

Editorial

ABPM in patients with heart failure: a long way to go

MAPA en insuficiencia cardiaca: un largo camino por andar

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Heart failure (HF) is a chronic disabling disease that affects 3% of primary care patients (and 15% of those older than 80 years).¹ The most common risk factor is hypertension (HT).² The pathophysiologic mechanisms underlying ventricular failure include acute or chronic alterations to ventricular loading conditions (preload and afterload) and/or changes to myocardial function (contractility and relaxation).³ It can be assumed that a patient with essential HT will have loading alterations that can negatively affect ventricular function, either directly or via compensatory neurohormonal activation that can interfere with cardiac function. Effective BP control is therefore crucial for preventing HF onset and unrelenting progression. It is important to recall that HF has an overall 5-year mortality of 50% to 75%, and that no major prognostic differences have been observed between patients with different left ventricular ejection fraction (LVEF) ranges.⁴ The 4 pharmacologic pillars of HF and reduced LVEF (HFrEF, defined by an LVEF \leq 40%) are sacubitril-valsartan, β -blockers, aldosterone blockers, and sodium-glucose cotransporter 2 inhibitors. These drugs all lower BP, but there is insufficient evidence on optimal targets for patients with HF and its different subtypes. BP in patients with HF is usually measured in clinical settings and/or in the home or usual place of residence. Office readings, however, must be interpreted with caution as they can fluctuate significantly, and they are clearly surpassed by ambulatory BP monitoring (ABPM) readings obtained using classic self-measurement techniques, or in time perhaps validated cuffless devices.⁵

ABPM has not been widely studied in the setting of HF. Ambulatory control provides a much higher number of readings than office-based assessments, and it can also modify perceptions of controlled or uncontrolled BP, typically formed within minutes based on a few readings. To our knowledge, no studies have estimated the prevalence of white coat HT (high mean office BP and normal out-of-office BP) or masked HT (normal office BP and high out-of-office BP) in patients with HF. ABPM also captures both daytime and nighttime BP, allowing for better interpretation of underlying neurohormonal mechanisms. The riser pattern (a higher mean systolic BP [SBP] at night than during the day, attributable to a loss of physiological circadian rhythms) has been

found to be more common in patients with HF and preserved LVEF (HFpEF, defined by an LVEF \geq 50%) than in those with HFrEF (LVEF $<$ 40%).⁶ The riser pattern has also been linked to a higher incidence of cardiovascular and all-cause mortality.⁷ A day-to-night dip in SBP of less than 10% (the nondipper pattern) has also been associated with a 5-fold increase in HF hospitalization and mortality in older patients (mean age, 76 years) with chronic HF.⁸ We are unaware of any studies linking nighttime BP patterns to LVEF.

In a recent article published in *Revista Española de Cardiología*, Bagudá et al.⁹ report the results of a laudable study conducted in the HF units of 2 Spanish teaching hospitals. The authors performed 24-hour ABPM in 266 outpatients (mean age, 72 years) with stable HFrEF (46% of patients), HFmrEF (HF with mid-range EF, defined by an LVEF of 41%–49%) (23%), or HFpEF (31%). HT phenotypes and nighttime patterns were established using widely accepted definitions from the latest guidelines on HT and HF.^{2,10} Seventy-nine percent of patients in the series had a previous diagnosis of HT. Against this epidemiological background, the 4 HT phenotypes revealed by ambulatory vs office BP measurements (uncontrolled HT, controlled HT, white coat HT, and masked HT) were as follows:

1. Thirty patients (11%) had normal office BP ($<$ 140/90 mmHg) but high out-of-office BP (\geq 130/80 mmHg). In other words, they had masked HT.
2. Twenty-seven patients (10%) had a diagnosis of uncontrolled HT (high office BP \geq 140/90 mmHg) confirmed by ABPM (\geq 130/80 mmHg). Thus, 57 patients (21%) thus were found to have uncontrolled out-of-office BP, information that was undoubtedly of great value for guiding possible changes to HF treatment strategies.
3. A total of 181 patients (68%) had a diagnosis of controlled HT (normal office BP \geq 140/90 mmHg) confirmed by ABPM ($<$ 130/80 mmHg).
4. Twenty-eight (11%) of 55 patients with high office BP (\geq 140/90 mmHg) had normal out-of-office BP ($<$ 130/80 mmHg). In other words, they had white coat HT, a diagnosis that does not normally result in treatment modifications and provides peace of mind for both patients and physicians.

In short, ABPM confirmed controlled BP in 209 patients (79%) in the study by Bagudá et al.⁹, showing its potential for guiding

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decisions on possible changes to pharmacologic treatments in patients with HF. If we accept that ABPM measurements should take priority over in-office or routine home measurements obtained using an arm cuff or other method, we can safely assume that ABPM will have helped guide several treatment decisions, including the probable decisions to maintain current antihypertensive treatment in 79% of patients (68% with controlled HT and 11% with white coat HT) and to modify it (or other treatments) in 21% (11% with masked HT and 10% with uncontrolled HT).

In their analysis of nighttime BP, Bagudá et al.⁹ observed the nondipper pattern (day-to-night dip in SBP of < 10%) in 43% of patients and the riser (reverse) pattern (higher mean SBP at night) in 26%. In other words, most patients with HF (69%) had pathologic nighttime BP values, which are usually attributed to a marked adrenergic overdrive.¹¹ This pathologic profile was not predominant in any of the 3 LVEF categories.

One relevant finding of the above study was that patients with HFrEF had a significantly lower (and clinically relevant) mean daytime SBP (109 mmHg) than those with HFmrEF (117 mmHg) or HFpEF (119 mmHg). The differences were even greater for nighttime SBP (9–13 mmHg vs 8–10 mmHg for daytime measurements), with mean values of 103 mmHg in patients with HFrEF, 112 mmHg in those with HFmrEF, and 116 mmHg in those with HFpEF. One important conclusion to emerge is that patients with HF and an LVEF ≤ 40% have significantly lower and potentially more harmful daytime and in particular nighttime SBP than those with a higher LVEF.

The study was performed in the HF units of 2 hospitals, but given the paucity of data in this area (or at least that of complete, systematically collected data), the epidemiological profile described by Bagudá et al.⁹ may be reasonably representative of what could be considered optimal care practice. It also provides important insights into the current situation and treatment of HF in patients with different HT/LVEF phenotypes and daytime and nighttime ABPM patterns.

The authors are to be commended because their findings raise several questions. Should ABPM be performed in all patients with HF or only in those with uncontrolled office HT (BP ≥ 140/90 mmHg) to rule out white coat HT? How can masked HT be identified in patients with HF? Should therapeutic action be taken in patients with HF with a nondipper or riser pattern detected by ABPM or in patients with an LVEF ≤ 40% and a mean daytime BP < 110–120 mmHg? What are the hospitalization and mortality rates in patients with HF and the different HT phenotypes identified by ABPM? In relation to the first question, 58 patients (22%) were incorrectly diagnosed by office BP assessment (they actually had masked or white coat HT). This rate is significant, although lower than that reported in a study of almost 105 000 patients (mostly from primary care settings) in the Spanish ABPM registry, which found that 40.6% of patients with essential HT had been misclassified using office-based BP measurements.¹² The above findings highlight the usefulness of ABPM in improving HT diagnosis in patients with HF. A correct diagnosis is important to ensure that patients, particularly frail ones, are receiving the right pharmacologic treatments. Probability-based studies aiming to optimize the use of ABPM, such as a recent study that designed a scoring system to screen for masked HT in patients with essential HT,¹³ could be very useful, particularly considering the poor prognosis associated with masked HT.¹⁴ Other questions relating

to the prognostic value of ABPM will need to be answered through prospective studies such as that of Camafort-Babkowski et al.,¹⁵ analyzing patients with stable HFpEF.

Significant efforts are required to advance research on the role of ABPM in HF, but therein lies a strong opportunity for those seeking to answer any of the above questions or many others yet to be formulated. These endeavors should provide clearer insights into how to best treat acute or chronic HF according to the 3 current LVEF categories, the 4 HT phenotypes offered by ABPM, and the 4 accepted nighttime patterns. As Jorge Bucay said “The path marks a direction. And a direction is much more than a result”.

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CONFLICTS OF INTEREST

None.

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