Relation Between Right Ventricular Function and Increased Right Ventricular [18F]Fluorodeoxyglucose Accumulation in Patients With Heart Failure

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Background—Left heart failure is characterized by alterations in metabolic substrate utilization, and metabolic modulation may be a future strategy in the management of heart failure. Little is known about cardiac metabolism in the right ventricle and how it relates to other measures of right ventricular (RV) function. This study was designed to measure glucose metabolism in the right ventricle, as estimated by [18F]fluorodeoxyglucose (FDG) positron emission tomography imaging and to determine the relation between RV function and FDG uptake in patients with heart failure.

Methods and Results—A total of 68 patients underwent cardiac [18F]FDG positron emission tomography scanning with measurement of RV FDG uptake as a standardized uptake value. Perfusion imaging was acquired at rest with rubidium-82 or [13N]ammonia. RV function was determined by equilibrium radionuclide ventriculography. Relative RV FDG uptake was determined as the ratio of RV to LV standardized uptake value. Fifty-five percent of these patients had ischemic cardiomyopathy. The mean LV and RV ejection fractions were 21% and 35%, respectively. There was a correlation between RV ejection fraction and the ratio of RV to LV FDG uptake whether the entire LV myocardium (r = 0.40, P = 0.001) or LV free wall (r = 0.43, P = 0.001) was used. This relation persisted in the subgroup with nonischemic cardiomyopathy (r = 0.37, P = 0.04). RV FDG uptake was weakly related to increased RV systolic pressure but not related to LV size, function, or FDG uptake. The correlation between RV ejection fraction and RV/LV FDG was maintained after partial-volume correction (r = 0.68, P = 0.001).

Conclusions—RV dysfunction is associated with an increase in RV FDG uptake, the magnitude of which may be correlated with severity. (Circ Cardiovasc Imaging. 2011;4:59-66.)

Key Words: heart failure ■ right ventricle ■ FDG PET

Right ventricular (RV) dysfunction is associated with poor prognosis in patients with preexisting coronary artery disease, significant heart failure (HF), and pulmonary artery hypertension.1–3 Alterations in myocardial substrate utilization have been implicated in the pathogenesis of contractile dysfunction and HF.4–6 Studies of substrate utilization in left HF have demonstrated that fatty acid utilization may be unchanged or slightly increased in early HF but substantially decreased in advanced HF; with a concomitant increase of glucose utilization in early HF and a subsequent decline in advanced HF as insulin resistance develops in the myocardium.8–11 Whether the shift toward glucose utilization represents an adaptive or a maladaptive response predisposing the heart to further myocardial dysfunction also remains uncertain. Few have studied RV metabolism in detail. Despite this, several lines of evidence suggest that RV metabolism may be different.12–14

Clinical Perspective on p 66

It has been demonstrated that there are substantial differences among patients in their tendency to develop right HF.15 Recent literature suggests that the heterogeneity in clinical course may be caused by a polymorphic variation in gene expression and that the link between cardiac contractile function and gene expression may be altered energy substrate metabolism.16 An understanding of the metabolic changes associated with RV failure and dysfunction may lead to a potential target for the use of metabolic modulation in the treatment and prevention of right HF. This study was de-
signed to measure glucose metabolism in the right ventricle estimated by [18F]fluorodeoxyglucose (FDG) positron emission tomography (PET) scans and to determine the relation between RV function and FDG uptake in patients with HF.

**Methods**

**Patient Population**

Sixty-eight patients were enrolled from 2 sources. First, 60 consecutive adult patients (>18 years of age) with a history of congestive HF who had a left ventricular (LV) ejection fraction (EF) ≤35% as documented by equilibrium radionuclide ventriculography (ERVG), symptoms consistent with New York Heart Association functional class II-III despite optimal medical therapy, and a QRS duration ≥130 ms based on baseline ECG who had been recruited for the PREDICT study17 were prospectively enrolled at the University of Ottawa Heart Institute. This study evaluated the effect of lateral wall scar on reverse remodeling and the clinical response to cardiac resynchronization therapy. For the most part, these patients did not have severe pulmonary hypertension. To include a patient population with a wider range of pulmonary hypertension, we also included a cohort of 8 consecutive patients with ischemic cardiomyopathy and significant pulmonary hypertension who were enrolled in the CADRE database, a regional registry study evaluating cardiac PET use in Ontario.18 Ischemic etiology was defined as having both a documented history of myocardial infarction and evidence of significant coronary artery disease on coronary angiography (at least 1 stenosis ≥70% in ≥2 major arteries). Significant pulmonary hypertension was defined as a mean pulmonary artery pressure ≥40 mm Hg, or an RV systolic pressure (RVSP) >50 mm Hg on right heart catheterization or echocardiography done within 60 days of the PET study. All patients enrolled provided informed consent for inclusion. The study was approved by the human research ethics board of the University of Ottawa Heart Institute.

**Study Procedures**

**Echocardiographic Assessment of Wall Thickness**

Transthoracic echocardiographic analysis was performed with a Phillips Sonos 5500 ultrasound system. The pressure difference between the right chambers was calculated from the modified Bernoulli equation: gradient (ΔP), in mm Hg, =4v², where v=accelerated velocity across a stenosis, and the measured Doppler velocity of the regurgitant tricuspid flow jet. The RVSP (in mm Hg) and right atrial pressure were assessed by standard recommended techniques.

Wall thickness was measured from 2-dimensional echocardiographic images. Each measurement was taken 3 times and then averaged. The diastolic RV free wall was measured in a subcostal view, and parasternal views were obtained to measure posterolateral LV wall thickness.19

**ERVG Imaging and Analysis**

Patients underwent ERVG planar imaging at baseline. The ERVGs were acquired with a standard ECG-gated equilibrium technetium-99m red blood cell blood pool imaging protocol.20,21 For quantitative analysis of global RVEF and LVEF, data obtained from the left anterior oblique view was used.

**PET Rest Perfusion and Metabolic Imaging Protocol**

Patients underwent a rest perfusion and a metabolism PET scan. All patients were required to fast before the PET study and underwent monitoring of blood glucose levels. Patients were positioned in a Siemens/CTI (Siemens, Knoxville, Tenn) ECAT ART camera (n = 52) or a GE Discovery Rx/VCT camera (n = 16). A 4-minute cesium-137 single-transmission scan22,23 was performed for attenuation correction in the Siemens/CTI scanner. For the GE PET system, a scout scan and low-dose computed tomography (15-cm field of view) were performed.

PET perfusion images were acquired at rest according to a standard protocol with rubidium-82 or [15N]ammonia as described previously. Immediately after the transmission scan, 8 to 10 MBq/kg of rubidium-82 or 5 to 10 MBq/kg of [15N]ammonia was administered intravenously. Static perfusion images were acquired.24,25 For FDG imaging, nondiabetic patients were studied after an oral glucose load; whereas an insulin-euglycemic clamp was used for those with diabetes.26–27 A standard dose of 5 MBq/kg (<550 MBq) of FDG was injected intravenously as a bolus 30 minutes after the glucose load. For diabetic patients with insulin-euglycemic clamp, plasma glucose levels were checked every 5 minutes. FDG injection was performed after 3 stable plasma glucose levels were obtained and stable glycemia was established (optimal glycemia = 5 mmol/L). FDG PET image acquisition was started 45 minutes after FDG injection to ensure accurate myocardial tracer uptake.28

**Image Processing**

Transverse PET images were reconstructed by Fourier rebinning--filtered back-projection with a 12-mm 3-dimensional Hann window of the ramp filter. Photon attenuation and scatter corrections were applied by ECAF v7.2 software. Automatic reorientation of the images into short-axis sections was achieved with FlowQuant software.29

**Determination of RV and LV Uptakes and Data Analyses**

Areas of maximal LV and RV uptake were identified visually on the short-axis transaxial images. One to four regions of interest (size ≥2×2 pixels) were drawn on the whole RV free wall, interventricular septum, whole LV, and LV lateral wall of the end-diastolic transaxial slice. The standardized uptake value (SUV), a well-established index of tissue FDG uptake per unit volume, was calculated as follows30:

\[ \text{SUV}_\text{RV} = \frac{\text{count (counts per second per pixel)} \times \text{body weight (kg)} \times \text{injected dose (mCi)} \times \text{calibration factor (cps/mCi)}}{\text{peak LV SUV}} \]

All measurements were performed twice for both right and left ventricles.

In the subset of patients with RV and LV wall thickness measurements available, partial-volume recovery coefficients (RPCs) were calculated by convolution with a gaussian kernel representing the reconstructed PET image resolution. Partial volume recovery--corrected FDG activity values were then calculated as SUV/RC.

**Analyses**

The primary objective of this study was to determine the relation between RVEF and RV glucose uptake, estimated from RV FDG SUV as well as the ratio of mean RV SUV to mean LV SUV. The value of mean RV SUV/peak LV SUV (in regions confirmed to have normal perfusion) was also determined to correct for any variability in FDG uptake in the left ventricle owing to previous scar. Secondary objectives included determination of the relation between RV FDG uptake and LV size, LVEF and LV size, and RV FDG uptake and estimates of pulmonary pressures.

**Statistical Analysis**

All values are expressed as mean ±SD for normally distributed data and medians with first and third quartiles for nonnormally distributed data. Pearson correlation and simple linear regression analyses were used to relate LVEF and markers of metabolism. Correlation coefficients were compared by a Z test after Fisher Z-transforms. Comparisons between the tertiles of RV FDG groups were made with χ² tests for unequal variances and χ² tests, where appropriate. All probability values were 2-sided and a probability value of <0.05 was considered statistically significant. All statistical analyses were performed with STATA software, version 9.2 (Stata Corp, College Station, Tex).

**Results**

**Baseline Characteristics**

Table 1 demonstrates the characteristics of the study population. All patients had advanced HF, with a mean LVEF of 21% and a mean RVEF of 35%. Seventy-five percent of patients were in New York Heart Association class III. Forty-five percent of the subjects had nonischemic cardiomyopathy. Moderate pulmonary hypertension was common, with a median RVSP of 52 mm Hg (minimum, 25 mm Hg; maximum, 81 mm Hg). The average RV wall thickness was 0.45 ± 0.07 mm (median, 0.45 mm; interquartile range, 0.42 to 0.5 cm).
RV Function and FDG Uptake
There was a statistically significant relation between increased FDG uptake in the right ventricle and decreases in RVEF \((r = 0.32, P = 0.008; \text{Figures 1 and 2})\). FDG uptake in the right relative to the left ventricle was represented as (1) the ratio of RV SUV to LV SUV for the entire LV myocardium and (2) the ratio of RV SUV to LV SUV when limited to uptake in the LV free wall, given the high proportion of subjects with baseline left bundle branch block. The ratio of normal RV to whole LV SUV in healthy volunteers without cardiomyopathy ranged from 0.26 to 0.31, according to data obtained in our laboratory. The relation between RVEF and relative RV:LV FDG uptake was also significant, regardless of whether the whole LV was used \((r = 0.40, P = 0.01)\) or the measurement was isolated to the LV lateral wall FDG uptake alone \((r = 0.43, P = 0.001; \text{Figure 3})\).

The significance of the RV-LV ratio was due to increased RV FDG uptake, as there was no correlation between LV glucose uptake and RV function or size.

Because FDG uptake may be variable, particularly in ischemic cardiomyopathy, we evaluated the ratio of RV uptake to LV peak uptake (in segments confirmed to have normal perfusion). Significant relations were observed similar to those for the ratio of the mean SUVs. To further evaluate the effect of potential variability in FDG uptake in the left ventricle of patients with ischemic cardiomyopathy, the data were stratified by history of ischemic versus nonischemic cardiomyopathy. Both groups continued to display a relation between increased RV/LV FDG uptake and decreases in RVEF \((r = -0.37, P = 0.04 \text{ for nonischemic cardiomyopathy, and } r = -0.47, P = 0.004 \text{ for those with})\.

Table 1. Baseline Characteristics of Study Cohort

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total (N=68)</th>
<th>T1 or T2 (n=46)</th>
<th>T3 (n=22)</th>
</tr>
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<tr>
<td>Median age (interquartile range) or mean±SD, y</td>
<td>69 (60–76)</td>
<td>65±12</td>
<td>67±11</td>
</tr>
<tr>
<td>Female sex</td>
<td>9 (13%)</td>
<td>16%</td>
<td>5%</td>
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<tr>
<td>Ischemic cardiomyopathy</td>
<td>37 (55%)</td>
<td>50%</td>
<td>67%</td>
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<td>NYHA class III</td>
<td>51 (75%)</td>
<td>72%</td>
<td>80%</td>
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<tr>
<td>Left bundle branch block</td>
<td>48 (71%)</td>
<td>75%</td>
<td>62%</td>
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<tr>
<td>History of hypertension</td>
<td>31 (46%)</td>
<td>50%</td>
<td>38%</td>
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<tr>
<td>Current smoker</td>
<td>13 (19%)</td>
<td>23%</td>
<td>10%</td>
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<td>Diabetes</td>
<td>30 (45%)</td>
<td>40%</td>
<td>62%</td>
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<tr>
<td>History of revascularization</td>
<td>37 (55%)</td>
<td>59%</td>
<td>48%</td>
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<td>Therapies</td>
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<td>(\beta)-blockers</td>
<td>64 (94%)</td>
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<td>86%</td>
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<td>ACEI or ARB</td>
<td>65 (95%)</td>
<td>95%</td>
<td>99%</td>
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<td>Diuretics</td>
<td>62 (92%)</td>
<td>89%</td>
<td>95%</td>
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<td>Digoxin</td>
<td>29 (42%)</td>
<td>38%</td>
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<td>(interquartile range)</td>
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<tr>
<td>RVEF</td>
<td>35% (23–43%)</td>
<td>36±11%</td>
<td>25±11%*</td>
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<tr>
<td>LVEF</td>
<td>21% (16–27%)</td>
<td>23±9%</td>
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<tr>
<td>LVESEV</td>
<td>226 (170–276) mL</td>
<td>237±109 mL</td>
<td>247±73 mL</td>
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<td>LVEDV</td>
<td>285 (242–340) mL</td>
<td>307±116 mL</td>
<td>310±82 mL</td>
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<tr>
<td>RVSP</td>
<td>50 (42–62) mm Hg</td>
<td>49±11 mm Hg</td>
<td>52±16 mm Hg</td>
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<tr>
<td>Metabolic parameters, median</td>
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<td>(interquartile range) or mean±SD</td>
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<tr>
<td>RV SUV</td>
<td>1.7 (1.2–2.4)</td>
<td>1.7±0.9</td>
<td>2.2±1.9*</td>
</tr>
<tr>
<td>LV SUV</td>
<td>2.9 (2.1–3.6)</td>
<td>2.7±1.0</td>
<td>2.5±1.0</td>
</tr>
<tr>
<td>RV SUV–LV SUV ratio</td>
<td>0.60 (0.51–0.75)</td>
<td>0.52±0.1</td>
<td>0.87±0.23*</td>
</tr>
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</table>

T indicates tertile; NYHA, New York Heart Association; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ESV, end-systolic volume; and EDV, end-diastolic volume.

*Denotes a significant difference between tertile 1/2 and tertile 3 \((P<0.05)\).

Figure 1. Examples of increased RV-LV FDG uptake in the right ventricle in 2 patients with ischemic cardiomyopathy. Short-axis images of myocardium demonstrate increased FDG uptake in the RV free wall. Patient 1 is a 68-year-old male with a previous anteroseptal myocardial infarction. His LVEF was 20% with an RVEF of 30%. The RV SUV was 1.9, with a whole LV SUV of 2.55 and an RV-LV ratio of 0.75. Patient 2 is a 59-year-old male with a previous coronary artery bypass graft, an LVEF of 18%, and an RVEF of 37%. The RV SUV was 1.7, with a whole LV SUV of 2.58 and an RV-LV ratio of 0.66.
ischemic cardiomyopathy). There was also no change between absolute RV FDG uptake and RVEF in this stratified analysis ($r = -0.33$, $P = 0.05$ for nonischemic cardiomyopathy and $r = -0.33$, $P = 0.04$ for ischemic cardiomyopathy). In contrast, LVEF was not related to RV FDG uptake ($r = 0.06$, $P = 0.65$) nor the RV-LV FDG uptake ratio ($r = -0.09$, $P = 0.49$). When compared with subjects in the first and second tertiles of RV SUV values, subjects in the third tertile had significantly lower RVEFs (25±11% vs 36±25%, $P = 0.001$) than in the other 2 tertiles (Table 1).

**Partial-Volume Effect on RV FDG Uptake Versus RV Function Relations**

To consider the effect of partial volume on FDG measurements owing to differences in RV wall thickness, we evaluated the relations or RV SUV parameters to RVEF in 26 patients who underwent echocardiography within 60 days of FDG PET. Table 2 shows the relations for the entire data set and the subset of patients who underwent echocardiography without and with partial-volume correction of their FDG data. After correction for partial volume, the correlation coefficients for the relation between RV FDG SUV parameters and RV function remained significant ($P \leq 0.03$ for all 3 relations; Figure 4) and were numerically greater than values from the original data set (Table 2). Statistical comparisons between correlation coefficients were not significantly different, although there was a trend for significant improvement in the relation between RVEF and RV/whole LV FDG SUV data when a partial-volume correction was applied ($P = 0.095$).

**Pulmonary Hypertension and RV Uptake**

The majority of patients in this study had some degree of pulmonary venous hypertension, with a median RVSP or (systolic pulmonary artery pressure, if available) at baseline of 52 mm Hg. Worsening pulmonary hypertension was weakly related to increased RV FDG uptake ($r = 0.36$, $P = 0.04$) but not LV FDG uptake ($r = -0.02$, $P = 0.91$), nor was it related to the ratio of whole RV to LV uptake ($r = 0.05$, $P = 0.85$; Figure 5). When a partial-volume correction was considered in a smaller cohort ($n = 16$ for whom both RVSP and RV wall thickness could be determined), the correlation coefficient was not significantly different because the relation was no longer statistically significant ($r = 0.19$, $P = 0.43$ for the subset without a partial-volume correction and $r = 0.05$, $P = 0.85$ for the subset with a partial-volume correction; Table 2).

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**Figure 2.** Pearson correlation between RVEF and RV FDG SUV (A) ($r = -0.32$, $P = 0.008$) and RV FDG SUV in those in the third tertile only ($r = -0.2$, $P = 0.37$) (B).

**Figure 3.** Pearson correlation between RVEF and the ratio of RV FDG SUV to whole LV SUV ($r = -0.40$, $P = 0.001$) and RVEF and the ratio of RV FDG SUV to LV lateral wall SUV ($r = -0.43$, $P = 0.001$).
Reproducibility of RV FDG Image Analysis

To evaluate intraobserver test reproducibility, a subset of scans from 30% of the total population were reread by the same image analyst (M.A.) 6 months later. The intraclass correlation for the whole LV uptake was 0.94 (interquartile range, 0.84 to 0.98; \( P < 0.0001 \)) and 0.99 (interquartile range, 0.97 to 0.99; \( P < 0.001 \)) for the whole RV. To evaluate interobserver test reproducibility, the same subset of scans was read by 2 independent image analysts (M.A. and M.Z.). The interclass correlation was 0.99 (interquartile range, 0.97 to 0.99; \( P < 0.0001 \)) for the whole LV uptake and 0.95 (interquartile range, 0.87 to 0.98; \( P < 0.001 \)) for the whole RV.

**Discussion**

This study demonstrates that RV glucose uptake increases with decreasing RVEF. The ratio of uptake in the right versus the left ventricle increased with progressive RV dysfunction, independent of a history of ischemia and baseline LV glucose...
metabolism. Finally, in a cohort of patients with known LV failure, the severity of pulmonary hypertension was weakly correlated to RV glucose metabolism. Importantly, RV FDG SUV parameters and RVEF relations were not adversely affected by a partial-volume recovery correction.

Alterations in myocardial substrate metabolism have been implicated in the pathogenesis of HF and contractile dysfunction. Animal models of left HF have demonstrated that the progression from cardiac hypertrophy to ventricular dysfunction is associated with a decrease in the expression of genes coding for fatty acid oxidation and a shift in metabolism, with glucose becoming the primary energy substrate. A similar shift in metabolism has been demonstrated in patients with idiopathic dilated cardiomyopathy on PET imaging. However, it has also been proposed that the reliance of the myocardium on glucose may produce a relatively energy-deficient state that, in the long term, could result in decreased contractile performance. It is possible that although glucose metabolism may be beneficial in early HF, in the long term this may lead to maladaptive myocardial responses contributing to the development of worsening HF. An understanding of the alterations and clinical significance of myocardial metabolism in HF is an important initial step in developing strategies to target metabolic modulation as a potential therapy for patients with HF.

The evaluation of RV failure is an important goal in the management of pulmonary artery hypertension, although few studies have examined RV metabolism in detail. Increased RV free-wall myocardial glucose utilization has been demonstrated in rat models of pulmonary artery hypertension. Okikawa and colleagues demonstrated that RV FDG accumulation increased in accordance with the severity of pulmonary vascular resistance in patients with pulmonary artery hypertension. In contrast to these results, a similar study by Kluge et al suggested that in patients with pulmonary artery hypertension, the increased ratio of right-left FDG accumulation with increased pulmonary vascular resistance was unrelated to increased RV FDG uptake, but a corresponding decreased LV FDG accumulation. Importantly, these authors did identify a significant linear relation between RV FDG uptake and increasing Tei index, an echocardiographic marker of progressive RV dysfunction.

Consistent with the work of Kluge and colleagues, the current study also demonstrated a linear relation between RV function and RV FDG uptake and a weak but statistically significant relation between worsening pulmonary pressure and increased RV FDG. However, in contrast to Kluge et al, we did not identify any relation between RVSP and changes in LV FDG uptake. This difference may be explained by the fact that in the study by Kluge et al, all subjects had normal LV systolic function, whereas in the current study, all subjects had significant LV dysfunction. It is possible that differences in methodology, including glucose loading protocols (with or without acipamox), and patient populations may explain the heterogeneity of the results regarding RV FDG and pulmonary vascular resistance. For example, the presence of RV infarction could potentially contribute to decreased RV FDG uptake. However, the relation between RVEF and RV FDG metabolism was similar when the data were stratified by ischemic and nonischemic cardiomyopathy. In addition, the findings were not altered when corrected for the peak LV SUV in normally perfused segments. Finally, some heterogeneity may have been due to the potential time delay between PET scanning and echocardiographic assessment of pulmonary pressures, owing to underlying lability in this measurement. Further studies are needed to determine whether the increased RV FDG accumulation promotes or results from progressive RV failure.

Some methodologic limitations require discussion. It is known that FDG uptake is only a surrogate for true glucose metabolism; thus, it is possible that the true difference in glucose metabolism between the 2 ventricles or as it relates to RV function or afterload might be missed or underestimated. Nevertheless, FDG uptake does provide an in vivo means to probe alterations in glucose metabolism in the human right ventricle. Although our inter- and intra-observer variabilities were excellent, data on the reproducibility of RV FDG measurements are limited. Future studies should consider defining its reliability to further characterize potential utility in measuring RV metabolism. RVEF and volumes were measured with planar ERVG imaging. Although planar ERVG is accurate and reproducible for the left ventricle, the technique is less robust for the right ventricle. This methodology may also contribute to some underestimation of the correlation between RVEF and RV metabolism. Cardiac magnetic resonance imaging is often considered the best technique to quantify RV volumes and size. However, in this population, 25% of the patients had either a pacemaker or an implantable cardioverter/defibrillator, thus making a significant proportion of the study population ineligible for magnetic resonance imaging. Measurements of RV hypertrophy were estimated by echocardiographic analysis, which also has limitations owing to poorer resolution compared with magnetic resonance imaging.

Nevertheless, given the potential impact of the partial-volume effect due to differences in RV wall thickness, we evaluated the relations of RV SUV parameters to RVEF in a subset of patients. After correction for partial volume, correlation coefficients were either similar or numerically increased when compared with the data without partial-volume correction. This has 2 important implications: (1) the relations of RV FDG SUV parameters and RVEF are likely valid observations and (2) the partial-volume
correction is important when measuring and interpreting glucose utilization in the right ventricle.

The correlation coefficients for the relation of RV SUV parameters to RVSP numerically decreased in the echocardiography subset and after partial-volume correction. These relations were no longer significant (with or without a partial-volume correction). This was likely due to loss of statistical confidence from a much smaller sample size. As such, conclusions regarding RV and RV FDG relations must be made with caution. Whether the relation is partly driven by a partial-volume effect (in contrast to the RVEF vs RV FDG parameters, which were not adversely affected by partial-volume correction) will require evaluation in larger studies.

Many of the patients in the current study were evaluated on the Siemens/CTI ECAT ART PET system, which has a resolution inferior to most current PET/computed tomography systems. This may have contributed to some of the variability observed and further emphasizes the importance of partial-volume correction. Data on invasive pulmonary pressures were not available for all subjects, thus limiting the robustness of volume correction. Data on invasive pulmonary pressures were not available for all subjects, thus limiting the robustness of partial-volume correction. This was likely due to loss of statistical confidence of the RVEF versus RV FDG parameter observations and the importance of partial-volume correction in the analysis of RV metabolism by PET. Whether a shift toward glucose metabolism in the failing right ventricle has potential long-term significance as a marker that could influence therapy for RV dysfunction and failure requires further evaluation in prospective studies. Such studies are now ongoing.

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Disclosures
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CLINICAL PERSPECTIVE

Despite significant improvements in the management of heart failure, morbidity and mortality remain high. The comorbid association of right ventricular (RV) dysfunction with left heart failure identifies patients with a particularly poor prognosis. There has been recent clinical interest in the role of metabolic modulation in the treatment of left ventricular dysfunction. An understanding of the metabolic changes in the right ventricle may serve as a potential target for the management of RV dysfunction. This study was designed to characterize myocardial metabolism in the right ventricle of patients with left ventricular failure. RV dysfunction was associated with an increase in RV glucose uptake. This metabolic change was correlated with the severity of RV dysfunction. Larger, prospective studies are required to define the potential clinical implications of this metabolic adaptation.
Relation Between Right Ventricular Function and Increased Right Ventricular [18F]Fluorodeoxyglucose Accumulation in Patients With Heart Failure

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