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ABSTRACT

Estimating the value of medical innovation is a continual challenge. In this research, we quantify the impact of antihypertensive therapy on U.S. blood pressures, risk and number of heart attacks, strokes, and deaths. We also consider the potential for further improvements. We estimate the value of innovation using equations relating blood pressure to adverse outcomes from the Framingham Heart Study. Our results show that without antihypertensive therapy, 1999-2000 average blood pressure for the U.S. population age 40 plus would have been 10-13 percent higher. 86,000 excess premature deaths from cardiovascular disease (2001), and 833,000 hospital discharges for stroke and heart attacks (2002) would have occurred. Life expectancy would be 0.5 (men) and 0.4 (women) years lower. At guideline care, there would have been 89,000 fewer premature deaths (2001) and 420,000 fewer hospital discharges for stroke and heart attack (2002) than observed. Our analysis suggests that antihypertensive therapy has had a significant impact on cardiovascular health outcomes but that mortality gains would have been approximately twice as high if guideline care had been achieved for all.

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Hypertension is an established risk factor for conditions including coronary heart disease (CHD), myocardial infarction (MI), and stroke. Over 50 million adults were hypertensive in 1999-2000, including 43 percent of adults age 40 and over (i.e., had Stage I or II blood pressures, or reported taking antihypertensive medicine). The risk of developing hypertension increases with age; lifetime risk is estimated at approximately 90 percent for individuals with normal blood pressures at age 55 or 65 and who survive to age 80 to 85.1 The economic burden of CHD and stroke is substantial. Applying the methodologies of Rice et al. and Hodgson and Cohen to 2002 data, the total economic burden of CHD and stroke is estimated at $120.6 and $48.9 billion, respectively.2,3

Drug therapy for hypertension has improved over the past four decades. Only limited drug therapy existed in the late 1950s and early 1960s; the only FDA-approved drug therapies for hypertension were vasodilators (approved in 1946), peripherally acting agents (1953) and diuretics (1958).[4] Most patients who would be treated immediately today with effective drug therapy were untreated, or treated less effectively. For example, while *Harrison's Principles of Internal Medicine* (1962) found that diastolic blood pressure was important in determining mortality, it justified treatment only in *properly selected cases,* stating, "A woman who has tolerated her diastolic pressure of 120 for 10 years without symptoms or deterioration does not need specific treatment for hypertension."[5] As late as 1971-75, only nine percent of the population age 40 and over with hypertension reported in NHANES taking medication for it, and blood pressures were poorly controlled; 79 percent of this treated group still had Stage I or II blood pressures. Thus, the earlier time period could be called "drug-naïve" with regard to widespread use of a range of effective drug therapies.

Over time, additional antihypertensive drugs became available – calcium channel blockers and beta blockers (1970s and 1980s), and ACE inhibitors and angiotensin receptor antagonists (1980s and 1990s). In 1999-2000, we calculate that 61 percent of the population age 40 and over with hypertension reported in NHANES taking medication.

Our objective is to estimate the actual impact of antihypertensive therapy on the U.S. population over the past four decades and identify potential further improvements if guideline care were achieved. Despite documented major strides in cardiovascular disease management, there has been no systematic estimate of the total impact of antihypertensive therapy on health outcomes. We use national survey data to estimate the impact of drug treatment on average blood pressures, and the best-available published risk equations to estimate the impact on the risk and number of MI, strokes, and deaths. Because the objective is to estimate the "real world" impact of antihypertensive therapy on outcomes for the entire population, representative national survey data were used, rather than estimates derived from highly controlled clinical trials.

METHODS

Data Sources

We use data on 5,046 individuals age 30-79 from the 1959-62 National Health Examination Survey (NHES) and 2,284 individuals age 40-79 from the 1999-2000 National Health and Nutrition Examination Survey (NHANES); U.S. life expectancy tables from the National Center for Health Statistics (NCHS); hospital discharge figures from the National Hospital Discharge Survey (NHDS); and published risk equations from the Framingham Heart Study. All observations from NHES and NHANES populated with the required data were included in calculations, which were performed using SAS, release 9.1.

Statistical Analysis

Determinants of hypertension, including body mass index (BMI), diabetes, family history, excessive alcohol use, high salt diet, exercise, race, age and sex were identified from literature searches. Data on BMI, diabetes, race, age and sex were used to model observed blood pressure among a drug-naïve sample (the 1959-62 NHES sample).

The relationship between blood pressure (BP) and these explanatory factors was estimated using ordinary least squares (OLS) regression, with separate equations for systolic and diastolic blood pressure, and for men and women.

The explanatory variables are race (BLACK, OTHER, with white being the omitted category), AGE (nine variables corresponding to five-year cohorts beginning with age 35, age 30-34 being the omitted category), BMI, BMI^2 (to account for possible non-linearities in the effect of BMI on BP), and DIABETES (either reporting taking insulin or having a blood glucose level>140 mg/dL) DIABETES, BMI, and BMI^2 terms control for the impact of secular population changes in the levels of diabetes and obesity.

To predict BP in the absence of antihypertensive drug therapy, the estimated structural relationship based on data from 1959-62 is applied to observed values of the explanatory variables for individuals in the 1999-2000 NHANES sample. Following JNC-V definitions, predicted and observed systolic (SBP) and diastolic (DBP) BPs for each individual are assigned to five categories: Optimal, Normal, High Normal, Stage I and Stage II Hypertension.[6] The resulting distribution of predicted BPs for 1999-2000 is compared to the distribution of observed BPs. Controlling for other identified risk factors for which data are available, the difference between the two distributions is attributed to antihypertensive therapy. Two risk factors – sodium intake and exercise – could not be included in the predictive model because data are available only for 1999-2000. A sensitivity analysis of the impact of these variables on BP, using 1999-2000 data, was conducted.

The impact of antihypertensive therapy on risk of death and total deaths from CHD is estimated using Framingham Heart Study risk equations estimated by Wilson *et al.*[7], together with NCHS population life tables and cause of death statistics. The impact of antihypertensive therapy on risk of stroke and MI, and hospital discharges for stroke and MI is estimated using Framingham Heart Study risk equations estimated by Anderson *et al*.[8], and NHDS hospital discharge statistics.

The Wilson *et al.* risk equations are generated from a hazard model predicting the probability of a CHD event (angina, MI, or death from CHD) within the next 10 years, and include variables for age, total cholesterol, HDL-C, BP, diabetes and smoking. These equations are applied to each individual in the NHANES sample based on the individual's observed characteristics. For each individual, the probability of a CHD event within 10 years is calculated based on (1) observed BP and (2) predicted BP in the absence of antihypertensive therapy, and a relative risk ratio calculated by dividing the two. The calculated relative risks are averaged across individuals within each sex-age cohort, according to sample population weights. Since the variable of interest is death from major cardiovascular disease, average risks are combined with the probabilities for men and women of a given age, n, surviving to age n+1 (from the NCHS life tables),[9] and with the share of deaths due to major cardiovascular disease (ICD/10 I00-I78) by sex-age cohort (from NCHS cause of death tables by sex and age).[10] By adjusting for the share of total mortality due to death from major cardiovascular disease, increases in the risk of a CHD event in the next 10 years are assumed to increase the risk of death from major cardiovascular disease in the next 10 years proportionally. Improvement in life expectancy attributable to antihypertensive therapy is calculated as the difference between life expectancy at birth reported by NCHS (with observed BPs) and calculated with predicted BPs. Predicted life expectancy is derived by adjusting the risk of death in each year for men and women age 40 and above by the relative risk described for the corresponding sex-age cohort, and summing over all ages.

The Anderson *et al.* risk equations are generated from separate parametric models predicting the probability of stroke and MI for men and women of given characteristics (age, BP, smoking, ratio of total cholesterol to HDL-C, diabetes and ECG-LVH) over the next 4 to 12 years. The recommended specification incorporating SBP is used. For each individual, the risk of stroke and MI, respectively, in the next five years is calculated based on (1) observed BP and (2) predicted BP in the absence of antihypertensive therapy, and a relative risk ratio calculated by dividing the two. The number of avoided hospital discharges is calculated by applying the relative risk ratio to hospital discharge statistics for stroke (ICD/9 430-438) and MI (ICD/9 410) by sex and age from the NHDS for 2002. These risk equations allow us to isolate the effect of BP from other factors such as secular changes in serum cholesterol levels, diabetes rates and smoking, which also affect CHD risk.

RESULTS

Predictive Structural Model of Untreated Blood Pressure

OLS multivariate regression relating SBP and DBP to risk factors was used with a sample of 2,382 men and 2,664 women age 30-79 from the 1959-62 NHES. Results appear in Table 1.

Table 1

Figure 1 is derived by applying the structural relationship from the untreated BP model for men and women, respectively, to observed characteristics of individuals in the 1999-2000 NHANES sample. The predicted SBPs and DBPs without antihypertensive drug therapy and observed BPs for each individual are assigned to five categories: Optimal, Normal, High Normal, Stage I and Stage II Hypertension. If an individual's predicted systolic and diastolic BPs are in different categories, the more hypertensive category is assigned.

Figure 1

For example, observed BPs for men age 40-49, treated or not, were: 36 percent Optimal, 26 percent Normal, 21 percent High Normal, 14 percent Stage I and 3 percent Stage II Hypertensive. For the same cohort, predicted BPs without treatment were: 22

percent Optimal, 22 percent Normal, 19 percent High Normal, 27 percent Stage I and 10 percent Stage II Hypertensive.

Calculating weighted average BPs for each sample, we find that in the absence of antihypertensive drug therapy, average BPs for the population over 40 would be 10-13 percent higher (10.0 to 10.7 percent for men and 10.4 to 12.9 percent for women for SBP and DBP, respectively). Women age 70-79 exhibited the greatest difference between predicted and observed BPs, with 61 percent predicted and 29 percent observed with Stage II hypertension, compared to 41 and 12 percent, respectively, for men age 70-79. However, these women also experienced rates of Stage II hypertension 2.5 times higher than their male counterparts.

Statistical Sensitivity Analyses

To allow for nonlinearities, the continuous BMI variables in the BP prediction equation were replaced by six categories from the literature: "less than or within normal BMI range", "marginally overweight", "overweight", "very overweight", "severely obese", and "morbidly/super obese".[11] The relationship between BP and BMI is linear for women and weakly concave for men.

Variables measuring reported sodium intake and exercise habits (available only for 1999-2000) were also included in the model. Both have been identified as hypertension risk factors in the literature. Neither increased the explanatory power of the model significantly; the predicted BP distributions were nearly identical with or without these variables. Variables for sodium intake were never statistically significant and often had incorrect signs, while the exercise variable was occasionally significant. They had no significant impact on BMI or BMI^2coefficients, and increased the R^2 measure only at the third decimal point. To assess possible non-normality in the model's error terms, a log-transformation of the dependent variable was regressed on the same variables. This specification did not increase the model's explanatory power nor the precision of the estimated coefficients significantly.

Impact of Blood Pressure on Life Expectancy and Total Deaths from Major Cardiovascular Disease

Applying the Wilson *et al.* risk equation for CHD events within the next 10 years to each individual in the 1999-2000 sample, and averaging the effect over the entire population, the total increase in life expectancy associated with antihypertensive drug therapy is 0.5 years (men) 0.4 years (women). Averaged over only the population with predicted Stage I or Stage II hypertension, the values are 0.9 years (men) and 0.6 years (women). Applying the change in risk of death to total deaths for each sex-age cohort, an estimated 86,000 excess premature deaths from cardiovascular disease (50,000 men, 36,000 women) would have occurred in 2001 among the U.S. population age 40 plus in the absence of antihypertensive drug therapy. For adults age 40 plus, observed 2001 total deaths and deaths from major cardiovascular disease are four and nine percent lower, respectively, than predicted levels with untreated BPs. Due to potential competitive mortality, these estimates represent reductions in premature deaths due to cardiovascular disease; reductions in total mortality from all causes in a given year may be lower.

If all patients with Stage I or Stage II hypertension who reported being untreated had been treated, as recommended in JNC 7[12(p 1211)], and all treated patients achieved normal BPs, we calculate an additional 89,000 fewer premature deaths from major cardiovascular disease in 2001 than actually occurred. This is likely an underestimate because it assumes that those dying are no more likely to have high BP than the general population.

Impact of Blood Pressure on MI and Stroke

Similarly, for each individual in the NHANES 1999-2000 sample, we calculate the separate risks of stroke or MI within the next five years, applying the Anderson *et al.* equations. In the absence of antihypertensive drug therapy, there would have been 572,000 more hospital discharges for stroke in 2002 (162,000 men, 410,000 women), and 261,000 more discharges for MI (87,000 men, 174,000 women). This is a 38 percent reduction in discharges for stroke and a 25 percent reduction for MI, compared to predictions with untreated BPs. Most of this improvement represents stroke and MI avoided altogether, rather than delayed. Even if all the predicted reduction in discharges were for patients with predicted Stage II hypertension (those most likely to have a stroke in the future), we calculate only seven percent of discharges for stroke and five percent for MI would have occurred in the future; the remainder were avoided altogether.

We also calculate considerable improvements if guideline care were achieved. If all untreated patients with Stage I or Stage II hypertension had been treated, and all treated patients achieved normal BPs, there would have been an additional 278,000 (stroke) and 142,000 (MI) fewer hospital discharges in 2002 than actually occurred. This is likely an underestimate because it assumes that those discharged for stroke and MI are no more likely to have high BP than the general population.

Table 2

DISCUSSION

By many measures, hypertension is much better treated now than in the past. Although further efforts are still needed to bring health benefits to all, awareness, treatment, and control of hypertension have shown considerable improvement since NHANES II (1976-80)[12(p 1207)]. The estimated impact of antihypertensive therapy on BP is substantial: approximately 10 percent (SBP) and 13 percent (DBP) for both men and women age 40 plus, compared to predicted untreated BP. Better-controlled BP translated into nine percent fewer deaths from major cardiovascular disease in 2001, and 38 percent and 25 percent fewer hospital discharges for stroke and MI in 2002, respectively, compared to predicted levels in the absence of drug therapy. Placing the estimated reduction of approximately 86,000 deaths in context, U.S. deaths from motor vehicle accidents totaled approximately 42,000 in 2001.[13]

Figure 2

This study quantifies the impact of antihypertensive drugs in terms of avoided premature deaths from cardiovascular disease and excess hospitalizations for stroke and MI. Assigning a monetary value to these outcomes and drawing on figures from the literature for 1998, we calculate a benefit-cost ratio of over 12:1 for men and 11:1 for women by comparing life expectancy benefits to antihypertensive drug expenditures. We assume each year of additional life in good health is valued at $90,000 (a generally accepted value of $100,000, less $10,000 in average support costs during non-working years paid by government),[14] and expenditures on antihypertensive drugs average $2,600 for men (over a 73.8-year expected lifespan) and $3,248 for women (over a 79.5-year expected lifespan).[15] We discount both costs and benefits at three percent a year, the inflation-adjusted U.S. Treasury bond yield.

Previous studies have concluded that antihypertensive treatment is cost-effective; Weinstein reports estimates of the number of quality-adjusted life years achieved with each $1 million spent on antihypertensive drug therapy as between 20-50 (antihypertensive treatment, DBP 95-104) to more than 200 (beta-blockade post-myocardial infarction, high risk). As a benchmark, $1 million spent for dialysis for end-stage renal disease is estimated to yield 10-20 quality-adjusted life years.[16]

Reduced hospitalizations for stroke and MI increase calculated net benefits further. Dividing figures from the literature for total hospital costs for CHD and cerebrovascular disease[17, 18] by corresponding hospital discharges, and assuming hospital costs represent 70 percent of direct medical costs in the year following stroke and MI,[19] we estimate 2002 total direct medical costs avoided due to fewer strokes and MI of $10.7 billion and $5.8 billion, respectively. Including the impact of antihypertensive drugs on quality of life and work productivity would increase the benefit–cost ratio further.

While the benefits have been substantial, significant opportunities remain to extend drug treatment to more who could benefit. We estimate life expectancy could increase an additional 0.3 years (men) and 0.1 years (women) if therapy were extended to all with Stage I or Stage II hypertension not currently treated with medication, and an additional 0.2 years for both men and women if those treated achieved normal BP. These figures translate into an additional 89,000 avoided premature deaths from major cardiovascular disease, and an additional 278,000 (stroke) and 142,000 (MI) avoided hospital discharges, compared with actual 2002 levels.

Study Limitations

The methodology used is a residual analysis. It controls for all risk factors for which data are available, and confirms that omitted sodium and exercise variables are unlikely to have a significant impact. There remains, however, the possibility that unknown factors may have contributed to differences between observed and predicted BPs. In particular, some researchers suggest a downward cohort effect due to unknown, population-level factors. Using the same datasets, Goff *et al*. find that the 10^{th}, 50^{th}, and 90^{th} percentile levels of observed BP by age were lower for more recent birth cohorts. However, because observed BPs include both treated and untreated BPs, the observed cohort effect is confounded by the impact of treatment. Even in lowest-decile SBPs, where "pharmacologic management is unlikely to have had any impact,"[20] 1999-2000 NHANES data show nine percent age 40 plus reported taking hypertension medication (increasing to 29 percent age 60 plus). We adjust for this confounding treatment effect by calculating the ratio of untreated to all lowest-decile SBPs in the 1999-2000 sample (by age), and adjusting the authors' estimates thereby. Assuming the improvement in the lowest decile, as adjusted, to be entirely due to a cohort effect would reduce our estimate of the impact of BP improvements modestly, from a nine- to an eight-percent reduction in 2001 premature deaths from cardiovascular disease.

Our calculations assume the 1959-62 data provide a reliable predictive basis for calculating the structural relationship between BP and explanatory variables, notably BMI and BMI^2, and that the sample is "drug-naïve." While NHANES data are available for subsequent time periods (1971-75, 1976-80, 1988-94, and 1999-2000), their suitability for estimating this relationship is compromised by the increasing proportion being treated with antihypertensive drugs, thereby rendering the remaining untreated population increasingly unrepresentative. When similar equations are estimated in later years with only the remaining untreated individuals, the predicted impact of a change in BMI (and BMI^2) on BP was always positive, but generally became smaller over time, consistent with an increasingly selective untreated population. While treatment rates were low in 1959-62 and few effective clinical options were available, to the degree that some individuals were treated effectively, predicted untreated BPs for 1999-2000, and therefore the estimated impact of treatment on mortality and morbidity, would be understated. The study design may also overestimate strokes and MI avoided due to a "survival bias"; specifically, in the absence of effective BP control, the total population with hypertension would have been smaller and some individuals who had a stroke or MI in 2002 would have died previously. However, there is a likely countervailing underestimation effect, in that those discharged for stroke and MI are assumed to be no more likely to have high BP than the general population. The net impact of these effects is unknown.

Finally, the study design relies on Framingham Heart Study risk equations. To the degree that these equations were estimated on an essentially all-white population, and that cardiovascular risk for the same level of BP may be higher for non-white populations, all other factors held constant, we have underestimated the impact of treatment.

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Tables and Figures

Table 1. Structural Model of Untreated Systolic and Diastolic Blood Pressure for Men and Women ages 30 to 79

Variable	Systolic Blood Pressure						Diastolic Blood Pressure					
	Men			Women			Men			Women		
	Coefficient		Standard Error	Coefficient		Standard Error	Coefficient		Standard Error	Coefficient		Standard Error
Black	7.605	*	1.327	8.114	*	1.341	5.662	*	0.790	5.631	*	0.741
Other	-5.246	***	3.043	-13.337	*	3.683	0.190		1.811	-3.367	***	2.036
Age 35-39	0.139		1.489	0.958		1.536	1.330		0.886	1.233		0.849
Age 40-44	2.371		1.470	6.577	*	1.589	3.348	*	0.875	3.940	*	0.879
Age 45-49	4.641	*	1.503	10.912	*	1.587	4.599	*	0.894	5.223	*	0.877
Age 50-54	8.139	*	1.560	15.185	*	1.689	4.886	*	0.929	5.870	*	0.934
Age 55-59	11.501	*	1.635	21.105	*	1.717	5.063	*	0.973	7.149	*	0.949
Age 60-64	15.238	*	1.688	27.409	*	1.833	5.017	*	1.005	7.817	*	1.013
Age 65-69	19.443	*	1.816	34.495	*	1.842	3.680	*	1.081	7.742	*	1.018
Age 70-74	24.767	*	2.014	42.432	*	2.020	3.289	*	1.199	4.821	*	1.117
Age 75+	29.205	*	2.344	36.163	*	2.495	2.715	**	1.395	3.157	**	1.379
BMI	2.567	*	0.806	1.722	*	0.551	2.007	*	0.480	1.295	*	0.305
BMI 2	-0.025	***	0.015	-0.006		0.010	-0.021	**	0.009	-0.008		0.005
Diabetes	0.987		3.806	6.733	**	2.902	1.125		2.265	-0.297		1.604
Number of Observations	2382			2664			2382			2664		
R-Squared	0.2015			0.4006			0.1249			0.2191		
Adj. R-Squared	0.1967			0.3975			0.1197			0.2149		

Note: * indicates significance at the 1% level; ** at the 5% level; *** at the 10% level.

Omitted category for race is white; for age, omitted category is 30-34.

Table 2: Predicted Achieved and Potential Further Improvements in Life Expectancy and Deaths Avoided, 2001, and Hospital Discharges for MI and Stroke Avoided, 2002, Men and Women

Sex and age	Deaths from Major Cardiovascular Disease, 2001				Hospital Discharges for Stroke, 2002				Hospital Discharges for Myocardial Infarction, 2002			
	10-Year Relative Risk of CHD Event *	Observed	Avoided Due to AHRT Therapy **	Improvement Possible if Guideline Blood Pressures Achieved	5-Year Relative Risk of Stroke *	Observed	Avoided Due to AHRT Therapy **	Improvement Possible if Guideline Blood Pressures Achieved	5-Year Relative Risk of Myocardial Infarction *	Observed	Avoided Due to AHRT Therapy **	Improvement Possible if Guideline Blood Pressures Achieved
TOTAL		**910,126**	**86,098**	**89,150**		**915,022**	**571,930**	**278,160**		**794,080**	**260,701**	**142,696**
MALE		**420,330**	**49,884**	**49,951**		**417,415**	**161,994**	**122,527**		**461,245**	**86,866**	**67,312**
40 to 44 years	1.027 1.017 , 1.037	7,508	937	845	1.482 1.400 , 1.565	8,991	4,336	621	1.284 1.207 , 1.361	26,079	7,395	1,110
45 to 49 years	1.027 1.017 , 1.037	12,418	1,270	1,145	1.312 1.235 , 1.388	19,011	5,923	2,148	1.179 1.126 , 1.231	27,741	4,954	1,793
50 to 54 years	1.056 1.038 , 1.073	18,442	3,261	2,324	1.233 1.171 , 1.295	23,094	5,388	5,101	1.103 1.070 , 1.137	45,416	4,697	6,171
55 to 59 years	1.056 1.038 , 1.073	22,489	3,689	2,629	1.334 1.255 , 1.414	32,509	10,870	6,929	1.166 1.116 , 1.217	55,019	9,142	6,453
60 to 64 years	1.064 1.051 , 1.077	27,831	5,067	4,266	1.512 1.434 , 1.589	45,942	23,517	10,925	1.244 1.204 , 1.285	49,159	12,003	6,407
65 to 69 years	1.064 1.051 , 1.077	36,221	6,506	5,478	1.378 1.316 , 1.440	48,040	18,161	15,124	1.187 1.155 , 1.218	56,299	10,508	8,880
70 to 74 years	1.042 1.032 , 1.053	51,535	5,855	6,681	1.391 1.318 , 1.464	66,281	25,923	22,573	1.189 1.151 , 1.228	53,954	10,218	9,771
75 years and over	1.042 1.032 , 1.053	243,886	23,299	26,584	1.391 1.318 , 1.464	173,547	67,875	59,105	1.189 1.151 , 1.228	147,578	27,949	26,727
Improvement in Life Expectancy			**0.5**	**0.5**								
FEMALE		**489,796**	**36,214**	**39,199**		**497,607**	**409,936**	**155,632**		**332,835**	**173,835**	**75,384**
40 to 44 years	1.048 1.035 , 1.061	3,610	979	213	1.665 1.565 , 1.765	7,795	5,184	402	1.633 1.412 , 1.855	7,638	4,838	344
45 to 49 years	1.048 1.035 , 1.061	5,350	1,308	284	1.670 1.556 , 1.785	16,598	11,128	1,451	1.601 1.425 , 1.778	11,502	6,918	910
50 to 54 years	1.042 1.026 , 1.057	8,240	1,514	842	1.422 1.337 , 1.506	28,323	11,943	5,514	1.323 1.231 , 1.415	13,905	4,487	2,061
55 to 59 years	1.042 1.026 , 1.057	10,846	1,837	1,022	1.635 1.523 , 1.746	26,132	16,582	5,155	1.446 1.323 , 1.569	29,537	13,165	4,139
60 to 64 years	1.072 1.057 , 1.088	15,058	4,009	2,387	1.660 1.545 , 1.776	37,766	24,938	9,855	1.404 1.312 , 1.496	26,852	10,844	4,930
65 to 69 years	1.072 1.057 , 1.088	22,845	5,479	3,262	1.823 1.695 , 1.952	42,976	35,389	12,373	1.526 1.407 , 1.645	36,247	19,081	7,219
70 to 74 years	1.023 1.016 , 1.030	37,808	2,607	3,856	1.902 1.771 , 2.032	64,382	58,050	23,024	1.553 1.448 , 1.658	34,794	19,232	9,369
75 years and over	1.023 1.016 , 1.030	386,039	18,482	27,332	1.902 1.771 , 2.032	273,635	246,722	97,858	1.553 1.448 , 1.658	172,360	95,270	46,411
Improvement in Life Expectancy			**0.4**	**0.3**								

* 95% confidence interval.
**Antihypertensive Therapy.

Sources:
National Center for Health Statistics, DataWarehouse, Deaths from 358 Selected Causes by 5-Year Age Groups, Race, and Sex: Each State and the District of Columbia, 2001.
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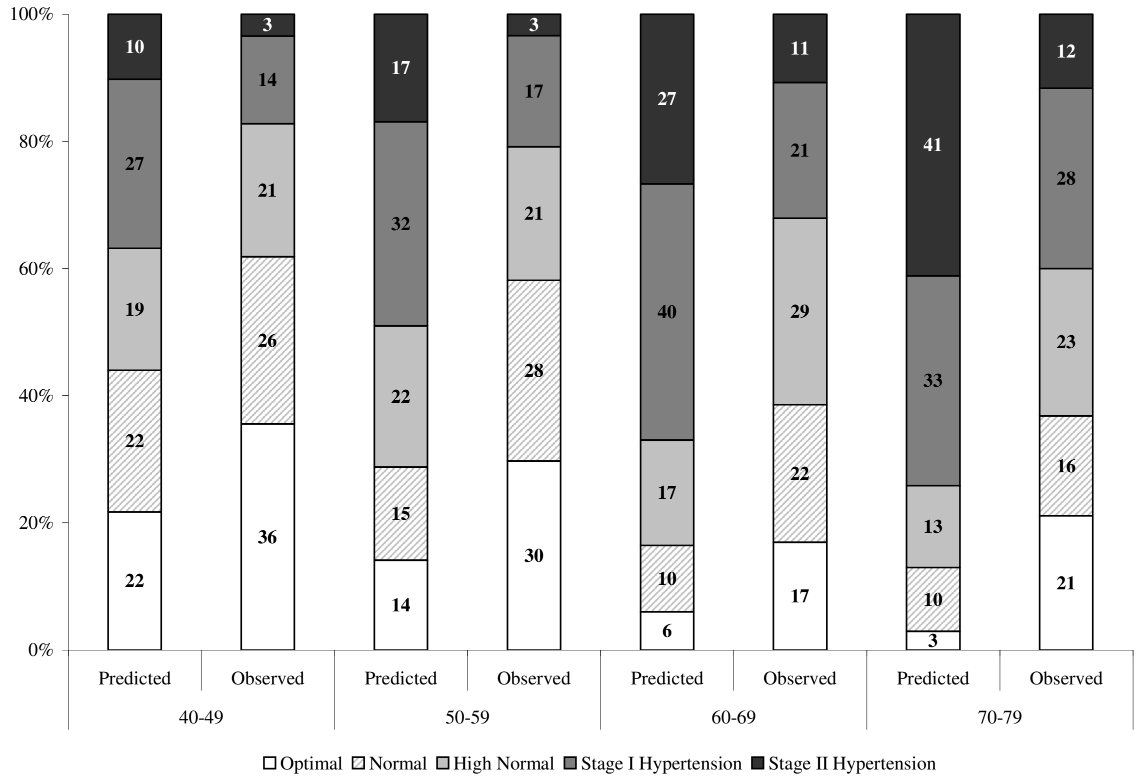
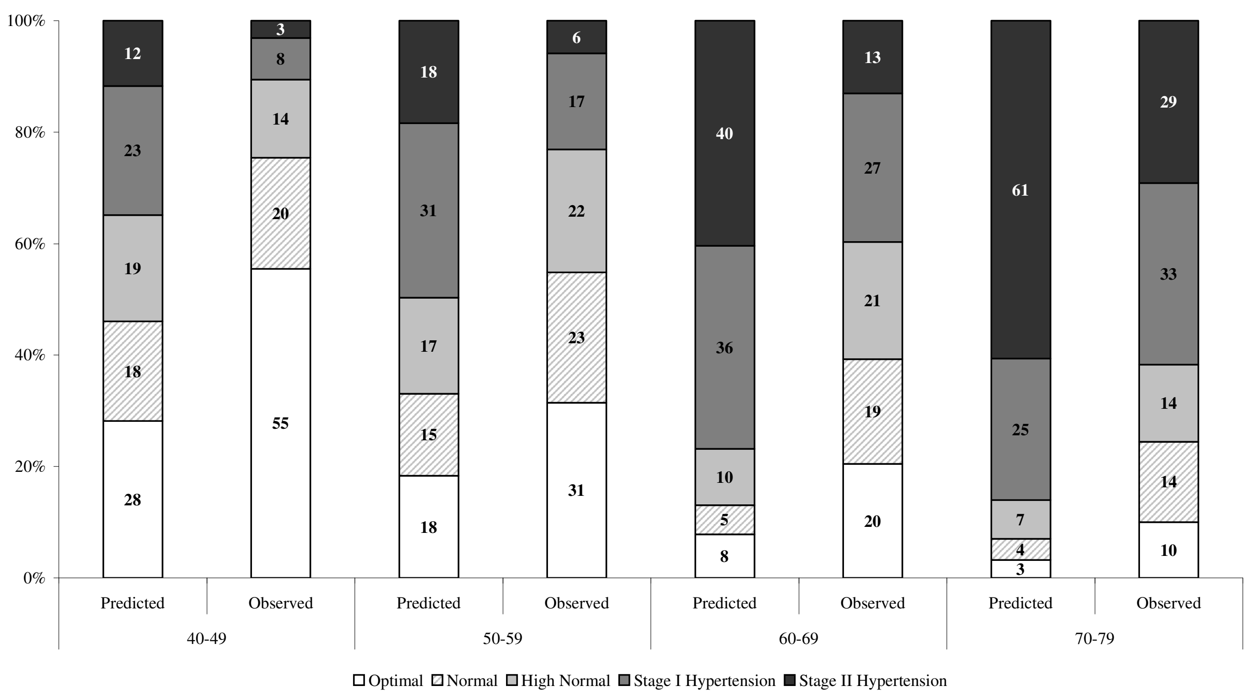
Wilson PWF, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation*. AHA; 1998.
Anderson KM, Odell PM, Wilson PWF, Kannel WB. Cardiovascular disease risk profiles. *Am Heart J*. 1990; 121: 293-8.

Figure 1: Predicted and Observed Blood Pressure, Men, 1999-2000



Predicted and Observed Blood Pressure, Women, 1999-2000



Optimal: SBP<120, DBP< 80; Normal: SBP 120-129, DBP 80-84; High Normal: SBP 130-139, DBP 85-89; Stage I: SBP 140-159, DBP 90-99; Stage II Hypertension: SBP>=160, DBP>=100

Figure 2: Predicted and Observed Deaths, 2001, and Hospital Discharges for Stroke and MI, 2002, Men and Women