Assessment of Left Ventricular Function and Contractile Reserve in Patients with Hypertension

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ABSTRACT

Background: Hypertension is a prevalent and well-recognized cardiovascular risk factor, which may lead to left ventricular (LV) systolic impairment through chronic pressure overload.
Aim of the work: An impaired contractile reserve (CR) may be an early manifestation of left ventricular (LV) systolic dysfunction in hypertensive patients. Using normotensive patients as controls, we examined LV CR and its correlates in hypertensive patients. **Material and methods:** The study was conducted prospectively on fifty cases. 30 cases (24 males and 6 females aged 55.57 ± 10.07) had hypertension. All patients were subjected to complete history taking including comorbidities, risk factors and full clinical examination. Patients with significant coronary or valvular disease, previous myocardial infarction or revascularization and diabetes were excluded. LV ejection fraction (LVEF) & global longitudinal strain (GLS) were measured at rest and at low-dose dobutamine.
Results: Hypertensive patients, compared to control had significantly impaired GLS at rest (-19.00 ± 2.34% vs. -20.50 ± 1.52%, P <0.015) and at low dobutamine dose (-19.90 ± 2.33% vs. -22.60 ± 1.88 %, P <0.001). Absolute and relative GLS CR were significantly lower in hypertensive patients (-0.88 ± 0.45% vs.-2.12 ± 0.77% and 4.70 ± 2.76% vs. 10.33 ± 3.78 %, respectively, both P < 0.001). Absolute and relative LVEF CR were significantly lower in hypertensive patients (3.31 ± 0.97 % vs. 7.09 ± 1.67% and 5.37 ± 1.64% vs. 11.61 ± 3.19%, respectively, both P < 0.001).
Conclusion: Compared to control, hypertensive patients with no other co-morbidities had impaired LV GLS at rest and impaired CR despite normal LVEF.
Keywords: Hypertension, Ventricular function, Contractile reserve, Echocardiography, Speckle tracking strain.

INTRODUCTION

Hypertension is a prevalent and well-recognized cardiovascular risk, which may lead to left ventricular (LV) systolic impairment through chronic pressure overload. LV hypertrophy (LVH) is a compensatory process in response to increase wall stress. However, this initially useful adaptive mechanism later becomes 'a pathological change' in the myocardium. Whilst, LVH has been shown to be a powerful independent predictor for cardiovascular morbidity and mortality [1].

Multiple recent studies have shown that LV ejection fraction (LVEF) lacks accuracy and sensitivity in detecting early subclinical impairment. In contrast, LV strain analysis has been proven to be more sensitive in the detection of early subclinical LV systolic dysfunction, when the LVEF is still within 'normal' limits [2].

LV systolic dysfunction in the early stage can be subtle and sub-clinical where resting systolic function may remain preserved, but contractile reserve (CR) to inotropic stimulation may be blunted. A depressed CR has been documented in patients with aortic stenosis [3], mitral regurge [4], aortic regurge [5] and in the diabetic cardiomyopathy [6] and is thought to represent an early manifestation of LV dysfunction. Similarly, impaired CR may be an early manifestation of LV systolic impairment in hypertensive patients.

AIM OF THE WORK

The aim of this study was to examine CR and its correlates in hypertensive patients and to examine the relationship between impaired CR and resting LV systolic function as assessed by LV deformation. A group of normotensive patients was recruited as controls.

PATIENT AND METHODS

This was a prospective observational study that was conducted from July 2018 to June 2019 and included 50 cases who presented to stress echocardiography test. 30 patients with hypertension as study group and twenty normotensive as control group. Complete history was taken (personal, past, present & family) and complete physical examination (SBP, DBP, HR, RR, Temp & BMI).

Exclusion criteria:
- Diabetes mellitus and moderate to severe left sided valvular diseases.
- Conduction abnormalities as well as pacemaker and ongoing arrhythmias.
- Congenital heart disease, poor echocardiographic window, previous myocardial infarction or coronary revascularisation.

The study protocol was approved by Al-Azhar University, Faculty of Medicine. A chart review was performed, and data were collected including patient demographics, medical history and physical examination.

Ethical consideration and written informed consent:
An approval of the study was obtained from Al-Azhar University Academic and Ethical

Committee. Every patient signed an informed written consent for participation in the study.

Echocardiographic data were collected as follow:

A) Resting conventional 2D echocardiography included (End-diastolic and end-systolic dimensions of the LV/ End-diastolic thickness of inter-ventricular septum and LV posterior wall /Ejection fraction).

B) Low dose dobutamine echocardiography was performed to all participants. Patients who were on B-blockers or non-dihydropyridine calcium blockers had their low-dose echocardiography assessment performed at 20 micg/kg/min. For the remaining patients, the low-dose assessment of them was performed at 10 micg/kg/min. These doses were chosen as they have been previously demonstrated to be safe and effective in detecting CR, without affecting heart rate, blood pressure (BP) or loading conditions [7].

C) 2D strain imaging by speckle tracking for deformation analysis: Endocardial borders was traced at the end-diastolic frame in apical four chamber view (AP4). End diastole will be defined by the QRS complex or as the frame after mitral valve closure. The software tracks speckles along the endocardial border throughout the cardiac cycle and peak global longitudinal strain (GLS) were measured automatically generating regional data from 6 segments and three cardiac cycles and were averaged. Normal resting peak global longitudinal strain (GLS) was defined as less than -20% [8].

D) Assessment of Left Ventricular Contractile Reserve (LV CR): LVEF and strain analysis were performed both at rest and at low-dose dobutamine. CR was measured as the following: (1) Absolute CR was calculated as the difference in LVEF and GLS between the low-dose and their corresponding resting values. (2) Relative CR was defined as the ratio of absolute CR to the corresponding resting values and was expressed as percentage. Impaired CR was defined as an absolute CR of < 5% as measured by LVEF [9].

Statistical methods

Recorded data were analyzed using the statistical package for social sciences, version 20.0 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were expressed as mean ± standard deviation (SD). Qualitative data were expressed as frequency and percentage.

The following tests were done:

- Independent-samples t-test of significance was used when comparing between two means.
- Chi-square (x^2) test of significance was used in order to compare proportions between qualitative parameters.
- The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the p-value was considered significant as the following: Probability (P-value)
  - P-value <0.05 was considered significant.
  - P-value <0.001 was considered as highly significant.
  - P-value >0.05 was considered insignificant.

RESULTS

All patients were subjected to history taking and full clinical examination, resting 2D conventional echocardiography, low dose dobutamine echocardiography and 2D strain imaging by speckle tracking. All data were collected and tabled and statistically analyzed. The mean age was 55.57 ± 10.07 years and its p-value was 0.456 (ranged from 30-75 years). Males represented 24 patients (48 %) of the study population and the male to female ratio was (0.92: 1)

Table (1): Comparison between study group and control group concerning demographic data

	Demographic data	Study group	Control group	t/x2#	p-value
Age (years)	Mean±SD Range	55.57±10.07 36-77	53.20±12.06 35-82	0.566	0.456
Sex	Female Male	16 (53.3%) 14 (46.7%)	10 (50.0%) 10 (50.0%)	0.053#	0.817

t-Independent Sample t-test; #x^2: Chi-square test p-value > 0.05 NS

Table (2): Comparison between study group and control group regarding risk factors

	Risk factors	Study group	Control group	X2	p-value
Special Habits	Absent	24 (80.0%)	14 (70.0%)	0.658	0.417
	Smoke	6 (20.0%)	6 (30.0%)		
Past History	HTN	30 (100.0%)	0 (0.0%)	50.000	<0.001**
	Not HTN	0 (0.0%)	20 (100.0%)		
Family history	Negative	13 (43.3%)	20 (100.0%)	17.172	<0.001**
	Positive	17 (56.7%)	0 (0.0%)		

*t-Independent Sample t-test; #x^2: Chi-square test , p-value>0.05 NS; **p-value <0.001 HS.* This table showed statistically significant difference between groups according to past history and family history and risk factors.

Table (3): Comparison between study group and control group as regards examinations

Examinations	Study group	Control group	t-test	p-value
SBP **Mean ± SD** **Range**	**132.50 ± 6.12** **120-150**	**120.50 ± 7.76** **110-135**	**37.153**	**<0.001****
DBP **Mean ± SD** **Range**	**85.33 ± 5.24** **80-100**	**79.00 ± 5.76** **70-90**	**16.194**	**<0.001****
HR **Mean ± SD** **Range**	**83.40 ± 11.55** **68-115**	**76.65 ± 9.84** **56-102**	**4.598**	**0.037***
RR **Mean ± SD** **Range**	**16.90 ± 1.03** **15-19**	**16.60 ± 1.05** **15-19**	**1.007**	**0.321**
Temp. **Mean ± SD** **Range**	**38.56 ± 8.59** **35.5-84**	**36.86 ± 0.23** **36.5-37.2**	**0.781**	**0.381**
BMI [wt/(ht)^2] **Mean ± SD** **Range**	**26.50 ± 3.47** **22.5-34.8**	**25.70±2.92** **22.5-32.5**	**0.719**	**0.401**

t-Independent Sample t-test
p-value>0.05 NS; *p-value <0.05 S; **p-value <0.001 HS

This table showed statistically significant difference between groups according to SBP, DBP, HR, RR, Temp and BMI.

Table (4): Comparison between study group and control group according to IVSD, PWD, LVIDd, LVIDs, EF % and GLS% at resting echocardiography

Resting Echocardiography	Study group	Control group	t-test	p-value
IVSD Mean ± SD Range	1.18 ± 0.14 0.9-1.5	0.90 ± 0.17 0.6-1.2	41.007	<0.001**
PWD Mean ± SD Range	1.12 ± 0.16 0.78-1.4	0.89 ± 0.15 0.7-1.23	27.331	<0.001**
LVIDd Mean ± SD Range	4.66 ± 0.55 3.58-5.81	4.83 ± 0.46 4.02-5.6	1.188	0.281
LVIDs Mean ± SD Range	3.04 ± 0.49 1.99-4.15	3.13 ± 0.40 2.61-3.88	0.567	0.455
EF % Mean ± SD Range	61.34 ± 4.57 52.8-70.5	61.44 ± 4.38 53.5-70	0.006	0.937
GLS% Mean ± SD Range	-19.00 ± 2.34 -22.8_-14	-20.50 ± 1.52 -25.2_-18.8	6.397	0.015*

t-Independent Sample t-test;p-value>0.05 NS; *p-value <0.05 S; **p-value <0.001 HS

This table showed statistically significant difference between groups according to LVSD, PWD and GLS%.

Table (5): Comparison between study group and control group regarding EF% and GLS% at low dose dobutamine echocardiography

Low Dose Echo	Study group	Control group	t-test	p-value
EF % :				
Mean ± SD	64.35 ± 4.93	67.19 ± 4.67	6.398	0.053
Range	55.2-74.5	58-77.2		
GLS % :				
Mean ± SD	-19.90 ± 2.33	-22.60 ± 1.88	18.508	<0.001**
Range	-23.4_-15.1	-28.5_-20.2		

t-Independent Sample t-test
*p-value>0.05 NS; *p-value <0.05 S; **p-value <0.001 HS*
This table showed statistically significant difference between groups as regards GLS% at low dose echocardiography.

Table (6): Comparison between study group and control group according to absolute and relative EF% and GLS at Left ventricular contractile reserve.

LV CR	Study group	Control group	t-test	p-value
Absolute of EF% Mean ± SD Range	3.31 ± 0.97 1_5	6.79 ± 1.67 4.5_11.2	85.563	<0.001**
Relative of EF (%) Mean ± SD Range	5.37 ± 1.64 1.64_8.6	10.61 ± 3.19 8_19.4	69.414	<0.001**
Absolute of GLS% Mean ± SD Range	-0.88 ± 0.45 -2_-0.2	-2.12 ± 0.77 -4.1_-0.8	52.299	<0.001**
Relative of GLS (%) Mean ± SD Range	4.70 ± 2.76 0.96_14.2	10.33 ± 3.78 4_21.2	37.043	<0.001**

*t-Independent Sample t-test; **p-value <0.001 HS*
This table showed highly statistically significant increased mean of control group compared to study group concerning absolute of EF, relative of EF (%), absolute of GLS and relative of GLS (%) of Left ventricular contractile reserve.

Table (7): Comparison between baseline and at low dose echocardiography regarding EF% in each group

EF%	Study group	Control group
Baseline	61.34 ± 4.57	61.44 ± 4.38
Low Dose Echo	64.35 ± 4.93	67.19 ± 4.67
Paired Sample t-test	4.30	8.288
p-value	0.021*	<0.001**

*t-Paired Sample t-test; **p-value <0.001 HS*
This table showed statistically significant increased mean of at low dose echocardiography compared to baseline according to EF% in each group.

Table (8): Comparison between baseline and at low dose echo regarding GLS% in each group

GLS%	Study group	Control group
Baseline	-19.00 ± 2.34	-20.50 ± 1.52
Low Dose Echo	-19.90 ± 2.33	-22.60 ± 1.88
Paired Sample t-test	5.58	11.492
p-value	0.008*	<0.001**

*t-Paired Sample t-test; **p-value <0.001 HS*
This table showed statistically significant increased mean of low dose echo compared to baseline according to GLS% in each group.

DISCUSSION

In patients with treated hypertension without DM or significant ischemic heart disease, despite having normal LVEF at rest, subclinical LV systolic dysfunction was evident when assessed by GLS compared to normotensive patients. Furthermore, hypertensive patients had an impaired ability to augment LV contractility in response to inotropic stimulation by dobutamine, as evident by the impaired GLS at low-dose dobutamine and a reduced CR. GLS at rest and with low-dose dobutamine, and CR were correlated with LV wall thickness. In addition, GLS at rest and with low-dose dobutamine were correlated with prevailing systolic BP.

The aim of this study was to detect the impaired contractile reserve (CR) as an early manifestation of left ventricular (LV) systolic dysfunction in hypertensive patients comparing them with normotensive patients as control group by using low-dose dobutamine stress echocardiography & 2D speckle tracking echo (2D-STE).

Mean ± SD age between hypertensive group and normotensive group was 55.57 ± 10.07 vs. 53.20 ± 12.06 years, respectively and its p-value was 0.456) ranged from 30-75 years. This is nearly concordant with **Matle et al.** [10] who discussed the role of speckle tracking echocardiography in assessing LV CR in hypertensive patients. They studied 129 cases with mean age of 60.0 ± 8.7 vs. 56.7 ± 10.2 years, respectively and its p-value was 0.051. Also, concordant with **Narayanan et al.** [11] who discussed cardiac mechanics in mild hypertensive heart disease with Speckle-Strain Imaging Study. The mean age was 53 ± 12 vs. 49 ± 13, and its p-value was NS.

Males represented 24 patients (48 % of the study population) and the male to female ratio was (0.92: 1) and its p-value was 0.817 which is nearly concordant with **Matle et al.** [10] who represented 68 males (52 % of the study population) and the male to female ration (1.11: 1), whereas **Narayanan et al.** [11] represented 73 males (70 % of study population) and the male to female ratio was (2.3 : 1).

➤ *Our study concluded presence of statistically significant difference between hypertensive and normotensive groups in the following predictor variables :*

Our study revealed significant difference between hypertensive and normotensive groups in blood pressure. We revealed SBP of 132.50 ± 6.12 vs. 120.50 ± 7.76, respectively and its p-value was < 0.001, which is nearly concordant with **Matle et al.** [10] who revealed 143.9 ± 16.3 vs. 129.1 ± 11.6, respectively and its p-value was < 0.001. We revealed DBP of 85.33 ± 5.24 vs. 79.00 ± 5.76, respectively and its p-value was <0.001, which is nearly concordant with **Matle et al.** [10] who revealed 84.0 ±

12.7 vs. 80.1 ± 8.6 and its p-value was 0.034 and nearly concordant with **Narayanan et al.** [11] who revealed SBP of 137 ± 19 vs.118 ± 13, respectively and its p-value was <0.001 and revealed DBP of 82 ± 10 vs. 73 ± 8, respectively and its p-value was <0.001.

Our study revealed significant difference between hypertensive and normotensive groups in heart rate. We revealed 83.40 ± 11.55 vs. 76.65 ± 9.84, respectively and its p-value was 0.037, which is discordant with **Matle et al.** [10] who revealed 65.3 ± 10.5 vs. 64.2 ± 9.0 and its p-value was 0.525.

Our study revealed significant difference between hypertensive and normotensive groups in Inter-ventricular septum dimension (IVSd). We revealed 1.18 ± 0.14 vs. 0.90 ± 0.17, respectively and its p-value was <0.001, which is nearly concordant with **Matle et al.** [10] who revealed 1.1 ± 0.2 vs. 1.0 ± 0.2 and its p-value was 0.003 and nearly concordant with **Narayanan et al.** [11] who revealed 1.1 ± 0.2 vs. 0.8 ± 0.1, respectively and its p-value was < 0.001.

In our study, there was significantly significant difference between hypertensive and normotensive groups in posterior wall thickness (PWTd). We revealed 1.12 ± 0.16 vs. 0.89 ± 0.15 respectively and its p-value was <0.001, which is nearly concordant with **Matle et al.** [10] who revealed 1.1 ± 0.1 vs. 1.0 ± 0.2, respectively and its p-value was 0.001 and nearly concordant with **Narayanan et al.** [11] who revealed 1.1 ± 0.2 vs. 0.8 ± 0.1 respectively and its p-value was < 0.001.

In our study, there was significantly difference in LV GLS between hypertensive and normotensive groups at rest. We revealed -19.00 ± 2.34% vs. -20.50 ± 1.52% respectively and its p-value was 0.015, which is nearly concordant with **Matle et al.** [10] who revealed -17.1 ± 1.8% vs. -19.4 ± 1.5% respectively and its p-value was < 0.001. Also nearly concordant with **Imbalzano et al.** [12] who revealed significantly difference in LV GLS between hypertensive patients with normal geometry (No LVH) and normotensive groups (−18 ± 1.9% vs. −20.4 ± 2.5%, respectively and its p-value was 0.02). And significant difference in LV GLS between hypertensive patients with abnormal geometry (LVH) and normotensive groups (−15.9 ± 3.3% vs. −20.4 ± 2.5%. respectively and its p-value was <0.001). Moreover, it is concordant with.

Kouzu et al. [13] who found significantly impaired longitudinal strain in the hypertensive group, (concentric, −15.1 ± 4.0%, eccentric, −15.9 ± 4.4% vs. control −18.9 ± 3.3%, its P value < 0.05) with LVH being the only independent

In our study, there was significant difference in LV GLS between hypertensive and normotensive groups at low dose dobutamine infusion. We revealed -19.90 ± 2.33% vs. -22.60 ± 1.88% respectively and its p-value was < 0.001, which is nearly concordant

with **Matle et al.** [10] who revealed -18.1 ± 2.3% vs. -22.6 ± 2.4%, respectively and its p-value was < 0.001. Moreover, numerous studies have found LVEF to be insensitive in detecting subclinical myocardial dysfunction in a variety of cardiovascular conditions, and only detects abnormalities in an advanced stage of hypertensive heart disease. In contrast, **Drazner** [14] discussed the progression of hypertensive heart disease and proved that strain analysis to be more sensitive and accurate in detecting subclinical myocardial dysfunction as mentioned above.

In our study, There was significantly impaired absolute GLS CR between hypertensive compared to normotensive group. We revealed -0.88 ± 0.45% vs. -2.12 ± 0.77%, respectively and its p-value was < 0.001, which is nearly concordant with **Matle et al.** [10] who revealed -1.0 ± 2.8% vs. -3.2 ± 2.2%, respectively and its p-value was < 0.001.

In our study, there was significantly impaired relative GLS CR between hypertensive compared to normotensive group. We revealed 4.70 ± 2.76% vs. 10.33 ± 3.78%, respectively and its p-value was <0.001, which is nearly concordant with **Matle et al.** [10] who revealed 5.0 ± 5.6% vs. 16.4 ± 11.7%, respectively and its p-value was < 0.001. Besides, **Badran et al.** [15] found significantly impaired reserve functional with exercise, in hypertensive patients compared to controls (10 ±16% vs. 17±6%, respectively and its p-value was < 0.001).

Our study demonstrated that patients with hypertension had impaired CR when assessed by GLS but not by LVEF.

Tan et al. [16] found significantly lower GLS in hypertensive patients both at rest and on exercise compared to healthy controls. the patients had reduced systolic longitudinal function (reserve index 0.97 ± 1.34 vs. 2.32 ± 1.24, respectively and its p-value was 0.001). However, the hypertensive group in their study had a significantly higher body mass index compared to controls, and a significant percentage of patients (20%) had a history of coronary artery disease and diabetes.

These co-morbidities and obesity have all been found to independently affect GLS and their results may therefore be confounded by these co-morbidities in the hypertensive group. In our study, both coronary artery disease and diabetes were excluded, our results would be more reflective of the true impact of hypertension on LV systolic deformation. Previous studies demonstrating impaired GLS in patients with hypertension used healthy subjects as controls. We used normotensive patients with comparable cardiovascular risk factors. Furthermore, we excluded patients with diabetes and significant coronary artery disease, and therefore were able to examine GLS in a more homogenous patient population, where the only major factor adversely affecting LV deformation was hypertension.

> *Our study concluded no statistically significant difference between hypertensive and normotensive groups in the following predictor variables :*

No significant correlation between prevailing resting systolic or diastolic BP and LVEF and LV GLS, and there were no correlations between prevailing BP and CR measured by LVEF and LV GLS, which is nearly concordant with **Matle et al.** [10].

Our study revealed no significant difference between hypertensive and normotensive groups in smoking [6 (20.0%) vs.6 (30.0%) respectively and its p-value was 0.417], which is nearly concordant with **Matle et al.** [10].

Our study revealed no significant difference between hypertensive and normotensive groups in Body Mass Index (BMI), which is nearly concordant with **Matle et al.** [10] who revealed 29.0 ± 4.4 vs. 27.8 ± 4.4, respectively and its p-value was 0.128.

Our study revealed no significant difference between hypertensive and normotensive groups in Left venticular internal dimensions (LVIDd) (sysolic & diastolic) that was 4.66 ± 0.55 vs.4.83 ± 0.46 respectively and its p-value was 0.281 and LVIDs that was 3.04 ± 0.49 vs.3.13 ± 0.40 respectively and its p-value was 0.455, which is nearly concordant with **Matle et al.** [10] who revealed LVIDd that was 4.6 ± 0.5 vs. 4.6 ± 0.4 respectively and its p-value was 0.9 and LVIDs that was 2.9 ± 0.6 vs. 2.6 ± 0.4, respectively and its p-value was 0.119.

In our study, there was no significant difference in LVEF between hypertensive and normotensive groups at rest. We revealed 61.34 ± 4.57% vs. 61.44 ± 4.38%, respectively and its p-value was 0.937, which is nearly concordant with **Matle et al.** [10] who revealed 64.5 ± 6.0% vs. 64.5 b ± 6.6% respectively and its p-value was 0.986, which is nearly concordant with **Narayanan et al.** [11] who revealed 68 ± 6% vs. 66 ± 7%, respectively and its p-value was NS.

In our study, there was no significant difference in LVEF between hypertensive and normotensive groups at low dose dobutamine infusion. We revealed 64.35 ± 4.93% vs. 67.19 ± 4.67%, respectively and its p-value was 0.053, which is nearly concordant with **Matle et al.** [10] who revealed 72.1 ± 6.5% vs. 71.2 ± 7.0 %, respectively and its p-value was 0.424.

LIMITATIONS

A Significant limitation is the relatively small sample size in our study. As the absence of ischemic heart diseases was a strict exclusion criterion. As diabetes mellitus and ischemic diseases are highly prevalent diseases among people, so a large number of screened cases had to be excluded. We believed that inclusion of these diseases would confound our results as these conditions have been shown to cause left ventricular systolic dysfunction. Nonetheless, compared to most of other published studies to assess left ventricular contractile reserve, our study had a

strict exclusion criteria to avoid any expected potential confounders.

In our study, we only used the prevailing blood pressure, which was measured just prior to the commencement of the dobutamine stress echocardiography. This blood pressure reading may not reflect overall blood pressure control for the patient. Using blood pressure measurements of 24 hours ambulatory monitoring might be more reflective of the overall average blood pressure, but this was beyond the scope of our study.

In our study, B-blocker treatment may have negative inotropic effect and chronotropic effect on the left ventricle and its response to dobutamine. So we performed the low-dose dobutamine stress test at 20 mic/kg/min for patients on such treatment, as compared to 10 mic/kg/min for cases who were not.

CONCLUSION

In patients with hypertension with no other co-morbidities like diabetes mellitus or ischemic heart disease, with normal resting left ventricular EF not only have impaired left ventricular GLS at rest but also lack contractile reserve with low-dose dobutamine. The impaired contractile reserve was correlated to left ventricular wall thickness independent of prevailing blood pressures. Assessment of contractile reserve by GLS may have incremental value in early diagnosis and subclinical left ventricular systolic dysfunction in patients with hypertension.

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