Home-Measured Blood Pressure Is a Stronger Predictor of Cardiovascular Risk Than Office Blood Pressure
The Finn-Home Study

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Abstract—Previous studies with some limitations have provided equivocal results for the prognostic significance of home-measured blood pressure (BP). We investigated whether home-measured BP is more strongly associated with cardiovascular events and total mortality than is office BP. A prospective nationwide study was initiated in 2000 to 2001 on 2081 randomly selected subjects aged 45 to 74 years. Home and office BP were determined at baseline along with other cardiovascular risk factors. The primary end point was incidence of a cardiovascular event (cardiovascular mortality, nonfatal myocardial infarction, nonfatal stroke, hospitalization for heart failure, percutaneous coronary intervention, or coronary artery bypass graft surgery). The secondary end point was total mortality. After a mean follow-up of 6.8 years, 162 subjects had experienced a cardiovascular event, and 118 subjects had died. In Cox proportional hazard models adjusted for other cardiovascular risk factors, office BP (systolic/diastolic hazard ratio [HR] per 10/5 mm Hg increase in BP, 1.13/1.13; systolic/diastolic 95% confidence interval [CI], 1.05 to 1.22/1.05 to 1.22) and home BP (HR, 1.23/1.18; 95% CI, 1.13 to 1.34/1.10 to 1.27) were predictive of cardiovascular events. However, when both BPs were simultaneously included in the models, only home BP (HR, 1.22/1.15; 95% CI, 1.09 to 1.37/1.05 to 1.26), not office BP (HR, 1.01/1.06; 95% CI, 0.92 to 1.12/0.97 to 1.16), was predictive of cardiovascular events. Systolic home BP was the sole predictor of total mortality (HR, 1.11; 95% CI, 1.01/1.23). Our findings suggest that home-measured BP is prognostically superior to office BP. On the basis of the results of this and previous studies, it can be concluded that home BP measurement offers specific advantages more than conventional office measurement. (*Hypertension*. 2010;55:1346-1351.)

Key Words: epidemiology ■ blood pressure ■ hypertension ■ blood pressure monitoring

Hypertension, accounting for more than 7 million deaths annually, is currently one of the most important challenges facing public health care worldwide, and it has been recently identified as the leading global risk factor for mortality.[1] A meta-analysis of individual data from 1 million adults found each increase of 10 mm Hg in systolic office-measured blood pressure (BP) to increase the risk of cerebrovascular mortality by 40% and the risk of mortality from ischemic heart disease by 30%.[2]

Hypertension, however, cannot be prevented, detected, treated, or controlled without accurate and practical methods for BP measurement. Conventional office BP measurement performed by a doctor or a nurse has been the only method available on a large scale for the past century. However, during the past decade, the popularity of home BP measurement has exploded as small, easily and reliably operated automatic devices have been introduced to the market. A large part of the popularity of home BP monitoring can be attributed to its ease of use compared with other methods of measurement, particularly as it allows patients to measure their BP in their own homes.[3] Home BP measurement also seems to have medical advantages as it is free from the white-coat effect and is more strongly associated with hypertensive target organ damage than office BP.[4–6] More importantly, some studies,[7–9] though not all,[10,11] have suggested that home BP could have a stronger predictive power for future cardiovascular (CV) events than office BP.

Despite the numerous advantages of home BP measurement, the diagnosis and treatment of hypertension are still mainly based on office BP values. One reason for this is that outcome data on the prognostic significance of home BP has been limited and has provided equivocal results.[7–11] Previous studies that have assessed the association between home BP and prognosis have also had several limitations. They have been performed in a single community,[8–11] have included a limited number of patients,[10] have been performed with a

selected hypertensive study population,[7] have used a self-measurement protocol different from the proposed (but not established) guidelines,[7–11] have provided no data on total CV morbidity and mortality,[8,9,11] or have had a low number of recorded CV events.[8,10,11] As a result, hypertension guidelines have not yet recommended home BP measurement as the method of choice for measuring BP.[12,13]

The purpose of this study is to elucidate the prognostic significance of home-measured BP in a large-scale outcome study. This study investigated, for the first time in an unselected nationwide population sample using an up-to-date home-monitoring schedule, whether home-measured BP is more strongly associated than office BP with (1) overall CV events and (2) total mortality.

Materials and Methods

Subjects

The study sample was drawn from the participants of a multidisciplinary epidemiological survey, the Health 2000 study, which was carried out in Finland from the fall of 2000 to the spring of 2001. The study population was a stratified 2-stage cluster sample of 8,028 subjects drawn from the population register to represent Finnish adults aged 30 years or older. The stratification and sampling procedures have been previously described in detail.[14]

Of the subjects aged 45 to 74 years (n=4,388), 84% (n=3,672) agreed to participate in the interview and attended the health examination, and 2,120 subjects also participated in the home BP measurement substudy (Finn-Home study). Home measurement of BP was not performed on all subjects willing to participate because of the limited number of home monitors (approximately 800), and study subjects were selected on the basis of monitor availability. The characteristics of the study population are identical to the general Finnish population aged 45 to 74 years, as previously reported.[15] Subjects who had missing laboratory or health examination data (n=39) were excluded from the study. After subjects with 1 or more exclusion factors were removed, the study population consisted of 2,081 subjects aged 45 to 74 years.

The study protocol of the Health 2000 survey was approved by the Epidemiology Ethics Committee of the Helsinki and Uusimaa hospital region, and all participants gave signed informed consent.

Flow of the Study

At an initial health interview at the subject's home, basic background and sociodemographic information and information about health, illnesses, and the use of medication were gathered by centrally trained interviewers. Participants of the home measurement substudy then received home monitors for measuring BP during the week following the health interview. A physical examination was performed on each subject 1 to 6 weeks later at a local health center by centrally trained doctors and nurses. Each subject's height, weight, and office BP were measured, and fasting blood samples for serum lipids and glucose were taken. Details of the methodology of the project have been published elsewhere.[14]

BP Measurements

Office BP was measured by a nurse with a conventional, calibrated, mercury sphygmomanometer from the sitting individual's right arm after a 10-minute rest. The last 5 minutes of rest were spent in the measuring room with the cuff around the right upper arm. BP was measured using a pressure cuff of appropriate size and methods that were in accordance to current guidelines.[12,13] Systolic BP and diastolic BP were defined according to Korotkoff sounds I and V. Means of 2 measurements performed at a 2-minute interval were used to determine office BP.

Home BP was self-measured with a validated, automatic oscillometric device (Omron model HEM-722C, Omron Corp, Tokyo, Japan) according to the current guidelines.[16,17] Subjects received written instructions and individual guidance on how to measure BP correctly. Preparations for self-measurement of BP were the same as for clinic BP. Seated BP was measured twice, at an approximately 2-minute interval every morning between 6 AM and 9 AM and every evening between 6 PM and 9 PM on 7 consecutive days. Home BP was determined as the mean of 14 duplicate measurements (28 measurements). The mean number of performed home BP measurements was 26.7±3.7.

Follow-Up

Follow-up data were accumulated until December 31, 2007. Mortality data were obtained from the national mortality register based on death certificates. The 10th version of the International Classification of Diseases, Injuries, and Causes of Death (ICD) has been in use in Finnish death certificates and hospital discharge reports since 1996. Two independent investigators classified the deaths as CV or non-CV. ICD codes I21 to I25 (chronic or acute ischemic heart disease), I61 (intracerebral hemorrhage), I63 (cerebral infarction), I46 (sudden cardiac arrest), I11 (hypertensive heart disease), I71.3 (ruptured abdominal aortic aneurysm), and I70.2 (peripheral vascular disease) were classified as CV deaths. Heart failure is not approved as a primary cause of death in Finland because an underlying reason must always be identified.

Data on hospitalization due to heart failure and nonfatal coronary and stroke events were obtained from the national hospital discharge register. ICD codes I21 to I23 were classified as acute coronary events, ICD codes I61 and I63 as acute stroke events, and subjects with an ICD code I50 were classified as being hospitalized because of acute heart failure. In addition, information on coronary interventions and coronary artery bypass graft surgery performed was obtained from the hospital discharge register. The data of the Finnish hospital discharge register and national mortality register have been validated on stroke and coronary heart disease diagnoses.[18,19]

The primary end point was the combination of CV mortality, nonfatal myocardial infarction, nonfatal stroke, hospitalization for heart failure, percutaneous coronary intervention, and coronary artery bypass graft surgery. Only the first event was included in this analysis. The secondary end point was total mortality.

Statistical Analyses

We used Cox proportional hazard models for multivariate analyses. Association of home and office BP with the end points was analyzed by estimation of the hazard ratios and their 95% confidence intervals per 10/5 mm Hg increase in systolic/diastolic BP. If reported, the models were adjusted for gender, age, use of antihypertensive medication, past history of CV disease (history of stroke, heart failure, or ischemic heart disease), smoking status (daily use of tobacco products), presence of diabetes (fasting serum glucose level ≥7.0 mmol/L or a history of use of oral hypoglycemic agents or insulin injection), and presence of hypercholesterolemia (fasting serum total cholesterol level of ≥7.0 mmol/L or use of statins).

Categorical variables were compared using the χ^2 test and continuous variables using the Student t test. A probability value <0.05 was considered statistically significant. Data are reported as mean±standard deviation. Database management and statistical analysis were performed with SAS software (SAS Institute, Cary, NC), version 9.1.

Results

Entry and Follow-Up Data

The general characteristics of the study population are reported in Table 1. Overall, men had slightly worse risk factor profiles compared with women. The population characteristics are very close to those of the general Finnish population aged 45 to 74 years, as previously reported.[15] Office BP was significantly higher than home BP in the whole population (137.4±20.2/83.7±10.6 versus 129.8±18.8/80.4±9.5, P<0.001).

Table 1. Baseline Characteristics of the Study Population (n=2081)

Characteristic	Male	Female
No. of subjects	964 (46.3)	1117 (53.7)
Age, years (SD)	56.0 (8.2)	56.6 (8.8)
BMI, kg/m² (SD)	27.6 (3.8)	27.3 (5.0)
Current smokers, n (%)	229 (23.8)	178 (15.9)*
Diabetes, n (%)	85 (8.8)	45 (4.0)*
Previous CV event, n (%)	129 (13.4)	100 (9.0)*
Pharmaceutical treatment for, n (%)		
Hypertension	205 (21.3)	267 (23.9)
Diabetes	48 (5.0)	31 (2.8)*
Hypercholesterolemia	100 (10.4)	86 (7.7)*
Office BP, mm Hg (SD)		
Systolic	138.1 (18.7)	136.8 (21.5)
Diastolic	86.0 (10.4)	81.8 (10.4)*
Home BP, mm Hg (SD)		
Systolic	132.5 (16.4)	127.4 (20.4)*
Diastolic	82.5 (9.3)	78.5 (9.3)*
Fasting glucose, mmol/L (SD)	5.8 (1.5)	5.5 (1.2)*
Fasting total cholesterol, mmol/L (SD)	6.1 (1.1)	6.2 (1.1)

*$P<0.05$; differences between men and women. BMI indicates body mass index.

The follow-up period ended on December 31, 2007, and the mean follow-up time was 6.8 years, resulting in 14 081 person-years of follow-up. During the follow-up period, there were 118 deaths (incidence, 8.4/1000 person-years), of which 37 (incidence, 2.6/1000 person-years) were of CV origin. The causes of death and their respective frequencies are reported in Table 2. During the follow-up period, 162 subjects had at least 1 CV event (incidence, 11.5/1000 person-years). The origins of these events are reported in Table 3.

BP and CV Risk

In a univariate analysis, male gender, age, body mass index, smoking, diabetes, hypercholesterolemia, previous CV events, office BP, and home BP were associated with future CV events. The same applied for total mortality, except for

Table 2. Causes of Death

Causes	n	% of Deaths	% of CV Deaths
All deaths	118	100.0	NA
Deaths of CV origin	37	31.4	100.0
Ischemic heart disease	26	22.0	70.3
Stroke	5	4.2	13.5
Hypertensive heart disease	2	1.7	5.4
Aortic aneurysm rupture	2	1.7	5.4
Sudden cardiac arrest	1	0.9	2.7
Peripheral vascular disease	1	0.9	2.7
Deaths of non-CV origin	81	68.6	NA
Cancer	44	37.3	NA
Injury or poisoning	14	11.9	NA
Other	23	19.5	NA

NA indicates not available.

Table 3. First Cardiovascular Events During Follow-Up

Event	n	% of Population	% of CV Events
Total no. with ≥1 CV event	162	8.0	100.0
Nonfatal myocardial infarction	35	1.7	21.6
Nonfatal stroke	35	1.7	21.6
Hospitalization for heart failure	27	1.3	16.7
Death of CV origin	25	1.2	15.4
Percutaneous coronary intervention	21	1.0	13.0
Coronary artery bypass graft surgery	19	0.9	11.7

body mass index, hypercholesterolemia, and diastolic office BP, for which no association was found. In addition, high home and office heart rate were associated with total mortality (Supplemental Table, available online at http://hyper.ahajournals.org).

In an unadjusted Cox regression model, all BPs were predictive of CV events and total mortality, except for diastolic office BP, which was not predictive of total mortality (Table 4). After adjustment for other risk factors for CV disease, all BP measurements were still predictive of CV risk, but only systolic home BP was predictive of total mortality ($P=0.04$, Table 4).

When systolic home BP and systolic office BP were entered in the same adjusted multivariate model (Table 5), only systolic home BP was a significant predictor of CV events ($P<0.001$), whereas systolic office BP was not a significant predictor of CV events ($P=0.80$). When entering diastolic home BP and diastolic office BP in the same model, a similar result was found, and only diastolic home BP was a significant predictor of CV events ($P=0.002$), whereas diastolic office BP was not a significant predictor of CV events ($P=0.19$).

The Figure shows the calculated absolute 6.8-year risk of all-cause mortality and CV events. The increase in CV risk per 1 mm Hg increase in BP was greater for home BP than for office BP.

Discussion

This study demonstrates in an unselected nationwide population that home and office BP are both predictive of overall CV events. However, home BP values provide prognostic information about CV risk and total mortality above and beyond that of office BP, even with a low number of measurements. The changes in CV risk are steeper with an increase in home compared with office BP.

Our study has been able to elucidate the important prognostic value of home measured BP, having the benefit of data on total CV mortality and morbidity in an unselected nationwide population using an up-to-date home monitoring schedule. The previously published studies concerning this topic have provided equivocal results and have suffered from some limitations. To date, 4 studies have provided prognostic information for home BP: the Presioni Arteriose Monitorate e

Table 4.   Hazard Ratios for Occurrence of CV Events With a Systolic/Diastolic BP Increase of 10/5 mm Hg

	Unadjusted Model				Adjusted Model*			
	Fatal and Nonfatal CV Events (n=162)		Total Mortality (n=118)		Fatal and Nonfatal CV Events (n=162)		Total Mortality (n=118)	
BP Variable	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value	HR (95% CI)	P Value
Systolic BP								
Home	1.41 (1.31–1.51)	<0.001	1.28 (1.18–1.40)	<0.001	1.23 (1.13–1.34)	<0.001	1.11 (1.01–1.23)	0.04
Office	1.24 (1.16–1.33)	<0.001	1.15 (1.06–1.25)	<0.001	1.13 (1.05–1.22)	0.001	1.05 (0.96–1.15)	0.32
Diastolic BP								
Home	1.21 (1.14–1.28)	<0.001	1.11 (1.02–1.21)	0.02	1.18 (1.10–1.27)	<0.001	1.08 (0.98–1.12)	0.14
Office	1.12 (1.04–1.20)	0.003	0.93 (0.85–1.01)	0.09	1.13 (1.05–1.22)	0.002	0.95 (0.87–1.04)	0.26

*Data adjusted for gender, age, smoking status, history of cardiovascular events, presence of diabetes mellitus, presence of antihypertensive medication, and presence of hypercholesterolemia. HR indicates hazard ratio; CI, confidence interval.

Loro Associazioni (PAMELA), Self-Measurement of Blood Pressure at Home in the Elderly: Assessment and Follow-up (SHEAF), Didima, and Ohasama studies.[7–11] In the PAMELA and Didima studies, the overall ability to predict death was not greater for home BP than for office BP.[10,11] However, only 56 CV deaths and 67 CV events were recorded in the PAMELA and Didima studies, respectively. This resulted in low statistical study power, and no definite conclusions could be drawn. A third population study, the Japanese Ohasama study, concluded that systolic home BP had a stronger predictive power for CV mortality than did screening BP, although again only 52 CV deaths were recorded.[8] However, the results of the Ohasama study appear to be the most reliable of the previous studies because subsequent follow-up studies of the Ohasama population have confirmed these findings with additional stroke and CV mortality data, although cardiac morbidity or procedure data are not available.[9] One limiting factor of all 3 previously published population studies (PAMELA, Didima, and Ohasama) is also that they have involved populations living in a single community, instead of nationwide populations, limiting their general applicability. In addition to population studies, 1 study with a study cohort consisting of hypertensive patients having 324 recorded CV events, the SHEAF study, has been published.[7] The authors of that study concluded that home BP measurement had a better prognostic accuracy than office BP mea-

surement. However, the study included only elderly, treated hypertensive patients and no data relating to the changes in treatment were collected, so its results cannot be extrapolated to the population level. In addition, the self-measurement protocols of all 4 previously published studies differ from the currently proposed guidelines, although no established worldwide protocol yet exists.[7–11]

The increase in CV risk increases more steeply with home BP than with office BP. This observation has been made previously in the PAMELA and Ohasama studies.[11,20] However, this steeper relation between home BP and risk can be explained by the fact that home BP values are the means of a

Table 5.   Adjusted Hazards Ratio for Occurrence of Fatal and Nonfatal CV Events With a Systolic/Diastolic BP Increase of 10/5 mm Hg

BP Variable	HR (95% CI)	P Value
Systolic BP		
Home	1.22 (1.09–1.37)	<0.001
Office	1.01 (0.92–1.12)	0.80
Diastolic BP		
Home	1.15 (1.05–1.26)	0.002
Office	1.06 (0.97–1.16)	0.19

Both BP values were included in the Cox proportional hazards model. Data adjusted for gender, age, smoking status, history of cardiovascular events, presence of diabetes mellitus, presence of antihypertensive medication, and presence of hypercholesterolemia. HR indicates hazard ratio; CI, confidence interval.
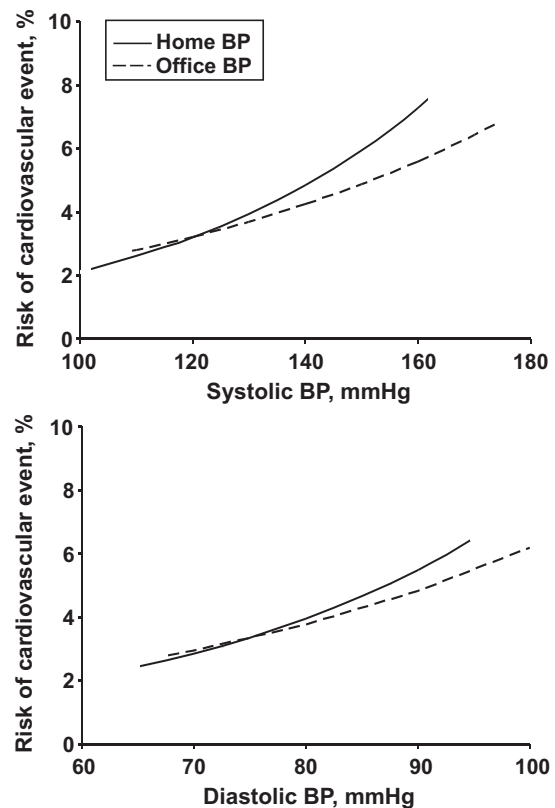

Figure. Office and home systolic and diastolic BP as predictors of 6.8-year risk of CV events. The values have been plotted to span the 5th to 95th percentile interval. The figure is adjusted for other CV risk factors.

large number of values and are consequently distributed over a narrower range than are the means of office BP values, which are usually obtained from a very few measurements. The narrower range of home BP values does not by itself necessarily imply a greater predictive ability. Our results still clearly indicate that physicians must keep in mind that an increase in BP leads to a greater increase in risk if based on home values than on office values, especially when the BP is highly elevated.

The results of our study confirm that home BP measurement provides physicians with BP values that reflect their patients' true BP more accurately than conventional office BP measurements. Besides having a prognostic superiority and a stronger association with end-organ damage,[4–6] home BP measurement also offers other clear advantages over conventional office BP measurement. Home BP measurement allows the identification of white-coat and masked hypertension with readings under standardized conditions, little measurement variability, and good reproducibility.[21] Home monitoring is also the method most preferred by patients, and it can lead to better BP control by increasing awareness of hypertension and compliance with drug treatment.[3,22,23]

Some of the benefits of home BP measurement, such as a better prognostic value compared with office BP, have also been demonstrated for ambulatory BP monitoring.[24] However, ambulatory monitoring is a costly, laborious, and uncomfortable procedure, usually performed in special hypertension clinics. The potential benefit of ambulatory monitoring on a population level will undoubtedly be minuscule and will be far more costly. Home BP monitoring, on the other hand, can be easily performed in the primary healthcare environment. In Finland, 60% of the patients treated for hypertension already possessed a home BP monitor in 2006.[25] Furthermore, the shortcomings of home BP measurement, such as poor accuracy of some home monitors,[13,26] reporting bias,[27] and lack of patient instruction, can be avoided with good patient training and by using only validated and calibrated home monitors.

There are some limitations in our study. Office BP was measured on only 1 day, and home BP readings were performed twice daily for 7 days. Therefore, we cannot exclude the possibility that taking office BP values over multiple days could have increased the association between office BP and morbidity. However, office BP was very meticulously assessed, which is often not the case in the hectic everyday practice of the clinician. Furthermore, home BP measurement always produces a greater number of measurements than office measurement in actual practice. Further follow-up of our study cohort is also necessary to validate our results and to obtain enough events for disease subtype analyses.

Perspectives

Results from our nationwide prognostic study show that home-measured BP is prognostically superior to office BP. On the basis of the results of this study and data from previous studies, it can be concluded that home BP measurement offers specific advantages over conventional office measurement. Home monitoring of BP is a convenient, accurate, and widely available option with no risk of white-coat and masked hypertension and should become the method of choice for diagnosing and treating hypertension. A paradigm shift is needed in BP measurement, as evidence-based medicine suggests that office BP measurement should mainly be restricted to screening purposes.

Disclosures

None.

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