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The Relative Utility of Home, Ambulatory, and Office Blood Pressures in the Prediction of End-Organ Damage

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Abstract

Background—Home blood pressure (HBP) monitoring plays an increasingly important role in the diagnosis and treatment of hypertension. We evaluated the independent value of HBP compared to ambulatory blood pressure (ABP) and office blood pressure (OBP) in the prediction of cardiovascular end-organ damage in normotensive subjects and untreated patients with mild hypertension.

Methods—163 subjects underwent measurements of OBP, HBP, ABP, and echocardiography. A physician using a mercury column sphygmomanometer performed three OBP measurements. ABP was recorded using a noninvasive ambulatory monitor (mean 35.4 awake readings per subject). Participants took HBP readings with an automatic, oscillometric device over a 10-week period (mean 277.9 readings per subject). Left ventricular mass index (LVMI) was calculated from measurements obtained from 2-dimensionally guided M-mode or linear tracings on echocardiography.

Results—For systolic and diastolic blood pressures (SBP/DBP), the correlation coefficients of LVMI with OBP, awake ABP, and HBP were .29/.27, .41/.26, and .47/.35 respectively (all $p < .01$). In a multivariate regression analysis in which age, sex, body mass index, OBP, awake ABP, and HBP were included, only age, sex, and HBP were significant predictors of LVMI. When only the first 12 home readings were used, the superiority of HBP was no longer evident.

Conclusions—In contrast to OBP and ABP, HBP measurements, when averaged over a 10-week period, are independently related to LVMI. HBP adds prognostic information over and above OBP and ABP in the prediction of cardiovascular end-organ damage, but this relation appears to depend on the number of readings taken.

Keywords

Hypertension; home blood pressure monitoring; ambulatory blood pressure monitoring; echocardiography

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Introduction

Elevated blood pressure is a strong, independent risk factor for incident cardiovascular disease.¹ The bulk of our knowledge about the prognostic value of elevated blood pressure is based on the traditional method of taking auscultatory measurements in a clinic setting. However, there is evidence that ambulatory blood pressure (ABP) is a better predictor of target end-organ damage and cardiovascular events than office blood pressure (OBP) in the general population as well as in hypertensive patients.²⁻⁴ Thus, it is generally accepted that ABP monitoring (ABPM) is a superior method of blood pressure assessment, and is increasingly viewed as the “gold standard” for clinical practice.⁵

Another way of measuring blood pressure is by self-monitoring at home. The risks of target end-organ damage⁶ and cardiovascular events⁷ are more strongly correlated with home blood pressure (HBP) than with OBP measurements. Although ABP has been proposed as the best measure of target organ damage, ABPM is expensive and impractical for most patients. These limitations, however, are far less applicable to HBP monitoring.⁸

Although ABP and HBP both appear to be related to left ventricular mass index (LVMI), a marker of cardiovascular end-organ damage,⁶⁻⁹⁻¹⁰ only one previous study in moderately to severely hypertensive patients, a group already at increased cardiovascular risk, has examined the independent predictive utility of these measures vis-a-vis their relation to LVMI.¹¹ Before HBP monitoring is routinely recommended for blood pressure assessment, the relative predictive value of HBP in comparison to ABP needs to be assessed in a broader group of individuals.

Therefore, the aim of the present study was to assess the relative utility of HBP, ABP, and OBP measurements for the prediction of elevated LVMI in mildly hypertensive patients and normotensive individuals.

Methods

Study Population

This study was conducted as part of an investigation of psychobehavioral mechanisms of white coat hypertension.¹²⁻¹³ Briefly, patients with Stage 1 hypertension (140-159 mmHg/90-99 mmHg), according to Joint National Committee (JNC VI) criteria, were recruited initially from the Weill Cornell Hypertension Center of New York Presbyterian Hospital and later from Mount Sinai Medical Center in New York City. Normotensive participants were recruited through advertisements. For this study, participants were eligible if they: (1) were aged between 18 and 80 years, (2) were willing, with physician's permission, to come off antihypertensive medication for 2 weeks prior to the first study visit, and remain off for the duration of the study, and (3) had no history of overt cardiovascular disease.

A total of 329 participants were enrolled between June 1998 and August 2003. Those recruited prior to May 15, 2002 or between November 20, 2002 and February 15, 2003 had an opportunity to have an echocardiogram (N=233). 211 of the subjects had usable OBP, ABP, and HBP data, and 177 (83.9%) of these had an echocardiogram to assess LVMI. Compared to the 34 non-participants who did not have an echocardiogram, the 177 participants were more likely to be female (60.5% of the participants vs. 50.0% of the non-participants, $p=.047$). However, there were no differences in age ($p=.25$) or body mass index (BMI, $p=.50$) between the two groups. LVMI could not be determined from 14 of the 177 echocardiograms due to poor acoustic penetration, resulting in 163 participants who are the focus of the present report. Written informed consent was obtained from all subjects, and the

study was approved by the Institutional Review Boards of Weill Medical College of Cornell University and Mount Sinai School of Medicine.

Blood Pressure Assessments

Participants attended visits on two consecutive days (Day 1 and Day 2). Starting on Day 1, ABP measurements were performed using an oscillometric Spacelabs ABP monitor (Model 90207, Redmond, WA) over a period of 36 hours. For about three-quarters of the recordings, the ABPM was programmed to take a blood pressure reading every 30 minutes throughout the monitoring period. For the remaining recordings, measurements were taken at 15-minute intervals, between 6 A.M. and 10 P.M., and every 30 minutes between 10 P.M. and 6 A.M. For the purposes of this study, the analyses of ABP readings were restricted to the first 24 hours of monitoring, as most of the published studies examining the relationship of LVMI with both ABP and HBP utilized 24-hour ABPM.^{6-9,10} Awake and sleep systolic blood pressure (SBP) and diastolic blood pressure (DBP) levels were defined based on diary reports of the times subjects woke up and went to sleep. The mean numbers of valid measurements used to compute the mean awake and sleep ABP were 35.4 ± 12.7 and 14.9 ± 4.0 respectively.

The subject, still wearing the ABP monitor, returned the next day (Day 2) where physician-obtained OBP readings were taken. The participant was escorted into an examination room and rested for at least 5 minutes, after which the physician entered and took three OBP measurements using a mercury column sphygmomanometer and stethoscope.

After the initial ABP recording, HBP was measured over a 10-week period using an Omron HEM-747 IC, an automatic, oscillometric HBP monitor (Omron Health Care, Vernon Hills, IL), which has previously been validated.¹⁴⁻¹⁶ The patients were instructed to take three HBP measurements on four days a week in the morning and evening; they were also asked to take three additional measurements on two occasions (at mid-morning and mid-afternoon) on two days a week, for a total of 36 measurements a week. The mean number of valid HBP readings per subject was 277.9 ± 111.2 .

Subjects were additionally classified as hypertensive by OBP, ABP, and HBP measurements. Office hypertension was defined as a SBP ≥ 140 mmHg or DBP ≥ 90 mmHg. Ambulatory hypertension, based on mean awake blood pressure, and home hypertension were both defined as SBP ≥ 135 or DBP ≥ 85 mmHg, which are internationally accepted limits.^{17,18}

Measurement of Cardiovascular End-organ Damage: Left Ventricular Mass Index

M-mode and two-dimensional echocardiograms were performed approximately 10 weeks after the initial assessment visits. Researchers blinded to patients' clinical characteristics performed left ventricular measurements from two-dimensionally guided M-mode tracings according to the recommendations of the American Society of Echocardiography.¹⁹ If an M-mode tracing was technically inadequate, linear measurements were performed from the two-dimensional study.²⁰ Up to six echocardiographic cycles were measured and averaged. Left ventricular mass (LVM) was calculated using the corrected American Society of Echocardiography formula: $0.8 \times (1.04 \times [(IVS + LVIDd + PWT)^3 - LVIDd^3]) + 0.6$. LVM index (LVMI) was calculated by dividing LVM by estimated body surface area, calculated from height and weight. LVMI was also used to identify subjects with left ventricular hypertrophy (LVH), defined as LVMI ≥ 125 g/m² for males and ≥ 110 g/m² for females as previously described.²¹

Statistical Analyses

Results are presented as mean $\pm$ SD. Fisher's exact test and independent samples t-tests were used to compare proportions and means respectively. Paired t-tests were used to test for differences in mean levels of the various blood pressure measures. Pearson correlations were used to assess the associations of the OBP, ABP, and HBP measurements with LVMI. To assess the independent predictive utility of the different blood pressure measurements, multiple regression models were estimated including age, sex, and BMI as covariates, and the blood pressure measurements as primary predictors. Separate analyses were performed for SBP and DBP. Variance inflation factors were calculated to examine the possible existence of substantial multicollinearity among the predictors. The validity of the regression model's inferential statistics was assessed using the bootstrap technique.^{22,23} One thousand random bootstrap samples were drawn (with replacement) from the full sample. Statistical significance was defined by $\alpha=0.05$ level, 2-tailed. Statistical analyses were performed using SPSS v.11 (SPSS Inc, Chicago, IL) and SAS v.8.2 (SAS Institute, Cary, NC).

Results

Table 1 shows the characteristics of the study participants. There were significant differences among the blood pressure measures (OBP, awake ABP, sleep ABP, and HBP) for both SBP ($p<.001$) and DBP ($p<.001$). T-tests indicated that HBP measurements were significantly lower than both OBP and awake ABP measurements. There were no significant differences in SBP or DBP between the OBP and awake ABP measurements. Sleep ABP levels were significantly lower than HBP, OBP, and awake ABP.

The Relations Among Blood Pressure Levels and Cardiovascular End-Organ Damage

The correlation coefficients of LVMI with OBP, awake ABP, sleep ABP, and HBP were .29/.27 (SBP/DBP; $ps<.001$), .41/.26 (SBP/DBP; $p<.001$ for SBP and $p<.01$ for DBP), .32/.18 (SBP/DBP; $p<.001$ for SBP and $p=.02$ for DBP), and .47/.35 (SBP/DBP; $ps<.001$) respectively. For SBP, the correlations of LVMI with awake ABP and HBP did not differ significantly, but both were significantly greater than the correlation of LVMI with OBP ($p<.05$ for ABP and OBP comparison, $p<.01$ for HBP and OBP comparison). However, the correlation of LVMI with sleep ABP was significantly less than with HBP ($p<.01$), and was not significantly different than with OBP. For DBP, none of the differences among the correlations of LVMI with awake ABP, HBP, and OBP were significant. Further, the correlation of LVMI with sleep ABP was significantly less than its correlation with HBP ($p<.01$).

Separate age-, sex-, and BMI-adjusted models were examined for each of the blood pressure measures. Each blood pressure measure was a significant predictor of LVMI, independent of age, sex, and BMI: OBP ($p=.01$ for SBP, $p<.01$ for DBP); awake ABP ($ps<.01$ for SBP and DBP); and HBP ($ps<.01$ for SBP and DBP). The percentages of variance in LVMI that were predicted by age, sex, BMI, and each of the blood pressure measures are shown in Table 2 (Models 1-7). Sleep systolic ABP (data not shown in Table 2) was also a significant predictor ($p=.02$) of LVMI, independent of age, sex, and BMI ($R^2=.21$). Sleep diastolic ABP was not a significant predictor of LVMI ($p=.22$), after adjusting for age, sex, and BMI.

A multiple regression model (Table 3) was then estimated predicting LVMI from age, sex, BMI, and all 3 blood pressure measures (OBP, ABP, and HBP). Because the relation of LVMI with awake ABP appeared to be stronger than with sleep ABP in our study, awake ABP was used in this model. As Table 3 shows, age and sex were significant predictors of LVMI, when controlling for all other predictors. Of the blood pressure measures, only HBP

(SBP and DBP) emerged as a significant independent predictor of LVMI. The variance inflation factors for OBP, awake ABP and HBP ranged from 1.97 to 3.69 (both SBP and DBP), indicating no strong evidence of multicollinearity among the predictors.²³ As expected, the bootstrap analysis resulted in larger estimates of standard errors and somewhat weaker p-values. Nevertheless, the conclusions were similar for SBP ($p=.01$ for age, $p=.02$ for sex, $p=.19$ for BMI, $p=.25$ for OBP, $p=.49$ for awake ABP, $p=.02$ for HBP), and DBP ($p<.001$ for age, $p=.004$ for sex, $p=.06$ for BMI, $p=.69$ for OBP, $p=.65$ for awake ABP, $p=.09$ for HBP), although diastolic HBP was not quite statistically significant.

To determine the incremental variation in LVMI accounted for by HBP, over and above OBP and awake ABP, a multivariate regression model was estimated including age, sex, and BMI on the first step, OBP on the second step, awake ABP on the third step, and then HBP on the final step (see Table 2, Models 8-11). For SBP only, awake ABP was statistically significant when added to the model that included age, sex, BMI, OBP. For both SBP and DBP, HBP was statistically significant when added to the model that included age, sex, BMI, OBP, and awake ABP.

Table 4 shows the number and percentage of patients who had LVH, by presence of office, ambulatory, and home hypertension. LVH was significantly more common in subjects who had home hypertension compared to subjects who had normal HBP levels (10.1% vs 2.1%). In contrast, the associations of LVH with office and ambulatory hypertension were not statistically significant.

To determine whether findings would be similar with a smaller number of HBP readings, post-hoc analyses were performed with the HBP levels averaged over the first 12 assessments. Both home SBP and DBP were significantly correlated with LVMI ($r=.34$, $p<.001$ for SBP; $r=.22$, $p=.006$ for DBP), but these correlations were lower than those for the full set of home readings and for awake ABP. In the multivariate regression analysis including age, sex, BMI and the three SBP measures (office, awake ambulatory, and home), age ($p=.02$), sex ($p=.01$), BMI ($p=.04$), and ABP levels ($p=.006$) were significant predictors of increased LVMI. In the analysis including the three DBP measures, age ($p<.001$), sex ($p=.007$), and BMI ($p=.03$) were the only significant predictors. OBP and HBP were not significant independent predictors of LVMI for either SBP ($p=.72$ for office, $p=.72$ for home) or DBP ($p=.23$ for office, $p=.96$ for home). Further, awake ambulatory DBP ($p=.18$) was not a significant independent predictor of LVMI. Finally, when the diagnosis of home hypertension was based on the average of 12 readings, there was no longer a significant association between LVH and home hypertension ($p=.17$).

Discussion

The results of our study show that HBP, measured over a 10-week period, was an independent predictor of LVMI after adjusting for age, sex, BMI, OBP, and awake ABP. In contrast, neither OBP nor awake ABP were independently related to LVMI. In addition, a diagnosis of hypertension based on HBP monitoring was related to echocardiographic LVH while diagnoses based on ABPM and OBP were not.

Other investigators have found that ABP and HBP are both correlated with LVMI.^{6-9,10} However, these studies did not examine whether these measures are redundant or independent in their relation with LVMI. Our findings confirm the correlations reported in these earlier studies, but suggest that while awake ambulatory SBP has independent prognostic value over and above OBP, only HBP (for both SBP and DBP) is independently associated with an increased risk of cardiovascular end-organ damage over and above OBP and ABP. Thus, when sufficient numbers of readings are taken, HBP may be a better

indicator than ABP or OBP of cardiovascular end-organ damage. Our results are consistent with data published by Jula et al.,¹¹ who found that HBP, measured twice, two times a day for seven consecutive days (total of 28 readings), was independently associated with LVMI after adjusting for age, sex, OBP, and various ABP measurements in patients with moderate to severe hypertension. Our study sample was comprised of normotensive participants and mild hypertensive patients, indicating that the superior predictive value of HBP over ABP and OBP for cardiovascular end-organ damage may generalize to a broader population.

In the Ohasama study, the first to establish the prognostic value of home readings, the prediction of risk of stroke became stronger with more home readings; the highest predictive value was found with a minimum of 14 and an average of 25 measurements.²⁴ Similarly, a recently published systematic review conducted by Verbeek et al.⁸ concluded that the minimum number of home measurements needed to obtain a reliable estimate of a subject's usual blood pressure is 12 (i.e. two in the morning and two in the evening for three consecutive days). Our findings that significant correlations were observed between HBP and LVMI, even when HBP was analyzed using the first 12 readings, are supportive of these conclusions. However, in this latter scenario, HBP was no longer independently predictive of LVMI, over and above OBP and ABP. With the full set of HBP measurements, we speculate that HBP was the only independent predictor of increased LVMI because the mean number of HBP readings, and therefore the resultant measurement reliability, was substantially greater than the number of ABP and OBP readings.

Our results are at odds with a recent report from the Pressioni Arteriose Monitorate e Loro Associazioni (PAMELA) study.²⁵ Although HBP in the PAMELA study was determined by averaging only two measurements, there was an increasing risk of cardiovascular death in subjects in whom HBP and ABP were normal to those with elevations in one or both blood pressure levels, suggesting that HBP and ABP have independent predictive values. It is likely that the prognostic value of HBP monitoring increases progressively with the number of measurements. Thus, the minimum number of HBP readings needed to maintain at least an equivalent predictive value compared to ABP is unclear, and should be further investigated. This question is particularly relevant as HBP monitoring, based on hundreds of readings over a 10-week period, as performed in our study, may be considered impractical for many patients. However, the device we used did not require the subject to write down the readings, and monitors now being developed have memories that can accommodate several hundred readings. Therefore, this is less likely to be an issue in the future. Further, the minimum number of HBP readings is also likely to be dependent on the study sample and machine characteristics. A sample better trained in HBP monitoring, a sample with less day-to-day variability, or a more reliable machine, may reduce the number of HBP measurements required.

HBP monitoring is an economical and widely available method of blood pressure assessment. Many HBP monitors have passed the British Hypertension Society and American Association of Medical Instrumentation validation criteria.^{14,26,27} The monitors are easy to use, as oscillometric measurement algorithms are used, which do not require location of one's brachial artery for cuff placement. The method requires little work on the part of the health provider, unlike ABPM which must be downloaded at the physician's office, and the data uploaded to software programs designed for this purpose. From the clinician's point of view, HBP monitoring may hold many advantages over ambulatory monitoring.

There are potential limitations of our study. Given the relatively small percentage of patients with LVH, caution is warranted in interpreting the finding that LVH was only related to home and not office or ambulatory hypertension. Three OBP measurements were performed

during one visit, and ABPM was performed only once. Therefore, we cannot exclude the possibility that HBP would not have been an independent predictor of LVMI, if a larger number of OBP and/or ABP assessments were obtained. Another limitation is that OBP and ABP measurements were performed at the beginning of the study, closer to when antihypertensive medications were withdrawn in treated patients, while HBP readings were obtained over the study period. Because echocardiograms were performed approximately at the end of the 10-week period, it is possible that the relation between LVMI and HBP was less influenced by any lingering effects of medications beyond the 2-week washout period. Finally, in our study, HBP levels were significantly lower than awake ABP levels, yet the same diagnostic threshold (i.e. 135/85 mmHg) was used to classify home and ambulatory hypertension respectively. However, it is generally accepted that the diagnostic threshold for both HBP and awake ABP is 135/85 mmHg.²⁸ Further, since HBP readings are taken at rest, and awake ABP readings include periods of activity, it is not unusual for awake ABP readings to be higher. ok

In conclusion, our data suggest that, compared to OBP and ABP, HBP measurements, when averaged over a 10-week period, are independently related to cardiovascular end-organ damage. Further research is needed to determine the minimum number of readings required for HBP to be at least an equivalent predictor of cardiovascular end-organ damage and long-term cardiovascular events compared to ABP, and to determine the threshold for HBP at which treatment should be initiated or adjusted.^{8,29}

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Table 1

Sample characteristics*

Characteristics	Total Sample (N=163)
Age, y	53.9 ± 14.5
Sex, % female	60.1
Race	
% White (Non-Hispanic)	66.3
% White (Hispanic)	8.6
% Black (Non-Hispanic)	13.5
% Black (Hispanic)	0.6
% Asian/Indian/Pacific Islander	6.1
% Native American/Alaskan Native	0.6
% Other	4.3
Body Mass Index, kg/m ²	25.9 ± 4.9
Office	
Systolic blood pressure, mmHg	134 ± 21
Diastolic blood pressure, mmHg	83 ± 12
% Hypertensive [†]	46.0
Ambulatory (awake)	
Systolic blood pressure, mmHg	134 ± 14
Diastolic blood pressure, mmHg	82 ± 10
% Hypertensive [‡]	57.1
Ambulatory (sleep)	
Systolic blood pressure, mmHg	119 ± 14 ^{//}
Diastolic blood pressure, mmHg	69 ± 11 ^{//}
Home	
Systolic blood pressure, mmHg	130 ± 16 [§]
Diastolic blood pressure, mmHg	79 ± 10 [§]
% Hypertensive [‡]	42.3
Left Ventricular Mass, g	145 ± 46
Left Ventricular Mass Index, g/m ²	78.6 ± 18.7

* Data are expressed as percentage or mean ± SD.

[†] Defined by systolic blood pressure ≥ 140 or diastolic blood pressure ≥ 90.

[‡] Defined by systolic blood pressure ≥ 135 or diastolic blood pressure ≥ 85.

[§] ps<.001 vs. OBP and vs. awake ABP.

^{//} ps<.001 vs. OBP, vs. awake ABP, and vs. HBP.

Table 2

The variance in LVMI explained by age, sex, body mass index, office, awake ambulatory, and home blood pressure levels

Model	R ²	p value
1 - Age, sex, body mass index	.15	<.001
2 - Age, sex, body mass index, office SBP	.19 [*]	<.001
3 - Age, sex, body mass index, awake ambulatory SBP	.24 [†]	<.001
4 - Age, sex, body mass index, home SBP	.27 [†]	<.001
5 - Age, sex, body mass index, office DBP	.19 [†]	<.001
6 - Age, sex, body mass index, awake ambulatory DBP	.20 [†]	<.001
7 - Age, sex, body mass index, home DBP	.21 [†]	<.001
8 - Age, sex, body mass index, office SBP, awake ambulatory SBP	.24 [‡]	<.001
9 - Age, sex, body mass index, office SBP, awake ambulatory SBP, home SBP	.28 [§]	<.001
10 - Age, sex, body mass index, office DBP, awake ambulatory DBP	.20	<.001
11 - Age, sex, body mass index, office DBP, awake ambulatory DBP, home DBP	.23 ^{//}	<.001

DBP=diastolic blood pressure. LVMI=left ventricular mass index. SBP=systolic blood pressure.

* p=.01 vs. Model 1.

[†] p<.01 vs. Model 1.

[‡] p=.001 vs. Model 2.

[§] p=.004 vs. Model 8.

^{//} p=.02 vs. Model 10.

Table 3

Regression analysis for predictors of LVMI

	Unstandardized Coefficient		Standardized Coefficient		t		p value	
	B	Std. Error	Beta		SBP	DBP	SBP	DBP
Age	.24	.36	.10	.09	.19	.28	2.44	3.83
Sex	-6.32	2.74	2.80	2.80	-.17	-.20	-2.31	-2.72
BMI	.35	.49	.28	.28	.09	.13	1.24	1.74
OBP	-.11	.07	.09	.16	-.12	.04	-1.15	.42
Awake ABP	.13	-.11	.17	.25	.10	-.05	.76	-.43
HBP	.45	.58	.16	.24	.38	.31	2.92	2.42
							.004	.02

Predictors with significant p values are in bold type. ABP=ambulatory blood pressure. BMI=body mass index. DBP=diastolic blood pressure. HBP=home blood pressure. LVMI=left ventricular mass index. OBP=office blood pressure. SBP=systolic blood pressure.

Table 4

Associations of LVH with office, ambulatory, and home hypertension.

	Office Hypertension [*]		Awake Ambulatory Hypertension [†]		Home Hypertension [‡]	
	Yes (n=75)	No (n=88)	Yes (n=93)	No (n=70)	Yes (n=69)	No (n=94)
LVH						
Present	6 (8.0%)	3 (3.4%)	6 (6.5%)	3 (4.3%)	7 (10.1%)	2 (2.1%)
Absent	69 (92.0%)	85 (96.6%)	87 (93.5%)	67 (95.7%)	62 (89.9%)	92 (97.9%)
p value [§]	.30		.73		.04	

^{*} Defined by systolic blood pressure ≥ 140 or diastolic blood pressure ≥ 90 .[†] Defined by systolic blood pressure ≥ 135 or diastolic blood pressure ≥ 85 .[‡] Fisher's Exact Test. LVH=left ventricular hypertrophy.