The aim of the study was to determine the prognostic significance and determinants of myocardial salvage assessed by cardiovascular magnetic resonance (CMR) in reperfused ST-segment elevation myocardial infarction.

In acute myocardial infarction, CMR can retrospectively detect the myocardium at risk and the irreversible injury. This allows for quantifying the extent of salvaged myocardium after reperfusion as a potential strong end point for clinical trials and outcome.

We analyzed 208 consecutive ST-segment elevation myocardial infarction patients undergoing primary angioplasty 12 h after symptom onset. T2-weighted and contrast-enhanced CMR was used to calculate the myocardial salvage index (MSI). Patients were categorized into 2 groups defined by the median MSI. The primary end point of the study was occurrence of major adverse cardiovascular events defined as death, reinfarction, and occurrence of new congestive heart failure within 6 months after the index event.

The median MSI was 48 (interquartile range 27 to 73). Major adverse cardiovascular events were significantly lower in the MSI ≥ median group (2.9% vs. 22.1%, p < 0.001). The stepwise Cox proportional hazards model revealed that the MSI was the strongest predictor of major adverse cardiovascular events at 6-month follow-up (p < 0.001). All prognostic clinical (symptom onset to reperfusion), angiographic (Thrombolysis In Myocardial Infarction flow grade before angioplasty), and electrocardiographic (ST-segment resolution) parameters showed significant correlations with the MSI (p < 0.001 for all).

This study for the first time demonstrates that the MSI assessed by CMR predicts the outcome in acute reperfused ST-segment elevation myocardial infarction. Therefore, MSI assessment has important implications for patient prognosis as well as for the design of future trials intended to test new reperfusion therapy efficacy. (Myocardial Salvage Assessed by Cardiovascular Magnetic Resonance—Impact on Outcome; NCT00952224). (J Am Coll Cardiol 2010; 55:2470–9) © 2010 by the American College of Cardiology Foundation

Myocardial salvage is the principal mechanism by which patients with acute myocardial infarction benefit from reperfusion therapies (1). To assess the efficacy of reperfusion therapy, it is necessary to determine how much myocardium is salvaged by measuring the final infarct size in relation to the initial myocardium at risk. The most widely practiced technique for directly measuring myocardial salvage currently is single-photon emission computed tomography (SPECT). This approach has been applied successfully in trials (2–6) and has confirmed that the degree of myocardial salvage is an independent predictor of outcome (7). However, this approach is limited by its low spatial resolution and the need for injection of the isotope in the acute setting of coronary occlusion, which could interfere with patient care (2,8). Moreover, imaging must be completed within 3 h, which is difficult during off-hours and the requirement of 2 separate perfusion studies leads to additional radiation exposure.

Recently, a landmark study showed that the area of high T2 signal in cardiovascular magnetic resonance (CMR) reflects the area at risk in acute reperfused myocardial infarction (9). Clinically, Friedrich et al. (10) confirmed that the proportion
of myocardial salvage can be assessed retrospectively in humans by comparing T2-weighted (edematous myocardium) and late enhancement CMR images. Furthermore, trials demonstrated that myocardial salvage assessment by CMR is a reproducible tool that identifies and quantifies myocardium salvage with excellent agreement with SPECT and angiographic scores of myocardial salvage (11–13). However, in contrast to the extensive clinical SPECT experience, there are no prognostic myocardial salvage data using CMR as an end point in clinical trials.

The aim of the present CMR study was therefore to determine the prognostic significance and determinants of myocardial salvage assessed by CMR in acute reperfused myocardial infarction.

**Methods**

**Study population.** This prospective trial was conducted at a single tertiary care center between November 2006 and May 2008. The study protocol was approved by the local ethics committee, and all patients gave written informed consent. Patients with infarction undergoing primary percutaneous coronary intervention (PCI) were eligible if the onset of symptoms was less than 12 h before PCI and if they had ST-segment elevation of at least 0.1 mV in ≥2 extremity leads or at least 0.2 mV in ≥2 precordial leads.

To ensure that CMR findings reflected acute myocardial injury, patients were not enrolled if they had a previous myocardial infarction. Further exclusion criteria were previous fibrinolysis and patients with contraindications to CMR at study entry such as implanted pacemakers, defibrillators, claustrophobia, or metallic intracranial implants.

**Primary angioplasty and subsequent treatment.** Primary PCI was performed according to standard clinical practice. The decision to use of bare-metal or drug-eluting stents was left to the discretion of the interventional cardiologist. Additional use of intra-aortic balloon counterpulsation or brillators, claustrophobia, or metallic intracranial implants.

**Abbreviations and Acronyms**
- CK = creatine kinase
- CMR = cardiovascular magnetic resonance
- LV = left ventricle/ventricular
- MACE = major adverse cardiovascular events
- MO = microvascular obstruction
- MSI = myocardial salvage index
- PCI = percutaneous coronary intervention
- SPECT = single-photon emission computed tomography
- TIMI = Thrombolysis In Myocardial Infarction

**Electrocardiographic and enzymatic analysis.** For electrocardiographic interpretation, the cumulative ST-segment resolution approximately 90 min after PCI, expressed as the percentage, was calculated by 2 blinded observers as described previously (15). Categorization was performed in complete (≥70%), partial (<70% to 30%), and no (<30%) ST-segment resolution (15). Plasma samples for creatine kinase (CK) and the CK-myocardial band fraction were collected on admission and subsequently during the hospitalization every 6 h for 2 days. CMR. Myocardial salvage was determined by CMR on days 1 to 4 after the index event. The area at risk, infarct size, and microvascular obstruction (MO) were acquired on a 1.5-T scanner (Intera CV, Philips Medical Systems, Best, the Netherlands). Left ventricular (LV) function was assessed by a standard steady-state free precession technique. For area at risk determination, short-axis slices covering the whole ventricle using a T2-weighted triple inversion recovery breath-hold pulse sequence (repetition time 2× R-R interval; echo time 80 ms; flip angle 180°; voxel size 0.71 × 0.71 × 8.0 mm) were obtained using a body coil. Late enhancement images covering the whole ventricle were acquired approximately 15 min after intravenous administration of 0.2 mmol/kg body weight of gadobutrol (Gadovist, Bayer Schering Pharma, Berlin, Germany). A 3-dimensional inversion recovery turbo gradient echo sequence (repetition time 2.8 ms; echo time 1.1 ms; flip angle 15°; typical spatial resolution 1.8 × 1.8 × 5 mm; 2 stacks; pre-pulse delay 180 to 280 ms) was used for image acquisition.

**Image analysis.** Offline image analysis was performed on an independent workstation with dedicated software (ViewForum release 5.2, Philips Medical Systems) by fully blinded observers. Infarct size, area at risk, and MO were expressed as percentages of the LV volume, given by the sum of the volume of edema, late enhancement, and MO regions for all slices divided by the sum of the LV myocardial cross-sectional volumes (%LV).

The area of abnormal signal intensity was measured in the T2-weighted images and in the corresponding late enhancement images by manual delineation in each of the short-axis images. A central core of hypointense signal within the area of increased T2 signal intensity, which is deemed to be hemorrhagic infarction, was included in the area at risk assessment (16). Care was taken to exclude increased signal intensity from the blood pool adjacent to the endocardium due to slow flow. By consensus, a myocardial region was regarded as affected if at least 10 adjacent myocardial pixels...
revealed a signal intensity of >2 SDs of remote myocardium for edema and >5 SDs in late enhancement images (17). In patients with MO, these dark areas were included for infarct size analysis and the area of MO was assessed separately. The following parameters were calculated as described previously (10,18):

a. Area at risk = volume edema/volume LV mass
b. Percentage of infarct size = volume infarct/volume LV mass
c. Percentage of MO = volume MO/volume LV mass
d. Myocardial salvage = area at risk minus infarct size
e. Myocardial salvage index (MSI) = area at risk minus infarct size/area at risk.

The CMR core laboratory has excellent reproducibility and low interobserver and intraobserver variability for infarct size and MSI assessment (13,19). For MSI assessment, the bias and limits of agreement are 0.3 ± 5.0 (13).

**Clinical end points.** The primary end point of this study was the occurrence of major adverse cardiovascular events (MACEs), defined as a composite of death, reinfarction, and new congestive heart failure within 6 months after the index event. In a secondary analysis, the individual components of the primary end point were analyzed. Post-hospital follow-up included 1 outpatient visit at 6 months. The diagnosis of reinfarction was based on clinical symptoms, new ST-segment changes, and increase in the CK-myocardial band level above the reference limit in patients with normalized values after the index event or if there was an increase of at least 50% from the last non-normalized measurement. New congestive heart failure was defined as any congestive heart failure (rales, dyspnea, New York Heart Association functional class III to IV) occurring >24 h after the index event. To avoid double counting of patients with >1 event, each patient contributed only once to the composite MACE end point.

**Statistical analysis.** Patients were grouped by the median MSI into a < median MSI and a ≥ median MSI group. Each categorical variable is expressed as the number and percentage of patients. Most continuous variables had non-normal distribution and are therefore presented as medians together with the interquartile range.

<table>
<thead>
<tr>
<th>Variable</th>
<th>MSI &lt; Median (n = 104)</th>
<th>MSI ≥ Median (n = 104)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edema (%)LV</td>
<td>35.2 (29.5–43.1)</td>
<td>37.3 (29.1–47.3)</td>
<td>0.34</td>
</tr>
<tr>
<td>Infarct size (%)LV</td>
<td>25.7 (19.4–32.6)</td>
<td>10.3 (5.4–14.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Late microvascular obstruction (%LV)</td>
<td>0.9 (0.4–1.9)</td>
<td>0.3 (0.0–0.9)</td>
<td>0.001</td>
</tr>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>46.5 (38.3–53.9)</td>
<td>55.1 (45.8–62.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Left ventricular end-diastolic volume (ml)</td>
<td>148.7 (125.0–167.3)</td>
<td>131.7 (111.2–154.7)</td>
<td>0.005</td>
</tr>
<tr>
<td>Left ventricular end-systolic volume (ml)</td>
<td>77.3 (59.3–96.6)</td>
<td>58.3 (43.8–75.5)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are presented as median and interquartile range.

LV = left ventricle; MSI = myocardial salvage index.

**Figure 1 Study Flow Diagram**

CMR = cardiac magnetic resonance; MSI = myocardial salvage index; STEMI = ST-segment elevation myocardial infarction.
Differences between groups were assessed by the Fisher exact or the chi-square test for categorical variables and by the Student $t$ test for continuous data with normal distribution. Otherwise, the nonparametric Wilcoxon rank-sum test was used. Correlation analyses were done by using Pearson or Spearman tests, as indicated.

Univariable and multivariable linear regression analyses were performed to characterize predictors of the MSI. Categorical variables included sex, smoking, hypertension, hypercholesterolemia, diabetes, infarct location, number of diseased vessels, Killip class on admission, and TIMI flow before and after PCI. Continuous variables included age, symptom onset-to-reperfusion time, door-to-balloon time and ST-segment resolution 90 min after PCI. Multivariable regression was performed using only variables with a probability value $<0.05$ in univariable regression analyses.

For the combined clinical end point, the Kaplan–Meier method was applied, and differences were assessed by the log-rank test. Simple Cox regression analysis using the same variables as defined previously as well as the CMR variables in Table 1 were used to identify predictors of MACEs during 6 months. Hazard ratios with their corresponding 95% confidence intervals are reported. All variables that appeared to be associated with MACEs at the $p < 0.05$...
level in univariable analysis were then tested in a multiple Cox regression analysis, based on a stepwise algorithm with the p value set at 0.05 for entering and 0.1 for exclusion. The extent of late MO was a better predictor of MACEs than the presence of late MO in univariable analysis and was therefore included in the multivariable Cox regression model.

For additional comparison of the prognostic value of the MSI, infarct size and late MO with regard to MACEs as well as mortality, receiver-operator characteristic curves were generated, and the areas under the curves were calculated. All statistical tests were performed with SPSS software, version 15.0 (SPSS, Inc., Chicago, Illinois). A 2-tailed p value <0.05 was considered statistically significant.

Results

Of 267 eligible ST-segment elevation myocardial infarction patients, this prospective CMR study included 208 patients (Fig. 1). The main reasons for exclusion from the study were the lack of a CMR examination (n = 23) and previous myocardial infarction (n = 29). Reasons for not undergoing CMR are listed in Figure 1. In all remaining 208 patients, clinical outcome data were available and CMR image quality was suitable to assess myocardial salvage.

Patient characteristics. Demographic and clinical characteristics are shown in Table 2. The baseline characteristics (age, sex, risk factors) were similar between groups. Patients with an MSI ≥ median had a significantly higher frequency of anterior myocardial infarction and lower Killip class at presentation.

The MSI values according to hours from symptom onset to treatment are shown in Figure 2, where the MSI was highest within the first 2 h but decreased thereafter. The time from symptom onset to reperfusion was significantly shorter in the MSI ≥ median group (p = 0.003). There was a significant inverse correlation of symptom duration and the MSI (r = −0.330, p < 0.001). The door-to-balloon time showed no significant differences between groups.

Patients with anterior myocardial infarction had a significantly higher MSI compared with patients with nonanterior myocardial infarction (58.6 [interquartile range 33.2 to 73.9] vs. 39.9 [interquartile range 25.4 to 70.8]; p = 0.01).

Angiographic analysis, ST-segment resolution, and enzymatic analysis. Patients with an MSI ≥ median had a significantly higher frequency of TIMI flow grade 3 before PCI (p = 0.005). The MSI was significantly higher in patients with residual TIMI flow (TIMI flow grade 2 to 3) before PCI (p < 0.001) (Fig. 3A). After PCI, the majority of patients had TIMI flow grade 3 in both groups (86% vs. 91%, p = 0.36). TIMI flow before and after PCI correlated significantly with the MSI (r = 0.317, p < 0.001 and r = 0.160, p = 0.03, respectively).

Of the 208 patients 104 (50%) had complete, 67 (32%) had partial, and 37 (18%) had no ST-segment resolution 90
min after PCI. ST-segment resolution as a continuous variable was significantly better in the MSI ≥ median group ($p = 0.007$) and patients with a MSI ≥ median also had a significantly lower peak CK and CK-myocardial band level ($p < 0.001$). MSI was significantly higher in patients with complete ST-segment resolution after PCI ($p < 0.001$) (Fig. 3B). The extent of ST-segment resolution 90 min after the primary PCI correlated with the MSI ($r = 0.287$, $p < 0.001$).

**CMR.** The median time between the index event and CMR was 3 days (interquartile range 2 to 4) for both groups. Results of CMR were available in all 208 patients. In all patients, a high transmural T2 signal abnormality was observed in the infarct region. In 9 (4%) patients, CMR detected no late enhancement (aborted infarction) (20). Figure 4A is an example of an acutely occluded right coronary artery with corresponding high signal in the inferior left and right ventricular walls. The evolution of myocardial edema in a patient with left anterior descending artery occlusion is shown in Figure 4B. The localization of late enhancement was in the same region of the edema in all cases. The median amount of edema was 35.5%LV (interquartile range 29.2 to 44.9) with no significant differences between groups. The
The median infarct size was 16.3% LV (interquartile range 10.0 to 26.4), significantly smaller than myocardium at risk ($p < 0.001$) (Table 1). The median calculated MSI was 48.3 (interquartile range 27.7 to 73.2).

Late MO was identified in 134 (64%) patients with a significantly higher occurrence in the MSI < median group (85 vs. 49 patients, $p < 0.001$). Infarct size and the extent of late MO were also significantly larger in the MSI < median group. LV ejection fraction was significantly higher in the MSI < median group, whereas end-diastolic and end-systolic volume indexes were significantly lower in this group at this early stage after the index event (Table 1).

### Predictors of myocardial salvage

In a multivariable regression model adjusted for significant variables in univariable regression analysis using the MSI as the dependent variable, complete ST-segment resolution ($p < 0.001$), time from symptom onset to reperfusion ($p = 0.002$), anterior myocardial infarction ($p = 0.004$), and TIMI flow grade ≥ 2 before PCI ($p = 0.03$) were the strongest predictors of MSI (Table 3).

### Clinical outcome

At 6-month follow-up, there were 10 cardiac deaths (9.6%) in the < median MSI group and 1 (1%) in the MSI ≥ median group ($p = 0.003$) (Fig. 5A). In the MSI ≥ median group, the patient died of a right ventricular infarction. Nonfatal reinfarctions and congestive heart failure occurred significantly more often in the < median MSI group (13 vs. 2 events). Consequently, at 6-month follow-up, MACEs were significantly lower in the MSI ≥ median group (2.9% vs. 22.1%, $p < 0.001$) (Fig. 5B).

In addition to the MSI, several established markers of increased patient risk were associated with increased MACEs at 6-month follow-up by simple Cox regression analysis (Table 4). Using stepwise multiple Cox regression analysis, only MSI remained an independent predictor of the combined clinical end point.

Despite significant predictors in univariable analyses, late MO and LV ejection fraction were unable to predict 6-month MACEs in stepwise multivariable Cox regression analysis.

Receiver-operator characteristic curve analyses further illustrated that the MSI is the strongest indicator of MACEs and especially of mortality compared with infarct size and late MO (Fig. 6).

### Discussion

To our best knowledge this the largest prospective CMR outcome study to date and the first to report introducing myocardial salvage as a novel strong CMR outcome parameter in acute reperfused myocardial infarction. The major

![Figure 5](https://example.com/figure5.png)

**Figure 5** Mortality and Major Adverse Cardiovascular Event Rate

Kaplan-Meier curve of the incidence of all-cause mortality (A) and the cumulative incidence of death, reinfarction, and new congestive heart failure (B) during the first 6 months after randomization. MSI = myocardial salvage index.
findings are as follows: 1) patients with a MSI ≥ median have a significantly lower mortality and MACE rate at 6-month follow-up; 2) the strongest predictors of the MSI are complete ST-segment resolution, time from symptom onset to reperfusion, anterior myocardial infarction, and TIMI flow grade ≥ 2 before PCI; and 3) the amount of myocardial salvage is the strongest inverse correlate of MACEs and mortality at 6-month follow-up. Thus, quantifying the extent of the salvaged area after revascularization using CMR might serve as a novel, strong end point for clinical trials investigating the success of reperfusion strategies.

**Myocardial salvage assessment by CMR.** The myocardial area at risk is defined as the myocardial tissue within the perfusion bed distal to the culprit lesion of the infarct-related artery (21). Recently, it has been shown, that in the setting of reperfused (9) and nonreperfused (22) infarction, high signal intensity areas in T2-weighted images accurately visualize the area at risk. The result of reperfusion therapy can therefore be assessed clinically by calculating myocardial salvage as the difference between myocardium at risk and final infarct size. Friedrich et al. (10) applied this technique to patients and systematically assessed myocardial salvage by CMR in 92 reperfused infarction patients. This group demonstrated that the area at risk identified with T2 imaging was consistently transmural and exceeded areas of irreversible injury defined by late enhancement, resulting in a mean myocardial salvage of 16 ± 11%. Despite acquiring only 3 short-axis slices, these data are in line with the current study, which calculated a mean myocardial salvage of 18 ± 12% from a 3-dimensional volume set, and a recent published study with a mean salvage of 14 ± 10% (11). Therefore, myocardial salvage assessment using CMR is a robust method for quantifying the extent of the salvaged area after reperfusion. However, further improvements in signal intensity and reduction in artifacts of T2 imaging is necessary.

**Myocardial salvage as clinical outcome parameter.** The accurate identification of the area at risk in acute myocardial infarction is crucial to the evaluation of future reperfusion therapies and their impact on myocardial salvage. The quantification of myocardial salvage might be a better indicator of therapeutic efficacy in myocardial infarction trials than infarct size. SPECT with a technetium tracer injection before reperfusion has been the most widely used method for quantifying myocardial salvage. However, the accuracy of SPECT is limited by the attenuation of the radiation and the need for patient positioning. CMR, on the other hand, provides images with excellent spatial and temporal resolution and can accurately quantify myocardial salvage. The use of CMR in this context is particularly appealing because it can provide detailed spatial information about the extent of myocardial salvage and can be used to monitor changes in myocardial salvage over time.

**Table 4: Predictors of Major Adverse Cardiac Events in Univariable and Stepwise Multivariable Cox Regression Analysis**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariable Hazard Ratio (CI) p Value</th>
<th>Stepwise Multivariable Hazard Ratio (CI) p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Killip class on admission</td>
<td>2.01 (1.30–3.09) 0.002</td>
<td>—</td>
</tr>
<tr>
<td>TIMI flow grade after PCI</td>
<td>0.57 (0.36–0.88) 0.01</td>
<td>—</td>
</tr>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>0.95 (0.92–0.98) &lt;0.001</td>
<td>—</td>
</tr>
<tr>
<td>ST-segment resolution (%)</td>
<td>0.99 (0.99–1.00) 0.04</td>
<td>—</td>
</tr>
<tr>
<td>Late microvascular obstruction (%LV)</td>
<td>1.10 (1.03–1.17) 0.004</td>
<td>—</td>
</tr>
<tr>
<td>Infarct size (%LV)</td>
<td>1.08 (1.05–1.12) &lt;0.001</td>
<td>—</td>
</tr>
<tr>
<td>MSI</td>
<td>0.95 (0.93–0.97) &lt;0.001</td>
<td>0.93 (0.91–0.96) &lt;0.001</td>
</tr>
</tbody>
</table>

CI = confidence interval; other abbreviations as in Tables 1 and 2.

**Figure 6: Receiver-Operator Characteristic Curves of the Prognostic Value of the MSI, Infarct Size, and Late MO**

Receiver-operator characteristic curves showing the prognostic sensitivity and specificity of the myocardial salvage index (MSI), infarct size (IS), and late microvascular obstruction (MO) with regard to major adverse cardiovascular events (A) and mortality (B) at 6-month follow-up. AUC = area under the curve.
practiced technique for assessing the area at risk and has been successfully used to compare the efficacy of various reperfusion strategies. (2–6) Furthermore, this approach has confirmed that the degree of myocardial salvage is an independent predictor of outcome (7). However, there are many drawbacks to its widespread use including: 1) need for 2 scans; 2) need for tracer administration; 3) access to a gamma camera <3 h of the tracer administration; 4) interfering with patient management in the acute setting; 5) application of radiation dose of both scans; and 6) lower spatial resolution, particularly of subendocardial infarcts (8).

Thus, it is desirable to find methods for assessing area at risk and subsequent myocardial salvage that are more clinically feasible to evaluate outcomes.

Our study for the first time clearly confirms that CMR is a promising tool that can quantify the success of revascularization reflected by the amount of salvaged myocardium with a strong predictive value of subsequent clinical outcome. So far, infarct size and MO were the most used CMR end points in clinical trials because previous studies have shown that these parameters are associated with global and regional functional recovery as well as MACEs (23–25). However, these trials had small sample sizes and traditional prognostic markers and scores have not been investigated. In addition, “soft” clinical end points were included in MACEs such as revascularization or hospital admission for unstable angina (24). In the present study, only harder end points were included and clinical, angiographic, and electrocardiographic parameters, which are known from earlier research to affect prognosis in ST-segment elevation myocardial infarction, showed significant correlations with the MSI (15,26,27). This may explain why these parameters are associated with survival. In particular, the strong correlation of ischemic time with the MSI is remarkable. It emphasizes that the extent of the salvaged myocardium assessed by CMR is in accordance with the wavefront phenomenon, initially postulated by Reimer et al. (28). Median myocardial salvage was extremely large when PCI was performed within the first 2 h of symptom onset, whereas it was twice smaller with an additional 2 to 4 h of reperfusion delay, and our data unequivocally show the “flat” part of the salvage curve thereafter (29). These data thus provide additional support for the relationship between myocardial salvage to ischemic time and confirm current recommendations for patient transport to tertiary centers for the primary PCI as soon after symptom onset as possible (14,30). However, some patients showed significant myocardial salvage after a prolonged ischemic time. This may depend on several clinical factors, including the extent of collateral circulation and the presence or absence of a history of ischemic preconditioning (27).

**Clinical implications.** Prognosis in patients with acute reperfused myocardial infarction is directly related to the amount of myocardial salvage, which strengthens the use of myocardial salvage as a strong surrogate end point for clinical trials investigating the success of reperfusion strategies. Particularly in myocardial infarction trials, there is increasing interest in measures that can be used as a surrogate end point, allowing a much smaller sample size, faster trials, and a reduction of costs. CMR might be the optimal method to assess myocardial salvage because it can be done retrospectively with 1 scan, without interfering with patient care in the acute setting, at high spatial resolution and without ionizing radiation.

**Study limitations.** Some limitations need attention. First, some patients had to be excluded from CMR myocardial salvage assessment. Because the baseline characteristics of patients undergoing and those not undergoing CMR were similar, a potential selection bias is unlikely. Second, the influence of several clinical variables that were not available and might affect myocardial salvage were not examined, such as collateral blood flow and preconditioning. However, angina onset and recurrence are generally subjective and difficult to assess.

**Conclusions**

This study clearly demonstrated that myocardial salvage assessed with CMR predicts MACEs and mortality in patients with acute reperfused myocardial infarction. Therefore, the retrospective assessment of myocardial salvage after PCI has important implications for patient prognosis and improving clinical outcomes, as well as for the design of future trials intended to test the efficacy of reperfusion modalities.

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Key Words: cardiovascular magnetic resonance • magnetic resonance imaging • myocardial infarction • myocardial salvage • prognosis.