Influence of afterload on left ventricular radial and longitudinal systolic functions: a two-dimensional strain imaging study

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Aims This study aimed to assess the influence of afterload alteration on radial (R) and longitudinal (L) left ventricular (LV) systolic regional functions.

Methods and results We analysed systolic myocardial function by two-dimensional strain (2D-S) and sonomicrometry (SS) in an experimental pig model of aortic banding. Both radial and longitudinal functions were analysed in six open-chest pigs under various loading conditions: baseline and graded aortic banding (subsequent increase in LV pressure of 10, 20, and 40 mmHg). Both systolic 2D-Slong and 2D-Srad were significantly correlated with SSlong and SSrad (r = 0.63, P < 0.001 and r = 0.65, P < 0.01, respectively). At a low increase in LV afterload, 2D-Srad was still preserved whereas 2D-Slong significantly decreased. When LV afterload was subsequently increased, both 2D-Srad and 2D-Slong significantly decreased. Difference in dependence to wall stress might explain these different behaviours.

Conclusion 2D-S shows a different response in longitudinal and radial functions to increased afterload. Longitudinal function is early impaired, whereas radial function remains preserved. This finding justifies the combined assessment of both radial and longitudinal regional myocardial functions to characterize myocardial dysfunction and might help to better identify the transition to heart failure in pressure-overload cardiomyopathy.

KEYWORDS
Left ventricular systolic function; Afterload; Strain

Introduction
The influence of acute changes in afterload on myocardial performance has been evaluated in various models (isolated muscle preparations, intact heart) that have demonstrated the afterload dependence of all the ejection phase indices of systolic properties.1 Therefore, global conventional indices of systolic function such as left ventricular (LV) fractional shortening and LV ejection fraction lack sufficient accuracy to quantify the changes in myocardial contractility and to early identify the transition from compensated hypertrophy to heart failure in pressure-overload cardiomyopathies.2–4 The recent parameters of regional systolic function such as strain and strain rate are also affected by afterload changes but might be less load-dependent than conventional parameters.5–8 Owing to the complex anatomical orientation of myocardial fibres, the analysis of regional myocardial function requires the information on both radial and longitudinal functions. Two-dimensional strain (2D-S) is a new ultrasonic modality based on the speckle-tracking analysis and provides the unique opportunity to get the regional deformation parameters from a single grey-scale image both in the longitudinal and the radial directions.9–12

The radial function of the left ventricle is due mainly to the contraction of the circumferential myocardial fibres in the mid-wall, whereas the longitudinal function is governed by the subendocardial fibres. Since the subendocardium is more vulnerable to increased wall stress, ischaemia, and interstitial fibrosis, we hypothesized that the combined measurement of longitudinal and radial shortening of the ventricle might be a more sensitive marker of subclinical changes in LV performance in pressure-overload than the assessment of global function by conventional methods.2,13–15

In this study, we sought to analyse the consequences of pressure-overload on longitudinal and radial strains in a pig model of pressure-overload by progressive aorta banding.

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Methods

Animal preparation

This study is in accordance with the American Association for Accreditation of Laboratory Animal Care.

Surgical preparation

Sixteen farm pigs, weighing 28 ± 4 kg, were pre-medicated with droperidol (1 mg/kg sc) and anaesthetized with pentobarbital (15 mg/kg iv). Additional intravenous administration of pentobarbital was performed when needed. Pigs were ventilated with room air through a tracheotomy tube, and tidal volume and rate were adjusted to provide physiological pH and blood gases. Body temperature was monitored with a rectal thermometer and kept constant by means of a heating pad. Cannulae were inserted into the right jugular vein (for administration of drugs and fluids) and the left carotid artery (for measurement of blood pressure). A high-fidelity micro-manometer catheter (Millar, Houston, TX, USA) was inserted into the right carotid artery and advanced in the mid-part of the LV cavity for recording LV pressure and its first derivative, LV dp/dt max. ECG limb leads, arterial and LV pressures, and LV dp/dt_max were monitored continuously on a Gould recorder (Gould Inc.). A median sternotomy was performed. The pericardium was opened and suspended. The distal part of the ascending aorta was exposed in order to place a snare, for graded aortic banding. One segment of the left anterior descending (LAD) was isolated just before the first diagonal branch.

In order to analyse radial function, two pairs of ultrasonic crystals were inserted via a small scalpel incision in the inner and outer layers of the anterior wall and orientated parallel to the short axis. In order to analyse longitudinal function, another pair of ultrasonic crystals was placed next to the other crystals, parallel to the long axis. The correct position of the pairs of ultrasonic crystals was checked at the end of each experiment after euthanasia.

The animals were allowed 30 min after these surgical procedures to stabilize.

Echocardiography

Epicardial echocardiography was performed using a Vivid 7 (GE VingMed, Milwaukee, WI, USA) equipped with a 2.5 MHz phased-array transducer. All measurements were performed with the heart suspended in a pericardial cradle. The image sector angle was 60° and the frame rate was 76 frames per second. We stored five consecutive cardiac cycles on an optical disk for further analysis. Using Echocap software (GE VingMed), systolic longitudinal strain and transverse strain (as a surrogate of radial function) were measured within the myocardial segment equipped with sonomicrometry, at end systole as assessed by aortic valve closure from the single apical two-chamber view. The LV end-diastolic and end-systolic volumes were measured in the apical two- and four-chamber views using Simpson’s method. The LV end-diastolic and end-systolic diameters were measured as well as LV posterior wall thickness by M-mode in the short-axis view. The sphericity index was calculated at end systole and at end-diastole as the LV minor-axis dimension (short-axis) divided by the major-axis dimension (length).16

Experimental protocol

In order to compare 2D-S and SS indices, myocardial function was serially assessed at baseline, after increasing LV afterload by aortic banding (LV-aorta pressure gradient was progressively increased from 10 (n = 6), 20 (n = 16), 40 (n = 13) to 60 mmHg (n = 7)), during myocardial ischaemia induced by a 10 min ligation of the LAD coronary artery.

In order to evaluate the influence of increased afterload on radial and longitudinal functions, six pigs were explored at baseline, and after consecutive and graded aortic banding leading to 10, 20, and 40 mmHg of LV-aorta pressure gradients. A period of stabilization (10 min) was allowed between each stage.

Data acquisition

Haemodynamics

Heart rate, arterial and LV blood pressures, and LV dp/dt_max were averaged over five continuous cardiac cycles in sinus rhythm at baseline, during aortic banding, during coronary occlusion, and after recovery.

Regional myocardial function by sonomicrometry

By calculating the instantaneous length in percentage of end-diastolic length, we analysed the longitudinal (SS_long) and the radial (SS_rad) functions by measuring the segment fractional shortening (SS) (Figure 1). SS (%) was defined as: [(EDL – ESL)/EDL] × 100%, where ESL and EDL are end-systolic and end-diastolic lengths, respectively. ESL and EDL were obtained from five cardiac cycles in each sample period. EDL was measured at the onset of the rapid increase in LV dp/dt_max, whereas ESL was measured at peak negative LV dp/dt.

Regional myocardial function by two-dimensional strain

Echocap workstation (GE VingMed) enabled the measurements of systolic longitudinal (2D-S_long) and radial (2D-S_rad) strains within the mid-anterior segment (at the position of the sonomicrometry microcrystals) in each sample period by tracking the natural acoustic markers (specckles) between the endocardium and the epicardium borders, as described previously.7 Five beats were averaged for each of these measurements (Figure 1).

Estimation of left ventricular wall stress

LV end-systolic meridional wall stress was estimated as 0.334 × LV pressure × LVs/PWs [1 + (PWs/LVs)], where LV pressure is the LV systolic pressure, LVs the systolic left ventricular diameter, and PWS the systolic posterior wall thickness.17

Statistical analysis

To test the reproducibility of 2D-S, 10 measurements were randomly re-analysed. Inter- and intra-observer variabilities were calculated as the difference between the two observations divided by the mean of the observations and were expressed as both absolute numbers and percentages.

Values were expressed as mean ± SD. The differences between echocardiographic and sonomicrometric variables before and after load alterations were tested with ANOVA for repeated measurements with a contrast analysis (each aortic banding stage vs. baseline). When differences in variances were found, ordinal logistic fit was applied. The 2D-S and SS were correlated using the simple linear regression analysis. Bland and Altman graphs were drawn to complete the assessment of the correlation between SS and 2D-S.18

The reliability of observer variability and the reproducibility of the echocardiographic and sonomicrometric parameters were, furthermore, assessed using the intraclass correlation coefficients (ICC) as described by Baumgartner.19 Reliability was considered to be ‘good’ if ICC ranged from 0.61 to 0.81 and ‘almost perfect’ if it exceeded 0.81.

A P-value of < 0.05 was considered as statistically significant. SPSS 10.0 statistical software (SPSS Inc., Chicago, IL, USA) was used for analysis.

Results

Among the 16 pigs included in the present study, comparative assessment of systolic regional myocardial function
was performed at baseline and during aortic banding, and mid-LAD coronary artery occlusion, representing therefore 81 matched measurements of deformation by sonomicrometry and 2D-S. During aortic banding, both systolic strain and systolic segmental shortening gradually decreased. During LAD occlusion, both systolic strain (2D-S\textsubscript{long}: 2.02 ± 5.45% vs. −16 ± 3%; 2D-S\textsubscript{rad}: −4.6 ± 8.14% vs. 37.6 ± 9.2%; \( P < 0.01 \)) and systolic shortening (SS\textsubscript{long}: 2.4 ± 1.03% vs. −19.4 ± 2.4%; SS\textsubscript{rad}: −4.5 ± 2.3% vs. 39.7 ± 4.4%; \( P < 0.01 \)) dramatically decreased from baseline values. Adequate analysis of longitudinal function was obtained in 95% of measurements by SS and in 98% by 2D-S. Adequate analysis of radial function was obtained in 92% of measurements by SS and in 98% by 2D-S. Intra- and inter-observer reproducibility are displayed in Table 1. SS and 2D-S measurements were significantly correlated in both longitudinal (\( r = 0.63, \ P < 0.05 \)) and radial (\( r = 0.65, \ P < 0.05 \)) deformation (Figure 2). The ICC were excellent to assess the agreement between echocardiographic and sonomicrometric both for longitudinal (ICC = 0.90) and radial (ICC = 0.97) functions.

### Haemodynamic data

All pigs had comparable heart rate, cardiac index, and LV \( \frac{dp}{dt\text{\textsubscript{max}}} \) at baseline and throughout the experiment (Table 2). Left-ventricular end-systolic pressure and end-systolic wall stress significantly increased with moderate and severe aortic banding vs. baseline.

### Conventional echographic data

LV end-diastolic volume significantly increased with moderate and severe aortic banding and was associated with a slight increase in sphericity index. Wall thickness did not significantly increase.

Assessment of global systolic function by LV fractional shortening showed no change with mild banding but a
Influence of increased afterload on radial and longitudinal myocardial functions

As soon as a slight banding (LV-aorta pressure gradient = 10 mmHg) was performed, longitudinal deformation was early impaired as demonstrated by 2D-S (Δ = −6.8 ± 4.1%, P < 0.05 vs. baseline values) and at a lesser extent by SS (Table 3; Figure 1). Furthermore, longitudinal strain was severely depressed when the afterload was increased.

Conversely, radial function was preserved and maintained near to baseline values as demonstrated by 2D-S (Δ = +7.8 ± 10.4%, P = NS) and by SS (Table 3) after a slight banding (LV-aorta pressure gradient: 10 mmHg) (Figures 1 and 3).

This difference between longitudinal and radial functions regarding the afterload mismatch may be explained by the influence of end-systolic meridional wall stress on these two components of myocardial function. Indeed, as shown in Figure 4, there was an inverse relationship between 2D-Slong and end-systolic wall stress (r = 0.68, P < 0.001), whereas 2D-Srad was less affected than LV wall stress (r = 0.50, P = 0.02).

Discussion

In this model of acute pressure-overload, we used the speckle-tracking analysis to quantify from a single apical view the longitudinal and the radial systolic functions. We demonstrated that systolic longitudinal strain was a sensitive marker of subclinical changes induced by pressure-overload since it early and dramatically fell as afterload increased, whereas LV fractional shortening and radial strain were still preserved. Thus, despite normal conventional indices of systolic function (LV fractional shortening and LV dP/dt max), longitudinal 2D-S early revealed systolic abnormalities during mild aorta banding.

Indeed, LV systolic function, as assessed by LV fractional shortening, was not altered during mild increase in LV afterload. In contrast, there was a clearly detectable reduction in regional longitudinal shortening as assessed by both SS and 2D-S. This significant reduction in longitudinal shortening increased with a concomitant increase in LV pressure. In addition to the reduction in longitudinal deformation, strain curves demonstrated a post-systolic deformation increasing significantly with increased afterload (Figure 1).

Conversely, with moderate to mild aortic banding, radial function was preserved, acting therefore as a compensatory phenomenon to the decrease in longitudinal deformation to

significant decrease with moderate to important increase in LV-aorta gradient (Table 2).

Intra- and inter-observer variabilities for longitudinal two-dimensional strain and ε assessment

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Mean difference</th>
<th>Coefficient variation (%)</th>
<th>Intraclass coefficient</th>
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<tr>
<td></td>
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<tr>
<td>Intra-observer variability</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2D-Srad (%)</td>
<td>21.6</td>
<td>2.9</td>
<td>14.1</td>
<td>0.96</td>
</tr>
<tr>
<td>2D-Slong (%)</td>
<td>9.2</td>
<td>1.4</td>
<td>15.1</td>
<td>0.98</td>
</tr>
<tr>
<td>Inter-observer variability</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2D-Srad (%)</td>
<td>23.1</td>
<td>3.0</td>
<td>15.5</td>
<td>0.96</td>
</tr>
<tr>
<td>2D-Slong (%)</td>
<td>8.7</td>
<td>1.3</td>
<td>16.5</td>
<td>0.93</td>
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</table>

Values are mean ± SD, n = 10. Intra- and inter-observer variabilities were calculated as the difference between the two observations divided by the means of the observations and expressed as both absolute numbers and percentage.

Figure 2  (A) Left panel: plot showing the relation between left ventricular longitudinal shortening (SSlong) by sonomicrometry and two-dimensional strain echocardiography (2D-Slong). Right panel: Bland–Altman plot showing the mean difference (dotted middle line) and 95% limits of agreement (dashed lines). (B) Left panel: plot showing the relation between left ventricular radial shortening (SSrad) by sonomicrometry and two-dimensional strain echocardiography (2D-Srad). Right panel: Bland–Altman plot showing the mean difference (dotted middle line) and 95% limits of agreement (dashed lines).
maintain a normal LV fractional shortening. This phenomenon has been described in hypertensive patients in whom was reported a supra normal LV ejection fraction leading to the suggestion that LV systolic function could be increased in those patients. As a part of a compensatory mechanism, in many hypertensive patients, LV chamber function is maintained a normal LV fractional shortening. This phenomenon has been described in hypertensive patients in whom was reported a supra normal LV ejection fraction leading to the suggestion that LV systolic function could be increased in those patients. As a part of a compensatory mechanism, in many hypertensive patients, LV chamber function is maintained a normal LV fractional shortening.

Our results are in agreement with previous studies that pointed out that regional changes in deformation are determined by both the fibre structure of the myocardium and its interaction with local wall stress. Myocardium is a complex three-dimensional structure consisting of myocytes orientated in different directions with their own intrinsic contractile properties. The subendocardial fibres are mainly longitudinally orientated, whereas mid-wall fibres are circumferential. Both fibre orientation and local geometry result in a non-uniform wall stress distribution, with a decrease from endo- to epicardium and from equator to apex. All indices of systolic function are altered by acute interaction among the different myocardial fibres causes the magnitude of endocardial displacement to depend negatively on the magnitude of wall stress. Indeed, in our study, we observed a higher relationship between wall stress and longitudinal strain than with radial strain.

Our results are in agreement with previous studies that pointed out that regional changes in deformation are determined by both the fibre structure of the myocardium and its interaction with local wall stress. Myocardium is a complex three-dimensional structure consisting of myocytes orientated in different directions with their own intrinsic contractile properties. The subendocardial fibres are mainly longitudinally orientated, whereas mid-wall fibres are circumferential. Both fibre orientation and local geometry result in a non-uniform wall stress distribution, with a decrease from endo- to epicardium and from equator to apex. All indices of systolic function are altered by acute or chronic changes in preload, afterload, and contractility and influenced by remodelling. It has been recently suggested that myocardial strain and strain rate are less load-dependent than other shortening parameters and should represent contractility. However, most of these studies did not incorporate in the analysis, factors such as preload, afterload, or LV geometrical changes. In addition, the respective influence of loading conditions on longitudinal

### Table 2: Echocardiographic and haemodynamic parameters at baseline and over the afterload modifications

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Aortic banding + 10 mmHg</th>
<th>Aortic banding + 20 mmHg</th>
<th>Aortic banding + 40 mmHg</th>
<th>Recovery</th>
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<tbody>
<tr>
<td><strong>Haemodynamic parameters</strong></td>
<td></td>
<td></td>
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<tr>
<td>Heart rate (bpm)</td>
<td>101 ± 13</td>
<td>100 ± 13</td>
<td>99 ± 15</td>
<td>98 ± 18</td>
<td>96 ± 21</td>
</tr>
<tr>
<td>Cardiac index (l/min/m²)</td>
<td>3.6 ± 0.3</td>
<td>3.8 ± 0.7</td>
<td>3.5 ± 0.4</td>
<td>3.3 ± 0.4</td>
<td>4.6 ± 2.1</td>
</tr>
<tr>
<td>LV dP/dt max (mmHg/s)</td>
<td>1621 ± 244</td>
<td>1463 ± 128</td>
<td>1478 ± 360</td>
<td>1330 ± 139</td>
<td>1335 ± 228</td>
</tr>
<tr>
<td>LV ESV (mmHg)</td>
<td>89 ± 13</td>
<td>96 ± 7</td>
<td>110 ± 10*</td>
<td>134 ± 10*</td>
<td>79 ± 12*</td>
</tr>
<tr>
<td>LV EDV (mmHg)</td>
<td>33 ± 6</td>
<td>37 ± 5</td>
<td>38 ± 3</td>
<td>40 ± 3</td>
<td>31 ± 12</td>
</tr>
<tr>
<td>Wall stress (Kdynes/m²)</td>
<td>56 ± 11</td>
<td>55 ± 10</td>
<td>77 ± 19*</td>
<td>82 ± 36</td>
<td>50 ± 14</td>
</tr>
<tr>
<td><strong>Echocardiographic parameters</strong></td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>LV EDV (mL)</td>
<td>33 ± 4</td>
<td>37 ± 4</td>
<td>42 ± 3*</td>
<td>48 ± 6*</td>
<td>36 ± 4*</td>
</tr>
<tr>
<td>LV EDD (mm)</td>
<td>32 ± 5</td>
<td>35 ± 3</td>
<td>38 ± 2*</td>
<td>40 ± 2*</td>
<td>36 ± 6</td>
</tr>
<tr>
<td>LV ESD (mm)</td>
<td>24 ± 5</td>
<td>26 ± 5</td>
<td>30 ± 3*</td>
<td>32 ± 3*</td>
<td>27 ± 3*</td>
</tr>
<tr>
<td>LV PWT (mm)</td>
<td>9.2 ± 1</td>
<td>10.6 ± 0.5</td>
<td>10.8 ± 0.7</td>
<td>10.8 ± 1.2</td>
<td>10.2 ± 0.7</td>
</tr>
<tr>
<td>LV fractional shortening (%)</td>
<td>28 ± 6</td>
<td>29 ± 4</td>
<td>22 ± 6*</td>
<td>21 ± 6*</td>
<td>25 ± 4</td>
</tr>
<tr>
<td>Sphericity index</td>
<td>0.62 ± 0.09</td>
<td>0.66 ± 0.09</td>
<td>0.69 ± 0.05*</td>
<td>0.70 ± 0.04*</td>
<td>0.65 ± 0.10</td>
</tr>
</tbody>
</table>

Values are mean ± SD, n = 6. LV EDP, left ventricular end-diastolic pressure; LV ESP, left ventricular end-systolic pressure; LV EDV, left ventricular end-diastolic volume; LV EDD, left ventricular end-diastolic diameter; LV ESD, left ventricular end-systolic diameter; LV PWT, left ventricular posterior wall thickness. Data were analysed using ANOVA for repeated measurements.

*P < 0.05 vs. baseline.
†P < 0.05 vs. aortic banding 10 mmHg.
‡P < 0.05 vs. aortic banding 20 mmHg.
‡‡P < 0.05 vs. aortic banding 40 mmHg.

### Table 3: Measurements of longitudinal and radial deformation at baseline and during afterload increase

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Banding (10 mmHg)</th>
<th>Banding (20 mmHg)</th>
<th>Banding (40 mmHg)</th>
<th>Recovery</th>
<th>One-way ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>SS_rad (%)</td>
<td>39.7 ± 4.4</td>
<td>39.1 ± 3.5</td>
<td>37.0 ± 3.1</td>
<td>26.1 ± 4.4*</td>
<td>38.9 ± 3.7*</td>
<td>P &lt; 0.001; F = 7.8</td>
</tr>
<tr>
<td>SS_long (%)</td>
<td>−19.4 ± 2.4</td>
<td>−17.2 ± 2.8</td>
<td>−16.9 ± 4.1*</td>
<td>6.9 ± 8.0*</td>
<td>11.1 ± 7.1*</td>
<td>P &lt; 0.05; F = 3.5</td>
</tr>
<tr>
<td>ZD_s_rad (%)</td>
<td>37.6 ± 9.2</td>
<td>45.5 ± 12.8</td>
<td>23.8 ± 5.7*</td>
<td>22.0 ± 6.4*</td>
<td>36.6 ± 12.1*</td>
<td>P &lt; 0.001; F = 10.6</td>
</tr>
<tr>
<td>ZD_s_long (%)</td>
<td>−16.0 ± 3.0</td>
<td>−9.2 ± 2.3*</td>
<td>−6.5 ± 3.2*</td>
<td>−5.8 ± 3.2*</td>
<td>14.6 ± 3.5*</td>
<td>P &lt; 0.001; F = 12.2</td>
</tr>
</tbody>
</table>

Data were analysed using ANOVA for repeated measurements, n = 6. SS, sonomicrometric assessment of segmental shortening (%); long, longitudinal; rad, radial.

*P < 0.05 vs. baseline.
†P < 0.05 vs. aortic banding 10 mmHg.
‡P < 0.05 vs. aortic banding 20 mmHg.
‡‡P < 0.05 vs. aortic banding 40 mmHg.
and radial functions has not been specifically assessed. The wall stress difference between mid-wall and longitudinal fibres might explain why we observed an early decrease in longitudinal function, while radial function was preserved. Since the longitudinal fibres show a larger radius of curvature, the increase in stress on the longitudinally orientated endocardial fibres is more pronounced compared with the circumferential fibres. Therefore, circumferential fibres, generating the radial deformation of the myocardium, are much better in generating higher pressure and will retain normal functional parameters for a longer time.

This observation can be extended to other pathological conditions where longitudinal function is early affected and radial function still preserved. Recently, Reant et al. used an open-chest animal model to examine the validity of strain measurements under varying conditions of flow-limiting and non-flow-limiting coronary artery stenosis at rest and during dobutamine infusion. Interestingly, longitudinal strain was abnormal at rest during flow-limiting stenosis and during stress with non-flow-limiting stenosis, whereas radial strain was only abnormal during stress in the setting of flow-limiting stenosis. An alternative explanation is that the longitudinal fibres located in the subendocardium are more susceptible to ischaemia and are therefore affected earlier in the ischaemic cascade.

Conversely, rapid decrease in afterload such as in aortic valve percutaneous replacement leads to an acute increase in longitudinal strain, despite the lack of significant change in LV ejection fraction.

These findings suggest that the combination of regional indices of longitudinal and radial functions and LV geometry might be more accurate for the detection of early alteration in systolic performance in pressure-overload cardiomyopathy, especially in asymptomatic patients.

Previous studies have also demonstrated the higher sensitivity of longitudinal function in detecting subclinical alteration in LV function in other clinical settings such as diabetes mellitus.

These studies were based on Doppler tissue imaging and its derived velocity and strain rate measurements that depend on Doppler angle and are limited to the segments in which motion and deformation are aligned with the ultrasound beam. In addition, velocity measurements reflect myocardial motion more than function and are influenced not only by contractility and wall stress but also by the interaction with the adjacent segments and the overall cardiac motion. The new ultrasound technique based on grey-scale B-mode images, the speckle-tracking echocardiography (2D-S), enables the assessment of myocardial deformation or strain during the cardiac cycle by automatic tracking of myocardial segments. Strain measurement using STE is independent of the insonation angle, therefore allowing complete evaluation of all myocardial segments. However, we could not perform any direct comparison between Doppler- and speckle-tracking imaging-based strain, since tissue Doppler imaging was not acquired in this present study.
Limitations

Compared with previous studies, the accuracy of 2D-S in measuring strain in both longitudinal and radial directions was not as satisfactory when compared with SS. The bias that we reported was similar to previously reported correlations, but the limit of agreements were higher. Many factors might have affected our results: we only induced a decrease in regional myocardial function, whereas previous studies both increased (dobutamine infusion or saline loading) and decreased (ischaemia) strain values\(^\text{10,20,28}\) using 2D-S, we performed our measurements using the Echopac software, exactly in the same way as it is routinely used for clinical studies. Therefore, we analysed regional myocardial function averaging one segment deformation, i.e. on a larger area than the region of interest corresponding to the crystal positions, whereas previous studies limited the size of region of interest.\(^\text{20}\) However, both SS and 2D-S could similarly detect myocardial function abnormalities induced by increase in afterload and differences in longitudinal and radial functions.

We focused our analysis on systolic function and did not explore the diastolic function since this open-chest and open-pericardium model modifies per se diastolic parameters.

Conclusion

Quantifying LV myocardial regional deformation in two orthogonal directions provides a better understanding of subclinical decrease in LV function. Longitudinal and radial LV functions are not similarly affected by afterload variations.

Author’s contribution

E.D.: conception, performance of the echocardiography, and review of the results and the paper; C.B.: review of the echocardiographic exams and writing of the paper; H.T.: assisting in conception and writing and reviewing of the paper; L.E.: assisting the performance of the exams and in the reviewing; J.L.: assisting during the experiments; M.O.: assisting in the performance of the study and reviewing of the paper; G.D.: reviewing of the paper and assistance all over the study.

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Conflict of interest: none declared.

References


