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Cardiovascular Magnetic Resonance Imaging

WARREN J. MANNING, M.D.

Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts, USA

Introduction

The combined attributes of superior image quality and lack of ionizing radiation exposure with flexibility for assessment of cardiac anatomy, ventricular function, viability, perfusion, valvular function, great vessel anatomy, blood flow, and native coronary artery and coronary artery bypass graft integrity give cardiovascular magnetic resonance (CMR) unmatched potential for the comprehensive evaluation of the cardiovascular system. However, the complexity in both acquisition and interpretation, lack of standardization, and relative scarcity of CMR training sites and trained CMR practitioners have hampered the clinical utilization of CMR. Infrastructure costs, relatively unfavorable reimbursement (vs. nuclear and echo), and training issues all combine to make CMR a relatively small "player" in the noninvasive cardiovascular imaging field (estimated < 500,000 studies in the United States in 2005), although tremendous growth will occur in the coming years as the education of practitioners increases, reimbursement and training issues are overcome, and studies defining the value of CMR unique information are published. The Society for Cardiovascular Magnetic Resonance (www.scmr.org), an international society focused on CMR, already has over 1,500 members.

Currently accepted clinical applications of CMR continue to expand rapidly (Table I).[1] Hardware (gradients and high field systems) advances now allow for subsecond data acquisitions with "real-time" imaging, while software advances and novel contrast agents promise to exploit CMR's advantages over competing noninvasive imaging methods further. Cardiovascular magnetic resonance training guidelines for both those in fellowship[2,3] and practitioners who have completed fellowship have now been developed.[3–5]

Address for reprints:

Warren J. Manning, M.D.
Beth Israel Deaconess Medical Center
Cardiovascular Division
330 Brookline Avenue
Boston, MA 02215, USA
e-mail: wmanning@bidmc.harvard.edu

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Cardiovascular Magnetic Resonance: Technical Considerations

A review of magnetic resonance (MR) physics is beyond the scope of this review, and readers are referred elsewhere.[6,7] Unlike other imaging techniques, CMR images may depict blood and other tissues as "bright," "dark," or of an intermediate intensity depending on the specific sequence that is employed, the use of exogenous contrast, whether the tissue of interest (e.g., blood) is moving rapidly or slowly, and whether blood flow is laminar or turbulent. The most common CMR approaches are the spin-echo (typically "black blood"), the k-space segmented gradient echo, and the steady-state free precession (SSFP), including true fast imaging with steady-state precision (TrueFISP), balanced fast field flow (FFE), and fast imaging employing steady-state excitation (FIESTA) (typically "bright blood" sequences). Prepulses to highlight or suppress specific tissues may then be added. Cardiovascular magnetic resonance "tagging" methods provide for application of a "grid" with subsequent observation as the intersections become distorted because of motion during the cardiac cycle.

Exogenous intravenous contrast (e.g., gadolinium [Gd-DTPA]) may be used in combination with both spin-echo and gradient-echo approaches and has been particularly valuable for assessment of myocardial fibrosis/scar for viability evaluation, assessment of regional myocardial perfusion, and for characterization of tumors/masses.[8] These extracellular CMR contrast agents (approved by the U.S. Food and Drug Administration [FDA] for noncardiac applications) are not nephrotoxic and display a highly favorable anaphylaxis profile.[9] They are commonly used for three-dimensional (3-D) MR angiography, during which image acquisition is performed during the first passage of the contrast agent through the vascular bed of interest. Another very useful CMR sequence is phase velocity mapping, in which the velocity of blood perpendicular to the imaging plane is encoded, providing localized flow data somewhat analogous to pulsed Doppler echocardiography. This approach is particularly valuable for quantifying valvular regurgitation and flows through the great vessels.

The vast majority of CMR applications depend on accurate R-wave detection for electrocardiographic (ECG) triggering. In the presence of a rate-controlled irregularly irregular rhythm,

TABLE I Current clinical applications of cardiovascular magnetic resonance (CMR) [a]

1. Indications for CMR in acquired diseases of the vessels
   a. Diagnosis and monitoring of thoracic aortic aneurysm, dissection, aortic wall hematoma, and penetrating ulcer
   b. Assessment of pulmonary artery dilation and dissection
   c. Characterization of pulmonary vein stenosis
2. Indications for CMR in coronary artery disease
   a. Assessment of global and regional left and right ventricular systolic function at rest and with pharmacologic stress
   b. Assessment of regional myocardial perfusion at rest and with stress
   c. Determination of viability
3. Indications for CMR in valvular heart disease
   a. Assessment of the severity of aortic stenosis and mitral stenosis
   b. Quantitative assessment of mitral and aortic regurgitation
4. Indications for CMR in cardiomyopathies and pericardial disease
   a. Differentiation of ischemic versus nonischemic cardiomyopathy and underlying etiology (including hypertrophic cardiomyopathy, noncompaction, arrhythmic right ventricular cardiomyopathy, and iron deposition)
   b. Characterization of mass, biventricular volumes, and ejection fraction
   c. Identification of pericardial thickening, circumferential and local pericardial effusions
5. Indications for CMR in congenital heart disease
   a. Anomalous coronary artery disease
   b. Quantification of intracardiac shunt
   c. Characterization of simple and complex coronary anatomy
   d. Identification of aortic and pulmonary pathology (e.g., coarctation, patent ductus arteriosus)
   e. Characterization of anomalies of the ventricles
   f. Anomalous pulmonary venous drainage

[a] Adapted from Ref. No. 1.

such as atrial fibrillation, image quality will be acceptable,[10] but high-grade ventricular ectopy or a regularly irregular rhythm (e.g., trigeminy) often leads to significant image degradation. In these situations, real-time CMR is often used, although this approach has reduced spatial and temporal resolution.[11]

Specific Considerations for Cardiovascular Magnetic Resonance in the Cardiac Patient

In addition to general restrictions regarding CMR (ferromagnetic intracranial clips, TENS units, intra-auricular implants, shrapnel, etc; Table II),[12,13] there are also special considerations for CMR (and general MR scanning) in the cardiac patient. A comprehensive discussion of CMR safety with regard to implanted devices is beyond the scope of this review, and readers are referred to www.mrisafety.com.[14] At current field strengths (1.5 and 3.0 tesla [T]), CMR imaging is considered safe for both bioprosthetic and mechanical heart valves;[14] however, a local image artifact (loss of signal/image distortion) will occur in the region surrounding the valve prosthesis (Fig. 1). Similarly, sternotomy wires, thoracic vascular clips, and ostial coronary artery bypass graft markers are not a contraindication to CMR imaging, but localized artifacts will be present, thereby limiting assessment of adjacent structures. The relative size of the image artifact is increased with gradi-

TABLE II Cardiovascular magnetic resonance (CMR) safety at ≤ 1.5 T

Safe for CMR scanning (but will cause local image artifacts)
Coronary artery stents
Coronary artery bypass graft markers
Sternotomy wires
Prosthetic valves (bioprosthetic and mechanical)
Safe for CMR scanning if done with special precautions/protocols[12,13]
Modern (2000+) pacemakers
Unsafe for CMR scanning
Pulmonary artery catheters with pacing lead components
Retained permanent pacing leads not connected to a generator
Older (pre-2000) pacemakers, implantable cardiodefibrillators

For a more complete and continuously updated list, readers are directed to www.mrisafety.com.

FIG. 1 Single image from a cine cardiovascular magnetic resonance in the two-chamber view. Note the artifact (black arrow) resulting from a bileaflet mechanical mitral valve prosthesis. LV = left ventricle.

ent echo and SSFP sequences versus spin-echo sequences. While the composition metal of intracoronary stents will cause a local susceptibility effect (dark area), no adverse events have been reported at 1.5 T.[15] In April 2005, the FDA approved both the Boston Scientific and Johnson & Johnson drug-eluting stents for immediate postimplant MR study.

The area of safety with regard to CMR scanning among patients with cardiac pacemakers and implantable cardiodefibrillators is currently in a state of flux. When CMR is the only imaging modality available, uncomplicated CMR has been performed at a low field strength, at 0.2 T[16] and at 1.5 T, using modern (post 2000) pacemakers in nonpacing-dependent patients using specific protocols and safeguards.[12, 13] The situation of retained permanent pacemaker leads is also considered a relative contraindication to CMR because of the potential for lead heating.[14]

Thoracic Aorta and Great Vessels

For many years, CMR has had its greatest clinical impact on the assessment of the thoracic aorta in the patient with known or suspected thoracic aortic aneurysm or aortic dissection (Fig. 2). Cardiovascular magnetic resonance compares favorably with spiral or multidetector computed tomography (CT) because it employs no ionizing radiation and has no requirement for potentially nephrotoxic iodinated contrast. Comprehensive data regarding the presence of a dissection, entry and exit points, presence of intraluminal thrombus, involvement of the great vessels, and coexistent aortic insufficiency and pericardial effusion are readily obtained.[17, 18] The sine qua non of aortic dissection for spin-echo imaging is the identification of an intimal "flap" separating the true and false lumen. A breath-hold 3-D contrast-enhanced (CE) magnetic resonance angiogram (MRA) is then obtained to define the aortic lumen either with[19] or without[20] ECG gating. Cine SSFP or gradient echo acquisitions can then be obtained if a dissection flap is identified. In addition to the classic finding of an intimal flap, an intramural hematoma can also be identified as eccentric aortic wall thickening.[21] In experienced hands, CMR, CT with iodinated contrast, and multiplane transesophageal echocardiography (TEE) have similarly high sensitivity, specificity, and accuracy for identification of thoracic aortic dissection.[17, 18, 22, 23] Cardiovascular magnetic resonance and CT have specific advantages (compared with multiplane TEE) for providing information regarding the extent of the dissection into the great vessels and abdominal aorta. Both TEE and CMR also permit determination of aortic valve involvement and aortic insufficiency (Fig. 2), although valve morphology is better defined by TEE. Multidetector CT, TEE, and CMR can also provide information regarding the involvement of the proximal coronary arteries. Study time and access are important factors in the choice of imaging test. We generally recommend CMR (or CT) for patients who are hemodynamically stable, and CMR for follow-up studies in younger patients with chronic aneurysms or dissection, and to refer stable older patients with good renal function to CT and utilize TEE for those with clinical instability, claustrophobia, or renal dysfunction.

In addition to aortic aneurysm and dissection, CMR is also useful for the assessment of congenital aortic lesions such as aortic coarctation. Phase velocity methods may be used to quantify velocity gradients.[24]

Pulmonary Embolism, Pulmonary Artery, and Pulmonary Vein Assessment

Compared with imaging of the aorta, CMR of the pulmonary artery is more technically demanding of patient breath-

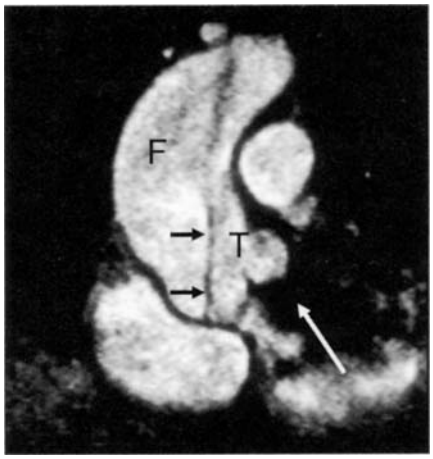


FIG. 2 Coronal diastolic frame from a cine gradient echo cardiovascular magnetic resonance in a patient with an ascending aortic dissection (black arrows). Note the dissection flap begins immediately above the aortic valve leaflet. Flow is seen in both the true and the false lumen. A prominent diastolic signal void (white arrow) from aortic regurgitation is also seen. F = false lumen, T = true lumen.

hold cooperation because of artifacts related to motion as the lungs expand and collapse. Typically, 3-D CE-MRA is used for pulmonary artery assessment.[25,26] Similar to conventional x-ray angiography or CT, pulmonary emboli present as an abrupt discontinuation (signal void) of the arterial lumen. Pulmonary artery dissection is a rare condition that may mimic aortic dissection in symptoms, for which pulmonary artery 3-D CE-MRA as well as gradient echo and spin-echo methods have been shown to be quite accurate.[27]

With increasing interest in pulmonary vein ablation as a mainstream therapy for patients with atrial fibrillation, monitoring of patients for asymptomatic pulmonary vein stenosis has become important. Pulmonary vein CE-MRA has been shown to be a very accurate method for monitoring the size of the pulmonary arteries[28–30] and for identifying stenoses (Fig. 3).[28,30,31] More recently, CMR methods for identifying left atrial and pulmonary vein scar have been reported[32] and offer the potential to assess the anatomic adequacy of ablation methods noninvasively (Fig. 4).

Quantitative Assessment of Ventricular Volumes and Mass

Although rarely used for "first-line" assessment, volumetric cine CMR is becoming increasingly recognized as the clinical "standard of reference" for the quantitative assessment of left and right ventricular volumes, global ejection fraction, regional systolic function, and biventricular mass in patients with known disease. Advantages of CMR include the ability to obtain high temporal and spatial resolution tomographic data in true short- and long-axis orientations, the outstanding endocardial border definition provided by current SSFP sequences, and the relative ease of data analysis. Semiautomated methods allow for the delineation of the endocardial and epicardial borders with both high accuracy and reproducibility for determination of ventricular volumes, stroke volume, and ejection fraction in normal, hypertrophied, and focally deformed ventricles.[33–35] Compared with M-mode or two-dimensional (2-D) echocardiography, which requires acoustic windows and is associated with suboptimal results in many obese and elderly patients, comprehensive, high temporal, and spatial resolution true short-axis volumetric CMR data sets are easily acquired in nearly all subjects in < 8 min.[36] Normative CMR data are sequence specific[37] and have identified racial differences.[38] Although in theory 3-D echocardiography and cardiac CT offer similar volumetric data, the superior spatial (vs. 3-D echocardiography) and temporal (vs. CT) resolutions of CMR make it the strong preference when accurate and reproducible assessments are needed. Volumetric CMR is especially valuable for quantitative information regarding left ventricular hypertrophy and volumes in patients with asymmetric hypertrophy, and defining cardiomyopathies (e.g., noncompaction; Fig. 5)[39] or monitoring ventricular volumes in patients with regurgitant valvular lesions. Left ventricular aneurysms may be recognized as severe wall thinning (< 4 mm) and diastolic bulging of the left ventricular free wall (Fig. 6). Left ventricular pseudoaneurysm or false aneurysms may also be readily identified on CMR because of their lack of myocardium in the wall of the aneurysm[40] with a relatively narrow neck. The superior reproducibility of CMR for ventricular volumes thereby provides more clinical utility in the monitoring of patients.[35,41,42] Volumetric CMR methods are also ideal for regional left ventricular assessment with the 17-segment model (6 basal; 6 mid; 4 apical; true apex)[43] generally used. Left ventricular mural thrombi may be identified on spin-echo images as a density/

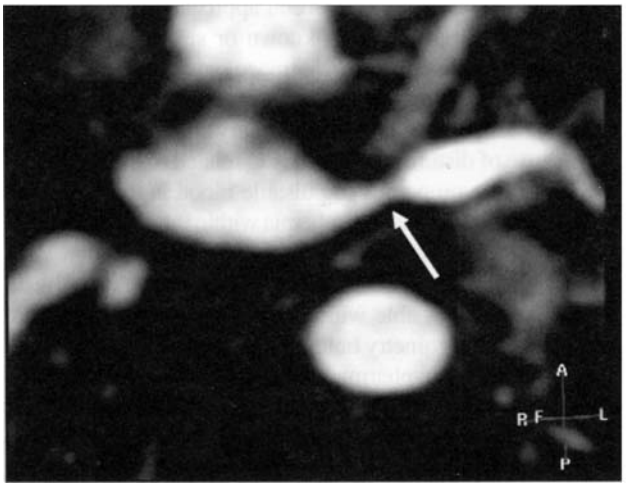


FIG. 3 Pulmonary vein contrast-enhanced magnetic resonance angiogram in a 65-year-old man with atrial fibrillation who underwent pulmonary vein ablation 1 month previously. Note the focal stenosis (white arrow) of the proximal left lower pulmonary vein. Courtesy of Dr. Thomas Hauser.

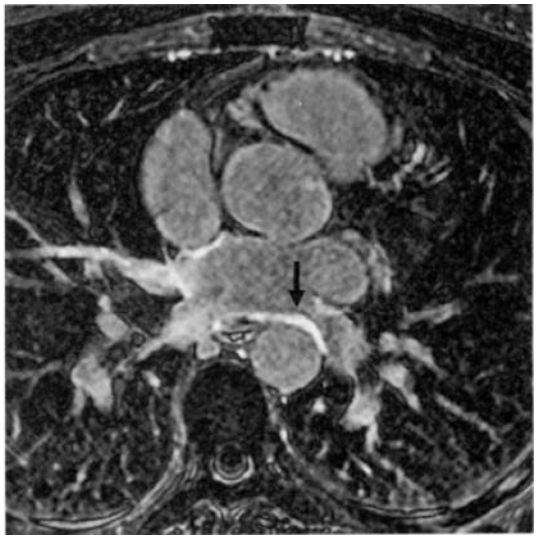
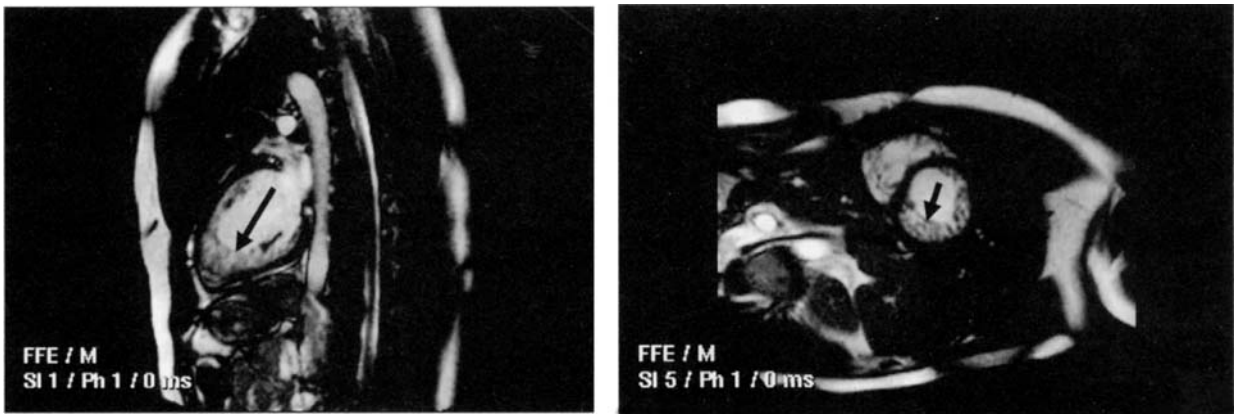
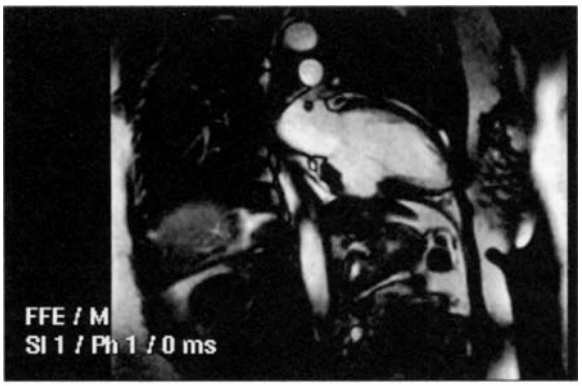


FIG. 4 Delayed-enhanced cardiovascular magnetic resonance in a patient who underwent pulmonary vein ablation for treatment of atrial fibrillation. Note the area of hyperenhancement (black arrow) in the posterior left atrium near the left lower pulmonary vein, indicative of a scar. Courtesy of Dr. Dana Peters.

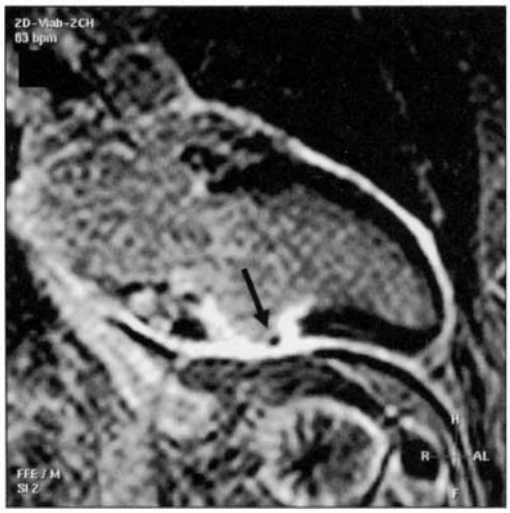


(A) (B)

FIG. 5 Cine steady-state free precession images in the (A) two-chamber and (B) short-axis orientations demonstrating prominent trabeculations (black arrows) consistent with noncompaction.



(A)



(B)

FIG. 6 (A) Cine steady-state free precession two-chamber and (B) delayed-enhanced cardiovascular magnetic resonance in a 54-year-old patient with an inferior infarction and aneurysm. Note the basal half of the inferior wall displays transmural hyperenhancement with a small thrombus (B, black arrow) along the subendocardial border.

mass filling the left ventricular apex[44] or as filling defects on cine gradient echo or SSFP imaging. Cardiovascular magnetic resonance methods may also depict transmural hyperenhancement in the wall of a true aneurysm, with a subendocardial area of hypoenhancement corresponding to a chronic left ventricular thrombus.[45, 46] Accurate quantitative evaluation of right ventricular volumes, ejection fraction, and mass is also a relatively unique attribute of CMR.[47] For regional assessment of both right and left ventricular systolic function, myocardial tagging techniques have been shown to be more sensitive for quantitation of local dysfunction,[48, 49] although their clinical role remains to be defined.

Detection of Coronary Artery Disease

In addition to ventricular volumes and global/regional systolic function, CMR offers several approaches for detecting and evaluating patients with known or suspected coronary artery disease. These include pharmacologic stress testing with beta agonists (regional dysfunction) or vasodilators (perfusion deficits), viability imaging, and coronary artery imaging.

Because of distortions of the ECG related to the magnetohydrodynamic properties of pulsatile blood in the aorta, the ECG is uninterpretable for ischemia within the CMR environment. Thus, close patient supervision and real-time monitoring of wall motion for dobutamine stress are imperative. Physiologic stress is possible within the CMR environment, and supine bicycle ergometry units have been developed for such an application,[50] but pharmacologic stress is more commonly used. In combination with graded doses of dobutamine (analogous to dobutamine stress echocardiography), cine images are acquired in the four- and two-chamber orientations along with three short-axis levels (base, mid, apical) at baseline and at each level of dobutamine. Data suggest that dobutamine stress CMR is more sensitive for the detection of coronary artery disease versus dobutamine stress echocardiography.[51–53] This superiority is directly related to the enhanced ability of CMR to

visualize/define all of the myocardial segments.[51] A study that compared dobutamine CMR stress with vasodilator CMR stress found dobutamine wall motion CMR to be superior.[54] The combination of CMR resting left ventricular ejection fraction and inducible ischemia has prognostic value among patients with known coronary artery disease.[55] Cardiovascular magnetic resonance tagging methods may offer superior sensitivity,[56] but they are less commonly used.

Cardiovascular magnetic resonance myocardial perfusion methods have advanced considerably over the past 5 years and now offer comprehensive three to six short-axis levels during the first passage of Gd-DTPA (0.05 mmol/kg) administered as a tight bolus into the right antecubital fossa. A rotational long-axis acquisition has also been proposed.[57] Both visual and quantitative methods (up-slope) have been utilized and validated in animal models.[58,59] Comparison studies with x-ray angiography and radionuclide imaging are very favorable,[60–62] with particular benefit for combined perfusion and delayed-enhancement (DE) approaches.[63] Cardiovascular magnetic resonance protocols of myocardial perfusion may include assessment of perfusion at rest and at peak stress, or a single peak-vasodilator assessment with normal resting systolic function used as a surrogate for normal perfusion. Vasodilator perfusion CMR has demonstrated an improvement in myocardial perfusion reserve after percutaneous coronary intervention,[64] and it has impaired subendocardial perfusion in syndrome X.[65]

Native Coronary Artery Disease Integrity

Cardiovascular magnetic resonance is used routinely for evaluation of vascular beds throughout the body, but coronary magnetic resonance imaging (MRI) is more technically challenging because of the small caliber, tortuosity, and motion related to the respiratory and cardiac cycles. As a result, coronary artery CMR continues to be a field of rapid evolution, with recent competition from coronary CT angiography (CTA) methods. Advantages of coronary CTA include the relative ease and speed of data acquisition as well as sophisticated reconstruction tools. Strengths of coronary CMR include both the lack of substantial ionizing radiation[66,67] and the need for potentially nephrotoxic/anaphylactic iodinated contrast or the need to induce bradycardia with beta blockade. Another disadvantage of coronary CTA is the difficulty with lumen integrity assessment among high-risk patients[68] and older patients due to prominent epicardial calcium.[69] Preliminary data suggest that epicardial calcium does not provide the same interference with coronary CMR depiction of the lumen (Fig. 7), and that coronary CMR and coronary CTA provide similar accuracy.[70]

Since the initial descriptions of 2-D breath-hold coronary CMR,[71,72] the field has advanced to 3-D acquisition methods (double-oblique slab or larger axial stack analogous to coronary CTA) with submillimeter spatial resolution and superior reconstruction capabilities. The spatial resolution of 3-D coronary MRI remains inferior to coronary CTA and x-ray coronary angiography, thereby precluding quantitative assessments although the magnitude of the local signal void does correlate with angiographic stenosis.[72] Data acquisition also remains relatively prolonged at 10 to 20 min. Despite this limitation, the feasibility of identifying stenoses in the proximal and midcoronary segments has been demonstrated in several single-center studies.[73–75] At present, some approaches (free-breathing navigator with real-time motion correction) remain vendor specific, and thus multicenter trials have been vendor specific. We continue to prefer a targeted 3-D free-breathing segmented k-space gradient echo sequence[75] using patient-specific delay and short acquisition (< 90 ms/R-R interval) periods.[76,77] With this approach, high signal intensity (bright blood) represents normal, laminar blood flow, with low signal (signal void) at sites of stenosis and focal turbulence (Fig. 7). A multicenter trial of over 100 patients from seven international sites demonstrated high sensitivity but only modest specificity for identifying focal stenoses, with very high accuracy for discriminating between patients with multivessel disease and no disease.[78] For this reason, we offer coronary CMR as a clinical option for patients presenting with dilated cardiomyopathy in the absence of a history of acute infarction. Preliminary data from a group of patients with depressed left ventricular systolic function[79] suggest that

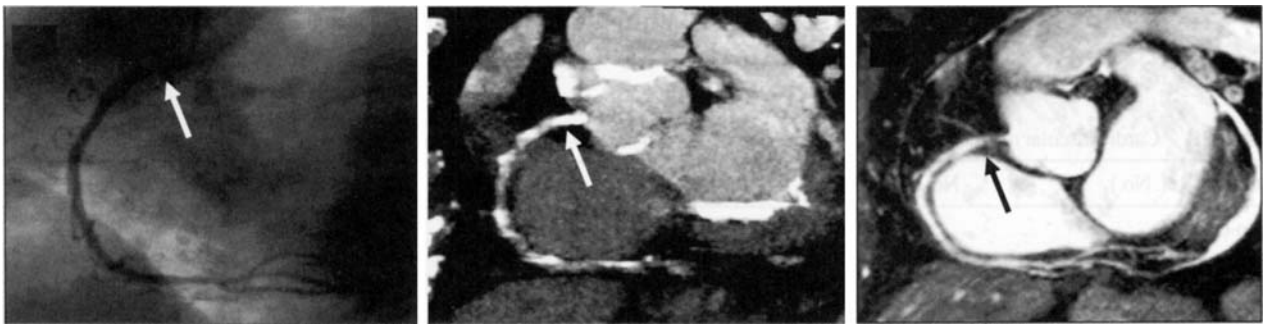


FIG. 7 Right coronary artery (RCA) (A) projection x-ray angiogram, (B) 16-slice multidetector computed tomography (CT), and (C) coronary cardiovascular magnetic resonance (CMR) in a patient with prominent epicardial calcium. Note the ostial RCA stenosis (arrows) is visible on the x-ray angiogram and the coronary CMR, but not on the multidetector CT because of extensive calcium. Courtesy of Dr. David Maintz.

coronary CMR is superior to DE-CMR for discriminating between these two subsets. Targeted 3-D approaches using SSFP methods[80] or a "whole heart" SSFP methodology that supports extensive reconstructions (Fig. 8),[81, 82] somewhat analogous to multidetector CT acquisitions (though inferior in spatial resolution) have been advocated. Comparative data suggest longer vessel segments identified, with improved signal-to-noise ratio and contrast-to-noise ratio with SSFP whole heart acquisitions,[81, 82] but similar diagnostic results. Preliminary data suggest similar overall results with 3.0-T coronary CMR.[83] It is very likely that coronary CMR methods will get faster and more automated with subsequent application of powerful CT analytic tools to the 3-D CMR data sets.

Although not yet routine because of issues of spatial resolution, another advantage of coronary MRI versus coronary CTA is the application of phase velocity flow methods to assess coronary artery blood flow and flow reserve. For patients who have experienced a myocardial infarction, phase velocity CMR can accurately evaluate the presence of antegrade flow in the infarct-related artery.[84] The noninvasive determination of patency influences therapy and prognosis in these patients.

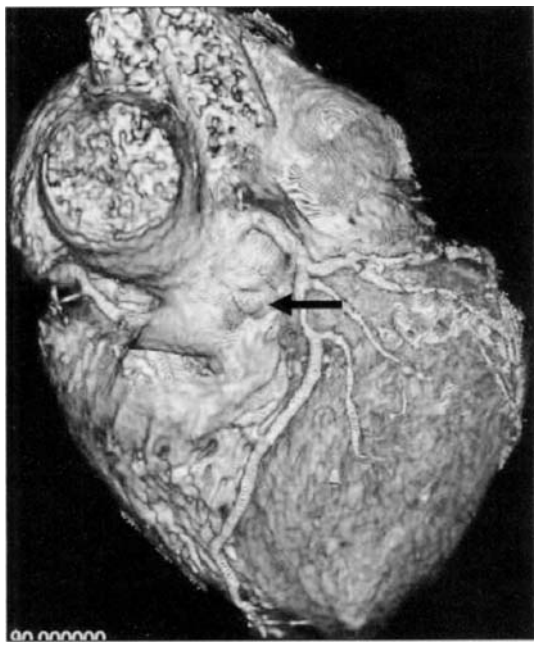


FIG. 8 Whole heart coronary magnetic resonance imaging reconstruction in a patient with a proximal left anterior descending stenosis. Courtesy of Dr. Hajima Sukama.

Coronary Artery Bypass Graft Patency

Compared with native coronary artery CMR, imaging of coronary artery bypass grafts (both saphenous veins and internal mammary arteries) is facilitated by their relatively stationary anterior location, straight and predictable course, and their greater lumen diameter. Adequate flow is visualized as a signal void (spin-echo) or as a bright signal (gradient echo, contrast imaging) in the anatomic location corresponding to the expected graft position. Identification of flow in at least two contiguous slices, or obtained at different planes perpendicular to the expected bypass graft course, suggests patency. If flow is suggested at only one level, graft patency is considered "indeterminate," and if there is no evidence of flow in any portions of the graft, the graft is considered "occluded." Spin-echo (dark blood), gradient echo (bright blood), and Gd-DTPA-enhanced 3-D coronary CMR have been reported to have higher sensitivity (95 to 100%) for patency of both saphenous venous and internal mammary grafts (Table III).[85–89] Focal disease can be identified using a 3-D coronary CMR sequence.[90] The addition of phase velocity imaging of graft flow[91] is also useful for discriminating vein graft patency, especially for jump grafts.

Implanted metallic clips, markers, or intracoronary stents[15] will have a local signal void, precluding assessment in these regions. These artifacts, which are commonly located in very close proximity to the coronary arteries or grafts, preclude the assessment of these vessels. As mentioned earlier, both drug-eluting stents currently marketed in the United States are FDA approved for CMR scanning immediately after implantation.

Myocardial Viability

Few applications of CMR have been so rapidly embraced by the clinical community as its ability to characterize myocardial fibrosis and thereby derive prognostic data regarding

TABLE III Cardiovascular magnetic resonance assessment of coronary artery bypass graft patency

Authors (Ref. No.)	No. of patients	No. of grafts	Percent patent	Sensitivity	Specificity
Spin-echo					
White *et al.* (85)	25	72	69	0.86	0.72
Rubinstein *et al.* (86)	20	47	62	0.90	0.72
Galjee *et al.* (87)	47	84	74	0.98	0.85
Gradient echo					
Aurigemma *et al.* (88)	45	45	73	0.88	1.00
Galjee *et al.* (87)	47	84	74	0.98	0.88

clinical viability—the likelihood that resting regional left ventricular dysfunction will improve with mechanical revascularization. Extensive correlative CMR and histologic studies in animal models have demonstrated that extracellular Gd-DTPA will localize/concentrate to areas corresponding to scar/fibrosis on histology and can be recognized using DE-CMR imaging.[92,93] Histologic correlation of DE-CMR is superior to nuclear methods because of the superior spatial resolution of CMR.[93] Using an inversion recovery sequence with imaging 10 to 20 min following injection of 0.1 to 0.2 mmol/kg of Gd-DTPA, areas of hyperenhancement correspond to scar/fibrosis (Fig. 6) with highly reproducible results.[94,95] The DE-CMR studies have demonstrated that the lack of hyperenhancement is a very strong predictor of functional viability, while the presence of >50% transmural hyperenhancement is a powerful predictor for the lack of functional recovery.[96–98] An intermediate finding (1–49% transmurality) is less useful. For this group, regional systolic response to low-dose dobutamine appears superior.[99] Delay-enhanced CMR also compares favorably with electromechanical mapping[100] and clinical positron-emission tomography.[101]

The DE-CMR method appears to be particularly superior to wall thinning for the discrimination of viable myocardium. Anecdotal reports[102] and preliminary data from a multicenter series[103] reported that 20% of subjects with thinned, akinetic segments had a lack of hyperenhancement in those segments. Following mechanical revascularization, these CMR-viable segments demonstrated markedly improved systolic thickening in addition to local hypertrophy/normalization of diastolic wall thickness. Delay-enhanced CMR also appears to be superior to global left ventricular ejection fraction for identification of patients with underlying substrate for sustained ventricular tachycardia.[104]

Nonischemic Cardiomyopathies

Initial studies suggested that hyperenhancement may be specific for coronary artery disease.[105] Subsequent studies have shown that hyperenhancement may occur in a variety of nonischemic myopathic conditions, including hypertrophic cardiomyopathy (Fig. 9),[106–108] Fabry's disease,[109] sarcoido-

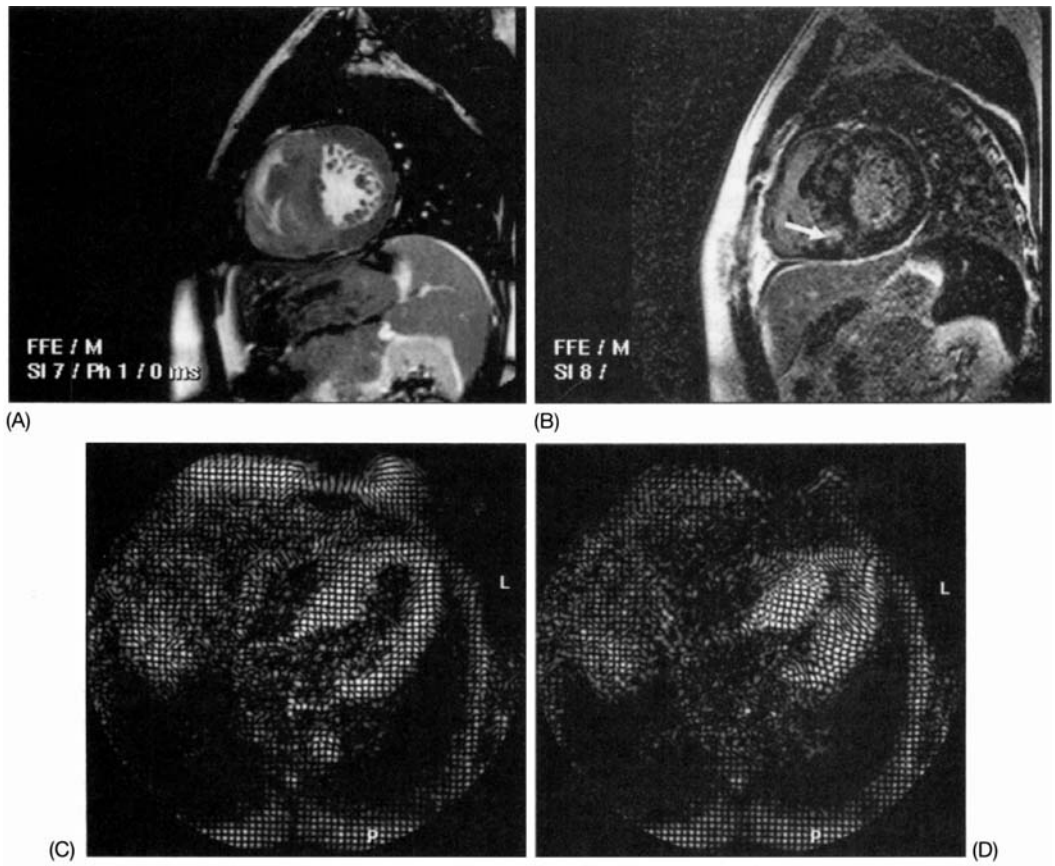


FIG. 9. (A) Cine steady-state free precession short-axis and corresponding (B) delayed-enhanced cardiovascular magnetic resonance in a 17-year-old patient with hypertrophic cardiomyopathy. Note the prominent septal hypertrophy with an area of hyperenhancement at the juncture of the right ventricular free wall and septum (white arrow). Tagged (C) end-diastolic and (D) end-systolic images from another patient with hypertrophic cardiomyopathy. Note the absence of tag distortion/contraction in the hypertrophied basal half of the septum. (A) and (B) courtesy of Dr. Martin Maron; (C) and (D) courtesy of Dr. Eli Gelfand.

sis,[110] myocarditis,[111, 112] and Churg-Strauss syndrome.[113] A diffuse pattern of hyperenhancement is seen in amyloid cardiomyopathy.[114] Though not totally specific for myocardial infarction, subendocardial hyperenhancement corresponding to a coronary artery distribution is far more common among patients with ischemic cardiomyopathy, while patients with nonischemic myopathies more often demonstrate a hyperenhancement pattern of the midventricular or epicardial layers that does not correspond to a coronary distribution.[107, 109, 111–113] Two studies[115, 116] suggested that the pattern may be used to characterize the cause of a dilated cardiomyopathy–although 15 to 25% of patients with a "coronary disease" hyperenhancement pattern had a nonischemic myopathy. In patients with dilated cardiomyopathy given carvedilol, the absence of hyperenhancement predicts a regional improvement in systolic function, global improvement in left ventricular ejection fraction, and a decrease in left ventricular cavity size.[117]

The ability of CMR to acquire images of the entire heart in true tomographic planes makes it ideal for the evaluation of patients with hypertrophic cardiomyopathies, which is especially important for those with focal/asymmetric hypertrophy. Both DE-CMR[106–108] and investigative CMR "tagging" methods[118] may assist in the assessment of these patients, though the latter remains to be more fully elucidated. Serial CMR examinations may be useful for monitoring ventricular remodeling and infarction size following alcohol ablation.[119]

In addition to biventricular volumetric and mass data, CMR may confirm iron deposition[120] as the cause of depressed systolic function in patients with suspected hemochromatosis. Septal T2* (measurement of transverse relaxation, which is shorter than T2 because of inhomogeneities) measurements reflect myocardial iron stores, with a T2* of < 20 ms indicative of iron overload.[121]

The ability of CMR to identify focal areas of fat and fibrosis is particularly valuable in the evaluation of patients with suspected arrhythmogenic right ventricular cardiomyopathy. This condition, in which the right ventricular free wall myocardium is diffusely or focally replaced with fatty or fibrous tissue with cavity dilation and focal wall thinning (or aneurysm), is associated with ventricular arrhythmias and sudden death. Spin-echo MR imaging can be used for identifying transmural or focal fatty infiltration in the right ventricular free wall, as well as focal wall thinning.[122, 123] Delay-enhanced CMR with right ventricular free wall hyperenhancement has also been described,[124] with the clinical history best able to discriminate hyperenhancement due to right ventricular infarction (Fig. 9) from that of a primary cardiomyopathy.

Valvular Heart Disease

The clinical adoption of CMR in the care of patients with valvular heart disease is also expanding rapidly. Once almost solely within the province of echocardiography, the unique quantitative nature of CMR with regard to ventricular volumes and function, as well as the ease in calculating regurgitant volumes, has brought CMR to the clinical arena for these patients. Valve morphology (e.g., bicuspid valve) is easily recognized by CMR with acquisition of a cine SSFP data set through the plane of the aortic valve. For now, leaflet thickening, calcification, vegetations, abscesses, and minor degrees of mitral valve prolapse remain largely within the province of echocardiography, although obvious prolapse and partial leaflet flail are readily identified on cine CMR imaging.

For the assessment of aortic valve stenosis, two CMR approaches are utilized: a morphologic/2-D assessment with planimetry of the maximum systolic aortic valve area[125] on orthogonal cine SSFP sequences, and a Continuity Equation "equivalent."[126] Similar to 2-D transthoracic echocardiography and TEE approaches, a weakness of the "anatomic" CMR method is the orientation of the slice in patients with markedly deformed valves, while difficulties with the Continuity Equation approach include orientation of the imaging plane perpendicular to the maximal velocity jet and dephasing artifacts due to turbulence in patients with severe aortic stenosis. Analogous approaches are used for assessing mitral stenosis, including 2-D planimetry of the mitral valve area as defined by a cine acquisition oriented orthogonal to the valve plane,[127] and with the use of phase velocity mapping at the level of the mitral leaflet tips,[128] thereby applying a pressure-half-time equivalent measurement. Difficulties with the former again include proper orientation orthogonal to flow, while limitations of the latter include the relatively poor temporal (vs. Doppler echocardiography) resolution and diastolic artifacts among the many patients with coexistent mitral stenosis and atrial fibrillation.

The use of CMR for the quantitative assessment of valvular regurgitation is much more direct and highly quantitative. While mitral and aortic regurgitation often cause a signal void due to local turbulence/dephasing in the receiving chamber in a manner somewhat analogous to color Doppler,[129] the use of SSFP sequences with very short echo times has led to attenuation and near elimination of the dephasing artifact.[130] Fortunately, CMR offers a more quantitative approach. Using phase velocity mapping, flow is measured across the aortic valve (from a practical perspective, this is often obtained in the axial plane at the level of the bifurcation of the pulmonary artery). Such an assessment provides a direct quantitative assessment of aortic regurgitation. For mitral regurgitation, we generally quantify the regurgitant volume as the difference between left ventricular stroke volume (derived from the contiguous short axis left ventricular stack) and forward flow out of the aorta. Another option is to measure mitral regurgitation volume directly using phase velocity mapping at the level of the mitral annulus. We have found the latter approach to be more technically challenging because of base-to-apex motion of the annulus during systole and eccentric, high-velocity mitral regurgitation jets, which sometimes lead to errors, analogous to some of the limitations of quantitative Doppler echocardiography.

Beyond simple calculation of regurgitant volume, CMR provides for the ready determination of regurgitant fraction (regurgitant volume/stroke volume), regurgitant volume index (regurgitant volume/end-diastolic volume), and effective for-

ward ejection fraction (net forward stroke volume/end-diastolic volume). Comparative CMR and Doppler echocardiographic studies in patients with mitral and aortic regurgitation have defined regurgitant fraction thresholds (Table IV).[131] These same measures can be applied equally well for pulmonic and tricuspid regurgitation. Another advantage of CMR versus echocardiography and invasive measures is its ability to quantify easily the regurgitant volumes attributable to each valve in the presence of serial regurgitant lesions (e.g., mitral regurgitation and aortic regurgitation). Preliminary data suggest that novel CMR measures such as effective forward ejection fraction may also provide a unique parameter to guide surgical intervention in the population with mitral regurgitation.[132]

TABLE IV Cardiovascular magnetic resonance (CMR) regurgitant fraction versus Doppler echocardiographic correlation

Regurgitant severity	CMR regurgitant fraction
Mild	≤15%
Moderate	16%–25%
Moderate–severe	26%–48%
Severe	>48%

Adapted from Ref. No. 131.

Cardiac Tumors and Masses

Although the high spatial resolution of CMR allows for depiction of intracavitary tumors/masses (e.g., myxoma), these intracavitary "masses" are generally well appreciated and characterized using conventional echocardiography (transthoracic and/or TEE); however, the sensitivity and accuracy of transthoracic echocardiography for mural left ventricular thrombi have recently been called into question by an operative series that suggested far superior accuracy of DE-CMR (Fig. 6).[46] Cardiovascular magnetic resonance also has great value for paracardiac and extracardiac tumors that extend into the myocardium, cardiac chambers, and/or neighboring mediastinal structures (e.g., venae cavae, pulmonary veins). The ability of 3-D CMR data sets to be reconstructed in any orientation helps in guiding the surgical approach in such situations.

Although rarely difficult to diagnose from echocardiographic images, benign lipomatous hypertrophy of the interatrial septum as visualized on transthoracic echocardiography or TEE may sometimes lead to the misdiagnosis of an atrial septal "tumor." The characteristic, very intense signal from fatty tissue[133] with suppression using a fat saturation prepulse readily allows for the CMR diagnosis of this benign disorder.

Pericardium

The normal pericardium extends around the heart as a thin black line between visceral and parietal pericardial fat on spin-echo CMR imaging. Normal CMR pericardial thickness is ≤3 mm.[134] Among patients presenting with constrictive cardiomyopathy, often following recurrent pericarditis or mediastinal radiation, the pericardium is thickened, a finding that is readily appreciated by ECG-triggered spin-echo CMR. Gradient echo methods are slightly less reliable for measuring pericardial thickness.[135] Computed tomography is also valuable in this situation and is better suited for the specific assessment of pericardial calcifications. While CMR (and CT) will accurately quantify focal pericardial thickening, the presence of thickened pericardium alone is not diagnostic of constrictive physiology, and constriction may be present in the absence of pericardial thickening.[136] Cardiovascular magnetic resonance tagging methods demonstrate adherence of the pericardium to underlying epimyocardium.[137] Among patients with constriction, CMR also frequently demonstrates thickened pericardium in concert with an enlarged inferior vena cava, along with right atrial and right ventricular enlargement[135] and abnormal septal motion (with real-time CMR). Although echocardiography is generally adequate for circumferential effusions, CMR depicts transudative effusions as areas of high intensity, and it may be particularly helpful in the delineation of loculated effusions, especially in patients with suboptimal echocardiographic windows. Delineation of hemorrhagic and transudative effusions is another attribute of CMR.

Congenital Heart Disease

An extensive review of CMR applications for congenital heart disease is beyond the scope of this review. It has great utility for both simple and complex congenital heart disease. While hemodynamically significant, atrial septal and ventricular septal defects are usually identifiable by transthoracic echocardiography and/or TEE, phase velocity CMR is highly accurate and valuable for quantifying the pulmonary:systemic flow ratio in patients with known defects[138] or for evaluating patients with dilated right-sided chambers. As previously mentioned, CMR is specifically valuable in the characterization of congenital heart disease outside of the cardiac chambers. These include aortic coarctation, anomalous pulmonary venous drainage (Table I),[1] and complex congenital heart disease in patients who have undergone corrective or palliative surgery. For these patients, CMR defines structural components and their relationships, including serial evaluation and planning of subsequent surgical interventions.

Compared with coronary artery CMR for assessment of stenoses, coronary CMR for identification and characterization of anomalous coronary arteries is a widely utilized clinical tool. This condition is found in < 2% of the population and is generally benign. However, there is an increased risk of sudden death and myocardial infarction when the anomalous vessel courses between the aorta and pulmonary artery (Fig. 10). Even among patients with anomalous coronary arteries identified by invasive x-ray angiography, the anatomic course of the vessel may be misinterpreted because of the projection method or operator inexperience, especially with the declin-

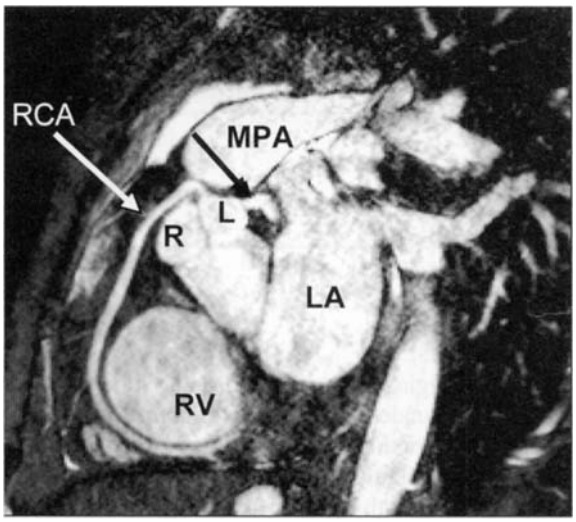


FIG. 10 Three-dimensional coronary cardiovascular magnetic resonance in a patient with an anomalous RCA arising from the left sinus. RCA = right coronary artery; MPA = main pulmonary artery, R = right sinus, L = left sinus, RV = right ventricle, LA = left atrium. The left main artery is also seen (black arrow). Courtesy of Dr. Franz Aepfelbacher.

ing routine use of right-heart catheterization, making coronary CMR a preferred approach. Several studies have now reported on the value of CMR in this condition,[139–142] including the finding of initial misinterpretation by conventional x-ray angiography.[140–142]

Cardiovascular Magnetic Resonance: Future Perspectives

Over the past decade, there has been tremendous clinical growth in CMR. The recent introduction of high-field (e.g., 3.0-T) CMR systems and the application of parallel imaging methods[143] in CMR have the potential to decrease the time needed dramatically for CMR study completion. Moreover, investigations of the use of CMR for detection of subclinical disease are ongoing[144] and are expected to expand further the clinical role of CMR in clinical care. Finally, the enhancement of real-time CMR has facilitated the exciting birth of interventional CMR methods, including placement of percutaneous valves and atrial septal defect devices, as well as guidance for electrophysiologic procedures. Interventional CMR is expected to have its greatest initial impact in the pediatric population[145] for which radiation exposure is of greatest concern. Ongoing training and sequence optimization along with multicenter trials will lead to continued utilization of CMR in the care of our patients.

References

1. Pennell DJ, Sechtem UP, Higgins CB, Manning WJ, Pohost GM, Rademakers FE, van Rossum AC, Shaw LJ, Yucel EK; European Society of Cardiology; Society for Cardiovascular Magnetic Resonance: Clinical indications for cardiovascular magnetic resonance (CMR): Consensus Panel report. *J Cardiovasc Magn Reson* 2004;6:727–765
2. Pohost GM, Kim RJ, Kramer CM, Manning WJ; Society for Cardiovascular Magnetic Resonance: Task Force 12: Training in advanced cardiovascular imaging (cardiovascular magnetic resonance [CMR]): Endorsed by the Society for Cardiovascular Magnetic Resonance. *J Am Coll Cardiol* 2006;47:910–914
3. Guidelines for credentialing in cardiovascular magnetic resonance (CMR). Society for Cardiovascular Magnetic Resonance (SCMR) Clinical Practice Committee. *J Cardiovasc Magn Reson* 2000;2: 233–234
4. Weinreb JC, Larson PA, Woodard PK, Stanford W, Rubin GD, Stillman AE, Bluemke DA, Duerinckx AJ, Dunnick NR, Smith GG; American College of Radiology: American College of Radiology clinical statement on noninvasive cardiac imaging. *Radiology* 2005;235:723–727
5. Budoff MJ, Cohen MC, Garcia MJ, Hodgson JM, Hundley WG, Lima JA, Manning WJ, Pohost GM, Raggi PM, Rodgers GP, Rumberger JA, Taylor AJ, Creager MA, Hirshfeld JW Jr, Lorell BH, Merli G, Rodgers GP, Tracy CM, Weitz HH: ACCF/AHA clinical competence statement on cardiac imaging with computed tomography and magnetic resonance: A report of the American College of Cardiology Foundation/American Heart Association/ American College of Physicians Task Force on Clinical Competence and Training. *J Am Coll Cardiol* 2005;46:383–402
6. *Cardiovascular Magnetic Resonance*, (Eds. Manning WJ, Pennell DJ) pp. 3–17. Philadelphia: Churchill Livingstone, 2002
7. *MRI and CT of the Cardiovascular System*. 2nd edition. (Eds. Higgins CB, de Roos A). Philadelphia: Lippincott Williams & Wilkins, 2005
8. Edelman RR: Contrast-enhanced MR imaging of the heart: Overview of the literature. *Radiology* 2004;232:653–668
9. Goyen M, Debatin JF: Gadopentetate dimeglumine-enhanced three-dimensional MR-angiography: Dosing, safety, and efficacy. *J Magn Reson Imaging* 2004;19:261–273
10. Hundley WG, Meshack BM, Willett DL, Sayad DE, Lange RA, Willard JE, Landau C, Hillis LD, Peshock RM: Comparison of quantitation of left ventricular volume, ejection fraction, and cardiac output in patients with atrial fibrillation by cine magnetic resonance imaging versus invasive measurements. *Am J Cardiol* 1996; 78:1119–1123
11. Kerr AB, Pauly JM, Hu BS, Li KC, Hardy CJ, Meyer CH, Macovski A, Nishimura DG: Real-time interactive MRI on a conventional scanner. *Magn Reson Med* 1997;38:355–367
12. Martin ET, Coman JA, Shellock FG, Pulling CC, Fair R, Jenkins K: Magnetic resonance imaging and cardiac pacemaker safety at 1.5-Tesla. *J Am Coll Cardiol* 2004;43:1315–1324
13. Roguin A, Zviman MM, Meininger GR, Rodrigues ER, Dickfeld TM, Bluemke DA, Lardo A, Berger RD, Calkins H, Halperin HR: Modern pacemaker and implantable cardioverter/defibrillator systems can be magnetic resonance imaging safe: In vitro and in vivo assessment of safety and function at 1.5 T. *Circulation* 2004;110: 475–482
14. Shellock FG: *Reference Manual for Magnetic Resonance Safety, Implants, and Devices*. 2005 edition. Philadelphia: W.B. Saunders, 2005
15. Kramer CM, Rogers WJ Jr, Pakstis DL: Absence of adverse outcomes after magnetic resonance imaging early after stent placement for acute myocardial infarction: A preliminary study. *J Cardiovasc Magn Reson* 2000;2:257–261
16. Gimbel JR, Johnson D, Levine PA, Wilkoff BL: Safe performance of magnetic resonance imaging on five patients with permanent cardiac pacemakers. *Pacing Clin Electrophysiol* 1996;19:913–919
17. Nienaber CA, von Kodolitsch Y, Nicolas V, Siglow V, Piepho A, Brockhoff C, Koschyk DH, Spielmann RP: The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med* 1993;328:1–9

18. Laissy JP, Blanc F, Soyer P, Assayag P, Sibert A, Tebboune D, Arrive L, Brochet E, Hvass U, Langlois J: Thoracic aortic dissection: Diagnosis with transesophageal echocardiography versus MR imaging. *Radiology* 1995;194:331–336
19. Goldfarb JW, Holland AE, Edelman RR: Single breath-hold multislab and cine cardiac-synchronized gadolinium-enhanced three-dimensional angiography. *Magn Reson Imaging* 2001;19:1267–1274
20. Krinsky GA, Reuss PM, Lee VS, Carbognin G, Rofsky NM: Thoracic aorta: Comparison of single-dose breath-hold and double-dose non-breath-hold gadolinium-enhanced three-dimensional MR angiography. *Am J Roentgenol* 1999;173:145–150
21. Murray JG, Manisali M, Flamm SD, VanDyke CW, Lieber ML, Lytle BW, White RD: Intramural hematoma of the thoracic aorta: MR image findings and their prognostic implications. *Radiology* 1997;204:349–355
22. Keren A, Kim CB, Hu BS, Eyngorina I, Billingham ME, Mitchell RS, Miller DC, Popp RL, Schnittger I: Accuracy of biplane and multiplane transesophageal echocardiography in diagnosis of typical acute aortic dissection and intramural hematoma. *J Am Coll Cardiol* 1996;28:627–636
23. Sommer T, Fehske W, Holzknecht N, Smekal AV, Keller E, Lutterbey G, Kreft B, Kuhl C, Gieseke J, Abu-Ramadan D, Schild H: Aortic dissection: A comparative study of diagnosis with spiral CT, multiplanar transesophageal echocardiography, and MR imaging. *Radiology* 1996;199:347–352
24. Oshinski JN, Parks WJ, Markou CP, Bergman HL, Larson BE, Ku DN, Mukundan S Jr, Pettigrew RI: Improved measurement of pressure gradients in aortic coarctation by magnetic resonance imaging. *J Am Coll Cardiol* 1996;28:1818–1826
25. Hatabu H, Gaa J, Kim D, Li W, Prasad PV, Edelman RR: Pulmonary perfusion and angiography: Evaluation with breath-hold enhanced three-dimensional fast imaging steady-state precession MR imaging with short TR and TE. *Am J Roentgenol* 1996;167:653–655
26. Meaney JF, Weg JG, Chenevert TL, Stafford-Johnson D, Hamilton BH, Prince MR: Diagnosis of pulmonary embolism with magnetic resonance angiography. *N Engl J Med* 1997;336:1422–1427
27. Stern EJ, Graham C, Gamsu G, Golden JA, Higgins CB: Pulmonary artery dissection: MR findings. *J Comput Assist Tomogr* 1992;16:481–483
28. Dill T, Neumann T, Ekinci O, Breidenbach C, John A, Erdogan A, Bachmann G, Hamm CW, Pitschner HF: Pulmonary vein diameter reduction after radiofrequency catheter ablation for paroxysmal atrial fibrillation evaluated by contrast-enhanced three-dimensional magnetic resonance imaging. *Circulation* 2003;107:845–850
29. Hauser TH, Yeon SB, McClennen S, Katsimaglis G, Kissinger KV, Josephson ME, Rofsky NM, Manning WJ: A method for the determination of proximal pulmonary vein size using contrast-enhanced magnetic resonance angiography. *J Cardiovasc Magn Reson* 2004; 6:927–936
30. Kato R, Lickfett L, Meininger G, Dickfeld T, Wu R, Juang G, Angkeow P, LaCorte J, Bluemke D, Berger R, Halperin HR, Calkins H: Pulmonary vein anatomy in patients undergoing catheter ablation of atrial fibrillation: Lessons learned by use of magnetic resonance imaging. *Circulation* 2003;107:2004–2010
31. Hauser TH, Yeon SB, McClennen S, Katsimaglis G, Kissinger KV, Josephson ME, Rofsky NM, Manning WJ: Subclinical pulmonary vein narrowing after ablation for atrial fibrillation. *Heart* 2005;91: 672–673
32. Peters DC, Wylie JV, Kissinger KV, Hauser T, Botnar RM, Essebag V, Josephson ME, Manning WJ: Detection of pulmonary vein ablation with high resolution MRI. *J Cardiovasc Magn Reson* 2006; 8(1):4–5
33. Cranney GB, Lotan CS, Dean L, Baxley W, Bouchard A, Pohost GM: Left ventricular volume measurement using cardiac axis nuclear magnetic resonance imaging. Validation by calibrated ventricular angiography. *Circulation* 1990;82:154–163
34. Mogelvang J, Stokholm KH, Saunamaki K, Reimer A, Stubgaard M, Thomsen C, Fritz-Hansen P, Henriksen O: Assessment of left ventricular volumes by magnetic resonance in comparison with radionuclide angiography, contrast angiography and echocardiography. *Eur Heart J* 1992;13:1677–1683
35. Grothues F, Smith GC, Moon JC, Bellenger NG, Collins P, Klein HU, Pennell DJ: 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
36. Salton CJ, Chuang ML, O'Donnell CJ, Kupka MJ, Larson MG, Kissinger KV, Edelman RR, Levy D, Manning WJ: Gender differences and normal left ventricular anatomy in an adult population free of hypertension. A cardiovascular magnetic resonance study of the Framingham Heart Study Offspring cohort. *J Am Coll Cardiol* 2002;39:1055–1060
37. Alfakih K, Plein S, Thiele H, Jones T, Ridgway JP, Sivananthan MU: Normal human left and right ventricular dimensions for MRI as assessed by turbo gradient echo and steady-state free precession imaging sequences. *J Magn Reson Imaging* 2003;17:323–329
38. Drazner MH, Dries DL, Peshock RM, Cooper RS, Klassen C, Kazi F, Willett D, Victor RG: Left ventricular hypertrophy is more prevalent in blacks than whites in the general population: The Dallas Heart Study. *Hypertension* 2005;46:124–129
39. Petersen SE, Selvanayagam JB, Wiesmann F, Robson MD, Francis JM, Anderson RH, Watkins H, Neubauer S: Left ventricular noncompaction: Insights from cardiovascular magnetic resonance imaging. *J Am Coll Cardiol* 2005;46:101–105
40. 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;14:603–606
41. Bellenger NG, Burgess MI, Ray SG, Lahiri A, Coats AJ, Cleland JG, Pennell DJ: Comparison of left ventricular ejection fraction and volumes in heart failure by echocardiography, radionuclide ventriculography and cardiovascular magnetic resonance: Are they interchangeable? *Eur Heart J* 2000;21:1387–1396
42. Johnson DB, Foster RE, Barilla F, Blackwell GG, Roney M, Stanley AW Jr, Kirk K, Orr RA, van der Geest RJ, Reiber JH, Dell'Italia LJ: Angiotensin-converting enzyme inhibitor therapy affects left ventricular mass in patients with ejection fraction > 40% after acute myocardial infarction. *J Am Coll Cardiol* 1997;29:49–54
43. Cerqueira MD, Weissman NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey WK, Pennell DJ, Rumberger JA, Ryan T, Verani MS; American Heart Association Writing Group on Myocardial Segmentation and Registration for Cardiac Imaging: Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation* 2002;105:539–542
44. McNamara MT, Higgins CB: Magnetic resonance imaging of chronic myocardial infarcts in man. *Am J Roentgenol* 1986;146: 315–320
45. Mollet NR, Dymarkowski S, Volders W, Wathiong J, Herbots L, Rademakers FE, Bogaert J: Visualization of ventricular thrombi with contrast-enhanced magnetic resonance imaging in patients with ischemic heart disease. *Circulation* 2002;106:2873–2876
46. Srichai MB, Junor C, Rodriguez LL, Stillman AE, Grimm RA, Lieber ML, Weaver JA, Smedira NG, White RD: Imaging and pathologic characteristics of left ventricular thrombus: A comparison of contrast-enhanced magnetic resonance imaging, transthoracic echocardiography, and transesophageal echocardiography with surgical or pathologic validation (abstr). *Circulation* 2004; 110:III–404
47. Grothues F, Moon JC, Bellenger NG, Smith GS, Klein HU, Pennell DJ: Interstudy reproducibility of right ventricular volumes, function, and mass with cardiovascular magnetic resonance. *Am Heart J* 2004;147:218–223
48. Fayad ZA, Ferrari VA, Kraitchman DL, Young AA, Palevsky HI, Bloomgarden DC, Axel L: Right ventricular regional function using MR tagging: Normals versus chronic pulmonary hypertension. *Magn Reson Med* 1998;39:116–123

49. Kramer CM, Rogers WJ, Theobald TM, Power TP, Petruolo S, Reichek N: Remote noninfarcted region dysfunction soon after first anterior myocardial infarction. A magnetic resonance tagging study. *Circulation* 1996;94:660–666
50. Roest AA, Kunz P, Lamb HJ, Helbing WA, van der Wall EE, de Roos A: Biventricular response to supine physical exercise in young adults assessed with ultrafast magnetic resonance imaging. *Am J Cardiol* 2001;87:601–605
51. Nagel E, Lehmkuhl HB, Bocksch W, Klein C, Vogel U, Frantz E, Ellmer A, Dreysse S, Fleck E: Noninvasive diagnosis of ischemia-induced wall motion abnormalities with the use of high-dose dobutamine stress MRI: Comparison with dobutamine stress echocardiography. *Circulation* 1999;99:763–770
52. Hundley WG, Hamilton CA, Thomas MS, Herrington DM, Salido TB, Kitzman DW, Little WC, Link KM: Utility of fast cine magnetic resonance imaging and display for the detection of myocardial ischemia in patients not well suited for second harmonic stress echocardiography. *Circulation* 1999;100:1697–1702
53. Schalla S, Klein C, Paetsch I, Lehmkuhl H, Bornstedt A, Schnackenburg B, Fleck E, Nagel E: Real-time MR image acquisition during high-dose dobutamine hydrochloride stress for detecting left ventricular wall-motion abnormalities in patients with coronary arterial disease. *Radiology* 2002;224:845–851
54. Paetsch I, Jahnke C, Wahl A, Gebker R, Neuss M, Fleck E, Nagel E: Comparison of dobutamine stress magnetic resonance, adenosine stress magnetic resonance, and adenosine stress magnetic resonance perfusion. *Circulation* 2004;110:835–842
55. Hundley WG, Morgan TM, Neagle CM, Hamilton CA, Rerkpattanapipat P, Link KM: Magnetic resonance imaging determination of cardiac prognosis. *Circulation* 2002;106:2328–2333
56. Kuijpers D, Ho KY, van Dijkman PR, Vliegenthart R, Oudkerk M: Dobutamine cardiovascular magnetic resonance for the detection of myocardial ischemia with the use of myocardial tagging. *Circulation* 2003;107:1592–1597
57. Wang Y, Moin K, Mathew ST, Akinboboye O, Reichek N: Myocardial first-pass perfusion assessment using rotational long-axis MRI. *J Magn Reson Imaging* 2005;22:53–58
58. Al-Saadi N, Nagel E, Gross M, Bornstedt A, Schnackenburg B, Klein C, Klimek W, Oswald H, Fleck E: Noninvasive detection of myocardial ischemia from perfusion reserve based on cardiovascular magnetic resonance. *Circulation* 2000;101:1379–1383
59. Epstein FH, London JF, Peters DC, Goncalves LM, Agyeman K, Taylor J, Balaban RS, Arai AE: Multislice first-pass cardiac perfusion MRI: Validation in a model of myocardial infarction. *Magn Reson Med* 2002;47:482–491
60. Wolff SD, Schwitter J, Coulden R, Friedrich MG, Bluemke DA, Biederman RW, Martin ET, Lansky AJ, Kashanian F, Foo TK, Licato PE, Comeau CR: Myocardial first-pass perfusion magnetic resonance imaging: A multicenter dose-ranging study. *Circulation* 2004;110:732–737
61. Panting JR, Gatehouse PD, Yang GZ, Jerosch-Herold M, Wilke N, Firmin DN, Pennell DJ: Echo-planar magnetic resonance myocardial perfusion imaging: Parametric map analysis and comparison with thallium SPECT. *J Magn Reson Imaging* 2001;13:192–200
62. Schwitter J, Nanz D, Kneifel S, Bertschinger K, Buchi M, Knusel PR, Marincek B, Luscher TF, von Schulthess GK: Assessment of myocardial perfusion in coronary artery disease by magnetic resonance: A comparison with positron emission tomography and coronary angiography. *Circulation* 2001;103:2230–2235
63. Klem I, Heitner JF, Shah DJ, Sketch MH Jr, Behar V, Weinsaft J, Cawley P, Parker M, Elliott M, Judd RM, Kim RJ: 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;47:1630–1638
64. Al-Saadi N, Nagel E, Gross M, Schnackenburg B, Paetsch I, Klein C, Fleck E: Improvement of myocardial perfusion reserve early after coronary intervention: Assessment with cardiac magnetic resonance imaging. *J Am Coll Cardiol* 2000;36:1557–1564
65. Panting JR, Gatehouse PD, Yang GZ, Grothues F, Firmin DN, Collins P, Pennell DJ: Abnormal subendocardial perfusion in cardiac syndrome X detected by cardiovascular magnetic resonance imaging. *N Engl J Med* 2002;346:1948–1953
66. Hunold P, Vogt FM, Schmermund A, Debatin JF, Kerkhoff G, Budde T, Erbel R, Ewen K, Barkhausen J: Radiation exposure during cardiac CT: Effective doses at multi-detector row CT and electron-beam CT. *Radiology* 2003;226:145–152
67. Coles DR, Smail MA, Negus IS, Wilde P, Oberhoff M, Karsch KR, Baumbach A: Comparison of radiation doses from multislice computed tomography coronary angiography and conventional diagnostic angiography. *J Am Coll Cardiol* 2006;47:1840–1845
68. Shaw LJ, Raggi P, Schisterman E, Berman DS, Callister TQ: Prognostic value of cardiac risk factors and coronary artery calcium screening for all-cause mortality. *Radiology* 2003;228:826–833
69. Hoffmann U, Moselewski F, Cury RC, Ferencik M, Jang IK, Diaz LJ, Abbara S, Brady TJ, Achenbach S: Predictive value of 16-slice multidetector spiral computed tomography to detect significant obstructive coronary artery disease in patients at high risk for coronary artery disease: Patient-versus segment-based analysis. *Circulation* 2004;110:2638–2643
70. Kefer J, Coche E, Legros G, Pasquet A, Grandin C, Van Beers BE, Vanoverschelde JL, Gerber BL: Head-to-head comparison of three-dimensional navigator-gated magnetic resonance imaging and 16-slice computed tomography to detect coronary artery stenosis in patients. *J Am Coll Cardiol* 2005;46:92–100
71. Manning WJ, Li W, Edelman RR: A preliminary report comparing magnetic resonance coronary angiography with conventional angiography. *N Engl J Med* 1993;328:828–832
72. Pennell DJ, Bogren HG, Keegan J, Firmin DN, Underwood SR: Assessment of coronary artery stenosis by magnetic resonance imaging. *Heart* 1996;75:127–133
73. Sommer T, Hackenbroch M, Hofer U, Meyer C, Flacke S, Schild H: Submillimeter 3D coronary MR angiography with real-time navigator correction in 112 patients with suspected coronary artery disease (abstr). *J Cardiovasc Magn Reson* 2001;4:28
74. Bogaert J, Kuzo R, Dymarkowski S, Beckers R, Piessens J, Rademakers FE: Coronary artery imaging with real-time navigator three-dimensional turbo-field-echo MR coronary angiography: Initial experience. *Radiology* 2003;226:707–716
75. 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;99:3139–3148
76. Jahnke C, Paetsch I, Schnackenburg B, Bornstedt A, Gebker R, Fleck E, Nagel E: Coronary MR angiography with steady-state free precession: Individually adapted breath-hold technique versus free-breathing technique. *Radiology* 2004;232:669–676
77. Plein S, Jones TR, Ridgway JP, Sivananthan MU: Three-dimensional coronary MR angiography performed with subject-specific cardiac acquisition windows and motion-adapted respiratory gating. *Am J Roentgenol* 2003;180:505–512
78. Kim WY, Danias PG, Stuber M, Flamm SD, Plein S, Nagel E, Langerak SE, Weber OM, Pedersen EM, Schmidt M, Botnar RM, Manning WJ: Coronary magnetic resonance angiography for the detection of coronary stenoses. *N Engl J Med* 2001;345:1863–1869
79. Hauser TH, Yeon SB, Appelbaum E: Discrimination of ischemic vs. non-ischemic cardiomyopathy among patients with heart failure using combined coronary MRI and delayed enhancement CMR (abstr). *J Cardiovasc Magn Reson* 2005;7:94
80. Maintz D, Aepfelbacher FC, Kissinger KV, Botnar RM, Danias PG, Heindel W, Manning WJ, Stuber M: Coronary MR angiography: Comparison of quantitative and qualitative data from four techniques. *Am J Roentgenol* 2004;182:515–521
81. Weber OM, Martin AJ, Higgins CB: Whole-heart steady-state free precession coronary artery magnetic resonance angiography. *Magn Reson Med* 2003;50:1223–1228
82. Sakuma H, Ichikawa Y, Suzawa N, Hirano T, Makino K, Koyama N, Van Cauteren M, Takeda K: Assessment of coronary arteries

with total study time of less than 30 minutes by using whole-heart coronary MR angiography. *Radiology* 2005;237:316–321
83. Sommer T, Hackenbroch M, Hofer U, Schmiedel A, Willinek WA, Flacke S, Gieseke J, Traber F, Fimmers R, Litt H, Schild H: Coronary MR angiography at 3.0 T versus that at 1.5 T: Initial results in patients suspected of having coronary artery disease. *Radiology* 2005;234:718–725
84. Kefer J, Coche E, Legros G, Pasquet A, Grandin C, Van Beers BE, Vanoverschelde JL, Gerber BL: Head-to-head comparison of three-dimensional navigator-gated magnetic resonance imaging and 16-slice computed tomography to detect coronary artery stenosis in patients. *J Am Coll Cardiol* 2005;46:92–100
85. Hundley WG, Clarke GD, Landau C, Lange RA, Willard JE, Hillis LD, Peshock RM: Noninvasive determination of infarct artery patency by cine magnetic resonance angiography. *Circulation* 1995; 91:1347–1353
86. White RD, Caputo GR, Mark AS, Modin GW, Higgins CB: Coronary artery bypass graft patency: Noninvasive evaluation with MR imaging. *Radiology* 1987;164:681–686
87. Rubinstein RI, Askenase AD, Thickman D, Feldman MS, Agarwal JB, Helfant RH: Magnetic resonance imaging to evaluate patency of aortocoronary bypass grafts. *Circulation* 1987;76:786–791
88. Galjee MA, van Rossum AC, Doesburg T, van Eenige MJ, Visser CA: Value of magnetic resonance imaging in assessing patency and function of coronary artery bypass grafts. An angiographically controlled study. *Circulation* 1996;93:660–666
89. Aurigemma GP, Reichek N, Axel L, Schiebler M, Harris C, Kressel HY: Noninvasive determination of coronary artery bypass graft patency by cine magnetic resonance imaging. *Circulation* 1989;80: 1595–1602
90. Vrachliotis TG, Bis KG, Aliabadi D, Shetty AN, Safian R, Simonetti O: Contrast-enhanced breath-hold MR angiography for evaluating patency of coronary artery bypass grafts. *Am J Roentgenol* 1997;168:1073–1080
91. Langerak SE, Vliegen HW, de Roos A, Zwinderman AH, Jukema JW, Kunz P, Lamb HJ, van Der Wall EE: Detection of vein graft disease using high-resolution magnetic resonance angiography. *Circulation* 2002;105:328–333
92. Langerak SE, Kunz P, Vliegen HW, Jukema JW, Zwinderman AH, Steendijk P, Lamb HJ, van der Wall EE, de Roos A: MR flow mapping in coronary artery bypass grafts: A validation study with Doppler flow measurements. *Radiology* 2002;222:127–135
93. Fieno DS, Kim RJ, Chen EL, Lomasney JW, Klocke FJ, Judd RM: Contrast-enhanced magnetic resonance imaging of myocardium at risk: Distinction between reversible and irreversible injury throughout infarct healing. *J Am Coll Cardiol* 2000;36:1985–1991
94. Wagner A, Mahrholdt H, Holly TA, Elliott MD, Regenfus M, Parker M, Klocke FJ, Bonow RO, Kim RJ, Judd RM: Contrast-enhanced MRI and routine single photon emission computed tomography (SPECT) perfusion imaging for detection of subendocardial myocardial infarcts: An imaging study. *Lancet* 2003;361:374–379
95. Mahrholdt H, Wagner A, Holly TA, Elliott MD, Bonow RO, Kim RJ, Judd RM: Reproducibility of chronic infarct size measurement by contrast-enhanced magnetic resonance imaging. *Circulation* 2002;106:2322–2327
96. Thiele H, Kappl MJ, Conradi S, Niebauer J, Hambrecht R, Schuler G: Reproducibility of chronic and acute infarct size measurement by delayed enhancement-magnetic resonance imaging. *J Am Coll Cardiol* 2006;47:1641–1645
97. Kim RJ, Wu E, Rafael A, Chen EL, Parker MA, Simonetti O, Klocke FJ, Bonow RO, Judd RM: The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med* 2000;343:1445–1453
98. Schvartzman PR, Srichai MB, Grimm RA, Obuchowski NA, Hammer DF, McCarthy PM, Kasper JM, White RD: Nonstress delayed-enhancement magnetic resonance imaging of the myocardium predicts improvement of function after revascularization for chronic ischemic heart disease with left ventricular dysfunction. *Am Heart J* 2003;146:535–541
99. Gerber BL, Garot J, Bluemke DA, Wu KC, Lima JA: Accuracy of contrast-enhanced magnetic resonance imaging in predicting improvement of regional myocardial function in patients after acute myocardial infarction. *Circulation* 2002;106:1083–1089
100. Wellnhofer E, Olariu A, Klein C, Grafe M, Wahl A, Fleck E, Nagel E: Magnetic resonance low-dose dobutamine test is superior to SCAR quantification for the prediction of functional recovery. *Circulation* 2004;109:2172–2174
101. Perin EC, Silva GV, Sarmento-Leite R, Sousa AL, Howell M, Muthupillai R, Lambert B, Vaughn WK, Flamm SD: Assessing myocardial viability and infarct transmurality with left ventricular electromechanical mapping in patients with stable coronary artery disease: Validation by delayed-enhancement magnetic resonance imaging. *Circulation* 2002;106:957–961
102. Kuhl HP, Beek AM, van der Weerdt AP, Hofman MB, Visser CA, Lammertsma AA, Heussen N, Visser FC, van Rossum AC: Myocardial viability in chronic ischemic heart disease: Comparison of contrast-enhanced magnetic resonance imaging with (18)F-fluorodeoxyglucose positron emission tomography. *J Am Coll Cardiol* 2003;41:1341–1348
103. John AS, Dreyfus GD, Pennell DJ: Images in cardiovascular medicine. Reversible wall thinning in hibernation predicted by cardiovascular magnetic resonance. *Circulation* 2005;111:e24–e25
104. James O, Kim HW, Weinsaft J, Wu E, Elliott M, Zenker N, Parker MA, Bonow RO, Kim RJ, Shah DJ: Demonstration and prediction of the potential reversible nature of thinned myocardium by CMR (abstr). *J Cardiovasc Magn Reson* 2005;7:69–70
105. Bello D, Fieno DS, Kim RJ, Pereles FS, Passman R, Song G, Kadish AH, Goldberger JJ: Infarct morphology identifies patients with substrate for sustained ventricular tachycardia. *J Am Coll Cardiol* 2005;45:1104–1108
106. 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: 21–28
107. Choudhury L, Mahrholdt H, Wagner A, Choi KM, Elliott MD, Klocke FJ, Bonow RO, Judd RM, Kim RJ: Myocardial scarring in asymptomatic or mildly symptomatic patients with hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2002;40:2156–2164
108. Moon JC, Reed E, Sheppard MN, Elkington AG, Ho SY, Burke M, Petrou M, Pennell DJ: The histologic basis of late gadolinium enhancement cardiovascular magnetic resonance in hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2004;43:2260–2264
109. van Dockum WG, ten Cate FJ, ten Berg JM, Beek AM, Twisk JW, Vos J, Hofman MB, Visser CA, van Rossum AC: Myocardial infarction after percutaneous transluminal septal myocardial ablation in hypertrophic obstructive cardiomyopathy: Evaluation by contrast-enhanced magnetic resonance imaging. *J Am Coll Cardiol* 2004;43:27–34
110. Moon JC, Sachdev B, Elkington AG, McKenna WJ, Mehta A, Pennell DJ, Leed PJ, Elliott PM: Gadolinium enhanced cardiovascular magnetic resonance in Anderson-Fabry disease. Evidence for a disease specific abnormality of the myocardial interstitium. *Eur Heart J* 2003;24:2151–2155
111. Vignaux O, Dhote R, Duboc D, Blanche P, Dusser D, Weber S, Legmann P: Clinical significance of myocardial magnetic resonance abnormalities in patients with sarcoidosis: A 1-year follow-up study. *Chest* 2002;122:1895–1901
112. Mahrholdt H, Goedecke C, Wagner A, Meinhardt G, Athanasiadis A, Vogelsberg H, Fritz P, Klingel K, Kandolf R, Sechtem U: Cardiovascular magnetic resonance assessment of human myocarditis: A comparison to histology and molecular pathology. *Circulation* 2004;109:1250–1258
113. 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;97:1802–1809
114. Smedema JP, van Paassen P, van Kroonenburgh MJ, Snoep G, Crijns HJ, Tervaert JW: Cardiac involvement of Churg Strauss syn-

drome demonstrated by magnetic resonance imaging. *Clin Exp Rheumatol* 2004;22(suppl 36):S75–S78
115. Maceira AM, Joshi J, Prasad SK, Moon JC, Perugini E, Harding I, Sheppard MN, Poole-Wilson PA, Hawkins PN, Pennell DJ: Cardiovascular magnetic resonance in cardiac amyloidosis. *Circulation* 2005;111:186–193
116. McCrohon JA, Moon JC, Prasad SK, McKenna WJ, Lorenz CH, Coats AJ, Pennell DJ: Differentiation of heart failure related to dilated cardiomyopathy and coronary artery disease using gadolinium-enhanced cardiovascular magnetic resonance. *Circulation* 2003;108:54–59
117. Casolo G, Minneci S, Manta R, Sulla A, Del Meglio J, Rega L, Gensini G: Identification of the ischemic etiology of heart failure by cardiovascular magnetic resonance imaging: Diagnostic accuracy of late gadolinium enhancement. *Am Heart J* 2006;151:101–108
118. Bello D, Shah DJ, Farah GM, Di Luzio S, Parker M, Johnson MR, Cotts WG, Klocke FJ, Bonow RO, Judd RM, Gheorghiade M, Kim RJ: Gadolinium cardiovascular magnetic resonance predicts reversible myocardial dysfunction and remodeling in patients with heart failure undergoing beta-blocker therapy. *Circulation* 2003; 108:1945–1953
119. Kramer CM, Reichek N, Ferrari VA, Theobald T, Dawson J, Axel L: Regional heterogeneity of function in hypertrophic cardiomyopathy. *Circulation* 1994;90:186–194
120. Amano Y, Takayama M, Amano M, Kumazaki T: MRI of cardiac morphology and function after percutaneous transluminal septal myocardial ablation for hypertrophic obstructive cardiomyopathy. *Am J Roentgenol* 2004;182:523–527
121. Johnston DL, Rice L, Vick GW III, Hedrick TD, Rokey R: Assessment of tissue iron overload by nuclear magnetic resonance imaging. *Am J Med* 1989;87:40–47
122. Anderson LJ, Holden S, Davis B, Prescott E, Charrier CC, Bunce NH, Firmin DN, Wonke B, Porter J, Walker JM, Pennell DJ: Cardiovascular T2-star (T2*) magnetic resonance for the early diagnosis of myocardial iron overload. *Eur Heart J* 2001;22:2171–2179
123. Carlson MD, White RD, Trohman RG, Adler LP, Biblo LA, Merkatz KA, Waldo AL: Right ventricular outflow tract ventricular tachycardia: Detection of previously unrecognized anatomic abnormalities using cine magnetic resonance imaging. *J Am Coll Cardiol* 1994;24:720–727
124. Globits S, Kreiner G, Frank H, Heinz G, Klaar U, Frey B, Gossinger H: Significance of morphological abnormalities detected by MRI in patients undergoing successful ablation of right ventricular outflow tract tachycardia. *Circulation* 1997;96:2633–2640
125. Tandri H, Saranathan M, Rodriguez ER, Martinez C, Bomma C, Nasir K, Rosen B, Lima JA, Calkins H, Bluemke DA: Noninvasive detection of myocardial fibrosis in arrhythmogenic right ventricular cardiomyopathy using delayed-enhancement magnetic resonance imaging. *J Am Coll Cardiol* 2005;45:98–103
126. Kupfahl C, Honold M, Meinhardt G, Vogelsberg H, Wagner A, Mahrholdt H, Sechtem U: Evaluation of aortic stenosis by cardiovascular magnetic resonance imaging: Comparison with established routine clinical techniques. *Heart* 2004;90:893–901
127. Caruthers SD, Lin SJ, Brown P, Watkins MP, Williams TA, Lehr KA, Wickline SA: Practical value of cardiac magnetic resonance imaging for clinical quantification of aortic valve stenosis: Comparison with echocardiography. *Circulation* 2003;108:2236–2243
128. Djavidani B, Debl K, Lenhart M, Seitz J, Paetzel C, Schmid FX, Nitz WR, Feuerbach S, Riegger G, Luchner A: Planimetry of mitral valve stenosis by magnetic resonance imaging. *J Am Coll Cardiol* 2005;45:2048–2053
129. Lin SJ, Brown PA, Watkins MP, Williams TA, Lehr KA, Liu W, Lanza GM, Wickline SA, Caruthers SD: Quantification of stenotic mitral valve area with magnetic resonance imaging and comparison with Doppler ultrasound. *J Am Coll Cardiol* 2004;44:133–137
130. Higgins CB, Wagner S, Kondo C, Suzuki J, Caputo GR: Evaluation of valvular heart disease with cine gradient echo magnetic resonance imaging. *Circulation* 1991;84(suppl):I198–I207
131. Suzuki J, Caputo GR, Kondo C, Higgins CB: Cine MR imaging of valvular heart disease: Display and imaging parameters affect the size of the signal void caused by valvular regurgitation. *Am J Roentgenol* 1990;155:723–727
132. Gelfand EV, Hughes S, Hauser TH, Yeon SB, Goepfert L, Kissinger KV, Rofsky NM, Manning WJ: Severity of mitral and aortic regurgitation as assessed by cardiovascular magnetic resonance: Optimizing correlation with Doppler echocardiography. *J Cardiovasc Magn Reson* 2006;8:503–507
133. Gelfand EV, Hauser TH, Goepfert L, Kissinger KV, de la Torre R, Manning WJ: Preoperative effective forward left ventricular ejection fraction in patients with chronic severe mitral regurgitation predicts left ventricular function following mitral valve surgery (abstr). *J Cardiovasc Magn Reson* 2006;8(1):167–168
134. Levine RA, Weyman AE, Dinsmore RE, Southern J, Rosen BR, Guyer DE, Brady TJ, Okada RD: Noninvasive tissue characterization: Diagnosis of lipomatous hypertrophy of the atrial septum by nuclear magnetic resonance imaging. *J Am Coll Cardiol* 1986;7: 688–692
135. Sechtem U, Tscholakoff D, Higgins CB: MRI of the normal pericardium. *Am J Roentgenol* 1986;147:239–244
136. Masui T, Finck S, Higgins CB: Constrictive pericarditis and restrictive cardiomyopathy: Evaluation with MR imaging. *Radiology* 1992;182:369–373
137. Talreja DR, Edwards WD, Danielson GK, Schaff HV, Tajik AJ, Tazelaar HD, Breen JF, Oh JK: Constrictive pericarditis in 26 patients with histologically normal pericardial thickness. *Circulation* 2003;108:1852–1857
138. Kojima S, Yamada N, Goto Y: Diagnosis of constrictive pericarditis by tagged cine magnetic resonance imaging. *N Engl J Med* 1999;341:373–374
139. Hundley WG, Li HF, Lange RA, Pfeifer DP, Meshack BM, Willard JE, Landau C, Willett D, Hillis LD, Peshock RM: 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;91:2955–2960
140. McConnell MV, Ganz P, Selwyn AP, Li W, Edelman RR, Manning WJ: Identification of anomalous coronary arteries and their anatomic course by magnetic resonance coronary angiography. *Circulation* 1995;92:3158–3162
141. Post JC, van Rossum AC, Bronzwaer JG, de Cock CC, Hofman MB, Valk J, Visser CA: Magnetic resonance angiography of anomalous coronary arteries. A new gold standard for delineating the proximal course? *Circulation* 1995;92:3163–3171
142. Vliegen HW, Doornbos J, de Roos A, Jukema JW, Bekedam MA, van der Wall EE: Value of fast gradient echo magnetic resonance angiography as an adjunct to coronary arteriography in detecting and confirming the course of clinically significant coronary artery anomalies. *Am J Cardiol* 1997;79:773–776
143. Taylor AM, Thorne SA, Rubens MB, Jhooti P, Keegan J, Gatehouse PD, Wiesmann F, Grothues F, Somerville J, Pennell DJ: Coronary artery imaging in grown up congenital heart disease: Complementary role of magnetic resonance and x-ray coronary angiography. *Circulation* 2000;101:1670–1678
144. McGee KP, Debbins JP, Boskamp EB, Blawat L, Angelos L, King KF: Cardiac magnetic resonance parallel imaging at 3.0 Tesla: Technical feasibility and advantages. *J Magn Reson Imaging* 2004; 19:291–297
145. Botnar RM, Stuber M, Kissinger KV, Kim WY, Spuentrup E, Manning WJ: Noninvasive coronary vessel wall and plaque imaging with magnetic resonance imaging. *Circulation* 2000;102: 2582–2587
146. Razavi R, Hill DL, Keevil SF, Miquel ME, Muthurangu V, Hegde S, Rhode K, Barnett M, van Vaals J, Hawkes DJ, Baker E: Cardiac catheterisation guided by MRI in children and adults with congenital heart disease. *Lancet* 2003;362:1877–1882