Rev Port Cardiol. 2018;37(4):329–331

Revista Portuguesa de
Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

EDITORIAL COMMENT

The time has come to change ambulatory blood pressure monitoring from 24-hour to 48-hour for the diagnosis of hypertension and cardiovascular risk assessment☆

Chegou a altura de abandonar a MAPA de 24 horas e adotar a de 48 horas para o diagnóstico de hipertensão e correta avaliação do risco cardiovascular

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Available online 1 May 2018

Despite circadian variations in blood pressure (BP) and the changeability of measured BP values, the diagnosis of normotension or hypertension continues to be based on static measurements obtained at one specific moment, as if this gave a true measure of the individual's BP status.

While these measurements are valid in large-scale population studies, they have little prognostic value in a given individual due to BP variation over the 24-hour period[1] and to the white-coat effect, both of which can lead to misdiagnosis and incorrect treatment, or no treatment at all. When assessed by ambulatory blood pressure monitoring (ABPM), one-third of individuals with suspected hypertension based on office assessment have normal ambulatory BP; in other words, they have white-coat hypertension. Furthermore, one in four patients with hypertension that is apparently resistant to treatment have controlled BP on ABPM, i.e., pseudoresistance.[2] It is therefore no wonder that the 2011 recommendations of the UK National Institute for Health and Clinical Excellence on the management of hypertension[3] propose using ABPM to confirm the diagnosis of hypertension when office BP is ≥140/90 mmHg. Nor is it surprising that 24-hour ABPM is considered the method of choice to quantify BP and to provide a more accurate diagnosis of hypertension and assessment of cardiovascular risk.[4]

However, nothing is perfect, and 24-hour ABPM also has its limitations, particularly the inconvenience of measurements being taken every 20 or 30 minutes and the low reproducibility of dipping patterns.

The reproducibility of these patterns is of considerable importance. Although the limitations of ABPM do not call into question their prognostic value[5] or the results of reference trials,[6,7] they do mean that 24-hour ABPM is less useful for calculating risk in individual patients. By contrast, nocturnal BP, which is most closely related to the risk of cardiovascular and renal events, is highly reproducible on 24-hour ABPM and thus remains the gold standard of ambulatory BP for determining cardiovascular risk in hypertensive patients.[8]

The above issues are relevant to the study by Araújo et al. published in this issue of the *Journal*,[9] which aims to analyze ABPM circadian patterns and their determinants in a population of normotensive and hypertensive patients.

☆ Please cite this article as: Machado AP. Chegou a altura de abandonar a MAPA de 24 horas e adotar a de 48 horas para o diagnóstico de hipertensão e correta avaliação do risco cardiovascular. Rev Port Cardiol. 2018;37:329–331.

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Unfortunately, 24-hour ABPM is not a suitable method for establishing circadian BP profiles, due to their low reproducibility in 24-hour recordings, and therefore does not serve the authors' purpose. The limited reproducibility of circadian patterns on 24-hour ABPM has been recognized for more than two decades,[10,11] and some authors have suggested that correct classification of dippers and non-dippers can be achieved through repeated ABPM recordings. However, the low reproducibility of circadian BP patterns on repeated 24-hour ABPM in the same individual[11] means that this is unlikely to succeed, even though the reproducibility of 24-hour ABPM is still superior to that of office BP measurements.

How can the low reproducibility of circadian patterns on 24-hour ABPM be explained? When BP is monitored on two consecutive days, it is found to be significantly higher on the first day than on the second. This pressor effect of ABPM is seen in the first four hours of measurement in 74% of untreated individuals and in the first nine hours of measurement in 72% of hypertensive individuals receiving antihypertensive medication.[12] However, the reproducibility of nocturnal BP, which is more closely related to the risk of cardiovascular events, is not affected.[6,13]

This ABPM pressor effect explains the change in dipping pattern from one recording to another, because nocturnal BP remains unchanged. On day 1, since waking BP levels are higher, there is a more marked drop from waking to sleeping BP. This favors dipping patterns. On day 2 of recording, since waking BP does not increase as much, the difference between waking and sleeping is attenuated, which favors the non-dipping pattern. As a result, one-third of individuals classified as dippers in the first 24 hours become non-dippers on the second day of recording.[12]

In reality, a 24-hour BP recording is too short to accurately characterize circadian variations. This limitation of ABPM is overcome if the recording time is increased from 24 to 48 hours and measurements are made at one-hour intervals.[13]

According to Hermida et al.,[13] the reproducibility of any estimated parameter in a time series depends more on the duration of monitoring than on sampling rate. Mean sleeping and waking systolic and diastolic BP can therefore be calculated with much greater accuracy when the duration of BP monitoring is increased from 24 to 48 hours, despite the marked reduction in the frequency of measurements, which are taken at intervals of one or even two hours.[13]

The results of numerous studies support the validity of 48-hour ABPM and its superiority over 24-hour ABPM in analyzing BP variability, diagnosing hypertension, assessing response to treatment, and stratifying cardiovascular risk.[14–16]

It should be pointed out that the relative lack of data on the participants in Araújo et al.'s study limits the interpretation of some of its results and calls into question the validity of its conclusions. In addition to age, gender, body mass index and treatment status, other parameters should have been collected and included in the analysis due to their ability to affect the occurrence of nocturnal hypertension and non-dipping patterns. These include the presence of diabetes,[17] African ancestry,[18] high salt intake[19] and impaired renal function.[20] Non-dipper and reverse dipper (riser) patterns are associated with a decline in renal function in individuals with chronic renal disease, while the reverse dipper pattern is commonplace in end-stage chronic renal disease.[21]

Some data on antihypertensive treatment in the study population, such as drug class and time of administration, should also have been collected, due to their impact on dipping and their importance in interpreting the results. Diuretics should have been taken into account because they can restore nocturnal BP fall due to their natriuretic effect and restoration of sodium balance,[22] and the time of administration should have been recorded because it can change the circadian BP pattern.[8,14,23] Taking antihypertensive drugs whose therapeutic effect lasts less than 24 hours will tend to produce a non-dipper pattern when taken on rising. In contrast, taking them at bedtime (especially angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers and calcium channel blockers, which have a shorter therapeutic half-life) improves BP control and helps to restore the dipper pattern in hypertensive patients.[23,24]

Some of the conclusions of Araújo et al. are unsupported by the evidence, such as when the authors question the value of the supposed relationship between a non-dipper pattern and cardiovascular risk because this pattern is also found in the normotensive population. The risk of cardiovascular events is in fact known to increase in normotensive patients with reduced nocturnal BP fall, a phenomenon that has been termed the normotensive non-dipper paradox.[25] Contrary to the implication of the authors' argument, the non-dipper pattern is not benign, since normotensive non-dippers have greater left ventricular mass[25–27] and higher cardiovascular mortality.[6]

This pattern in normotensive patients may be due to the pressure natriuresis described by Guyton: elevated blood pressure promotes the excretion of sodium and water, reducing blood volume.[28]

The lack of key data on patients and their treatment warrants some caution in proposing relationships between circadian patterns and their determinants, especially since the observed patterns have a tendency to change in the same individual, due to their low reproducibility on 24-hour ABPM.

The time has come to replace 24-hour ABPM with 48-hour monitoring for the diagnosis of hypertension and accurate cardiovascular risk assessment.

Conflicts of interest

The author has no conflicts of interest to declare.

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