Blood Pressure, Left Ventricular Hypertrophy, and Congestive Heart Failure: A Continuum
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Abstract
The left ventricular hypertrophy (LVH) predicts adverse outcomes in hypertension. However, it is a crude and imprecise index of risk occurring late in the evolution of complicated hypertension. Heart failure (HF) is a major complication of hypertension, but its onset and syndromes are heterogeneous, and clinical definitions and risk thresholds are imprecise. Once LVH occurs, the window to the development of HF has opened. Imaging techniques may provide early insights into the structural basis of HF and track ventricular remodeling. Ventricular-arterial coupling analysis techniques provide an added opportunity to understand how HF evolves within this window, and so inform tailored management of hypertension and adverse LV loading (hydraulic and myocyte afterload) conditions in the continuum from LVH to overt HF.

Key words: Arterial hemodynamics, arterial stiffness, heart failure, hypertension, left ventricular hypertrophy, ventricular-arterial coupling

Brachial Cuff Blood Pressure Provides Limited Insight into Heart Failure Mechanisms
Elevated blood pressure (BP) relates to long-term prognosis including the development of heart failure (HF),¹ and its control will effectively prevent HF.² In addition to absolute systolic and diastolic pressures, recording widened pulse pressure, patterns of variability, ambulatory BP phenotypes, and even non-linear patterns of pressure values provide mechanistic insights into the pathophysiology of complicated hypertension. Nonetheless, the brachial cuff BP can have limited value in informing the development of the left ventricular hypertrophy (LVH) and complex evolution of HF later in hypertension. HF is not always heralded by the presence of LVH despite the presence of impaired diastolic function.³ Structural remodeling, which follows treatment of hypertension or HF, may occur within the myocardium as well as in large conduit arteries,⁴ with limited structural and functional information provided by brachial cuff measurements.

Central aortic BP also relates to prognosis, ventricular remodeling, and complications of hypertension.⁵ Its effects in hypertension can be seen through the differential influence of medications or heart rate.⁶ Due to the heart rate dependence of pulse amplification between the aorta and brachial artery,⁷ beta-blockers have been shown to have a reduced effect on regression of LVH compared to other antihypertensive agents for a similar decrease in brachial systolic pressure.⁸ Being a more proximate measure of the ventricular-arterial (V-A) coupling interface, it may more closely reflect LV loading conditions; however, large cohort studies are awaited to establish the clinical value of central aortic pressure in treatment and management of hypertension and associated cardiac complications.⁹

The definition and onset detection of HF, which is a clinical syndrome, remains multifaceted¹⁰ and cannot be evaluated by simply recording arterial pressures. Even though hypertension is a risk factor for the development of HF, a rise in BP accompanies clinical improvement and predicts a better prognosis in HF.¹¹ Despite the increased understanding of various phenotypes of hypertension,¹²,¹³ it remains unclear which are the best parameters obtained from a 24 h ambulatory BP recording,¹⁴ for example, which influence or promote LVH and predict the development of HF.

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Left Ventricular Hypertrophy is a Maladaptive Response

LVH is a well-defined structural biomarker for increased risk in hypertension, in particular for the development of HF.[23] Once HF occurs, it presages a poorer clinical outcome. Targeting LVH is appropriate and effective in hypertension.[24] It is useful to consider the finding of LVH as representing a failing heart, evidenced by deranged neurohumoral, microcirculatory, and inflammatory biomarkers which are recognized hallmarks of HF.[17]

Techniques such as cardiac magnetic resonance imaging, echocardiography, and positron emission tomography in LVH may show significant abnormalities consistent with myocardial dysfunction, which are not accompanied by symptoms.[18] The use of arterial and pulse wave analysis techniques[19] provides a further opportunity for HF to be more clearly understood and managed much earlier along the pathophysiologic continuum, as hypertension evolves to complicated forms.

“LV remodeling,” a term used to describe structural heart changes without hypertrophy, may be an earlier marker for abnormal myocardial function which may warrant intervention.[20] Although LVH or remodeling may only be crude indicators of impaired LV performance, they mark prominent red flags along the risk continuum.

Assessing Ventricular-Arterial (V-A) Dynamics in Understanding HF

Assessing V-A coupling using various modalities provides more detailed understanding of mechanisms of HF and other cardiovascular disease. V-A coupling has also been proposed as a means to manage a variety of cardiovascular syndromes including HF,[21] providing insights into pathophysiology, energetics, fibrosis, and remodeling.

V-A coupling reflects the fundamental notion that the ventricle, aortic valve, aorta, and peripheral arteries are separate organs in series. Ventricular stroke volume affects arterial performance, and dynamic arterial characteristics affect ventricular function. BP is the result of flow generated by the heart meeting the resistance of the arterial tree. The development of LVH and HF (either HF with reduced ejection fraction [HFrEF] or HF with preserved ejection fraction [HFpEF]) is a result of the maladaptive interaction of the heart and the arterial tree.[22]

The “gold standard” technique of measuring ventricular performance is based on pressure-volume loop relationships within the ventricular chamber, which is load independent. This uses the ratio of effective arterial elastance (EA) to LV endsystolic elastance (EES).[23] However, this technique does not take into account the pulsatile characteristics of V-A coupling nor does it impart any information about the myocardium itself. It appears to be of limited value in assessing HFpEF. This has led to recent calls to include comprehensive measures of pulsatile arterial dynamics in assessing V-A coupling.[24] This seems to be useful in HFpEF, which happens to be the earliest manifestation of HF in hypertension.

Various hemodynamic modalities available to assess V-A coupling include pulse wave velocity (PWV) and reflection analyses, wave intensity analysis, wave power analysis, global longitudinal strain (GLS) and tissue Doppler echocardiography, measurement of characteristic impedance of the proximal aorta, and obtaining PWV to GLS ratios.[17,24,25] Fibrosis, inflammation, and oxidative stress are common biochemical pathways linking impaired V-A function.[26] Their direct evaluation requires detailed techniques which are not yet widely available in routine clinical use but can be inferred by current V-A modalities.

Arterial load consists of steady and pulsatile components. Total peripheral resistance is the measure of the steady component and depends on microvascular properties such as stiffness and reflectivity. The pulsatile component of LV afterload is influenced by the elastic properties of conduit vessels, which includes the aorta and distal muscular arteries, and wave propagation phenomena, including intensity and timing of peripheral wave reflection.[24] Pulsatile LV load may then be measured by the characteristic impedance of the proximal aorta, the magnitude and timing of wave reflections, and the total arterial compliance.

Furthermore, LV afterload should be distinguished from myocardial afterload. LV afterload is defined as the hydraulic load imposed on the LV by arterial pressure, and myocardial afterload is the wall stress imposed on myocytes to generate fiber shortening.[27] It is now understood that complex patterns of LV afterload and wall stress evolve as patients develop the syndrome of heart failure.[28-30] Carotid to femoral pulse wave velocity (PWV), aortic characteristic impedance, and the magnitude and timing of wave reflections during systole summate the impact of arterial load on LV function throughout the cardiac cycle, linked in turn to clinical syndromes and cardiovascular events.[31-32]

Dividing myocardial dysfunction syndromes into HF with reduced ejection fraction (HFrEF) and HF with preserved ejection fraction (HFpEF) by arbitrary cutoff values belies the variations and complexities of loading and structural conditions which provoke HF at the V-A interface, as well as the varied influence of neurohumoral, cellular, and biochemical alterations.[32]

While imaging techniques such as echocardiography, cardiac magnetic resonance, and isotopic techniques provide valuable information regarding structural changes and clinical risk in HF from hypertension,[33] novel techniques extending their use in assessing V-A coupling have emerged and now may also be applied to tailor treatment in heart failure syndromes.[25]

In HFpEF, arterial waveform indices (which are measures of pulsatile function) have been shown to match the ability of echocardiographic tissue Doppler parameters in establishing the diagnosis.[34] A recent study described the utility of tailoring heart failure therapy by measuring and adjusting aortic pulsatility in HFrEF,[34] employing radial applanation tonometry to estimate the central aortic pressure. This is based on the idea that the true hydraulic load of a failing LV occurs at the level of
the central aorta and cannot be strictly assessed by peripheral BP cuff measurements. However, in HFrEF, it has been shown that effective treatment of HF with angiotensin receptor blockade and nepriylin inhibition is not associated with remodeling of the proximal aorta, as measured by applanation tonometry and echocardiography.

Conclusions

As an adverse adaptation in hypertension, LVH may precede the clinical syndrome of HF, but is poorly predicted by brachial cuff pressure alone. Central aortic pressure may be a better predictor of the left ventricular hypertrophy and reflect ventricular loading conditions.

More refined assessment of arterial load at the V-A interface has the potential to translate into more effective and individualized management of HF and adds to the value of imaging and biomarker techniques.

Elevated BP initiates the progression from LV remodeling to failure through a process involving altered LV loading conditions, distorted myocyte structure or hypertrophy, neurohumoral, and circulatory changes [Figure 1]. Recognizing this continuum will enable the clinician to supplement brachial pressure readings with myocardial imaging techniques and newer modalities of dynamic arterial analysis for assessing severity and prognosis in hypertension.

The challenge is to find the best way to bring these novel techniques to the bedside in the least complex way.

Figure 1: The continuum pathway for blood pressure, left ventricular hypertrophy, and heart failure

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