitive to reductions in SBP and MAP. As recently described, increased pulsatile load during acute coronary occlusion resulted in a 3-fold decline in ejection fraction, marked fall in SBP, and doubling of ischemic mass. This may contribute to the greater severity of CHF and left ventricular dysfunction seen in the elderly after MI, independent of the severity of coronary stenosis.

Several issues regarding our study methods should be addressed. Brachial artery BPs by sphygmomanometry are less accurate in measuring central pressure than more invasive or technologically advanced methods. However, this type of BP measurement is easily obtained in a clinical setting, and in an elderly population, such as ours, peripheral and central PP are better correlated than in younger subjects. We also used the average of 3 BPs taken by Hypertension Detection and Follow-up Program protocol to increase the accuracy of our measurements. In other prospective studies, even a single, casual BP measurement yielded significant positive associations between PP and CVD events.

While we used HCFA diagnoses to identify end points, a validation study found that ICD-9 codes for CHF had sensitivities and positive predictive values, respectively, of 0.89 and 0.71 in any position, and 0.85 and 0.87 in the principal position. In a study of Medicare admissions in 1991 and 1992 in 4 states, the principal diagnosis of CHF was confirmed by clinical criteria in 95.7% of cases. When we restricted our analysis to CHF as the principal diagnosis, the magnitude of association was not materially altered, although the CI widened due to the smaller number of end points. Hospitalization is a reasonable but imperfect indicator of incident CHF. While most patients require hospitalization at some point in their disease course, this method is biased toward selection of sicker patients and will not identify nonhospitalized cases of CHF. A potential limitation is that some subjects may have had CHF at baseline. Given the natural history of CHF, with its poor prognosis and high rate of
hospitalization, we sought to address this by excluding subjects at baseline with a prior HCFA diagnosis in the preceding 3 years or with evidence of active drug therapy consistent with CHF treatment. Whether our findings apply to populations in middle age requires further study. The pathophysiology of arterial stiffness suggests that the association between PP and CHF risk may apply predominantly to the elderly. We do not have data about left ventricular function in these subjects but speculate that a link may exist between progressive arterial stiffness, with its adverse effects on ventricular relaxation, loading conditions, and myocardial perfusion, and the increase in diastolic heart failure observed in the elderly.

In summary, in this prospective study of a community-based elderly cohort, PP was independently and linearly associated with risk of CHF. Pulse pressure may be a clinically useful tool to identify elderly subjects at risk for CHF, who may benefit from treatment to improve arterial compliance. More research is needed to further examine the relationship of arterial compliance with its cardiovascular sequelae, as well as the possible benefits of therapies specifically designed to prevent or treat the vascular pathology associated with elevated pulse pressure.

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