Blood Pressure in Adulthood and Life Expectancy With Cardiovascular Disease in Men and Women: Life Course Analysis

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Hypertension 2005;46;280-286; originally published online Jun 27, 2005;
DOI: 10.1161/01.HYP.0000173433.67426.9b
Hypertension is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0194-911X. Online ISSN: 1524-4563

Blood Pressure in Adulthood and Life Expectancy With Cardiovascular Disease in Men and Women

Life Course Analysis

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Abstract—Limited information exists about the consequences of hypertension during adulthood on residual life expectancy with cardiovascular disease. We aimed to analyze the life course of people with high blood pressure levels at age 50 in terms of total life expectancy and life expectancy with and without cardiovascular disease compared with normotensives. We constructed multistate life tables for cardiovascular disease, myocardial infarction, and stroke using data from 3128 participants of the Framingham Heart Study who had their 50th birthday while enrolled in the study. For the life table calculations, we used hazard ratios for 3 transitions (healthy to death, healthy to disease, and disease to death) by categories of blood pressure level and adjusted by age, sex, and confounders. Irrespective of sex, 50-year-old hypertensives compared with normotensives had a shorter life expectancy, a shorter life expectancy free of cardiovascular disease, myocardial infarction, and stroke, and a longer life expectancy lived with these diseases. Normotensive men (22% of men) survived 7.2 years (95% confidence interval, 5.6 to 9.0) longer without cardiovascular disease compared with hypertensives and spent 2.1 (0.9 to 3.4) fewer years of life with cardiovascular disease. Similar differences were observed in women. Compared with hypertensives, total life expectancy was 5.1 and 4.9 years longer for normotensive men and women, respectively. Increased blood pressure in adulthood is associated with large reductions in life expectancy and more years lived with cardiovascular disease. This effect is larger than estimated previously and affects both sexes similarly. Our findings underline the tremendous importance of preventing high blood pressure and its consequences in the population. **(*Hypertension*. 2005;46:280-286.)**

Key Words: blood pressure ■ cardiovascular diseases

Elevated blood pressure (BP) is a major modifiable risk factor for cardiovascular disease (CVD) and mortality.[1,2] Suboptimal BP (>115 mm Hg systolic BP [SBP]) is estimated to be responsible for 62% of cerebrovascular disease and 49% of coronary heart disease.[3] The relationship between BP and CVD risk is continuous and independent of other risk factors.[1,4,5] BP control has been shown to be effective in reducing CVD and mortality, although below expectations from observational evidence.[1,6,7] However, few studies have looked at the impact of BP on life expectancy (LE),[8,9] and none have evaluated its effects on LE with and without CVD. Therefore, whether improving the level of BP in the population will lead to more or fewer years lived with CVD remained uncertain.

The answer to this question might seem obvious, but examples from other risk factors such as smoking or obesity show that results can be counterintuitive. Obesity is associated with a shorter LE and an increase in LE with CVD. Therefore, control of obesity would lead to less cardiovascular morbidity (M. Pardo Silva, unpublished data, 2003). Smoking is also associated with shorter LE but with fewer years lived with CVD because smokers, on average, die younger because of other causes, not reaching older ages at which CVD rates are higher.[10] Therefore, reducing smoking will lead to an increase in total LE but also to more cardiovascular morbidity.

The objective of this study was to determine the impact of increased BP levels at age 50 (independent of BP-lowering treatment) on total LE and LE with and without CVD (or myocardial infarction [MI] or stroke). We constructed multistate life tables (MSLTs) using the mortality and CVD experience from 46 years of follow-up of the original Framingham Heart Study (FHS).

Methods

Study Base

The FHS is a cohort study involving 5209 respondents 28 through 62 years of age at enrollment, residing in Framingham, Mass, between

Received March 14, 2005; first decision April 8, 2005; revision accepted May 3, 2005.

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This article has been presented previously in an oral presentation in the American Heart Sessions 2003 (Orlando, Fla, November 2003), therefore, it has been published as an abstract in the abstract supplement of *Circulation* for the American Heart Sessions.

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Hypertension **is available at http://www.hypertensionaha.org** **DOI: 10.1161/01.HYP.0000173433.67426.9b**

1948 and 1951. Data on occurrence of CVD and mortality were gathered by standardized biennial examinations, regular surveillance of hospital admissions, death registries, and medical records over a follow-up period of up to 46 years.[11]

In the present investigation, we followed participants from age 50 years onward. The participants may have reached this age at any point during the follow-up of FHS. Therefore, start of follow-up in our study did not correspond to FHS enrollment. Eligible participants were those enrollees in FHS who were in the study at age 50, were free of CVD at that age, and had BP measured during ≥1 examination (2 measurements) in the 5 years previous to reaching that age (1364 men and 1764 women).

Baseline Assessments

BP, weight, and total serum cholesterol at age 50 were estimated from the information collected through the biennial examinations. We estimated risk factor status at age 50 from linear regression on age using all data from visits during the 5 years before this age. Height and physical activity level were estimated from the closest measurement available, favoring those taken at <50 years of age. Body mass index (BMI) was calculated as weight (kg)/height squared (m^2).[12] Smoking status was self-reported and classified in 3 categories: never-smoker, ever-smoker, and current smoker. We updated the information on smoking history at enrollment with the latest available information on smoking at <50 years of age. Level of education was assessed according to the highest degree achieved at FHS enrollment. Diabetes was defined as either a random blood glucose measurement ≥200 mg/dL during a regular examination at <50 years of age or as treatment with insulin or oral hypoglycemic agent.

BP Level Classification

Participants were classified in 3 groups on the basis of their BP levels and independent of BP-lowering treatment using the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7) criteria: group I <120 mm Hg SBP and <80 mm Hg diastolic BP (DBP), also called normal BP; group II 120 to 139 mm Hg SBP or 80 to 89 mm Hg DBP (called high-normal); and group III ≥140 mm Hg SBP/≥90 mm Hg DBP (called hypertension).[1]

Data Analysis

LEs with and without disease are the result of several incidences: the incidence of the disease, the mortality after the disease, and mortality without the occurrence of the disease. To calculate LE with and without disease, we constructed MSLTs for CVD, MI, and stroke. In these MSLTs, we described 3 different potential states: disease-free, prevalent (history of) disease, and death. The possible transitions from 1 state to the other were from disease-free to death or to prevalent disease, and from prevalent disease to death. No backflows were permitted, and we considered only the first entry into a state and not subsequent disease events.[13,14] To construct these MSLTs, different steps were followed.

First, we assessed the risk associated with the different levels of BP for each of the 3 transitions. Hazard ratios for subjects with high BP levels compared with normotensives were calculated for non-CVD death among participants free of CVD, CVD incidence, and death among individuals with prevalent CVD. We performed Cox proportional hazards analyses with age as the time scale and stratified by sex. Analyses were performed for each transition using STATA version 8.2 for Windows (Stata Corporation). We tested the proportionality of hazards assumption by analysis of the Schoenfeld residuals. Statistical significance was set at the 5% level.[15,16] In the Cox analyses, we assessed the effect of the potential confounders by adjusting for current smoking status (at age 50), level of education (high school or higher versus the rest), diabetes, and BMI. Information on all these variables was present for 3123 participants (99.8%). Data on cholesterol levels and physical activity were not available for >40% of the participants and were therefore not considered. All the analyses were done for CVD, MI, and stroke separately. From the several adjustments for potential confounding, only adjustment for BMI substantially changed the hazard ratios associated with BP. Therefore, we additionally performed these analyses stratified by BMI. On the basis of BMI, subjects were classified in 2 groups: group 1 with BMI <25 kg/m^2 (normal weight) and group 2 with BMI ≥25 kg/m^2 (overweight and obesity).[12] For this stratified analysis, 34 subjects with BMI <18.5 kg/m^2 were excluded. Therefore, the final analyses involved 3089 participants (1341 men and 1748 women).

Second, we used Poisson regression to calculate smoothed age-specific transition rates for each transition.[14] We assessed graphically the goodness-of-fit of the model to the empirical rates in 1-year intervals for the different transitions. We calculated the transition rates for the different BP groups by age directly without assuming proportional hazards.

Finally, after the Cox proportional hazards analyses for assessment of the risk associated with different levels of BP and the Poisson regression models used to calculate smoothed age-specific transitions rates for each transition, we transformed the crude transition rates into probabilities (assuming that within each single 1-year age interval, the hazard is constant) and created MSLTs for the total population stratified by level of BP and sex. We also created MSLTs additionally stratified for BMI. All these MSLTs represented populations 50 years of age and free of CVD at baseline. On the basis of LEs of the total FHS sample, the LE at 90 years of age was assumed to be a constant 4.53 years for men and 5.05 years for women in all groups of BP level.[13]

All life table calculations were done in S-plus 2000 (S-Plus 2000 Professional for Windows; Math Soft Inc) and confidence intervals (CIs) were obtained using a bootstrap procedure with 2000 replicates.[17]

Sensitivity Analysis

To test the robustness of our findings and to evaluate the effect of high BP among populations with different age, we repeated the analyses for participants at age 40 and at age 60.

Results

The average follow-up was 27.5 years. Weight and BMI increased with increasing BP. For smoking status, diabetes, and education the relationship with BP was less clear (Table 1).

BP and Risk of Disease and Mortality

Overall, we saw an increase in risk of disease (CVD, MI, and stroke) among participants with increased BP levels (groups 2 and 3; Table 2) as expected. Significant increases in mortality were observed among hypertensives but not among participants with high-normal BP levels compared with normotensives. In general, hazard ratios changed only slightly after adjusting for age, sex, and potential confounders (Table 2).

There was a clear dose-response relationship with BP category. The higher the BP levels, the higher the risk of incident disease. This increase in risk of incident CVD was >113% for hypertensives compared with normotensives and 41% for participants with high-normal levels compared with normotensives. The effect was similar for MI but stronger for stroke (Table 2).

The relationship of BP with the transition from a disease-free (without incident MI, stroke, or any CVD) status to death was less pronounced. The analyses excluding incident MI or stroke after the age of 50 showed a significant increased in risk of mortality among participants with elevated BP compared with normotensives. The risk of mortality among individuals without CVD was significantly increased for hypertensives compared with normotensives. This was not the case for the group with high-normal levels (Table 2).

The relationship of baseline BP with mortality after the onset of disease was only significant for hypertensives in the analyses of

TABLE 1. Baseline Characteristics by BP Category and Sex*

Sex	Characteristics	BP Category by JNC-7 Criteria‡ Group 1	Group 2	Group 3
Men	No. (%)	305 (22)	584 (43)	475 (35)
	SBP, mean (SD), mm Hg	112.6 (5.1)	127.2 (6.4)	149.4 (15.4)
	DBP, mean (SD), mm Hg	75.0 (4.6)	82.5 (5.1)	93.8 (8.8)
	Weight, mean (SD), kg	74.1 (10.7)	78.5 (10.9)	80.7 (11.8)
	Height, mean (SD), kg	1.73 (0.07)	1.73 (0.07)	1.72 (0.07)
	BMI†, mean (SD), kg/m^2	24.8 (3.1)	26.3 (3.2)	27.2 (3.6)
	BMI† ≥25, No. (%)	137 (44.9)	385 (65.9)	354 (74.5)
	Diabetes at age 50, No. (%)	6 (2)	7 (1.2)	10 (2.1)
	Smoking status, No. (%)			
	Never smoker	27 (8.9)	82 (14)	60 (12.6)
	Former smoker	31 (10.1)	85 (14.6)	65 (13.7)
	Current smoker	247 (81)	417 (71.4)	350 (73.7)
	High school graduated or higher, No. (%)	176 (57.7)	352 (60.3)	284 (59.8)
Women	No. (%)	469 (26)	754 (43)	541 (31)
	SBP, mean (SD), mm Hg	112.3 (5.3)	127.8 (6.7)	155.8 (17.7)
	DBP, mean (SD), mm Hg	73.7 (4.9)	81.2 (5.2)	93.6 (9.7)
	Weight, mean (SD), kg	61.2 (8.8)	64.1 (10.7)	69.1 (13.0)
	Height, mean (SD), kg	1.60 (0.06)	1.59 (0.06)	1.59 (0.06)
	BMI†, mean (SD), kg/m^2	23.9 (3.4)	25.2 (3.9)	27.3 (5.3)
	BMI† ≥25, No. (%)	137 (29.2)	343 (45.5)	336 (62.1)
	Diabetes at age 50, No. (%)	3 (0.6)	3 (0.4)	10 (1.8)
	Smoking status, No. (%)			
	Never smoker	179 (38.2)	363 (48.1)	287 (53.0)
	Former smoker	40 (8.5)	75 (10.0)	51 (9.5)
	Current smoker	250 (53.3)	316 (41.9)	203 (37.5)
	High school graduated or higher, No. (%)	316 (67.4)	484 (64.2)	320 (59.1)

*Subjects included were free of CVD at baseline and with no missing data BP. Total No. was 3128.
†BMI was calculated as weight in kg/height^2 in m^2.
Group 1 stands for normotensives, group 2 for participants with high-normal BP levels, and group 3 for hypertensives.

mortality subsequent to CVD or stroke after the age of 50. Hypertensives with MI did not have a significant increase in the risk of mortality after disease. In general, participants in group 2 of BP, with CVD, MI, or stroke did not have an elevated risk of mortality when compared with normotensives (Table 2). The higher relative risks of disease and mortality associated with elevated BP were similar when stratified by sex (data not shown).

BP and LE (Total, With and Without CVD)

For men and women, increased levels of BP were associated with strong reductions on total LE and LE without CVD. Participants with elevated BP (groups 2 and 3) lived more years with CVD, MI, and stroke than normotensives (Table 3).

The strong effect of increased BP levels on disease incidence, combined with a weaker effect on nondisease-related mortality and on mortality after the onset of disease, led to the shorter total LE and LE free of CVD and a longer LE with CVD for groups 2 and 3 compared with group 1 (Table 3).

Increased BP levels compared with normotension represented a decrease in LE of 5.1 (95% CI, 3.5 to 6.7) years for hypertensive men and 1.7 (95% CI, 0.1 to 3.2) years in men with high-normal BP, respectively. These reductions were in a similar direction for women but at a lower magnitude. Compared with the overall population, having hypertension (group III) at age 50 was associated with a reduction in total LE of 2.6 (95% CI, 1.8 to 3.5) and 2.9 years (95% CI, 2.2 to 3.7) for men and women, respectively.

Hypertensive men at age 50 lived 3.4 (95% CI, 2.6 to 4.2) fewer years without CVD than the overall population and 7.2 (95% CI, 5.6 to 9) fewer years than men with normal BP. Similar differences were observed in women. Hypertension was also associated with more years lived with CVD compared with normotensives: in men, 2.1 (95% CI, 0.9 to 3.4) years and in women, 2.3 (95% CI, 1.0 to 3.4) years. Smaller increases were seen for participants with high-normal BP compared with normotensives.

In hypertensive men, the increase in years lived with CVD was mainly attributable to the increase in years lived with MI (52%) and stroke (28%). For hypertensive women, the increase in years lived with MI (39%) and stroke (13%) explained to a lesser extent the increase in years lived with CVD (Figure).

TABLE 2. Hazard Ratios by Transition in Men and Women Combined at Age 50, Derived from 46 Years of Follow-Up

Transition	Outcome	BP Category*	HR† (95% CI)	HR‡ (95% CI)
Incident disease	CVD	High-normal	**1.43** (1.25–1.64)	**1.41** (1.23–1.62)
		Hypertension	**2.28** (1.99–2.61)	**2.13** (1.84–2.46)
	MI	High-normal	**1.47** (1.18–1.83)	**1.49** (1.19–1.86)
		Hypertension	**2.26** (1.82–2.82)	**2.18** (1.73–2.75)
	Stroke	High-normal	**1.60** (1.21–2.12)	**1.58** (1.19–2.11)
		Hypertension	**2.79** (2.11–3.69)	**2.68** (2.00–3.58)
Nondisease to death	CVD	High-normal	1.01 (0.85–1.20)	1.06 (0.89–1.26)
		Hypertension	**1.26** (1.05–1.53)	**1.34** (1.10–1.64)
	MI	High-normal	1.12 (0.98–1.28)	**1.17** (1.02–1.34)
		Hypertension	**1.61** (1.40–1.84)	**1.69** (1.46–1.95)
	Stroke	High-normal	1.10 (0.97–1.25)	1.14 (1.00–1.29)
		Hypertension	**1.50** (1.32–1.71)	**1.54** (1.34–1.77)
Disease to death	CVD	High-normal	1.03 (0.97–1.21)	1.05 (0.89–1.24)
		Hypertension	**1.22** (1.04–1.43)	**1.29** (1.10–1.52)
	MI	High-normal	0.86 (0.66–1.10)	0.86 (0.67–1.11)
		Hypertension	1.02 (0.79–1.31)	1.04 (0.81–1.35)
	Stroke	High-normal	1.18 (0.84–1.66)	1.16 (0.82–1.63)
		Hypertension	**1.45** (1.04–2.02)	**1.50** (1.07–2.10)

HR indicates hazard ratio.
Bold HRs are significant at the 0.05 level.
Classification is according to the JNC-7.
*Normotensive is always the reference category.
†Adjusted for age and sex (participants with information on all potential confounders; n=3089).
‡Adjusted for age, sex, smoking status, prevalent diabetes, and BMI at age 50 and education level (n=3089).

BP, BMI, and LE (Total, With and Without CVD)

Additional stratification for BMI showed similar differences in LEs for the group with BMI of <25 compared with the group with BMI of ≥25 in the overall population (Table 4).

Sensitivity Analysis

Analyses for participants with hypertension defined at age 40 or at age 60 showed similar differences in LEs compared with the group at age 50 (please see online supplement, available at http://www.hypertensionaha.org).

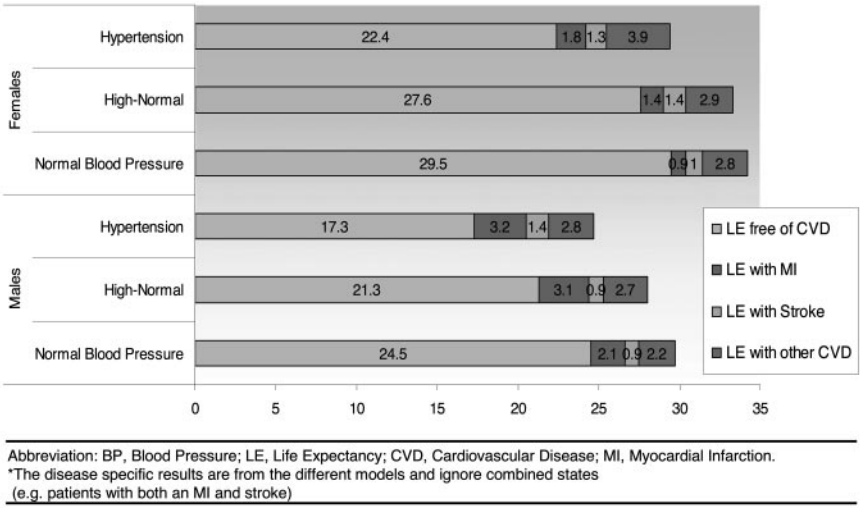
Discussion

Increased levels of BP at age 50 are associated with large decreases in total LE and LE free of CVD and increases in the number of years lived with CVD, MI, and stroke for men and

TABLE 3. LE in Years at Age 50 by Category of BP and Stratified by Sex

Sex	BP Category*	LE Free of CVD (95% CI)	LE With CVD (95% CI)	LE With MI (95% CI)	LE With Stroke (95% CI)	Total LE (95% CI)
Men	Overall Population	20.7 (20.0–21.3)	6.6 (6.1–7.1)	2.9 (2.6–3.3)	1.1 (0.9–1.3)	27.3 (26.6–27.9)
	Normal BP	24.5 (23.1–25.8)	5.2 (4.3–6.2)	2.1 (1.5–2.7)	0.9 (0.5–1.4)	29.7 (28.4–30.9)
	High-normal	21.3 (20.3–22.2)	6.7 (6.0–7.5)	3.1 (2.6–3.7)	0.9 (0.7–1.3)	28.0 (27.1–28.9)
	Hypertension	17.3 (16.3–18.3)	7.4 (6.6–8.2)	3.2 (2.6–3.8)	1.4 (1.1–1.8)	24.6 (23.6–25.6)
Differences in LE	High-normal vs normal	**−3.2** (−4.8–−1.5)	**1.5** (0.4–2.7)	**1.0** (0.2–1.8)	0.1 (−0.5–0.6)	**−1.7** (−3.2–−0.1)
	Hypertension vs normal	**−7.2** (−9.0–−5.6)	**2.1** (0.9–3.4)	**1.1** (0.3–2.0)	**0.6** (0.0–1.1)	**−5.1** (−6.7–−3.5)
	Hypertension vs overall	**−3.4** (−4.2–−2.6)	**0.8** (0.1–1.5)	0.3 (−0.2–0.8)	**0.4** (0.1–0.6)	**−2.6** (−3.5–−1.8)
Women	Overall population	26.5 (25.8–27.0)	5.8 (5.4–6.3)	1.4 (1.2–1.6)	1.3 (1.1–1.5)	32.3 (31.7–32.8)
	Normal BP	29.5 (28.3–30.9)	4.7 (3.9–5.8)	0.9 (0.6–1.6)	1.0 (0.6–1.6)	34.3 (33.1–35.5)
	High-normal	27.6 (26.5–28.4)	5.7 (5.1–6.5)	1.4 (1.1–1.8)	1.4 (1.0–1.8)	33.3 (32.4–34.2)
	Hypertension	22.4 (21.3–23.4)	7.0 (6.2–7.9)	1.8 (1.4–2.2)	1.3 (1.1–1.7)	29.4 (28.4–30.3)
Differences in LE	High-normal vs normal	**−2.0** (−3.7–−0.5)	1.0 (−0.2–2.2)	**0.5** (0.1–1.0)	0.4 (−0.3–1.0)	−1.0 (−2.5–−0.5)
	Hypertension vs normal	**−7.2** (−8.7–−5.4)	**2.3** (1.0–3.4)	**0.9** (0.3–1.4)	0.3 (−0.3–0.8)	**−4.9** (−6.4–−3.4)
	Hypertension vs overall	**−4.1** (−5.0–−3.2)	**1.2** (0.5–1.8)	**0.4** (0.3–0.8)	0.1 (−0.2–0.4)	**−2.9** (−3.7–−2.2)

Bold LE differences are significant at the 0.05 level.
*Classification according to JNC-7.



Abbreviation: BP, Blood Pressure; LE, Life Expectancy; CVD, Cardiovascular Disease; MI, Myocardial Infarction.
*The disease specific results are from the different models and ignore combined states
(e.g. patients with both an MI and stroke)

Effect of BP level on LE by type of CVD and sex.

women. The effects of baseline BP level on LE and LE with and without CVD showed a dose-response relationship.

For hypertensives, the decrease in total LE was between 5.1 (males) and 4.9 (females) years compared with normotensive participants. The reduction in LE free of CVD for hypertensives was 7.2 years for both sexes. In addition to a shorter LE, hypertensives lived >2 years more with CVD compared with participants with normal BP. Also, for participants with high-normal BP compared with normotensives, there was a decrease in LE free of CVD and an increase in LE with CVD but almost half that observed among hypertensives.

The harmful effects of increased BP on LE and CVD observed in our study were independent of BMI. Similar effects were seen among participants above and below a BMI of 25. The same applies to age. Participants at age 40 and 60 with hypertension experienced similar reductions in LE. This suggests that efforts to reduce high BP levels should be increased similarly in lean and obese adult populations and starting as soon as age 40. It is possible that this effect is similar in populations aged <40 or >70; however, we did not have sufficient data to test this.

We decided not to consider treatment of hypertension in the classification of our population and our analyses because we wanted to evaluate the effect of increased BP levels, which can arise as well in hypertensives under treatment. Besides, BP control in the Unites States during the time most of the FHS participants were ≈50 years of age (the 1950s and 1960s) was poor (only 31% of hypertensives were receiving treatment, and BP levels were controlled <140/90 mm Hg in only 10%).[1] Also, guidelines and treatment of hypertension have changed substantially with time, as has the coding of hypertension treatment in the FHS. These factors limit the comparability between different time periods.

Our study has several advantages when compared with other studies. Two studies previously compared the relationship of BP in adulthood with total mortality and found similar effects but of smaller magnitude.[8,9] Both studies used only a single reading of BP levels at baseline, which, because of random variation in an individual's BP, will result in nondifferential misclassification of exposure and therefore in a systematic underestimation of the effect of BP on CVD and mortality.[18] We used all BP measurements between ages 45 and 50 to predict BP at age 50; each participant had ≥2 different measures of BP, therefore lowering the probability of dilution of the effect of BP on CVD. This could be the reason why we found larger effects. Furthermore, these studies did not investigate the LE with and without CVD separately, and only 1 of them also reported effects on women, and they used shorter follow-up periods. Thus, our study is the first report from a large and continuously followed cohort showing in both sexes the effect of high BP levels on total LE and LE with and without CVD, MI, and stroke.

A relevant limitation of our study is that we could not evaluate the effect of high BP levels completely independent of other risk factors of CVD such as cholesterol and physical activity. Although we accounted for some risk factors at baseline by the exclusion of participants with prevalent CVD and the correction for BMI in our analyses, data for physical activity and serum cholesterol were incomplete, unreliable, or unavailable for a large proportion of our population. It is possible that part of the observed differences in LE within the 3 BP groups could be explained by the differences in blood lipid concentration and physical activity levels. However, the extent of this "lipid" and "physical inactivity" effect cannot be calculated with the available data. Other important risk factors for CVD (sex, age, BMI, smoking, and education) were taken into account in our analysis.

Most of the data used in our analyses were collected ≥3 decades ago. The historic character of the Framingham studies limits the extrapolation of the findings obtained through analyses of the Framingham studies to today's populations. Great advances in health promotion and in the diagnosis, prevention, and treatment of CVD have occurred since the Framingham studies started. However, these results do indicate the potential health impact of hypertension in the absence of appropriate treatment.

The life course perspective used in this study allowed us to show that the harmful effects of hypertension are not limited to decreasing the total LE among those who experience it, but it is also associated with an important reduction in the number

TABLE 4. LE (Total, With and Without CVD) in Years at Age 50 by Category of BP, Stratified by Sex and BMI*

Sex	BP Category†	LE Free of CVD (95% CI)	LE With CVD (95% CI)	Total LE (95% CI)
Men, BMI <25	Overall	22.2 (21.1–23.2)	5.6 (4.9–6.3)	27.8 (26.6–28.8)
	Normal BP	25.3 (23.5–26.9)	4.4 (3.4–5.6)	29.8 (28.0–31.3)
	High-normal	21.7 (20.0–23.3)	5.9 (4.7–7.1)	27.7 (25.9–29.2)
	Hypertension	18.5 (16.5–20.7)	6.8 (5.2–8.5)	25.3 (22.8–27.6)
Differences in LE	Hypertension vs overall	**−3.6** (−5.6-−1.8)	1.2 (0.1–2.8)	**-2.5** (−4.7-−0.6)
	Hypertension vs normal	**−6.8** (−9.4-−3.9)	**2.3** (0.6–4.5)	**−4.4** (−7.3-−1.6)
Men, BMI ≥25	Overall	19.8 (19.1–20.7)	7.2 (6.6–7.8)	27.0 (26.2–27.7)
	Normal BP	23.8 (21.7–26.1)	6.1 (4.6–7.9)	29.9 (27.9–32.0)
	High-normal	21.2 (20.1–22.5)	7.1 (6.2–8.1)	28.3 (27.1–29.4)
	Hypertension	16.9 (15.8–18.0)	7.6 (6.8–8.5)	24.5 (23.4–25.7)
Differences in LE	Hypertension vs overall	**−3.0** (−4.0-−2.0)	0.5 (−0.2–1.2)	**−2.5** (−3.4-−1.6)
	Hypertension vs normal	**−6.9** (−9.3-−4.3)	1.6 (−0.5–3.3)	**−5.4** (−7.7-−3.2)
Women, BMI <25	Overall	27.8 (26.9–28.7)	5.4 (4.8–6.0)	33.2 (32.4–34.0)
	Normal BP	30.3 (28.7–31.7)	3.7 (2.9–4.7)	34.1 (32.6–35.3)
	High-normal	28.2 (27.0–29.4)	5.8 (4.9–7.0)	34.0 (32.7–35.2)
	Hypertension	23.1 (21.5–25.0)	7.1 (6.0–8.6)	30.2 (28.6–31.7)
Differences in LE	Hypertension vs overall	**−4.7** (−6.2-−3.1)	**1.8** (0.8–3.0)	**−3.0** (−4.5-−1.7)
	Hypertension vs normal	**−7.3** (−9.3-−4.9)	**3.4** (1.9–5.0)	**−3.9** (−5.9-−1.7)
Women, BMI ≥25	Overall	24.9 (24.1–25.8)	6.5 (5.8–7.1)	31.4 (30.6–32.2)
	Normal BP	27.6 (25.4–30.1)	7.2 (5.4–9.4)	34.8 (32.6–37.0)
	High-normal	26.8 (25.5–28.1)	5.8 (4.8–6.9)	32.7 (31.4–33.8)
	Hypertension	21.9 (20.6–23.3)	7.0 (6.0–7.9)	28.9 (27.8–30.2)
Differences in LE	Hypertension vs overall	**−3.0** (−4.1-−2.0)	0.5 (−0.3–1.3)	**−2.5** (−3.5-−1.5)
	Hypertension vs normal	**−5.7** (−8.3-−3.0)	0.2 (−2.6–1.8)	**−5.9** (−8.3-−3.3)

Ref indicates reference.
Bold LE differences are significant at the 0.05 level.
*BMI calculated as weight in kg/height^2 in m^2.
†Classification according to the JNC-7.

of years lived without CVD and an increase in the time spent with this disease.

Perspectives

Our findings indicate that the harmful effects of hypertension are not limited to decreasing the total LE among those who experience it. Hypertension is also associated with an important reduction in the number of years lived without CVD and an increase in the time spent with this disease. Our results also show that the association between increased BP levels and total LE is higher than estimated previously,[8,9] emphasizing the global need to improve BP control. Although similar analyses of treated populations are needed, our results suggest that optimizing the control of BP to keep it at normal levels and avoiding hypertension could potentially lead to a longer LE, and notwithstanding the subsequent aging of the population, also to a reduction of CVD in the general population.

Acknowledgments

O.H.F., A.P., L.B., and C.dL. were partly funded by the Netherlands Heart Foundation (grant 98.138) and the Netherlands Organization for Scientific Research (grants 904-66-093 and 2200.0126). A.P. was also partly funded by VicHealth (fellowship grant 2002-0191). These funding organizations did not participate in the design and conduct of the study, collection, management, analysis, and interpretation of the data, nor preparation, review, or approval of the manuscript. We thank the Framingham Heart Study coordinators for access to the original data set. The Framingham Heart Study is conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with Framingham Heart Study investigators. This manuscript was prepared using a limited access data set obtained from the NHLBI and does not necessarily reflect the opinions or views of the Cardiovascular Health Study or NHLBI. This manuscript has been reviewed by NHLBI for scientific content and consistency of data interpretation with previous Framingham Heart Study publications.

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