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Author Response to LTE JCH-16-0356

Title

Cardiac magnetic resonance imaging provides a new insight in hypertensive heart disease – a reply

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Conflict of interest

Dr. Chiara Bucciarelli-Ducci is a Consultant for Circle Cardiovascular Imaging Inc.,
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Letter to Editor

We thank the readers for their interest in our article(1).

With regards to the request for correlation with office and ambulatory blood pressure readings, these data are provided in the manuscript; end-diastolic wall thickness (EDWT) correlated with office SBP ($R=0.43$, $P<.001$) and office DBP ($R=0.32$, $P<.005$) but did not correlate with ABPM SBP ($R=0.24$, $P=.12$), ABPM DBP ($R=0.18$, $P=.27$), or ABPM mean arterial pressure ($R=0.18$, $P=.27$).

We agree that echocardiography is currently the imaging modality most frequently used to investigate subjects with arterial hypertension, but cardiovascular magnetic resonance imaging (CMR) has the potential to offer a 'one-stop' comprehensive assessment of subjects with hypertension, both to screen for secondary causes and identify end-target organ damage(2).

We elected to perform CMR rather than echocardiography because of 1) its increased spatial resolution and tissue contrast and 2) the suboptimal imaging due to the relatively high prevalence of concomitant obesity in our cohort. We have not performed a direct comparison of echocardiographic and CMR findings but agree it would be interesting to perform such an analysis.

We agree that left ventricular (LV) geometry is an interesting parameter. We provide data on absolute wall thickening relative to the LV end-diastolic diameter to correct for changes in LV cavity size. Absolute wall thickening is determined by both end-

diastolic wall thickness and radial strain. The proportional decrease in cavity size during systole is more closely determined by end-diastolic dimension than volume. Correction of 1-dimensional measurement (absolute wall thickening) by another 1-dimensional measurement (diameter) intuitively makes more sense than by a 3-dimensional measurement (volume).

We provided information on longitudinal shortening, which is mathematically equivalent to longitudinal engineering strain (but with an opposite sign). We chose to calculate mid wall myocardial shortening instead of mean circumferential strain as there is a large strain gradient between the epicardium and endocardium.

We agree the interaction between diffuse myocardial interstitial fibrosis and myocardial mechanics in hypertensive heart disease is an interesting area. Further to the study by Kuruvilla et al.(3), our group has shown that the prevalence and degree of LV fibrosis and central aortic distensibility vary amongst different hypertensive heart disease LV phenotypes(4,5). We agree that such new insights are important in furthering our understanding of the pathophysiology of hypertensive heart disease and our appreciation of different potential aetiologies and drivers of the hypertensive state. Such insights should help us to tailor existing anti-hypertensive treatments more effectively and highlight potential substrate for new anti-hypertensive strategies.

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