

STATE-OF-THE-ART PAPER

Cardiac Magnetic Resonance Imaging for the Interventional Cardiologist

Gemma A. Figtree, MBBS, DPHIL,*† Jacob Lønborg, MD,*§
Stuart M. Grieve, MBBS, DPHIL,*‡ Michael R. Ward, MBBS, PhD,*†
Ravinay Bhindi, MBBS, PhD*†

Sydney, Australia; and Copenhagen, Denmark

Cardiac magnetic resonance imaging is a noninvasive technique for assessing heart structure and function without the need for ionizing radiation. Its ability to precisely outline regions of myocardial ischemia and infarction gives it an important role in guiding interventional cardiologists in revascularization. Its ability to characterize and precisely quantify abnormal regurgitant flow volumes or abnormal shunts also makes it a valuable tool for many noncoronary interventions. This review will discuss the evidence for cardiac magnetic resonance in guiding complex therapies in the catheter laboratory, as well as practical issues that need to be addressed to allow the application of this powerful tool to an increasing number of our patients. (J Am Coll Cardiol Intv 2011;4:137–48) © 2011 by the American College of Cardiology Foundation

Magnetic resonance imaging (MRI) has revolutionized medical imaging. However, its application to the heart is still evolving rapidly because of complexities in studying a moving organ. Unique challenges of *cardiac* MRI (cardiac magnetic resonance [CMR]) relate to the need for specialized hardware (“coils”) and complex software that is unique to the heart. Technology in the field is continuing to develop rapidly, allowing CMR to improve diagnostic accuracy in our patients without the use of invasive or radiation-dependent tests. Furthermore, CMR has the advantage over echocardiography and radionuclide scintigraphy in that images can be obtained in any tomographic plane without limitations imposed by body habitus.

Its application to cardiovascular research is also invaluable, allowing precise quantification of myocardial infarction (MI) size, myocardium salvage index, left ventricular (LV) function, and response to novel treatments. This review will discuss the basic protocols used for CMR, and then focus on its multiple potential roles for the interventional cardiologist.

CMR physics: technical considerations for a standard examination. For MRI, a powerful magnetic field (most commonly 1.5- or 3.0-T) is used to align the nuclear magnetization of hydrogen atoms (protons) present in the water and fat of the body. Radiofrequency electromagnetic fields are used to modify the alignment of magnetization in this large magnetic field. When the radiofrequency source is switched off, the magnetic vector returns to its resting state, emitting a radio wave signal that is received by coils placed around the body part of interest. Characteristics of this received signal are used to construct an image. Different tissues relax at different rates when the transmitted radiofrequency pulse is switched off—a fact that is exploited to generate clinically important image contrast. The time taken for the magnetic vector to return to its equilibrium state is called T_1 relax-

From the *North Shore Heart Research Group, Kolling Institute, University of Sydney, Sydney, Australia; the †Department of Cardiology, Royal North Shore Hospital, Sydney, Australia; the ‡Department of Radiology, Royal Prince Alfred Hospital, Sydney, Australia; and the §Department of Cardiology, Rigshospitalet, Copenhagen, Denmark. Supported by North Shore Heart Research Foundation, Sydney, Australia, and Sydney Medical Foundation, Australia. Dr. Lønborg was supported by Danish Heart Foundation, Danish Cardiovascular Research Academy, and Rigshospitalet Research Foundation. All other authors have reported that they have no relationships to disclose.

Manuscript received June 24, 2010; revised manuscript received September 10, 2010, accepted September 17, 2010.

ation. Gadolinium hastens T_1 relaxation, and thus causes a local signal increase in the area of increased gadolinium concentration when using appropriately T_1 -weighted sequences. T_2 relaxation describes the loss of phase coherence of the magnetic vectors that form the signal.

To allow for the creation of a CMR image, pulsed magnetic field gradients are applied in 3 dimensions before the acquisition of the signal. For 2 of these dimensions, this gradient is invariant with each pulse. However, with the third dimension, the gradient is a variable strength pulse allowing for spatial reconstruction via a process known as “phase encoding.” Therefore, each MRI image must be acquired in several steps and, in the case of the heart, over several heartbeats. It is possible to acquire CMR data in “real-time” during single heartbeats using fast imaging techniques. However, this involves substantial tradeoffs in terms of spatial and temporal resolution and is reserved for applications where the real-time data during free breathing is helpful, such as for constrictive pericarditis (1,2). Instead, most CMR acquisitions use breath-hold and electrocardiogram (ECG) gating, allowing coordination of the acquisition to the correct phase of the cardiac cycle with the assumption that the heart is perfectly periodic. This process works well for most patients; however, in patients unable to perform breath holds, “navigator” sequences have also been developed that “tag” the diaphragm, and allow for free-breathing (3).

Abbreviations and Acronyms

CAD = coronary artery disease

CMR = cardiac magnetic resonance

CT = computed tomography

ECG = electrocardiogram

LGE = late gadolinium enhancement

LV = left ventricle

MI = myocardial infarction

MRI = magnetic resonance imaging

SPECT = single-photon emission computed tomography

3D = 3-dimensional

CMR physics: a standard examination. A standard CMR acquisition protocol is outlined in Table 1. After an initial scout sequence to locate anatomical landmarks, specific acquisition sequences are used to examine: 1) cardiac function; 2) tissue characterization; 3) flow of blood from the left and right ventricular outflow tracts; 4) perfusion of the myocardium (with and without adenosine); and 5) areas of fibrosis or infiltration (late gadolinium enhancement [LGE]). In contrast to a cardiac computed tomography (CT), where a volume of data covering the whole heart is acquired, specific planning of imaging planes is required prospectively as illustrated in Figure 1. Contiguous short-axis planes are acquired for the evaluation of biventricular function, volumes, and mass. Post-acquisition planimetry using either semiautomated or manual methods defines the endocardial and epicardial borders in end diastole and end systole (illustrated in Fig. 2A). The accuracy of global LV volume measurements by CMR is well established (4,5). As a 3-dimensional (3D) dataset is acquired, all

segments of the LV are well seen, and subtle wall motion abnormalities are easier to detect than in transthoracic echocardiography (6–8). Contractility is reported for each myocardial segment (Fig. 2B) to allow for easy interpretation with coronary artery anatomy.

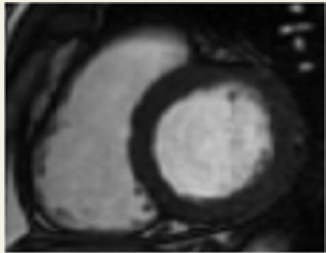
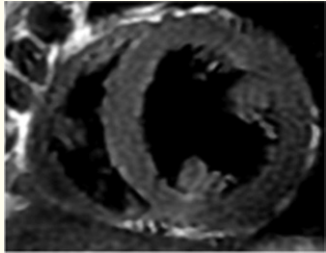
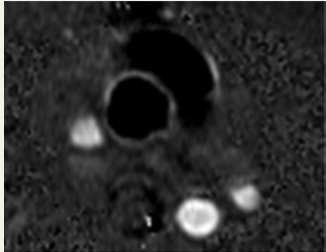
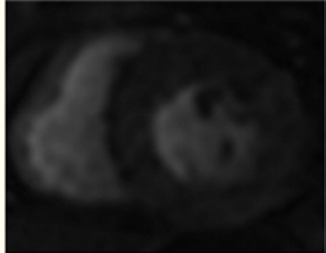
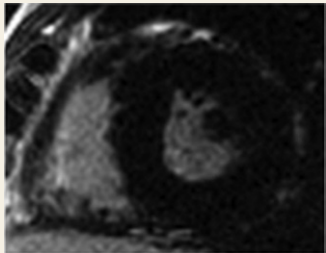
Specific Applications of CMR to Interventional Cardiology

CMR has a role in a broad range of patients presenting with suspected coronary artery disease (CAD). Its major contributions to this patient cohort are in the assessment of segmental wall motion abnormalities, reversible ischemia, myocardial edema, and irreversible myocardial injury. Cine sequences are used in almost all CMR studies to assess function. The application of perfusion (with or without pharmacological “stress”), T_2 -weighted, and LGE sequences depends on the clinical question being asked. This review will briefly discuss CMR techniques used for specific patient populations.

CMR in patients with acute coronary syndrome. Even after short periods of coronary artery occlusion, ischemic myocardium becomes edematous because of Na^+ accumulation and ceases to contract normally (9). Cell death occurs over time within the ischemic myocardium starting from the subendocardial myocytes, and progressing as a wave front into the mid-myocardium and then the subepicardium over hours (10). The ability of CMR to detect edematous myocardium using T_2 -weighted sequences effectively identifies a myocardium that was ischemic before reperfusion, and thus “at risk” of infarction and commonly termed “area at risk” (11–16). This can assist in identifying patients who have had aborted MI with minimal troponin elevation (13) and in guiding the interventionalist to the culprit vessel in the setting of multiple coronary artery stenoses of moderate degree. This method of identifying myocardium at risk has been validated by comparisons with the area at risk determined by histopathology (11) and fluorescein uptake in animal models (17), as well as by technetium Tc 99m tetrofosmin single-photon emission computed tomography (SPECT) (18), and to angiographic and contrast-enhanced CMR measurements of area at risk (16). T_2 -weighted imaging also identifies regions of hemorrhage, which are seen as areas of signal loss within the edema (19).

In contrast to T_2 -weighted imaging of edematous myocardium, LGE CMR identifies regions of irreversible injury, and thus completed infarction. Ex vivo MRI has demonstrated that the spatial extent of infarction identified using late enhancement CMR was the same as the spatial extent of myocyte necrosis in an animal model of MI (20). Infarct size determined by LGE also closely correlates with peak cardiac enzymes (21,22) and SPECT (23,24). Cardiac magnetic resonance is demonstrably superior to SPECT in detecting subendocardial and nonanterior infarcts, in part, because CMR

Table 1. Different Sequences for Different Tasks

Focus of CMR Sequences	Technical Detail	Example Images
Cine images: cardiac volumes, mass, and function	The SSFP sequence provides excellent contrast between the myocardium and the blood pool. The cardiac cycle is divided into 12 to 30 "phases." Individual images of each of these phases are then stitched together to form a "cine" movie of the cardiac cycle.	
Structure and tissue characterization	A family of sequences called short tau inversion recovery are used to enhance tissue characterization. These use multiple pulses to null the signal from the blood pool, creating a "black blood" effect and, thus, emphasizing signal properties from the myocardium. The sequences can be relatively T ₁ - or T ₂ -weighted. T ₁ -weighting can assist in identification of fatty infiltration, such as in ARVC. T ₂ -weighting is used to identify regions of myocardial edema in inflammation or ischemia.	
Blood flow and velocity	Velocity-encoded sequences allow the quantification of flow across cardiac valves. They achieve this by using special gradient pulses that "sensitize" the images to flow in a precise and quantifiable manner, permitting the calculation of velocity and, hence, of bulk flow.	
Myocardial perfusion	CMR is able to image the change in T ₁ -weighted signal intensity of the myocardium during the passage of gadolinium contrast agent. Imaging is performed with each cardiac cycle (usually 3 to 6 short-axis slices). In regions where blood flow is normal, the myocardium appears bright as the gadolinium perfuses the capillaries, whereas dark areas represent ischemic myocardial regions of low perfusion.	
LGE in regions of fibrosis or increased interstitial space	LGE-CMR uses the slow clearance of gadolinium from regions of increased interstitial space, classically fibrosis, infiltration, or cellular necrosis. The most validated sequence to assess this is a 2D-segmented inversion recovery fast gradient echo sequence. Images are acquired starting from 5 to 10 min after intravenous administration of a dose of 0.1 to 0.2 mmol/kg of gadolinium-DTPA. The T ₁ (inversion recovery time) is adjusted to null the normal myocardium before acquiring a short-axis stack through the ventricles, as well as 4-, 2-, and 3-chamber image planes.	

A standard plan for CMR acquisition includes: 1) a scout; 2) black blood axials and coronals; 3) bSSFP cine images in specific cardiac planes (4-chamber shown); 4) black blood images; 5) perfusion images; 6) flows sequences; and 7) LGE images.

ARVC = arrhythmogenic right ventricular cardiomyopathy; bSSFP = balanced steady-state free precession; CMR = cardiac magnetic resonance; DTPA = diethylenetriaminepentaacetic acid; LGE = late gadolinium enhancement; 2D = 2-dimensional.

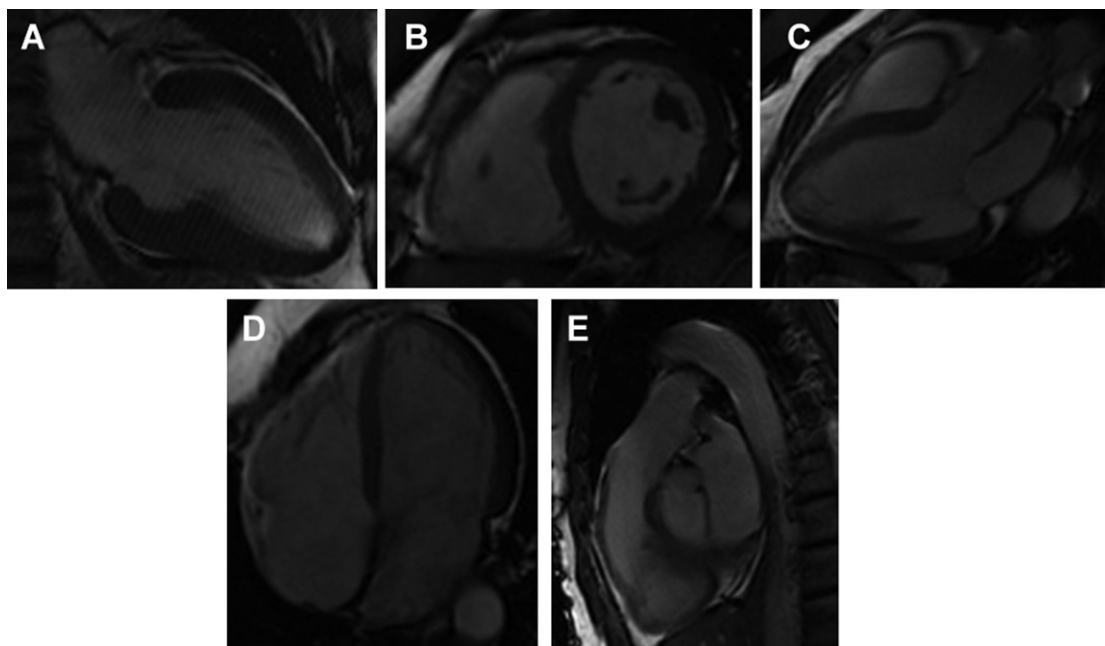


Figure 1. Standard Imaging Planes Acquired Prospectively During CMR

The panels show: (A) the long-axis view of left atrium and left ventricle (LV); (B) the short-axis view through LV at the basal level; (C) the LV outflow tract or 3-chamber view; (D) the 4-chamber or horizontal long-axis view; and (E) the right ventricular outflow tract. CMR = cardiac magnetic resonance.

has greater spatial resolution (25,26). The transmural extent of late enhancement, which reflects the nonviable myocardium, can guide the interventionalist as to the likelihood of benefit from revascularization in patients with acute and chronic MI (21,27–29). Persistent microvascular obstruction (no-flow phenomenon) is also seen as a residual perfusion defect after contrast administration (Fig. 3) (30) and has been shown to be a predictor of mortality (30,31).

An important role for CMR is to detect MI by LGE in patients in whom the troponin and ECG changes are either nonspecific or normalized as shown in a case of an out-of-hospital cardiac arrest with only moderate stenosis at angiography (Fig. 4). The territory of the late enhancement assists the interventionalist in identifying the culprit vessel if there are multiple stenoses or if a culprit is not readily identifiable. Cardiac magnetic resonance has been shown to be substantially more sensitive than ECG at detecting previous silent MIs (32–34). CMR is also useful in assessing complications arising from acute MI including LV pseudoaneurysm (35), ventricular septal defect (36), mitral regurgitation (37), and ventricular thrombus (38,39).

Assessment of patients with chest pain but normal coronary arteries. CMR is a useful tool in the assessment of patients with acute chest pain, ECG changes and elevated troponin, but normal coronary arteries, or a high clinical suspicion of a noncoronary cause. In myocarditis, edema is the hallmark of inflammation caused by increased cellular membrane

permeability. This results in increased T_2 -weighted signal in the affected myocardium, as well as late enhancement. These changes are classically in the mid-wall or subepicardium compared with the subendocardial abnormalities seen with coronary-related myocardial injury (40,41). Cardiac magnetic resonance has been proven useful in differentiating between MI, myocarditis, and takotsubo cardiomyopathy (42). Therefore, in young patients with chest pain, and troponin rise/ECG changes but minimal risk of CAD, CMR can be used to help avoid invasive coronary angiography as illustrated in the case shown in Figure 5. Furthermore, CMR can visualize the thoracic aorta, which may also allow detection of an aortic dissection as an alternative cause of chest pain presentation.

CMR in patients with known chronic CAD. Cardiac magnetic resonance is proving itself a valuable tool in the assessment of patients with known ischemic heart disease being considered for revascularization, assisting clinicians in the identification of ischemic myocardium, and evaluating viability in regions that have impaired contractility. The transmural extent of late enhancement in a particular dysfunctional segment predicts recovery of function after percutaneous intervention as shown in multiple elegant studies (21,27–29). Figure 6 illustrates the use of CMR for assessing regions of reversible ischemia and viability in a patient with known CAD and LV dysfunction. Patients with CAD and LV dysfunction, in whom viability can be demon-

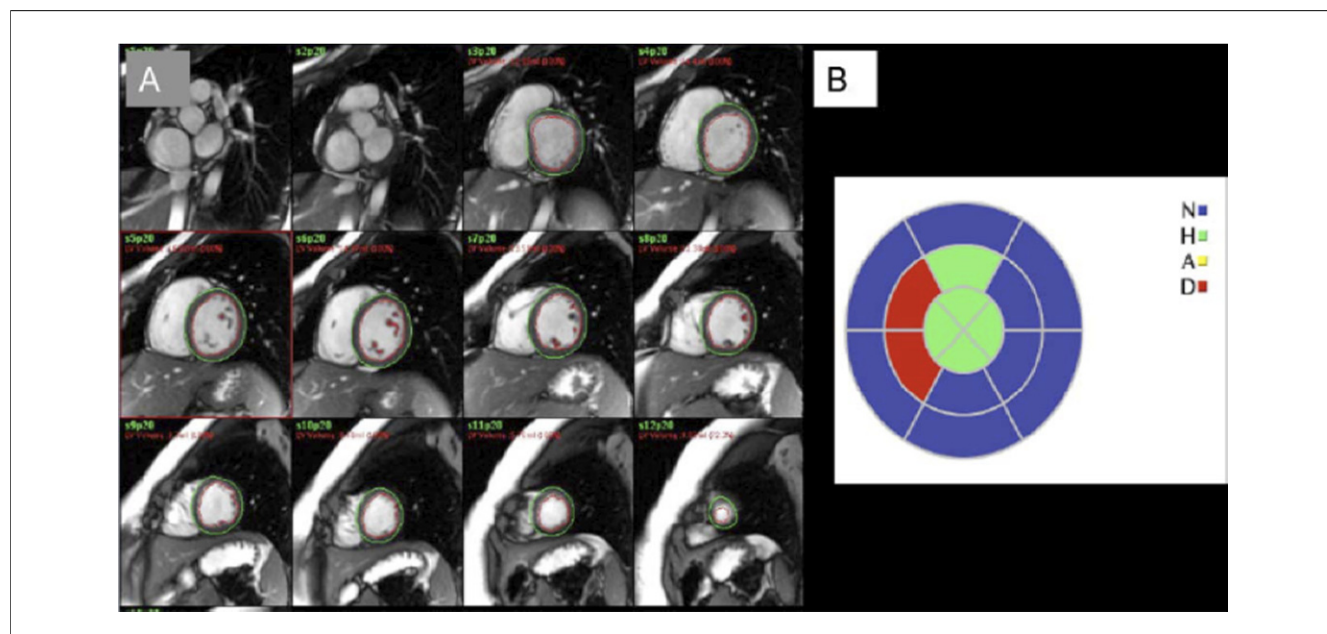


Figure 2. CMR Accurately Assesses Ventricular Volumes

(A) Semiautomated methods are used to define the endocardial and epicardial borders of a short-axis stack of the LV allowing calculation of LV volumes and ejection fraction. A snapshot is shown of GE ReportCARD analysis software (GE Healthcare, Milwaukee, Wisconsin). (B) Contractility is reported according to a standard 16-segment model of the LV myocardium. A = akinetic; D = dyskinetic; H = hypokinetic; N = normal. Abbreviations as in Figure 1.

strated, have improved survival after revascularization. Conversely, patients with nonviable myocardium have no survival benefit from revascularization (43). This has important implications suggesting that CMR can be used to prevent unnecessary procedures that are costly and may harm the patient.

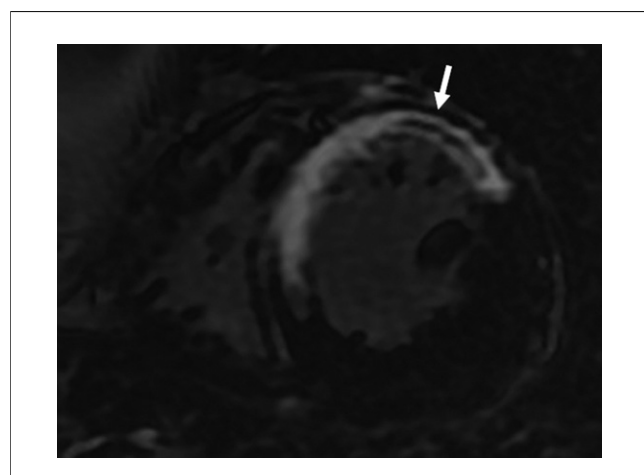


Figure 3. Microvascular Obstruction Seen in Extensive MI With Contrast-Enhanced CMR

Region of persistent microvascular obstruction (low signal: **black**) (arrow) with a region of transmural myocardial infarction (MI) (high signal: **white**) seen on late gadolinium enhancement (LGE) imaging in a patient with late presentation anterior ST-segment elevation MI. Images were acquired on a GE 3-T MR750 (GE Healthcare). Abbreviations as in Figure 1.

CMR and the evaluation of chest pain in patients with suspected CAD. Physicians now have numerous investigations to choose from to assess for myocardial ischemia under conditions of physiological or pharmacological stress. Head-to-head comparisons of CMR perfusion imaging with SPECT suggest a higher diagnostic accuracy of CMR in the detection of epicardial CAD, a difference that might be more pronounced in patients with multivessel disease (44,45). The addition of anatomic, functional, and LGE data provided by CMR without the need for radiation suggests that it has an important role in the assessment of patients suspected of having CAD. The combination of perfusion and LGE CMR has been shown to have a sensitivity of 89%, a specificity of 87%, and accuracy of 88% for diagnosis of CAD with >70% stenosis (46). Perfusion CMR also gives the physician important prognostic information, because the 3-year event-free survival with a normal CMR perfusion scan is 99% compared with 84% in patients with an abnormal scan (47).

Dobutamine stress magnetic resonance is also used to detect myocardial ischemia and assess viability and is particularly useful in patients in whom gadolinium administration is contraindicated such as those with renal dysfunction. The general principles, protocols, and the safety profile are similar to that of dobutamine echocardiography. However, compared with echocardiography, the improved image quality of CMR leads to higher diagnostic accuracy (47), particularly in those individuals in whom echocardiographic

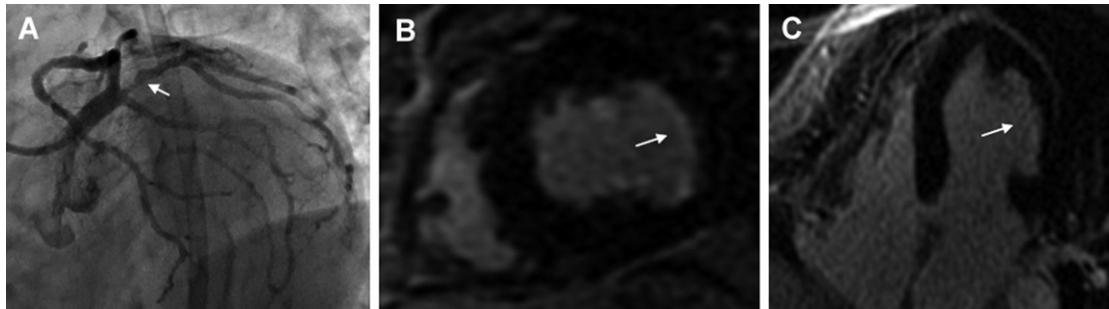


Figure 4. CMR Identifies a Culprit Lesion Causing VF Arrest

A 50-year-old man was referred to our center after successful resuscitation from an out-of-hospital ventricular fibrillation (VF) arrest. Troponin and electrocardiogram (ECG) were nonspecifically abnormal, and attributable to the period of cardiac arrest. Initial angiography reported a lesion in the ramus intermediate (arrow) and mid left anterior descending coronary artery (LAD) that was considered nonobstructive (A). Although transthoracic echocardiography was unremarkable, CMR was performed to exclude a structural abnormality that may be associated with "sudden death" before a planned implantation of an automatic implantable cardioverter-defibrillator (AICD). No evidence of arrhythmogenic right ventricular cardiomyopathy (ARVC) or hypertrophic cardiomyopathy was observed, but subendocardial LGE (arrow) was clearly seen consistent with infarction in the territory of the ramus intermediate territory (B, C). Coronary pressure wire demonstrated that the lesion in the ramus intermediate, considered moderate at initial angiography, was hemodynamically significant with a fractional flow reserve of 0.75. Given the CMR evidence for infarction (arrow) in this territory, and its hemodynamic significance at pressure wire, the lesion was stented. Implantation of an AICD was no longer recommended. The CMR images were acquired using a GE 1.5-T HDxt (GE Healthcare). Abbreviations as in Figures 1 and 3.

image quality is suboptimal. Ischemia is defined as a new wall motion abnormality, or a biphasic response to the increasing dose of dobutamine, and has a sensitivity of 86%

and specificity of 86% (48). Furthermore, the presence of a wall motion abnormality on dobutamine stress magnetic resonance is associated with increased risk of MI and cardiac

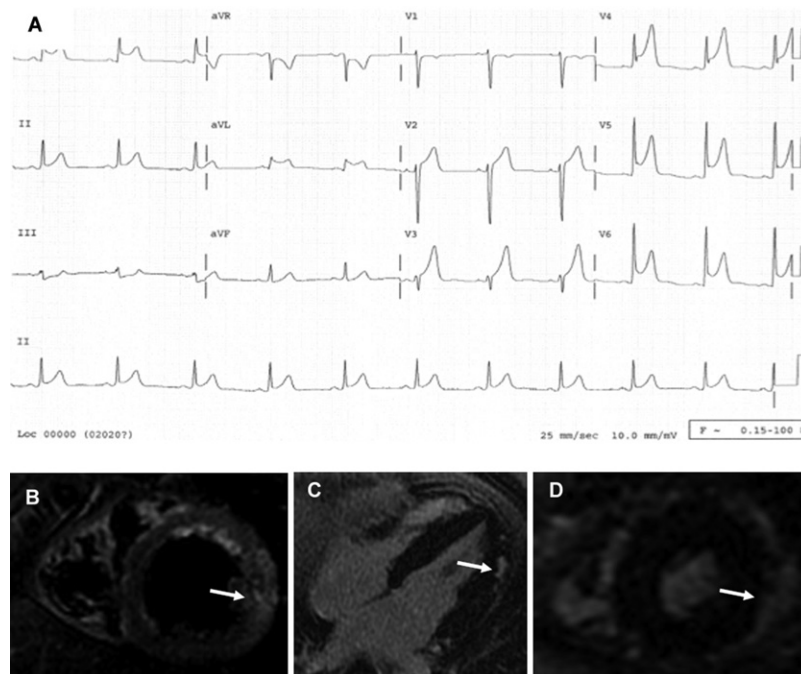


Figure 5. Use of CMR to Determine Cause of Troponin Rise and ECG Changes in Young Patient With No Cardiovascular Risk Factors

Recently in our center, a 23-year-old man presented with severe chest pain and ST-segment elevation in the anterolateral leads (A). CMR demonstrated low normal LV function and features of acute segmental myopericarditis: increased signal on T_2 -weighted double-inversion recovery images was observed in the subepicardial wall (arrows, B), and subepicardial and pericardial LGE was seen in the same territory (arrows, C, D). Invasive coronary angiography was avoided. The CMR images were acquired using a GE 1.5-T HDxt (GE Healthcare). Abbreviations as in Figures 1, 3, and 4.

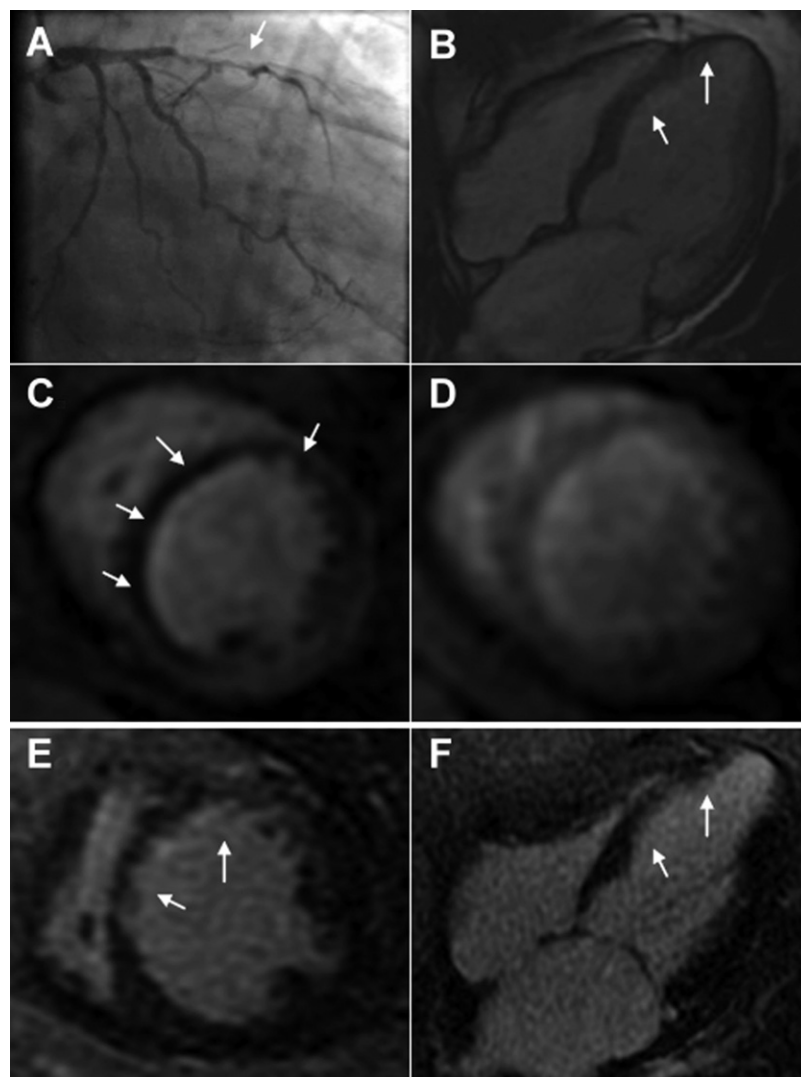


Figure 6. CMR for the Assessment of Ischemia and Viability

A 65-year-old woman presented 4 days after a prolonged episode of chest pain with shortness of breath. Angiography showed a long segment of subtotal occlusion in the LAD just beyond the first diagonal branch (A), but an akinetic anterior wall led the interventionalist to question its viability and, therefore, the appropriateness of revascularization given the late presentation. CMR confirmed akinesis of anterior wall, anteroseptum, and apex (B). An extensive perfusion defect was observed with adenosine (arrow, C), with a large proportion of reversibility (D). The LGE images (E, F) showed subendocardial infarction in the LAD territory (white signal, arrows), but substantial viability (black myocardium), and the lesion was stented with improvements in anterior wall systolic function. This case highlights the ability of CMR to distinguish between hibernating and infarcted myocardium as the cause of akinesis. The CMR images were acquired using a GE 1.5-T HDxt. Abbreviations as in Figures 1, 3, and 4.

death compared with a subject with no abnormalities and an ejection fraction of >40% (49).

Compared with fractional flow reserve measured at the time of angiography, adenosine perfusion CMR has also been used to assess for significant functional coronary stenoses and has been shown to have a sensitivity and specificity of 91% and 94%, respectively (50).

The need for higher accuracy for noninvasive tests has been discussed extensively in the literature. Given the relatively poor positive predictive value of more tradi-

tional screening tests for CAD, as well the recently highlighted low yield of invasive angiography in patients with chronic chest pain (51), CMR with either adenosine perfusion, or dobutamine stress imaging should be considered before angiography in patients assessed as having an intermediate probability of obstructive CAD based on previous investigations. The higher diagnostic accuracy of these CMR investigations may reduce the number of invasive coronary angiograms performed yielding negative results.

CMR in the assessment of cardiomyopathy. Exclusion of CAD is an important aspect of assessing patients with dilated cardiomyopathy. Traditionally, this may involve coronary angiography, and more recently, there has been an increasing use of CT coronary angiography. However, CMR has been shown to be a powerful technique to distinguish dilated cardiomyopathy from LV dysfunction secondary to CAD (52). This may not only reduce the number of invasive coronary angiograms performed unnecessarily in this cohort of patients, but also avoid incorrect assignment of dilated cardiomyopathy to patients who, on CMR, show clear evidence of MI despite an apparently unremarkable angiogram, as may occur for recanalized coronary artery occlusion. Although late enhancement can be seen in dilated cardiomyopathy secondary to fibrosis, its predominant mid-wall localization clearly distinguishes it from the subendocardial pattern of late enhancement seen in coronary artery-related infarction (52).

Coronary artery imaging with CMR. Imaging of the coronary arteries using CMR is limited primarily by difficulties with cardiac motion. The current spatial resolution is not as high as for CT, and acquisition planes must be prescribed in advance and take considerable time using current protocols. Predominantly, CMR is used to exclude anomalous origin of vessels and to look for the consequences of coronary artery stenosis and thrombosis, that is, perfusion defects or infarction. A substantial body of work has built up over the past decade demonstrating the ability of MRI to characterize plaque components (53). Although there may be future improvements in coronary artery imaging, particularly aiming to use the potential of CMR for characterization of plaque, this is not currently a clinical application. In contrast, for vein grafts, CMR may be closer to clinical application. Studies have shown both spin echo (54,55) and gradient echo (56,57) sequences to achieve approximately 90% accuracy in predicting graft patency. Flow measurements by velocity-encoded sequences may also contribute to the identification of diseased vein grafts (58).

Noncoronary Interventions and the Role of CMR

Structural heart disease. Interventional cardiologists are increasingly involved in the percutaneous treatment of structural heart disease. The precise assessment of anatomy, function, and flow by CMR has given it a key role in the management of these patients. The lack of radiation and reduction in need for invasive procedures makes CMR ideal for serial studies that are often required in patients with complex abnormalities. Cardiac magnetic resonance is valuable in the assessment of atrial septal defects and ventricular septal defects assisting assessment for suitability of percutaneous closure (36,59–61). Measurement of shunt volumes by MR phase-contrast techniques have been shown to be accurate when compared with invasively measured $Q_p:Q_s$ ratios (62). Furthermore, a recent study by Teo et al. (63)

has shown excellent agreement between CMR and transesophageal echocardiography for estimation of maximum defect size ($R = 0.87$), with CMR also being successful in assessing the margins of the defect. Compared with echocardiography, CMR allows more accurate imaging of the right side of the heart and has been applied reliably to detect improvements in right ventricular function and volume following percutaneous atrial septal defect closure (64). In addition, the ability of CMR to evaluate associated anomalous pulmonary venous return is superior to transthoracic echocardiography (65). CMR is invaluable in assessing complex anatomy in conditions such as tetralogy of Fallot, and its superiority to echocardiography in quantifying ventricular volumes greatly assists decisions about timing and strategy in surgery or interventional procedure.

CMR for real-time guiding of cardiac interventions. In addition to pre-operative assessment of structural heart disease, CMR has the potential to guide percutaneous interventional procedures in real time. Hybrid laboratories have been developed, and the feasibility of such an approach has been shown in animal models (66–68), as well as humans with congenital heart disease (68) and aortic coarctation (69). Operators use nonmetallic guidewires and fill catheters with diluted solutions of iron oxide particles to allow for tracking by CMR. Reduction or elimination of exposure to ionizing radiation is a major advantage of such an approach, as well as improved 3D anatomical information and physiological data to guide the procedures. However, many practical and safety issues remain before such an approach becomes mainstream, and real-time MR image acquisition, reconstruction, and display must improve (70–72).

Cardiac electrophysiology procedures. Radiofrequency catheter ablation has advanced to be the first line treatment of many cardiac arrhythmias. These include tachycardias, resulting from atrioventricular reentrant tachycardia accessory pathway, and atrial flutter (73). Clinical indications have now expanded to include arrhythmias such as atrial fibrillation (74) and scar-related ventricular tachycardia (75). X-ray fluoroscopy has been invaluable in placement of catheters for these procedures, but is limited by poor 3D visualization and inability to visualize soft tissue anatomy. One approach to improve targeting of catheters to particular anatomical sites is the use of electrospatial mapping systems.

The most frequent use of CMR in the electrophysiology field is in the pre-procedure planning of pulmonary vein isolation for atrial fibrillation. Although CT is most commonly used for acquiring the 3D data to be integrated into the mapping systems, CMR with 3D angiography offers an excellent alternative with the advantage of no radiation exposure to the patient (76,77). An example of a 3D CMR pulmonary venogram and its use for pulmonary vein isolation is illustrated in Figure 7. The ability of CMR to visualize ablation scar may also assist electrophysiologists in the future in the assessment of failed ablations. The use of CMR for



Figure 7. Contrast-Enhanced 3D MR Left Atrial and Pulmonary Venogram and CARTO Map

(A) Contrast-enhanced 3-dimensional (3D) magnetic resonance (MR) left atrial and pulmonary venogram for radiation-free imaging and integration to pulmonary vein mapping software (CARTO navigation system, Biosense Webster, Inc., Diamond Bar, California) before pulmonary vein isolation for atrial fibrillation. (B) A representative CARTO Map is provided demonstrating foci of radiofrequency lesions (red dots). The CMR images were acquired using a GE 1.5-T HDxt (GE Healthcare). Abbreviations as in Figure 1.

assisting ablation of monomorphic ventricular tachycardia is in the investigational stages but shows promise, as reviewed by Koldaivelu et al. (78). The possible use of real-time CMR to guide electrophysiology procedures using nonferromagnetic catheters is also being explored with many obvious practical issues to be overcome to reach mainstream (79,80).

Research Applications of CMR in Interventional Cardiology

CMR in clinical studies. Cardiac magnetic resonance has played a key role in clinical studies of novel therapeutic techniques applied in the cardiac catheter laboratory. Its precise and reproducible measurement of LV volumes and function makes it the ultimate tool for assessing the impact of new approaches in the catheter laboratories and their impact on ventricular remodeling, resulting in a substantial reduction in the numbers of subjects required to achieve appropriate statistical power compared with studies using echocardiography of up to one-tenth (81). The precise quantification of infarct size with LGE imaging is also a large advance. However, investigators still face difficulties in assessing variable myocardial territory at risk depending on the location of the coronary artery occlusion. The ability of CMR to quantify infarct size as a proportion of the area at risk, and thus calculate the myocardium salvage index, using T₂-weighted and late enhancement sequences, dramatically increases the power of studies examining the impact of interventions designed to limit

infarct size. Recently, the salvage index has been shown to predict the clinical outcome in patients with MI (82). The ability of CMR to characterize hemorrhage (19) and microvascular obstruction (19,30,31) within the myocardium adds an additional dimension for clinical trials with demonstrated clinical relevance.

Molecular imaging. The recent design of molecular contrast agents that tag antibodies or specific proteins with iron oxide or gadolinium chelate-, micro-, or nanoparticles has opened the door for MRI to be used for molecular imaging. This approach has been demonstrated in several animal models of cardiovascular disease, for example in targeting vascular cellular adhesion molecule in models of cerebral ischemia-reperfusion (83) and glycoprotein IIb/IIIa expressed by activated platelets (84). The ability to perform in vivo quantitation of expression of molecules important in the pathogenesis of cardiovascular disease will be a crucial advance in the assessment of novel pharmacotherapies. The inevitable but still-distant transition of molecular CMR into the clinical arena may allow for early detection of cardiovascular disease and improved identification of vulnerable plaque.

Diffusion tensor imaging and the heart. The structural organization of myocytes, particularly their “array” or “disarray,” is a key determinant of mechanical and electrical properties of the heart. Diffusion tensor imaging is an MRI technique that enables the measurement of the restricted diffusion of water in tissue and has recently emerged in the

field of neuroimaging as an excellent tool to perform tractography of white matter. Its application to the heart has enormous potential particularly in understanding the impact of myocardial ischemia and infarction and remodeling of the ventricle after such insults (85).

Limitations of CMR

Safety and practical issues faced when performing CMR. Despite the benefits of CMR, some practical problems are commonly faced. Claustrophobia may result in patients not tolerating the scan, a problem in approximately 2% of patients (86). Inability to breath hold or hear breathing instructions also create problems during the acquisition phase. Another major limitation for the application of MRI to cardiac imaging is the presence of a pacemaker or implantable cardiac defibrillator. An illustration of the important diagnostic role attributed to MRI can be observed in the efforts being invested in the development of implantable devices to be MR compatible. Coronary artery stents have little to no ferromagnetic material and are generally considered safe. Magnetic resonance imaging safety information has been obtained for many of the bare-metal and drug-eluting coronary stents, which have been reported to be safe for patients undergoing MRI procedures at 3-T or less. Specific information can be obtained from the manufacturers or on online data sites (87). Many prosthetic valves are MR safe, but do cause some artifacts. Similarly, stainless steel sternal wires can induce susceptibility artifacts particularly affecting images of the right ventricular free wall.

Perfusion and LGE sequences depend on the ability to administer gadolinium to the patient, a decision that is predominantly influenced by renal function. The rare, but serious side effect of nephrogenic systemic fibrosis involves fibrosis of the skin, connective tissue, joints, lung, liver, muscles, and heart and develops rapidly over days to weeks (88). It has only been observed in patients with renal dysfunction, specifically with glomerular filtration rate <30 ml/min/1.73 m². Thus, in most institutions individuals with a glomerular filtration rate <50 ml/min/1.73 m² are not administered gadolinium unless there is an extreme indication with no alternative diagnostic approach.

Perceived cost and accessibility issues of CMR. The perceived cost and inaccessibility of CMR is important to consider. Imaging tests for cardiovascular diseases contribute a large proportion of health care costs in the developed world. Although an argument is often made that "simple" tests such as an exercise stress test are underused, careful consideration needs to be made about the costs of unnecessary further investigations may occur because of high false-positive rates, as well as the inherent inability of exercise stress tests to detect vulnerable plaque that may have caused unstable angina or a small MI, but does not itself reach hemodynamic significance in regard to its obstruction of coronary blood flow during increased de-

mand. Given the high diagnostic accuracy of CMR, particularly in the assessment of patients with chest pain and suspected CAD, as well as its ability to assess function, perfusion, and previous infarction in 1 study (61), it has the potential to decrease overall test use, increase early diagnosis, decrease hospitalization and length of stay, and reduce invasive procedure use.

Conclusions

CMR is emerging as an indispensable tool to the interventional cardiologist. The high spatial and temporal resolution images examining myocardial function, perfusion, and infarction assists in diagnosis and design of appropriate interventional strategies. Improved understanding by interventionalists of the full potential of CMR will help to minimize the use of work-up involving radiation and invasive procedures and will lead to a more targeted approach in the catheter laboratory.

Acknowledgments

The authors would like to thank the MR radiographers and staff of North Shore Radiology.

Reprint requests and correspondence: Dr. Ravinay Bhindi, North Shore Heart Research Group, Kolling Institute (University of Sydney), Royal North Shore Hospital, St. Leonards, New South Wales 2065, Australia. E-mail: rbhindi@med.usyd.edu.au.

REFERENCES

1. Francone M, Dymarkowski S, Kalantzi M, Bogaert J. Real-time cine MRI of ventricular septal motion: a novel approach to assess ventricular coupling. *J Magn Reson Imaging* 2005;21:305-9.
2. Francone M, Dymarkowski S, Kalantzi M, Rademakers FE, Bogaert J. Assessment of ventricular coupling with real-time cine MRI and its value to differentiate constrictive pericarditis from restrictive cardiomyopathy. *Eur Radiol* 2006;16:944-51.
3. Botnar RM, Stuber M, Danias PG, Kissinger KV, Manning WJ. Improved coronary artery definition with T2-weighted, free-breathing, three-dimensional coronary MRA. *Circulation* 1999;99:3139-48.
4. Longmore DB, Klipstein RH, Underwood SR, et al. Dimensional accuracy of magnetic resonance in studies of the heart. *Lancet* 1985;1:1360-2.
5. Rehr RB, Malloy CR, Filipchuk NG, Peshock RM. Left ventricular volumes measured by MR imaging. *Radiology* 1985;156:717-9.
6. Underwood SR, Rees RS, Savage PE, et al. Assessment of regional left ventricular function by magnetic resonance. *Br Heart J* 1986;56:334-40.
7. Peshock RM, Rokey R, Malloy GM, et al. Assessment of myocardial systolic wall thickening using nuclear magnetic resonance imaging. *J Am Coll Cardiol* 1989;14:653-9.
8. Azhari H, Sideman S, Weiss JL, et al. Three-dimensional mapping of acute ischemic regions using MRI: wall thickening versus motion analysis. *Am J Physiol* 1990;259:H1492-503.
9. Jennings RB, Murry CE, Steenbergen C Jr., Reimer KA. Development of cell injury in sustained acute ischemia. *Circulation* 1990;82 Suppl 3:II2-12.
10. Reimer KA, Jennings RB. The "wavefront phenomenon" of myocardial ischemic cell death. II. Transmural progression of necrosis within the framework of ischemic bed size (myocardium at risk) and collateral flow. *Lab Invest* 1979;40:633-44.

11. Aletras AH, Tilak GS, Natanzon A, et al. Retrospective determination of the area at risk for reperfused acute myocardial infarction with T2-weighted cardiac magnetic resonance imaging: histopathological and displacement encoding with stimulated echoes (DENSE) functional validations. *Circulation* 2006;113:1865-70.
12. Abdel-Aty H, Zagrosek A, Schulz-Menger J, et al. Delayed enhancement and T2-weighted cardiovascular magnetic resonance imaging differentiate acute from chronic myocardial infarction. *Circulation* 2004;109:2411-6.
13. Abdel-Aty H, Cocker M, Meek C, Tyberg JV, Friedrich MG. Edema as a very early marker for acute myocardial ischemia: a cardiovascular magnetic resonance study. *J Am Coll Cardiol* 2009;53:1194-201.
14. Friedrich MG, Abdel-Aty H, Taylor A, Schulz-Menger J, Messroghli D, Dietz R. The salvaged area at risk in reperfused acute myocardial infarction as visualized by cardiovascular magnetic resonance. *J Am Coll Cardiol* 2008;51:1581-7.
15. O'Regan DP, Ahmed R, Neuwirth C, et al. Cardiac MRI of myocardial salvage at the peri-infarct border zones after primary coronary intervention. *Am J Physiol Heart Circ Physiol* 2009;297:H340-6.
16. Wright J, Adriaenssens T, Dymarkowski S, Desmet W, Bogaert J. Quantification of myocardial area at risk with T2-weighted CMR: comparison with contrast-enhanced CMR and coronary angiography. *J Am Coll Cardiol Img* 2009;2:825-31.
17. Garcia-Dorado D, Oliveras J, Gili J, et al. Analysis of myocardial oedema by magnetic resonance imaging early after coronary artery occlusion with or without reperfusion. *Cardiovasc Res* 1993;27:1462-9.
18. Carlsson M, Ubachs JF, Hedstrom E, Heiberg E, Jovinge S, Arheden H. Myocardium at risk after acute infarction in humans on cardiac magnetic resonance: quantitative assessment during follow-up and validation with single-photon emission computed tomography. *J Am Coll Cardiol Img* 2009;2:569-76.
19. Ganame J, Messalli G, Dymarkowski S, et al. Impact of myocardial haemorrhage on left ventricular function and remodelling in patients with reperfused acute myocardial infarction. *Eur Heart J* 2009;30:1440-9.
20. Kim RJ, Fieno DS, Parrish TB, et al. Relationship of MRI delayed contrast enhancement to irreversible injury, infarct age, and contractile function. *Circulation* 1999;100:1992-2002.
21. Choi KM, Kim RJ, Gubernikoff G, Vargas JD, Parker M, Judd RM. Transmural extent of acute myocardial infarction predicts long-term improvement in contractile function. *Circulation* 2001;104:1101-7.
22. Wu E, Judd RM, Vargas JD, Klocke FJ, Bonow RO, Kim RJ. Visualisation of presence, location, and transmural extent of healed Q-wave and non-Q-wave myocardial infarction. *Lancet* 2001;357:21-8.
23. Klein C, Nekolla SG, Bengel FM, et al. Assessment of myocardial viability with contrast-enhanced magnetic resonance imaging: comparison with positron emission tomography. *Circulation* 2002;105:162-7.
24. Ibrahim T, Nekolla SG, Hornke M, et al. Quantitative measurement of infarct size by contrast-enhanced magnetic resonance imaging early after acute myocardial infarction: comparison with single-photon emission tomography using Tc99m-sestamibi. *J Am Coll Cardiol* 2005;45:544-52.
25. Ibrahim T, Bulow HP, Hackl T, et al. Diagnostic value of contrast-enhanced magnetic resonance imaging and single-photon emission computed tomography for detection of myocardial necrosis early after acute myocardial infarction. *J Am Coll Cardiol* 2007;49:208-16.
26. Wagner A, Mahrholdt H, Holly TA, et al. Contrast-enhanced MRI and routine single photon emission computed tomography (SPECT) perfusion imaging for detection of subendocardial myocardial infarcts: an imaging study. *Lancet* 2003;361:374-9.
27. Mahrholdt H, Wagner A, Parker M, et al. Relationship of contractile function to transmural extent of infarction in patients with chronic coronary artery disease. *J Am Coll Cardiol* 2003;42:505-12.
28. Selvanayagam JB, Kardos A, Francis JM, et al. Value of delayed-enhancement cardiovascular magnetic resonance imaging in predicting myocardial viability after surgical revascularization. *Circulation* 2004;110:1535-41.
29. Kim RJ, Wu E, Rafael A, et al. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med* 2000;343:1445-53.
30. Wu KC, Kim RJ, Bluemke DA, et al. Quantification and time course of microvascular obstruction by contrast-enhanced echocardiography and magnetic resonance imaging following acute myocardial infarction and reperfusion. *J Am Coll Cardiol* 1998;32:1756-64.
31. Hombach V, Grebe O, Merkle N, et al. Sequelae of acute myocardial infarction regarding cardiac structure and function and their prognostic significance as assessed by magnetic resonance imaging. *Eur Heart J* 2005;26:549-57.
32. Kim HW, Klem I, Shah DJ, et al. Unrecognized non-Q-wave myocardial infarction: prevalence and prognostic significance in patients with suspected coronary disease. *PLoS Med* 2009;6:e1000057.
33. Kwong RY, Chan AK, Brown KA, et al. Impact of unrecognized myocardial scar detected by cardiac magnetic resonance imaging on event-free survival in patients presenting with signs or symptoms of coronary artery disease. *Circulation* 2006;113:2733-43.
34. Barbier CE, Bjerner T, Johansson L, Lind L, Ahlstrom H. Myocardial scars more frequent than expected: magnetic resonance imaging detects potential risk group. *J Am Coll Cardiol* 2006;48:765-71.
35. Harrity P, Patel A, Bianco J, Subramanian R. Improved diagnosis and characterization of postinfarction left ventricular pseudoaneurysm by cardiac magnetic resonance imaging. *Clin Cardiol* 1991;14:603-6.
36. Sechtem U, Pflugfelder P, Cassidy MC, Holt W, Wolfe C, Higgins CB. Ventricular septal defect: visualization of shunt flow and determination of shunt size by cine MR imaging. *AJR Am J Roentgenol* 1987;149:689-92.
37. Garcia-Fuster R, Rodriguez I, Estornell J, Martinez-Leon J. A combined approach for ischemic mitral valve regurgitation: scar plication and the role of magnetic resonance imaging. *J Thorac Cardiovasc Surg* 2008;135:1169-72.
38. Mollet NR, Dymarkowski S, Volders W, et al. Visualization of ventricular thrombi with contrast-enhanced magnetic resonance imaging in patients with ischemic heart disease. *Circulation* 2002;106:2873-6.
39. Sechtem U, Theissen P, Heindel W, et al. Diagnosis of left ventricular thrombi by magnetic resonance imaging and comparison with angiocardiology, computed tomography and echocardiography. *Am J Cardiol* 1989;64:1195-9.
40. Friedrich MG, Strohm O, Schulz-Menger J, Marciniak H, Luft FC, Dietz R. Contrast media-enhanced magnetic resonance imaging visualizes myocardial changes in the course of viral myocarditis. *Circulation* 1998;97:1802-9.
41. Mahrholdt H, Goedecke C, Wagner A, et al. Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. *Circulation* 2004;109:1250-8.
42. Eitel I, Lucke C, Grothoff M, et al. Inflammation in takotsubo cardiomyopathy: insights from cardiovascular magnetic resonance imaging. *Eur Radiol* 2010;20:422-31.
43. Allman KC, Shaw LJ, Hachamovitch R, Udelson JE. Myocardial viability testing and impact of revascularization on prognosis in patients with coronary artery disease and left ventricular dysfunction: a meta-analysis. *J Am Coll Cardiol* 2002;39:1151-8.
44. Schwitler J, Wacker CM, van Rossum AC, et al. MR-IMPACT: comparison of perfusion-cardiac magnetic resonance with single-photon emission computed tomography for the detection of coronary artery disease in a multicentre, multivendor, randomized trial. *Eur Heart J* 2008;29:480-9.
45. Sakuma H, Suzawa N, Ichikawa Y, et al. Diagnostic accuracy of stress first-pass contrast-enhanced myocardial perfusion MRI compared with stress myocardial perfusion scintigraphy. *AJR Am J Roentgenol* 2005;185:95-102.
46. Klem I, Heitner JF, Shah DJ, et al. Improved detection of coronary artery disease by stress perfusion cardiovascular magnetic resonance with the use of delayed enhancement infarction imaging. *J Am Coll Cardiol* 2006;47:1630-8.
47. Jahnke C, Nagel E, Gebker R, et al. Prognostic value of cardiac magnetic resonance stress tests: adenosine stress perfusion and dobutamine stress wall motion imaging. *Circulation* 2007;115:1769-76.
48. Nagel E, Lehmkuhl HB, Bocksch W, et al. Noninvasive diagnosis of ischemia-induced wall motion abnormalities with the use of high-dose dobutamine stress MRI: comparison with dobutamine stress echocardiography. *Circulation* 1999;99:763-70.

49. Hundley WG, Morgan TM, Neagle CM, Hamilton CA, Rerkpatanapipat P, Link KM. Magnetic resonance imaging determination of cardiac prognosis. *Circulation* 2002;106:2328-33.
50. Watkins S, McGeoch R, Lyne J, et al. Validation of magnetic resonance myocardial perfusion imaging with fractional flow reserve for the detection of significant coronary heart disease. *Circulation* 2009;120:2207-13.
51. Patel MR, Peterson ED, Dai D, et al. Low diagnostic yield of elective coronary angiography. *N Engl J Med* 2010;362:886-95.
52. McCrohon JA, Moon JC, Prasad SK, et al. Differentiation of heart failure related to dilated cardiomyopathy and coronary artery disease using gadolinium-enhanced cardiovascular magnetic resonance. *Circulation* 2003;108:54-9.
53. Saam T, Hatsukami TS, Takaya N, et al. The vulnerable, or high-risk, atherosclerotic plaque: noninvasive MR imaging for characterization and assessment. *Radiology* 2007;244:64-77.
54. Gomes AS, Lois JF, Drinkwater DC Jr., Corday SR. Coronary artery bypass grafts: visualization with MR imaging. *Radiology* 1987;162:175-9.
55. Jenkins JP, Love HG, Foster CJ, Isherwood I, Rowlands DJ. Detection of coronary artery bypass graft patency as assessed by magnetic resonance imaging. *Br J Radiol* 1988;61:2-4.
56. White RD, Caputo GR, Mark AS, Modin GW, Higgins CB. Coronary artery bypass graft patency: noninvasive evaluation with MR imaging. *Radiology* 1987;164:681-6.
57. Hoogendoorn LI, Pattynama PM, Buis B, van der Geest RJ, van der Wall EE, de Roos A. Noninvasive evaluation of aortocoronary bypass grafts with magnetic resonance flow mapping. *Am J Cardiol* 1995;75:845-8.
58. Langerak SE, Vliegen HW, Jukema JW, et al. Value of magnetic resonance imaging for the noninvasive detection of stenosis in coronary artery bypass grafts and recipient coronary arteries. *Circulation* 2003;107:1502-8.
59. Didier D, Higgins CB. Identification and localization of ventricular septal defect by gated magnetic resonance imaging. *Am J Cardiol* 1986;57:1363-8.
60. Hundley WG, Li HF, Lange RA, et al. Assessment of left-to-right intracardiac shunting by velocity-encoded, phase-difference magnetic resonance imaging. A comparison with oximetric and indicator dilution techniques. *Circulation* 1995;91:2955-60.
61. Pennell DJ, Sechtem UP, Higgins CB, et al. Clinical indications for cardiovascular magnetic resonance (CMR): Consensus Panel report. *J Cardiovasc Magn Reson* 2004;6:727-65.
62. Petersen SE, Voigtlander T, Kreitner KF, et al. Quantification of shunt volumes in congenital heart diseases using a breath-hold MR phase contrast technique-comparison with oximetry. *Int J Cardiovasc Imaging* 2002;18:53-60.
63. Teo KS, Disney PJ, Dundon BK, et al. Assessment of atrial septal defects in adults comparing cardiovascular magnetic resonance with transthoracic echocardiography. *J Cardiovasc Magn Reson* 2010;12:44.
64. Schoen SP, Kittner T, Bohl S, et al. Transcatheter closure of atrial septal defects improves right ventricular volume, mass, function, pulmonary pressure, and functional class: a magnetic resonance imaging study. *Heart* 2006;92:821-6.
65. Greil GF, Powell AJ, Gildein HP, Geva T. Gadolinium-enhanced three-dimensional magnetic resonance angiography of pulmonary and systemic venous anomalies. *J Am Coll Cardiol* 2002;39:335-41.
66. Lederman RJ, Guttman MA, Peters DC, et al. Catheter-based endomyocardial injection with real-time magnetic resonance imaging. *Circulation* 2002;105:1282-4.
67. Arepally A, Karmarkar PV, Weiss C, Rodriguez ER, Lederman RJ, Atalar E. Magnetic resonance image-guided trans-septal puncture in a swine heart. *J Magn Reson Imaging* 2005;21:463-7.
68. Raval AN, Telep JD, Guttman MA, et al. Real-time magnetic resonance imaging-guided stenting of aortic coarctation with commercially available catheter devices in Swine. *Circulation* 2005;112:699-706.
69. Krueger JJ, Ewert P, Yilmaz S, et al. Magnetic resonance imaging-guided balloon angioplasty of coarctation of the aorta: a pilot study. *Circulation* 2006;113:1093-100.
70. Geva T, Marshall AC. Magnetic resonance imaging-guided catheter interventions in congenital heart disease. *Circulation* 2006;113:1051-2.
71. McVeigh ER, Guttman MA, Kellman P, Raval AN, Lederman RJ. Real-time, interactive MRI for cardiovascular interventions. *Acad Radiol* 2005;12:1121-7.
72. Muthurangu V, Razavi RS. The value of magnetic resonance guided cardiac catheterisation. *Heart* 2005;91:995-6.
73. Calkins H, Yong P, Miller JM, et al., for the Atakr Multicenter Investigators Group. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction: final results of a prospective, multicenter clinical trial. *Circulation* 1999;99:262-70.
74. Pappone C, Rosanio S, Oreto G, et al. Circumferential radiofrequency ablation of pulmonary vein ostia: a new anatomic approach for curing atrial fibrillation. *Circulation* 2000;102:2619-28.
75. Marchlinski FE, Callans DJ, Gottlieb CD, Zado E. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. *Circulation* 2000;101:1288-96.
76. Kato R, Lickfelt L, Meininger G, et al. Pulmonary vein anatomy in patients undergoing catheter ablation of atrial fibrillation: lessons learned by use of magnetic resonance imaging. *Circulation* 2003;107:2004-10.
77. Mansour M, Refaat M, Heist EK, et al. Three-dimensional anatomy of the left atrium by magnetic resonance angiography: implications for catheter ablation for atrial fibrillation. *J Cardiovasc Electrophysiol* 2006;17:719-23.
78. Kolandaivelu A, Lardo AC, Halperin HR. Cardiovascular magnetic resonance guided electrophysiology studies. *J Cardiovasc Magn Reson* 2009;11:21.
79. Nazarian S, Kolandaivelu A, Zviman MM, et al. Feasibility of real-time magnetic resonance imaging for catheter guidance in electrophysiology studies. *Circulation* 2008;118:223-9.
80. Lardo AC, McVeigh ER, Jumrussirikul P, et al. Visualization and temporal/spatial characterization of cardiac radiofrequency ablation lesions using magnetic resonance imaging. *Circulation* 2000;102:698-705.
81. Grothues F, Smith GC, Moon JC, et al. Comparison of interstudy reproducibility of cardiovascular magnetic resonance with two-dimensional echocardiography in normal subjects and in patients with heart failure or left ventricular hypertrophy. *Am J Cardiol* 2002;90:29-34.
82. Eitel I, Desch S, Fuernau G, et al. Prognostic significance and determinants of myocardial salvage assessed by cardiovascular magnetic resonance in acute reperfused myocardial infarction. *J Am Coll Cardiol* 2010;55:2470-9.
83. McAteer MA, Sibson NR, von Zur Muhlen C, et al. In vivo magnetic resonance imaging of acute brain inflammation using microparticles of iron oxide. *Nat Med* 2007;13:1253-8.
84. von Zur Muhlen C, von Elverfeldt D, Moeller JA, et al. Magnetic resonance imaging contrast agent targeted toward activated platelets allows in vivo detection of thrombosis and monitoring of thrombolysis. *Circulation* 2008;118:258-67.
85. Sosnovik DE, Wang R, Dai G, Reese TG, Wedeen VJ. Diffusion MR tractography of the heart. *J Cardiovasc Magn Reson* 2009;11:47.
86. Francis JM, Pennell DJ. Treatment of claustrophobia for cardiovascular magnetic resonance: use and effectiveness of mild sedation. *J Cardiovasc Magn Reson* 2000;2:139-41.
87. Shellock FG. MRI Safety. Available at: http://www.mrisafety.com/safety_article.asp?subject=184. Accessed January 17, 2011.
88. Kribben A, Witzke O, Hillen U, Barkhausen J, Daul AE, Erbel R. Nephrogenic systemic fibrosis: pathogenesis, diagnosis, and therapy. *J Am Coll Cardiol* 2009;53:1621-8.

Key Words: cardiac magnetic resonance ■ coronary artery disease ■ ischemia ■ myocardial viability.