Quantitative Magnetic Resonance Perfusion Imaging Detects Anatomic and Physiologic Coronary Artery Disease as Measured by Coronary Angiography and Fractional Flow Reserve

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Objectives
To evaluate the ability of quantitative perfusion cardiac magnetic resonance (CMR) to assess the hemodynamic significance of coronary artery disease (CAD) compared with well-established anatomic and physiologic techniques.

Background
Fractional flow reserve (FFR) is considered by many investigators to be a reliable stenosis-specific method to determine hemodynamically significant CAD. Quantitative perfusion CMR is a promising noninvasive approach to detect CAD but has yet to be validated against FFR.

Methods
This is a prospective study in patients with suspected CAD who underwent coronary angiography, FFR, and CMR assessments. The quantitative myocardial perfusion reserve (MPR) was calculated in 720 myocardial sectors (8 sectors/slice). The MPR was calculated from the ratio between stress and rest myocardial flow based on signal intensity time curves using deconvolution analysis. Stress was simulated with adenosine for both FFR and MPR. The MPR assessments were compared to FFR (n = 44 coronary segments) and quantitative coronary angiography (n = 108 segments) in the corresponding coronary territories.

Results
The MPR was 1.54 ± 0.36 in segments with FFR <0.75 (n = 14) and 2.11 ± 0.68 in those with FFR >0.75 (n = 30; p = 0.0054). An MPR cutoff of 2.04 was 92.9% (95% CI 77.9 to 100.0) sensitive and 56.7% (95% CI 32.8 to 80.6) specific in predicting a coronary segment with FFR <0.75. The MPR was 1.54 ± 0.49 in coronary segments with ≥50% diameter stenosis (DS) (n = 47) and 2.13 ± 0.80 in segments with <50% DS (n = 61; p < 0.001). An MPR cutoff of 2.04 was 85.1% (95% CI 71.1 to 99.2) sensitive and 49.2% (95% CI 33.6 to 64.8) specific in predicting CAD with ≥50% DS.

Conclusions
Quantitative perfusion CMR is a safe noninvasive test that represents a stenosis-specific alternative to determine the hemodynamic significance of CAD. (J Am Coll Cardiol 2007;50:514–22) © 2007 by the American College of Cardiology Foundation

In patients with coronary artery disease (CAD), revascularization is deemed appropriate based on combined physiologic and anatomic data. Quantitative coronary angiography (QCA) remains a well-established technique in the anatomic assessment of CAD (1,2), whereas fractional flow reserve (FFR) is an accurate stenosis-specific method to evaluate the hemodynamic significance of CAD (3,4). Both methods are well validated and routinely applied to determine severity of CAD (1,5,6). However, the invasive nature of these diagnostic modalities limits their broad application for screening purposes.

Cardiac magnetic resonance (CMR) is a promising noninvasive and ionizing-free radiation imaging modality to evaluate CAD. The first pass of gadolinium through the myocardium can be plotted as signal intensity (SI) versus time. Both relative and absolute blood flows can be quantified with reproducibility by first-pass magnetic resonance perfusion (7,8). The high spatial resolution of CMR allows regional quantification of transmural flow gradients (9).
Myocardial perfusion reserve (MPR) can be calculated from the ratio of simulated stress and rest flows derived from the myocardial SI time curves.

Perfusion CMR has compared favorably with other noninvasive modalities to detect CAD (10–13). Pilot studies have shown the correlation between CMR and invasive assessment of coronary flow reserve (CFR) using intracoronary Doppler flow wire or positron emission tomography (8,10,14). However, CFR is highly influenced by the microcirculation status and is not considered a stenosis-specific method to define the hemodynamic significance of CAD (15,16). As a result, CFR has been replaced by FFR as the central invasive physiologic technique to define CAD severity. Whether perfusion CMR correlates with FFR and represents a noninvasive stenosis-specific assessment of ischemia and hemodynamically significant stenoses remains to be evaluated.

The present study investigated the ability of CMR to assess the physiologic significance of CAD. We compared quantitative perfusion CMR using MPR with stenosis-specific physiologic assessments determined by FFR and anatomic assessments by QCA.

Methods

We prospectively enrolled 37 consecutive patients, between 18 and 80 years of age, with suspected CAD who underwent coronary angiography, FFR, and CMR assessments. Patients were excluded if they had a myocardial infarction within 14 days of either procedure, high-degree atrioventricular block, hypotension (systolic blood pressure <90 mm Hg), severe chronic obstructive pulmonary disease, decompensated congestive heart failure (New York Heart Association functional class III or IV), a ferromagnetic metallic implant, or claustrophobia or were pregnant or lactating. All CMR assessments were done within 2 months of the angiography and FFR evaluations. Coronary revascularization was not performed between CMR and angiography assessments. The protocol was approved by the institutional review board. Informed consent was obtained before study procedures.

Angiography and fractional flow reserve assessments. Guiding catheters (6-F) without side holes were used. Cine angiographies were performed in at least 2 orthogonal projections after 100 to 200 μg intracoronary nitroglycerin infusion. All cineangiographies were recorded on digital compact discs. The QCA was performed off line by an independent core laboratory (University of Florida Cardiovascular Imaging Core Laboratories, Jacksonville, Florida). Interpolated reference vessel diameter, minimal lumen diameter, and percentage diameter stenosis (DS) were calculated using an automated contour detection algorithm (CAAS II Analysis System, Pie Medical, Maastricht, the Netherlands) with at least 2 orthogonal angiographic views. A straight (nontapered) portion of the catheter was used for image calibration. Measurements were performed by an independent analyst who was blinded to the clinical, FFR, and MPR data. Side branches distal and proximal to the stenotic segment were used to define the target segment for quantitative analysis. This methodology has been found to be highly accurate (−0.01 ± 0.18 mm) and reproducible (correlation coefficient = 0.94) (17). The QCA was performed in all 3 major epicardial coronary arteries in each patient, regardless of the visual estimation of stenosis.

Intracoronary pressure was measured using a 0.014-inch pressure guide wire (WaveMap Pressure System; Volcano Therapeutics, Rancho Cordova, California) across the target stenosis. The ratio of the distal and the aortic pressure on maximal hyperemia, or pressure-derived fractional flow reserve (FFR = Pd/Pa), has been extensively validated (3,4). Following 100 to 200 μg of intracoronary nitroglycerin injection, FFR was calculated after intravenous adenosine at 140 μg/kg/min for at least 2 min. All measurements, as well as calibration, pressure equalization, baseline and hyperemic trans-stenotic gradients, and FFR were recorded. The stenosis was considered physiologically significant if the FFR was ≤0.75 (3).

CMR quantification of myocardial perfusion reserve. Contrast-enhanced magnetic resonance imaging (MRI) using gadolinium-based contrast agents has been used to evaluate myocardial perfusion patterns (18,19). Imaging postprocessing was performed using commercial workstations (Leonardo, Siemens, Munich, Germany; and Mass Flow MR 6.1, Medis, Leiden, the Netherlands). In the present study, SI time curve editing was performed using a dedicated workstation (Mass Flow, version 6.1, Medis), which allowed objective and automated assessment of myocardial SI time curves to derive myocardial blood flow data with minimal interference by the analyst. Measurements were performed by 2 independent MRI experts blinded to the angiographic and FFR results. Adequate CMR image quality for quantification was prospectively determined by evaluating the contrast injection, image resolution, and SI curve. Quantification requires a very rapid bolus by a power injector that ideally forms a single sharp spike occurring at time zero. Spatial resolution should be <2.5 mm to resolve transmural variations in blood flow. Temporal resolution should be 1 to 2 image frames per heartbeat. The SI curve for each sector should be compared with the wash-in curve of the left ventricular (LV) blood pool, looking for the characteristic upslope and delayed and reduced peak signal amplitude.

After manual correction of images for gross cardiac motion, endocardial and epicardial contours were drawn to define the myocardium for the base, middle, and apical slices. The myocardium was then segmented into 8 equi-
distant radial sectors per slice (Fig. 1). The sectors were ordered in a clockwise orientation, with sector number 1 beginning at the insertion of the right ventricle to the anterior septal wall of the LV. The distribution of the arterial territories within the 24 segments was based on the 17-segment model for tomographic imaging of the heart (20). The left anterior descending artery perfusion territory was assigned to sectors 8, 1, and 2 for the base, middle, and apical slices. The diagonal branches perfusion territory was assigned to sectors 1 and 2 for the middle and apical slices. The circumflex and obtuse marginal branches perfusion territories were assigned to sectors 3 and 4. The circumflex territory included the base, middle, and apical slices, and the obtuse marginal branches territory involved the middle and apical slices. The right coronary and posterior descending artery perfusion territories were assigned to sectors 5, 6, and 7. The right coronary artery territory included the base, middle, and apical slices, and the posterior descending artery territory was assigned to the apical slice. The posterior lateral artery perfusion territory was assigned to sectors 4 and 5 of the apical slice (Fig. 1).

Constrained deconvolution analysis using a Fermi function was applied to the first-pass SI curves and provided an adjusted or absolute myocardial blood flow measurement using a custom C++ software program previously developed by our group (7–9). The Fermi function models the probability that a contrast molecule has left the myocardium as a function of time. Deconvolution of the LV blood pool SI curve with the Fermi function yields a theoretic myocardial SI curve. The theoretic curve is compared against the actual measured myocardial SI data points using least squares and a repetitive Marquardt-Levenberg algorithm. The initial amplitude of the Fermi function corresponds to absolute myocardial blood flow (21–24). This technique has been validated using radioactive microspheres (25). The MPR was then calculated as the ratio of myocardial blood flow at maximal hyperemia divided by the myocardial blood flow at rest. The MPR was then normalized by rate pressure product, which was calculated for rest and hyperemia at the maximum rate of adenosine infusion, as previously described and validated (7,21,24,25). The reproducibility of CMR first-pass imaging has also been reported and showed...
good intraobserver (correlation coefficient = 0.8 to 0.85) and interobserver (correlation coefficient = 0.81 to 0.97) agreements (26).

**CMR imaging protocol.** All patients underwent first-pass contrast-enhanced CMR perfusion analysis imaging obtained at rest and stress on a Sonata 1.5 Tesla (Siemens-Sonata) magnet (maximum slew rate 150 T/m/s; gradient strength 40 mT/m) using a 6-channel body coil. Scout images were obtained initially to determine the cardiac geometry and to plan subsequent scans. Imaging was performed at rest and simulated stress during intravenous injection of 0.1-mmol/kg gadolinium-DPTA (Magnevist; Berlex Laboratories Inc., St. Louis, Missouri) contrast by power injector (MedRad, Indianola, Pennsylvania) at a rate of 10 ml/s. The pulse sequence used was a single-shot gradient echo sequence with saturation-recovery magnetization preparation for T1 weighting and linear k-spacing. The parameters were set to repetition time/echo time flip angle of 2.4 ms/1.2 ms/18° and a slice thickness of 10 mm. Sixty images per slice location were acquired with a spatial resolution of 2 to 3 mm. Simulated stress imaging was performed using an infusion of adenosine at a concentration of 140 μg/kg/min for 4 min. First-pass perfusion imaging was obtained at the end of injection, and after 3 min 45 s of adenosine infusion. Subsequently, the scan was repeated during resting conditions, at least 5 min after the adenosine infusion had been stopped. Perfusion was then determined in 3 LV short-axis slices. The first slice was located closer to the base of the heart, the second in the middle of the LV, and the third slice closer to the apex just distal to the base of the papillary muscles. Patients were asked to perform shallow breathing for the duration of the scan to minimize respiratory motion. Blood pressure, heart rate, and any serious adverse reactions caused by the simulated stress throughout the CMR imaging examination were monitored by a physician and nursing personnel.

For cine magnetic resonance imaging, an electrocardiogram-gated breath hold, segmented truefisp sequence was used with RT/TE/flip angle 33 ms/6 ms/25°. In-plane spatial resolution of the cine sequence was 2 mm × 1.4 mm with a slice thickness and increment of 10 mm. The temporal resolution was 30 to 50 ms with 14 to 16 cardiac phases per plane.

**Comparisons between MPR, FFR, and QCA.** First, MPR values for all coronary segments, as described in the preceding, were plotted and compared with QCA and FFR data without any guidance (blind analysis) from the angiographic data regarding specific target segments. The minimum MPR values for the corresponding coronary segment in each slice (base, middle, and apex) were averaged and used for comparison.

In a second analysis, the location of the stenosis defined by QCA was used to guide the selection of MPR values from specific myocardial slices corresponding to the stenosis location (proximal, middle, or distal). This allowed a more stenosis-specific comparison among MPR, QCA, and FFR assessments. Thus, in an ostial stenosis we considered MPR values from all 3 slices, whereas for proximal and middle located stenosis, only MPR values from the middle and apical slices were averaged for comparison. As in the blind analysis, the lowest MPR from an artery’s territory was used from each pertinent slice. At least 2 MPR values from different slices were averaged for comparison. The exception occurred with apical stenosis, of which the 2 lowest MPR values from the artery’s territory were averaged. It is important to note that, although the selection of MPR values was not blinded to QCA measurements, the calculation of MPR for each sector (8 per slice) was automatically calculated using a computerized workstation by analysts unaware of the angiographic data.

**Statistical analysis.** Data are presented as mean ± standard deviation. Frequencies are presented as percentage. Receiver-operating curve (ROC) analysis was performed using MedCalc (MedCalc Software, Mariakerke, Belgium) to define sensitivity, specificity, and optimal cutoff values of MPR to determine anatomic (≥50% DS) and hemodynamically (FFR ≤0.75) significant CAD. Positive predictive value (PPV) and negative predictive value (NPV) were also derived using the MPR cutoff from the ROC analysis. The 95% confidence intervals (CIs) for sensitivity and specificity were analyzed using SAS 9.1 (SAS Institute Inc., Cary, North Carolina) with PROC SURVEYFREQ and PROC SURVEYMEANS. Analysis of variance single factor analysis was also performed on the MPR data using an FFR of ≤0.75 and DS of ≥50% as cutoff for those patients with CAD.

**Results**

Demographics of the study population are reported in Table 1. There were no significant demographic differences between patients with single-vessel CAD who had an FFR of >0.75 or ≤0.75. One patient was excluded because of wire-induced coronary spasm during FFR measurements. Six patients were excluded because of inadequate MRI image quality for quantitative assessment. There were no complications related to CMR procedures, and all patients tolerated the procedure well. The MPR and FFR values were compared in 44 coronary segments, and comparison between QCA and MPR involved 108 segments from 30 patients. Figure 2 illustrates a typical correlation between MPR, angiography, and FFR. A typical appearance of a perfusion defect during stress MRI is illustrated in Figure 3.

**Blind MPR comparisons with FFR and QCA.** The MPR and FFR values were compared in 44 corresponding coronary segments (Table 2). The average MPR in segments with FFR ≤0.75 (n = 14) was 1.50 ± 0.45 and with FFR >0.75 (n = 30) was 2.07 ± 0.66 (p = 0.0059). The ROC curve demonstrated an MPR cutoff of 1.97 that was 85.7% sensitive (95% CI 57.2 to 97.8) and 60.0% specific (95% CI 40.6 to 77.3) with a PPV of 50% and an NPV of 90% in predicting an FFR of ≤0.75.

The MPR and QCA data were compared in 108 corresponding coronary segments (Table 2). The average MPR in segments with ≥50% DS (n = 47) was 1.54 ± 0.51 and with <50% DS (n = 61) was 2.02 ± 0.77 (p <
The myocardial perfusion reserve (MPR) data are reported in graphics for individual slices of cardiac magnetic resonance (CMR) (S1 to S8; first graphic corresponding to left ventricle blood pool). Purple and green lines represent the fitting curves for rest and stress, respectively. The MPR values (Pr) are depicted for each slice at the bottom of each graphic. Rest and stress flow are also reported in the bar graph, with each bar corresponding to 1 CMR slice. Note that slice S4 shows an average MPR of 1.79, which corresponds to the left circumflex artery territory. Coronary angiography (middle bottom panel) revealed a 65% stenosis in the first obtuse marginal branch of the left circumflex artery, which had a fractional flow reserve of 0.65. Middle upper panel shows the middle CMR slides with the quantitative contours. Perfusion magnetic resonance imaging images at both stress and rest are shown in the right panels. It is important to note that the perfusion defect was not visualized in the stress image, and was only detected by the quantitative method. DS = diameter stenosis; FFR = fractional flow reserve.
ROC analysis demonstrated an MPR cutoff of 2.04 that was 85.1% sensitive (95% CI 71.1 to 99.2) and 49.2% specific (95% CI 33.6 to 64.8) with a PPV of 59% and an NPV of 83% in predicting significant (50% DS) CAD (Fig. 5). The average MPR in segments with ≥70% DS (n = 14) was 1.40 ± 0.38 and with <70% DS (n = 94) was 1.94 ± 0.76 (p = 0.01). The ROC analysis demonstrated an MPR cutoff of 1.85 that was 92.9% sensitive (95% CI 66.1 to 98.8) and 50% specific (95% CI 39.5 to 60.5) with a PPV of 22% and an NPV of 98% in predicting significant (≥70% DS) CAD.

In segments that were most likely to be hemodynamically significant, according to the invasively derived data, i.e., FFR ≤0.75 and ≥50% DS (n = 12), the average MPR was 1.60 ± 0.34. In segments that were least likely to have significant CAD, i.e., FFR >0.75 and <50% DS (n = 20), the average MPR was 2.25 ± 0.73.

### Discussion

The present results suggest that quantitative CMR can be safely used to determine the hemodynamic significance of coronary stenosis and to exclude the presence of significant CAD with a high degree of accuracy. Beyond the ability of CMR to detect the presence of ischemia (10,11), these findings support the hypothesis that MPR represents a stenosis-specific measure of the functional significance of CAD. Cardiac magnetic resonance could therefore be used as a highly sensitive screening tool to exclude hemodynamically significant CAD. Likewise, CMR, with its higher spatial resolution compared with single-photon emission computerized tomography (SPECT) and its ability to simultaneously evaluate myocardial viability, mechanics using tagging or phase-based techniques, and hemodynamics, could be used synergistically with multislice computerized tomographic angiography to further stratify patients who have been identified with ambiguous coronary stenosis (27).

Myocardial perfusion reserve, similar to other approaches that use adenosine as the pharmacologic stimuli, may be affected by endothelial dysfunction and the microcirculation status (28–32). Thus, the relatively lower specificity of MPR compared with both FFR and QCA is likely associated with the high prevalence of hypertension, diabetes, and dyslipidemia, which are all known risk factors for impaired endothelium and myocardial flow-mediated dependent vasodilation (33). In addition, there were ≥2 vessels with significant CAD in 73% of the patients. In the present study, side branches were also included and coronary segments supplying small myocardium territories compared. Yet, MPR showed high degrees of agreement with both FFR and QCA. Although the FFR has been shown to be stenosis specific and an accurate determinant of the need for

### Table 2

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<tr>
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<th>Blinded MPR</th>
<th>Stenosis-Specific MPR</th>
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<tbody>
<tr>
<td>FFR ≤0.75</td>
<td>1.50 ± 0.45*</td>
<td>1.54 ± 0.36*</td>
</tr>
<tr>
<td>FFR &gt;0.75</td>
<td>2.07 ± 0.66</td>
<td>2.11 ± 0.68</td>
</tr>
<tr>
<td>≥50% DS</td>
<td>1.54 ± 0.51*</td>
<td>1.54 ± 0.49*</td>
</tr>
<tr>
<td>&lt;50% DS</td>
<td>2.02 ± 0.77</td>
<td>2.13 ± 0.80</td>
</tr>
<tr>
<td>≥70% DS</td>
<td>1.46 ± 0.53*</td>
<td>1.40 ± 0.38*</td>
</tr>
<tr>
<td>&lt;70% DS</td>
<td>1.86 ± 0.72</td>
<td>1.94 ± 0.76</td>
</tr>
<tr>
<td>FFR ≤0.75 + ≥50% DS</td>
<td>1.56 ± 0.46*</td>
<td>1.60 ± 0.34*</td>
</tr>
<tr>
<td>FFR &gt;0.75 + &lt;50% DS</td>
<td>2.2 ± 0.72</td>
<td>2.25 ± 0.73</td>
</tr>
<tr>
<td>FFR ≤0.75 + ≥70% DS</td>
<td>1.49 ± 0.46*</td>
<td>1.56 ± 0.29*</td>
</tr>
<tr>
<td>FFR &gt;0.75 + &lt;70% DS</td>
<td>2.09 ± 0.66</td>
<td>2.14 ± 0.68</td>
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*p < 0.05 indicates comparison between each category.

DS = diameter stenosis; FFR = fractional flow reserve; MPR = myocardial perfusion reserve.
coronary revascularization (3–6,34), it might still be influenced by microvascular disease resulting in elevated values despite significant epicardial CAD (34,35). Discrepancy between QCA data and physiologic measurements has been known since the early studies comparing CFR and QCA (36,37). The possibility of using other pharmacologic agents, such as dobutamine, makes CMR an attractive noninvasive test for patients who are not candidates for CFR, FFR, or other adenosine-based ischemia tests (38).

According to the present study, which includes a stable population, a patient with a normal MPR (≥2.04) in all coronary segments has a very low probability of having hemodynamically significant CAD. Whether CMR can be used as a guide for therapeutic decision-making in patients with known CAD remains to be evaluated in future studies with a larger patient population and long-term follow-up (39).

The MPR cutoff values observed in the present study correlated well with previously reported significant CFR values observed in patients with CAD (1,40). Serruys et al. (1) reported that a CFR >2.5 with a <35% DS had excellent short- and long-term outcomes after percutaneous transluminal coronary angioplasty. Di Mario et al. (40) demonstrated that a CFR >2.0 with <35% DS was associated with similar clinical outcomes. We and others (8,10,41,42) have shown the equivalence and direct correlation between CFR and MPR values. Bedaux et al. (41) reported an average MPR of 2.7 ± 1 and CFR of 3.1 ± 0.6 (p < 0.01) for nonstenosed vessels. Similar to the present study findings, CFR has also been shown to strongly correlate with SPECT imaging and QCA (1,40,43,44).

The sensitivity and specificity of MPR in detecting CAD when FFR was used as the gold standard in the present study compared favorably with earlier reports comparing SPECT with FFR (45–47). Hacker et al. (46) reported a sensitivity of 80% and a specificity of 76% for SPECT in detecting a target vessel with an FFR <0.75.

The MPR and QCA data, although correlated, were not as strong as the correlation observed between MPR and FFR in the present study. Similar correlation coefficients...
between SPECT and QCA data have been reported (47,48). The present data correlate favorably with a recent meta-analysis demonstrating a sensitivity of 89% and specificity of 65% when SPECT was compared with coronary angiography in detecting CAD (48). The limitation of angiography to determine the hemodynamic significance of CAD and its inability to define microvascular disease has been previously reported (49) and likely explains some of the discordance between the noninvasive physiologic data and QCA.

The MPR cutoff values to exclude significant CAD in the present study are relatively higher than those previously reported (11,50). The differences in patient demographics and degrees of epicardial and microvascular CAD between studies may explain these variations in MPR cutoff values. The MPR values have recently been shown to be affected by gender, age, and CAD risk factors (32).

The benefit of a noninvasive highly sensitive diagnostic test to evaluate for and exclude physiologically significant CAD is unquestionable. Although further studies are needed to further establish the value of this imaging approach, the present data suggest that CMR through the use of quantitative MPR can be used as an alternative screening tool to both exclude and localize hemodynamically significant CAD.

Study limitations. The relatively small study population represents a potential limitation of this study. Our analysis, however, was based on coronary segments that included 44 and 108 comparisons between MPR, FFR, and QCA, respectively. Indeed, a larger study population would have increased the statistical power and potentially improved the specificity of MPR compared with FFR and QCA. Although not pre-specified in the present study, a larger sample size would allow subgroup analyses such as specific evaluation of patients with FFR between 0.75 and 0.8 and those with poor LV function.

The CMR images in 6 patients did not meet the minimal requirements for quantitative analysis, which required adequate images at rest and stress, as described in the Methods section. Suboptimal images in the present study were mainly associated with motion artifact or inadequate timing of the contrast bolus. Therefore, the present study findings apply only to patients with optimal CMR image acquisition. Hopefully, recent CMR technologic advancements such as 3-T scanning, 32-channel scanners, and 32-element cardiac array coils will improve the CMR image quality and lead to fewer patients with suboptimal image quality. Nevertheless, the seminal data reported in this study comparing MPR and FFR for the first time suffice to demonstrate the potential value of MPR as an alternative noninvasive test to demonstrate the physiologic significance of CAD.

Coronary flow reserve data would have been beneficial to determine the relative contribution of microvascular disease to MPR values, and such data have been previously reported (8,10). However, CFR data alone do not provide an objective evaluation of epicardial CAD. The present study was aimed at investigating the ability of MPR to reproduce FFR data, the current gold standard method to assess the hemodynamic status of epicardial CAD with minimal influence of microcirculation. The FFR is not absolutely independent of the microcirculatory status, but, unlike CFR, microvascular dysfunction has an opposite effect on FFR measurements, i.e., it may increase FFR values. The MPR and FFR data correlated well in the present study despite this peculiar aspect of FFR assessment of coronary physiology, which may further strengthen our conclusions regarding the ability of perfusion MRI to evaluate CAD. To our knowledge, such a comparative study between MPR and FFR had not been done previously.

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