Is it only diastolic dysfunction? Segmental relaxation patterns and longitudinal systolic deformation in systemic hypertension

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Aims To investigate changes in longitudinal systolic function estimated by strain echocardiography in relation to global diastolic dysfunction and alterations in segmental relaxation patterns.

Methods and results We studied 75 hypertensive patients, of whom 45 had diastolic dysfunction and normal EF, and 30 matched controls. All subjects had 2D and colour Doppler myocardial imaging. Mean longitudinal strain (S) and strain rate (SR) were averaged from the basal and mid-LV segments assessed in the longitudinal axis. Early to late diastolic SR ratio <1.1 was defined as altered segmental relaxation [segmental diastolic dysfunction (DD)]. The total number of segmental DD out of the 18 basal-mid-apical segments was calculated for all the participants. Longitudinal systolic function estimated by mean strain and SR was decreased in the hypertensive group, but was further deteriorated in the diastolic dysfunction group compared with controls. Altered Segmental Relaxation was highly correlated with longitudinal systolic dysfunction expressed by strain (r: 0.56) or SR (r: 0.57). A septal mitral annular Ea cut-off of 5.9 cm/s predicted longitudinal systolic dysfunction with a sensitivity of 81% and a specificity of 70%. A multiple linear regression model proved LVMI, systolic blood pressure (SBP) and age as independent predictors of diastolic and longitudinal systolic dysfunction and BMI to independently related to diastolic dysfunction.

Conclusion Longitudinal systolic dysfunction may be present in hypertensive patients with diastolic dysfunction, especially when septal Ea < 5.9 cm/s. Altered segmental relaxation pattern is highly correlated with longitudinal systolic dysfunction. LV hypertrophy, SBP and aging are important determinants of both diastolic and longitudinal systolic dysfunction, whereas obesity appears to contribute to the appearance of diastolic dysfunction.

KEYWORDS
Strain; Diastolic dysfunction; Hypertension

Introduction

Recent epidemiological data have strengthened proof of the well-recognized relationship between blood pressure (BP) and cardiovascular disease, and have confirmed the overwhelming importance of systolic BP (SBP) as a determinant of risk.1

Hypertension constitutes one of the most common causes of diastolic dysfunction and is a major contributor to the pathogenesis of a large proportion of heart failure cases in a population-based sample.2,3 Diastolic dysfunction is commonly associated with left ventricular hypertrophy (LVH), a common feature of systemic hypertension, which is characterized by a disproportionate involvement of non-myocytic elements in the myocardium.4

Previous studies have found that longitudinal systolic dysfunction may be present in hypertensive patients with diastolic dysfunction or diastolic heart failure.5,6

Novel deformation parameters have the potential to quantify systolic and diastolic myocardial function with high spatial and temporal resolution, irrespective of adjacent segments’ tethering or translation effects.7,8 Altered segmental relaxation based on strain echocardiography is regarded a better method to evaluate changes of diastolic function in hypertension, since it is evident before global indices of diastolic dysfunction become informative.9

The aim of the present study is to investigate changes in longitudinal systolic function estimated by strain echocardiography in relation to global diastolic dysfunction and...
alterations in segmental relaxation patterns in hypertensive patients.

Methods

Study subjects

The study population consisted of 75 patients with essential hypertension, recruited from the hypertension outpatient clinic and echocardiography department, and 30 aged matched healthy volunteers for control. Patients were excluded if they were not in sinus rhythm, or if they had a history of coronary artery disease, regional wall motion abnormalities, mitral or aortic stenosis, congenital disease, cardiomyopathy, mitral or aortic regurgitation, pericardial disease, or cor pulmonale. From the hypertensive group (HTN-ALL), a subgroup of patients had diastolic dysfunction, (n: 45) symptomatic or asymptomatic (HTN-DD).

Diagnosis of global diastolic dysfunction was based on known conventional echocardiographic criteria [using E/A, deceleration time (DT), IVRT (isovolumic relaxation time), pulmonary veins S, D, and A flow velocities], along with newer tissue Doppler parameters (Ea, and E/Ea), validated against tau.10-13 All patients, originally had >Grade 1 essential hypertension (systolic >140 mmHg and/or diastolic <90 mmHg) according to their medical records, with a history of hypertension for more than 1 year, currently under medical treatment. An informed consent was given by all the participants. The study was approved by the Local Research Ethics Committee.

Echocardiography

All subjects were studied by standard two-dimensional and Doppler echocardiography. We used a Toshiba Aplio model SSA-770A, with a phased array transducer (2.8–4.5 MHz). Parasternal and apical projections were obtained according to the recommendations of the American Society of Echocardiography (ASE).14 LV ejection fraction was derived from Simpson's modified biplane method.14 LV mass was estimated with the area-length formula as described in detail in the ASE document on LV quantification.15 LV mass index then was calculated by the formula: LVmass/BSA, where BSA=body surface area. Relative wall thickness (RWT) was estimated according to the formula: RWT = 2 x posterior wall thickness/LVEDD, where LVEDD = left ventricular end-diastolic diameter.

Pulsed-wave Doppler of transmitral LV inflow was performed in the apical four-chamber view, with the sample volume placed at the level of mitral valve tips. The following measurements of global LV diastolic function were determined: peak velocities of E and A wave and their ratio E/A, deceleration time of E wave (DT) and LV IVRT.

Pulsed wave tissue Doppler recordings from the septal and lateral site were also recorded from the apical four-chamber view. The pulsed sample volume of 5 mm was placed over the mitral annulus and the average of three consecutive cardiac cycles of peak diastolic velocities during early filling (Ea) was measured. Mean Ea was estimated averaging the septal and lateral values. LV filling pressures were estimated by calculating the E/ Ea mean ratio.

Longitudinal (Meridional) end-systolic wall stress (LWS) was calculated according to the formula:16 LWS = BP x R/2WT(1 + WT/R), where R = end-systolic radius, WT = wall thickness, and BP = systolic blood pressure.

Pulse pressure (PP) was estimated by the formula: PP = SBP – DBP. Mean arterial pressure (MAP) was estimated by the formula: MAP = DBP + 1/3 (SBP – DBP).

Tissue Doppler imaging (TDI) samples acquisition for strain–strain rate analysis

Tissue Doppler images of cineloops of three cardiac cycles from the lateral, septal, anterior, inferior anteroseptal and posterior wall, from the apical long-axis view, were acquired separately at end-expiratory apnoea and stored digitally. To optimize the tissue velocity signals, the two-dimensional image was optimized, to obtain a clear differentiation between the myocardium and the blood pool. We used the narrowest image sector angle possible (30°), to achieve maximum colour Doppler frame rate, typically >150 frames/s. The recorded wall was positioned in the centre of the sector, so that the direction of motion interrogated was as near as possible parallel to the direction of the insonating beam, giving an insonation angle <15° in all the recordings.

The insonation frequency was set at 2.8 MHz. Filter harmonic imaging was set as a standard for all the recordings. The pulse repetition frequency (PRF) was adjusted accordingly, avoiding aliasing, typically ~4.2 kHz.

Strain–strain rate off-line analysis

Analysis of strain and strain rate (SR) parameters was performed off line, using the incorporated USTQ-770A program of Toshiba Aplio System.

A sample volume ROI (region of interest) was placed within the area of interest, so that there was no migration beyond the limits of the selected myocardium. The automatic ROI tracking mode was activated in order to ensure that measurements reflected motion of a myocardial tissue segment throughout the cardiac cycle. In this study, we used an oval size of 9 mm x 6 mm for longitudinal measurements. Early and late diastolic SR parameters SRE and SRE of basal, mid, and apical segments were recorded from each LV wall in the longitudinal direction, in a 18-segment model. SRE/SRE < 1.1 was regarded as an index of altered segmental relaxation – segmental diastolic dysfunction (DD)9 (Figure 1). The total number of segmental DD was calculated for all the participants.

The systolic SR parameters of basal and the mid-segments were analysed from each LV wall in the longitudinal direction. The myocardial systolic strain parameters were calculated by integrating the SR profiles over time and compensating for drifting over the cardiac cycle. The average of values of three consecutive cycles were calculated for each parameter. Mean longitudinal S and SR were estimated averaging the values of each of the 12 segments assessed.

Statistical analysis

Data were analysed using SPSS 12 software (SPSS, Chicago, IL, USA). Continuous variables, expressed as mean ± SD, were compared using Student's t-test for independent groups. When the assumptions to use the Student's t-test were not satisfied, comparison between groups was performed using the Mann–Whitney U-test. The two-tailed chi-square (χ²) was used to test the null hypothesis for categorical variables.

Pearson's correlation was used to evaluate bivariate linear relations. Differences between groups were also tested for significance using analysis of variance, with subgroup analysis by the Scheffe F-test.

Multiple linear regression analysis was used to assess the influence of selected variables on parameters of diastolic and longitudinal systolic function, applying a stepwise method. Receiver operating characteristic (ROC) curves were constructed for detection of cut-off values. For ROC curves of diastolic dysfunction, septal Ea and strain values were taken into account. Patients having a mean longitudinal strain beyond the range of normality were regarded abnormal.

Bland-Altman's plot of differences was used to assess interobserver and intraobserver variability in 20 randomly selected patients. A P < 0.05 for a two-tailed test was considered significant.

Results

Characteristics of the study population

General and standard echocardiographic parameters of the study groups are summarized in Tables 1 and 2. Risk
factors for coronary artery disease between control (C) and hypertensives were the following – hypercholesterolemia: 20% vs. 56%, \( P < 0.05 \), diabetes 4% vs. 3.3%, \( P: \text{NS} \), smoking 8% vs. 18%, \( P < 0.05 \), family history for CAD: 12% vs. 15%, \( P: \text{NS} \). The percentages of patients taking certain medications were the following – ACE inh. 68%, b-blockers 34%, Ca blockers 39%, diuretics: 24%, A blockers 15% and statin 49%. There were no significant differences in risk factors or in medications between HTN-DD and HTN-ALL.

**Global systolic function**

The global systolic function as expressed by EF was similar between hypertensive patients whether with diastolic dysfunction or not and normal individuals (Table 1).

**Longitudinal diastolic function (Ea) by tissue Doppler**

Hypertensive patients had decreased septal and mean Ea, which were further deteriorated in the group with diastolic dysfunction (HTN-DD) (Table 2). Annular Ea correlations with other echocardiographic indices are presented on Table 3.

**Altered segmental relaxation**

Hypertensive patients had a greater number of segments (Figure 2) with altered relaxation pattern (Segmental DD), and these were more extensive in the group with diastolic dysfunction. Segmental DD was highly correlated with longitudinal strain and SR, as well as with mean annular Ea (Figure 3).

**Longitudinal systolic function by strain echocardiography**

Longitudinal systolic function estimated by mean strain and SR decreased in the hypertensive group but further deteriorated in the diastolic dysfunction group, compared with control (Table 2). Moreover, S and SR were positively correlated with diastolic septal and mean mitral annular Ea and negatively with L VMI. Correlations of strain and SR parameters with other echocardiographic indices are presented in Table 3.

From the hypertensive group with diastolic dysfunction, 51% (23/45) and 53% (24/45) were out of the range of

Table 1 General and echocardiographic characteristic of the study groups

<table>
<thead>
<tr>
<th></th>
<th>Control (n: 30)</th>
<th>HTN ALL (n: 75)</th>
<th>HTN-DD (n: 45)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48.58 ± 8</td>
<td>50.9 ± 10.1</td>
<td>51.8 ± 11.3</td>
</tr>
<tr>
<td>Male</td>
<td>61% (18)</td>
<td>65% (45)</td>
<td>61% (27)</td>
</tr>
<tr>
<td>WT (mm)</td>
<td>8.7 ± 1.1</td>
<td>10.9 ± 1.9*</td>
<td>12.0 ± 1.5*§</td>
</tr>
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<td>RWT</td>
<td>0.36 ± 0.05</td>
<td>0.47 ± 0.1*</td>
<td>0.51 ± 0.08*§</td>
</tr>
<tr>
<td>LVMI(gr/m²)</td>
<td>89.1 ± 17.4</td>
<td>111.3 ± 25.9*</td>
<td>121.2 ± 23.2*§</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.93 ± 0.13</td>
<td>1.89 ± 0.23</td>
<td>1.88 ± 0.27</td>
</tr>
<tr>
<td>BMI</td>
<td>25.8 ± 3.4</td>
<td>28.8 ± 4.7*</td>
<td>28.7 ± 4.4*</td>
</tr>
<tr>
<td>HTN (yrs)</td>
<td>–</td>
<td>9.1 ± 7.2*</td>
<td>10.3 ± 6.5*§</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>122.4 ± 7.7</td>
<td>144.6 ± 19.8*</td>
<td>149.8 ± 19.5*§</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>79.2 ± 4.1</td>
<td>82.9 ± 8.8*</td>
<td>83.8 ± 9.8*</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>93.3 ± 4.9</td>
<td>102.8 ± 11.6*</td>
<td>105.0 ± 12.1*§</td>
</tr>
<tr>
<td>PP (mmHg)</td>
<td>43.4 ± 7.3</td>
<td>61.5 ± 16.8*</td>
<td>66.0 ± 16.1*§</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>47.7 ± 6.4</td>
<td>47.1 ± 7.4</td>
<td>46.7 ± 7.7</td>
</tr>
<tr>
<td>LVESD (mm)</td>
<td>30.6 ± 3.9</td>
<td>30.1 ± 4.8</td>
<td>29.5 ± 4.1</td>
</tr>
<tr>
<td>LVEDD (mm/m²)</td>
<td>24.7 ± 2.7</td>
<td>24.9 ± 2.9</td>
<td>24.8 ± 2.1</td>
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<tr>
<td>EF (%)</td>
<td>66.2 ± 5.1</td>
<td>67.7 ± 7.9</td>
<td>68.0 ± 9.3</td>
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<td>DD (ms)</td>
<td>191.8 ± 33.2</td>
<td>218.9 ± 53.5*</td>
<td>235.9 ± 55.1*§</td>
</tr>
<tr>
<td>IVRT (ms)</td>
<td>82.5 ± 25.2</td>
<td>126.5 ± 19.6*</td>
<td>131 ± 16.5*§</td>
</tr>
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<td>E (cm/s)</td>
<td>66.0 ± 11.0</td>
<td>65.2 ± 14</td>
<td>63.0 ± 13.5*§</td>
</tr>
<tr>
<td>A (cm/s)</td>
<td>53.1 ± 14.8</td>
<td>66.6 ± 16.5*</td>
<td>72.5 ± 15.5*§</td>
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<tr>
<td>E/A</td>
<td>1.31 ± 0.38</td>
<td>0.97 ± 0.35*</td>
<td>0.83 ± 0.20*§</td>
</tr>
<tr>
<td>HR</td>
<td>68.1 ± 9.1</td>
<td>69.9 ± 12.2</td>
<td>69.0 ± 14.5</td>
</tr>
</tbody>
</table>

HTN-DD = hypertensives with diastolic dysfunction; HTN-ALL = the whole hypertensive group; \( P < 0.05 \): HTN-ALL or HTN-DD vs. normal subjects. \( P < 0.05 \): HTN-DD vs. HTN-ALL.

Figure 1 Sample from interrogation of the septal wall along with representative strain rate curve of one region with abnormal relaxation pattern.
normality for longitudinal systolic SR and longitudinal systolic strain, compared with controls.

Filling pressures

Filling pressures were increased in the hypertensive group but were more pronounced in the diastolic dysfunction group, compared with control (Table 2). Filling pressures were inversely correlated with longitudinal systolic Strain and SR deformation parameters (Table 3).

Prediction of longitudinal systolic dysfunction

When septal Ea was regarded as an index of diastolic dysfunction, the value $Ea = 5.9 \text{ cm/s}$ predicted longitudinal systolic dysfunction based on strain with sensitivity: 83%, specificity: 71% (area under the curve: 0.815, std. error: 0.043, $P < 0.001$, asymptotic 95% CI: 0.730–0.900).

| Table 2 | Echocardiographic characteristics of the study groups |
|---------------------------------|---------------------------------|---------------------------------|
| Mean longitudinal strain rate (1/s) | 1.62 ± 0.18 | 1.46 ± 0.23* | 1.37 ± 0.19* |
| Mean longitudinal strain (%) | 20.2 ± 1.8 | 18.3 ± 2.4* | 17.4 ± 2.1* |
| LVS (Kdynes/cm²) | 83.5 ± 10.6 | 75.5 ± 21.4* | 71.5 ± 21.9* |
| Mean mitral annulus (Ea) (cm/s) | 10.2 ± 1.7 | 7.5 ± 1.8* | 6.3 ± 1.4* |
| Septal mitral annulus (Ea) (cm/s) | 9.6 ± 2.1 | 6.7 ± 1.3* | 5.6 ± 1.5* |
| Filling pressures E/Ea mean | 6.4 ± 0.9 | 8.6 ± 2.9* | 10.1 ± 3.2* |
| Segmental DD | 5 ± 3 | 9 ± 4* | 11 ± 3* |

HTN-DD = hypertensive with diastolic dysfunction; HTN-ALL = the whole hypertensive group; mean mitral annulus Ea = septal + lateral, *$P < 0.05$: HTN-ALL or HTN-DD vs. control, SP < 0.05: HTN-DD vs. HTN-ALL. Segmental DD = number of segments with altered relaxation pattern.

| Table 3 | Correlations between strain, SR, Ea, and echocardiographic parameters |
|---------------------------------|---------------------------------|---------------------------------|
| Mean longitudinal strain rate | Mean longitudinal strain | Mean annular Ea |
| Pearson correlation | $r$ | $r$ | $r$ |
| LVMI | $-0.52^*$ | $-0.51^*$ | $-0.56^*$ |
| WT | $-0.56^*$ | $-0.57^*$ | $-0.58^*$ |
| RWT | $-0.46^*$ | $-0.49^*$ | $-0.60^*$ |
| Age | $-0.50^*$ | $-0.47^*$ | $-0.64^*$ |
| BMI | $-0.08$ | $-0.09$ | $-0.28^*$ |
| SBP | $-0.45^*$ | $-0.46^*$ | $-0.52^*$ |
| DBP | $-0.15$ | $-0.09$ | $-0.13$ |
| MAP | $-0.33^*$ | $-0.27^*$ | $-0.40^*$ |
| PP | $-0.48^*$ | $-0.51^*$ | $-0.54^*$ |
| DT | $-0.22$ | $-0.19$ | $-0.31^*$ |
| IVRT | $-0.33^*$ | $-0.31^*$ | $-0.38^*$ |
| A | $0.28^*$ | $0.26^*$ | $0.35^*$ |
| E/A | $0.34^*$ | $0.41^*$ | $0.66^*$ |
| E/Ea | $-0.41^*$ | $-0.43^*$ | $-0.77^*$ |
| Segmental DD | $-0.57^*$ | $-0.56^*$ | $-0.68^*$ |
| Mean annular Ea | $0.54^*$ | $0.52^*$ | -- |

Independent predictors

A multiple linear regression model was used to assess independent predictors of diastolic and longitudinal systolic function. Mitral annular Ea and Mean Strain were regarded as indices of diastolic dysfunction and longitudinal systolic dysfunction respectively and were entered in the statistical model as dependent variables. All the significant univariate parameters were entered as independent variables. LVMI, SBP and age were proved as independent predictors for both diastolic dysfunction and longitudinal systolic dysfunction, whereas BMI was also found to be independently associated with diastolic dysfunction (Table 3).

Reproducibility

Reproducibility and variability were as follows – interobserver SR (SR differences between observers): arithmetic
mean $= 0.005$, standard deviation $= 0.095$, with 95% limits of agreement ($\pm 1.96SD$): $-0.183$ to $0.193$.

Interobserver S (strain differences between observers): arithmetic mean $= 0.050$, standard deviation $= 0.851$, with ($\pm 1.96SD$): $-1.62$ to $1.72$.

Intraobserver SR (SR differences): arithmetic mean $= -0.008$, standard deviation $= 0.074$, with ($\pm 1.96SD$): $-0.154$ to $0.138$.

Intraobserver S (strain differences): arithmetic mean $= 0.0850$, standard deviation $= 0.5878$, with ($\pm 1.96SD$): $-1.07$ to $1.24$.

Discussion

The present study’s main finding is that hypertensive patients who appear with diastolic dysfunction, frequently have longitudinal systolic dysfunction based on strain echocardiography, especially when mitral annular septal diastolic velocity is $\leq 5.9$ cm/s.

Altered segmental relaxation is a novel method of evaluating regional changes and is better correlated with longitudinal systolic Strain and SR compared with conventional indices of diastolic dysfunction. LV hypertrophy, SBP and age are independently related to both diastolic and longitudinal systolic dysfunction, whereas obesity appears to contribute significantly to the appearance of diastolic dysfunction.

The term ‘diastolic dysfunction’ refers to an abnormality of diastolic distensibility, filling or relaxation of the left ventricle, regardless of whether the EF is normal or abnormal and whether the patient is symptomatic or asymptomatic. This study’s hypertensive cohort with diastolic dysfunction consisted of a mix of patients with symptomatic and asymptomatic diastolic dysfunction with normal EF. Deformation parameters can evaluate regional systolic function, and it has been shown that the systolic SR (SR) is related to invasive measures of contractility, whereas strain correlated well with changes in stroke volume-EF, and therefore is more closely related to changes in global haemodynamics than changes in contractility.

According to our findings, a substantial number of hypertensive patients with diastolic dysfunction (51%) appeared to have depressed mean SR, and a similar proportion (53%) showed decreased longitudinal haemodynamic performance, as depicted by strain.

It is generally accepted that myocardial fibres responsible for longitudinal motion and function are placed close to the subendocardium, contrary to the circumferential mid-wall myocardial fibres commissioned for radial function. Stein et al. found a higher intramyocardial pressure in the endocardium than in the epicardium, and this could be one explanation why longitudinal fibres and function seem to be vulnerable to subendocardial ischaemia and fibrosis, findings...
not uncommon in hypertension. At the same time, increased afterload increases mechanical stress, which, through internal transduction mechanisms and the secretion of Angiotensin II and Endothelin 1, provokes hypertrophy and increased collagen synthesis, an environment that favours diastolic dysfunction.

However, the high relation between longitudinal systolic and diastolic dysfunction might have more explanations. Diastole is an energy-dependent process (in the first part) and also includes systolic components of the active systole of ascending fibres in isovolumic relaxation. Moreover, intrinsic myocytic impairment constitutes part of the pathology in hypertensive disease, as evidenced by recent studies showing the association between impaired LV filling and sub-normal high energy phosphate metabolism, calcium movement abnormalities and beta-adrenergic receptor density, the last of which strongly depends on both systolic myocardial velocity and early diastolic Ea. Furthermore, recent evidence also suggests that subendocardial myocardium constitutes a distinct functional-anatomical compartment with a separate contribution to cardiac performance in both normal and diseased hearts. That compartment is characterized by its own biochemical pathways that seem to play an important role in the natural history of diastolic dysfunction and diastolic heart failure. That background could explain the high correlation between mean longitudinal SR/S, Ea and segmental DD in our study, in addition to the well-described ischaemia-induced subendocardial fibrosis.

The placement within the range of normal longitudinal systolic function of a number of patients with DD underlines the fact that diastolic dysfunction is an antecedent event, compared with systolic dysfunction. The progression of diastolic dysfunction is accompanied by deterioration of longitudinal systolic dysfunction, indicating that the two conditions are related to various degrees, proportionally to the pathophysiological substrate of the subjects under study, or that they could constitute a continuous spectrum of disease processes with the same natural history. That continuous spectrum is also suggested by the cut-off value of Septal Ea to predict the presence of longitudinal systolic dysfunction, provided in our study.

Altered segmental relaxation by strain echocardiography estimates directly regional tissue structural changes and appears to delineate better the relation between diastolic and longitudinal systolic dysfunction as presented in our study (Table 3) (Figure 3).

Previous studies also found longitudinal function to be decreased in various percentages in patients with DHF or DD. However, the diagnostic accuracy of deformation parameters is expected to be higher, because they directly estimate tissue function, rather than indirectly estimating annular velocity. For this reason, the mean values of the basal-mid-parts of the left ventricle, as provided in our study, are expected to represent more accurately the functional condition of that part of the heart.

It is also important to emphasize that the group of patients with diastolic dysfunction also showed evidence of LVH. LVMI independently and directly affected both diastolic and longitudinal systolic dysfunction, underlining the importance of LVH in hypertensive disease, as previous studies have shown.

Finally, our study also confirmed that obesity (BMI) is an important risk factor for diastolic dysfunction, a finding that is also supported by other studies.

Clinical significance

A substantial number of patients with an apparently common diastolic dysfunction might also have longitudinal systolic dysfunction, based on strain echocardiography, despite their normal EF. A cut-off value of ≤5.9 for Septal Ea should raise the suspicion for the concurrent presence of longitudinal systolic abnormalities. Altered segmental relaxation patterns appear to be an important alternative method for the evaluation of the hypertensive patient, given the high correlation with global diastolic and longitudinal systolic parameters.

Limitations

The coexistence of coronary artery disease has not been excluded in all the hypertensive patients, as only 51% of the subjects had undergone tests to detect ischaemia. However, there was no evidence for CAD, such as wall motion abnormalities, ECG findings or chest pain on exertion. Furthermore, the incidence of smoking, diabetes and family history for CAD was low in the study group, making the presence of CAD unlikely. Additionally, there was no evidence of decreased S or SR in territories supplied by certain coronary arteries in any of the patients under study.

Independent confirmation of global DD using invasive haemodynamic measurements was not performed. However, Doppler-derived indices for estimation of diastolic function have been validated against tau and are widely used to assess global DD in large population-based studies.

Left ventricular end diastolic index was normal (<32 mm/m²) and actually matched between the control and hypertensive patients, reducing the probability of diastolic left ventricular dysfunction secondary to a high end systolic load or significant preload changes.

Conclusion

Longitudinal systolic dysfunction maybe be present in hypertensive patients with diastolic dysfunction, despite their normal overall systolic function based on EF.

Altered segmental relaxation patterns correlate highly with longitudinal systolic dysfunction.

A cut-off value of ≤5.9 for Septal Ea appears to be associated with concurrent presence of longitudinal systolic dysfunction. LV hypertrophy, SBP and aging are important determinants of both diastolic and longitudinal systolic dysfunction, whereas obesity appears to contribute to the appearance of diastolic dysfunction.

Conflict of interest: none declared.

References


