Hypertension Staging Through Ambulatory Blood Pressure Monitoring

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This issue of Hypertension includes an article by Bur et al1 that focuses on the comparison between clinic and ambulatory blood pressure (ABP) values in patients with moderate to severe hypertension. The primary goal of the Bur study was to obtain a classification of hypertensive patients, based on the ABP values corresponding to the clinic blood pressure (BP) values that have been used to stage hypertension by the World Health Organization–International Society of Hypertension (WHO-ISH) and the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC VI) guidelines.2,3 An additional goal was to evaluate whether this ABP-based classification has prognostic value, as shown for the prognostic value of the clinic BP staging. Both clinic BP and ABP were measured in 736 hypertensive patients (557 of whom were under treatment) at the time of their first admission to the local Hypertension Unit. All patients then entered a follow-up period with an average duration of 52 months (range, 6 to 96 months), during which only clinic BP was obtained. During the observation time, 82 patients had nonfatal cardiovascular events and 9 patients died of cardiovascular causes.

The article adds interesting information to the existing database on the clinical value of ABP. In particular, it contributes to the available knowledge on the prognostic importance of ABP as well as on its relation to clinic BP in the context of treating patients in a hypertension center.4

Stratifying patients into different risk categories on the basis of ABP values requires studies that (1) establish in populations or in large groups of hypertensive patients the relation of cardiovascular morbidity and mortality with the different 24-hour ABP values selected5,6 and (2) evaluate how prognosis of patients is modified when ABP is reduced by treatment, leading to a change in the ABP-based staging. This information is only partly available, however, because the association between the incidence of cardiovascular disease and ABP has been examined in only a few studies of suitable size.7 Furthermore, the few intervention studies addressing the prognostic value of treatment-induced changes in ABP have been undermined by problems such as a low number of BP measurements during ABP monitoring, use of surrogate end points rather than morbid or fatal events, uncontrolled experimental designs, small numbers of patients (and thus insufficient statistical power to back the study conclusions), and lack of ABP measurements during antihypertensive treatment.6

The available evidence clearly indicates that the upper limits of normal 24-hour average ABP are markedly <140/90 mm Hg. However, because of these problems, it does not provide any classification of hypertensive patients on the basis of ABP levels that might parallel the staging of hypertension, based on clinic BP, proposed by WHO-ISH and JNC VI guidelines.2,3

Bur et al1 have made an attempt to cope with at least some of the above problems. The results of their study confirm previous findings that the relation between clinic BP and ABP, although statistically significant, is by no means close. They also confirm the observations made in previous studies that ABP is significantly lower that clinic BP, especially in moderate and severe hypertension,4–7 because the discrepancy between clinic BP and ABP increases with increasing clinic BP values. Based on these findings, and consistent with previously published recommendations,2–7 the authors correctly emphasize that the classification of hypertension by WHO-ISH or JNC VI clinic BP criteria should not be directly transferred to ABP data. Finally, and most importantly, the results of Bur et al confirm and extend previous observations on the prognostic value of ABP by showing that staging hypertension by ABP values is indeed related to prognosis of hypertensive patients.

It should be acknowledged that it is difficult to carry out controlled studies on the prognostic value of different ABP levels. This is because, as mentioned above, the study size needs to be large and the observation period to be prolonged in order to obtain a sufficiently high number of events that permit conclusions with high statistical power. In particular, if the purpose is not just to show the prognostic value of ABP (a rather likely finding) but whether ABP is prognostically superior to or adds to the prognostic value of clinic BP, the study size and duration need to be substantially increased. Finally, to assess whether cardiovascular protection depends more on treatment-induced reduction in ABP than in clinic BP (or whether knowledge of ABP reduction by treatment adds to the estimate of protection based on clinic BP reduction), additional requirements need to be fulfilled. These include (1) frequent collection of ABP data during treatment, (2) need to avoid excessive treatment nonhomogeneity, and

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(3) physicians’ different attitudes and biases on optimal ABP levels to be achieved.

This has not been done in the study by Bur et al. First, the patients included during the 52-month follow-up period had a limited number of cardiovascular events. This problem was made more serious by the fact that the attempt to stage patients on the basis of ABP values required their subdivision into subgroups, which showed only few events and often trivial event differences. Second, ABP was obtained (as it was the case for previous studies) only at the time of the initial evaluation, which was followed in the next 52 months by a modification of antihypertensive treatment, because clinic BP showed on average a substantial reduction. This does not detract from the authors’ conclusion that the ABP values originally obtained at the initial evaluation had a prognostic significance. It does not allow, however, clarification of whether determination of this prognostic significance was modified by the new values subsequently obtained after treatment changes, which is an important issue, given the evidence on the prevailing prognostic importance of BP values achieved by treatment. This is especially important because selection of the treatments was unrestricted and the patients were given different drugs or drug combinations, which may have been responsible for differences in cardiovascular protection beyond those accounted for by BP reductions. Third, identification in the study by Bur et al of normal ABP values based on the regression between clinic BP and the corresponding clinic BP-ABP difference is open to criticism. This is not because of the use of their specific statistical approach, which in previous population studies has led to valuable data. It is because the present study involved mainly patients under antihypertensive treatment (75.7%), in whom the differences between clinic BP and ABP may have reflected a differential effect of antihypertensive drugs on the two pressures. This possible explanation is supported by the fact that different drugs have different trough-to-peak ratios and by the evidence that usually the effects of antihypertensive treatment are more pronounced on clinic BP than on ABP.

However, the authors should be given credit for their finding that in the hypertensive patients in whom clinic BP was in the lowest WHO/ISH or JNC VI stage, 24-hour ABP was similar or only slightly above the normal values calculated for population studies. This means that in this range, we can expect a certain degree of correspondence between these two different BP estimates. This is an important observation, although it is valid only for average group data and not for individual patients.

Two final comments deserve to be made. First, evidence is available that the prognostic power of ABP values is a function of their reproducibility, which means that averaging data from two ABP recordings may improve it. This methodological issue has not been addressed in the present study, in which initial clinic BP was measured in two different occasions, whereas ABP monitoring was obtained only once. Second, it is not clear in the study by Bur et al whether ABP staging carries an additional prognostic contribution over and above that classically provided by clinic BP. As mentioned above, this is of critical importance because the dilemma we face is not to abandon clinic BP in favor of ABP but to determine whether information on ABP refines the risk assessment made possible by clinic BP to an extent that justifies the additional cost of the ABP monitoring procedure. This could have been addressed by Bur et al, however, by comparing the Kaplan-Meier curves on cardiovascular events based on clinic BP and on ABP or, alternatively, by assessing the prognostic value of ABP after adjusting for the information provided by clinic BP.

Despite these problems, the effort made by Bur et al to provide us with indications on how to classify the severity of hypertension based on ABP data does represent an important step toward the use of ABPM in the prognostic stratification of hypertensive patients, although the data obtained in this study are not robust enough yet to recommend changes in current treatment strategies.

Additional studies are still needed to this aim, including longitudinal investigations on the predictive role of ABP values obtained during a follow-up period in a large sample of patients and intervention studies that might provide evidence on a reduction in cardiovascular risk after treatment-induced reductions in ABP levels. This could be achieved by including serial ABP recordings in clinical trials, either in large longitudinal population risk factor studies, and in large mortality trials addressing the benefits of antihypertensive therapy.

Until the data are available, use of ABP measurements in the routine treatment of hypertensive patients should still be recommended only in selected cases, as a complement to home blood pressure measurements, and to repeatedly and carefully obtained clinic BP readings.

References


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