Prognostic Value of Right Ventricular Function in Patients After Acute Myocardial Infarction Treated With Primary Percutaneous Coronary Intervention

M. Louisa Antoni, MD; Roderick W.C. Scherptong, MD; Jael Z. Atary, MD; Eric Boersma, PhD; Eduard R. Holman, MD, PhD; Ernst E. van der Wall, MD, PhD; Martin J. Schalij, MD, PhD; Jeroen J. Bax, MD, PhD

Background—Data on the association between right ventricular (RV) function and adverse events after acute myocardial infarction (AMI) are scarce. The purpose of the current study was to evaluate the relation between RV function and adverse events in patients treated with primary percutaneous coronary intervention for AMI.

Methods and Results—Consecutive patients admitted with AMI treated with primary percutaneous coronary intervention underwent echocardiography within 48 hours of admission to assess left ventricular and RV function. RV function was quantified with RV fractional area change (RVFAC), tricuspid annular plane systolic excursion, and RV strain. The end point was defined as a composite of all-cause mortality, reinfarction, and hospitalization for heart failure. All patients (n=621) were followed prospectively, and during a mean follow-up of 24 months, 86 patients reached the composite end point. RVFAC, tricuspid annular plane systolic excursion, and RV strain were all univariable predictors of worse outcome. After multivariable analysis, only RVFAC (hazard ratio, 0.96; 95% CI, 0.92 to 0.99) and RV strain (hazard ratio, 1.08; 95% CI, 1.03 to 1.13) independently predicted the composite end point. In addition, RV strain provided incremental value to clinical information, infarct characteristics, left ventricular function, and RVFAC.

Conclusions—RV function provides strong prognostic information in patients treated with primary percutaneous coronary intervention for AMI. (Circ Cardiovasc Imaging. 2010;3:264-271.)

Key Words: echocardiography □ myocardial infarction □ prognosis

The prognosis of patients after acute myocardial infarction (AMI) is determined by the interaction of a large number of factors. Besides the importance of clinical parameters, several studies have described the use of 2D echocardiography for the identification of patients who are at risk of adverse outcome.1 These investigations revealed that the presence of left ventricular (LV) dysfunction, on 2D-echocardiography shortly after AMI, is one of the most important prognostic parameters.2,3 Therefore, noninvasive assessment of LV function has become essential for post-AMI risk stratification.

Clinical Perspective on p 271

The relevance of right ventricular (RV) function, on the other hand, is poorly defined in post-AMI patients. The involvement of the RV during inferior AMI has been defined as a strong predictor of major complications and in-hospital mortality.4,5 Some evidence is available that RV dysfunction is associated with an adverse prognosis in post-AMI patients with moderate to severe LV dysfunction.6,7 In patients who undergo primary percutaneous coronary intervention (PCI), however, the degree of LV dysfunction is generally mild and the clinical relevance of RV dysfunction in that currently growing population of post-AMI patients is unknown. Therefore, the aim of the current study was to investigate the relation between RV function and adverse events in post-AMI patients treated with primary PCI. In addition to traditional measurements that are recommended to quantify RV function with 2D-echocardiography, RV strain was assessed. This novel technique enables direct quantification of myocardial deformation and is a sensitive tool to detect RV dysfunction.8-11

Methods

Patient Selection and Study Protocol

Since February 2004, consecutive patients admitted with AMI treated with primary PCI were included in an ongoing registry. All patients were treated according to the institutional AMI protocol,
which is driven by the most recent guidelines. This protocol, designed to improve care around AMI, includes structured medical therapy and outpatient follow-up, as described previously. In addition, 2D echocardiography is performed within 48 hours of admission. This echocardiogram was used to assess LV and RV function. All patients were followed prospectively and the occurrence of adverse events was noted. Patients of whom more than 6 months of follow-up data were lacking were considered lost to follow-up and excluded from further analysis.

Echocardiography Images were obtained with patients in the left lateral decubitus position using a commercially available system (Vivid 7, General Electric-Vingmed, Horten, Norway). Data acquisition was performed at a depth of 16 cm in the parasternal and apical views using a 3.5-MHz transducer. During breath-hold, M-mode and 2D images were obtained and 3 consecutive beats were saved in cine-loop format. Analysis was performed offline by 2 independent observers using dedicated software (EchoPac version 108.1.5, General Electric-Vingmed). The reference limits of all echocardiographic parameters were defined according to American Society of Echocardiography Guidelines. The LV end-systolic volume (LVEDV) and end-diastolic volume (LVEDV) were assessed and LV ejection fraction (LVEF) was calculated using the biplane Simpson method.

In addition, the LV was divided into 16 segments, and each segment was analyzed individually and scored based on its motion and systolic thickening (1, normokinesis; 2, hypokinesis; 3, akinesis; and 4, dyskinesis). Subsequently, wall motion score index (WMSI) was calculated as the sum of the segment scores divided by the number of segments scored.

Left atrial (LA) size was quantified by calculating the volume according to the ellipsoid model. Severity of mitral regurgitation (MR) was graded semiquantitatively from the jet area of color-flow Doppler data and by measuring the width of the vena contracta. MR was characterized as: mild, jet area/LA area <0.7 cm; moderate, jet area/LA area 20% to 40% and vena contracta width 0.3 cm; moderate, jet area/LA area 20% to 40% and vena contracta width 0.3 to 0.6 cm; and severe, jet area/LA area >40% and vena contracta width ≥0.7 cm.

Tricuspid regurgitation (TR) severity was graded based on jet right atrial area ratio. When the jet area occupied <10% of the right atrial area, TR was graded as trivial, 10% to <20% as mild, 20% to <33% as moderate, ≥33% as severe. In addition, the diameter of inferior vena cava and its respiratory variation were measured 1.0 to 2.0 cm from the junction with the right atrium in the subcostal view, as recommended by the guidelines.

To assess diastolic function, pulsed-wave Doppler of the mitral valve inflow was obtained by placing the sample volume between the aortic valve and the mitral valve inflow, and the ratio of early diastolic mitral inflow velocity to peak early diastolic mitral outflow velocity (E/A) was calculated. The E/A ratio was obtained by dividing E by E', which was measured using color-coded tissue Doppler imaging at the septal side of the mitral annulus in the apical 4-chamber view.

RV Function Analysis RV fractional area change (RVFAC) was analyzed by tracing the RV end-diastolic area (RVEDA) and end-systolic area (RVSA) in the apical 4-chamber view using the formula (RVEDA-RVSA)/RVEDA×100. Tricuspid annular plane systolic excursion (TAPSE) was measured in the RV free wall. In the 4-chamber view, the M-mode cursor was placed through the tricuspid annulus in such a way that the annulus moved along the M-mode cursor and the total displacement of the RV base from end-diastole to end-systole was measured. Peak systolic longitudinal strain of the RV free wall was measured in the 4-chamber view using speckle-tracking analysis. This novel software analyzes motion by tracking frame-to-frame movement of natural acoustic markers in 2 dimensions. All images were recorded with a frame rate of >40 fps for reliable analysis. The RV endocardial border was manually traced at end-systole and the automatically created region of interest was adjusted to the thickness of the myocardium. Peak systolic longitudinal strain was determined in the 3 segments of the RV free wall (basal, mid, and apical), and RV strain was calculated as the mean value of all segments. Segments were discarded if tracking was of poor quality. Strain analysis was feasible in 85% of segments.

Statistical Analysis Continuous data are presented as mean±standard deviation and categorical data are presented as frequencies and percentages. Differences in characteristics between patient groups were evaluated using the unpaired Student t test and χ² test.

The primary aim was to assess the association between RV function and adverse events after adjusting for clinical and echocardiographic covariates. Separate multivariable models were constructed for RVFAC, TAPSE, and RV strain using Cox proportional hazards analysis to evaluate the individual prognostic importance of the different RV function measurements. Selection of parameters for consideration for entry in the multivariable models was based both on clinical judgment and univariable statistical significance. Based on these considerations, adjustments in the multivariable models were made for age, Killip class ≥2, right coronary artery (RCA) as culprit vessel, multivessel disease, peak cardiac troponin T (cTnT) level, LVEF, WMSI, E/E’ ratio, and moderate or severe MR. Peak creatine phosphokinase level and LVESV were not included in multivariable analyses to avoid collinearity with peak cTnT level and LVEF.

In addition, multiple variable analysis was performed for all events individually. Nonfatal reinfarction was defined based on criteria of typical chest pain, elevated cardiac enzyme levels, and typical changes on the ECG. Hospitalization for heart failure (HF) was defined as hospitalization for new-onset or worsening HF. Because only a small number of 29 patients reached the end point of HF, no further subdivision was made for the cause of HF.

To further investigate the clinical relevance of RV dysfunction, the population was stratified into 2 groups according to RV function. For RVFAC and TAPSE, cut-offs were defined according to the guidelines: 32% and 1.5 cm, respectively. The normal value of RV strain has been reported to be 29.3±3.6%. Patients were therefore divided according to the mean value ±2 SD, which is the lower limit of normal RV strain (−22.1%). Event rates were plotted in Kaplan-Meier curves for the composite end point, the study population was divided by the previously mentioned cut-offs, and groups were compared using the log-rank test. The date of last contact for patients without events was used in Kaplan-Meier analysis. Finally, univariable and multivariable Cox proportional hazards analyses were performed for RVFAC, TAPSE, and RV strain, dichotomized by the cut-offs.

The incremental value of RV function, in addition to known risk factors for adverse outcome (age, Killip class ≥2, RCA as culprit vessel, multivessel disease, peak cTnT level, LVEF, WMSI, E/E’ ratio, and moderate or severe MR), was established. For this purpose, those characteristics were entered in the Cox proportional hazard model in a stepwise fashion. Subsequently, RVFAC and RV strain were entered individually. In addition, RV strain was entered into the model of RVFAC to test further incremental value. Global χ² values including significance levels were calculated.

All statistical tests were 2-sided, and a probability value <0.05 was considered statistically significant.

Results Patient Characteristics and Follow-Up A total of 682 patients were included. Nine (1.3%) patients died before echocardiographic assessment could be performed, and in 22 (3.2%) patients echocardiographic assessment was not available within 48 hours of admission due to logistic reasons. Thirty patients (4.4%) were lost to follow-up and were excluded from further analysis. The study population consisted of the remaining 621 consecutive patients by guest on November 6, 2017
admitted with AMI treated with primary PCI. Tables 1 and 2 summarize the clinical and echocardiographic characteristics of the population. Mean age was 60 ± 12 years, and most patients were men (78%). Mean LVEF, RVFAC, TAPSE, and RV strain were 45 ± 8%, 37 ± 9%, 1.7 ± 0.2 cm, and −22 ± 7%, respectively.

Fifty-seven patients (10%) presented with congestive HF defined as Killip class ≥2. Patients with congestive HF had significantly lower TAPSE (1.6 ± 0.2 cm versus 1.7 ± 0.2 cm, P = 0.01) and RV strain (−19 ± 6% versus −22 ± 7%, P = 0.02). No differences were observed in RVFAC and patients with and without congestive HF (37 ± 10% versus 37 ± 9%, P = 0.71).

The RCA was the culprit vessel in 217 patients (35%). No differences in RVFAC (36 ± 9% versus 38 ± 9%, P = 0.07), TAPSE (1.7 ± 0.2 cm versus 1.7 ± 0.2 cm, P = 0.28), and RV strain (−21 ± 7% versus −22 ± 7%, P = 0.38) were observed in patients with and without inferior AMI.

LV dysfunction (defined as LVEF < 40%) was observed in 151 patients (24%). When comparing RV function in patients with and without LV dysfunction, no significant differences were observed in RVFAC (37 ± 9% versus 38 ± 9%, P = 0.31) and RV strain −21 ± 7% versus −22 ± 7%, P = 0.09). However, TAPSE was significantly lower in patients with LV dysfunction compared with patients without LV dysfunction (1.6 ± 0.2 cm versus 1.7 ± 0.2 cm, P = 0.02).

TAPSE was the only RV function measurement that differed significantly in patients with multivessel disease compared with patients without multivessel disease (1.6 ± 0.2 cm versus 1.7 ± 0.2, P = 0.03).

### Table 1. Baseline Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>All Patients (n = 621)</th>
<th>Event (n = 86)</th>
<th>Event-Free (n = 535)</th>
<th>P (Event vs Event-Free)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>60 ± 12</td>
<td>65 ± 14</td>
<td>60 ± 11</td>
<td>0.001</td>
</tr>
<tr>
<td>Male sex</td>
<td>486 (78%)</td>
<td>67 (78%)</td>
<td>419 (78%)</td>
<td>0.93</td>
</tr>
<tr>
<td>Killip class ≥2</td>
<td>57 (10%)</td>
<td>26 (33%)</td>
<td>31 (6%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Current smoking</td>
<td>313 (51%)</td>
<td>42 (49%)</td>
<td>271 (51%)</td>
<td>0.83</td>
</tr>
<tr>
<td>Diabetes</td>
<td>61 (10%)</td>
<td>14 (16%)</td>
<td>47 (9%)</td>
<td>0.03</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>253 (41%)</td>
<td>28 (33%)</td>
<td>225 (42%)</td>
<td>0.11</td>
</tr>
</tbody>
</table>

### Table 2. Baseline Echocardiographic Characteristics

<table>
<thead>
<tr>
<th></th>
<th>All Patients (n = 621)</th>
<th>Event (n = 86)</th>
<th>Event-Free (n = 535)</th>
<th>P (Event vs Event-Free)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVESV, mL</td>
<td>58 ± 22</td>
<td>67 ± 32</td>
<td>56 ± 20</td>
<td>0.006</td>
</tr>
<tr>
<td>LVEDV, mL</td>
<td>105 ± 34</td>
<td>111 ± 43</td>
<td>104 ± 33</td>
<td>0.20</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>45 ± 8</td>
<td>41 ± 9</td>
<td>46 ± 8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>WMSI</td>
<td>1.5 ± 0.3</td>
<td>1.7 ± 0.3</td>
<td>1.5 ± 0.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.0 ± 0.4</td>
<td>1.0 ± 0.4</td>
<td>1.0 ± 0.4</td>
<td>0.10</td>
</tr>
<tr>
<td>Deceleration time, ms</td>
<td>211 ± 74</td>
<td>199 ± 68</td>
<td>213 ± 75</td>
<td>0.11</td>
</tr>
<tr>
<td>E/E’ ratio</td>
<td>13 ± 6</td>
<td>15 ± 8</td>
<td>13 ± 5</td>
<td>0.05</td>
</tr>
<tr>
<td>Moderate or severe MR</td>
<td>44 (7%)</td>
<td>14 (17%)</td>
<td>30 (6%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Moderate or severe PR</td>
<td>2 (0.3%)</td>
<td>0 (0%)</td>
<td>2 (0.4%)</td>
<td>0.60</td>
</tr>
<tr>
<td>Moderate or severe TR</td>
<td>24 (4%)</td>
<td>5 (7%)</td>
<td>19 (4%)</td>
<td>0.17</td>
</tr>
<tr>
<td>LA volume index, mL/m²</td>
<td>16 ± 6</td>
<td>17 ± 6</td>
<td>16 ± 6</td>
<td>0.29</td>
</tr>
<tr>
<td>RVDA, cm²</td>
<td>15 ± 4</td>
<td>17 ± 5</td>
<td>15 ± 4</td>
<td>0.001</td>
</tr>
<tr>
<td>RVSA, cm²</td>
<td>10 ± 4</td>
<td>11 ± 4</td>
<td>9 ± 3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RVFAC, %</td>
<td>37 ± 9</td>
<td>33 ± 8</td>
<td>39 ± 9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>TAPSE</td>
<td>1.7 ± 0.2</td>
<td>1.6 ± 0.3</td>
<td>1.7 ± 0.2</td>
<td>0.07</td>
</tr>
<tr>
<td>RV strain, %</td>
<td>−22 ± 7</td>
<td>−17 ± 7</td>
<td>−22 ± 7</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

PR indicates pulmonary regurgitation.

### Table 3. Cox Univariable Predictors for the Composite End Point

<table>
<thead>
<tr>
<th></th>
<th>HR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, per 1-y increase</td>
<td>1.05</td>
<td>1.02–1.07</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Killip class ≥2, yes/no</td>
<td>5.05</td>
<td>3.15–8.08</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Prior myocardial infarction, yes/no</td>
<td>2.57</td>
<td>1.42–4.65</td>
<td>0.002</td>
</tr>
<tr>
<td>Multivessel disease, yes/no</td>
<td>1.88</td>
<td>1.21–2.92</td>
<td>0.005</td>
</tr>
<tr>
<td>Peak cTnT level, per 1 µg/L increase</td>
<td>1.02</td>
<td>1.02–1.03</td>
<td>0.001</td>
</tr>
<tr>
<td>Peak cTnT level, per 1 µg/L increase</td>
<td>1.08</td>
<td>1.06–1.10</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEF, per 5 mL increase</td>
<td>1.07</td>
<td>1.03–1.12</td>
<td>0.001</td>
</tr>
<tr>
<td>LVF, per 1% increase</td>
<td>0.93</td>
<td>0.91–0.96</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>WMSI, per 1 unit increase</td>
<td>10.95</td>
<td>5.02–23.90</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RVFAC, %</td>
<td>3.38</td>
<td>1.90–6.02</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RVFAC, %</td>
<td>0.94</td>
<td>0.92–0.97</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RVFAC &lt;32%, yes/no</td>
<td>2.22</td>
<td>1.39–3.54</td>
<td>0.001</td>
</tr>
<tr>
<td>TAPSE, per 1 cm increase</td>
<td>0.10</td>
<td>0.03–0.38</td>
<td>0.001</td>
</tr>
<tr>
<td>TAPSE &lt;1.5 cm, yes/no</td>
<td>4.00</td>
<td>2.45–6.53</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RV strain, per 1% increase</td>
<td>1.10</td>
<td>1.06–1.14</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>RV strain &lt; −22.1%, yes/no</td>
<td>3.43</td>
<td>1.87–6.29</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Table 4. Cox Multivariable Model With RVFAC for the Composite End Point

<table>
<thead>
<tr>
<th>Killip class ≥2, yes/no</th>
<th>HR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multivessel disease, yes/no</td>
<td>1.92</td>
<td>1.04–3.56</td>
<td>0.04</td>
</tr>
<tr>
<td>Peak cTnT level, per 1 µg/L increase</td>
<td>1.05</td>
<td>1.01–1.08</td>
<td>0.008</td>
</tr>
<tr>
<td>LVEF, per 1% increase</td>
<td>0.95</td>
<td>0.91–0.99</td>
<td>0.01</td>
</tr>
<tr>
<td>RVFAC, per 1% increase</td>
<td>0.96</td>
<td>0.92–0.99</td>
<td>0.007</td>
</tr>
</tbody>
</table>

Table 5. Cox Multivariable Model With RV Strain for the Composite End Point

<table>
<thead>
<tr>
<th>Killip class ≥2, yes/no</th>
<th>HR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multivessel disease, yes/no</td>
<td>2.03</td>
<td>1.07–3.86</td>
<td>0.03</td>
</tr>
<tr>
<td>Peak cTnT level, per 1 µg/L increase</td>
<td>1.06</td>
<td>1.01–1.10</td>
<td>0.01</td>
</tr>
<tr>
<td>LVEF, per 1% increase</td>
<td>0.95</td>
<td>0.91–0.99</td>
<td>0.01</td>
</tr>
<tr>
<td>RV strain, per 1% increase</td>
<td>1.08</td>
<td>1.03–1.13</td>
<td>0.002</td>
</tr>
</tbody>
</table>

During a mean follow-up of 24±15 months, 86 patients (14%) reached the composite end point: 51 patients died (8%), 16 patients (3%) had a nonfatal reinfarction, and 29 patients (5%) were hospitalized for HF. Differences in clinical and echocardiographic characteristics between patients who reached the composite end point and patients who remained event-free are shown in Tables 1 and 2.

**RV Function and Association With Outcome**

Table 3 shows the significant univariable predictors of the composite end point. In addition to clinical characteristics and echocardiographic measurements of LV function, RV function significantly predicted worse outcome. RVFAC, TAPSE, and RV strain were univariable predictors of the composite end point. After adjusting RVFAC, TAPSE, and RV strain for other variables that predicted adverse outcome, RVFAC and RV strain independently predicted the occurrence of the composite end point (Tables 4 and 5). However, TAPSE did not remain significant in the multiple variable analysis (hazard ratio [HR], 0.88; 95% CI, 0.16 to 4.81; P=0.88).

Incremental Value of RV Function in Addition to Traditional Risk Factors

Global \( \chi^2 \) scores were calculated to assess the incremental value of RV function. RV function quantified by RVFAC and RV strain provided incremental value to clinical information (age and Killip class ≥2), infarct characteristics (RCA as culprit vessel, multivessel disease, and peak cTnT level), and LV systolic and diastolic function (LVEF, WMSI, and E/E’ ratio and moderate or severe MR). In addition, RV strain was added to the RVFAC model, which was demonstrated to increase the predictive power of the model even further (Figure 2).

**Discussion**

The major finding of the present study was that RVFAC, TAPSE, and RV strain were strong predictors of the composite end point all-cause mortality, reinfarction, and hospitalization for HF. In addition, the prognostic value of several traditional risk factors, including Killip class, peak cardiac enzymes, multivessel disease, and LV function, was again confirmed. After adjusting for known risk factors of adverse outcome after AMI, RVFAC (HR, 0.96; 95% CI, 0.92 to 0.99) and RV strain (HR, 1.08; 95% CI, 1.03 to 1.13) independently predicted the composite end point. In addition, the cut-off for RV strain at <−22.1% was associated with an adjusted HR of 2.18 for the occurrence of the composite end point. Moreover, RV strain <−22.1% provided incremental value over clinical information, infarct characteristics, LV function, and RVFAC for the prediction of adverse outcome in post-AMI patients. However, RV function failed to provide prognostic information for the prediction of nonfatal reinfarction individually.

**Quantification of RV Function**

Multiple methods have been described to quantify RV function with 2D echocardiography. In clinical practice, qualita-
ative assessment of RV function is usually performed, whether or not in combination with TAPSE or RVFAC. Both measurements are simple to perform and associated with prognosis, particularly in patients with LV dysfunction after AMI. In contrast to previous studies, the current study evaluated the importance of RV function in a large population of post-AMI patients treated with primary PCI and relatively preserved LV function. In addition to TAPSE and RVFAC, we assessed RV strain. Although RVFAC, TAPSE, and RV strain are highly correlated, they measure different aspects of RV function. RVFAC is the most commonly used measurement to assess RV contractility. However, the measurement of RVFAC is experience-dependent and reproducibility is often poor. Therefore, RVFAC may not adequately reflect contractility. TAPSE is another frequently used measurement to assess RV function and reflects the longitudinal systolic excursion of the lateral tricuspid valve annulus, which may not fully reflect RV contractility. Strain is a novel technique that enables angle-independent measurement of active myocardial deformation. Previous studies indicated that subtle but clinically relevant decreases in ventricular function can be detected using strain, and we therefore hypothesized that this may also apply for subtle changes in RV function after AMI. Peak RV longitudinal strain, which quantifies the maximal shortening in the RV free wall from apex to base, is likely to be a good estimator of RV function because 80% of the stroke volume is generated by longitudinal shortening of the RV free wall.

Figure 1. Cumulative incidence of adverse events. Patients are stratified by RVFAC (A), tricuspid annular plane systolic excursion (B), and RV strain (C).
Clinical information (age, Killip class) value to known risk factors for adverse outcome related to -axis). Addition of RVFAC and RV strain provides incremental y-function for the prediction of long-term outcome has been underestimate. Although RV dysfunction was reported to recover to some extent after AMI, recently the value of RV function has been well recognized in patients with inferior AMI and LV function, was demonstrated to be of incremental value in addition to RVFAC. In addition, RV strain may detect RV dysfunction earlier than RVFAC because the Kaplan-Meier estimates showed earlier divergence for RV strain than RVFAC (Figure 1).

TAPSE has been found to correlate with LVEF, which is an important predictor of adverse outcome in AMI patients and thus may explain the earlier separation on the graphs. This indicates that RV strain may be superior to traditional measures of RV function for the prediction of adverse events after AMI.

**RV Function and Outcome**

In the past, the clinical importance of RV function has been underestimated. Although RV dysfunction was reported to recover to some extent after AMI, recently the value of RV function for the prediction of long-term outcome has been well recognized in patients with inferior AMI and LV dysfunction. Mehta et al. showed in a meta-analysis that RV strain, even after correction for clinical information, infarct characteristics, and LV function, was demonstrated to be of incremental value in addition to RVFAC. In addition, RV strain may detect RV dysfunction earlier than RVFAC because the Kaplan-Meier estimates showed earlier divergence for RV strain than RVFAC (Figure 1).

The results of the current study point out for the first time, to our knowledge, that reduced strain of the RV is a strong independent predictor of adverse events in post-AMI patients. RV strain, even after correction for clinical information, infarct characteristics, and LV function, was demonstrated to be of incremental value in addition to RVFAC. In addition, RV strain may detect RV dysfunction earlier than RVFAC because the Kaplan-Meier estimates showed earlier divergence for RV strain than RVFAC (Figure 1).

TAPSE has been found to correlate with LVEF, which is an important predictor of adverse outcome in AMI patients and thus may explain the earlier separation on the graphs. This indicates that RV strain may be superior to traditional measures of RV function for the prediction of adverse events after AMI.

**RV Strain**

Although strain was primarily developed for the measurement of LV deformation, previous reports have demonstrated the usefulness of RV strain in several populations to detect subtle changes in RV function. Measurement of longitudinal strain of the RV is a reliable method for the assessment of RV function, because 80% of the stroke volume is generated by longitudinal shortening of the RV free wall. To the best of our knowledge, this is the first study to examine the value of RV strain in post-AMI patients. RV strain provided incremental value to traditional measurements of RV function, and the quantification of RV strain is simple to perform and highly feasible.

**Clinical Implications**

The results of the current study suggest that routine assessment of RV function should be implemented in the follow-up of AMI patients. RV strain measured early after AMI appeared to be superior to RVFAC and TAPSE for the risk stratification of AMI patients and could facilitate in the identification of patients who are at risk for adverse events.

**Limitations**

RV infarction complicates about 50% of inferior AMI. ST-segment elevations and Q waves in the right precordial leads have shown to have a high diagnostic accuracy for RV infarctions. Unfortunately, in the present study, right precordial leads were not applied during electrocardiography; however, no significant differences in RV function parameters were observed in patients with and without inferior infarction.

For the current study, the cut-off for RV strain was chosen at 2 SD from the normal RV strain in a group of 60 healthy subjects. Normal limits of RV strain derived from larger populations are currently lacking. Therefore, future research should aim at defining normal limits for RV strain and validating these cut-offs in relation to clinical end points.

Although RV function at baseline was a good predictor of outcome in AMI patients, the predictive value of RV function at different periods after AMI could not be addressed. Changes in RV function could occur in the first weeks after AMI, and serial assessment of RV function would be interesting.

**Conclusions**

RV function provides strong prognostic information in AMI patients treated with primary PCI. RV strain is an indepen-
dent predictor of all-cause mortality, reinfarction, and hospi-
talization for HF. In addition, RV strain provides incremental
value over clinical information, infarct characteristics, LV
function, and RVFAC. Quantitative assessment of RV func-
tion with RV strain may improve the risk stratification of
patients after AMI.

Disclosures
Dr Bax received grants from GE Healthcare, BMS Medical Imaging,
St Jude Medical, Medtronic, Boston Scientific, Biotronik, and
Edwards Lifesciences. Dr Schalij received grants from Boston
Scientific, Medtronic, and Biotronik.

References
1. Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M,
Hochman JS, Krumholz HM, Kushner FG, Lamas GA, Mullany CJ,
Ornato JP, Pearle DL, Sloan MA, Smith SC Jr, Alpert JS, Anderson JL,
LF, Hunt SA, Jacobs AK. ACC/AHA guidelines for the management
of patients with ST-elevation myocardial infarction–executive summary: a
report of the American College of Cardiology/American Heart Asso-
ciation Task Force on Practice Guidelines (Writing Committee to Revise
the 1999 Guidelines for the Management of Patients With Acute Myo-
2. Moller JE, Hillis GS, Brandt PW, Whitlock RM, Wild CJ. Left
ventricular end-systolic volume as the major determinant of survival
Right ventricular function assessed by two-dimensional strain and
longitudinal strain is a sensitive marker for right ventricular deterioration
M, Just H. Right ventricular infarction as an independent predictor of
5. Amundsen BH, Helle-Valle T, Edvardsen T, Torp H, Crosby J, Lyseggen
JG, Bosch J, Viergever EP, van RC, Padmos I, Sedney MI, van Exel HJ,
Matsunaga A, Duran CM. Right ventricular dysfunction and risk of heart failure and mortality after acute myocardial infarction. Am J Cardiol.
6. Sutherland GR, Di SG, Claus P, D’hooge J, Bijnen B. Strain and strain
rate imaging: a new clinical approach to quantifying regional myocardial
Doeyden MS, Tajik AJ. Clinical utility of Doppler echocardiography and tissue
doppler imaging in the estimation of left ventricular filling pressures: a
8. Samad BA, Alam MJ, Jensen-Urstad K. Prognostic impact of right ven-
tricular involvement as assessed by tricuspid annular motion in patients
James C, Tichnell C, Russell S, Judge D, Corretti M, Bluemke D, Calkins H,
Abraham TP. Utility of tissue Doppler and strain echocardiography in
arrhythmogenic right ventricular dysplasia cardiomyopathy. Am J Cardiol.
10. Giusca S, Dambrauskaite V, Scheurwegs C, D’hooge J, Claus P, Defor-
11. Poppescu BA, Antonini-Canneri F, Temporelli PL, Gianuzzi P, Bosimini
E, Gentile F, Maggioni AP, Tavazzi L, Piazza R, Ascione L, Stoian I,

patients with acute myocardial infarction. Am J Heart. 2007;153:
14.e1–e11.
12. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka
PA, Picard MH, Roman MJ, Seward J, Shanewise JS, Solomon SD,
Spencer KT, Sutton MS, Stewart WJ. Recommendations for chamber
quantification: a report of the American Society of Echocardiography’s
Guidelines and Standards Committee and the Chamber Quantification
Writing Group, developed in conjunction with the European Association
of Echocardiography, a branch of the European Society of Cardiology.
13. Zogbi WA, Enriquez-Sarano M, Foster E, Grayburn PA, Kraft CD,
Levine RA, Nihoyannopoulos P, Otto CM, Quinones MA, Rakowski H,
Stewart WJ, Waggoner A, Weissman NJ. Recommendations for eval-
uation of the severity of native valvular regurgitation with two-
14. Matsunaga A, Duran CM. Progression of tricuspid regurgitation after
repaired functional ischemic mitral regurgitation. Circulation. 2005;112:1-
453–1457.
15. Naqvi TZ, Padmanabhan S, Raffi F, HyluHN, Mirocha J. Comparison
of usefulness of left ventricular diastolic versus systolic function as a
predictor of outcome following primary percutaneous coronary angio-
16. Lester SJ, Tajiik AJ, Nishimura RA, Oh JK, Khandheria BK, Seward JB.
Unlocking the mysteries of diastolic function: deciphering the Rosetta
17. Naqvi TZ, Padmanabhan S, Raffi F, HyluHN, Mirocha J. Comparison
of usefulness of left ventricular diastolic versus systolic function as a
predictor of outcome following primary percutaneous coronary angio-
18. Lester SJ, Tajiik AJ, Nishimura RA, Oh JK, Khandheria BK, Seward JB.
Unlocking the mysteries of diastolic function: deciphering the Rosetta
19. Samad BA, Alam MJ, Jensen-Urstad K. Prognostic impact of right ven-
tricular involvement as assessed by tricuspid annular motion in patients
20. Sutherland GR, Di SG, Claus P, D’hooge J, Bijnen B. Strain and strain
rate imaging: a new clinical approach to quantifying regional myocardial
21. Alpert JS, Thyesen K, Antman E, Bassand JP. Myocardial infarction
redefined: a consensus document of The Joint European Society of
Cardiology/American College of Cardiology Committee for the redefi-
Doeyden MS, Tajik AJ. Echocardiographic tissue deformation imaging of right ventricular systolic function in endurance athletes. Eur Heart J.
2009;30:969–977.
James C, Tichnell C, Russell S, Judge D, Corretti M, Bluemke D, Calkins
H, Abraham TP. Utility of tissue Doppler and strain echocardiography in
arrhythmogenic right ventricular dysplasia cardiomyopathy. Am J Cardiol.
24. Giusca S, Dambranskaite V, Scheurwegs C, D’hooge J, Claus P, Defor-
1436–1448.
26. Carlson M, Ugander M, Heiberg E, Arheden H. The quantitative rela-
tionship between longitudinal and radial function in left, right, and total
heart pumping in humans. Am J Physiol Heart Circ. Physiol. 2007;293:
H636–H644.
27. Haddad F, Doyle R, Murphy DJ, Hunt SA. Right ventricular function in cardiovascular disease, II: pathophysiology, clinical importance, and
management of right ventricular failure. Circulation. 2008;117:
1717–1731.
28. Poppescu BA, Antonini-Canneri F, Temporelli PL, Gianuzzi P, Bosimini
E, Gentile F, Maggioni AP, Tavazzi L, Piazza R, Ascione L, Stoian I,
Cereda E, Poppescu AC, Nicolosi GL. Right ventricular functional recovery after acute myocardial infarction: relation with left ventricular function and septal ventricle septum motion. GISSI-3 echo substudy. Heart.
CLINICAL PERSPECTIVE

The prognosis of patients after acute myocardial infarction (AMI) is determined by the interaction of a large number of factors. Besides the importance of clinical parameters, several studies have described the importance of left ventricular function as one of the most important prognostic parameters. On the other hand, data on the association between right ventricular (RV) function and adverse events after AMI are scarce. In the current study, the prognostic value of RV function in 612 consecutive patients admitted with AMI treated with primary percutaneous coronary intervention, who underwent echocardiography within 48 hours of admission, was evaluated. RV function was quantified with RV fractional area change, tricuspid annular plane systolic excursion, and RV strain. During a mean follow-up of 24 months, 86 patients reached the end point defined as a composite of all-cause mortality, reinfarction, and hospitalization for heart failure. RV fractional area change (hazard ratio, 0.96; 95% CI, 0.92 to 0.99) and RV strain (hazard ratio, 1.08; 95% CI, 1.03 to 1.13) were both independent predictors of the composite end point. In addition, RV strain provided incremental value to baseline clinical information, infarct characteristics, left ventricular function, and RV fractional area change for the prediction of the composite end point. In conclusion, RV function provides useful prognostic information in patients treated with primary percutaneous coronary intervention for AMI.
Prognostic Value of Right Ventricular Function in Patients After Acute Myocardial Infarction Treated With Primary Percutaneous Coronary Intervention
M. Louisa Antoni, Roderick W.C. Scherptong, Jael Z. Atary, Eric Boersma, Eduard R. Holman, Ernst E. van der Wall, Martin J. Schalij and Jeroen J. Bax

Circ Cardiovasc Imaging. 2010;3:264-271; originally published online February 27, 2010; doi: 10.1161/CIRCIMAGING.109.914366

Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2010 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circimaging.ahajournals.org/content/3/3/264