The clinical role of cardiovascular magnetic resonance (CMR) continues to expand, supported by ongoing technological advances that have shortened acquisition times while maintaining and often improving image quality. New applications of CMR in cardiovascular imaging continue to emerge, and results from larger clinical trials are beginning to define the role of CMR in a range of clinical scenarios. Currently accepted indications for CMR include the assessment of congenital heart disease, the great vessels, acquired myocardial and pericardial disease, and chronic coronary artery disease (CAD). The role of CMR in the assessment of acute coronary syndromes (ACS) is less well established. However, evidence is accumulating that CMR provides often unique information in chest pain syndromes that can aid in the detection and differential diagnosis of ACS, guide clinical decision making, and improve risk stratification after an event.

After reviewing the relevant CMR methodology, the present article puts forward the current evidence for CMR in ACS and gives an outlook of future developments.

CMR Methods
The following CMR methods are most commonly used to assess ACS (Figure 1) and can be incorporated into a clinical protocol that can be performed within an hour.

Cine Imaging
The assessment of global and regional left ventricular (LV) and right ventricular (RV) function by CMR is typically based on a cine data set aligned in the true LV short axis that covers the heart in 10 to 12 consecutive 2-dimensional slices. Alternatively, 3-dimensional cine data sets covering the entire heart in a single breath-hold can be acquired. In addition to its high tissue contrast, the main advantage of CMR over other imaging modalities is that imaging planes can be defined freely and reproducibly. Consequently, CMR is the most accurate and reproducible imaging modality for the assessment of global ventricular volumes and function. In addition, regional contractile function can be assessed either by visual interpretation of cine loops or by measuring wall motion, thickening, and strain using myocardial tagging methods. Myocardial tagging during low-dose dobutamine stress has been used to measure parameters of diastolic dysfunction such as the time to peak untwist, which may identify coronary stenosis. After acute myocardial infarction (AMI), low-dose dobutamine cine CMR can be used to predict viability and functional recovery. High-dose dobutamine stress CMR has high diagnostic accuracy to identify inducible LV wall motion abnormalities indicative of flow-limiting coronary stenosis.

First-Pass Myocardial Perfusion
Current first-pass myocardial perfusion CMR methods track the passage of a bolus of a T1-shortening contrast agent injected into a peripheral vein. Data acquired during intravenous vasodilator stress (most commonly with adenosine) delineate relatively underperfused regions associated with myocardial ischemia. The spatial resolution of CMR myocardial perfusion imaging of 2 to 3 mm is vastly superior to other imaging modalities, so subendocardial ischemia can be identified more reliably. Recent developments have seen further improvements in spatial resolution to ≈1 mm in the imaging plane, and acquisition at 3 T promises improved signal-to-noise ratio and diagnostic yield.

Early and Late Gadolinium Enhancement
After acute ischemic injury, the myocardial distribution of extracellular gadolinium-based contrast agents is increased because of the presence of sarcolemmal disintegration and abnormal washout kinetics. In chronic MI, the...
presence of fibrotic tissue increases the distribution volume of the contrast agents. The resulting differences in contrast distribution between normal and injured myocardium can be delineated with T1-sensitive inversion-recovery CMR methods. Imaging within the first few minutes after contrast administration is the method of choice to delineate MVO, which prevents contrast delivery to the infarct core and thus results in low signal on T1-weighted imaging. Acutely injured and chronically infarcted tissue without MVO, on the other hand, retains contrast agent and therefore appears bright. The preferred imaging time for scar is between 10 and 20 minutes after contrast agent administration, when the differences between scar, normal myocardium, and blood pool are maximal. This method is referred to in the literature variably as late gadolinium-enhanced CMR (the currently preferred term), late contrast-enhanced CMR, delayed contrast-enhanced CMR, or hyperenhancement CMR. It has become the reference standard for the in vivo assessment of myocardial viability because of its very high spatial definition and high contrast to normal myocardium, which allows detailed assessment of the spatial distribution of scar. Because of its high spatial resolution, late gadolinium-enhanced CMR can detect infarction in as little as 1 mL tissue, substantially less than other in vivo methods. The technique has been validated extensively in animal models, showing excellent agreement with histology, and has been applied in numerous recent human studies. Most notably, it was shown that CMR is more sensitive in detecting subendocardial MI than single-photon emission computed tomography or positron emission tomography in chronic CAD that the extent of scar on CMR predicts the potential for functional recovery after revascularization.

Figure 2 shows a case example of early and late gadolinium enhancement after acute MI.

**T2-Weighted Imaging**

Myocardial edema is a feature of many forms of acute myocardial injury that are associated with inflammation. Edema alters myocardial T2 relaxation and can therefore be detected with T2-weighted CMR imaging. After acute MI, T2-weighted CMR can be used to delineate the ischemic risk region, which typically extends beyond the scar (Figure 3). This is discussed in greater detail later. However, both the relatively small contrast-to-noise ratio between edematous and normal myocardium (≈2 to 3) and artifacts from slow-flowing blood at the subendocardial border can make interpretation of T2-weighted images more challenging than other CMR methods, although recent methodological developments promise to improve these limitations.

**Coronary MR Angiography**

CMR imaging can be used to delineate coronary morphology and to detect at least proximal coronary stenosis. Coronary MR angiography, however, is rarely used in ACS, in which invasive angiography is a routine test and noninvasive coronary imaging has little to add to the diagnostic process.

**Summary**

In summary, CMR offers a wide range of tools that can be used for the detection, differential diagnosis, and management of patients with acute and chronic manifestations of CAD. Data acquisition times for most CMR methods continue to be reduced, allowing multiparametric assessment in a single imaging session. In the following sections, the estab-
Established and evolving clinical applications of CMR in ACS are discussed.

**Detecting and Differentiating ACS**

**Detection of ACS**

Patients with suspected ACS are increasingly managed with early interventional strategies. However, in low-risk patients or in the presence of concomitant medical problems that increase the risk of complications from cardiac catheterization, an initial noninvasive functional test may be preferred. In this context, CMR presents an attractive alternative to established diagnostic methods. A study by Kwong and colleagues suggested that CMR imaging may identify ACS more accurately than conventional markers. In 161 consecutive patients presenting to the emergency room with cardiac chest pain but no evidence of MI, CMR was performed within 12 hours of presentation. The CMR protocol comprised myocardial perfusion at rest, cine imaging, and late gadolinium enhancement. The study reports a sensitivity and specificity of 84% and 85%, respectively, of CMR for detecting ACS defined as 70% coronary stenosis or positive stress test within 8 weeks of the index event. Detection of regional wall motion abnormalities was the most powerful part of the CMR study in this setting, in which perfusion abnormalities may be normal between episodes of pain and infarction may not yet be established. CMR was more sensitive than ECG, troponin, and the Thrombolysis in Myocardial Infarction (TIMI) risk score and was the strongest predictor of ACS on multivariate logistic regression analysis.

CMR is also helpful in differentiating acute from chronic MI by combining late gadolinium enhancement with T2-weighted imaging, which will delineate the edema associated with acute infarction. In a study of 73 patients with acute and chronic MI by Abdel-Aty and colleagues, CMR was 96% sensitive in differentiating acute from chronic MI. The incremental value of T2-weighted imaging for the detection of ACS was the subject of a recent study of 64 consecutive patients presenting with chest pain to the emergency room with negative cardiac enzymes and no ECG changes suggestive of coronary ischemia. Adding T2-weighted imaging and LV wall thickness measurements to a core CMR protocol of cine and late gadolinium enhancement imaging increased the specificity, positive predictive value, and overall accuracy in detecting ACS from 84% to 96%, 55% to 85%, and 84% to 93%, respectively. CMR provided incremental value in the detection of ACS over and above traditional risk stratification, with the changes detected by CMR occurring before the rise in cardiac enzymes (6±12 hours).

Besides detecting early changes after ACS, CMR is useful in the setting of delayed presentations in which cardiac markers may have returned to normal, whereas abnormalities on T2-weighted CMR can persist for several weeks.

**Differential Diagnosis of ACS**

CMR can facilitate the differential diagnosis of ACS, particularly in the context of a normal coronary angiogram. In a study of 27 patients with troponin-positive chest pain and normal x-ray coronary angiogram, half showed subepicardial, or midwall, late gadolinium enhancement, suggesting myocarditis, and the other half demonstrated subendocardial, or transmural, enhancement typical of MI. Another study of 61 patients presenting with troponin-positive chest pain and normal coronary angiography showed that contrast-enhanced
CMR was able to identify a cause in 65% of cases, with the commonest being myocarditis (50%), followed by MI and cardiomyopathy. Laissy and colleagues had similar results in a study of 55 patients, including 24 patients with clinically suspected myocarditis and normal coronary angiography and 31 with a history of atypical MI and coronary stenosis. In all but 1 patient with myocarditis, CMR perfusion was normal, and late gadolinium-enhanced CMR showed either epicardial or diffuse nonsegmental enhancement, whereas MI patients showed typical endocardial enhancement. The pattern of CMR abnormalities in myocarditis may even predict long-term outcome. Figure 4 shows an example of late gadolinium enhancement in a patient with myocarditis.

Takotsubo cardiomyopathy, or apical ballooning syndrome, is a syndrome with distinctive features such as acute chest pain and shortness of breath, ST-segment elevation on ECG, and release of cardiac enzymes. Consequently, it can mimic AMI at clinical presentation. The diagnosis is usually suspected during invasive coronary angiography, which typically reveals nonobstructed coronary arteries and an apical wall motion abnormality crossing coronary supply territories. CMR can reliably diagnose the abnormal apical contraction that characterizes the syndrome, and late gadolinium-enhanced CMR shows the absence of myocardial necrosis in this syndrome and reliably predicts recovery (Figure 5). A study by Eitel et al demonstrated the ability of CMR to distinguish apical AMI from apical ballooning syndrome without infarction and myocarditis in patients with angiographically normal coronary arteries and characteristic wall motion abnormalities.

In summary, CMR may be a useful and accurate test to detect the presence of ACS and can be considered an additional diagnostic tool to differentiate ACS from chronic MI and from disease entities with similar clinical presentations, such as myocarditis.

Management of ACS

Non-ST-Elevation ACS

Current guidelines for the management of non-ST-elevation ACS recommend that low-risk patients with normal biomarkers should undergo a stress test (nuclear perfusion imaging or stress echocardiography) within 72 hours as an alternative to inpatient admission. Plein et al showed that CMR can also be used safely in the context of ACS. A CMR study incorporating cine imaging, rest and stress perfusion, coronary MRA, and late gadolinium enhancement was performed within 2 to 5 days of non-ST-elevation ACS in 72 patients. CMR reliably predicted the presence of coronary stenosis requiring revascularization on subsequent x-ray coronary angiography, particularly when several of the CMR modules were interpreted in combination (sensitivity, 96%; specificity, 83%). Furthermore, this was superior to the prediction based on the TIMI risk score. Figure 6 gives a case example of a patient presenting with a biomarker-negative ACS in whom CMR identified unknown previous MI and inducible ischemia in 2 separate coronary territories.

Ingkanisorn et al subsequently showed that CMR adds significant prognostic value in the prediction of future diagnosis of CAD, MI, or death over clinical risk factors. Using adenosine stress perfusion CMR, they studied 135 troponin-negative patients presenting to the emergency room with chest pain. CMR had 100% sensitivity and 93% specificity for predicting the development of CAD at the 1-year follow-up.

ST-Elevation MI

After acute ST-elevation MI (STEMI), patients may not receive definitive revascularization at the time of initial presentation for a variety of reasons, including late presentation, concomitant medical problems that exclude reperfusion strategies, or widespread or complex coronary disease that is

Figure 4. CMR images from a 49-year-old woman presenting with chest pain and breathlessness. Cardiac serum biomarkers were raised, and an ECG showed widespread T-wave inversion. Coronary angiography showed normal coronary arteries with no evidence of coronary atheroma. CMR shows a regional wall motion abnormality predominantly in the inferior segments (A, diastole; B, systole). Late gadolinium enhancement images (C) show extensive epicardial hyperenhancement in the inferior and near-transmural enhancement in the lateral segments. Based on all available results, a clinical diagnosis of myocarditis was made. The patient declined cardiac biopsy.

Figure 5. CMR images from a 52-year-old woman presenting with an episode of severe chest pain associated with marked anterior ST elevation and raised serum biomarkers. Coronary angiography revealed normal coronary arteries with no atherosomatous disease. An LV angiogram showed a marked apical wall motion abnormality, raising the suspicion of takotsubo syndrome. CMR confirmed the typical diastolic apical “ballooning” on cine images (A, B) and the absence of scar on late gadolinium-enhanced images (C).
not suitable for percutaneous revascularization. The detection and quantification of scar by late gadolinium-enhanced CMR is increasingly used to guide revascularization decisions in these patients. In a group of 50 patients with MI, 6 of whom were studied within 2 weeks of infarction, Kim et al.\(^{34}\) showed that late gadolinium-enhanced CMR predicts reversible myocardial dysfunction. Ninety percent of the myocardial segments studied that contained hyperenhancement of between 51% and 75% of tissue and virtually all of those with transmural infarction did not improve after revascularization. Conversely, 256 of 339 hypokinetic segments (78%) containing no hyperenhancement had improved contractility after revascularization. A subsequent study by Nijveldt et al.\(^{58}\) in 60 patients with recent AMI confirmed that segments with >75% transmural enhancement are unlikely to function completely at follow-up, whereas in about half of the segments with <25% transmural enhancement, function improved completely. These results were supported by Bodi et al.\(^{59}\) who showed that the amount of viable myocardium on CMR predicts functional recovery in thrombolysed STEMI patients who receive posthoc revascularization.

In patients receiving thrombolytic therapy for the treatment of acute STEMI, functional assessment with exercise testing or a pharmacological imaging study is recommended for low-risk patients before discharge from hospital.\(^{60}\) Greenwood and colleagues,\(^{61}\) using a small sample of 35 patients and CMR imaging with adenosine stress perfusion, showed that viability and function assessment can be safely performed within a few days of acute STEMI and is more accurate than an exercise tolerance test for detecting residual ischemia.

**Complications of ACS**

In addition to supporting management decision making in ACS, CMR reliably detects important complications of AMI. Ventricular aneurysms and ventricular septum defects are clearly identified on cine images. With late gadolinium-enhanced CMR, these pathologies can be further characterized in the context of the acute injury and may aid planning of surgical or percutaneous procedures.\(^{62}\) Early or late gadolinium-enhanced imaging is also very useful for identifying LV thrombus\(^{63,64}\) (Figure 7).

**Summary**

In summary, there is early evidence that using CMR in ACS patients is safe and provides a comprehensive assessment of the sequelae of AMI that can help to guide patient management. CMR has the benefit over other imaging modalities of providing the most accurate information on cardiac morphology, function, and scar. In addition, CMR can determine the presence of residual myocardial ischemia and reliably detects complications associated with ACS.

**Risk Stratification After ACS**

In patients with known or suspected CAD, CMR has predictive value for future adverse cardiac events comparable to nuclear scintigraphy or stress echocardiography.\(^{65}\) Even small amounts of scar on late gadolinium-enhanced CMR provide...
ACS, several CMR measures are associated with prognosis of infarction also was associated with worse outcome. Wu et al showed that the amount of myocardium exhibiting an intermediate signal on late gadolinium-enhanced CMR images was a strong predictor of all-cause mortality. A subsequent study suggested that 1 mechanism for this increased mortality may be that the peri-infarct zone is a substrate for arrhythmias. Ingkanisorn et al suggested that the extent of the peri-infarct zone may reflect partial myocardial necrosis and edema. A study by Yan et al suggested that the extent of the peri-infarct zone is an independent predictor of post-MI mortality. In 144 patients with documented CAD and previous MI, they found that the amount of myocardium exhibiting an intermediate signal on late gadolinium-enhanced CMR images was a strong predictor of all-cause mortality. A subsequent study suggested that 1 mechanism for this increased mortality may be that the peri-infarct zone is a substrate for arrhythmias. CMR data on scar and peri-infarct zone could thus prove useful in the future to identify patients for an implantable cardioverter-defibrillator and cardiac resynchronization therapy.

**Infarct Size**

Infarct size measured by late gadolinium enhancement is directly associated with outcome. Tarantini and colleagues showed in 76 patients with acute revascularized MI that the amount of transmural necrosis on late gadolinium-enhanced CMR predicted adverse LV remodeling, with significant additional predictive value over infarct size and MVO. These findings were confirmed by Roes et al, who showed in 231 patients with healed MI that infarct size on late gadolinium-enhanced CMR was a stronger predictor of all-cause mortality than LV ejection fraction and LV volumes. Transmurality of infarction also was associated with worse outcome. Wu et al showed that acute infarct size as determined by CMR, which was independent of LV stunning and loading, directly relates to LV remodeling and is a stronger predictor of future events than measures of LV systolic performance.

**Peri-Infarct Zone**

On late gadolinium-enhanced CMR images, a border zone of intermediate signal can be observed between the infarct and surrounding tissue. This peri-infarct zone may reflect partial volume or partial myocardial necrosis and edema. A study by Yan et al suggested that the extent of the peri-infarct zone is an independent predictor of post-MI mortality. In 144 patients with documented CAD and previous MI, they found that the amount of myocardium exhibiting an intermediate signal on late gadolinium-enhanced CMR images was a strong predictor of all-cause mortality. A subsequent study suggested that 1 mechanism for this increased mortality may be that the peri-infarct zone is a substrate for arrhythmias. CMR data on scar and peri-infarct zone could thus prove useful in the future to identify patients for an implantable cardioverter-defibrillator and cardiac resynchronization therapy.

**Microvascular Obstruction**

Early contrast-enhanced CMR can reliably demonstrate the presence and extent of MVO after reperfused AMI. MVO results in poor tissue perfusion despite restoration of epicardial blood flow to the infarcted region. It is the consequence of clogging of the small myocardial arterioles with embolic debris, acute inflammation, platelet aggregation, and vasospasm. Studies have shown that the presence and extent of MVO after AMI as measured by early gadolinium enhancement CMR are associated with adverse ventricular remodeling and clinical outcome that is independent of the infarct size. Baks et al examined patients with CMR soon after primary percutaneous revascularization for STEMI and again at 5 months. They found that dysfunctional segments without MVO had early increased wall thickness and late partial functional recovery compared with segments with MVO that showed late wall thinning and no functional recovery. Nijveldt et al demonstrated that MVO had incre-

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>n</th>
<th>Initial CMR Scan</th>
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<th>CMR Method</th>
<th>Main Parameter Examined</th>
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<tr>
<td>Wu et al</td>
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<td>44</td>
<td>10±6 d after AMI</td>
<td>6-mo CMR, 16±5-mo clinical</td>
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<td>MVO</td>
<td>Predicts poor LV recovery with increased MACE</td>
<td>&lt;0.01</td>
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<td>24 h after PPCI</td>
<td>3-mo CMR</td>
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<td>Predicts poor LV recovery</td>
<td>0.02; 0.048</td>
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<tr>
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<td>Predicts poor LV recovery with increased MACE</td>
<td>0.0074</td>
</tr>
<tr>
<td>Baks et al</td>
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<td>5 d after PPCI</td>
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<td>First-pass perfusion</td>
<td>MVO</td>
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<td>0.006</td>
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<td>Tarantini et al</td>
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<td>Predicts poor LV recovery</td>
<td>&lt;0.001</td>
</tr>
<tr>
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<td>&lt;72 h after                 troponin-negative chest pain</td>
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<td>Adenosine stress perfusion</td>
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<td>Highly accurate for predicting CAD, MI, death</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Larose et al</td>
<td>2007</td>
<td>147</td>
<td>&gt;30 d after AMI</td>
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<td>Cine and LGE</td>
<td>RV function (EF &lt;40%)</td>
<td>Increased mortality independently of LV function</td>
<td>&lt;0.003</td>
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<td>Predicts mortality</td>
<td>0.005</td>
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<tr>
<td>Wu et al</td>
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<td>122</td>
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<td>LGE</td>
<td>Acute infarct size (&gt;18% LV)</td>
<td>Increased MACE</td>
<td>&lt;0.05</td>
</tr>
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PPCI indicates primary percutaneous coronary intervention; MACE, major adverse cardiovascular event; LGE, late gadolinium enhancement; and EF, ejection fraction.
ment diagnostic value over transmurality of infarction, particularly in segments with 75% to 100% transmural enhancement.

Area at Risk
The area at risk is defined as hypoperfused myocardium at the time of an ischemic episode. Currently, the clinical gold standard for determining the area at risk is single-photon emission tomography with injection of technetium-99m tracer into the occluded coronary artery before revascularization. In the experimental setting, myocardial contrast echocardiography also has been used.

T2-weighted CMR offers a potentially attractive alternative for the noninvasive measurement of area at risk. The increased water content of myocardium after acute ischemia/reperfusion injury leads to high signal on T2-weighted images and, combined with late gadolinium enhancement, allows the delineation of areas that are injured but not infarcted after reperfusion. The areas of T2 enhancement are invariably transmural and subsequently larger than the regions of late gadolinium enhancement, and the difference between them likely represents myocardial salvage.

The advantage of this method is that such a region can be retrospectively determined days after the acute event without the need for direct injection of agents into the coronary artery at the time of primary reperfusion. So far, no outcome studies based on CMR assessment of area at risk have been published.

RV Infarction
It is well recognized that involvement of the right ventricle in acute MI is associated with adverse clinical outcome. RV function is difficult to assess reliably with most imaging modalities, whereas it poses no particular challenge to CMR cine imaging. Kaandorp et al showed that RV infarction can be detected by late gadolinium enhancement and that the extent of scar tissue is linearly related to the severity of RV dilatation at a 6-month follow-up. Kumar et al found in 37 patients that acute RV infarction is detected more frequently by late gadolinium enhancement than by ECG and echocardiography. Finally, Larose et al have shown that RV ejection fraction measured by CMR is an important predictor of prognosis after AMI. In 147 consecutive patients studied late after MI, RV ejection fraction <40% was strongly associated with mortality (hazard ratio, 4.02) independently of patient age, LV infarct size, and LV ejection fraction.

Summary
In summary, CMR imaging provides several independent measures of prognosis after ACS that can all be obtained in a single imaging procedure and cannot be assessed equally with other imaging modalities. A potential role for CMR may thus be to offer an improved method for risk stratification in the early post-ACS period.

Future Perspectives
Owing to ongoing technological advances (eg, faster gradients, multichannel receiver coils, transform coding to accelerate data acquisition), scan times for many CMR methods continue to be shortened. Until now, this development has usually been invested into obtaining higher-quality images (eg, better spatial resolution, better temporal resolution, better signal-to-noise ratio). However, given the image quality achieved today, future improvements in imaging speed are likely to be reinvested into shortening scan time for a given CMR method. This speedup can be used to evaluate more patients for chronic disease and to answer numerous questions within a single short session for patients with ACS. A likely scenario for CMR in these patients will resemble one of the current uses of cardiovascular computed tomography in patients with chest pain: the triple ruling out of pulmonary embolism, aortic dissection, and MI in a single imaging session. Such an approach would look at wall motion abnormalities, edema, and resting perfusion first and then would use a higher bolus of contrast agent for a time-resolved 3-dimensional angiography, which allows assessment of the anatomy of the pulmonary arteries and the aorta within a single scan and the final assessment of late gadolinium enhancement to visualize scar tissue.

In vivo MRI of advanced plaques in human carotid arteries with contrast-enhanced techniques has identified lipid core with 85% sensitivity and 92% specificity. In addition, thin- and ruptured plaques can be identified in vivo by MRI in a high proportion of patients in whom it was subsequently identified on examination of postatherectomy specimens. Improving methods and the introduction of 3-T clinical imaging raise the possibility that in the near future such techniques may be applied to the coronary arteries.
More specific techniques to visualize activation within a plaque using targeted (molecular) contrast agents could be used as a potential predictor of plaque rupture, with the potential for identification of specific disease processes in vivo. This has already been demonstrated in animal models for fibrin-specific contrast agents, which allow the highlighting of ruptured plaques and thrombosis within the coronary arteries. Similarly, contrast agents designed to show earlier processes of plaque vulnerability such as myeloperoxidase activity and macrophage activation have been applied in animal models. If translated into clinical application, the ability to assess plaque activity noninvasively may overcome a major limitation of the current practice of cardiology.

**Which Patients With ACS Should Undergo CMR Imaging?**

- In patients presenting with suspected ACS but no angiographic evidence of coronary artery stenosis, CMR can contribute to the differential diagnosis of ACS from other acute myocardial diseases such as myocarditis.
- In low-risk patients presenting with suspected ACS, an early stress-perfusion CMR scan can be considered an alternative noninvasive stress test to risk stratify and discharge patients.
- Patients with established MI may undergo CMR after diagnostic angiography to determine the degree of myocardial necrosis and the likelihood of recovery to plan further revascularization strategies.
- After AMI, CMR also may be used to risk stratify patients using LV ejection fraction, infarct size and characteristics, and RV involvement. On the basis of these data, high-risk patients, for example, with poor ventricular function and a large scar burden can be selected for implantation of a cardioverter-defibrillator.
- A CMR protocol for these clinical scenarios that follows the recent recommendations of the Society of Cardiovascular Magnetic Resonance is given in Figure 1b.

**Conclusions**

CMR imaging is emerging as a versatile diagnostic tool for the management of the patient with suspected or established ACS. It provides additional information over other clinical tests for the detection, differential diagnosis, and prognostication after ACS owing to the high spatial definition and multimodal data it provides. Future larger studies will determine more fully the role of CMR in the setting of ACS.

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