Home Blood Pressure Is as Reliable as Ambulatory Blood Pressure in Predicting Target-Organ Damage in Hypertension


Background: Our objective was to assess the value of home blood pressure (BP) monitoring in comparison to office BP measurements and ambulatory monitoring in predicting hypertension-induced target-organ damage.

Methods: Sixty-eight untreated patients with hypertension with at least two routine prestudy office visits were included (mean age, 48.6 ± 9.1 [SD] years; 50 men). Office BP was measured in two study visits, home BP was measured for 6 workdays, and ambulatory BP was monitored for 24 h. All BP measurements were obtained using validated electronic devices. Target-organ damage was assessed by measuring the echocardiographic left-ventricular mass index (LVMI), urinary albumin excretion rate (AER) in two overnight urine collections, and carotid-femoral pulse-wave velocity (PWV) (Complior device; Colson, Garges-les-Gonesse, Paris, France).

Results: The correlation coefficients of LVMI with office BP were 0.24/0.15 (systolic/diastolic), with home BP 0.35/0.21 (systolic, \(P < .01\)), and with 24-h ambulatory BP 0.23/0.19, awake 0.21/0.16, and asleep 0.28/0.26 (asleep, both \(P < .05\)). The correlation coefficients of AER with office BP were 0.24/0.31 (diastolic, \(P < .05\)), with home BP 0.28/0.26 (both \(P < .05\)), and with 24-h ambulatory BP 0.25/0.24, awake 0.24/0.25 (diastolic, \(P < .05\)), and asleep 0.26/0.18 (systolic, \(P < .05\)). There was a trend for negative correlations between PWV and diastolic BP measurements (not significant). In multiple-regression models assessing independent predictors of each of the three indices of target-organ damage, systolic home BP and age were the only independent predictors of increased LVMI that reached borderline statistical significance.

Conclusions: These data suggest that home BP is as reliable as ambulatory monitoring in predicting hypertension-induced target-organ damage, and is superior to carefully taken office measurements. Am J Hypertens 2007; 20:616–621 © 2007 American Journal of Hypertension, Ltd.

Key Words: Self-measurement, home blood pressure, ambulatory blood pressure, left-ventricular hypertrophy, microalbuminuria, pulse-wave velocity.

Office blood pressure (OBP) measurement is still regarded as the reference method for hypertension diagnosis and follow-up.\(^1,2\) However, because of the white-coat and the masked hypertension phenomena, conventional measurements may be unrepresentative of the true blood pressure in about 30% of subjects attending hypertension clinics.\(^3\) Therefore, out-of-office blood-pressure monitoring, using either ambulatory blood pressure (ABP) or home blood pressure (HBP), is often necessary.\(^3\)

Several studies showed that ABP measurements are more reproducible than OBP measurements\(^1,4\) and are more closely related to target-organ damage\(^5,6\) and a risk of cardiovascular events.\(^3,7\) Therefore, ABP monitoring has found an important application in clinical practice and is regarded as an essential test for the diagnosis of the white-coat and the masked hypertension phenomena, as well as for the assessment of resistant and nocturnal hypertension.\(^3\)

Self-monitoring of blood pressure by patients at home has seen increasing use in clinical practice and is endorsed by hypertension societies worldwide.\(^1–3\) Studies showed that HBP measurements are as reproducible as ABP,\(^4\) and are...
devoid of the white-coat\textsuperscript{8} and masked hypertension effects.\textsuperscript{9} Therefore, HBP monitoring is regarded as an important adjunct to conventional OBP measurements in clinical practice.\textsuperscript{3,10} However, evidence of the value of HBP in predicting target-organ damage\textsuperscript{11–15} and cardiovascular events is still limited.\textsuperscript{16–19}

This prospective study was designed to assess the value of HBP monitoring, in comparison with ABP monitoring and OBP measurements, in predicting target-organ damage in hypertensive patients.

**Subjects and Methods**

**Subjects**

Untreated hypertensive subjects aged 20 to 75 years, with diastolic OBP $>90$ mm Hg on two routine prestudy office visits, were invited to participate. Participants had not received antihypertensive drug treatment for at least 6 months before study entry. Reasons for exclusion were systolic blood pressure $>200$ mm Hg and/or diastolic blood pressure $>120$ mm Hg anytime during the study, evidence of secondary hypertension, diabetes mellitus, congestive heart failure, coronary or valvular heart disease, serum creatinine $>1.5$ mg/dL, overt proteinuria or hematuria, and the use of any drug known to influence blood pressure at least 4 weeks before and during the study. All subjects gave informed consent for study participation.

**Blood-Pressure Measurements**

Blood pressure was measured in the office, at home, and with ambulatory monitoring. Office blood pressure was measured by trained physicians in two study visits 1 to 4 weeks apart, using a validated oscillometric device with memory capacity\textsuperscript{20} (bladder size, $23 \times 12$ cm or $28 \times 14$ cm where appropriate; Omron HEM-705CP; Omron Healthcare GmbH, Hamburg, Germany). Triplicate OBP measurements were performed at each visit after 5 min of sitting rest and with at least 1 min between recordings. Home blood pressure was self-monitored by patients who used the same device and cuff as for OBP measurements (Omron HEM-705CP). Participants were trained in the conditions of HBP measurement and the use of the device and were instructed to perform duplicate morning (7:00–10:00 AM) and evening (6:00–9:00 PM) measurements, after 5 min of sitting rest and with 1 min between readings, for 6 routine working days within 2 weeks. Apart from the device printout, a form was supplied to patients for reporting HBP readings. The ABP monitoring was performed on a routine working day before or after HBP monitoring, depending on the device’s availability and patient preference. Measurements were taken at 20-min intervals for 24 h, using a validated oscillometric device (bladder size, $23 \times 12$ cm or $30 \times 14$ cm where appropriate; SpaceLabs 90207 or 90217; SpaceLabs, Inc., Redmond, WA).\textsuperscript{20} Participants were instructed to follow their usual daily activities, but to hold still with the forearm extended during each ABP reading. The accuracy of the devices used for HBP and ABP monitoring was tested against a mercury column in each participant (Y-connector). Three successive readings were taken to ensure that blood-pressure values did not differ by $>5$ mm Hg from those of the mercury sphygmomanometer. For each individual, all OBP, HBP, and ABP measurements were taken on the same arm (nondominant).

**Assessment of Target-Organ Damage**

Assessment of target-organ damage was performed within 2 weeks after HBP monitoring. An M-mode echocardiogram was performed by a single observer (L.M.) who was not involved in blood-pressure monitoring. Left-ventricular mass was calculated from the average of three cardiac cycles, following American Society of Echocardiography recommendations,\textsuperscript{21,22} and was divided by body-surface area to provide the left-ventricular mass index (LVMI). Left-ventricular hypertrophy was defined as $LVMI \geq 125$ g/m$^2$ for men and $\geq 110$ g/m$^2$ for women. The urinary albumin excretion rate (AER) was assessed by means of two overnight urine collections obtained 1 to 2 weeks apart. Findings were averaged to give a single number per individual. The AER was measured with the immunoturbidimetric method. Microalbuminuria was defined as an AER of 15–150 $\mu$g/min, mormoalbuminuria $<15$ $\mu$g/min, and proteinuria $>150$ $\mu$g/min.\textsuperscript{23} Arterial distensibility was determined by measuring the carotid-femoral pulse-wave velocity (PWV), using a Complior device (Colson) that records pulse waves with two transducers (one positioned at the base of the neck for the common carotid artery and the other over the femoral artery) and that automatically calculates PWV.\textsuperscript{24}

**Analysis**

Subjects with $\leq 6$ OBP measurements, $<12$ valid HBP readings, or HBP readings taken for $<3$ days were excluded from the analysis. Subjects with ABP recordings providing $<14$ successful awake blood-pressure measurements or $<7$ asleep measurements were also excluded. Subjects with an assessment of $<2$ indices of target-organ damage (echocardiography, AER, and PWV) were excluded from the analysis.

All OBP readings of the two study visits and all HBP readings were averaged to give a single systolic and diastolic OBP and HBP value per individual. Data generated by the ABP monitor were batch-imported and organized in a relational database (Microsoft Access 2000, Microsoft Corp., Redmond, WA), using a Visual Basic program. This program, designed by L.G.R. for statistical analysis of ABP-derived data, reads the ASCII text files generated by the ABP monitor and performs multiple data procedures and analyses. Average awake and asleep ABP was calculated using individual patients’ sleeping times.
Table 1. Average office, home, and ambulatory blood pressure (mm Hg, mean ± SD)

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Office</td>
<td>147.7 ± 15.1‡</td>
<td>96.5 ± 9.4‡</td>
</tr>
<tr>
<td>Home</td>
<td>142.2 ± 14.4</td>
<td>92.3 ± 9.0</td>
</tr>
<tr>
<td>Ambulatory</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-h Awake</td>
<td>139.2 ± 13.7*</td>
<td>89.9 ± 9.7†</td>
</tr>
<tr>
<td>Awake</td>
<td>146.2 ± 14.0†</td>
<td>96.0 ± 9.8‡</td>
</tr>
<tr>
<td>Asleep</td>
<td>126.6 ± 14.1‡</td>
<td>79.3 ± 9.8‡</td>
</tr>
</tbody>
</table>

* P < .05 for differences from home blood pressure; † P < .01 for differences from home blood pressure.

The OBP, HBP, and ABP measurements were compared using Student’s paired t-tests with Bonferroni correction for multiple comparisons. Pearson correlations were used for the assessment of associations between methods of blood-pressure measurement and indices of target-organ damage. Multiple-regression models were used to investigate independent predictors of each of the studied indices of target-organ damage (LVMI, AER, and PWV), including age, sex, body mass index, OBP, HBP, and ABP, separately for systolic and diastolic measurements. Statistical analysis was performed using Minitab statistical software (release 13.31; Minitab, Inc., State College, PA). A P value of <0.05 was considered statistically significant.

Results

Seventy subjects were invited to participate in the study. Two subjects were excluded because of low average OBP, and 68 were included in the analysis (50 men, 74%). The mean age was 48.6 ± 9.1 (SD) years, and mean body mass index was 29.0 ± 4.0 (SD) kg/m². The average total cholesterol was 226.8 ± 45.1 (SD) mg/dL (29% had total cholesterol <200 mg/dL, 43% had total cholesterol at 200–250 mg/dL, and 29% had total cholesterol >250 mg/dL). Thirty-two percent of participants were current smokers, 46% were nonsmokers, and 22% were ex-smokers. The 10-year risk of fatal cardiovascular disease according to the SCORE Project for populations at low cardiovascular-disease risk was ≤1% in 54% of participants, 2–4% in 27%, 5–9% in 17%, and >10% in 2%. All participants had complete OBP, HBP, and ABP data. Seven participants had no measurements of AER, and four provided one instead of two urine samples. Two subjects were not assessed using echocardiography, five did not have PWV measurements, and four had unreliable PWV measurements.

Average blood-pressure values obtained by each measurement method, and comparisons among these measurements, are given in Table 1. Strong correlations were found between OBP and HBP (r = 0.79/0.83 for systolic/diastolic), OBP and 24-h ABP (0.76/0.77 for systolic/diastolic), and HBP and 24-h ABP (0.76/0.78 for systolic/diastolic) (P < .001 for all correlations). The mean LVMI was 108.3 ± 11.1 (SD) g/m², the median AER rate was 4.4 μg/min (Q1, 2.4; Q3, 11.9), and the mean PWV was 9.5 ± 2.4 (SD) m/sec. Ten participants (15%) had left-ventricular hypertrophy, and 10 (16%) had microalbuminuria. No significant correlations were found between LVMI, AER, and PWV.

The correlation coefficients of blood-pressure measurement with target-organ damage are presented in Table 2 and Figs. 1 and 2. Systolic HBP and systolic and diastolic asleep ABP were significantly correlated with LVMI. In addition, systolic HBP and asleep ABP, and diastolic HBP and awake ABP and OBP, were significantly correlated with AER. There was a consistent trend toward a negative correlation between PWV and all diastolic blood-pressure measurements (r values from −0.17 to −0.25), but none of these reached statistical significance. The LVMI was correlated with office pulse pressure (r = 0.25, P < .05) and home pulse pressure (r = 0.34, P < .01) but not with 24-h ambulatory pulse pressure (r = 0.12). There were no significant correlations of AER with pulse pressure (r = 0.08, 0.17, and 0.17 for office, home, and 24-h ambulatory pulse pressure, respectively), whereas PWV was positively correlated with office pulse pressure (r = 0.27, 0.16, and 0.17 for office, home, and 24-h ambulatory pulse pressure, respectively; P < .05 only for office pulse pressure).

In multiple-regression models assessing independent predictors of each of the three indices of target-organ damage, the correlation coefficients (r) between office, home, and ambulatory blood pressure and indices of target-organ damage are as follows:

Table 2. Correlation coefficients (r) between office, home, and ambulatory blood pressure and indices of target-organ damage

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>Left-ventricular mass index (g/m²)</th>
<th>Urinary albumin excretion rate (μg/min)</th>
<th>Pulse-wave velocity (m/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Office</td>
<td>0.24/0.15</td>
<td>0.24/0.31*</td>
<td>0.06/−0.17</td>
</tr>
<tr>
<td>Home</td>
<td>0.35†/0.21</td>
<td>0.28*/0.26*</td>
<td>−0.02/−0.21</td>
</tr>
<tr>
<td>Ambulatory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24-h</td>
<td>0.23/0.19</td>
<td>0.25/0.24</td>
<td>−0.06/−0.21</td>
</tr>
<tr>
<td>Awake</td>
<td>0.21/0.16</td>
<td>0.24/0.25*</td>
<td>−0.03/−0.20</td>
</tr>
<tr>
<td>Asleep</td>
<td>0.28*/0.26*</td>
<td>0.26*/0.18</td>
<td>−0.16/−0.25</td>
</tr>
</tbody>
</table>

Values are for systolic/diastolic.
* P < .05; † P < .01.
damage, age and systolic HBP were the only predictors of increased LVMI that reached borderline statistical significance (b coefficients /0.28; SE /0.15; P /0.06 for age; and b /0.33; SE /0.16; and P /0.05 for systolic HBP).

**Discussion**

This study compared HBP, ABP, and OBP measurements in terms of their relationship with target-organ damage in untreated hypertension. The main findings are that (1) HBP measurements appear to be as reliable as ABP monitoring in predicting target-organ damage, and (2) both methods are superior to carefully taken OBP measurements in a research setting.

Considerable effort was placed in this study to improve the reliability of OBP measurements: (1) participants entered the study after at least two prestudy office visits; (2) six OBP readings taken in two study visits were averaged; (3) OBP measurements were taken in a hypertension research setting; and (4) a validated automated oscillometric device was used. This approach prevented observer bias and significantly reduced the white-coat reaction. This is clearly demonstrated by the very small difference between OBP and awake ABP (1.5/0.5 mm Hg for systolic/diastolic, Table 1) and the strong correlation of OBP with HBP and ABP. Therefore, the findings regarding OBP in this study should not be seen as representative of the usual OBP but rather of “research clinic” blood-pressure measurements.

Another characteristic of this study is the relatively low prevalence of target-organ damage and total cardiovascular risk. Left-ventricular hypertrophy was present in 15% of participants, microalbuminuria was present in 16%, and the average PWV appeared to be lower than that previously reported in hypertensive subjects. However, the association between blood pressure–induced changes in target organs and the risk of cardiovascular events is continuous and is not dependent on threshold values. Particularly for AER, values lower than the current accepted threshold were shown to predict cardiovascular events in nondiabetic hypertensive patients, and therefore a lower diagnostic threshold was proposed (eg, >5 μg/min).

The value of ABP monitoring in predicting target-organ damage in hypertension was consistently demonstrated in several trials. A review of 19 trials showed strong correlations between ABP and LVMI, with correlation coefficients ranging from 0.42 to 0.49 for systolic blood pressure and 0.37 to 0.40 for diastolic blood pressure. A more recent review of 21 studies of LVMI clearly showed a stronger relationship with ABP than with OBP. In regard to AER, studies of nondiabetic hypertensive patients also showed a closer relationship with ABP (r = 0.30–0.45) compared with office measurements (r = 0.10–0.30). It should be mentioned, however, that in several studies with LVMI, the methodology for OBP measurements was poorly standardized. It was suggested that, when an adequate number of carefully standardized OBP measurements is obtained, the relationship between LVMI and ABP might not differ from that with OBP. This view is in line with the findings of this study, which showed the association between target-organ damage and carefully taken OBP to be as strong as with awake or 24-h ABP (Table 2, Figs. 1, 2). Nevertheless, the question is whether it is feasible to achieve highly standardized OBP measurements in the usual office setting in general practice.

In contrast to the large amount of data on the relationship of ABP with target-organ damage, the evidence on
HBP is limited. Mule et al compared OBP, HBP, and ABP in regard to their association with target-organ damage in 38 untreated hypertensive patients. However, ABP gave superior correlation coefficients with both LVMI (0.51/0.47) and AER (0.45/0.40), whereas the correlations with office measurements were weaker (0.26/0.27 and 0.25/0.16, respectively).

In the SAMPLE Study, 184 hypertensive patients with left-ventricular hypertrophy were assessed with OBP, HBP, and ABP monitoring and echocardiography at baseline and after 12 months of treatment. In that study, treatment-induced changes in LVMI were best predicted by the treatment-induced change in ABP (r about 0.40, P < .01). Interestingly, despite the fact that only two HBP readings were obtained (single morning and single evening), the average HBP was also predictive of the change in LVMI (r about 0.40, P < .05), whereas neither the usual supine OBP nor random zero device–taken OBP was predictive. Given that, in the present study, HBP (not ABP) proved to be the only blood-pressure measure that was able to predict an increase in LVMI, it might be argued that in the SAMPLE Study, HBP might have been at least as effective as ABP monitoring in predicting the change in LVMI, had more HBP readings been obtained.

A recent study of 225 hypertensive patients in primary care reported significant correlations of systolic HBP and daytime ABP with both LVMI (r = 0.33 and 0.32 for HBP and ABP, respectively) and AER (0.24 and 0.23). In addition, significant negative associations were found between carotid intima–media thickness and both diastolic HBP (r = −0.27) and daytime ABP (r = −0.26). In contrast, OBP failed to achieve significant correlations with any of the target-organ damage indices (r values, 0.10–0.19).

Despite the large sample size, the value of the above-mentioned study is limited, because echocardiography was performed in <20% of the participants, AER measurement in 33%, and carotid ultrasound in <40%, suggesting that different participants had different tests of target-organ damage.

Taken together, the above-mentioned data are in general agreement with our findings regarding ABP as well as HBP measurements. Regarding OBP, the highly standardized methodology for OB measurement in our study was expected to improve the reliability of these measurements in terms of target-organ damage prediction assessed by echocardiography and the measurement of AER. However, arterial stiffness, assessed by measuring PWV, failed to show any significant association with blood-pressure measurements in this study. A previous study, which included both hypertensive and normotensive patients, reported stronger correlations of PWV with HBP (r = 0.46/0.17 for systolic/diastolic) compared with OBP (r = 0.38/0.10). Furthermore, the association between PWV and pulse pressure was stronger for HBP (r = 0.49) compared with office measurements (r = 0.20). In another study in 24 untreated hypertensive patients assessed by brachial-ankle PWV, correlation coefficients with morning HBP were 0.28/0.22 (systolic/diastolic), and with evening HBP, 0.69/−0.01 (systolic/diastolic). That study also included 44 treated hypertensive patients in whom the correlation coefficients with PWV were 0.34/0.15 (systolic/diastolic) with morning HBP, and 0.20/0.11 (systolic/diastolic) with evening HBP. We have no plausible explanation for the lack of association with systolic blood pressures. However, the trend toward a negative association of PWV with diastolic blood pressures (r values from 0.17 to 0.25) is in line with the positive association of PWV with pulse-pressure measurements that also reflect arterial stiffness.

There are several limitations in this study that might have affected the findings. The low values of pulse pressure and PWV, and the low prevalence of left-ventricular hypertrophy and microalbuminuria, suggest that the study population might not be representative of the hypertensive patients currently seen in general practice. The low prevalence of target-organ damage, together with the relatively low statistical power because of the small sample size, might have contributed to the lack of significant correlation between BP values and arterial distensibility, as well as to the nonsignificant trend shown in the multivariate analysis. A larger study including subjects with more severe hypertension of longer duration would have probably provided more certain conclusions. In addition, the different indices of target-organ damage evaluated in this study have not been definitely shown to be really independent surrogate markers of cardiovascular risk. Few data are available regarding the effect of improvements in AER or LVMI on the risk of cardiovascular events, and a drug-induced increase in arterial distensibility was shown to have independent predictive value only in end-stage renal disease patients.

In conclusion, these data suggest that HBP measurements are as reliable as ABP monitoring in predicting hypertension-induced, target-organ damage as assessed by measurement of LVMI and AER, and that both methods are superior to carefully taken OBP measurements. It should be remembered, however, that cross-sectional studies are considered to be of lower relevance compared with longitudinal studies. Some longitudinal studies, conducted in different ethnic populations, showed the ability of HBP measurements to predict cardiovascular events, which is a much stronger end point than the intermediate end points investigated in the present study. Nevertheless, the evidence for the prognostic value of HBP monitoring is still limited, compared with the strong evidence for ABP monitoring.

References


