Ambulatory Blood Pressure Measurement: A Trove of Hidden Gems?

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Editorial Commentary

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One of the consequences of hypertension is increasing stiffness of the arteries, which leads to cardiovascular events and increased mortality.[1] Clearly this process is a continuous one and, contrary to acceptance that such change is inevitable, it is not now unreasonable to assume that if stiffening in the arterial system could be detected at an early rather than at a late stage, therapeutic interventions might be initiated to delay or even prevent its occurrence. However, measuring arterial stiffness requires special equipment and trained staff, facilities that are not generally available and that are, moreover, costly. It has been proposed recently that a measure of arterial stiffness could be obtained from the routine use of ambulatory blood pressure measurement (ABPM) by using the dynamic relationship between diastolic and systolic blood pressure over 24 hours, calculated as 1 minus the regression slope of diastolic on systolic blood pressure. The rationale underlying the ambulatory arterial stiffness index (AASI) is that average distending pressure varies during the day and that the relation between diastolic and systolic blood pressure, with this changing distending pressure, depends largely on the structural and functional characteristics of the large arteries.

In the Dublin Outcome Study, AASI predicted cardiovascular mortality in a large cohort of hypertensive individuals. Moreover, this prediction withstood additional adjustment for other risk factors, including pulse pressure. Interestingly, AASI was a stronger predictor of fatal stroke than pulse pressure in patients with ambulatory normotension suggesting that AASI may provide an early indication of arterial stiffness before sustained hypertension develops.[2] The demonstration that AASI closely correlated with aortic pulse wave velocity and that it correlated more closely with central and peripheral augmentation index than with 24-hour pulse pressure even after adjustment for major determinants of arterial stiffness and wave reflections gave support for the claim that AASI was indeed a "novel measure of arterial stiffness."[3]

The new index had been criticized, however, on the basis of not being a true measure of arterial stiffness but rather a "surrogate of a surrogate end-point"[4] and on the grounds that it is not clear whether AASI really measures large artery stiffness or if it is largely influenced by peripheral resistance.[5] The fact that AASI was a better predictor of fatal strokes, whereas 24-hour pulse pressure better predicted cardiac events in the Dublin Outcome Study, supports the hypothesis that AASI may depend on the mechanical properties of small arteries.[5]

In this issue of *Hypertension*, Leoncini et al[6] show that in untreated Italian patients with primary hypertension in whom the prevalence of microalbuminuria, left ventricular hypertrophy, and carotid abnormalities was 12%, 38%, and 19%, respectively, AASI was positively related to age, triglycerides, office and 24-hour systolic blood pressure, 24-hour pulse pressure, urinary albumin excretion, and carotid intima–media thickness. Even after adjusting for confounding factors, patients with target organ involvement had a higher AASI than those who did not, and each standard deviation increase in AASI doubled the risk of having subclinical organ damage. Moreover, the association between ambulatory stiffness and organ damage seems to be graded and linear: the higher the AASI, the greater the severity of organ involvement. The relevance of these findings to clinical practice is that the increasingly used technique of ABPM may provide us with a measure (whether it is surrogate or otherwise is irrelevant) that will herald the onset of arterial stiffness at an early stage in the atherosclerotic process even in subjects with ambulatory normotension.

This thought begs another. Traditionally, the information sought from the technique of ABPM in clinical practice has been confined to providing the mean of the 24-hour, daytime, and nighttime blood pressures,[7] with the necessity for recording nocturnal blood pressure often being questioned. Could it be that we have been blinded to the hidden gems that may be found in the relatively simple and inexpensive investigation of ABPM?

First, let us consider the valuable information yielded from ABPM by simply analyzing data within the windows of the 24-hour profile. Starting with the "white coat window," the first hour of ABPM recording during which the white coat effect of the medical environment may be carried over to the ABPM profile, we find evidence that this window can provide a more accurate diagnosis of white coat hypertension than other measures of blood pressure, as well as providing a means of stratifying risk in patients with white coat hypertension.[8]

Next there is the nocturnal window in which much happens to the cardiovascular system, especially in relation to blood pressure. The patterns of nocturnal blood pressure (nocturnal hypertension, nocturnal hypotension, dipping and nondipping, reverse dipping, and autonomic failure) have been largely ignored in clinical practice. Many studies evaluating morbidity and dipping status have supported the concept that a diminished nocturnal blood pressure fall is associated with

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a worse prognosis.[7] The results of the Dublin Outcome Study now confirm studies in other populations showing that nighttime blood pressure predicts outcome more accurately than any other measure of blood pressure: for each 10-mm Hg rise in mean nighttime systolic blood pressure, the mortality risk increased by 21%.[9] Further confirmation of the importance of nocturnal hypertension comes from a recent study showing that a nondipping pattern and increased nighttime diastolic blood pressure predicted the occurrence of congestive heart failure independently of antihypertensive treatment and established risk factors for cardiac failure. Furthermore, a nondipping pattern increased the risk of cardiac failure even after adjusting for office blood pressure measurement, thereby showing that ABPM once again conveys important information that cannot be obtained with conventional measurement.[10]

The preawakening window of the 24-hour profile, perhaps more correctly denoted as the "matinal" window, which has been dubbed "the blind spot" in current clinical practice,[11] has been implicated time and again as the period of the 24-hour profile when most cardiovascular events occur. Indeed, quite apart from the predictive importance of nocturnal and matinal hypertension and the nondipping nocturnal pattern, one must wonder at the lack of pharmacological interest in targeting this period for therapeutic intervention rather than persisting in the use of conventional blood pressure as the measure on which the efficacy of antihypertensive medication is judged. As has been suggested, a prospective randomized clinical trial to show whether treatment based on nighttime pressure will improve outcome is overdue.[9]

Finally, there is a wealth of statistical analysis to be obtained from ABPM not only in identifying different forms and patterns of hypertension,[7] but also in applying statistical analysis, such as measures of cardiovascular load and variability, not only to the circadian blood pressure but also to heart rate[12]; all of which surely must make it timely to reappraise the value of the technique not only in clinical practice but also in clinical research.

Disclosures

None.

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