Ventricular Geometry, Strain, and Rotational Mechanics in Pulmonary Hypertension

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**Background**—We tested the hypothesis that right ventricular (RV) pressure overload affects RV function and further influences left ventricular (LV) geometry, which adversely affects LV twist mechanics and segmental function.

**Methods and Results**—Echocardiographic images were prospectively acquired in 44 patients (age, 46±12 years; 82% women) with evidence of pulmonary hypertension (estimated pulmonary artery systolic pressure, 71±23 mm Hg) and in 44 age- and gender-matched healthy subjects. Patients with intrinsic LV diseases were excluded. RV lateral wall longitudinal strain (LS) and interventricular septal (IVS) LS were reduced in the pulmonary hypertension group compared with control subjects (−15.9±7.6% versus −25.5±6.1%, P<0.001; and −17.3±4.4% versus −20.2±3.9%, P=0.002, respectively), whereas LV lateral wall LS was preserved. RV lateral wall LS and IVS LS, but not LV lateral wall LS, correlated with pulmonary artery systolic pressure (r=0.56, P<0.01; r=0.32, P<0.01) and LV eccentricity index (r=0.57, P<0.01; r=0.57, P<0.01). IVS and LV lateral wall circumferential strain (CS) were both reduced in the pulmonary hypertension group. Although IVS CS and LV lateral wall CS correlated with pulmonary artery systolic pressure and LV eccentricity index, after adjustment of CS for LV eccentricity index, differences between groups persisted for IVS CS (P<0.01) but not LV lateral wall CS (P=0.09). LV torsion was decreased in patients with pulmonary hypertension compared with control subjects (9.6±4.9° versus 14.7±4.9°, P<0.001). LV torsion inversely correlated with pulmonary artery systolic pressure (r=−0.39, P<0.01) and LV eccentricity index (r=−0.3, P<0.01). LV untwisting rates were similar in both groups (P=0.7).

**Conclusions**—Chronic RV pressure overload directly affects RV longitudinal systolic deformation. RV pressure overload further influences IVS and LV geometry, which impairs LV torsion and segmental LS and CS, more for the IVS than for the free wall of the LV. (Circulation. 2010;121:259-266.)

**Key Words:** echocardiography ▪ hypertension, pulmonary ▪ myocardial contraction ▪ torsional force
had evidence of PAH defined as peak PASP >35 mm Hg estimated by Doppler echocardiography.6,7 We excluded participants with abnormal LV end-diastolic diameter >5.5 cm, LV wall thickness >1.5 cm, prior myocardial infarction, uncontrolled hypertension, significant aortic/mitral valve disease, and age >65 years because we thought that these patients might have alterations in LV function that were independent of their PH. Of 51 patients and family members, 44 met the study criteria and were included in the study. Of 44 patients, 42 had PH diagnosed previously by right heart catheterization. These 44 subjects constituted a heterogeneous group that included 16 patients with idiopathic PAH, 10 patients with familial PAH, 11 patients with PAH associated with connective tissue diseases, 4 patients with PH associated with sleep apnea disorders, 1 patient with PH associated with sarcoidosis, and 2 family members of patients with familial PAH who had estimated PASP >35 mm Hg. Similar echocardiographic data from age- and gender-matched 44 healthy control subjects were collected for comparison.

The study protocol was approved by the Cleveland Clinic Institutional Review Board.

Echocardiographic Study
All 44 participants with PH and 44 control subjects underwent a complete transthoracic echocardiographic study, including 2-dimensional, color flow, and spectral Doppler and high-frame-rate (70- to 140-Hz) imaging with a GE-Vingmed Vivid 7 system (GE-Vingmed, Horten, Norway). Agitated saline was used to enhance tricuspid regurgitation (TR) spectral Doppler in subjects with PH who had weak spectral Doppler signals.

Echocardiographic Analysis
PASP was estimated from the systolic pressure gradient between the RV and the right atrium by the peak continuous-wave Doppler velocity of the TR jet using the modified Bernoulli equation plus right atrial pressure estimated from the inferior vena cava size and collapsibility with respiration, as previously validated and described by others.6,8 RV end-diastolic and end-systolic areas were measured in the apical 4-chamber view by tracing the endocardial border of the RV and the tricuspid annular plane.8,9–11 RV fractional area change was calculated as follows: RV end-diastolic area minus RV end-systolic area divided by RV end-diastolic area.

In the apical 4-chamber view, the mean of these 2 strain measurements was considered positive; clockwise rotation was deemed negative. LV torsion was calculated as the maximal difference between the apical and basal rotation curves during systole.13,15 That maximal difference per second during early diastole was calculated as the LV untwisting rate.13,15

The digitally stored echocardiographic data were analyzed by an experienced sonographer and cardiologist who were unaware of patient’s clinical characteristics. All measurements represent an average of 3 to 5 consecutive cycles.

Reproducibility
The same 2 observers analyzed echocardiographic data from a nested case group of 10 randomly selected patients and control subjects, respectively. Measurements of RV LAT LS by the same observer and independent observers showed a mean±SD difference of 0.4±1.5% and 0.2±1.6%, respectively. Intraclass correlation coefficients between the same observer and 2 different observers were 0.92 and 0.93, respectively (P<0.001). Measurements of LV torsion by 2 independent observers showed a mean difference by the same observer of 0.03±1.74° and 0.30±1.58°, respectively. Intraclass correlation coefficients between the same observer and 2 different observers were 0.88 and 0.93, respectively (P<0.001).

Statistical Analysis
Continuous data are presented as mean and SD or median (range). Categorical data are presented as frequency. Comparisons of categorical and continuous variables between subjects with PH and control subjects were performed by the McNemar test and paired t test, respectively. Correlations between echocardiographic parameters and estimated PASP or LV end-diastolic eccentricity index were tested by linear regression analysis and Pearson correlation coefficients. The correlation coefficients were further compared among septal, LV lateral, and RV lateral (if applicable) walls by using SISA online software.16 ANCOVA was performed to test whether certain factors (PH and control subjects) have an effect after removing the variance for which quantitative covariates (peak PASP and LV end-diastolic eccentricity index) account. Statistical significance was defined as P<0.05.

Results
Clinical and echocardiographic characteristics in the PH and control groups are shown in Table 1. The mean age of participants with PH was 46±12 years; 82% were female. Participants with PH had a significantly larger RV, increased RV free wall thickness, impaired gross RV systolic and diastolic function, increased LV eccentricity index, and lower LV end-systolic and end-diastolic volume indexes. Estimated peak PASP in the PH group was 71±23 mm Hg. Of the participants with PH, 66% (29 of 44) had estimated peak PASP >60 mm Hg.

Relationship Between Estimated PASP and Biventricular Function
Estimated PASP Versus Biventricular LS
Patients with PH had significantly reduced (lower absolute values) RV LAT LS (−15.9±7.6% versus −25.5±6.1%; P<0.001) and IVS LS (−17.3±4.4% versus −20.2±3.9%; P=0.002) compared with control subjects, whereas LV LAT LS was similar between PH patients and control subjects (−18.0±5.2% versus −19.4±3.4%; P=0.24; Table 2). Regression analysis (Figure 1A, 1B, and 1C) showed that, after midpapillary muscle, and apical level), end-systolic circumferential strain (CS) curves were obtained. As with LS, peak CS was averaged from each level of the 2 septal segments and the 2 free wall segments to yield IVS CS and LV LAT CS. LV rotation in the 3 short-axis planes was determined as the average rotation of the 6 myocardial segments around the central axis. Counterclockwise rotation (as viewed from the apex) was considered positive; clockwise rotation was deemed negative. LV torsion was calculated as the maximal difference between the apical and basal rotation curves during systole.13,15 That maximal difference per second during early diastole was calculated as the LV untwisting rate.13,15
Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th></th>
<th>PH Patients (n=44)</th>
<th>Control Subjects (n=44)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>46±12 (45, 18–64)</td>
<td>46±12 (45, 8–61)</td>
<td>0.90</td>
</tr>
<tr>
<td>Female, n</td>
<td>36</td>
<td>36</td>
<td>0.79</td>
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<tr>
<td>Diabetes mellitus, n</td>
<td>2</td>
<td>0</td>
<td>0.17</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>115±17 (114, 85–140)</td>
<td>118±14 (114, 96–131)</td>
<td>0.44</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>65±11 (66, 45–88)</td>
<td>69±10 (69, 45–88)</td>
<td>0.32</td>
</tr>
<tr>
<td>Estimated pulmonary systolic pressure, mm Hg</td>
<td>71±23 (71, 37–125)</td>
<td>23±4 (22, 17–31)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RV end-diastolic area, cm²</td>
<td>26.3±9.5 (25, 12–49)</td>
<td>12.3±3.5 (12, 3–22)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RV end-systolic area, cm²</td>
<td>18.3±8.7 (16, 6–40)</td>
<td>5.8±1.9 (6, 2–10)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RV fractional area change, %</td>
<td>32±14 (32, 19–61)</td>
<td>60±13 (53, 25–96)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Tricuspid annular plane systolic excursion, cm</td>
<td>1.6±0.5 (1.6, 0.6–2.8)</td>
<td>2.4±0.5 (2.5, 1.1–3.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Right atrial volume index, mL/m²</td>
<td>35±21 (27, 11–92)</td>
<td>14±5 (13, 6–29)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV end-systolic area, cm²</td>
<td>9.7±3.0 (10, 4–19)</td>
<td>3.7±0.9 (4, 2–6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV end-systolic eccentricity index</td>
<td>1.55±0.69 (1.16, 0.84–2.13)</td>
<td>1.03±0.21 (0.94, 0.76–2.11)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV end-diastolic eccentricity index</td>
<td>1.24±0.32 (1.39, 0.47–3.50)</td>
<td>0.96±0.09 (1.04, 0.67–2.11)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>67±12 (65, 42–92)</td>
<td>63±9 (61, 47–78)</td>
<td>0.043</td>
</tr>
<tr>
<td>Mitral E velocity, cm/s</td>
<td>77±27 (76, 28–192)</td>
<td>76±16 (76, 37–112)</td>
<td>0.836</td>
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<td>Mitral E deceleration time, ms</td>
<td>200±63 (189, 107–349)</td>
<td>186±30 (183, 130–260)</td>
<td>0.202</td>
</tr>
<tr>
<td>Mitral E/A</td>
<td>1.14±0.46 (1.05, 0.50–2.50)</td>
<td>1.40±0.40 (1.41, 0.70–2.80)</td>
<td>0.015</td>
</tr>
<tr>
<td>LV end-diastolic volume, mL</td>
<td>65±26 (63, 14–135)</td>
<td>75±25 (70, 40–164)</td>
<td>0.078</td>
</tr>
<tr>
<td>LV end-diastolic volume index, mL/m²</td>
<td>35±13 (33, 8–62)</td>
<td>43±12 (42, 25–75)</td>
<td>0.001</td>
</tr>
<tr>
<td>LV end-systolic volume, mL</td>
<td>21±11 (21, 3–50)</td>
<td>28±12 (27, 10–75)</td>
<td>0.009</td>
</tr>
<tr>
<td>LV end-systolic volume index, mL/m²</td>
<td>11±5 (11, 2–23)</td>
<td>16±6 (16, 6–39)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stroke volume, mL</td>
<td>44±18 (41, 8–85)</td>
<td>47±18 (44, 22–124)</td>
<td>0.440</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>77±14 (79, 47–112)</td>
<td>70±9 (70, 48–91)</td>
<td>0.003</td>
</tr>
</tbody>
</table>

BP indicates blood pressure.

patients and control subjects were combined, the correlation between estimated peak PASP and strains was stronger for RV LAT LS (r=0.56, P<0.001) than for IVS LS (r=0.32, P=0.007 versus RV LAT LS) and lower still for LV LAT LS (r=0.06, P=0.027 versus IVS LS). After adjustment for estimated peak PASP as a covariate (Table 2), the differences between control subjects and patients were no longer significant, indicating that the differences in LS between the 2 groups were due primarily to the presence of PH.

**Estimated PASP Versus LV CS**

Patients with PH had significantly reduced IVS CS (−17.6±5.8% versus −23.1±4.87%; P<0.001) and LV LAT CS (−11.9±5.0% versus −14.5±6.1%; P=0.002; Table 2). Regression analysis (Figure 2A and 2B) showed that, after patients and control subjects were combined, the correlation between estimated peak PASP and CS was stronger with IVS LS (r=0.60, P<0.001) than with LV LAT CS (r=0.28, P<0.001 versus IVS LS). After adjustment for estimated peak PASP as a covariate, the differences between groups disappeared for both IVS CS (P=0.90) and LV LAT CS (P=0.09), again indicating that the differences in CS between the 2 groups are due primarily to the presence of PH.

**Estimated PASP Versus LV Torsion**

Finally, torsion was also significantly decreased in patients with PH compared with control subjects (9.6±4.9° versus 14.7±4.9°; P<0.001) and showed a moderately strong correlation with estimated peak PASP (r=-0.39, P<0.001; Figure 3). However, even after adjustment for estimated peak PASP as a covariate, patients with PH had significantly lower torsion (P=0.008). LV untwisting rates were similar in both groups (−90.2±46.3°/s versus −91.1±41.0°/s; P=0.72) and show poor correlation with PASP (r=−0.10, P=0.40), even after adjustment for peak PASP as a covariate.

**Relationship Between Estimated PASP and LV Diastolic Geometry**

Patients with PH had significantly increased LV diastolic eccentricity index compared with control subjects (1.24±0.32 versus 0.96±0.09; P<0.0001; Table 2). There was a moderately strong correlation between estimated peak PASP and LV diastolic eccentricity index (r=0.63, P<0.0001).

**Relationship Between LV Diastolic Geometry and LV Function**

**LV Diastolic Geometry Versus LV LS**

Regression analysis (Figure 2E and 2F) showed that, after patients and control subjects were combined, the correlation between LV diastolic eccentricity index and LS was stronger with IVS LS (r=0.57, P<0.0001) than with LV LAT LS (r=0.10, P<0.0001 versus IVS LS).
between diastolic eccentricity index and CS was stronger with IVS CS ($r=0.52$, $P<0.0001$) than with LV LAT CS ($r=0.21$, $P=0.0024$ versus IVS CS). After adjustment for LV diastolic eccentricity index as a covariate, differences between groups persisted for IVS CS ($P=0.0001$) but not LV LAT CS ($P=0.09$).

**LV Diastolic Geometry Versus LV Torsion**

Torsion showed a weak inverse correlation with LV diastolic eccentricity ($r=-0.30$, $P=0.006$; Figure 3). Adjusting for LV diastolic eccentricity index did not eliminate the effect of a PH diagnosis on LV torsion ($P=0.0004$). LV untwisting showed poor correlation with LV diastolic eccentricity index ($r=-0.08$, $P=0.5$), even after adjustment for peak LV diastolic eccentricity index as a covariate.

**Discussion**

The major findings of our study are the following: (1) RV free wall LS and IVS LS were significantly reduced in patients with PH, although LV LAT LS was preserved; (2) IVS CS and LV LAT CS, but not LS, were significantly reduced in patients with PH, but the magnitude of reduction was greater with IVS CS; and (3) LV systolic torsion was significantly reduced in patients with PH, whereas the LV early untwisting rate was similar in both groups.

### Chronic RV Pressure Overload and RV Longitudinal Deformation

Unlike the LV, the RV does not tolerate systemic arterial pressures because of its thinner wall and higher compliance. Chronic RV pressure overload leads to a gradual change in RV sarcomeres and myocytes, resulting in structural and functional deterioration. In our study, we demonstrated that in patients with chronic PH, RV longitudinal deformation is significantly reduced in magnitude compared with control subjects. Furthermore, we demonstrated that the severity of PH significantly correlates with the degree of RV strain reduction. These findings agree with the previous report by Pirat and colleagues, who showed impaired RV peak systolic strain (assessed by velocity vector imaging) in 58 patients with PH compared with 19 control subjects. Similar to our study, their study found that the PASP estimated by TR Doppler velocities was the best predictor of RV strain ($r=0.61$, $P<0.05$). Before the development of speckle tracking echocardiography, several investigators used tissue Doppler imaging to demonstrate reduced RV strain in patients with PH. Although tissue Doppler imaging has significant limitations caused by angle dependency, noise, and tethering, the major findings of those studies are in accordance with the present study. They also reported that RV LAT LS was significantly reduced in PH patients and correlated with pulmonary systolic pressures. Notably, our study further analyzed the data by adjusting LS using estimated PASP as a covariate. Once adjusted for PASP, the differences between control subjects and patients were no longer significant, suggesting that the differences in RV LAT LS between 2 groups are due predominantly to the presence of PH.

### Chronic RV Pressure Overload and Biventricular Geometric Alteration

Under normal conditions, the RV forms a crescent wrapped around the LV. In the milieu of PH, the RV is characterized by increased end-diastolic area and volume, a change in geometry to more spherical conformation, and various degrees of RV hypertrophy. We have confirmed that PH not only impairs RV function but also alters RV geometry. In our study, RV area and RV free wall thickness were significantly increased in patients with PH. Previous studies have reported that marked RV dilatation, increased RV pressures, and increased transseptal pressure gradient cause bowing of the IVS toward the LV as a result of anatomic contiguity of the 2 ventricles with a shared IVS within the restricted intrapericardial space, known as ventricular interdependence. As a result, septal and LV geometry is distorted by a decreased septal-free wall dimension and, conversely, increased anterior-posterior dimension. The elevated LV end-diastolic eccentricity index, ie, more D-shaped LV, in our PH group confirms the findings of Raymond et al and Manzel et al, who also reported increased LV end-diastolic eccentricity index in patients with PH. In this study, we found that LV end-systolic volume also was significantly decreased in the PH group. Previous studies supported the theory that encroachment on the LV by the IVS could impair LV filling if the increase in D1 was a result of leftward septal displacement. It was clearly shown that the remodeling in RV geometry and wall thickness, septal configuration, and LV geometry, and hence volume, accompanies the increase in RV pressure load.
In the present study, IVS LS was significantly reduced in patients with PH compared with control subjects, whereas LV LAT LS was maintained, findings that are concordant with those of Lopez-Candales and colleagues. They assessed RV LAT LS, IVS LS, and LV LAT LS by tissue Doppler imaging in PH patients and control subjects, showing that LV LAT LS was similar between the PH group and control subjects, whereas RV LAT LS and IVS LS were significantly impaired in patients with PH. Similarly, Huez et al studied LS by tissue Doppler imaging technique in 18 PH patients and 14 control subjects, showing LV LAT LS to be preserved. However, in contrast to our study and that of Lopez-Candales et al, the IVS LS was not reduced in their PH group.
In this study, we demonstrated the reduction in IVS LS to correlate significantly with both PASP and the LV diastolic eccentricity index. This implies a close relationship between septal geometry and IVS LS. Moon et al. elegantly demonstrated that septal configuration changes, including leftward shift, flattening, and thickening, can cause abnormal septal function and reduced septal output in the setting of reduced septal/LV preload. Their results support the idea that septal functional alteration is closely linked with septal geometric alteration and vice versa. In our cohorts, we found that the differences in IVS LS between the 2 groups are due mainly to PH and altered LV geometry and are not a primary abnormality of septal contractility. Strain represents a part of the stress-strain relationships, which define myocardial contractility.13,24–26 Thus, strain can be decreased even in the setting of normal contractility if regional or global stress (ie, afterload) is elevated. This is even more pronounced in the setting of RV circulation, which is especially sensitive to afterload elevation.13,25,26

LV Circumferential Deformation

In contrast to longitudinal deformation, both IVS CS and LV LAT CS were reduced in PH patients. However, the correlation between estimated PASP and CS was significantly stronger for the IVS than the LV LAT. This supports the finding that PH influences the IVS more than the LV LAT. The mechanism of impaired LV LAT CS, but preserved LV LAT LS, was unclear. A potential explanation could be that reverse septal curvature and a more D-shaped LV in patients with PH affect circumferential more than longitudinal myocardial fiber shortening. Wang et al. demonstrated that CS was preserved and LS was impaired in patients with diastolic heart failure. This implies that LS and CS do not necessarily vary in parallel. It also is reasonable that abnormalities in circumferential fiber function could propagate farther from the septum than longitudinal fibers. Nevertheless, previous studies have shown that longitudinal rather than circumferential shortening plays a major contribution of global LV systolic performance.28 Similar to estimated PASP, the correlation between LV diastolic eccentricity index and CS was also stronger with the IVS than the LV LAT. However, after adjustment for LV diastolic eccentricity as a covariate, differences between groups persisted for septal circumferential but not lateral CS.

LV Torsion and Untwisting Rate

It is well established that LV torsion is sensitive to changes in both regional and global LV dysfunction.13,26 However, our study has shown that LV torsion is significantly decreased in patients with chronic RV pressure overload, with moderate correlation to estimated PASP. However, significantly lower LV torsion persisted in patients with PH even after adjustment for estimated PASP and LV geometry as covariates. This may reflect decreased global preload present in the setting of PH. Supporting this postulate, Gan et al. showed that the LV filling rate measured by cardiac magnetic resonance imaging was significantly decreased in PH patients compared with control subjects. Furthermore, they found that the LV filling rate correlated significantly and inversely with leftward IVS curvature, confirming reduced LV preload in PH patients. The authors also concluded that the underfilled LV in the setting of PH could be due to either a decrease in RV output or a leftward displacement of IVS. Previous studies have investigated the hemodynamic determinants of LV torsion. Dong et al. demonstrated that preload, afterload, and contractility were predictors of LV torsion on multiple regression analysis. Furthermore, Gibbons Kroeker et al. studied the effect of preload, afterload, contractility, and heart rate on apical rotation. In their model, preload was a major determinant of apical rotation. In our study, we found that LV end-systolic and end-diastolic volume indexes were significantly lower in the PH group compared with the control group, suggesting reduced preload in patients with PH. These findings supported the idea that reduced LV preload in patients with PH may contribute to a significant reduction in LV torsion.

In addition to preload reduction, the interaction between myocardial fiber traction and RV dilation can be a potential explanation for reduced systolic torsion in PH group. It is known that subendocardial fibers form a right-handed helix and subepicardial fibers form a left-handed helix.29 Under normal physiological circumstances, epicardial traction dominates over endocardial movement during systole.25,31 Furthermore, there is evidence for a continuum of subepicardial RV muscle angles along the LV to the region of fibrous trigone.25 These suggest that if this balance of fiber rotation, especially in the septum, is altered and compressed by RV dilation in the setting of PH, the LV twist pattern might be affected. Thus, we believe that septal bowing and noncircular LV configuration may affect LV torque, resulting in a reduction in LV torsion in PH patients.

The LV untwisting rate provides additional noninvasive insight into LV elastic recoil and diastolic suction.13 Wang et al. demonstrated that LV untwisting rate was affected by
loading conditions. In their animal study, inferior vena caval occlusion led to a significant decrease in LV end-systolic volume and a significant increase in LV untwisting rate. Furthermore, the LV untwisting rate inversely correlated with LV end-systolic volume to compensate for LV filling. In our study, we found that LV untwisting rates were similar between the PH and control groups. The possible explanation for a lack of reduction in untwisting velocity in the PH group was most likely that early diastolic suction in the setting of reduced LV end-systolic volume in the PH group may be exaggerated to compensate for LV filling.

Study Limitations
Simultaneous invasive pulmonary pressure and vascular resistance measurements were not performed in patients with PH. However, Doppler interrogation of TR to estimate peak PASP has been validated and widely accepted, and although it has some limitations, its widespread use suggests that it remains the best noninvasive measure available.6–7 We also excluded patients with severe TR when the modified Bernoulli method was used. Another limitation of the study is the heterogeneous nature of the PH group; the group reflected a variety of disease causes and concurrent PH therapy, which may have affected the PASP and loading conditions of the ventricles. We did not exclude the potential confounding effects of RV volume overload caused by TR and pulmonic regurgitation on LV geometry and function. Among 44 patients with PH, 20, 10, 13, and 1 patient had no, mild (1+), moderate (2+), and severe TR (3 to 4+), respectively. Furthermore, none of our patients had severe (3 to 4+) pulmonic regurgitation. The contribution of RV volume overload cannot be excluded. Furthermore, because the RV geometry is quite complex, RV LAT LS might not optimally reflect overall RV function. Finally, we showed that differences in regional systolic function between groups can be, to a large degree, statistically accounted for by the variability of LV diastolic geometry. However, because we used regression analysis technique, this cannot be taken as a proof of causality. Still, one can safely assume that PASP plays a critical role in the altered LV geometry. Furthermore, LV geometry in diastole temporally precedes and functionally affects subsequent systolic strains of the IVS and LAT (the last assumption based on the Starling law of the heart), making the relationship between regional geometry and function quite probable.

Conclusions
In patients with PH, chronic RV pressure overload directly affects RV systolic function as manifested by impaired RV longitudinal deformation. RV pressure overload further influences IVS and LV geometry, which detrimentally impairs LV longitudinal and circumferential systolic deformation, more in the septum than more remote walls, and results in a decrease in LV torsion.

Sources of Funding
This work was supported in part by National Institutes of Health grant HL60917 (Dr Erzurum), National Space Biomedical Research Institute through NASA NCC 9-58 (Dr Thomas), and National Institute of Health, National Center for Research Services, CTSA (UL1RR024989), Cleveland, Ohio.

Disclosures
None.

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**CLINICAL PERSPECTIVE**

As a result of ventricular interdependence, analysis of concomitant intrinsic left ventricular (LV) diseases (ie, myocardial fibrosis) in patients with significant pulmonary hypertension (PH) is challenging. The farther a region is from the right ventricle (eg, the lateral wall), the less the impact will be that the PH has on LV strain. Furthermore, parameters based on circumferential fibers appear to propagate farther from the right ventricle than longitudinal parameters, likely reflecting continuity of the circumferential fibers from the septum to the lateral wall. Accordingly, a finding of abnormal LV lateral wall longitudinal strain in patients with PH might identify true LV dysfunction that is independent of PH. We further demonstrated that LV torsion correlated negatively with estimated pulmonary artery systolic pressure and with septal flattening. Abnormally reduced LV torsion in PH could represent an advanced stage of PH and geometric alteration. Interestingly, although torsion was reduced, untwisting velocity was similar regardless of pulmonary artery systolic pressure. This may reflect the known association of rapid untwisting with reduced LV end-systolic volume, a likely compensatory mechanism to maintain filling in the presence of reduced preload and/or exercise. Further study defining the relationship of LV torsion and untwisting with clinical outcomes and evaluating the effect of PH therapy on LV torsion is needed.
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_Circulation_. 2010;121:259-266; originally published online January 4, 2010;
doi: 10.1161/CIRCULATIONAHA.108.844340

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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