Published in final edited form as:

Cardiol Rev. 2011 ; 19(5): 246–254. doi:10.1097/CRD.0b013e31821f4d6a.

Cardiovascular Magnetic Resonance: Applications in Daily Practice

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Introduction

Over the last 10 years, the development of newer pulse sequences and applications in new clinical areas has enabled cardiovascular magnetic resonance (CMR) to emerge as a powerful tool for the physician to both diagnose and guide treatment of various cardiac pathologies. The greatest strengths of CMR include the assessment of ischemia and viability, evaluation of non-ischemic cardiomyopathies including myocarditis, pericardial disease, congenital heart disease, as well as tissue characterization of cardiac masses. Appropriate indications for CMR have been incorporated into ACC/AHA guidelines over the last decade and are summarized in Table 1.

Coronary Artery Disease

One of the most useful clinical applications of CMR is the evaluation of myocardial viability. Areas of fibrosis and scarring after myocardial infarction (MI) have an increased extracellular space compared to normal myocardium. Based on this principle, areas of prior MI will appear bright on a T₁-weighted (T₁-W) sequence performed 10-15 minutes after an injection of gadolinium contrast agent (2), (Figure 1). The superior spatial resolution of CMR allows detection of subendocardial infarction which may be missed by nuclear medicine techniques (3). The ability to quantify transmural extent of infarction has important clinical implications, particularly when patients are being referred for high-risk percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). It has been demonstrated that contractility after revascularization with PCI or CABG improves in 78% of dysfunctional segments that have no late gadolinium enhancement (LGE), and 60% in those with scar up to 25% of LV wall thickness (4). When analyzed by wall segments, anterior wall viability appears to contribute the most to improvement in left ventricular (LV) function post-CABG (5). It is worth noting that 65-70% of segments with subendocardial infarct that demonstrate severe hypokinesis, dyskinesis or akinesis will have an improvement in contractility after revascularization, stressing the importance of viability testing in patients with pre-operative LV dysfunction to distinguish hibernating myocardium from regions of transmural scar that are not likely to improve with revascularization (4,6).

The clinical dilemma of what to recommend for patients who have numerous segments with scar comprising 25-50% of LV wall thickness remains, as these segments have an intermediate probability of functional recovery (4,6). Using a 16-segment model and a transmural viability cutoff of 50%, patients with 10 or more normal and viable segments had an improvement in global LV ejection fraction $\geq 3\%$ after CABG (positive predictive value 87%, negative predictive value 89%), (7). Incorporating low-dose dobutamine testing (10

ug/kg for 10 minutes) into the CMR protocol may also be useful in this subset of patients. Those dysfunctional segments with up to 50% transmural infarct that demonstrate an improvement in contractility to normal with dobutamine have a significantly greater chance of functional recovery 4-6 months post-CABG when compared to patients without as robust of a response (8). Another study demonstrated that response to dobutamine was superior to transmural scar in discriminating those who would have an improvement function in segments with 1-74% transmural scar (9).

Complications of recent myocardial infarction such as ventricular septal defect and pseudoaneurysm formation can be evaluated by CMR (Figure 2). In addition, CMR in the early post-MI period can provide prognostic information. Acute infarct size by LGE and the presence of microvascular obstruction (MVO), areas of low signal on late enhanced images (Figure 2), are more predictive than LVEF or patency of infarct-related artery post-MI in predicting long-term prognosis and adverse LV remodeling (10). In addition, the presence of MVO is a significant predictor of MACE post MI, where there is only a trend toward increased MACE with larger infarct size (11).

Myocardial salvage index (MSI) has been shown to be an independent predictor of MACE in reperfused patients with acute MI (12). T₂-weighted (T₂-W) imaging of area of myocardial edema defines area at risk as validated in both animal models (13) and clinically (14). The salvage index is calculated as the (area at risk-final infarct size)/area at risk. The area at risk is obtained by identifying areas of myocardial edema (those regions >2 standard deviations in intensity when compared to normal myocardium), with all values represented as a percentage of total LV mass. A major need in the field is standardization of T₂-W sequences across CMR centers to enable MSI to become a quantitative tool for future clinical trials involving new reperfusion and infarct size reduction strategies (12,15).

An advantage of CMR stress testing over other modalities is the ability to assess ischemia, function, and diagnose small, otherwise unrecognized infarcts. Dobutamine stress CMR has a greater sensitivity and specificity than dobutamine stress echocardiography for detecting significant coronary artery disease (74% and 70% vs 86% and 86%) (16), and can be a particularly useful modality for stress testing in obese patients where images from a dobutamine stress echo may be poor quality and non-diagnostic. A multicenter trial comparing adenosine stress CMR to SPECT demonstrated that CMR is equivalent to, and may be superior to SPECT for the diagnosing CAD (17). The addition of late gadolinium enhanced imaging to CMR stress perfusion (Figure 3) increases specificity and accuracy of diagnosing coronary artery disease (18). When combining LGE and stress perfusion, the absence of LGE or a reversible perfusion defect is associated with a 98.1% negative annual event rate for death or MI, and the presence of LGE alone in patients without a history of prior MI was associated with an 11-fold increase in hazard ratio for death or MI (19). In patients with known or suspected CAD, the three-year event-free survival for patients who have a normal adenosine stress perfusion CMR and dobutamine stress CMR is 99.2% (20). In a larger cohort of over 1400 patients, dobutamine stress CMR alone demonstrating the absence of inducible wall motion abnormality in patients with low or intermediate risk of CAD was associated with a 99.5% event-free survival (21). Amongst patients with and LVEF 40-55%, an increase in dobutamine CMR wall motion score index was a predictor of MI and cardiac death (22).

Adenosine stress perfusion CMR has also been shown to be safe early after acute MI, and is more sensitive and specific than exercise treadmill testing (ETT) in predicting ≥70% coronary artery stenoses (23). The major limitation of CMR stress testing has been the inability to feasibly combine ETT with CMR due to the challenge of obtaining post-stress images in a similar position to rest images within a short time frame after exercise.

However, a pilot study incorporating ETT and CMR with use of an inflatable cushion mold to “recall” the patient's position in the MRI scanner minimizes the delay of post-stress imaging to under a minute, and may make exercise stress CMR a clinically viable test in the future (24).

Adenosine CMR is a cost-effective method for evaluating patients presenting to the emergency department for evaluation of chest pain without evidence of acute MI (25). A negative perfusion CMR study in this population has a negative predictive values of 100% for a diagnosis of CAD or adverse cardiovascular outcome at 1 year follow-up (26). There is also an emerging role for CMR in the evaluation of patients presenting with elevated troponin and either a low likelihood of acute coronary syndrome (ACS) or angiographically normal or non-obstructive coronary artery disease. Although the possible etiologies for troponin elevation is broad, the most likely diagnoses in patients with non-obstructed coronary arteries remain acute myocardial infarction due to spontaneous reperfusion of thrombus (27), myocarditis, and cardiomyopathy, especially Takotsubo or stress cardiomyopathy (28). The latter diagnosis may be made by CMR with T₂-W images positive for edema and the absence of LGE.

A pilot study in patients with elevated troponin and low clinical likelihood of ACS which employed a stepwise CMR algorithm used to rule out aortic dissection, acute MI, pulmonary embolus, or myopericarditis was able to yield a diagnosis in 27 of 29 patients (29). More importantly, the average scan time was 35± 8 minutes, which makes this feasible as an initial test to further risk stratify such patients. While the cost-effectiveness of such an approach needs to be investigated in larger study populations, the ability of CMR to correctly identify patients with ACS who should undergo angiography as well as prevent potentially unnecessary angiography in patients with non-coronary etiology of troponin elevation is promising.

Cardiomyopathies

Although a small group of patients with dilated cardiomyopathy (DCM) can have subendocardial or transmural LGE similar to patients with coronary artery disease, it is more common to have either no LGE or LGE in a mid-myocardial distribution (30). This can be a useful tool to distinguish ischemic from non-ischemic etiologies of cardiomyopathy, as those with ischemic cardiomyopathy will usually have LGE in a coronary distribution that is either subendocardial or transmural, but not mid-myocardial (30). In several prospective cohorts of patients with DCM, LGE predicted adverse cardiovascular outcomes and episodes of sudden cardiac death (SCD) and ventricular tachycardia, independent of adverse LV remodeling (31, 32).

While an echocardiogram is frequently the initial screening test for hypertrophic cardiomyopathy (HCM), CMR can be useful in cases of suspected apical variant HCM, as the apex can be difficult to adequately visualize by echo due to foreshortening or suboptimal windows (33). CMR can also identify cases of extreme wall thickness (>3 cm) where echo had underestimated the degree of hypertrophy, particularly in the anterolateral wall (34,35). Because LV wall thickness >3 cm is an established risk factor SCD (36), obtaining maximal wall thickness in HCM patients is important when considering ICD placement for primary prophylaxis. Typically, the pattern of LGE in HCM is that of focal or patchy enhancement in the mid-myocardium, most common in those segments with the greatest LV wall thickness (35) and at the RV insertion sites. It is, however, important to distinguish left ventricular hypertrophy (LVH) due to HCM from other disease states associated with LVH such as Anderson-Fabry disease, because such a diagnosis may lead to treatment with α -galactosidase enzyme replacement therapy. The LGE pattern of Anderson-Fabry disease is

focal, typically endocardial-sparing with a high predelection for the basal inferolateral wall (37).

Several studies have demonstrated an association between LGE and ventricular arrhythmias on Holter monitor (38), appropriate ICD discharge (39), and cardiac mortality in otherwise low-risk or asymptomatic HCM population (40). However, larger registries of HCM patients are needed to determine whether screening for specific patterns of LGE or quantifying LGE may improve the current guidelines for assessing risk of SCD (41).

Although CMR is able to identify areas of fat and fibrosis, the ability to reliably detect these features within the right ventricle is challenging even in experienced CMR centers (42), likely due to the presence of epicardial fat and the thin-walled RV. The finding of fat, however, is a non-specific finding and thus, there are no major or minor ARVC Task Force criteria based on CMR findings of fatty infiltration or DE of the RV. The most recent proposed modification of the Task Force Criteria incorporates one major and one minor criterion by CMR that may be met based on abnormal regional RV function with either abnormal global RV function or size (Table 2), (43). Routine calculation of the RV ejection fraction should be part of the assessment for ARVC, as a qualitative assessment of RV function may incorrectly classify patients with RV dysfunction as normal (44).

CMR can be a useful tool in establishing a diagnosis of cardiac amyloidosis. Characteristic DE patterns have been present in 69-97% (45, 46) of biopsy-proven cases. In one series, 100% of histologically confirmed cases of senile amyloid were detected by CMR (45). One of the hallmark findings of amyloidosis is the inability to find an inversion time to null the myocardium when preparing to obtain LGE images. This is due to diffuse infiltration of the myocardium with amyloid protein, resulting in delayed washout of gadolinium in this increased intracellular space. Difficulty nulling the myocardium can also occur in other conditions associated with diffuse LGE such as systemic sclerosis (47,48) and after orthotopic heart transplant (49).

The most common patterns of LGE in amyloidosis are either global subendocardial or global transmural, however, patchy LGE may also be seen (45). Other features of amyloidosis, such as biventricular dysfunction, increased wall thickness, biatrial enlargement, and pericardial effusion are also readily detected by CMR. Among patients with systemic amyloidosis, an abnormal CMR (defined as inability to null myocardium with an inversion time >300 ms) was associated with significantly decreased survival at 2 years compared to those with a negative CMR (14% vs 77% survival), (50). However, congestive heart failure was the only independent predictor of survival by multivariate analysis. Gadolinium kinetics, rather than the presence of LGE predicts prognosis, as a difference between T1 in the subepicardium and subendocardium of <23 ms measured 2 minutes after gadolinium administration is associated with decreased survival (51).

MRI is a useful way to non-invasively assess iron content of organs in disease states where iron deposition can occur and result in cardiomyopathy, such as hemochromatosis and thalassemia. Attenuation of signal intensity in the liver relative to skeletal muscle on spin-echo and gradient-recalled-echo (GRE) MR imaging correlates with increased hepatic iron (52,53). Newer GRE sequences have been developed that are able to measure myocardial T2*, which reflects differences in relaxation in tissues with iron deposition due to local inhomogeneity of magnetic fields (54,55). A T2* value of <20 ms is associated with reduced LV function (54). Cardiac MR can also be used to serially follow patients with thalassemia major to assess effectiveness of chelation therapies in decreasing myocardial iron content and improving LV function (56, 57).

The prevalence of cardiac sarcoidosis is 27% based on autopsy findings (58). The yield of endomyocardial biopsy for the diagnosis of cardiac sarcoidosis is low, likely due to the focal nature of involvement (59). Cardiac involvement of sarcoidosis by CMR may demonstrate areas of thinning, segmental wall motion abnormalities, focal areas of myocardial edema, and nodular mid-myocardial or sub-epicardial LGE (Figure 4). LGE is most commonly seen in basal and lateral segments (60). When compared to established Japanese Ministry Criteria, the presence of LGE identifies twice as many biopsy-proven cases of cardiac sarcoid when compared to established Japanese Ministry Criteria (61). As the characteristics of cardiac lesions appear to mirror systemic disease activity, CMR may be a non-invasive method for following success of corticosteroid therapy (62). In a cohort of patients with biopsy proven extracardiac sarcoidosis, those who had LGE had a higher rate of cardiac death compared with those who did not (61).

The superior spatial resolution of CMR as compared to echocardiography makes it an excellent technique for diagnosing cardiomyopathies that depend on accurate measurements of left ventricular wall thickness and identification of abnormal trabeculation, as in left ventricular non-compaction (LVNC). Thought to arise from cessation of the normal process of myocardial compaction early in-utero, the characteristic abnormal finding is a layer of abnormal, sponge-like trabeculation on top of a thin compacted layer of myocardium (63). The advantages of CMR over echocardiography in the evaluation of LVNC is the ability to analyze all myocardial segments by CMR, and the potential for underestimating the NC/C myocardium at end-systole by echo (64). By CMR, non-compaction is most prominent in the apical and lateral walls (65). Accepted echocardiographic criteria for LVNC include findings of a large network of non-compacted myocardium with confirmation of crypt-like recesses by color Doppler, and an end-systolic ratio of non-compacted to compacted myocardium of >2 (66). Specific CMR criteria have been proposed, which include a NC/C of >2.3 in diastole (65), and trabeculated LV mass $>20\%$ of total LV mass (67).

Myocarditis

In cases of possible myocarditis, CMR is an excellent test to obtain functional information as well as provide tissue characterization to aid in diagnosis. Areas of inflammation and edema will have either focal or diffuse increase in T_2 signal relative to areas of normal myocardium or skeletal muscle. Early after gadolinium administration, global enhancement of the myocardium may be visualized on steady-state free precession (SSFP) or fast-spin-echo T_1 -W images. The negative predictive value for acute myocarditis has been reported to be 100% for patients without areas of edema seen on T_2 -W images or early-post contrast global enhancement (68). The Lake Louise Consensus Criteria for diagnosing myocarditis by CMR require at least two of the following three conditions be met: 1) regional or global increase in signal intensity on T_2 -W images, 2) increased global myocardial early gadolinium enhancement ratio between myocardium and skeletal muscle in gadolinium-enhanced T_1 -W images, 3) at least one focal lesion with non-ischemic regional distribution on LGE images (69). Repeat imaging is recommended in 1-2 weeks after presentation if there is a high clinical suspicion but a negative or inconclusive initial CMR study (69). LGE is most commonly seen in the posterolateral wall, and may be either subepicardial or mid-myocardial (70), (Figure 5). In the convalescent phase, the majority of patients will have areas of persistent LGE which are smaller than in the acute phase due to healing and retraction of scar (71).

Pericardial diseases

CMR can offer both anatomical and functional information to aid in the diagnosis of pericardial diseases. The pericardium can be clearly visualized by CMR on black-blood and SSFP images, and is generally considered to be thickened if it measures ≥ 4 mm. Findings

on CMR suggestive of constrictive pericarditis include a dilated IVC, atrial enlargement, and pleural or pericardial effusions. Diastolic septal flattening by CMR has a sensitivity of 81% and specificity of 100% for constrictive pericarditis amongst patients with suspected disease (72). Obtaining a real-time cine image in a short axis, mid-ventricular plane is optimal to assess for ventricular interdependence, as diaphragmatic excursion during the respiratory cycle is clearly visualized in this view. In addition to cine images (Figure 6), myocardial tagging can identify areas of focal adhesion of the pericardium to myocardium (73). Areas of LGE in constrictive pericarditis correlate histologically with pericardial fibrosis (74). CMR can also be helpful in differentiating between constrictive pericarditis and restrictive cardiomyopathy, with a relative atrial volume ratio (left atrial volume/right atrial volume) cutoff of >1.32 yielding a sensitivity and specificity of 83% and 86% respectively for the diagnosis of constrictive pericarditis (75).

In the setting of acute pericarditis, the pericardium can appear bright on T_2 -W images, consistent with inflammation and corresponding diffuse LGE of the pericardium can be seen (76). While clinical assessment and echocardiography remain the mainstays of diagnosing pericardial tamponade, CMR can demonstrate findings of right atrial or right ventricular collapse, as well as the presence of a loculated effusion. CMR also can be useful in characterizing the size and location of pericardial cysts and diagnosing congenital absence of the pericardium (77).

Intracardiac Masses

CMR is able to characterize cardiac masses based on tissue composition and appearance on specific sequences (Table 3). Generally, tumors will demonstrate perfusion on first-pass perfusion images. Comparing pre-contrast and early post-contrast T_1 intensity of a mass is also a helpful tool, as most tumors will demonstrate an increase in signal intensity relative to pre-contrast T_1 images. Metastases to the heart can also be detected, appearing in the form of pericardial thickening or nodularity, pericardial effusion, or direct involvement of the myocardium. While breast cancer, lung cancer, and lymphoma are the most prevalent malignancies with cardiac involvement, melanoma has the greatest affinity for cardiac metastasis (78,79). For suspected cases of pericardial involvement, myocardial tagging is often a useful sequence for localizing areas of adherence to the myocardium.

When compared to non-contrast and contrast echocardiography, CMR is more sensitive in detecting LV thrombi, particularly those that are small or mural (80-82). It is important to realize that thrombus will have different CMR signal characteristics depending on the pulse sequence. In a black blood image, thrombus may appear slightly more intense or isointense relative to myocardium, while on cine-CMR it will appear as a hypointense filling defect relative to the blood pool. Repeating cine images that are suspicious for thrombus after contrast administration may improve resolution between suspected filling defects in the LV cavity and the blood pool. Typically, thrombus on DE imaging appears black, characteristic of avascular tissue (Figure 1). However, the appearance of thrombus on DE images may vary depending on age of thrombus, as organized thrombus occasionally takes up contrast (83,84), which can produce a heterogeneous pattern on late enhanced images. Using a long T_1 (600 ms) to prepare T_1 -W post-contrast images can also better delineate thrombus from myocardium. Thrombus will appear dark, as this longer T_1 surpasses the inversion time needed to null normal myocardium, which will appear gray (80).

LGE CMR imaging has been shown to be superior to cine MR as well as dark blood haste imaging (81,85) in the detection of left ventricular thrombus. In one study of patients with LV systolic dysfunction undergoing CMR primarily for the assessment of viability or etiology of cardiomyopathy, the overall prevalence of LV thrombus detected by LGE was

7% (85). Thrombus was more likely to be present in patients with ischemic cardiomyopathy, more severe LV dysfunction, and increased scar burden detected by LGE.

Valvular Heart Disease

While echocardiography still remains the accepted non-invasive modality for anatomic and functional evaluation of valvular heart disease, CMR can also be a useful method to evaluate severity of valvular disease as well as the hemodynamic effects of the lesion on chamber sizes and ventricular function (Figure 7). Through-plane velocity encoded imaging can provide information regarding forward and regurgitant flow in addition to peak velocity across a valve plane. In-plane velocity-encoded imaging can help localize the site of maximal stenosis in more challenging cases such as discrete subvalvular aortic stenosis. Criteria for grading aortic and mitral insufficiency by CMR have been proposed by correlating echocardiographic parameters with CMR regurgitant volume as follows: mild <15%, moderate 16-25%, moderate-severe 26-48%, and severe >48% (86). Assessment of aortic and mitral insufficiency by CMR can be particularly helpful in cases where the regurgitant jet is eccentric, and severity underestimated by echocardiography. Planimetry of mitral valve area by CMR correlates well with calculations by catheterization (Gorlin formula) and echocardiography (pressure-half time method), however, CMR tends to overestimate valve area by 5-8% (87). Grading severity of aortic stenosis by CMR can be performed by continuity equation similar to echocardiography, as well as determining peak and mean gradient (88). Due to artifact and resultant errors in velocity encoding, CMR is not optimal for assessing hemodynamics of mechanical valve prostheses (Figure 8).

Electrophysiology Applications

CMR has an integral role in guiding ablation therapies. MR angiography of the left atrium is helpful to operators when planning where to deliver ablation lesions, as variations in pulmonary vein anatomy are common, with only 40% of patients having typical anatomy of two separate right sided and left sided veins (89). Common variants include a common ostium for the left sided veins, and the presence of a right middle pulmonary vein. Electroanatomic mapping merged with 3D MRA data reduces fluoroscopy time for the procedure (90) and may also improve outcomes (91). Although measurement of left atrial volume by CMR is reproducible and is a predictor of successful atrial fibrillation ablation (92), quantification of left atrial scarring by DE pre-ablation has become a useful tool in helping to select optimal patients for ablation therapy, as those with more left atrial fibrosis have a greater chance of recurrence (93). More recently, degree of left atrial fibrosis has been shown to be an independent predictor of stroke, and might be used in the future along with traditional clinical predictors (CHAD score) to provide incremental risk stratification for stroke in patients with atrial fibrillation (94).

Given the observation that up to one-third of patients with severe LV dysfunction do not have symptomatic improvement with cardiac resynchronization therapy (95), additional information provided by CMR can help to select candidates likely to derive the most benefit. A combination of DE MR with assessment of dyssynchrony by myocardial tagging has a positive predictive value and negative predictive values of 93% and 100% respectively for predicting those who will have an improvement in functional class after CRT implantation (96).

Congenital Heart Disease

The anatomic imaging of congenital lesions is inherently complex, and can pose a challenge in many patients for whom echocardiography yields suboptimal images. CMR can be viewed as complementary to echocardiography in patients with complex congenital heart disease and is superior at imaging extracardiac anatomy and the great vessels. There are

several adult congenital populations in whom CMR can yield quantitative information to guide clinical decision making. The timing of pulmonic valve replacement for patients with repaired Tetralogy of Fallot who have severe pulmonic insufficiency is controversial. However, prospective studies propose a cutoff of RV volume indexed to BSA of between 150 mL/m²-160 mL/m² (97,98) for pulmonic valve replacement, as those with RV volumes above these values are less likely to normalize RV volumes post-op (Figure 9). With the possibility of approved percutaneous options for pulmonic valve replacement on the horizon (99), CMR can be used to measure RVOT dimensions for sizing of prostheses.

CMR combined with clinical assessment in patients without prior screening test has been proposed as a cost-effective method for detecting late complications after repair of coarctation of the aorta (100). In one retrospective analysis of patients with coarctation repair performed less than 2 years of age undergoing CMR for routine surveillance and clinical indications, re-coarctation of the aorta was seen in 68% of patients (101). Furthermore, patients with previous coarctation repair who have a bicuspid aortic valve have significantly larger measurements of the aortic root when compared to patients with tricuspid aortic valves (101, 102). CMR is a comprehensive test for this select group of patients, as it can demonstrate complications at the site of coarctation repair, assess aortic valve morphology (Figure 10) and function, and screen for the aortopathy that is associated with bicuspid aortic valves (1).

Conclusions

Cardiovascular magnetic resonance is a versatile tool to guide diagnosis and treatment of a wide range of cardiovascular diseases. The cornerstone of current CMR practice remains the assessment of viability, ventricular function, and myocardial tissue characterization. As the importance of information gleaned from CMR is increasingly recognized, CMR will become increasingly invaluable to patient care.

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Figure 1. Varying degrees of myocardial infarction transmuralities detected by CMR. (A) LGE image of subendocardial inferior wall infarct and apical thrombus. (B) PSIR LGE-image of an infarction in the inferior wall of intermediate transmuralities and a transmural infarction of the apex and anterior septum. PSIR=Phase-sensitive inversion recovery. DE=delayed enhancement.

Figure 2.

Sequelae of myocardial infarction. (A) Microvascular obstruction within a transmural infarction of the lateral wall seen by PSIR-LGE imaging. (B) Two ventricular septal defects within the apical septum shown on SSFP image. (C) Pseudoaneurysm of the anterior wall with thrombus, and transmural apical infarction by PSIR-LGE imaging.

Figure 3. Basal short axis rest perfusion image demonstrating perfusion defect in the inferolateral wall (A) and matching region of delayed enhancement on PSIR image (B) representing infarct with 50% transmural.

Figure 4. Sarcoidosis. PSIR-DE 4-chamber long-axis image demonstrating patchy, nodular LGE of the septum and lateral wall, and near-transmural apical LGE. LGE is also present in the RV free wall.

Figure 5. Myocarditis. (A) Increased signal in the sub-epicardium and mid-myocardium of the anterolateral wall by T₂-W imaging representing myocardial edema, with corresponding areas of delayed enhancement on PSIR-LGE image (B).

Figure 6. SSFP axial image in diastole (A) and systole (B) showing a large complex pericardial effusion adjacent to the right ventricle, and tethering of the pericardium to the distal RV free wall (arrows).

Figure 7. Axial SSFP image demonstrating ascending aortic aneurysm and moderate aortic insufficiency in a patient with Marfan's syndrome and previous aortic aneurysm repair.

Figure 8. Three-chamber SSFP image of mechanical St. Jude aortic valve shown in diastole (A), and systole (B), with significant artifact in the aortic root due to valve opening.

Figure 9.

Comprehensive CMR evaluation of adult with Tetralogy of Fallot. SSFP axial image demonstrating severe RV dilatation (A) with indexed RV end-diastolic volume of 302 mL/m², GRE RVOT image showing an dilated RVOT and severe pulmonic insufficiency (B), flow vs. time curve (C) generated from a velocity-encoded short-axis pulmonic valve image, also consistent with severe pulmonic insufficiency.

Figure 10.
SSFP image of a bicuspid aortic valve in systole, demonstrating fusion of non and right coronary cusps.

Table 1

Summary of appropriate indications for CMR (1).

Indications for CMR
Assessment of ventricular function and myocardial viability in heart failure
Assessment of infiltrative cardiomyopathies, hypertrophic cardiomyopathy, ARVC
Patients with CAD or heart failure when echocardiography does not provide accurate assessment of cardiac structure and function.
CMR stress testing for patients with intermediate probability of CAD and/or unable to exercise, or in patients with known coronary stenosis of unclear functional significance
Determine myocardial viability prior to revascularization
Evaluation of pericardial mass or constrictive pericarditis
Evaluation of left atrial and pulmonary vein anatomy in planning of atrial fibrillation ablation
Assessment of coarctation of the aorta or previous coarctation repair, pulmonic stenosis, congenital coronary anomalies, complex congenital heart disease
Characterization of cardiac mass or possible thrombus
Assessment of ascending aorta in patients with dilated aortic root or if the aorta is poorly visualized by echocardiography

Table 2

CMR criteria for ARVC, adapted from Revised Task Force Criteria for ARVC (36). A diagnosis of ARVC requires 2 major, 1 major plus 2 minor criteria, or 4 minor criteria from different categories.

Major	Minor
Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and 1 of the following: <ul style="list-style-type: none"> - Ratio of RV end-diastolic volume to BSA ≥ 110 mL/m² (male) or ≥ 100 mL/m² (female) - RV ejection fraction $\leq 40\%$. 	Regional RV akinesia or dyskinesia or dyssynchronous RV contraction and 1 of the following: <ul style="list-style-type: none"> - Ratio of RV end-diastolic volume to BSA ≥ 100 to <110 mL/m² (male) or ≥ 90 to <100 mL/m² (female) - RV ejection fraction $>40\%$ to $\leq 45\%$

Table 3

Tissue characterization of cardiac masses by CMR

Mass	T1-W	T2-W	Delayed enhancement	Other
Lipoma	Increased signal	Low signal	None	Decreased intensity with fat saturation; may appear as lipomatous hypertrophy of the intraatrial septum, sparing the fossa ovalis
Myxoma	Varying signal intensity	High signal	Heterogeneous enhancement	Appear dark on gradient-echo sequences. Most commonly seen in left atrium attached to septum with thin stalk. Carney's complex (autosomal dominant) LA and LV myxomas.
Rhabdomyoma	Increased or isointense	High Signal	Mild enhancement	Associated with tuberous sclerosis, most commonly seen in ventricle.
Fibroma	Hypointense	Isointense to skeletal muscle	Heterogeneous, Homogeneous, or no enhancement	May be calcified, intramural. Usually ventricular location.
Angiosarcoma	Varying signal intensity	Varying signal intensity	Heterogeneous enhancement	Most commonly seen right atrium. May appear as abnormal thickening of right atrial wall.