Ambulatory Blood Pressure Monitoring and Organ Damage

Giuseppe Mancia, Gianfranco Parati

Abstract—Several papers have suggested that 24-hour average blood pressure (BP) is superior to office BP in relation to hypertension target organ damage. This review article will specifically address the evidence provided in this regard by either cross-sectional or longitudinal studies. It will also critically discuss the available data supporting the concept that not only 24-hour average BP values, but also specific BP patterns occurring within the 24 hours may have clinical relevance. This is the case for daytime versus nighttime BP, the day/night BP difference, the morning BP rise, and overall BP variability. (Hypertension. 2000;36:894-900.)

Key Words: ambulatory blood pressure ■ hypertension, white coat ■ blood pressure variability ■ organ damage

Ambulatory blood pressure monitoring (ABPM) has made it clear that 24-hour average blood pressure (BP) values bear a limited correspondence with office BP values taken by a doctor or a nurse. Indeed, in the general population as well as in untreated and in treated hypertensive patients, the correlation coefficients between office systolic BP (SBP) or diastolic BP (DBP) and the corresponding 24-hour average values are rarely >0.50, indicating a wide between-subject discrepancy that can make one of them lower when the other is higher and vice versa.1-4

The question that has arisen from the above findings is obviously which pressure has the greater clinical significance and can thus be taken either as the best predictor of the patient’s risk, before starting antihypertensive treatment, or the best indicator of the patient’s protection induced by treatment. This paper will address this issue on the basis of cross-sectional and longitudinal studies in which ABP has been related to the organ damage accompanying hypertension.

Twenty-four–Hour Average BP and Organ Damage: Cross-Sectional Studies

A large number of studies have investigated whether, on a cross-sectional basis, the organ damage accompanying hypertension is more closely related to 24-hour average than to office BP. The results have almost invariably shown this to be the case regardless of whether the damage is quantified in the heart (left ventricular hypertrophy or dysfunction), in the kidney (proteinuria), in the brain (cerebral lacunae or white matter lesions as identified by nuclear magnetic resonance), in the small and large arteries, or by a comprehensive organ damage score.5-7

The largest database has recently been provided by the European Lacidipine Study on Atherosclerosis (ELSA), which aimed at determining the differential effect of long-term antihypertensive treatment with a calcium antagonist versus a β-blocker on the progression of carotid arteries atherosclerosis (ultrasonography) in more than 2200 hypertensive patients with no marked elevation in serum cholesterol nor diabetes mellitus.8 In the baseline condition, most patients had evidence of an atherosclerotic plaque or of a thickening of the intimal-medial layer of the carotid wall. Either the number of plaques or the size of the thickening were more closely related to 24-hour average SBP and pulse pressure than to the corresponding office values. Indeed, 24-hour average SBP or pulse pressure values were only second to age in their correlation with carotid artery wall status, their importance being also greater than that seen for serum cholesterol and other components of the lipid profile8 (Table 1).

In earlier studies, it was suggested that, among ABP values, daytime ABP, in particular the BP recorded during working hours, is the value displaying the closer correlation to organ damage when compared with office BP values.5 More recent data, however, collected in the frame of the Study on Ambulatory Monitoring of Pressure and Lisinopril Administration (SAMPLE), has provided evidence that the end-organ damage of hypertension is similarly related to daytime, nighttime and 24-hour average ABPs. This supports the concept that BP monitoring periods shorter than 24 hours might be sufficient in providing a clinical evaluation of hypertensive patients superior to that offered by office BP measurements.9

24-Hour Average BP and Organ Damage: Longitudinal Studies

Few longitudinal studies have so far examined the relationship between 24-hour average BP and organ damage.9-12 The

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evidence that has been obtained, however, also points to a greater importance of this pressure vis-à-vis the pressure taken in the doctor’s office, thereby confirming the suggestions made by cross-sectional studies.

Among the most relevant data are, again, those collected by the SAMPLE, which was planned and conducted to determine whether in hypertensive patients with a marked echocardiographic left ventricular hypertrophy, regression of hypertrophy by a 12-month treatment (consisting of an ACE inhibitor plus a diuretic if needed) was more closely related to reduction in 24-hour average than in office BP.9

As shown in Table 2, the 12-month treatment consistently reduced office BP, 24-hour average BP, and left ventricular mass index. The reductions in office and 24-hour BP showed a limited relationship to each other (rSBP/DBP = 0.47/0.40, n=184, P<0.01 for both), while only the latter, but not the former, showed a significant relationship with the degree of the left ventricular hypertrophy regression. Thus, in these patients, an organ damage of prognostic significance such as thickening and/or enlargement of the heart13,14 could be more clearly improved by a BP lowering intervention if 24-hour BP was controlled.

Such a superiority in the prediction not only of end-organ damage but also of clinical events has been more recently emphasized by the results of the SYST-EUR study, the European Study on Isolated Systolic Hypertension in the Elderly. In this study, the incidence of cardiovascular events and the mortality rate were more closely predicted over the follow-up period by ABP values than by office BP measurements.15 This establishes the superiority of ABPM over conventional measurements also on a longitudinal basis. Such an important issue is at present under investigation also in a number of other large controlled trials, such as the ELSA, the PHYLLIS, and the INSIGHT.

### TABLE 1. Correlative Baseline Data: Carotid vs Demographic and Clinical Measurements

<table>
<thead>
<tr>
<th></th>
<th>CBMmax</th>
<th>Mmax</th>
<th>Tmax</th>
<th>Number of Plaques per Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>r</strong></td>
<td><strong>Significance</strong></td>
<td><strong>r</strong></td>
<td><strong>Significance</strong></td>
<td><strong>r</strong></td>
</tr>
<tr>
<td>Age</td>
<td>0.35</td>
<td>P=0.0001</td>
<td>0.37</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>Body mass index</td>
<td>0.03</td>
<td>NS</td>
<td>0.04</td>
<td>NS</td>
</tr>
<tr>
<td>Duration of hypertension</td>
<td>0.09</td>
<td>P=0.0026</td>
<td>0.12</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>Clinic SBP</td>
<td>0.19</td>
<td>P=0.0001</td>
<td>0.20</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>Clinic DBP</td>
<td>-0.03</td>
<td>NS</td>
<td>-0.04</td>
<td>NS</td>
</tr>
<tr>
<td>Clinic pulse pressure</td>
<td>0.21</td>
<td>P=0.0001</td>
<td>0.22</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>Clinic heart rate</td>
<td>-0.05</td>
<td>NS</td>
<td>-0.05</td>
<td>NS</td>
</tr>
<tr>
<td>24-h ambulatory SBP</td>
<td>0.23</td>
<td>P=0.0001</td>
<td>0.24</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>24-h ambulatory DBP</td>
<td>0.03</td>
<td>NS</td>
<td>0.02</td>
<td>NS</td>
</tr>
<tr>
<td>24 h ambulatory pulse pressure</td>
<td>0.32</td>
<td>P=0.0001</td>
<td>0.35</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>24 h ambulatory heart rate</td>
<td>-0.11</td>
<td>P=0.0001</td>
<td>-0.13</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>Total cholesterol concentration</td>
<td>0.10</td>
<td>P=0.0005</td>
<td>0.12</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>HDL cholesterol concentration</td>
<td>-0.08</td>
<td>P=0.0051</td>
<td>-0.11</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>LDL cholesterol concentration</td>
<td>0.11</td>
<td>P=0.0001</td>
<td>0.14</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>LDL:HDL cholesterol ratio</td>
<td>0.13</td>
<td>P=0.0001</td>
<td>0.17</td>
<td>P=0.0001</td>
</tr>
<tr>
<td>Triglyceride concentration</td>
<td>0.08</td>
<td>P=0.0038</td>
<td>0.10</td>
<td>P=0.0004</td>
</tr>
<tr>
<td>Glucose concentration</td>
<td>-0.02</td>
<td>NS</td>
<td>-0.00</td>
<td>NS</td>
</tr>
<tr>
<td>Creatinine concentration</td>
<td>0.05</td>
<td>NS</td>
<td>0.08</td>
<td>P=0.0038</td>
</tr>
</tbody>
</table>

Spearman correlation coefficients (r) and P values. Calculations are based on data from 1173 patients for whom complete sets of data were available. CBMmax indicates mean of the maximum of IMT of the four far walls of the carotid bifurcations and distal common carotid arteries; Mmax, changes in the mean thickness of 12 different sites (right and left near and far walls, distal common, bifurcation and proximal internal carotid); and Tmax, overall mean maximum IMT. (Reprinted with permission from Reference 8).

### TABLE 2. Office and 24-Hour BP Values in Patients of the SAMPLE and Their Correlation With LVMI

<table>
<thead>
<tr>
<th></th>
<th>Baseline, n=206</th>
<th>Δ With treatment (12 m), n=184</th>
<th>Δ With treatment (12 m), n=184</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Office SBP/DBP, mm Hg</strong></td>
<td>165±15/105±5</td>
<td>-25±17/–18±7</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>24-h SBP/DBP, mm Hg</td>
<td>149±16/95±11</td>
<td>-18±17/–12±11</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>LVMI, g/m²</td>
<td>158±32</td>
<td>-26±22</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>ΔLVMI vs ΔOffice SBP/DBP, r</td>
<td>0.11/0.11</td>
<td>NS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ΔLVMI vs Δ24-h SBP/DBP, r</td>
<td>0.42/0.38</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data obtained at baseline and their changes after 1-year antihypertensive treatment are shown.
is brought about by sleep and then increases steeply when in the morning the subject awakes and resumes his/her daily activities. This increase occurs together with a peak incidence of myocardial infarction, sudden death, and stroke in the morning hours, which is why an enhanced morning incidence of cardiovascular morbid and fatal events.

Finally, the morning BP rise appears to be so closely linked with the transition from sleep to wakefulness and the resumption of physical activity that its alteration by drug treatment might be obtained at the price of an impairment of autonomic cardiovascular control that could endanger daytime BP homeostasis. The situation may be different, however, when the magnitude of the morning BP rise is artificially affected by an unbalanced 24-hour BP reduction by treatment. This may occur when short-lasting antihypertensive drugs are administered once a day (usually in the morning) to lower BP in hypertensive patients. In such a case, the early hours of the next morning may be characterized by a steeper BP rise, the physiological changes occurring at waking time being combined with the BP escape from the effects of treatment, a condition that might indeed contribute to a higher risk of cardiovascular events.

Thus, the main goal of treatment should probably be not to reduce the slope of the morning BP rise (which is also hard to define unless the precise time of awakening is identified by electroencephalographic and electromyographic recordings), but rather to homogeneously lower the whole 24-hour BP profile, without inducing major differences between the reduction of day and night values, and thus also without any adverse interference with the physiological morning BP rise.

Nighttime BP

A population survey has shown nocturnal fall in BP to be, on average, similar in subjects between 25 and 74 years of age, although a meta-analysis of a large number of smaller studies suggests that in individuals more than 75 years of age an attenuation may occur. The nocturnal fall in BP has also been found to be on average preserved in hypertension, unless in a few cases of secondary hypertension. The nocturnal fall in BP, however, can vary widely among individuals, which has led hypertensive subjects to be classified into 2 categories, ie, those whose nighttime average BP falls more than 10% (known as “nondippers”), organ damage is much greater than in those in whom it falls less than 10% (known as “dippers”), and that this is the case also for the organ damage progression and the incidence of cardiovascular disease.

Other studies, however, have not found these differences to be so pronounced and clear. Furthermore, evidence has been
obtained that in a given population the magnitude of the nocturnal BP fall is normally distributed (Figure 2), which makes the 10% threshold dividing dippers and nondippers arbitrary.1,27

Finally, in the SAMPLE (1) repetition of ABPM in absence of treatment, or during a treatment regimen that had achieved a stable antihypertensive effect, was accompanied by a 40% change in the dipping or nondipping status (Figure 3),27 indicating that this classification is not reproducible and (2) the dipping or nondipping status did not lead to a difference in the size of the left ventricular hypertrophy regression over the 12-month treatment, which only depended on the average 24-hour, daytime, or nighttime BP control that treatment could achieve (Figure 4).9,27 This clearly calls for more stringent criteria to investigate the importance of the day/night BP difference in the genesis of the organ damage accompanying hypertension. These criteria should account for the fact that, within individuals, this phenomenon may vary as a function of the physiological variations in sleep pattern and depth between different days. This was suggested by the findings, in the SAMPLE, of a limited, although statistically significant, relationship between the nighttime BP fall on 2 different occasions (n = 180, correlation of the differences between daytime and nighttime SBP/DBP values at 3 versus 12 months, r = 0.26/0.27 respectively, P < 0.01). This tendency for a higher frequency of reproducible dippers on the basis of DBP than on SBP (Figure 3) would need to be confirmed by additional studies and might depend on the known larger variability of SBP than DBP values, which might make also the day/night changes in SBP less reproducible than DBP changes.

It should also be considered that in the hypertensive population, daytime and nighttime BP values show a close relationship between each other, as do the daytime and nighttime BP changes induced by treatment (Figure 4).2,9 In other words, it should be considered that daytime and nighttime BP values are not truly independent but that they are interrelated variables, which should in most instances make their correlation with organ damage similar. This was indeed the case in the SAMPLE, in which their close relationship before and during treatment was accompanied by a similar correlation with left ventricular mass index (at echocardiography, LVM) and either daytime values, nighttime values, or the values of the day/night difference in pressure observed at entry (left) or their changes induced by a 1-year treatment (T) (right). Data are separately shown for SBP and DBP. Vertical lines on top of bars refer to 95% confidence intervals. Asterisks refer to statistical significance of correlation coefficients (Reprinted with permission from Reference 9).

Figure 3. Histograms refer to the percentage of patients in the SAMPLE in whom ambulatory blood pressure monitoring was repeated twice and who were classified in both instances as dippers (D-D) or as nondippers (ND-ND) or who shifted from being dippers to nondippers (D-ND) or from being nondippers to dippers (ND-D). Top panels refer to differences between daytime and nighttime values observed in 170 hypertensive patients in whom duplicate ambulatory blood pressure recordings were obtained at entry and under the final placebo wash-out period. Bottom panels refer to differences between daytime and nighttime values observed in 180 hypertensive patients in whom duplicate ambulatory blood pressure recordings were obtained after 3 and 12 months of active treatment, respectively. Left panels refer to SBP day/night differences. Right panels refer to DBP day/night differences (Reprinted with permission from Reference 27).

Figure 4. Top panels refer to correlations between day and night BP values recorded at entry while middle panels refer to the corresponding correlations between changes in daytime and nighttime BP values induced by a 1-year antihypertensive treatment. Data were obtained in 206 and 184 patients, respectively, included in the SAMPLE. Bars in the bottom panels illustrate the correlation coefficients between left ventricular mass index (at echocardiography, LVM) and either daytime values, nighttime values, or the values of the day/night difference in pressure observed at entry (left) or their changes induced by a 1-year treatment (T) (right). Data are separately shown for SBP and DBP. Vertical lines on top of bars refer to 95% confidence intervals. Asterisks refer to statistical significance of correlation coefficients (Reprinted with permission from Reference 9).
damage to autonomic cardiovascular regulation (diabetes) and/or because the extent of organ damage (severe hypertension, secondary hypertension, etc.) impairs the vessel ability to dilate and thus the vascular resistance to lower. Under these circumstances (which describe a truly non-dipping status), nighttime BP may contribute to an important and independent degree to the overall BP load on the cardiovascular system, as recently shown by the SYST-Eur study.15 Second, it has also been suggested that in a hypertensive subgroup, nighttime BP fall may be so pronounced as to make these patients (termed "extreme dippers") at risk of vital organ underperfusion.28 The reproducibility and clinical significance of this phenomenon, as well as its modifications by treatment, still need to be adequately investigated, however.

**BP Variability**

Twenty-four–hour BP varies not only because of a reduction during night sleep but also because of sudden, fast, and short-lasting changes that may occur both during the day and, to a lesser extent, during the night. As shown in Figure 1, when quantified as the standard deviations of the BP values recorded intra-arterially over the 48 half hours of a 24-hour monitoring period, this short-term BP variability increases when BP increases, which can also be seen when normotensive, mild, moderate, and severe hypertensive subjects are compared.16,17

It has repeatedly been shown that this phenomenon may have clinical relevance because hypertensive patients with similar 24-hour mean BP values have a greater comprehensive score for organ damage when their BP variability is greater.5,6,16,18-21,29-30 Furthermore, in patients with a greater BP variability, overall organ damage and left ventricular mass index increase more at follow-up than in those hypertensive patients in whom for the same 24-hour BP mean values, BP variability is less.31 Finally, carotid artery atherosclerosis has been found to independently correlate with SBP or pulse pressure variability in the hypertensive patients of the ELSA study (Mancia G, Parati G, Hennig M, Flatau B, Omboni S, Bond G, Zanchetti A, unpublished data, 2000), its relative importance closely following in the multivariate analysis the one attributable to 24-hour average BP values.

Whether it is the increased variability that increases organ damage or vice versa remains to be established to properly understand whether and to what extent this phenomenon can be regarded as a marker of rather than a factor leading to cardiovascular disease. A few studies, however, have shown an experimentally induced increase in BP variability, with no increase in average BP values, to be followed by cardiovascular damage.32 The hypothesis can thus be made that the organ damage accompanying hypertension is in part due to the extent of the BP variations. Thus not only average BP values but also upward and downward BP excursions around them should be reduced by treatment. This has so far been explored only to a limited degree because of the need to use intra-arterial ABPM to precisely quantify BP variability on a beat-to-beat basis. The need of continuous ABPM to properly quantify BP variability16 might explain also why other studies, in which only discontinuous ABPM techniques were available, have provided us with conflicting evidence on the occurrence of a significant relation between BP variability and end-organ damage after accounting for the prognostic value of 24-hour average BP values.33 Thus, whether BP variability does indeed represent an additional independent factor contributing to cardiovascular risk in hypertension needs to be confirmed by larger prospective studies in which BP variability might be properly assessed. Techniques allowing beat-to-beat BP to be monitored noninvasively in ambulatory patients34 hold promise for these studies to be more easily performed in the future.

**ABP Measurements, Organ Damage, and Antihypertensive Treatment**

ABPM is particularly useful in measuring the efficacy of antihypertensive therapy over the dosing interval, and indeed all newly marketed agents need to be evaluated by means of 24-hour ABP recordings in pivotal clinical trials. The major reasons are that, as mentioned above, office BP does not accurately reflect pretreatment and on-treatment ABP values, which, as shown above, are likely to have a superior clinical importance.2 Four other important reasons, supporting the use of ABPM in the assessment of the efficacy of antihypertensive drugs, are the following. First, ABP is not significantly modified by the white coat effect,35,36 which means that recruitment on the basis of this approach more adequately selects truly hypertensive individuals and allows the actual BP lowering effect of a given treatment to be more specifically assessed; Second, over weeks or months, ABP values are not substantially altered by placebo,37 which means that the placebo arm of the study can be avoided, eliminating a major ethical problem and reducing the number of patients to be studied; Third, 24-hour average BP values are much more reproducible (≈3 times) than office values,38 which means that small BP differences in 24-hour average values between different treatments can more easily achieve statistical significance even when the study size is limited.39 Fourth, with the use of 24-hour ABPM it is possible to determine whether a once-a-day drug does lower BP throughout the 24 hours in a homogeneous fashion, ie, without an excessive BP fall early after drug assumption and without a vanishing of the hypotensive effect later. The need of a precise quantification of this phenomenon has led to the definition of mathematical indices aimed at providing a comprehensive assessment of the homogeneity of the BP reduction induced by a given antihypertensive treatment, such as the trough-to-peak ratio and the smoothness index.40-43 Indeed, a nonhomogeneous distribution of the antihypertensive effect of a given treatment over 24 hours might endanger cardiac and cerebral perfusion and may adversely affect target organs by increasing the magnitude of BP variations, as recently documented by the inverse relationship that was found between a precise measure of the homogeneity of the antihypertensive effect such as the smoothness index and the on-treatment BP variability as quantified by 24-hour standard deviation.31

**Isolated Clinic Hypertension**

In some hypertensive individuals, the elevation of clinic BP is not accompanied by a similar BP elevation outside the clinical environment, ie, at home or over the 24 hours. This is believed to reflect an excessive emotional response to BP
measurements by a doctor or a nurse, which has led this condition to be termed “white-coat hypertension.” Data on whether white-coat hypertension is clinically relevant however are even more controversial than those on the day/night BP difference. First, the prevalence of white-coat hypertension in the population is not yet precisely established, although it is now clear that several earlier studies have probably overemphasized the frequency of this phenomenon because of their failure to take into account that the cut-off value dividing ABP normality, and abnormality is much lower than the corresponding clinic value, ie, much lower than 140/90 mm Hg. Second, although some studies have reported white-coat hypertensives as characterized by organ damage and/or cardiovascular risk factors, in other studies no organ damage has been found. Furthermore, no excessive cardiovascular morbidity events have been observed in white-coat hypertensive patients who were followed over time in studies that, however, lacked statistical power to prove the true innocence of this phenomenon. Third, there is reason to believe that the difference between clinic BP and ABP may depend on several factors other than the pressure response to ABP measurements in the clinical environment and that thus the term white-coat hypertension to identify a positive difference between clinic BP and average day/time ABP can be a misnomer. This is because the clinic BP-ABP difference (1) is not accompanied by a similar difference in heart rate as it should be if the greater clinic BP values were due to an emotional stimulus, (2) is greater in aged subjects in whom, however, the hemodynamic response to emotions is not greater, and (3) is negatively related to ABP (and thus to its modulation in daily life) and unrelated to the true BP response to the doctor, as measured in patients undergoing a medical visit while under continuous noninvasive BPM.

The controversy whether isolated clinic hypertension (the term that should be used instead of white-coat hypertension to indicate persistently elevated BP values in a clinic environment and persistently normal BP values at other times) represent an innocent or risky phenomenon was addressed also in a few recent studies of ours. We have observed that in hypertensive subjects with left ventricular hypertrophy, the difference between clinic BP and ABP is variably attenuated by long-term treatment but that this attenuation does not play any role in the regression of left ventricular hypertrophy, which depends exclusively on the treatment-induced reduction in ABP. We have also recently seen, however, that in the less severe hypertensive patients of the ELSA Study, for any given level of ABP, carotid artery wall abnormalities (thickening and plaques) displayed a tendency to be greater when the clinic-ABP difference was greater (Mancia G, Parati G, Hennig M, Flautau B, Omboni S, Bond G, Zanchetti A, unpublished observations, 2000). It is thus possible that when hypertension is in a more advanced stage, organ damage progression or regression depends on 24-hour BP values, whereas initially the clinic/daytime BP difference also plays a role, possibly because it reflects a BP tendency to vary more markedly in response to inner and outer influences. This suggests that the decision not to treat this condition should be taken with caution, and that, whenever it is taken, a close follow-up of the patient should be implemented.

### Conclusion: ABPM in the Clinical Practice

ABPM is thus not only critical in the assessment of the efficacy of antihypertensive drugs in clinical trials, but it is also applicable in clinical practice. Given some technical problems not yet fully solved (such as the limited accuracy of individual BP readings) and given the possible social impact of the nonnegligible cost of ABP recordings, this approach should not be adopted routinely in all hypertensive patients. It should rather be used in selected situations, as listed by the JNC VI and the 1999 WHO/ISH guidelines (Table 3). For example, the use of ABPM is helpful when the diagnosis of hypertension is uncertain, ie, when a persistently elevated office BP is accompanied by no organ damage at all and probably also by normal home values. It is also helpful in the evaluation of patients who do not respond to treatment despite the use of combined treatment or in those who exhibit drug treatment symptoms (eg, dizziness, syncope, etc) that may suggest the occurrence of hypotensive episodes. Both JNC VI and WHO/ISH guidelines agree, however, on the fact that this approach should not be used routinely because of its cost and uncertainty as to the prognostic value and the need of treatment of some condition it may shed light on, ie, isolated office or white-coat hypertension. Given the present lack of more consistent evidence, parameters such as the day/night BP difference and BP variability should probably still be regarded as research issues, although they might be considered in specific conditions.

Physicians should thus be left free to decide when ABPM should be performed, keeping in mind that they may increase the accuracy and the clinical significance of their office readings by increasing the number of clinical visits and by obtaining at each visit multiple BP measurements or by teaching their patients to repeatedly perform self BP measurements at home.

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