THe progression from hypertension to congestive heart failure

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The Progression From Hypertension to Congestive Heart Failure

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Objectives.—To study the relative and population-attributable risks of hypertension for the development of congestive heart failure (CHF), to assess the time course of progression from hypertension to CHF, and to identify risk factors that contribute to the development of overt heart failure in hypertensive subjects.

Design.—Inception cohort study.

Setting.—General community.

Participants.—Original Framingham Heart Study and Framingham Offspring Study participants aged 40 to 89 years and free of CHF. To reflect more contemporary experience, the starting point of this study was January 1, 1970.

Exposure Measures.—Hypertension (blood pressure of at least 140 mm Hg systolic or 90 mm Hg diastolic or current use of medications for treatment of high blood pressure) and other potential CHF risk factors were assessed at periodic clinic examinations.

Outcome Measure.—The development of CHF.

Results.—A total of 5143 eligible subjects contributed 72 422 person-years of observation. During up to 20.1 years of follow-up (mean, 14.1 years), there were 392 new cases of heart failure; in 91% (357/392), hypertension antedated the development of heart failure. Adjusting for age and heart failure risk factors in proportional hazards regression models, the hazard for developing heart failure in hypertensive compared with normotensive subjects was about 2-fold in men and 3-fold in women. Multivariable analyses revealed that hypertension had a high population-attributable risk for CHF, accounting for 39% of cases in men and 59% in women. Among hypertensive subjects, myocardial infarction, diabetes, left ventricular hypertrophy, and valvular heart disease were predictive of increased risk for CHF in both sexes. Survival following the onset of hypertensive CHF was bleak; only 24% of men and 31% of women survived 5 years.

Conclusions.—Hypertension was the most common risk factor for CHF, and it contributed a large proportion of heart failure cases in this population-based sample. Preventive strategies directed toward earlier and more aggressive blood pressure control are likely to offer the greatest promise for reducing the incidence of CHF and its associated mortality.

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HYPERTENSION is a cardinal precursor of congestive heart failure (CHF).[1,2] Although several other precursors of CHF also have been identified, including myocardial infarction,[3,4] diabetes,[5] valvular heart disease,[6] left ventricular hypertrophy,[7,8] and cardiomyopathies,[9,10] hypertension is the most common condition antedating heart failure in the general population.[1,2,11] The population-attributable risk of hypertension for CHF, the time course of the progression from hypertension to CHF, and the relative contributions of several mechanisms by which hypertension contributes to the pathogenesis of CHF have not been fully elucidated.

Prior studies document the efficacy of hypertension treatment in reducing the incidence of CHF,[12-17] but these intervention trials provide little insight into the mechanisms by which hypertension predisposes to CHF or by which hypertension treatment prevents or delays the onset of overt heart failure.

The Framingham Heart Study offers an opportunity to examine the association between hypertension and CHF in a population-based setting with comprehensive, long-term follow-up. It permits an assessment of the natural history of the progression from hypertension to overt left ventricular dysfunction and an exploration of potential mechanisms influencing the association between hypertension and heart failure.

METHODS

Study Sample

The Framingham Heart Study was designed as a prospective, population-based investigation of cardiovascular disease prevalence, incidence, and precursors. Study design and recruitment procedures have been published.[18] Beginning in 1948, the study enrolled a total of 5209 men and women between the ages of 28 and 62 years to undergo comprehensive evaluations. Each examination included a medical history, physical examination, blood pressure measurements, 12-lead electrocardiogram (ECG), and laboratory tests.

Examination cycles were repeated every 2 years. Beginning in 1971, the Framingham Offspring Study enrolled 5124 male and female subjects who were offspring (and spouses of offspring) of original Framingham Heart Study subjects; the design and recruitment procedures have been published.[19] Offspring Study examinations were similar to those performed on the original Framingham Heart Study cohort. The second, third, and fourth Offspring Study cycles were conducted 8, 12, and 16 years, respectively, after the initial examination cycle. A subset of Offspring Study subjects (n=880) who were selected because of high-risk parent characteristics were deemed ineligible for this study because of possible selection bias. To reflect more contemporary clinical experience, the baseline for this investigation was the first routine examination attended after January 1, 1970. Subjects with evidence of CHF at baseline and subjects who were younger than 40 years or older than 89 years were excluded. The follow-up period was through examination 20 (roughly 18 years) for original Framingham Heart Study subjects and through examination 4 (roughly 16 years) for Offspring Study participants.

Clinical Data and Outcomes

At each clinic examination, seated systolic and diastolic blood pressures were measured twice by the examining physician using a mercury-column sphygmomanometer. Hypertension was defined as a systolic blood pressure of 140 mm Hg or more or a diastolic blood pressure of 90 mm Hg or more on the average of 2 physician measurements or current use of medications for treatment of high blood pressure.[20] Stage 1 hypertension was defined as systolic blood pressure of 140 to 159 mm Hg or diastolic blood pressure of 90 to 99 mm Hg in subjects not receiving antihypertensive treatment, and stage 2 or greater hypertension was defined as systolic blood pressure of 160 mm Hg or more, diastolic blood pressure of 100 mm Hg or more, or current use of antihypertensive treatment. Diabetes was defined on the basis of a fasting blood glucose level greater than 7.77 mmol/L (140 mg/dL), 2 random nonfasting levels greater than 11.10 mmol/L (200 mg/dL), or the use of insulin or an oral hypoglycemic agent. Valvular heart disease was defined as the presence of a systolic murmur of grade 3/6 intensity or greater, any diastolic murmur, or a palpable thrill.

Standard 12-lead ECGs obtained at each examination cycle were interpreted for the presence of myocardial infarction and left ventricular hypertrophy. Criteria for left ventricular hypertrophy have been reported.[21,22] Briefly, increased QRS voltage criteria included the following: R wave greater than 1.1 mV in aVL, R wave greater than or equal to 2.5 mV in left precordial leads, S wave greater than or equal to 2.5 mV in right precordium, sum of right precordial S wave plus left precordial R wave of 3.5 mV or more, sum of limb lead RI plus SIII of 2.5 mV or more, or R wave or S wave in aVF of 2.0 mV or more. Left ventricular hypertrophy was present when increased voltage was associated with major ST-T repolarization changes (strain pattern).

Following each examination cycle, assessments of suspected coronary heart disease and CHF events were undertaken. Outside medical records of participants who did not attend an examination were obtained and evaluated for evidence of interim events. All suspected interim morbid and fatal cardiovascular events were reviewed by a panel of 3 physicians who evaluated pertinent Framingham Heart Study clinic records, outside physician records, hospitalization records, and pathology reports.

Criteria for myocardial infarction and angina pectoris (including stable angina and coronary insufficiency–prolonged angina with documented ischemic ECG changes) have been reported.[23] Recognized myocardial infarction was determined on the basis of hospitalization records documenting the presence at least 2 of the following findings: (1) new pathological Q waves or loss of R waves on review of serial ECGs; (2) elevation of cardiac enzymes; and (3) symptoms consistent with myocardial infarction. An unrecognized myocardial infarction was present when an ECG (obtained at a clinic visit or from an outside physician or hospitalization) revealed serial changes consisting of new pathological Q waves or loss of R waves in the absence of clinical recognition of an infarction.

The Framingham Heart Study has consistently used a set of clinical criteria for the diagnosis of CHF.[24] This diagnosis is established on the basis of the simultaneous presence of at least 2 major criteria or 1 major plus 2 minor criteria. Minor criteria were considered only if their presence could not be attributed to another disease process. Major criteria included paroxysmal nocturnal dyspnea, jugular venous distention, pulmonary rales, increasing heart size on chest x-ray film, acute pulmonary edema, third heart sound, central venous pressure of at least 16 cm H_2O, hepatojugular reflux, weight loss of 4.5 kg or more in response to diuretics, and autopsy evidence of pulmonary edema, visceral congestion, or cardiomegaly. Minor criteria included bilateral ankle edema, nocturnal dyspnea, dyspnea on ordinary exertion, hepatomegaly, pleural effusion, decrease in vital capacity by one third from prior maximum recorded value, and heart rate of at least 120 beats per minute.

Statistical Methods

The relations of hypertension and other risk factors (myocardial infarction, angina pectoris, diabetes, left ventricular hypertrophy, and valvular heart disease) to the incidence of CHF were examined using sex-specific proportional hazards regression models.[25] Subjects were followed until death, development of CHF, or date of last contact. Analyses were stratified by 10-year age groups. Two separate proportional hazards models were used to assess the impact of hypertension on risk for CHF. A static model assigned hypertension and risk factor status at the time of baseline examination (the first examination attended after January 1, 1970). Second, a dynamic model was developed with hypertension and other CHF risk factor status reassigned at each follow-up examination, with upward reclassification permitted only; a newly present risk factor was maintained thereafter. The dynamic model was used to calculate the population-attributable risk of hypertension for CHF. Population-attributable risk represents the percentage of heart failure cases that can be attributed to a risk factor given its prevalence and hazard ratio (HR); it assumes a causal relation between the risk factor and the occurrence of CHF. Population-attributable risk (expressed as a percentage) was defined as (100×prevalence×[hazard ratio–1])/(prevalence×[hazard ratio–1]+1), with prevalence calculated as the number of person-years of observation with a risk factor present divided by total observation time. Subjects with a risk factor prior to the baseline examination were classified as having it at baseline.

Cumulative incidence rates for CHF as a function of hypertension status at baseline were estimated using the Kaplan-Meier[25] method and plotted as a function of age and sex. Survival following the diagnosis of CHF was estimated similarly.

Descriptive data are presented as percentages or mean±SD. All analyses were conducted separately for men and women. All statistical modeling was performed with SAS software (SAS Institute Inc, Cary, NC) procedures LIFETEST[26] and PHREG.[27]

RESULTS

Study Sample

A total of 2334 men and 2809 women were eligible for this study; they contributed 72 422 person-years of follow-up. The characteristics of the study

Table 1.—Characteristics of Subjects at Baseline Examination

	Normotensive		Hypertensive	
	Men (n=1206)	Women (n=1435)	Men (n=1128)	Women (n=1374)
Person-years of follow-up	17324	22293	14327	18478
Age, mean±SD y*	55±10	55±10	60±10	64±10
Blood pressure, mean±SD mm Hg*				
Systolic	123±9	121±11	153±17	156±20
Diastolic	78±7	76±7	91±10	88±10
Use of antihypertensive drugs, %*	0	0	18.4	24.8
Myocardial infarction, %†	5.6	0.6	8.3	2.8
Angina pectoris, %†	6.0	2.8	9.8	9.5
Diabetes, %†	4.6	1.3	7.1	8.0
Left ventricular hypertrophy, %†	1.4	0.6	5.7	4.7
Valvular heart disease, %†	2.7	3.8	5.4	8.2

*At the baseline examination.
†At or prior to the baseline examination.

Table 2.—Characteristics of Hypertensive Congestive Heart Failure Cases at Examination Prior to Onset of Heart Failure

	Men (n=165)	Women (n=192)
Age, mean±SD y*	73±9	78±9
Blood pressure, mean±SD mm Hg*		
Systolic	149±21	150±22
Diastolic	81±13	77±12
Stage 1 hypertension, %†	24	18
Stage 2 or greater hypertension, %†	76	82
Myocardial infarction %†	52	34
Angina pectoris, %†	36	35
Angina pectoris without myocardial infarction, %†	12	21
Diabetes, %†	24	28
Left ventricular hypertrophy, %†	21	23
Valvular heart disease, %†	24	33

*At the clinic examination preceding congestive heart failure.
†At any time preceding congestive heart failure.

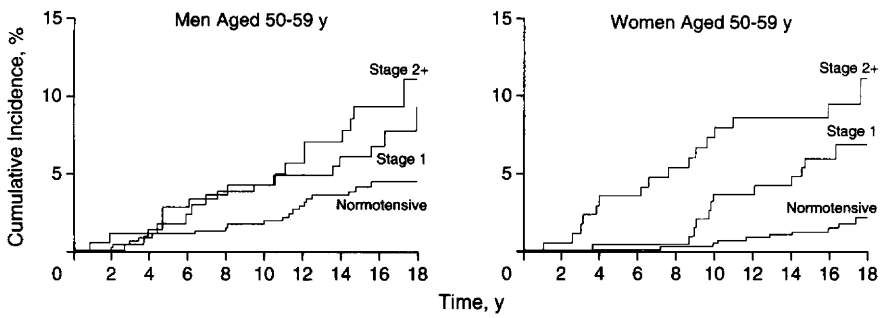

Figure 1.—Cumulative incidence of congestive heart failure in men and women aged 50 to 59 years according to hypertension status at baseline. Stage 1 hypertension was defined as systolic blood pressure of 140 to 159 mm Hg or diastolic blood pressure of 90 to 99 mm Hg in subjects not receiving antihypertensive treatment, and stage 2 or greater hypertension (stage 2+) was defined as systolic blood pressure greater than 160 mm Hg, diastolic blood pressure greater than 100 mm Hg, or the current use of antihypertensive treatment.

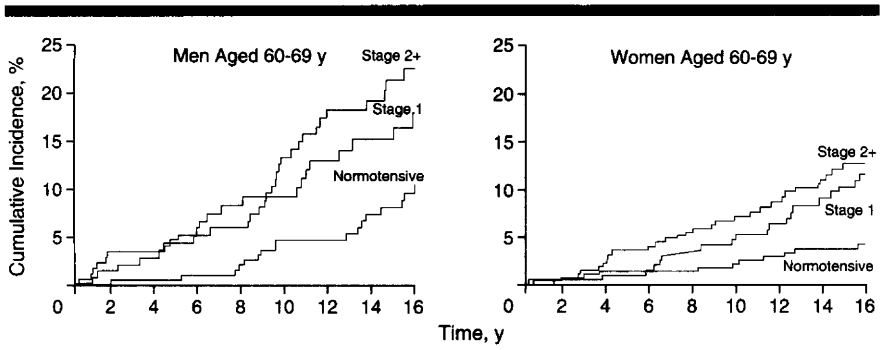

Figure 2.—Cumulative incidence of congestive heart failure in men and women aged 60 to 69 years according to hypertension status at baseline. Stage 1 hypertension was defined as systolic blood pressure of 140 to 159 mm Hg or diastolic blood pressure of 90 to 99 mm Hg in subjects not receiving antihypertensive treatment, and stage 2 or greater hypertension (stage 2+) was defined as systolic blood pressure greater than 160 mm Hg, diastolic blood pressure greater than 100 mm Hg, or the current use of antihypertensive treatment.

sample at baseline are presented in Table 1; criteria for hypertension were fulfilled in 2502 subjects (49%). During up to 20.1 years of follow-up (mean±SD, 14.1±0.7 years), 392 subjects developed CHF. Hypertension predated CHF in 91% of cases (357/392).

Incidence of CHF According to Hypertensive Status

Antecedent clinical characteristics of hypertensive heart failure cases are presented in Table 2. The mean age at diagnosis of CHF among hypertensive cases was 73 years in men and 78 years in women; 46% of hypertensive heart failure cases were men. Among hypertensive subjects who developed heart failure, 76% of men and 82% of women had stage 2 or greater hypertension or were receiving pharmacological treatment for high blood pressure. Coronary heart disease antedated the development of CHF in 64% of men and 55% of women with hypertension. Electrocardiographic evidence of left ventricular hypertrophy was present in 21% of hypertensive cases in men and in 23% of women.

Figures 1, 2, and 3 depict age- and sex-specific cumulative incidence of CHF according to hypertension status and severity at the baseline examination. The incidence of CHF was greater at increasing blood pressure levels and increased as a function of age and duration of follow-up.

Hypertension and Risk of CHF

Hypertension was associated with increased risk for developing CHF. Based on classification of hypertension and risk factor status at the baseline examination (static model), after age adjustment, subjects with hypertension had a 2- to 3-fold risk for CHF compared with normotensive subjects (in men, HR=2.04, 95% confidence interval [CI], 1.50-2.78; in women, HR=3.21, 95% CI, 2.20-4.67). Hypertension remained a strong risk factor after additional adjustment for angina pectoris, myocardial infarction, diabetes, left ventricular hypertrophy, and valvular heart disease (in men, HR=1.84, 95% CI, 1.35-2.51; in women, HR=2.60, 95% CI, 1.77-3.81).

When hypertension and other CHF risk factors were defined as time-dependent variables (dynamic model), the HRs were greater than in the static model. This is because many subjects who were normotensive at the baseline examination progressed to hypertension

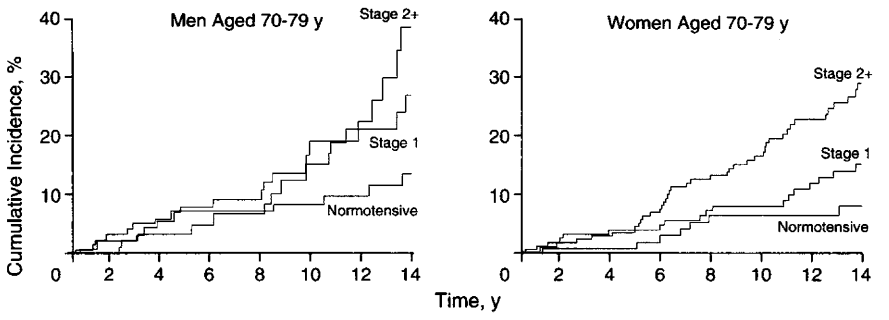

Figure 3.—Cumulative incidence of congestive heart failure in men and women aged 70 to 79 years according to hypertension status at baseline. Stage 1 hypertension was defined as systolic blood pressure of 140 to 159 mm Hg or diastolic blood pressure of 90 to 99 mm Hg in subjects not receiving antihypertensive treatment, and stage 2 or greater hypertension (stage 2+) was defined as systolic blood pressure greater than 160 mm Hg, diastolic blood pressure greater than 100 mm Hg, or the current use of antihypertensive treatment.

Table 3.—Population-Attributable Risk for the Development of Congestive Heart Failure*

Risk Factor	Sex	Age- and Risk Factor–Adjusted Hazard Ratio† (95% CI)	Prevalence, %‡	Population-Attributable Risk, %§
Hypertension	M	2.07 (1.34-3.20)	60	39
	F	3.35 (1.67-6.73)	62	59
Myocardial infarction	M	6.34 (4.61-8.72)	10	34
	F	6.01 (4.37-8.28)	3	13
Angina pectoris	M	1.43 (1.03-1.98)	11	5
	F	1.68 (1.23-2.30)	9	5
Diabetes	M	1.82 (1.28-2.58)	8	6
	F	3.73 (2.71-5.15)	5	12
Left ventricular hypertrophy	M	2.19 (1.49-3.21)	4	4
	F	2.85 (1.97-4.12)	3	5
Valvular heart disease	M	2.47 (1.70-3.60)	5	7
	F	2.13 (1.54-2.94)	8	8

*Based on dynamic model with reclassification of hypertension and risk factors at each follow-up examination. CI indicates confidence interval.

†Adjusted for angina pectoris, myocardial infarction, diabetes, left ventricular hypertrophy, and valvular heart disease.

‡Prevalence was the person-years of observation with a risk factor present divided by the total person-years of observation.

§Population-attributable risk was defined as the following: (100×prevalence×[hazard ratio−1])/(prevalence×[hazard ratio−1]+1).

prior to the onset of CHF (in men, age-adjusted HR=2.82, 95% CI, 1.85-4.32; in women, age-adjusted HR=5.62, 95% CI, 2.84-11.12; in men, age- and risk factor–adjusted HR=2.07, 95% CI, 1.34-3.20; in women, age- and risk factor–adjusted HR=3.35, 95% CI, 1.67-6.73).

Population-Attributable Risk for CHF

As shown in Table 3, among all eligible subjects, multivariable analyses using time-dependent modeling for hypertension and other risk factors revealed that hypertension carried the greatest population-attributable risk for the development of CHF of all risk factors considered (39% in men and 59% in women). Hypertension also had the highest prevalence of all risk factors in this sample (60% in men and 62% in women). Myocardial infarction, despite its lower prevalence, carried a high population-attributable risk, especially in men (34%).

Risk Factors for CHF Among Hypertensive Subjects

Among hypertensive subjects, the relations of the other risk factors to incidence of CHF were assessed in multivariable proportional hazards models (Table 4). Myocardial infarction had the greatest HR for CHF (5.54 in men and 5.99 in women). Angina pectoris (women only), diabetes, left ventricular hypertrophy, and valvular heart disease also increased the risk for CHF risk in hypertensive subjects.

Mortality Following the Development of CHF Among Hypertensive Subjects

Survival following the diagnosis of CHF was bleak; median survival following the diagnosis of heart failure in hypertensive subjects was 1.37 years in men and 2.48 years in women. At 5 years of follow-up, 76% of men and 69% of women were dead.

Table 4.—Risk Factors for Congestive Heart Failure Among Hypertensive Subjects*

Risk Factor	Sex	Age- and Risk Factor–Adjusted Hazard Ratio† (95% CI)
Myocardial infarction	M	5.54 (3.96-7.77)
	F	5.99 (4.33-8.27)
Angina pectoris	M	1.35 (0.95-1.92)
	F	1.71 (1.25-2.34)
Diabetes	M	1.78 (1.23-2.59)
	F	3.57 (2.59-4.94)
Left ventricular hypertrophy	M	1.97 (1.31-2.96)
	F	2.80 (1.93-4.05)
Valvular heart disease	M	2.40 (1.62-3.56)
	F	1.96 (1.41-2.73)

*Based on 165 congestive heart failure events in 1707 men and 192 events in 2118 women with hypertension prior to congestive heart failure. Based on dynamic model with reclassification of hypertension and risk factors at each follow-up examination. CI indicates confidence interval.

†Adjusted for angina pectoris, myocardial infarction, diabetes, left ventricular hypertrophy, and valvular heart disease.

COMMENT

This investigation used long-term follow-up of a carefully monitored cohort to study the relative risk and population-attributable risk of hypertension for the development of CHF and the time course of progression to overt heart failure as a function of hypertension status. We also examined the contributions of other risk factors to CHF incidence in hypertensive subjects. Hypertension antedated the development of CHF in 91% of cases. Hypertension was associated with a 2- to 3-fold risk for the development of CHF after adjusting for age and several other risk factors. Multivariable analyses revealed that hypertension had a high population-attributable risk for CHF, accounting for 39% of cases in men and 59% in women. Among hypertensive men and women, myocardial infarction, diabetes, left ventricular hypertrophy, and valvular heart disease were predictive of increased risk for CHF. These findings provide insight into the pathogenesis of hypertensive heart disease.

Hypertension and Risk for CHF

Hypertensive men and women had a substantially greater risk for the development of CHF than their normotensive counterparts. Results were consistent between a long-term (static) model that used hypertension status and other CHF risk factor status at baseline without opportunity for reclassification and a dynamic model that captured subjects with hypertension and other risk factors not present at baseline that developed in the course of follow-up.

Our results confirm and extend earlier findings from Framingham. Nearly a quarter century ago, the Framingham Heart Study reported that hypertensive subjects were more likely to develop heart failure than those who were

normotensive.[1] That study was based on 142 cases of CHF detected during the first 16 years of follow-up in Framingham (through the mid-1960s). In contrast, the present report is based on nearly 3 times as many cases of CHF that developed in a more contemporary era. Unlike the earlier report,[1] this investigation uses multivariable proportional hazards modeling (adjusting for age, angina pectoris, myocardial infarction, diabetes mellitus, left ventricular hypertrophy, and valvular heart disease) to determine the relative and population-attributable risks of hypertension for CHF. Kaplan-Meier plots of cumulative CHF incidence as a function of age and hypertension severity are provided. Additionally, newer definitions for hypertension are used and both static and dynamic models of hypertension are assessed. Finally, risk factors for CHF among hypertensive men and women are reported.

Risk Factors for CHF Among Hypertensive Subjects

Myocardial infarction is an important contributor to the pathogenesis of CHF.[3,28] In the present study, myocardial infarction was present in 52% of hypertensive men and 34% of hypertensive women with CHF; it was associated with a 5- to 6-fold risk for CHF after adjusting for age and other risk factors.

Left ventricular hypertrophy has been well documented as a precursor of CHF.[7,8,29] In the present study, the increased risk for CHF in hypertensive subjects with ECG evidence of left ventricular hypertrophy persisted even after adjusting for the other CHF precursors included in the multivariable analyses (HR=1.97 in men and HR=2.80 in women).

Diabetes also emerged as an important precursor of CHF with greater relative risk in women than in men. Earlier studies also documented increased risk for the development of CHF in diabetic patients,[5,30] but the mechanisms responsible for the increased risk are not fully understood. Increased myocardial fibrosis may play a role in the pathogenesis of CHF in diabetic hypertensive patients.[31] Also, there is growing evidence for the existence of a distinct diabetic cardiomyopathy,[32] which might contribute to the association of diabetes with increased risk for CHF.

Our study documented an important role of valvular heart disease in the development of CHF. Preexisting valvular heart disease was detected in about 30% of our hypertensive CHF cases. In hypertensive subjects, the presence of valvular heart disease was associated with a 2-fold hazard for CHF after adjusting for age and other risk factors. This finding is consistent with the clinical consequences of a variety of valvular disorders.[6]

Systolic vs Diastolic Left Ventricular Dysfunction in Hypertension

Congestive heart failure can result from systolic or diastolic left ventricular dysfunction. Overt heart failure resulting from diastolic left ventricular dysfunction may be clinically indistinguishable from that resulting from systolic dysfunction.[33] It has been estimated that this entity is observed in 30% to 50% of adult cases of CHF.[34-42]

Myocardial infarction is a principal cause of systolic left ventricular dysfunction[3,43-46]; however, fewer than half our hypertensive CHF cases had a history of antecedent myocardial infarction. As such, the findings of this study support a potentially important role for diastolic dysfunction in the pathogenesis of hypertensive heart failure. Our study also documented important contributions of diabetes mellitus, left ventricular hypertrophy, and valvular heart disease to the pathogenesis of hypertensive CHF. These CHF precursors may contribute to CHF in individuals with normal left ventricular systolic function.

Study Strengths and Limitations

The Framingham Heart Study provides an opportunity to study a large sample of hypertensive subjects in a setting in which referral bias is intrinsically low; hence, the natural history of the progression from hypertension to CHF can be assessed more thoroughly than in clinical series or intervention trials. The large sample size permitted analyses of the association of hypertension with CHF after adjustment for multiple risk factors. This is one of the largest observational studies of its kind: 5143 subjects with 72 422 person-years of observation, during which time 2502 hypertensive subjects contributed 357 cases of heart failure. Unlike some prior studies, the availability of longitudinal blood pressure data permitted the identification of hypertension in subjects who were normotensive at baseline. Additionally, with comparable numbers of men and women, we were able to separately consider the contributions of hypertension to incidence of CHF in each sex.

Since the study sample was overwhelmingly white, the conclusions about the relative contributions of hypertension to CHF may not apply to other racial or ethnic groups. The heart failure criteria used by the Framingham Study are clinical; many of the cases were diagnosed prior to the introduction of echocardiography in our study and, hence, the diagnosis of CHF is subject to misclassification.

Preventive Implications

Our data satisfy several criteria for a causal role[47,48] of hypertension in the pathogenesis of CHF. The biological plausibility (of elevated blood pressure causing CHF), the strength of the association (2- to 3-fold hazard), its persistence after adjusting for potential confounders (results of multivariable analyses), the demonstration of a temporal sequence (Kaplan-Meier plots), the presence of a dose-response relation (Kaplan-Meier plots), and the consistency of these findings with an earlier investigation[1] and with other observations[2] all point to the causal nature of the observed association. Further support of causality is provided by major hypertension trials, which have documented that treatment of hypertension[12-17] can dramatically reduce risk for CHF. The results of a recent meta-analysis suggesting that hypertension treatment in the elderly can reduce CHF incidence by 47%[17] are in close agreement with the population-attributable risk of hypertension for the development of CHF in our subjects (39% in men and 59% in women).

Although clinical trials have documented that several pharmacological interventions can improve survival in subjects with CHF,[3,43,44,46,49,50] the prognosis of this condition remains dismal.[7,51-55] The greatest opportunity for reducing the incidence of CHF and the excess mortality it carries is through preventive strategies directed toward earlier detection and more aggressive management of hypertension and other CHF risk factors. The present investigation provides additional rationale for a strategy of primary prevention of CHF.

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