Evolving Clinical Application of Cardiac MRI

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Over the past decade, cardiac magnetic resonance imaging (MRI) has emerged as a new technology representing the next major advance in noninvasive cardiac imaging. It provides unique and accurate data representative of cardiac structure, function, and perfusion at both the gross anatomical and myocardial levels. Cardiac MRI proves to be highly accurate and reproducible in many challenging areas in clinical cardiology, including diagnosis of constrictive pericarditis, differentiation of ischemic from dilated cardiomyopathy, confirmation of the diagnosis of myocarditis, and definition and quantification of myocardial viability. As compelling studies support its clinical utility, the evolution of cardiac MRI is gaining speed. In many cases, such as the diagnosis of anomalous origin of the coronary arteries, it is the gold standard diagnostic technique.
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Over the past 25 years, the development of noninvasive imaging techniques has transformed the clinical evaluation of patients with known or suspected heart disease. In the not-too-distant past, physicians relied primarily upon the stethoscope, the electrocardiogram (ECG), and the chest x-ray to detect cardiac structural and functional abnormalities and ischemia. The development of echocardiography, and, to a lesser extent, nuclear imaging techniques, has dramatically improved the ease and accuracy of noninvasive diagnosis for patients with congenital, valvular, myocardial, and ischemic abnormalities.

Cardiac magnetic resonance imaging (MRI) represents the next major advance in noninvasive cardiac imaging. It consists of a series of techniques and applications that are still evolving and are not yet universally available. Nevertheless, it has become clear that MRI adds a new dimension to cardiac diagnosis and provides data regarding cardiac structure, function, and perfusion that are, in many cases, superior to data from all other available techniques—invasive as well as noninvasive. Indeed, for many of the current clinical indications for imaging, cardiac MRI is the gold standard. The ability to achieve high fidelity spatial and temporal resolution imaging is accomplished without radiopharmaceuticals, intra-arterial injections, x-ray radiation, or an obligate need for exogenous contrast administration.

This article will review the basic concepts and clinical application of cardiac MRI, with a special focus on common clinical dilemmas. The focus is on enabling the practicing physician to understand the utility of cardiac MRI. Emphasis has been placed upon evaluation of common clinical disorders in which available MRI applications are particularly useful.

MRI Basics

Physics

Each atom in the human body is composed of a nucleus surrounded by rotating negatively charged electrons. The nucleus itself is composed of neutral particles (neutrons) and positively charged particles (protons). The latter rotate or spin (formally denoted as *precess*) within the nucleus, creating an electrical current perpendicular to which a magnetic field or dipole is generated.

Protons, specifically those protons present within hydrogen atoms contained in water (H_2O) molecules, are the principal targets of MRI. Since these hydrogen particles are always spinning, the human body is composed of billions of magnetic dipoles. Because these dipoles are arranged and aligned randomly, the net signal emitted from the body is zero. MRI works by utilizing powerful magnets to realign those dipoles so that their net signal is no longer zero and is detectable by special sensors.

When exposed to a magnetic field, proton dipoles will be realigned so that their direction points either toward or away from the magnetic field. Intermediate alignment is not an option. A slightly larger number of dipoles will be directed toward rather than away from the magnetic field. The net signal so generated is detectable by a sensor located in the MRI machine and transformed through sophisticated computerized and mathematical techniques into images.

MRI Techniques

The MRI techniques briefly discussed below are available in all advanced MRI machines with cardiac gating and are the mainstays of clinical cardiac MRI. (For a more complete description of the physics and data processing involved in the techniques, the reader is referred to Bushong SC[1] and Higgins CB and de Roos A.[2])

Spin Echo Imaging. In this technique, also referred to as *black blood imaging*, the tissue structure of the heart and vascular system is depicted as bright, and that of the blood pool is depicted as dark. Spin echo imaging is used to produce a still image. It is used mainly to define anatomy and structure and depict abnormalities, including myocardial masses, left ventricular (LV) hypertrophy, congenital abnormalities, zones of myocardial infarction, and infiltrative diseases of the myocardium.

White Blood Gradient Echo Imaging. In this technique, also known as *white blood imaging*, cine MRI is generated, displaying moving images of the heart in which cardiac tissues appear dark and the blood pool appears white via endogenous contrast due to either moving blood flow. This technique is used predominantly to define *functional* changes involving the right and left ventricles, including wall motion abnormalities and changes in blood flow patterns accompanying valvular and shunt lesions. More recently, improved techniques that seek to combine attributes of both spin echo and gradient echo, particularly exploiting the intrinsic properties of the underlying substrate, yield even greater cine imaging of the heart.

Phase Velocity Mapping. This technique is used to measure flow directly and thus to quantify degrees of valvular stenosis and regurgitation, estimate shunt size, and assess the severity of arterial obstruction while providing directional components to flow.[3]

Gadolinium Enhancement. Gadolinium chelate is the exogenous contrast agent of clinical MRI; its use entails no exposure to radiation and the agent is non-nephrotoxic.[4] Due to its high molecular weight and lipophobicity, it cannot enter living myocyte; however, it can be uptaken by those that are necrotic or whose cell membranes were damaged during the process of acute infarction. Gadolinium enhancement is therefore seen in the territories of acute and chronic infarction, allowing viable myocardial segments to be clearly distinguished from infarcted areas.[5] Gadolinium also accumulates in the large interstitial spaces within the collagen matrix of fibrotic tissue. Therefore, gadolinium enhancement

will also be seen in areas of scar formation secondary to remote myocardial infarction (MI) and interstitial fibrosis, such as occurs in patients with dilated or hypertrophic cardiomyopathy.

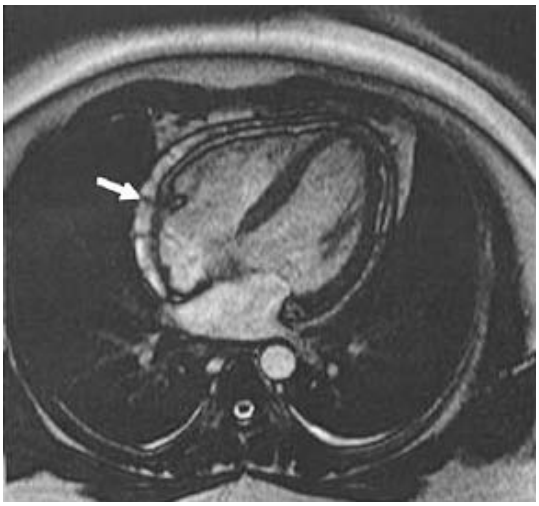


Figure 1. *Prominent pericardial thickening is indicated by the arrow. Reprinted from: Axel L. Assessment of pericardial disease by magnetic resonance and computed tomography.* J Magn Reson Imaging. *2004;19(6): 816-826.*[43] *Copyright 2004 John Wiley & Sons, Inc. Reprinted with permission of Wiley-Liss, Inc., a subsidiary of John Wiley & Sons, Inc.* www.medreviews.com

Clinical Applications

Constrictive Pericarditis

From a diagnostic perspective, constrictive pericarditis has always been among the most challenging entities in clinical cardiology. Patients classically present with fatigue, dyspnea, and variable signs of elevated systemic venous pressure—a clinical picture that closely mimics congestive heart failure. It may be particularly difficult to separate this syndrome from heart failure resulting from restrictive cardiomyopathy, with which it shares the functional abnormality of severe diastolic dysfunction. Complicated algorithms using subtle differences in physical examination, echocardiography, and hemodynamic characterization have been devised to differentiate these 2 conditions but, in practice, there is a great deal of overlap and diagnostic uncertainty. It is extremely important to distinguish these 2 diseases because patients with constrictive pericarditis often benefit from surgical intervention, whereas supportive medical therapy remains the only option for patients with restrictive cardiomyopathy.

Cardiac MRI provides a detailed examination of pericardial structure and precise measurement of pericardial thickness.[6,7] Its accuracy in this regard is markedly superior to that of echocardiography, which has been commonly used to obtain this measurement. In normal individuals, pericardial thickness by MRI is less than 3.5 mm; values in excess of 4 mm are abnormal and suggest fibrous pericarditis (Figure 1). In patients without clinical evidence of abnormal diastolic filling, this finding may be incidental. In patients who exhibit clinical and hemodynamic characteristics compatible with constrictive pericarditis, anatomical verification of a thickened pericardium can be crucial to appropriately direct therapy toward surgical intervention.[8,9]

Cine MRI and phase velocity mapping also furnish additional information useful in the diagnosis of constrictive pericarditis. These data include documentation of the limited expandability of the ventricle during diastole and the predominance of early as opposed to late diastolic filling, physiologic patterns that are characteristic of pericardial constriction.

More recently, the incorporation of radio frequency tissue tagging, in which a saturation grid is placed across the myocardium and pericardium, has been used to discern the adherence pattern between the visceral and parietal pericardium. Failure to demonstrate slippage between layers is pathognomonic of focal constriction; when a large area is affected, it suggests global constrictive pericarditis. This technique has been used by many clinicians to confirm diagnosis in patients with signs and symptoms of constrictive disease and is a radiographic equivalent to the view of the surgeon under direct visualization.

Cardiomyopathy

Cardiac MRI adds significant, clinically relevant information to that provided by echocardiography in patients with dilated, hypertrophic, and infiltrative cardiomyopathies as well as arrhythmogenic right ventricular dysplasia.

Dilated Cardiomyopathy. When confronted by a patient with new-onset heart failure and a reduced LV ejection fraction, clinicians frequently experience great difficulty in arriving at an accurate etiologic diagnosis. Determination of heart failure etiology is important for multiple reasons. Patients with ischemic cardiomyopathy have a worse prognosis than patients with nonischemic cardiomyopathy.[10] They may, in some cases, benefit from coronary revascularization; standard medical treatments, including antiplatelet agents, beta-blockers, angiotensin-converting enzyme inhibitors, and statins, are mandatory. A diagnosis of acute myocarditis raises the possibility of spontaneous recovery. In patients with nonischemic cardiomyopathy,

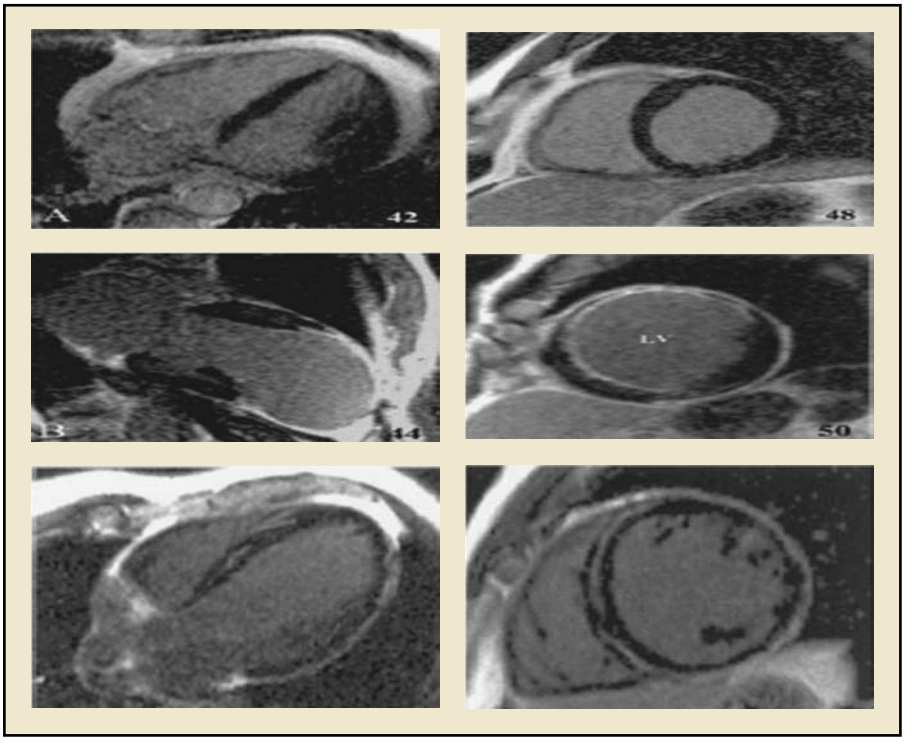
treatable causes (thyrotoxicosis, hemochromatosis, HIV, sarcoidosis, alcoholism, etc) must be identified or excluded. In certain situations in which genetic causes are found, familial screening and reproductive counseling are indicated.[11]

Coronary angiography has been adopted as the first diagnostic test of choice in most cardiac centers in patients with new-onset heart failure. If significant obstructive coronary artery disease (CAD) is found, patients are classified as having ischemic cardiomyopathy; however, use of this strategy frequently leads to an incorrect clinical diagnosis of heart failure etiology. In an autopsy series of 171 deaths occurring during the course of a multicenter heart failure trial,[12] it was determined that of those patients who had received a clinical diagnosis of idiopathic cardiomyopathy, 31% had significant coronary obstructive lesions at autopsy. Conversely, 17% of patients whose heart failure was thought to be ischemic in origin had no significant CAD.

It is becoming increasingly apparent that a diagnosis of ischemic cardiomyopathy based upon angiographic findings is often inaccurate. Patients with some degree of coronary atherosclerosis but no hemodynamically obstructive lesions or history of MI constitute a significant source of the error. Although it may be difficult at first glance to ascribe severe LV dysfunction to angiographically mild atherosclerosis, it is well known that the majority of acute myocardial infarctions are due to rupture of angiographically nonobstructive vulnerable atherosclerotic plaques and that up to one third of MIs are clinically silent. Patients may undergo a sequence of plaque rupture, thrombosis, infarction, and ventricular remodeling leading to dilated cardiomyopathy. At the same time, spontaneous thrombolysis may have removed the evidence that ventricular dysfunction is, in fact, related to CAD.

Recent studies indicate that cardiac MRI can be used to accurately differentiate dilated from ischemic cardiomyopathy based upon the presence and pattern of myocardial gadolinium enhancement. In a study by McCrohon and colleagues,[13] gadolinium enhancement with cardiac MRI was performed in 90 patients with congestive heart failure (CHF) and LV systolic dysfunction. Based upon angiography, 63 patients had previously been classified as having nonischemic dilated cardiomyopathy, whereas 27 patients with significant obstructive CAD had been classified as ischemic. An additional 15 subjects with coronary risk factors and normal coronary angiograms served as controls. None of the control subjects exhibited gadolinium enhancement, whereas all patients classified as ischemic demonstrated gadolinium enhancement indicative of prior transmural or subendocardial MI (Figure 2). Patients with angiographically diagnosed nonischemic dilated cardiomyopathy (n = 63) proved to be most interesting. These patients were classified into 3 subgroups based on the pattern of gadolinium enhancement: no enhancement (59%); subendocardial enhancement indistinguishable from that seen in patients with ischemic cardiomyopathy and previous MI (13%); and patchy or longitudinal striae of midwall enhancement (28%), a finding related

Figure 2. Top: *Two patients with DCM and no late gadolinium enhancement despite dilation and LV systolic dysfunction.* **Middle:** *Patient with heart failure related to CAD. Two-vessel infarction in the territory of the LAD and RCA. Thinning is clearly seen where gadolinium enhancement is nearly transmural, especially in the anterior wall.* **Bottom:** *Patient with DCM with midwall striae of enhancement. Gadolinium enhancement followed the ventricular longitudinal muscle fibers, and involved in particular the septum and basal to mid-LV regions. The pattern is clearly different from patients with heart failure related to CAD. DCM, dilated cardiomyopathy; LV, left ventricular; CAD, coronary artery disease; LAD, left anterior descending artery; RCA, right coronary artery. Reprinted with permission from McCrohon JA et al.*[13] www.medreviews.com



to the presence of interstitial fibrosis due to ventricular remodeling.

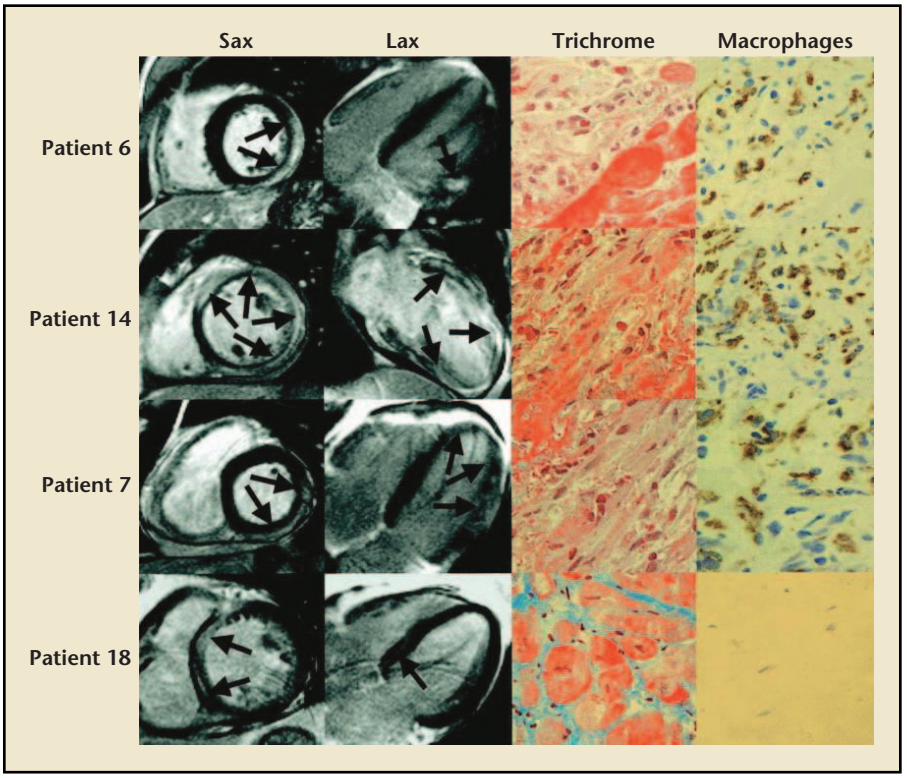
These data indicate that patients with systolic heart failure with no areas of infarct-related gadolinium enhancement have non–CAD-related cardiomyopathy, and angiographic confirmation is not required. The data also suggest that the sensitivity of gadolinium-enhanced (GE) cardiac magnetic resonance (GE CMR) to diagnose CAD-related systolic dysfunction is close to 100%, although pathologic studies will be required to confirm these results. Most importantly, the study points out that coronary angiography not infrequently misclassifies patients with CAD-related cardiomyopathy as nonischemic, due to the lack of angiographic obstructive lesions. By directly imaging areas of old infarction, GE CMR makes a correct diagnosis possible. The current procedural terminology (CPT) code most often used in the past 5 years is that for CHF, and the incorporation of gadolinium imaging to discern the presence or absence of viable myocardium is increasingly becoming the standard for patients presenting with systolic heart failure.

Myocarditis. As mentioned above, documentation of myocarditis in a patient with new-onset heart failure greatly alters the patient's likely prognosis. Unlike most forms of heart failure associated with reduced systolic function, spontaneous recovery occurs in up to 90% of patients with myocarditis.[14] However, in 5% to 10% of cases, patients progress to develop chronic dilated cardiomyopathy and, occasionally, sudden death.[15] In clinical practice, the diagnosis of acute myocarditis is fraught with considerable error because patients often present with a confusing and extremely variable array of signs and symptoms, including those associated with a recent viral illness, as well as nonspecific ECG findings that may include sinus tachycardia, ST-T changes, atrioventricular block, and ventricular tachyarrhythmias.[14] Even myocardial biopsy often fails to provide definitive information because histologic findings are often nonspecific, patchy, and subject to varying interpretation by expert pathologists.[16]

To determine whether cardiac MRI can accurately visualize areas of active myocarditis, Mahrholdt and colleagues[17] performed GE MRI in 32 patients diagnosed with acute myocarditis based upon clinical criteria. Endomyocardial biopsy was then obtained from the myocardial region exhibiting contrast enhancement (when present) and examined by histopathology. As is the case in patients with myocardial infarction, contrast enhancement occurs in the affected areas as the result of gadolinium uptake by damaged or necrotic myocardial cells.

Contrast enhancement was noted in 28 out of 32 patients (88%) (Figure 3), most often in the lateral wall, confirming previous autopsy studies showing a predominance of lateral wall involvement in active myocarditis. Of the 28 patients, biopsy was successfully obtained from the site and demonstrated gadolinium uptake in 21 patients. Nineteen of these patients (91%) showed histopathological evidence of active myocarditis with positive polymerase chain reaction (PCR)

Figure 3. *Results of cardiovascular magnetic resonance and histopathology of typical patients in whom biopsies were obtained from the area of contrast enhancement. The top 3 panels (patients 6, 14, and 7) show patients who have active myocarditis with myocyte damage and infiltration of macrophages. The bottom panel (patient 18) shows a patient without active myocarditis who was diagnosed with hypertropic obstructive cardiomyopathy. Sax, short axis; Lax, long axis. Reprinted with permission from Mahrholdt H et al.*[17] www.medreviews.com



indicative of active viral infection. The other 2 patients had histopathological findings consistent with hypertrophic cardiomyopathy, a condition previously associated with contrast enhancement primarily due to myocardial fibrosis. Histopathology in the 4 patients with no contrast enhancement showed no evidence of active myocarditis and had negative viral titers by PCR. After a follow-up period of 3 months, the area of contrast enhancement decreased from 9% to 3% of LV mass. The reduction was correlated with improvement in the LV ejection fraction from 47% to 60%. This study demonstrates that GE MRI is a valuable tool to identify patients with active myocarditis and to monitor disease progression. Additional data suggest that the extent of myocarditis detected by cardiac MRI is predictive of the eventual ejection fraction at 72 days.[18] In the correct setting, we use this technique to diagnose active myocarditis, sparing the patient invasive and potentially dangerous biopsy.

Determination of Myocardial Viability in Patients With CAD

It is well established that the prognosis of patients with CAD is directly related to the degree of LV dysfunction. As discussed above, the most frequent cause of ventricular dysfunction is myocardial damage secondary to CAD, MI, and subsequent ventricular remodeling. In some patients with CAD, ventricular dysfunction may also result from a process known as *myocardial hibernation*, in which segments of myocardium that are viable do not contract because of severe chronic restriction in myocardial blood flow due to high-grade obstructive coronary lesions. Identification of viable but dysfunctional myocardial segments is extremely important clinically because it indicates a high likelihood that contractility, ventricular function, and prognosis could be improved by coronary revascularization.[19,20] In contrast, absence of these findings implies that revascularization—which carries increased risks in patients with ventricular dysfunction—will not be effective in improving ventricular function or relieving symptoms of heart failure when present.[21,22]

Accurate differentiation of viable from infarcted myocardium has proved extremely challenging in clinical practice. Several diagnostic techniques have been studied extensively. They include myocardial perfusion imaging,[23] stress echocardiography,[24] and positron emission tomography (PET) scanning.[25] Instead of directly measuring or quantifying viable as opposed to infarcted tissue, these modalities rely upon the definition of physiological parameters that viable cells or myocardial segments *should* exhibit. Thus, thallium imaging seeks to define myocardial perfusion on the assumption that only living cells receive blood flow. Dobutamine stress echocardiography (DSE) looks at myocardial contractile reserve based upon the knowledge that catecholamine stimulation will often stimulate hibernating cells or segments to contract. PET scanning images glucose uptake and metabolism because only viable cells utilize metabolic substrates. All these tests are useful but have significant limitations, including the subjectivity and low sensitivity of DSE and the relatively low specificity of both thallium perfusion imaging and PET scanning. These techniques suffer from their indirect assessment of viability rather than direct quantification of viable myocardium.

Alone among available techniques, GE CMR provides the opportunity to image, measure, and quantify the extent of viable myocardium directly and therefore can predict the likelihood of future functional improvement following revascularization.[26] Because gadolinium cannot be uptaken by myocardial cells with intact cell membranes, areas of hyperenhancement on GE CMR represent either nonviable myocardial cells or scar tissue. Instead of looking for a physiological parameter that viable cells are supposed to exhibit, GE CMR determines viability by relating it to myocardial infarction, defining what is alive by knowing what is dead.

The use of GE CMR was evaluated in a study by Choi and colleagues[27] of 24 patients who presented with their first acute MI and were successfully revascularized; patients underwent cine MRI (for functional evaluation) and GE CMR within 7 days (scan 1) of the infarct and a follow-up study at 8 to 12 weeks (scan 2). The transmural extent of the infarct and wall thickening on both scans was defined using a 72-segment model.[27]

In the first group of scans, 1524 out of 1571 segments were dysfunctional. In the second group of scans, the transmural extent of the infarction was inversely related to improvement of the segment function (Figure 4). The best predictor of global improvement in LV function was the extent of dysfunctional but viable myocardium with an infarction extension of less than 25% of the LV wall thickness.

The study demonstrates that in the setting of acute MI, the transmural extent of the infarction defined by GE CMR predicts the improvement in contractile function. GE CMR has significant value in patients with CAD and LV systolic dysfunction; this was demonstrated in a recent study of 50 patients who underwent

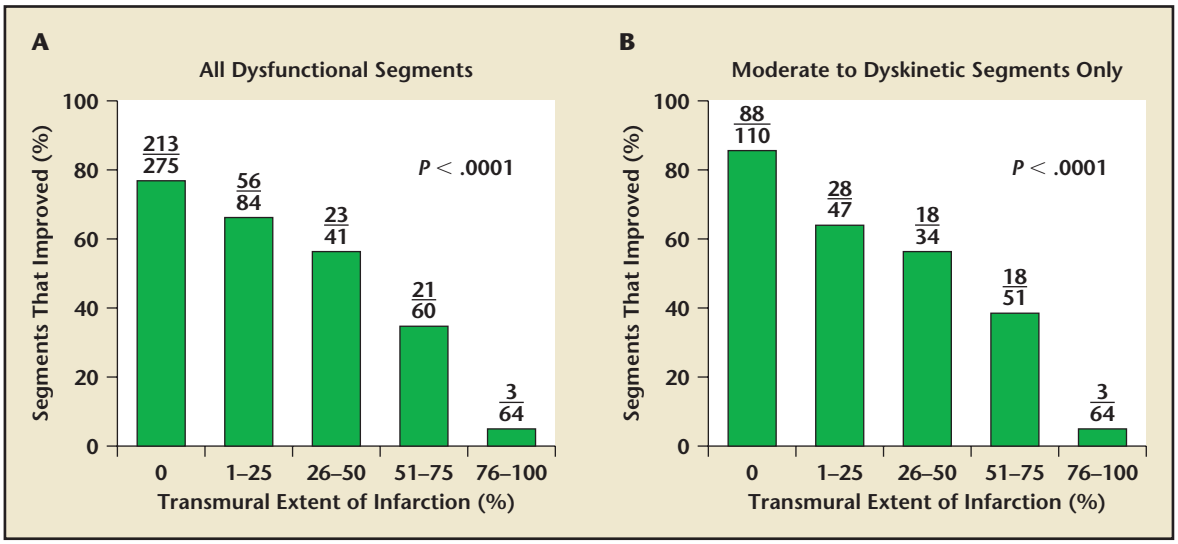


Figure 4. *Of all dysfunctional segments on scan 1, the likelihood of improvement in contractile function decreased with increasing transmural extent of infarction. The numbers above each column refer to the number of segments. Reprinted with permission from Choi KM et al.*[27]
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GE CMR and cine MRI before and after surgical or percutaneous revascularization.[28]

Before revascularization, 804 of the 2093 myocardial segments analyzed (38%) had abnormal contractility, and 694 segments (33%) had variable degrees of hyperenhancement. In the analysis of the 804 dysfunctional segments, the likelihood for the improvement in the regional contractility following revascularization was inversely related to the transmural extent of hyperenhancement ($P < .001$).

The percentage of the left ventricle that was both dysfunctional and not hyperenhanced before revascularization was strongly related to the improvement in segmental and global function ($P < .001$). The study clearly demonstrates that GE CMR can define reversible myocardial dysfunction before coronary revascularization. The ability of GE CMR to predict reversible myocardial dysfunction was tested in patients with CHF treated with beta blockers.[29] Forty-five patients with CHF underwent cine and GE CMR. Cine imaging was used to evaluate LV function and was repeated in 35 patients after 6 months of beta-blockade therapy. GE CMR showed scarring in 30 of 45 patients (67%). Scarring was found in 100% of patients with ischemic cardiomyopathy (28 patients) but in only 12% of patients with dilated cardiomyopathy (2 patients out of 17). In the 35 patients who were maintained on beta-blocker therapy, there was an inverse relationship between the extent of enhancement on GE CMR and the likelihood of contractile improvement 6 months later ($P < .001$).

Multivariate analysis showed that the amount of dysfunctional yet viable myocardium was an independent predictor of an improvement in LV function. In conclusion, in patients with CHF on beta-blocker therapy, GE CMR predicts the response in LV function, supporting the notion that in patients with LV systolic dysfunction, medical therapy often can achieve as much or more improvement than more aggressive therapies.[30,31]

Cardiac MRI has always shown favorable results when compared with other diagnostic modalities for the assessment of myocardial viability. For example, in a recent study, GE CMR was compared with PET scan in assessing viability in patients with CAD and LV dysfunction.[32] Twenty-six patients with ischemic cardiomyopathy underwent both GE CMR and PET scanning. In a 17-segment model, the extent of transmural enhancement was compared with the segmental F-fluorodeoxyglucose uptake by PET scan. In severely dysfunctional segments (n = 165), subendocardial enhancement was 9 + 14% in segments with normal metabolism/perfusion, 33 + 25% ($P < .05$) in segments with metabolism perfusion mismatch, and 80 + 23% ($P < .05$) in segments with matched defect.

The segmental glucose uptake by PET scan was inversely related to the subendocardial enhancement by GE CMR ($r = -0.86$; $P < .01$). The study shows that GE CMR allows assessment of myocardial viability with high accuracy compared with PET scan.

Overall, GE CMR is a very accurate clinical modality in distinguishing viable from nonviable myocardium at various levels of LV dysfunction and can predict improvement of segmental and global LV function in both acute and chronic CAD. These features, along with high spatial resolution, lack of imaging artifacts, and obviation of the need to perform a stress test, make GE CMR a gold standard technique to assess myocardial viability.

Coronary Imaging

CAD is the leading cause of mortality. The gold standard for diagnosis—coronary angiogram—is considered an invasive technique with risk of radiation and dye exposure. Thus, the development of a robust, noninvasive technique to accurately define CAD has always been desirable.

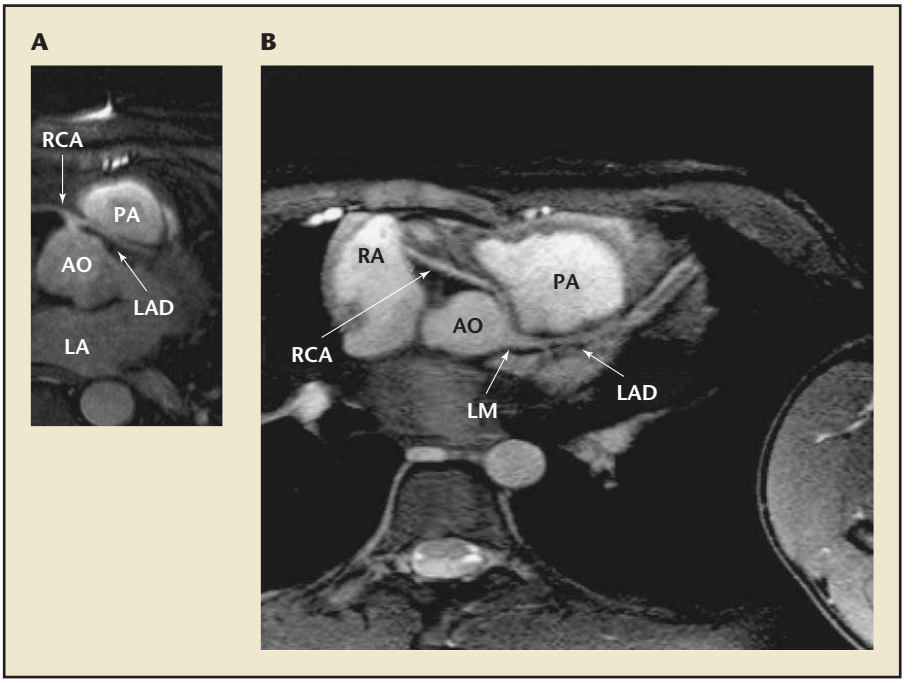
The use of cardiac MRI to visualize the coronary tree has improved significantly over the past several years due to the improvement in cardiac and respiratory navigator systems that prevent artifacts induced by cardiac and respiratory motion. The most striking description of coronary artery imaging was recently presented by Kim and colleagues.[33] They sought to demonstrate the utility for coronary imaging by CMR in a generic setting, choosing centers that had demonstrated prowess at cardiac, but not necessarily coronary, imaging. They recognized that there were many "recipes" for coronary acquisitions, and they provided all sites with one prescription and used a vendor-specific sequence and platform (Philips, Bothell, WA). This multicenter, international study enrolled 109 patients who underwent state-of-the-art 3-dimensional MRI prior to conventional invasive x-ray coronary angiography.

Eighty-four percent of the major coronary arteries were visualized by MRI (vs 100% by x-ray angiography). Eighty-three percent of clinically significant coronary artery abnormalities that were later identified on x-ray coronary angiography were also detected with the cardiac MRI scanner. Clinically, these results translated into an overall accuracy of 72% for the MRI scanner versus 100% accuracy for the standard x-ray angiographic technique. In detection of those patients at greatest risk of a life-threatening heart attack (eg, patients with significant left main artery or 3-vessel CAD), the overall accuracy of MRI was 100%. It should be noted that MRI was unable to successfully image the coronary arteries in 16% of study patients.

Despite this advancement, the routine use of CMR instead of coronary angiography to visualize the coronary tree is currently not recommended because the technique still suffers from limitations, including an inability to visualize intra-stent pathology and limited visualization of the distal coronary segments.[34] However, certain clinical indications compel interrogation of the present ability and potential applications of noninvasive coronary imaging by cardiac MRI.

Coronary artery anomalies and Kawasaki disease are the 2 exceptional conditions in which cardiac MRI has a major role in diagnosis and is considered the standard test of choice. Anomalous origin of the coronary artery is usually a benign condition. However, a coronary artery course between the aortic root and the right ventricular outflow tract (RVOT) is considered malignant (Figure 5). Compression of the artery between the major outflow tracts (usually during exercise) is associated with ischemic arrhythmias and sudden death. Bunce and colleagues[35] studied 26 patients suspected of

Figure 5. *These coronary MRI images depict* **(A)** *a malignant-type anomalous LAD originating from the proximal RCA and* **(B)** *a malignant-type anomalous origin of the RCA from the left coronary cusp. RCA, right coronary artery; PA, pulmonary artery; AO, aorta; LAD, left anterior descending artery; LA, left atrium; LM, left main artery; RA, right atrium; MRI, magnetic resonance imaging.* www.medreviews.com



having anomalous origin of coronary arteries by both MRI and coronary angiogram. MRI revealed 6 anomalous circumflex arteries passing behind the aortic root; 6 right coronary arteries passing between the aortic root and the RVOT; 9 left anterior descending arteries arising from the right sinus of Valsalva, 7 of which coursed between the aortic root and the RVOT; and 5 patients with minor anomalies. Of the 13 malignant courses of the coronary arteries detected by MRI, 8 were missed by coronary angiogram. This accuracy of MRI in the diagnosis of anomalous origin of the coronary arteries has been replicated in other studies.[36-38] These findings make cardiac MRI the gold standard test to diagnose anomalous origin of the coronary arteries.

Coronary artery abnormalities develop in 15% to 25% of patients with Kawasaki disease. This condition traditionally mandated the use of coronary angiography for initial diagnosis and frequent follow-up, which led to significant repetitive exposure to dye and radiation in the young age group that contracts this disease. The use of CMR in patients with Kawasaki disease has been evaluated in many studies and showed accurate results compared with cardiac catheterization, making CMR the test of choice for diagnosis and follow-up.[39-42]

Conclusion

Cardiac MRI is the most recent modality to demonstrate potential for clinical imaging of patients with varied cardiac manifestations of disease. However, in the past decade, it has quickly jumped to the forefront of the competing modalities to provide rapid, highly accurate, and reproducible cardiac information in a noninvasive manner. The evolution of MRI is gaining speed as evidenced by the growing sentiment, backed by compelling studies, that in many cases MRI is superior to current standards offered to patients today. Those institutions that have cardiac MRI or are seeking to acquire it will lead the next generation of advanced cardiac imaging. When coronary imaging by MRI has been shown to demonstrate efficacy, the consideration for alternative imaging will be even less necessary or compelling. Attributes of MRI include noninvasiveness; patient comfort; reduction in risk; exponential improvements in speed, ease, and accuracy; and recent documentation of robustness in a landmark coronary clinical trial. The next 5 years will see cardiac MRI become the preferred imaging modality for an increasing number of indications, including cardiomyopathy and anomalous coronary arteries, and the preferred screening test for patients with low and intermediate risk or for those who wish to avoid the risk of invasive angiography. ■

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Main Points

- Magnetic resonance imaging (MRI) adds a new dimension to cardiac diagnosis and provides data regarding cardiac structure, function, and perfusion that are, in many cases, superior to data from all other available techniques—invasive as well as noninvasive.
- Cardiac MRI provides a detailed examination of pericardial structure and precise measurement of pericardial thickness.
- Cardiac MRI adds significant, clinically relevant information to that provided by echocardiography in patients with dilated, hypertrophic, and infiltrative cardiomyopathies as well as arrhythmogenic right ventricular dysplasia.
- The routine use of cardiac MRI instead of coronary angiography to visualize the coronary tree is currently not recommended because the technique still suffers from limitations, including an inability to visualize intra-stent pathology and limited visualization of the distal coronary segments.
- Cardiac MRI is the gold standard test to diagnose anomalous origin of the coronary arteries.

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