Right ventricular free wall circumferential strain reflects graded elevation in acute right ventricular afterload

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Traditionally, regional myocardial function can be assessed via echocardiography and magnetic resonance imaging (MRI). The aim of this experimental study was to improve the understanding of alterations in regional RV function to a better understanding of alterations in regional RV function and can be measured directly by echocardiographic Doppler tissue imaging (30). However, the angle dependency of the Doppler method and the complexity of RV diastolic function (31, 32). Using the same concept, we also assessed the eccentricity index for the RV. RV and LV myocardial performance indexes (MPI) were calculated by using a previously derived formula (31, 32).

AN ACUTE INCREASE in right ventricular (RV) afterload is reflected by RV dysfunction. However, the existing quantitative approaches to RV function evaluation are typically limited to analysis of global rather than regional RV systolic function (6). Even though the McConnell et al. (17) sign represents regional RV wall motion evaluation, it has been considered an indirect measure (14). Recently, Jamal et al. (13) have demonstrated that strain can quantitate regional RV function under various loading conditions and can be measured directly by echocardiographic Doppler tissue imaging (30). However, the angle dependency of the Doppler method and the complexity of RV shape limit this approach. Consequently, only a few reports about RV longitudinal strain during RV overload exist (6, 18).

The aim of this experimental study was to improve the noninvasive characterization of RV contractility and contribute to a better understanding of alterations in regional RV function under various levels of acutely increased RV systolic pressure (RVSP). In preliminary tests, we confirmed that the RV of a pig was thick enough to provide speckle patterns suitable for motion tracking and regional strain analysis.

We hypothesized that 1) the RV free wall (RVFW) strain will reflect an acute gradual increase in RVSP and 2) the change in the RVFW strain will be more marked compared with a change in strain within the interventricular septum (IVS) because the IVS is affected by both left ventricle (LV) and RV loading conditions.

MATERIALS AND METHODS

Animal Preparation

The experimental protocol was approved by the Institutional Animal Care and Use Committee. Fourteen pigs (11 males, 43 ± 4 kg) were anesthetized and mechanically ventilated. Pressure in the aorta, LV, RV, and right atrium were measured with high-fidelity catheters. After the animal’s chest was opened, an external pneumatic cuff occluder was placed around the pulmonary artery. The pericardium was repaired and preservation of baseline hemodynamics verified.

Standard Echocardiography

Epicardial echocardiograms were obtained with a Vivid 7 scanner (GE Healthcare, Milwaukee, WI) and included measurements of parameters representative of conventional LV systolic and diastolic functions. An eccentricity index, a measure of the IVS displacement, was assessed in midshort-axis views at both end diastole and end systole as D2/D1, where D2 is the LV dimension parallel to the septum and D1 is the LV dimension perpendicular to D2 and bisecting the septum (24, 27). Using the same concept, we also assessed the eccentricity index for the RV. RV and LV myocardial performance indexes (MPI) were calculated by using a previously derived formula (31, 32).

Speckle Tracking Echocardiography

The endocardial border was traced at the end-systolic frame and an automated tracking algorithm (EchoPAC 7.0.0 for PC; GE Healthcare) outlined the myocardium. The calculated deformation (Fig. 1) was expressed as myocardial longitudinal, radial, and circumferential strain, and strain rate profiles were obtained and their peak systolic values measured at each gradual increase of RVSP. Time intervals from the starting point of the QRS wave on the electrocardiogram to the peak systolic strain or strain rate value were also measured at each level of RVSP.

Study Protocol

Scans and data were collected at baseline and during constrictions of the pulmonary artery graded to acute mild (>35 and ≤50 mmHg), moderate (≥50 and ≤60 mmHg), and severe (>60 mmHg) RV afterload (7, 25).

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Statistical Analysis

Data are expressed as means ± SD or as percentages where appropriate. One-way ANOVA with the post hoc Scheffe test was used for parametric comparisons of measurements obtained at baseline and during interventions. Linear regression analysis with the Pearson correlation coefficient was used to reveal relations between two continuous variables. All statistical analyses were performed with software (SPSS 12.0; SPSS, Chicago, IL), and a P value <0.05 was considered statistically significant.

Inter- and intraobserver variability. Two independent observers processed and measured images for 10 of the study animals for longitudinal, radial, circumferential strain, and strain rate separately. Each observer analyzed the same image two times. These results were compared, and correlation was found using Pearson’s correlation coefficients.

RESULTS

Baseline data were obtained from all 14 animals. Data at mild and moderate afterload were obtainable from 12 and 13 animals, respectively. Severe afterload obtainable data were limited to only six animals that tolerated this condition long enough to achieve a steady state.

RV and LV Hemodynamics

RV and LV hemodynamic data are shown in Table 1. Baseline RVSP was slightly above 30 mmHg because the occluder had to be placed tightly around the pulmonary artery. Both RV systolic and diastolic pressures increased through each stage of elevated afterload. LV systolic pressure was different at each intervention step, with a decreasing trend from baseline to moderate RV afterload when the difference with respect to baseline reached statistical significance.

RV and LV Function

RV and LV functional data are shown in Table 2. Both RV end-systolic and end-diastolic areas increased through the loading conditions, and this increase became statistically significant during severe afterload. Fractional area change of RV did not change significantly. There were no significant changes in LV ejection fraction and volume measurements. The LV eccentricity index increased in systole, whereas the RV eccentricity index decreased in systole with the exception of the severe overload condition. The RV eccentricity index in diastole also decreased with an increase of RV pressure overload.

Mitrall inflow and annular velocities, measured by Doppler echocardiography, did not show significant changes or trends with the interventions. There were no significant differences in tricuspid E and A components of inflow velocities or in their ratio, neither were there significant differences in tricuspid annular

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**Table 1. RV and LV hemodynamics at graded afterload**

|                  | Baseline (n = 14) | Mild (n = 12) | Moderate (n = 13) | Severe (n = 6) | P Value (ANOVA) |
|------------------|-------------------|--------------|-------------------|---------------|----------------
| RVSP, mmHg       | 31.0±4.3          | 41.1±2.7     | 52.7±3.4          | 61.7±1.6      | <0.001         |
| RVDP, mmHg       | 4.5±1.6           | 8.4±4.3      | 11.2±7.4          | 12.2±7.7*     | 0.008          |
| LVSP, mmHg       | 108.7±20.2        | 102.9±15.4   | 89.8±14.3†       | 98.1±18.5     | 0.005          |
| LVDP, mmHg       | 14.7±6.4          | 12.5±3.8     | 19.0±17.7         | 19.1±6.9      | 0.573          |
| HR, beats/min    | 86.2±16.0         | 92.7±15.0    | 88.4±13.4         | 94.3±14.7     | 0.589          |

Values are means ± SD; n, no. of pigs. DP, diastolic pressure; HR, heart rate; LV, left ventricle; RV, right ventricle; SP, systolic pressure. *P = 0.023 and †P = 0.024 vs. baseline.
velocities in systole or diastole. The MPI of both LV and RV were rather variable and showed no significant differences.

**RV Deformation**

Table 3 shows that there were no significant changes in RV deformation at increasing levels of afterload as measured by longitudinal and radial strains of the RVFW and IVS. Time to peak longitudinal and radial strains or strain rates of the RVFW and the IVS did not change either except for the time to peak radial strain rate of the RVFW, whose value obtained at the severe afterload condition was significantly prolonged compared with the moderate afterload. Table 4 presents circumferential RV deformation. There were no statistically significant differences in circumferential strain, strain rate, or time to peak circumferential strain and strain rate measured globally or regionally (in the RVFW and IVS). Traces in Fig. 2, A–C, demonstrate that the absolute RVFW circumferential strain values decreased from baseline to the moderate afterload condition, whereas Fig. 2, D–F, shows that circumferential deformation of the IVS was with no significant reduction under the same RVSP loading conditions. Data plot in Fig. 3 documents that absolute values of circumferential strains of the RVFW were continuously decreasing from baseline to the mild and moderate RVSP loading. However, from the moderate to severe level of loading, the mean value suggested a trend toward an increased absolute circumferential strain although the spread of values reflected a variable ability of the RV to withstand the severe level of RV afterload in those six animals that were able to at least temporarily tolerate this condition.

**Relation of RVFW and Global RV Circumferential Deformation to Changes in RVSP**

When the severe afterload condition was excluded, the RVFW circumferential strain correlated inversely with an increase in RVSP (Fig. 4A), whereas the time interval to peak global circumferential strain of the RV correlated directly but weakly with RVSP (Fig. 4B).

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**Table 2. RV and LV function at graded afterload**

<table>
<thead>
<tr>
<th></th>
<th>Baseline (n = 14)</th>
<th>Mild (n = 12)</th>
<th>Moderate (n = 13)</th>
<th>Severe (n = 6)</th>
<th>P Value (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RV</strong></td>
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</tr>
<tr>
<td>RVAs, cm²</td>
<td>4.4 ± 1.4</td>
<td>4.8 ± 1.4</td>
<td>5.4 ± 1.3</td>
<td>7.7 ± 2.3</td>
<td>0.001</td>
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<tr>
<td>RVAd, cm²</td>
<td>8.3 ± 1.7</td>
<td>8.0 ± 2.1</td>
<td>9.1 ± 2.2</td>
<td>12.1 ± 1.8</td>
<td>0.001</td>
</tr>
<tr>
<td>FAC, %</td>
<td>47.1 ± 11.7</td>
<td>38.9 ± 11.5</td>
<td>39.4 ± 8.2</td>
<td>36.7 ± 15.0</td>
<td>0.144</td>
</tr>
<tr>
<td>Systolic eccentricity index</td>
<td>4.1 ± 1.6</td>
<td>3.3 ± 1.3</td>
<td>2.7 ± 0.8</td>
<td>3.0 ± 2.1</td>
<td>0.044†</td>
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<tr>
<td>Diastolic eccentricity index</td>
<td>3.4 ± 0.8</td>
<td>3.1 ± 1.0</td>
<td>2.6 ± 0.7</td>
<td>2.4 ± 0.8</td>
<td>0.039</td>
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<tr>
<td><strong>LV</strong></td>
<td></td>
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<td>LVEF, %</td>
<td>56.7 ± 10.3</td>
<td>53.3 ± 13.6</td>
<td>54.8 ± 11.4</td>
<td>58.0 ± 11.0</td>
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<td>LVVs, ml</td>
<td>18.3 ± 6.2</td>
<td>14.6 ± 4.7</td>
<td>14.5 ± 5.8</td>
<td>16.2 ± 5.6</td>
<td>0.282</td>
</tr>
<tr>
<td>LVDd, ml</td>
<td>43.3 ± 17.3</td>
<td>33.8 ± 13.3</td>
<td>31.9 ± 9.4</td>
<td>38.3 ± 6.4</td>
<td>0.110</td>
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<tr>
<td>Systolic eccentricity index</td>
<td>1.03 ± 0.11</td>
<td>1.05 ± 0.13</td>
<td>1.18 ± 0.14</td>
<td>1.20 ± 0.17</td>
<td>0.008</td>
</tr>
<tr>
<td>Diastolic eccentricity index</td>
<td>1.05 ± 0.13</td>
<td>1.00 ± 0.06</td>
<td>1.10 ± 0.12</td>
<td>1.13 ± 0.20</td>
<td>0.096</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of pigs. Ad, end-diastolic area; As, end-systolic area; EF, ejection fraction; FAC, fractional area change; Vd, ventricular end-diastolic volume; Vs, ventricular end-systolic volume. *P < 0.05 vs. other overload conditions. †P value except severe afterload condition.

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**Table 3. RV longitudinal and radial deformation at graded afterload**

<table>
<thead>
<tr>
<th></th>
<th>Baseline (n = 14)</th>
<th>Mild (n = 12)</th>
<th>Moderate (n = 13)</th>
<th>Severe (n = 6)</th>
<th>P Value (ANOVA)</th>
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<td><strong>Longitudinal movement</strong></td>
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<tr>
<td>FW Strain, %</td>
<td>−33.8 ± 5.5</td>
<td>−27.4 ± 8.6</td>
<td>−29.8 ± 11.6</td>
<td>−25.8 ± 12.2</td>
<td>0.227</td>
</tr>
<tr>
<td>Strain rate, s⁻¹</td>
<td>−2.0 ± 0.4</td>
<td>−2.1 ± 0.7</td>
<td>−2.1 ± 1.0</td>
<td>−1.8 ± 0.6</td>
<td>0.802</td>
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<tr>
<td>Time to peak strain, ms</td>
<td>385 ± 63</td>
<td>379 ± 135</td>
<td>387 ± 84</td>
<td>358 ± 159</td>
<td>0.951</td>
</tr>
<tr>
<td>IVS Strain, %</td>
<td>−16.1 ± 6.2</td>
<td>−14.5 ± 4.7</td>
<td>−16.2 ± 5.6</td>
<td>−17.7 ± 6.2</td>
<td>0.698</td>
</tr>
<tr>
<td>Strain rate, s⁻¹</td>
<td>−1.2 ± 0.8</td>
<td>−1.1 ± 0.5</td>
<td>−1.2 ± 0.5</td>
<td>−1.4 ± 0.6</td>
<td>0.767</td>
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<tr>
<td>Time to peak strain, ms</td>
<td>378 ± 146</td>
<td>392 ± 76</td>
<td>385 ± 90</td>
<td>413 ± 94</td>
<td>0.932</td>
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<tr>
<td><strong>Radial movement</strong></td>
<td></td>
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<tr>
<td>FW Strain, %</td>
<td>48.6 ± 24.6</td>
<td>49.3 ± 19.9</td>
<td>41.8 ± 22.2</td>
<td>47.7 ± 27.3</td>
<td>0.838</td>
</tr>
<tr>
<td>Strain rate, s⁻¹</td>
<td>2.1 ± 0.6</td>
<td>1.9 ± 0.5</td>
<td>1.6 ± 0.4</td>
<td>1.8 ± 0.5</td>
<td>0.218</td>
</tr>
<tr>
<td>Time to peak strain, ms</td>
<td>386 ± 52</td>
<td>406 ± 49</td>
<td>391 ± 75</td>
<td>416 ± 91</td>
<td>0.733</td>
</tr>
<tr>
<td>IVS Strain, %</td>
<td>28.9 ± 12.9</td>
<td>30.1 ± 10.0</td>
<td>30.9 ± 10.4</td>
<td>31.3 ± 10.0</td>
<td>0.958</td>
</tr>
<tr>
<td>Strain rate, s⁻¹</td>
<td>1.3 ± 0.6</td>
<td>1.3 ± 0.4</td>
<td>1.3 ± 0.4</td>
<td>1.2 ± 0.4</td>
<td>0.913</td>
</tr>
<tr>
<td>Time to peak strain, ms</td>
<td>363 ± 102</td>
<td>371 ± 44</td>
<td>380 ± 62</td>
<td>356 ± 48</td>
<td>0.896</td>
</tr>
<tr>
<td>Time to peak strain, ms</td>
<td>179 ± 104</td>
<td>186 ± 150</td>
<td>104 ± 71</td>
<td>151 ± 102</td>
<td>0.290</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of pigs. FW, free wall; IVS, interventricular septum. *P = 0.044 vs. moderate overload condition.
Relation of RVFW Deformation Rate to Changes in RVSP

Radial strain rate of the RVFW showed significant but weak inverse linear correlation with RVSP when the severe afterload condition data were not considered ($r = 0.382, P = 0.011$).

Inter- and intraobserver variability. Interobserver variability was tested using 10 randomly selected loops by a second observer, revealing a good correlation between two independent observers measured by results for longitudinal, radial, and circumferential strain and strain rate ($r = 0.832$). The correlation within observer was also good ($r = 0.884$).

DISCUSSION

Our experimental study shows that acutely increasing RVSP to the mild and moderate afterload levels is functionally reflected in the RVFW (but not in the IVS) by a gradually decreasing magnitude of circumferential strain, whereas longitudinal and radial strains are not significantly changed. However, with a further increase in RVSP to a severe afterload level (i.e., RVSP exceeding 60 mmHg), the functional response becomes variable, ranging from an immediate RV failure to at least a temporary (~20 min in our tests), functional tolerance
RV Architecture and Motion

The RV is structurally and functionally divided into the following two parts: the inflow tract (IT; i.e., basal level of the RV) and outflow tract (OT) separated by crista supraventricularis (8). The IT is mainly composed of transverse fibers in the subepicardium and longitudinal fibers in the subendocardium, whereas the OT is composed of longitudinal fibers in both the subendocardial and subepicardial layers (10). The fibers in the IT region produce systolic longitudinal shortening and a radial motion, complemented by the resulting circumferential shortening (4). The fibers in the OT region produce an overall peristaltic RV motion (20).

There is evidence of crisscross oblique fibers within the apical IVS (1, 10). Some of these oblique fibers also contribute to the morphology of the RVOT (22). The fact that the IVS does not have a predominant RV or LV side is a critical aspect of this spatial geometry. Rather, the IVS is the central structure composed of the same crisscross oblique fibers as those within the LV free wall (12). This oblique fiber orientation of the IVS affords a greater ejection fraction for both the RV and LV (22), but, as a consequence, radial and circumferential movements of the IVS might be underrepresented compared with the RVFW.

Role of the Free Wall of the RV Function

RV performance is not significantly impaired by either cauterization of the entire RVFW (29) or replacement of the RVFW with a patch material (28) as long as the septum remains intact, but the crescent-shaped, thin RVFW gives the RV much greater compliance than the LV. These functional and anatomic characteristics explain significant RV dilatation in response to acutely increased afterload (3, 15). In our study, the systolic RV eccentricity index decreases with mild and moderate RV afterload (Table 2). This finding is consistent with enlargement of the RV mainly in the transverse direction (i.e., perpendicularly to the IVS) because of the RVFW compliance and explains the corresponding decreases of RVFW circumferential deformation accomplished by the transverse fibers (Fig. 5).

New Concepts for Assessing RV Function

In 2003, Jamal and colleagues (13) demonstrated in open-chest pig experiments that longitudinal deformation measurements derived from ultrasonic strain rate imaging allow accurate analysis and noninvasive quantification of regional RV contractile function under varying loading conditions by constraining the pulmonary artery (just as in our experiments). Unlike in the study by Jamal et al. (13), in our tests longitudinal deformation of the RV did not change significantly after pulmonary artery constriction (Table 3). The decrease in circumferential deformation of the RVFW was the only response to acute gradual increases in RV afterload. We propose a possible reason for this discrepancy. The measured segment of RVFW was different from ours. Instead of measuring IT and OT of the RV, we measured midlevel of the RVFW because the IT of the RVFW is connected with the IVS and the fibrous tricuspid annulus, whereas the OT is composed mainly of longitudinal fibers. Thus the movement of the IT cannot independently reflect RV afterload conditions, and OT deformation cannot represent transverse fiber motion.

Hori et al. (11) suggested that, from tissue Doppler data, both the velocity of the tricuspid annular leaflet in systole and the MPI are representative of RV contraction function. Ruan and Nagueh (26) reported a significant decrease of septal and tricuspid annular early diastolic velocities in idiopathic pulmonary hypertension. However, in that study, the mean value of pulmonary pressure in idiopathic pulmonary hypertension patients was >70 mmHg (i.e., extremely high), and the patient group was compared with a normal control group. In our study, the conventional parameters directly associated with global RV function did not change (Table 2). Although routinely used for analysis of global RV function, neither the mitral annular septal and lateral tissue Doppler velocities nor tricuspid annular tissue velocities in systole and diastole showed significant changes during acute increases in RV afterload (data not shown); therefore, these measurements could not be considered as reflecting the tested increases in RV afterload. Given the fact that strain and strain rate measurements of the RV wall by tissue Doppler echocardiography are best performed from the apical four-chamber view, the approach is limited to functional quantification of predominantly longitudinal RV fibers. Lindqvist et al. (16) suggested a clinical utility of 2D (i.e., speckle tracking-based) strain for evaluation of RV function. Edvardsen et al. (9) showed that strain measured by speckle tracking closely reflects the actual myocardial deformation present. Although used mainly for the evaluation of LV function, the speckle tracking technique is also used in the thin-walled RV (2, 21).
Rationale for Measuring Deformation of the Midlevel of the RV Wall During RV Pressure Overload

The basal level of the RVFW is connected with the IVS and the fibrous tricuspid annulus (22). Thus the movement of the basal RVFW cannot independently reflect deformation of the RVFW under changing afterload conditions. The distribution of the adrenergic receptors in the RV wall is not entirely clear, but, if there is a similarity to the LV in this regard, the distribution density of the receptors would be higher in the RV base than in the apex (20), and the basal RVFW would not represent the RVFW response in an acute setting of RV pressure overload.

Functional RV Responses to Pressure Overload

We demonstrated that the longitudinal and radial movements of both the RVFW and the IVS did not change significantly (Table 3), and circumferential strain and strain rate of the IVS and global RV wall did not significantly change either (Table 4) with an increase in RV afterload. The main role of the RVFW is supplying compliance to the RV rather than maintaining its mechanical performance. That explains the functional response of the mid-RVFW to changes in RV pressure loading and the lack of such a response within the other RV wall segments. The functional response of the mid-RVFW is demonstrated in our study with regard to circumferential strains, whose absolute values decreased with progression of RV afterload to mild and moderate conditions (Figs. 2, A–C and 3) and correlated closely with changes in RVSP (Fig. 4A). The time interval between the electrical starting point of a QRS wave on the electrocardiogram and the peak magnitude of global RV systolic circumferential strain were positively cor-

(Severe afterload excluded)

Fig. 4. Linear regression between RV systolic pressure (RVSP) and the RVFW circumferential strain and between RVSP and the time to peak global RV circumferential strain with exception of severe afterload condition. A: the absolute value of circumferential strain of the RVFW has a significant negative linear correlation with RVSP. B: with an increase in RVSP, the RVFW time to peak global circumferential strain delay. SP, systolic pressure.

Fig. 5. Mechanism of decrease in circumferential deformation of midlevel of RVFW. Increase of RV afterload in systolic period enhance the main role of the RVFW, supplying compliance to the RV rather than maintaining its mechanical performance by enlarging toward transverse direction opposite to IVS and eventually decrease the circumferential deformation accomplished by epicardial transverse fiber.
related with RVSP (Fig. 4B). This suggests that, with an increase in RVSP, it took proportionally longer to achieve the peak global systolic circumferential deformation. We explain the results of our study on a basis of interaction of adjacent muscle fibers of myocardium, i.e., using a similar physiological concept as in work by Claus and colleagues (5), but we have to account for the effect of RV pressure overload: Subepicardial transverse fibers were subject to an increased internal pressure developed in systole and acting through the subendocardial longitudinal myocardial muscle fibers (Fig. 5). As a result, the subepicardial fibers elongated in transverse direction and resulted in a decreased systolic circumferential strain.

The statistically significant but weak negative linear correlation between radial strain rate and RVSP ($r = -0.382, P = 0.011$) in our results may be explained by the fact that radial movement of the RVFW is accomplished not only by transverse fiber shortening but also by shortening of oblique fibers (23).

To our knowledge, there are no data about RVFW circumferential changes occurring in response to acute alterations in RV loading conditions. In 2007, Pettersen and colleagues (19) evaluated changes in RV regional wall mechanics in patients with chronically increased RV preload and afterload. The investigators demonstrated a shift in contraction patterns from longitudinal to predominantly circumferential shortening compared with the normal RV, without any differences in IVS shortening patterns. The predominantly circumferential free wall shortening may have represented an adaptive response to the systemic load. There was a relative increase in transverse fiber mass in that chronic setting and may have contributed to the predominant circumferential RVFW shortening. Thus, based on previous results (19) and our study results, RVFW circumferential movement appears to be a sensitive adaptive mechanism occurring in response to acute (decreasing circumferential shortening) and chronic (increasing circumferential shortening) increase in RV afterload. Based on Ref. 19 only, we speculate that RV circumferential strain changes would sensitively indicate changes in alteration of RV preload conditions.

Limitations

We do not provide comparisons with radionuclide angiography, magnetic resonance imaging, or three-dimensional echocardiography of the RV. However, these methods do not necessarily represent a gold standard and have their own limitations.

There are no data available from all animals for severe RV afterload because only 6 out of 14 pig hearts were able to tolerate the high RVSP condition.

Clinical Implications

In our study, the RVFW circumferential strain significantly decreased with the increasing grade of afterload severity. This suggests that circumferential strain could be a diagnostic parameter of clinically relevant acute elevations in RVSP loading. The change in circumferential movement of RV transverse fibers appears to reflect even a subtle increase in RVSP and could have utility in monitoring of the initial responses to therapy intended to reduce RV pressure overload. However, the study also implies that, at the severe level of RV afterload, circumferential strain is no longer an indicator of the functional status and that the preserved systolic deformation does not exclude an impending functional RV failure in the hearts that are capable of withstanding the severe loading conditions.

Conclusion

Measurement of circumferential strain of the RVFW by 2D speckle tracking can quantify regional RV dysfunction and noninvasively show acute gradual increases in RV afterload as long as its elevation does not reach the severe level.

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GRANT

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