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COMMENT

Home blood pressure monitoring for improved risk assessment in heart failure: are brachial measurements sufficient?

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Editorial Commentary: HTR-2024-1161. Komori T et al. "The prognostic impact of home blood pressure measurements in patients with stage B heart failure"

The increasing global burden of heart failure (HF) has been a significant driver for recent updates of universal definitions and classification of HF, which have been endorsed by professional societies world-wide [1]. The classifications are broad and detailed and encompass structural, functional and symptomatic development of the disease as it progresses along four main stages: (i) stage A, individuals "at-risk" for HF including the presence of hypertension; (ii) stage B, individuals classified as "pre-HF", without current or prior symptoms of HF, but having at least one structural or functional abnormality; (iii) stage C, individuals with current or prior symptoms due to existing structural or functional abnormalities; (iv) stage D, individuals with advanced HF and who have sever symptoms requiring hospitalisation or advanced therapy.

Progression of HF can be insidious, particularly in the pre-clinical phase of stage A and stage B, conditions in which elevated blood pressure (BP) is a significant risk factor, and which can be used as an early treatment target to mitigate developing cardiovascular risk [2]. Progression from stage A to stage B is significant, since, although at a decreasing rate, there is evidence of cardiac structural and morphological changes as well as biomarker increase in brain natriuretic peptide (BNP) [3]. Hence, in addition to high BP being a potential modifiable factor for overall cardiovascular risk, it is particularly important for assessment of risk carried by the progression of HF, and specifically by the level of elevated BP considered to be in the hypertensive state in the context of diurnal changes of BP.

The study by Komori et al. [4] in this issue of *Hypertension Research* investigates the association of hypertension as defined by home BP measurements (day and night) and incidence of cardiovascular events in patients diagnosed with stage B HF. The study is an extension of previous studies addressing associations of morning and evening BP [5] and nighttime BP [6] with cardiovascular risk, all being part of the extensive nationwide Japan Morning Surge-Home Blood Pressure (J-HOP) study conducted in 4310 participants with history or presence of risk factors for cardiovascular disease during the period of 2005–2012. The retrospective analysis by Komori et al. [4] was conducted in 568 J-HOP participants with stage B HF and with at least one cardiovascular risk factor. The mean follow-up period was 7.8 ± 3.6 years, during which time 66 events occurred. In the cohort analysed, stage B HF was defined according to the universal classification [1], with the presence of at least one of the following factors: (i) brain natriuretic peptide (BNP) ≥ 35 pg/mL, (ii) N-terminal pro b-type natriuretic peptide (NT-proBNP) ≥ 125 pg/mL, (iii) Troponin T > 0.014 ng/mL, (iv) left ventricular ejection fraction (LVEF) < 50%, (v) enlarged left ventricular dimensions in diastole (LVDd) (≥60 mm in men and ≥54 mm in women), (vi) enlarged left atrium (>40 mm in men, >38 mm in women), or (vii) increased left ventricular mass (>115 g/m^2 in men, >95 g/m^2 in women). Daytime home hypertension (morning and evening measurements) was defined as systolic BP (SBP) ≥ 135 mmHg, nighttime hypertension as SBP ≥ 120 mmHg and office hypertension as SBP ≥ 140 mmHg. Home and office BP were measured with a similar device (Omron, HEM-5001) and the same device was used for nighttime BP.

When accounting for all potential confounding factors in the models, the analysis showed a sustained significant association between nighttime hypertension and

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cardiovascular events in participants with stage B HF ($p = 0.03$). There was no significant association with other categories of hypertension. This suggests the potential importance of achieving optimal nighttime BP control to mitigate risk in stage BP HF. While these are significant and informative findings of the analysis, there are several factors that need to be considered to enhance the interpretations of the findings of the study.

An important consideration when assessing the role of the hemodynamic load due to arterial BP in HF is the effect of structural changes in the myocardium on ventricular-vascular coupling. During systole, the dynamic load on the contracting myocardial fibres is presented by the pressure in the ascending aorta, the pressure proximal to the heart. Under normal conditions, fibre shortening will accommodate changes in afterload so that stroke volume is commensurate with the regulated cardiac output based on metabolic demand. However, in the failing heart, there is a more pronounced dependency of fibre shortening on afterload, hence stroke volume is more strongly affected by changes in aortic pressure. The implication of this mechanism is that the operation of the heart, in terms of pump function (pressure-stroke volume) characteristics, changes from being closer to a constant flow source under normal conditions (or more strictly a constant power source) towards a constant pressure source in HF [7], suggesting that in HF, a small change in aortic BP can have a larger effect on stroke volume compared to the normal heart (Fig. 1A). This will have a significant hemodynamic effect in characterising ventricular-vascular coupling in HF [7]. A further consideration is the physiological amplification of the of the arterial pulse as it travels from the central aorta toward peripheral sites, suggesting that brachial SBP is usually higher than aortic SBP [8]. The amplification is heart rate dependent and has recently been shown to be associated with cardiovascular risk [9].

In the study by Komori et al. [4], 40.9% of participants in the cohort with events were on beta-blockers compared to 18.5% in the control group. Since beta-blockers lower heart rate, this results in aortic SBP being relatively higher for the same brachial SBP [10] (Fig. 1B). Hence a large proportion of the cohort with events had a much higher aortic SBP than the control group, as well as aortic SBP having a different distribution to brachial SBP in the events cohort. Furthermore, these effects could not be accounted for in the models since the aortic SBP values and distribution in the whole study group was not known. Indeed, changes in heart rate per se, independent of medication and as occur at night, would have an additional confounding effect in the association of BP and events in HF as central aortic BP would be a more relevant characterisation of ventricular afterload than brachial SBP. This notion has been supported by studies showing the relevance of pulsatile hemodynamics in

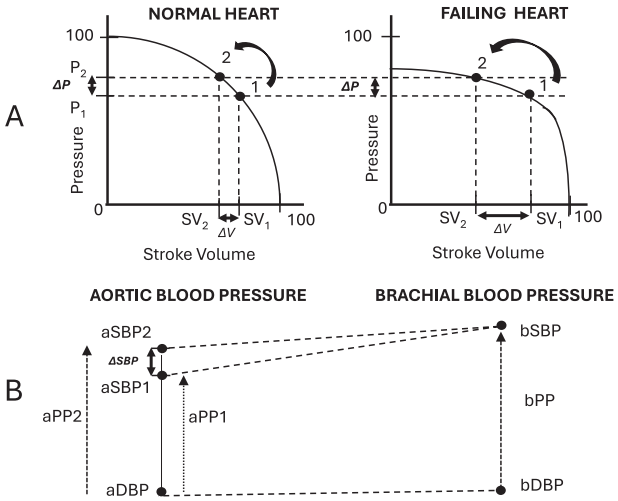

Fig. 1 **A** Schematic illustration of pump function curves for normal and failing heart. Pressure and stroke volume axes are normalised to a scale of 100% for maximum pressure (at zero ejection) and maximum ejection (zero pressure load). In heart failure, the curve is flattened towards the volume axis, increasing the ejection sensitivity to pressure afterload. When the operating point moves from "1" to "2" due to a similar pressure increase (ΔP), the decrease in stroke volume (ΔV) is much greater in heart failure. **B** Schematic illustration of pulse pressure amplification between the aorta (aPP1) and brachial artery (bPP) for similar diastolic pressure (aDBP, bDBP), with the corresponding aortic systolic pressure (aSBP1). For reduction in heart rate (as occurs with beta-blockers), a similar bPP would correspond to a higher aortic pulse pressure (aPP2), hence giving a higher aortic systolic pressure (aSBP2). This difference (ΔSBP) can be a significant factor in analysing effects of arterial blood pressure in heart failure due to the increased pressure sensitivity of ventricular contractility and ejection (panel **A**), and if not taken into account it can be a potentially significant confounding factor in the analysis of cardiovascular risk. The effect will be masked if only bSBP is used in analyses where there are changes in pulse amplification. It can only be taken into account if aortic pressure is known

HF [11, 12] including the use of the central aortic waveform to guide treatment in HF [13]. Hence, given the relatively higher aortic SBP in a large proportion of the stage B HF cohort analysed due to reduced heart rate effect of beta-blockers, it is conceivable that the significant association of nighttime hypertension as assessed by brachial SBP might well be underestimated.

The definition of stage B HF includes the presence of increase in circulating volume, as is manifest by elevated levels of BNP and NT-proBNP. This suggests that the increased vascular volume of the arterial compartment places the arterial wall under increased tension and so enhances arterial BP, while possibly also affecting arterial stiffness. It also includes presence of structural changes affecting cardiac contractility, making the ejecting ventricle more dependent on dynamic pressure afterload in the proximal aorta. These potentially confounding issues add to the list of limitations described by Komori et al. [4]. An additional finding of the analysis was that the hazard ratio

for nighttime home hypertension was attenuated when morning and nighttime home hypertension were included in the same model. This suggests that there may have been some overlap in the hypertensive cohorts as defined by brachial SBP. It is of interest to speculate whether such overlap might be clarified by noninvasive measurement of central aortic SBP in stage B HF.

Analyses of the J-HOP cohort have provided insightful information on the potential importance of home BP measurement in the population, and particularly in groups with elevated risk of the broad spectrum of cardiovascular disease [4–6]. The added value of the analysis by Komori et al. [4] is the first demonstration of the relevance and benefit of home BP measurement in stage B HF and the significant association of nighttime hypertension as defined by brachial SBP with cardiovascular events. An important caveat in the interpretation of the findings of the analysis is that the failing heart becomes increasingly sensitive to changes in the pressure afterload in the proximal aorta, which may be different to that quantified by brachial SBP. Inclusion of aortic SBP in the statistical models would have the potential for improved separation of the different levels of risk in the whole cohort. Notwithstanding the study limitations and confounding issues, the analysis reported in the study by Komori et al. [4] makes a significant contribution in the pathway to establishing BP treatment targets in HF defined as stage B by the universal classification criteria [1].

Compliance with ethical standards

Conflict of interest The author declares no competing interests.

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