The Assessment of Area at Risk and Myocardial Salvage After Coronary Revascularization in Acute Myocardial Infarction
Comparison Between CMR and SPECT

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OBJECTIVES  This study sought to compare cardiac magnetic resonance (CMR) and single-photon emission computed tomography (SPECT) for assessment of area at risk, scar size, and salvage area after coronary reperfusion in acute myocardial infarction.

BACKGROUND  Myocardial salvage is an important surrogate endpoint assessing the success of coronary reperfusion in acute myocardial infarction. SPECT, the established modality for assessment of myocardial salvage, requires radiopharmaceutical injection before revascularization and 2 examinations. The combination of T2 and late enhancement imaging in CMR can assess myocardial salvage in 1 examination, but up to now, data comparing both modalities are very limited.

METHODS  We analyzed 207 patients who were treated by primary revascularization in acute myocardial infarction and who underwent both SPECT and CMR for assessment of myocardial salvage. In CMR, T2-weighted turbo spin echo sequences for area at risk and contrast-enhanced inversion recovery gradient echo sequences were performed.

RESULTS  Image quality was insufficient in 27 patients (13%). In the remaining 180 patients, mean area at risk was 29.4 ± 18.7% of the left ventricle (LV), and infarct size was 14.7 ± 16.9% LV, resulting in a mean salvage area of 14.9 ± 15.1% LV in SPECT, whereas in CMR, mean area at risk was 28.0 ± 14.5% LV, and infarct size was 16.0 ± 13.5% LV, resulting in a mean salvage area of 11.9 ± 12.3%. Results of both modalities correlated well for area at risk (r = 0.80), scar size (r = 0.87), and salvage area (r = 0.66, all p < 0.0001).

CONCLUSIONS  Assessment of the salvage area by CMR using T2 and late enhancement imaging correlates well with the established modality of SPECT. CMR therefore may be an alternative to paired SPECT imaging for myocardial salvage assessment, but the contraindications of the modality and limitations in the established T2 imaging sequences currently cause a considerable rate of data loss. (J Am Coll Cardiol Img 2013;6:358–69) © 2013 by the American College of Cardiology Foundation
Myocardial salvage in CMR and SPECT

MATERIALS AND METHODS

Study population and design. Included in this study were all patients with acute ST-segment elevation myocardial infarction (STEMI) or non–ST-segment elevation myocardial infarction (NSTEMI) undergoing both contrast-enhanced CMR and Tc99m sestamibi myocardial perfusion SPECT for assessment of myocardial salvage after primary angioplasty between October 1, 2006, and October 1, 2011.

All patients who were treated by primary angioplasty for acute myocardial infarction and had no contraindications for CMR (pacemaker, internal defibrillator or other incompatible intracorporal foreign bodies, creatinine clearance below 50 ml/min [since May 2007], hemodynamic instability for >7 days after infarction, claustrophobia) were eligible for CMR.

Patients were diagnosed with acute myocardial infarction if they presented with chest pain lasting at least 20 min associated with electrocardiographic (ECG) changes (ST-segment elevation, new-onset left bundle branch block) and/or elevated cardiac enzyme levels and if the onset of symptoms was <72 h before PCI (12). Patients with prior myocardial infarction were excluded to ensure that CMR findings displayed acute myocardial injury. All patients gave written informed consent for both investigations.

Immediately after hospital admission, all patients were treated with PCI of the infarct-related artery. For the SPECT scan, patients received an intravenous injection of 1,000 MBq of technetium-99m (Tc99m) sestamibi before the initiation of the revascularization procedure. Image acquisition was performed within 6 to 8 h after the intervention. The SPECT scan for scar size assessment and the cardiac CMR were scheduled to take place 5 to 7 days after PCI.

Hypertension, hypercholesterolemia, diabetes, and a family history of premature coronary artery disease were defined by published criteria (13–16). Patients’ individual clinical and periprocedural risk was assessed by calculating TIMI (Thrombolysis In Myocardial Infarction) (17,18) and GRACE (Global Registry of Acute Coronary Events) risk scores (19). The latter is a group of scores created at the Center for Outcomes Research of the University of Massachusetts in a large multinational registry of patients with acute coronary syn-
dromes for prediction of morbidity and mortality during the initial hospital stay and during follow-up. The GRACE score used in this study assessed death or myocardial infarction between admission and 6 months later, and includes age, heart rate, systolic blood pressure, creatinine, Killip class, cardiac arrest at admission, elevated cardiac markers, and ST-segment deviation (20).

The study protocol was approved by the institutional ethics committee.

Myocardial perfusion SPECT. The methods used for the radionuclide studies have been previously described in detail (21,22). Image acquisition was performed with the patient in the supine position. Dual-head camera systems with low-energy, high-resolution collimators were used for the radionuclide studies. Images were acquired ECG-gated in a 64-by-64 matrix with an acquisition time of 40 s per image. A volumetric sampling tool was applied to create polar maps of the relative distribution of activity throughout the left ventricle (LV). Each polar map was adjusted for its own maximal value. The size of the defect was calculated with the use of a threshold of 50%, according to previously described methods (23,24). The difference between these immediate and follow-up measurements provided the degree of myocardial salvage. Initial perfusion defect, infarct size, and degree of myocardial salvage were expressed as a proportion of the LV.

Cardiac magnetic resonance. CMR was performed on a 1.5-T system (Siemens Avanto, Siemens Medical Solutions, Erlangen, Germany) equipped with a dedicated cardiac phased-array surface coil. For image acquisition, patients were positioned in a supine position, and images were acquired at repeated end-expiratory breath holds with ECG gating. Area at risk was assessed by T2-weighted turbo spin echo sequence acquired before contrast injection (slice thickness 8 mm; repetition time 2 RR intervals; echo time 99 ms; image matrix 145 × 192). The infarct scar was assessed 15 min after injection of 0.2 mmol/kg body weight of dimegluminingadopentetat (Magn evacist, Bayer HealthCare Pharmaceuticals, Berlin, Germany, until June 2009, Magnograf, Marotstrat, Jena, Germany, thereafter) on T1-weighted inversion-recovery turbo fast low-angle shot sequence (slice thickness 8 mm; repetition time 4.0 ms; echo time 1.5 ms; image matrix 175 × 256; flip angle 30°). The inversion time was individually adjusted to null normal myocardium. For both acquisitions, contiguous short-axis slices of the LV from base to apex, as well as 2- and 4-chamber views of the LV, were acquired at the same location. The CMR study was performed 4.1 days [interquartile range (IQR): 3.6 to 4.9 days] after PCI, and the median time interval between CMR and the second SPECT study was 3.1 h [IQR: 1.4 to 25.1 h].

For defect quantification, endocardial and epicardial contours were manually traced on each of the short-axis slices by an experienced reader. The defect size was then calculated automatically by comparison with manually marked, healthy remote myocardium and was expressed as the percentage of total LV myocardial volume (25).

Area at risk was defined as a region of hypoenhanced myocardium with signal intensity above 2 standard deviations of healthy remote myocardium as proposed by Friedrich et al. (9). For infarcted myocardium, the same algorithm was used with a threshold of 3 standard deviations, because at this cutoff, the correlation between CMR and SPECT is best (26). In consensus with previous studies, a defect was required to have at least 10 contiguous myocardial pixels of increased signal intensity (9,27). Microvascular obstruction as an area of hypoenhancement within the infarcted myocardium was delineated manually, since there is no robust automated detection algorithm available. Care was taken not to miss tissue between the manually delineated and the automatically detected scar areas. Microvascular obstruction was included in the infarct size calculation. Salvage area in CMR was defined as the difference between T2 defect and total infarct size assessed by late enhancement imaging. Images of SPECT and CMR were analyzed by different observers blinded to the results of the other modality. For image examples describing infarct characterization, see Figure 1.

Statistical analysis. Categorical variables were expressed as frequencies and percentages, continuous variables were expressed as mean ± SD or, for the more skewed clinical parameters and time intervals, as median [IQR]. Comparison between continuous variables was done using the Student t test or Wilcoxon test as appropriate; for categorical variables, the Fisher exact test was used. Correlation between CMR and SPECT was calculated using a linear regression model. Correlation coefficients were compared using Fisher’s Z-transform (28); correlation with clinical parameters was calculated using Spearman’s r. Statistical significance was accepted for 2-sided p values <0.05. The statistical
package R version 2.10.1 (29) was used for statistical analysis.

RESULTS

Study population and clinical characteristics. During the study period, 1,368 patients with acute myocardial infarction were treated by primary PCI. Of these, 254 had a recurrent infarction; 441 of the 1,114 patients with primary infarction underwent SPECT imaging for assessment of area at risk and scar size. From these, 207 patients also underwent CMR for T2 imaging and late enhancement imaging. Of these, 27 were excluded because the image quality of the CMR study was insufficient for automated analysis (all in T2 studies), resulting in a study population of 180 patients.

From the 441 patients undergoing SPECT, 26 patients (6%) had creatinine levels above 1.5 mg/dl at the time of discharge, 8 patients (2%) had a permanent pacemaker or an internal defibrillator, so in total, 8% of the patients undergoing SPECT were ineligible for CMR. Reasons for not performing CMR in the remaining patients were missing consent of the patients because of claustrophobia or other reasons, and logistical problems in scheduling the exam before discharge.

The age of the patients was 61 [IQR: 52 to 70] years, and 139 patients (77%) were male. The majority of the patients (149, or 87%) presented in stable condition in Killip class 1. STEMI was diagnosed in 121 patients (67%), whereas 59 patients (33%) had NSTEMI. A detailed summary of the patients’ characteristics is provided in Table 1.

Results of SPECT and CMR. In SPECT imaging, mean area at risk was 29.4 ± 18.7% LV, and infarct...
size was 14.7 ± 16.9% LV, resulting in a mean salvage area of 14.9 ± 15.1% LV. In the CMR studies, mean area at risk was 28.0 ± 14.5% LV, and total infarct size was 16.0 ± 13.5% LV, resulting in a mean salvage area of 11.9 ± 12.3%.

For the area at risk, there was no significant

Table 1. Clinical Characteristics and Conventional Risk Scores

<table>
<thead>
<tr>
<th></th>
<th>All Patients</th>
<th>NSTEMI (n = 59)</th>
<th>STEMI (n = 121)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>60.5 [51.5–69.8]</td>
<td>62.2 [53.1–70.0]</td>
<td>58.6 [50.6–69.8]</td>
<td>0.29</td>
</tr>
<tr>
<td>Male</td>
<td>139 (77)</td>
<td>40 (68)</td>
<td>99 (82)</td>
<td>0.039</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.2 [24.7–29.3]</td>
<td>27.1 [25.5–28.9]</td>
<td>27.2 [24.6–29.4]</td>
<td>0.57</td>
</tr>
<tr>
<td>Arterial hypertention</td>
<td>60 (33)</td>
<td>19 (32)</td>
<td>41 (34)</td>
<td>0.87</td>
</tr>
<tr>
<td>Diabetes</td>
<td>25 (14)</td>
<td>11 (19)</td>
<td>14 (12)</td>
<td>0.25</td>
</tr>
<tr>
<td>Current smoker</td>
<td>72 (40)</td>
<td>18 (31)</td>
<td>54 (45)</td>
<td>0.077</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>86 (48)</td>
<td>33 (56)</td>
<td>53 (44)</td>
<td>0.15</td>
</tr>
<tr>
<td>Family history of premature CAD</td>
<td>72 (40)</td>
<td>23 (39)</td>
<td>49 (41)</td>
<td>0.87</td>
</tr>
<tr>
<td>Prior revascularization</td>
<td>18 (10)</td>
<td>7 (12)</td>
<td>11 (9)</td>
<td>0.6</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>131 [120–150]</td>
<td>140 [128–150]</td>
<td>130 [120–146]</td>
<td>0.036</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>76 [66–85]</td>
<td>76 [67–85]</td>
<td>75 [64–85]</td>
<td>0.67</td>
</tr>
<tr>
<td>Creatinine, mg/dl</td>
<td>0.9 [0.8–1.0]</td>
<td>0.9 [0.8–1.0]</td>
<td>0.9 [0.8–1.1]</td>
<td>0.17</td>
</tr>
<tr>
<td>Killip class before PCI</td>
<td></td>
<td></td>
<td></td>
<td>0.13</td>
</tr>
<tr>
<td>1</td>
<td>149 (87)</td>
<td>48 (92)</td>
<td>101 (84)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>19 (11)</td>
<td>4 (8)</td>
<td>15 (13)</td>
<td></td>
</tr>
<tr>
<td>≥3</td>
<td>4 (2)</td>
<td>0 (0)</td>
<td>4 (3)</td>
<td></td>
</tr>
<tr>
<td>GRACE score</td>
<td>144 [117–166]</td>
<td>131 [109–159]</td>
<td>149 [122–170]</td>
<td>0.027</td>
</tr>
<tr>
<td>TIMI risk score</td>
<td>3 [1–5]</td>
<td>2 [1–4]</td>
<td>3 [1–5]</td>
<td>0.30</td>
</tr>
</tbody>
</table>

Values are median (interquartile range) or n (%). BMI = body mass index; CARG = coronary artery bypass graft; CAD = coronary artery disease; GRACE = Global Registry of Acute Coronary Events; NSTEMI = non-ST-segment elevation myocardial infarction; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction; TIMI = Thrombolysis in Myocardial Infarction.

Table 2. Infarct Characteristics

<table>
<thead>
<tr>
<th></th>
<th>All Patients</th>
<th>NSTEMI (n = 59)</th>
<th>STEMI (n = 121)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infarct location</td>
<td></td>
<td></td>
<td></td>
<td>0.21</td>
</tr>
<tr>
<td>Inferior</td>
<td>84 (47)</td>
<td>28 (48)</td>
<td>56 (46)</td>
<td></td>
</tr>
<tr>
<td>Lateral</td>
<td>23 (13)</td>
<td>9 (15)</td>
<td>12 (10)</td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>73 (40)</td>
<td>22 (37)</td>
<td>53 (44)</td>
<td></td>
</tr>
<tr>
<td>Cardiac markers (before PCI)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CK-MB, U/l</td>
<td>26 [17.5–59.2]</td>
<td>40.5 [25.7–70.3]</td>
<td>21.8 [16.4–49.9]</td>
<td>0.00033</td>
</tr>
<tr>
<td>Troponin T, ng/ml</td>
<td>0.15 [0.04–0.55]</td>
<td>0.37 [0.15–0.84]</td>
<td>0.08 [0.03–0.37]</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>TIMI flow before PCI</td>
<td></td>
<td></td>
<td></td>
<td>0.026</td>
</tr>
<tr>
<td>0</td>
<td>85 (47)</td>
<td>24 (41)</td>
<td>61 (50)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>18 (10)</td>
<td>2 (3.4)</td>
<td>16 (13)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>46 (26)</td>
<td>20 (34)</td>
<td>26 (22)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>31 (17)</td>
<td>13 (22)</td>
<td>18 (15)</td>
<td></td>
</tr>
<tr>
<td>TIMI flow grade post-PCI</td>
<td></td>
<td></td>
<td></td>
<td>0.64</td>
</tr>
<tr>
<td>0</td>
<td>2 (1.1)</td>
<td>0 (0)</td>
<td>2 (1.6)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>3 (1.7)</td>
<td>2 (3.39)</td>
<td>1 (0.8)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>15 (8)</td>
<td>3 (5)</td>
<td>12 (10)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>160 (89)</td>
<td>54 (92)</td>
<td>106 (88)</td>
<td></td>
</tr>
</tbody>
</table>

Values are median (interquartile range) or n (%). CK = creatine kinase; CK-MB = creatine kinase myocardial band; EF = ejection fraction; other abbreviations as in Figure 1.
difference between CMR and SPECT (difference $-1.4 \pm 11.3$, $p = 0.084$). CMR slightly overestimated infarct size when compared with SPECT (difference $1.3 \pm 8.4$, $p = 0.038$), resulting in a significantly smaller salvage area in CMR when compared with SPECT (difference $-2.8 \pm 11.5$, $p = 0.0015$).

Expressed as median and IQR, the size of the area at risk was 27.5% [IQR: 15.0 to 44.0] LV in SPECT and 28.9% [IQR: 18.6 to 39.4] LV in CMR ($p = 0.97$ for difference); infarct size was 7.0% [IQR: 1.7 to 25.0] LV in SPECT and 12.7% [IQR: 5.2 to 24.6] LV in CMR ($p = 0.06$ for difference), and salvage area was 9.0% [IQR: 3.0 to 16.5] LV in SPECT and 10.6% [IQR: 4.7 to 18.4] LV in CMR ($p = 0.22$ for difference).

Despite the overall good correlation between the 2 modalities, there were substantial differences in individual cases, as demonstrated in the imaging examples in Figure 1.

Although both area at risk and scar size were larger in STEMI patients than in NSTEMI patients (all $p < 0.009$), the salvage area showed no significant difference in both groups ($p = 0.66$ for SPECT and $p = 0.82$ for CMR). A detailed summary of the SPECT and CMR results is provided in Table 3.

Area at risk correlated well in both modalities ($r = 0.80$, $p < 0.0001$). Similar results were found for the scar size ($r = 0.87$, $p < 0.0001$). Correlation for salvage area was slightly weaker ($r = 0.66$); CMR underestimated the salvage area by more than 10% LV in 45 patients (25%) and overestimated by more than 10% LV in 24 patients (13%) when compared with SPECT (Fig. 2).

For external validation of the results, both modalities were compared with cardiac enzymes and cardiac function, as summarized in Table 4. For assessment of the area at risk, both SPECT and CMR T2 had no significant correlation with the initial cardiac markers, but a good correlation with the ejection fraction before PCI ($r = -0.61$ and $r = -0.46$, respectively). For scar size, both SPECT and CMR late enhancement correlated well with peak creatine phosphokinase ($r = 0.64$ and $r = 0.72$, respectively) and ejection fraction at the time of CMR ($r = -0.55$ and $r = -0.54$, respectively).

A subgroup analysis of the area at risk showed a consistently good correlation between the 2 modalities for different clinical parameters. There was no significant difference in correlation when comparing patients with STEMI and NSTEMI ($r = 0.80$ vs. $r = 0.76$, $p = 0.52$) and patients with a pre-interventional TIMI flow grade of 0 and a TIMI flow grade $\geq 1$ ($r = 0.75$ vs. $r = 0.82$, $p = 0.41$), but there was a trend towards a better correlation in patients with anterior and lateral wall infarction when compared with patients with inferior wall infarction ($r = 0.80$ vs. $r = 0.69$, $p = 0.10$) (Fig. 3).

Looking at the correlation to the time interval between start of symptoms and start of interventions, there was no significant correlation found in both modalities for the area at risk ($r = 0.05$, $p = 0.48$ for T2 CMR, $r = 0.08$, $p = 0.30$ for SPECT), scar size ($r = 0.04$, $p = 0.58$ for late gadolinium enhancement CMR, $r = 0.01$, $p = 0.92$ for SPECT), or salvage area ($r = 0.11$, $p = 0.14$ for CMR, $r = 0.11$, $p = 0.16$ for SPECT) (Fig. 4).

**DISCUSSION**

The key finding of this study is that both the area at risk assessed by T2 imaging in CMR and the salvage area calculated from the difference between this measurement and the total scar size in contrast-
enhanced CMR correlate well with the established method using Tc99m sestamibi SPECT.

**Area at risk.** T2-weighted imaging in CMR (30) and the acute perfusion scan in SPECT (23,31) assess slightly different pathophysiological changes of acute myocardial infarction. Triggered by an acute occlusion of an epicardial coronary artery, there will be impaired cellular metabolism causing electrolyte imbalances, release of water from protein bindings, and endothelial leakage, all leading to (reversible) cell swelling detectable by T2-weighted imaging (7,32–34). Although it could be demon-
Stratified in experimental studies that cell edema is detectable as early as 30 min after total occlusion of a coronary artery (35), the pathophysiological changes in clinical infarction are more variable, including subtotal coronary occlusion or spontaneous lysis of a total occlusion after varying time intervals (36,37). But it is also well known that the changes in T2 imaging persist for days and sometimes weeks after reperfusion, even when functional metabolism is recovering (7). Sestamibi, on the other hand, is easily incorporated into myocytes, but accumulates in the mitochondria because it cannot be metabolized further. Because sestamibi is rapidly excreted from blood circulation after injection, distribution in the myocardium is mainly influenced by the myocardial blood flow in the minutes after injection (38). Both methods may therefore produce different results in certain circumstances. CMR may show a smaller area at risk than SPECT in case of a very acute occlusion (which may not coincide with the start of symptoms); this may be the case in patient C in Figure 1. It may also show a larger area at risk after spontaneous lysis of a longer-lasting total occlusion or in case of a subtotal occlusion still allowing a basal cell metabolism. The inconclusive findings in patient B in Figure 1 could be explained by spontaneous lysis because the wall motion abnormality is out of proportion to the observed TIMI flow grade 2 distal to the culprit lesion.

Despite these methodical limitations, there was no significant difference in the area at risk between both modalities, and we found a good correlation between the 2 modalities. In addition, area at risk derived from both modalities correlated equally well with pre-intervention ejection fraction as a parameter for external validation.

There was a slight overestimation of the area at risk by CMR as compared with SPECT; this may be explained by the myocardial swelling, which leads to a thickening of the affected LV wall. This wall thickening is incorporated into the volumetric analysis of CMR but may be missed by the surface area–based quantification algorithm of SPECT. Another explanation could be an overestimation by detection of peri-infarct inflammation as suggested by Mewton et al. (39).

Our results confirm on a large scale 2 smaller studies by Carlsson et al. (10) and Sörensson et al. (11), who demonstrated similar correlations between manually traced lesions in CMR T2 imaging and SPECT in groups of 16 patients each. In addition, Berry et al. (40) found a similarly strong correlation ($r = 0.77$) between the area at risk assessed by T2-weighted CMR and the APPROACH (Alberta Provincial Project for Outcome Assessment in Coronary Heart Disease) lesion score, which estimates the area at risk by the actual location of the culprit lesion.

**Scar size and salvage area.** For scar size quantification, we could reproduce the good correlation between contrast-enhanced CMR and SPECT described by Ibrahim et al. (6) in a larger patient population. The same as described in their study, we found a slight overestimation of small scar size in CMR that can be explained by the higher spatial resolution of CMR compared with SPECT, resulting in the detection of small subendocardial scars missed by SPECT. Scar size in both modalities correlated equally well with functional parameters.
such as ejection fraction and end-systolic volume index and enzymatic infarct size.

There are several studies published assessing myocardial salvage assessed by CMR. Friedrich et al. (9) analyzed 92 patients with acute myocardial infarction and successful reperfusion, and found an area at risk consistently larger than the scar size in all patients, resulting in a salvage area of 16 ± 11% LV. Eitel et al. (41) analyzed 208 STEMI patients undergoing primary angioplasty and identified myocardial salvage, infarct size, and microvascular obstruction assessed by CMR as good predictors of major cardiac events during a 6-month follow-up. Larose et al. (42) found only a weak correlation

Figure 3. Subgroup Analysis for Area at Risk
Correlation between SPECT and CMR studies for the area at risk: NSTEMI versus STEMI (A), TIMI flow before intervention (B) and infarct localization (C). NSTEMI = non–ST-segment elevation myocardial infarction; TIMI = Thrombolysis In Myocardial Infarction; other abbreviations as in Figure 1.
between myocardial salvage assessed by CMR and LV dysfunction 6 months after the event in 101 patients undergoing early revascularization in STEMI, whereas acute LV function and the size of late gadolinium enhancement and microvascular obstruction showed better correlations.

But to date, no data exist on the direct comparison between myocardial salvage assessed by CMR and the current gold standard of SPECT. We compared the salvage areas derived from both modalities in a population of 180 patients with successful reperfusion in acute myocardial infarction, and despite the aforementioned methodical differences, we found a reasonably good correlation between the 2 modalities; but in 38% of the patients, the results of both methods diverged by more than 10% of the LV volume, a difference that may be clinically significant. Although the weaker correlation between the 2 modalities in this derived parameter can easily be explained mathematically, the timing of the examination may be an additional confounder. Infarct healing is still in progress during the first week after infarction, and infarct size as measured by CMR decreases by about 7% between day 1 and 7 (43). Therefore, the time interval between CMR and SPECT measurement may add to the variability between the 2 modalities.

The suboptimal correlation between CMR and SPECT has to be kept in mind when assessing the success of a coronary intervention by CMR. Follow-up studies are clearly needed to finally validate this method. Limitations of CMR. For assessment of salvage area, CMR has 2 clear advantages over SPECT. It is not associated with radiation exposure and only needs 1 exam about 5 days after the acute event when acute therapy normally is finished and the patient is fairly stabilized. But there are clear drawbacks, too. First, CMR has additional contraindications compared with SPECT, most of all the presence of pacemakers and implantable defibrillators and an impaired renal function. In addition, the procedure is not always tolerated well by the patients, due to claustrophobia or reluctance against a long examination, which may further complicate patient recruitment. Of all patients undergoing SPECT during the study period, 2% of the patients had a pacemaker or defibrillator, and 6% had impaired renal function at discharge.

Second, the T2-weighted turbo spin echo sequence has a low contrast-to-noise ratio, necessitating a quite low threshold of 2 SD for edema detection and is prone to artifacts that may necessitate individual sequence adjustments. Tachycardia, arrhythmias, and the sequela of hemodynamic impairment such as pleural effusion or congestive heart failure, all common, particularly after large infarctions, limit the image quality or prevent a valid image analysis at all. The exclusion rate of 13% because of insufficient image quality found in our analysis clearly limits the use of CMR as a surrogate endpoint in a clinical study.

There are new, promising developments in sequence technology that probably improve image quality, namely T2 mapping sequences and automated motion correction algorithms. These improvements may provide the robustness to dependably examine even hemodynamically compromised
patients and allow for further simplification and standardization, both of image acquisition and analysis.

**CONCLUSIONS**

We could demonstrate that the assessment of the area at risk by T2-weighted CMR imaging in CMR correlates well with the established modality of SPECT in patients undergoing primary PCI in acute myocardial infarction. A reasonably good correlation could be found between the salvage area derived from T2 imaging and scar quantification by late enhancement imaging in CMR and the salvage area derived from SPECT. CMR may therefore be an alternative to paired SPECT imaging for myocardial salvage assessment, but the contraindications of the modality and limitations in the established T2 imaging sequences currently may cause a considerable rate of data loss.

**Acknowledgments**
The authors thank the medical and technical staff members of the magnetic resonance tomography laboratory for their invaluable contribution.

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Key Words: area at risk, cardiac magnetic resonance, myocardial infarction, salvage area, Tc99m sestamibi SPECT.