O
ne 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 pres-
sure 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."

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"3 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.4
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 al6 show that in
untreated Italian patients with primary hypertension in whom
the prevalence of microalbuminuria, left ventricular hyper-
trophy, 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 pres-
sure, urinary albumin excretion, and carotid intima-media thick-
ness. 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 associ-
ation 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 record-
ing 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 pro-
vide 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.\textsuperscript{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\%.\textsuperscript{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.\textsuperscript{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,\textsuperscript{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.\textsuperscript{5}

Finally, there is a wealth of statistical analysis to be obtained from ABPM not only in identifying different forms and patterns of hypertension,\textsuperscript{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\textsuperscript{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.

**References**

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Ambulatory Blood Pressure Measurement: A Trove of Hidden Gems?
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