Distribution of myocardial work in arterial hypertension: insights from non-invasive left ventricular pressure-strain relations

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Declaration

Ethics Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The study was approved by the Comite Etico de Investigacion Clinica, Hospital Clinic, Barcelona, reference number: HCB/2015/0455.

Consent to participate

Informed consent was obtained from all individual participants included in the subjects.

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Conflict of interest

None.

Abstract

Background: A index of non-invasive myocardial work (MWI) can account for pressure during the assessment of cardiac function, potentially separating the influence of loading conditions from the influence of the underlying tissue remodelling. The aim is to assess LV function accounted for loading and explore hypertensive MWI distribution by comparing healthy individuals to hypertensive patients without and with localized basal septal hypertrophy (BSH).

Methods and results: An echocardiogram was performed in 170 hypertensive patients and 20 healthy individuals. BSH was defined by a basal-to-mid septal wall thickness ratio ≥ 1.4. LV speckle-tracking was performed, and the MWI calculated globally and regionally for the apical, mid and basal regions. An apex-to-base gradient, seen in regional strain values, was preserved in the distribution of myocardial work, with the apical region compensating for the impairment of the basal segments. This functional redistribution was further pronounced in patients with localized BSH. In these patients, segmental MWI analysis revealed underlying impairment of regional work unrelated to acute loading conditions.

Conclusions: Non-invasive MWI analysis offers the possibility to compare LV function regardless of blood pressure at the time of observation. Changes in MWI distribution can be seen in hypertension unrelated to the load-dependency of strain. Accentuated functional changes affirm the role of BSH as an echocardiographic marker in hypertension.

Keywords: hypertension, remodeling, myocardial work, basal septal hypertrophy, speckle tracking

Introduction

Myocardial deformation is influenced by remodelling of the myocardium - hypertrophy and myocardial damage, and by loading conditions - the load-dependency of strain. In clinical assessment of arterial hypertension both of these influences are relevant when interpreting left ventricular (LV) function. Systolic myocardial deformation is load dependent, showing decreased deformation in the setting of elevated afterload, as seen in experiments with aortic constriction in animal models [1] or handgrip exercises in healthy individuals [2]. Load dependency can, therefore, result in misrepresentation when assessing patients with high blood pressure. Moreover, in hypertension there is a continuous spectrum of LV remodelling influencing cardiac function. With prolonged exposure to an increased afterload, the LV undergoes structural and functional remodelling[3] resulting in concentric hypertrophy in a high percentage of patients [4]. However, the thickening of the myocardium is gradual and non-uniform [3], and in earlier stages some patients present with basal septal hypertrophy (BSH) [5,6] – localized remodelling recognized as a tell-sign of increased afterload [4]. In this setting, the heart has to boost performance to maintain the ejection of blood into the circulation [7], however, the way this increased workload is distributed inside a structurally changed LV is still widely unexplored.

The novel concept of LV pressure-strain loops [8] allows non-invasive estimation of an index of myocardial work (MWI), and moreover, by looking at segmental deformation, the exploration of regional distribution of the workload in different cardiac conditions. When investigating LV function with MWI, we can account for acute loading over the course of the cardiac cycle, potentially separating influences of loading conditions from the impact of chronic remodelling on regional deformation. Therefore, non-invasive MWI may offer the possibility to compare LV function in diseased hearts regardless of blood pressure at the time of observation. The aim is to assess LV function accounted for loading conditions and explore hypertensive MWI distribution by comparing healthy individuals to hypertensive patients without and with localized BSH. We hypothesise that remodelling seen in hypertension will influence the way myocardial work is regionally distributed inside the LV.

Materials and Methods

Studied population

The cohort consisted of 209 participants – 185 hypertensive patients and 24 healthy individuals. The participants were studied at two centres - Hospital Clinic Barcelona and the University Hospital Centre in Zagreb.

Clinically managed hypertensive patients treated during a minimum of 3 years with antihypertensive drugs, were included. Exclusion criteria were history of heart failure, moderate or severe valvular disease or previously known target organ disease. Healthy individuals included volunteers from the local community, presumed healthy, without prior history of hypertension, diabetes or other significant cardiac or non-cardiac diseases. All participants were assessed for cardiovascular risk factors, family medical history, comorbidities, and pharmacological treatment - followed by a cuff blood-pressure measurement and a comprehensive echocardiographic examination. The study was in accordance with the Declaration of Helsinki and the ethical standards of the responsible committee on human experimentation. All participants gave written informed consent.

Echocardiography

Examinations were performed in line with current recommendations[9] on a commercially available Vivid 9 system (GE, Vingmed Ultrasound, Horten, Norway) equipped with a M5S transthoracic transducer.

All patients were studied with conventional 2D, Doppler, and speckle tracking deformation imaging. The thickness of the anterior and inferior septum was measured at end-diastole in the basal- and mid-level in the parasternal long axis (PLAX) and 4-chamber cardiac views, respectively. Measurements were obtained starting from the interface between the myocardial wall and cavity to the transition of the LV to the RV septal myocardium [10]. The posterior wall thickness and LV internal diameter were measured in PLAX view. BSH was defined based on the basal-to-mid septal wall thickness ratio of ≥ 1.4 in either the 4-chamber or PLAX view. LV volumes were calculated using the Simpson biplane method, and LV mass was calculated by the linear method and normalized by body surface area, and sex-dependent cut-off values were applied to indicate LV hypertrophy [9].

Myocardial deformation of the LV was assessed using speckle tracking echocardiography software on 2D grayscale images obtained from the 4-chamber, 3-chamber and 2-chamber cardiac views, respectively. Images were analysed using GE Echopac software (GE Medical Systems, version 202.41.0) by manually tracing the endocardial border at end-systole of the LV. Longitudinal strain curves were generated, and end-systolic strain, determined at the aortic valve closure time, was measured in all LV segments based on the 18-segment model. LV global longitudinal strain (LVGLS) was calculated by averaging values of the 18 segments.

The MWI was calculated using the commercially available Echopac software for MWI assessment – the methodology was validated in previous publications [1,8]. To enable the exploration of regional strain and work distribution in the LV, the ventricle was divided into 18 segments. Three regions (basal, mid-cavity, and apical) were

each divided into six segments, enabling a practical exploration of the regional characteristics in each apical plane. MWI was calculated globally and segmentally, as well as regionally for the apical, mid and basal regions, following the 18-segment model [11,12]. The relative apical MWI was calculated using the formula based on previous methods of exploration of regional differences in the LV (average apical MWI/(average basal MWI + mid-MWI))[13]. Reproducibility analysis of LV strain and the basal septal wall thickness measurements (in both the 4-chamber and PLAX views) was performed in our associated work on the same hypertensive cohort [10]. Reproducibility of MWI assessment using the GE software was recently determined in a large multi-centre cohort[14]. The schematic of the myocardial work assessment is shown in the *Supplementary Figure 1*.

Statistical analysis

The data were analysed using IBM SPSS Statistics version 23.0. The quantitative variables were expressed as mean ± standard deviation or median and interquartile range based on the normality of their distribution evaluated by the Shapiro-Wilk test. The qualitative variables were expressed as a total number and percentage. Differences between groups were analysed for statistical significance with the ANOVA test when comparing variables with normal distribution and the Wilcoxon test for non-normally distributed variables. Post-hoc comparisons were assessed with the Bonferroni correction. When comparing categorical data, contingency tables and a Chi-square or the Fisher's exact test were used for comparison. The strength and direction of the linear relationships between pairs of continuous variables was performed using the bivariate Pearson Correlation, resulting in a correlation coefficient, R. A value of $p<0.05$ was considered statistically significant.

Results

General characteristics of the cohort

From the initial cohort of 209 participants, 15 hypertensive patients and 4 healthy controls were excluded from the myocardial work analysis due to missing cardiac images or insufficient quality of the 2-chamber, 3-chamber or 4-chamber images, respectively, (n=15), missing data on systolic blood pressure measurements (n=3), or large differences in heart rate between the different cardiac views included in the analysis (n=1). The excluded patients included 4 hypertensive patients with BSH. The clinical characteristics of the remaining cohort (n=190) are presented in *Table 1*. Based on the predefined criteria: 18% (n=31) of the hypertensive patients and none of the healthy controls were classified as having BSH. Hypertensive patients and healthy controls had comparable age and gender

characteristics (median age 57 vs. 54 years (p=0.009), male sex 56% vs. 46% (p=0.463)). The subgroup with BSH was older compared to the controls. Hypertensive patients had higher BMI and systolic blood pressure at initial presentation; however, when comparing the hypertensive subgroups, no differences were noted regarding blood pressure, duration of hypertension, body mass index, comorbidities, or antihypertensive therapy.

LV size and function

Characteristics of LV size and function are presented in *Table 2*. There was a trend of smaller LV size and an increased LV mass in the BSH subgroup. Though global LV systolic function, assessed by both ejection fraction and global longitudinal strain, was preserved in all groups, there were significant differences in segmental myocardial deformation. The average strain of the basal segments was significantly lower in the BSH subgroup – primarily based on the considerable reduction in systolic deformation of the basal anteroseptal and inferoseptal segments (*Fig. 1, left*) - resulting in a visible gradient of strain value declining from apex-to-base in the hypertensive subgroups.

Distribution of myocardial work in arterial hypertension

As expected, there was a moderate correlation between global MWI and systolic blood pressure (Pearson R=0.644, p<0.001**,** see *Supplementary Figure 2A*). Accordingly, there was a clear trend in higher values of global MWI in the hypertensive subgroups.

Segmental differences in performed work are demonstrated in *Figure 1*. Considering the basal segments, the inferoseptum performed a notably decreased amount of work in hypertensive patients, a finding notably more pronounced in the BSH subgroup (*Figure 2*)**.** Moreover, the basal septum, consisting of the anteroseptum and inferoseptum, performed a reduced percentage of total basal work in BSH patients. The relationship between the basal septal MWI and systolic blood pressure showed a trend of a positive relationship in healthy individuals (Pearson R 0.429, p=0.059), a significant and moderately strong positive correlation in non-BSH hypertensive patients (Pearson R 0.548, p<0.001), however, no relationship was seen in the BSH subgroup (Pearson R 0.213, p=0.249) (*Supplementary Figure 2B*)**.** The mid segments showed no differences in work across subgroups, however, at the apical level all segments showed a trend of performing significantly more work in hypertension.

The percentage of total global work performed by the basal segments was decreased in both hypertensive groups, notably more so in the BSH subgroup. This was coupled with an increase in the percentage of total work performed by the apical segments in both hypertensive groups, with a visible trend of a further increase in the BSH

patients (*Figure 3*). A demonstration of the MWI apex-to-base gradient in 4-chamber view is shown through average global and segmental pressure strain loops in *Figure 4*. These findings were further quantified using the relative apical MWI cacluation, showing an increase in both hypertensive groups, as compared to the healthy controls, and with a further trend of increase between the non-BHS and BSH patients (p=0.05) (**Table 2**).

Discussion

Findings

MWI analysis provides the possibility to explore LV function accounted for the effects of acute loading in hypertensive patients with preserved EF and GLS. An apex-to-base gradient in the distribution of myocardial work was preserved after adjustment for loading, implying apical compensation of basal impairment in hypertension is due to underlying myocardial remodelling. This functional redistribution was further pronounced in patients with localized BSH. In these patients, segmental MWI analysis revealed the effect of chronic remodelling on myocardial function – unmasking a low capacity of the thickened basal septum to adjust to an increased workload.

Left ventricular remodelling in arterial hypertension

Localized hypertrophy and deformation impairments in hypertension are deemed related to a non-homogeneous wall stress distribution in the LV with a decrease from base to apex[15,16]. Wall stress is dependent on local LV geometry, thus regions with a greater radius of curvature are exposed to higher wall stress. The basal septum has a greater radius of curvature compared to the free wall[17,18], and with the rise of blood pressure stress increases disproportionately. The consequential imbalance between elevated wall stress and locally developed force results in decreased local deformation. Over time, this imbalance may trigger local hypertrophy, a compensating mechanism intended to maintain normal local deformation[4]. Data from large cohort magnetic resonance studies support this hypothesis, showing that the basal septum is the thickest LV segment in the majority of hypertensive patients[19], a finding not seen in equivalent studies of the healthy population[17]. Moreover, the finding of BSH in hypertension has been linked to impairment of atrial and ventricular systolic and diastolic function[10]. Therefore, BSH in hypertension can indeed be used as a marker of LV remodelling, and MWI may be a novel tool with the potential to further explore the repercussions of this morphological finding on LV function.

Myocardial work - added value for assessing left ventricular function

MWI incorporates information on deformation throughout the cardiac cycle corrected for afterload throughout

the cycle; whereas peak systolic strain at aortic valve closure gives a pressure-non-adjusted, single point value of cardiac function. These two parameters are obviously related, and depict a similar message in the setting of normal blood pressure. However, when blood pressure is elevated, strain has less reliability to reflect underlying cardiac function, whereas MWI offers insight by quantifying myocardial function relieved of the influence of the loading conditions. To better illustrate this concept, we can examine these parameters in patients with arterial hypertension and cardiomyopathy [20]. In early stages of hypertensive heart disease, LV GLS can be preserved, indicating normal LV function. However, global MWI measurements demonstrate significantly increased values of work, which may ultimately lead to adverse remodelling. On the other hand, in the setting of intrinsic contractile dysfunction, as seen in a non-ischemic dilatated cardiomyopathy, deformation imaging and MWI will both show an equivalent decrease. In everyday practice, clinicians aim to integrate information on blood pressure when interpreting the results of deformation analysis. Here, MWI can provide a simplified, single parameter and single value integration of echo and blood-pressure data reflecting the patient's cardiac status.

Work is calculated as force applied over a distance. In MWI calculations, LV pressure is used as a surrogate for force (or, more specifically, for fibre stress, which is force applied on a unit of area) and the relative deformation of the heart (i.e. strain) as the surrogate for distance. Using LV pressure as a surrogate for fibre stress does not take into account LV geometry – the radius of curvature of the LV or the wall thickness. In consequence, we cannot calculate work, per se, but a measure of work – named MWI, quantified in the unit mmHg%. These limitations are applied to enable a non-invasive, accessible assessment of MWI based on 2D echocardiography and cuff blood pressure measurements. Nevertheless, since individual differences in LV shape and wall thickness inevitably exist, but cannot be accounted for with this methodology, the calculated absolute MWI values, in general, should not be directly compared between patients[8]..

Insights from myocardial work for the exploration of ventricular remodeling in hypertension

MWI can help distinguish impairment in BSH related to chronic remodelling from impairment associated with acute loading. *Fig. 2* shows a clear example – the strain bullseye plots depict a similar pattern of inferoseptal deformation impairment in acute high blood pressure in both the non-BSH and BSH patient. However, the MWI plots and segmental pressure-strain loops reveal a notably worse underlying impairment present in the BSH patient after correction for acute loading, as compared to that seen in the non-BSH patient. This can also be noterd in the whole cohort. As systolic pressure rises in the non-BSH subgroup, basal septal work increases accordingly, accounting for

the overall added workload imposed to the LV. However, this is not seen in the BSH subgroup, indicating chronic changes and impairment of the thickened myocardial septum, non-related to loading. The lack of a correlation between a rise in myocardial work following a rise in blood pressure shows that the thickened basal septum in BSH has a low capacity to adjust to an increased workload. Findings of mid-wall fibrosis predominantly localized in the basal and mid-ventricular septum in magnetic resonance studies of a hypertensive cohort may suggest a potential underlying cause.[19] Overall, our findings overlap with the hypothesis that BSH is a useful marker in defining a patient subgroup with an advanced impact of hypertension on cardiac function associated with LV remodelling. In a clinical setting, the capacity to account for loading conditions has considerable value, giving opportunity to assess a patient's condition regardless of blood pressure during the time of assessment, thus aiding in clinical decision making on the basis of pressure-accounted function.

MWI also offered insights into early change of LV function in hypertensive heart disease. The increased workload in hypertension is not distributed homogeneously inside the LV. An apex-to-base gradient seen in strain values is preserved in MWI distribution, with the apical region compensating for the impairment of the basal segments (*Fig. 3 and Table 2*). As this functional redistribution is seen in both strain and MWI distribution, it is clear that it is unrelated to the influence of acute loading, but a consequence of chronic changes already present in early stages of hypertensive heart disease. Therefore, the redistribution is accentuated in patients with BSH - a marker of more advanced LV remodelling. As MWI is reflective of oxygen consumption, the observed differences in regional work distribution might be associated with differences in regional blood flow and oxygen demand - differences that in long term exposure might lead to further adverse remodelling of the LV [8]. A recent longitudinal study demonstrated pathological regional distribution of work is related to regional LV remodelling, with the potential of reverse remodelling with therapy [21]. Therefore, we believe that differences in MWI distribution in hypertension may have a potential clinical implication as a signal for the need for a more stringent disease follow-up, and possibly therapy modification. Follow-up data will be crucial for understanding the clinical implications of the findings reported in this study.

Limitations

The study includes a medium size cohort of hypertensive patients clinically managed at the local practice in line with the current guidelines. Included patients reported stable home measurements of blood pressure since the previous visit, however, data on 24-hour blood pressure measurements was not available. Although the cohort is not

representative of the whole hypertensive spectrum, the changes in MWI distribution were quantifiable even in these clinically managed patients. The statistically significant age difference in our study should not result in any considerable differences in strain or MWI values, nevertheless, this could be a potential limitation.

The criteria used for the definition of BSH are not standardized [22]. In this setting, a previously used value of basal septal to mid-septal wall thickness ratio was selected to identify BSH [10]. Reassuringly, the prevalence of BSH in our cohort corresponded to that seen in preceding publications [5,23], and the related regional impairments concur. The linear method of LV mass assessment is flawed in the presence of asymmetric hypertrophy. Additional exploration of this topic can be found in [10]. Localized basal septal hypertrophy can also be seen in hypertrophic cardiomyopathy (HCM) and aortic stenosis. As noted, patients with moderate or severe valvular disease were excluded from analysis. Genetic data was not available in our study; however, we assessed data on family history of cardiac disease and sudden cardiac death finding none of the BSH patients had positive findings. Furthermore, the regional deformation impairment seen in the BSH patients was not similar to that seen in HCM [24]. Moreover, recent investigations of MWI analysis in HCM clearly show considerably lower values of regional MWI than those found in our hypertensive patients[25].

Conclusion

Non-invasive work analysis allows the comparison and exploration of function in hypertensive hearts regardless of blood pressure. An apex-to-base work gradient, unrelated to the effects of acute loading, demonstrates work redistribution in arterial hypertension. Accentuated changes in work redistribution affirm BSH as an echocardiographic marker in hypertensive heart disease.

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Figure Captions

Fig. 1 Group averages of segmental longitudinal strain and the myocardial work index

Bullseye plots of 18-segment left ventricular model showing group averages in segmental longitudinal strain at aortic valve closure (*left*) and averages of the segmental myocardial work index (*right*). An apex-to-base work gradient is visible in the hypertensive groups, as well as an impairment of the basal septal work in the BSH subgroup. The plots were generated using methods made openly available by the authors.

Fig. 2 Comparison of two hypertensive patients - without basal septal hypertrophy (BSH) (*left*) and with BSH (*right*).

Both patients have preserved global longitudinal strain values and elevated systolic blood pressure at the time of acquisition. (A) Bulls eye LV deformation plot showing segmental deformation of the LV. The inferoseptal segment demonstrates an equally reduced deformation in both ventricles (*yellow arrow*). (B) Bulls eye myocardial work index plot and the global (*red*) and segmental inferoseptal (*green*) LV pressure-strain loops. After correction for the acute loading with non-invasive MWI estimation, MWI plots and pressure-strain loops show a significantly reduced work index of the inferoseptum in the BSH patient as compared to the non-BSH patient. (*GLS – global longitudinal strain; HR – heart rate; BP – blood pressure; GWI – global work index)*

Fig. 3 Regional distribution of myocardial work in hypertension

(A) An 18-segment model of the left ventricle showing the apical (*green*), mid (*grey*) and basal (*blue*) regions. The septal region consisting of the antero- and inferoseptum is highlighted (*dark blue*). (B) A scatter plot showing the percentage of total myocardial work performed by the apical, mid and basal regions in the different study groups. An increased apex-to-base gradient can be recognized in patients with arterial hypertension as compared to healthy individuals (*black arrows*).

Fig. 4 Average global and segmental pressure-strain loops of the three subgroups

(A) Average global pressure-strain loops of the three subgroups. The area of the loop corresponds to the myocardial work index, incorporating information on deformation and LV intracavity pressure. A trend in larger loop area can be

seen in the hypertensive subgroups. (B) Average segmental pressure-strain loops of the six segments seen in the 4C view show an apex-to-base work gradient pronounced in the hypertensive subgroups.

Tables

Table 1 General characteristics

	Healthy controls (n=20)	Patients without BSH (n=139)	Patients with BSH (n=31)	Group P value
Age, years	54 (51-56)	57 (52-60)	58 (55-62)*	**0.004**
Male gender, n (%)	8 (44)	72 (52)	20 (65)	0.337
Duration of hypertension, years	-	8 (4-14)	8 (6-16)	0.213
BMI, kg/m^2	24.8±2.9	27.8±4.5*	28.2±3.9*	**0.017**
Diabetes mellitus, n (%)	0 (0)	14 (10)	5 (16)	0.218
Dyslipidaemia, n (%)	0 (0)	742 (52)	19 (61)	0.427
Body surface area, m^2	1.92±0.19	1.91±0.23	1.99±0.20	0.230
Systolic blood pressure, mmHg	120 (115-130)	135 (126-145)*	138 (130-155)*	**<0.001**
Diastolic blood pressure, mmHg	80 (74-85)	80 (70-86)	84 (76-89)	0.249
Heart frequency, beats per minute	63±12	68±11	68±11	0.220
Beta-blockers, n (%)	0 (0)	29 (21)	7 (23)	0.999
ACE-inhibitors, n (%)	0 (0)	49 (36)	13 (42)	0.541

Aldosterone receptor blockers, n (%)	0 (0)	61 (45)	15 (48)	0.842
Ca-channel blockers, n (%)	0 (0)	35 (26)	9 (29)	0.821
Diuretics, n (%)	0 (0)	45 (32)	12 (39)	0.656
Statins, n (%)	0 (0)	31 (23)	12 (39)	0.072

* - *P<0.05 versus healthy controls*

Table 2 LV dimensions and function

	Healthy controls (n=20)	Patients without BSH (n=139)	Patients with BSH (n=31)	Group P value
LV ejection fraction, %	58 (54-60)	55 (53-59)	62 (58-65)*†	**<0.001**
LV end-diastolic volume, ml	105 (88-115)	108 (94-129)	91 (80-104)†	**<0.001**
LV end-systolic volume, ml	45 (37-52)	47 (40-59)	35 (30-42))*†	**<0.001**
LV end-diastolic posterior wall thickness, cm	0.8 (0.8-0.9)	1.1 (1.0-1.2)*	1.1 (1.0-1.2)*	**<0.001**
LV end-diastolic diameter, cm	4.7 (4.4-5.1)	4.2 (3.9-4.5)*	4.2 (4.0-4.4)*	**0.001**
Relative wall thickness	0.37 (0.34-0.39)	0.50 (0.43-0.58)*	0.51 (0.46-0.56)*	**<0.001**
LV mass indexed to BSA, g/m^2	68 (54-81)	76 (66-89)	93 (81-100)*†	**<0.001**
E/A ratio	1.3 (1.0-1.5)	1.0 (0.8-1.2)*	0.9 (0.7-1.1)*†	**<0.001**
Global MWI, mmHg%	2098±373	2345±398*	2297±427	**0.038**
Relative apical MWI	0.60±0.09	0.68±0.97*	0.72±0.12*	**<0.001**
LV Global longitudinal strain, %	-21.05±2.33	-21.19±2.29	-20.42±2.44	0.253
Basal anteroseptum strain, %	-17.25±2.61	-15.15±3.47*	-13.89±4.27*	**0.005**

Basal inferoseptum strain, %	-16.69±2.47	-15.02±2.60*	-12.20±2.73*†	<**0.001**
Average strain of basal segments, %	-18.33±1.92	-17.22±2.09	-15.64±2.13*†	<**0.001**
Average strain of mid segments, %	-20.67±2.53	-20.29±2.26	-19.43±2.33	0.110
Average strain of apical segments, %	-24.14±3.72	-26.06±3.75	-26.19±3.9	0.096

** - P<0.05 versus healthy controls*

† - P<0.05 versus HTN patients without BSH

Supplementary data

Additional Figures

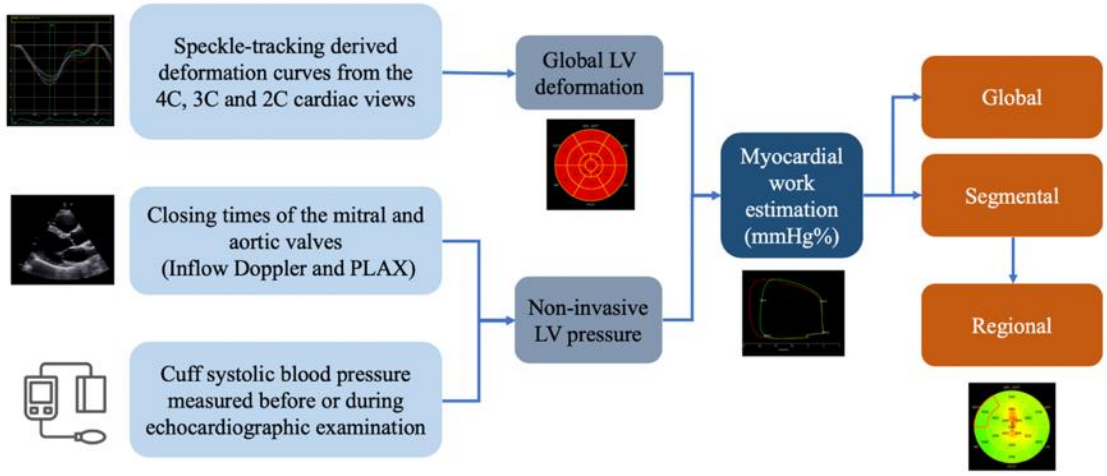
Figure legends

Supplementary Figure 1 A schematic presentation of myocardial work analysis.

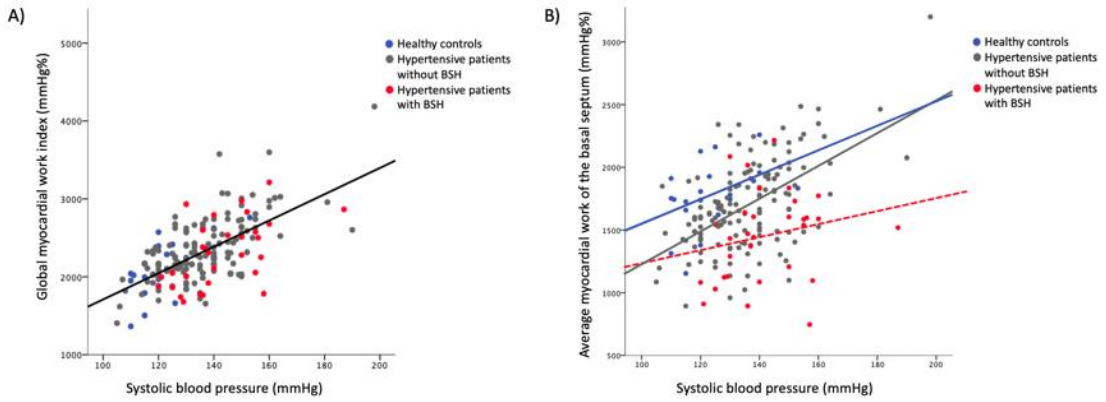
Supplementary Figure 2 (A) The linear relationship of global myocardial work index and the systolic blood pressure (Pearson R coefficient 0.644, p<0.001). (B) The linear relationship of the average myocardial work of the basal septum and systolic blood pressure is present in the healthy controls (Pearson R 0.429, p=0.059) and non-BSH hypertensive patients (Pearson R 0.548, p<0.001), however there is no relationship in the BSH subgroup (Pearson R coefficient 0.213, p=0.249).

Figures

Supplementary Figure 1



Supplementary Figure 2



Tables

Supplementary Table 1 – Linear relationship of the average myocardial work of the basal septum and systolic blood pressure

	Pearson R coefficient	P value
Healthy controls (n=20)	0.429	0.059
Hypertensive patients without BSH (n=139)	0.548	<0.001
Hypertensive patients with BSH (n=31)	0.213	0.249
BSH – basal septal hypertrophy		