Published in final edited form as:

Curr Treat Options Cardiovasc Med. 2013 August ; 15(4): 373–386. doi:10.1007/s11936-013-0253-6.

The Role of Cardiovascular Magnetic Resonance (CMR) and Computed Tomography (CCT) in Facilitating Heart Failure Management

R. Brandon Stacey, MD, MS¹ and W. Gregory Hundley, MD^{1,2}

¹Department of Internal Medicine (Section on Cardiology) at the Wake Forest University School of Medicine, Winston-Salem, North Carolina, 27157, USA

²Department of Radiology at the Wake Forest University School of Medicine, Winston-Salem, North Carolina, 27157, USA

Opinion Statement

Cardiovascular magnetic resonance (CMR) and cardiac computed tomography (CCT) offer advantages for detecting left or right ventricular dysfunction in patients with or suspected of heart failure. CMR does not expose patients to ionizing radiation, and thus is well-suited for functional assessments and serial studies. CCT provides high spatial resolution, making it useful for the identification of coronary arteriosclerosis associated with ischemic cardiomyopathy. In this review, the clinical applications of CMR and CCT are individually discussed, with comparisons made between them to examine the strengths of each modality. The major techniques for each modality are outlined, as well as their uses for the evaluation of cardiomyopathy in heart failure patients with reduced left ventricular ejection fraction, preserved left ventricular ejection fraction, and valvular heart disease. Finally, we review the utility of CMR and CCT in determining which patients will benefit from cardiac resynchronization therapy.

Keywords

Cardiovascular Magnetic Resonance; Cardiac Computed Tomography; Heart Failure; Cardiomyopathy; Valvular Heart Disease

Introduction

Heart failure, a clinical syndrome that results from a structural or functional cardiac disorder which prevents the ventricles from either filling with or ejecting blood, represents the most common Medicare diagnosis-related group in individuals over the age of 65 years [1].

Dyspnea and fatigue limit exercise tolerance and fluid retention, which promotes pulmonary congestion or peripheral edema, and represent the primary manifestations that impair the functional capacity as well as the quality of life of those with heart failure [2].

Cardiovascular magnetic resonance (CMR) and cardiac computed tomography (CCT) have

Address for Correspondence: W. Gregory Hundley, M.D. Cardiology Section Watlington Hall Wake Forest University School of Medicine Medical Center Boulevard Winston-Salem, North Carolina 27157-1045 Phone: (336) 716-6125 Fax: (336) 713-9188 ghundley@wakehealth.edu.

Compliance with Ethics Guidelines

Conflict of Interest Dr. Richard Stacey reported no potential conflicts of interest relevant to this article.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

recently emerged as tomographic imaging modalities with high utility for assessing patients with or suspected of heart failure [3]. The ability of these modalities to collect information related to the etiology of left ventricular (LV) dysfunction facilitates the diagnosis and management of patients with heart failure [3]. In this article, we review the clinical applications of CMR and CCT for the assessment and evaluation of LV dysfunction in patients with heart failure.

Techniques

Cardiovascular Magnetic Resonance (CMR)

Cardiovascular magnetic resonance is a versatile imaging modality. In one examination, one can assess an organ's structure, function, perfusion, or metabolism. All of these assessments can be accomplished without an interventional procedure, the administration of intravenous contrast materials, or exposure to ionizing radiation. Each of these aspects of CMR is particularly useful for assessing patients with heart failure.

Determination of cardiac structure is useful for classifying types of heart failure, whether they be related to a dilated or hypertrophic cardiomyopathy. Most commonly, identifying cardiac structure is accomplished using black-blood imaging, a technique that defines cardiac and vascular anatomy by nulling the signal from the blood to create a dark chamber cavity (Figure 1) [4]. Black-blood imaging may be weighted more toward T1 or T2 recovery [5]. T1-weighted black blood imaging displays fat as a higher signal, which can be useful for identifying fat infiltration of the right ventricular (RV) free wall in patients suspected of arrhythmogenic RV cardiomyopathy. T2-weighted black blood imaging can be used to identify acute myocardial edema that is present in myocardial inflammation associated with infarction or myocarditis (Figure 1). On these images, excess myocardial water is displayed as higher signal intensity.

In addition to assessing cardiac structure, CMR can be used to assess cardiac function. This is accomplished using cine white blood imaging techniques in which the motion of the blood through the heart provides contrast with the cavity and endocardial surface (Figure 2). In general, one of two imaging sequences is used: spoiled gradient echo (GRE) imaging or steady state free precession (SSFP) imaging [6]. In SSFP images, only fluid and fat have high signals, which improves the delineation of the endocardial surface [7]. The only drawback of SSFP relative to GRE is that turbulent flow may not be well visualized [3]. Inability to visualize turbulent flow becomes problematic when patients with heart failure exhibit abnormal flow vortices associated with valvular stenosis or regurgitation [3]. When these clinical situations are suspected, the GRE sequence is often implemented.

Left ventricular systolic and diastolic function can be readily assessed using these white blood imaging methods. Using 3-dimensional acquisitions and incorporating Simpson's rule analysis techniques, CMR can assess end-diastolic and end-systolic LV volumes with high precision [8], and from these volume measurements, the LV ejection fraction (LVEF) can be calculated. When compared to 2-dimensional echocardiographic methods, the accuracy of CMR allows for 2- to 7-fold reductions in sample size estimates when LV volumes, EF, and mass are planned as primary outcomes in clinical trials [9].

Right heart chamber sizes, volumes, EF, and regional wall motion abnormalities can also be assessed with CMR. Both axial and sagittal cine image acquisitions of the right heart are useful for excluding major and minor criteria associated with RV cardiomyopathy (Figure 3).

In addition to “global” measures of left or right ventricular volumes and EF, CMR can quantify regional changes in cardiac function including measures of myocardial strain. Strain analysis can be performed with CMR using several approaches [10-16]. Traditionally, many of these methods have incorporated tissue tagging, but more recently, feature tracking techniques have been developed that enable measurement of myocardial strain without needing to acquire additional images with tags (which increases the duration of the procedure) [17]. Additionally, LV diastolic function can be measured using assessments of strain rate [18].

Phase contrast GRE imaging is a form of GRE that is used to evaluate blood flow [19]. It involves creating a gradient, which induces a phase shift that is proportional to the velocity in the direction of the gradient [20]. On these images, the gray scale of the pixels in the image encodes for velocity and flow can be calculated by multiplying mean velocity by the area [20]. This technique is very accurate in measuring blood flow including cardiac and valvular output, regurgitant volume and regurgitant fraction [21].

CMR techniques are available to assess myocardial perfusion [22]. When implemented during pharmacologic stress, these perfusion methods can readily identify myocardial ischemia. Most perfusion techniques coordinate image acquisition around the first pass of gadolinium contrast through the heart. As contrast arrives within the LV myocardium, a decrease in contrast relative to the remaining myocardium causes a low signal intensity to occur on the image and indicates inducible ischemia that most often is associated with an epicardial coronary arterial luminal narrowing of >50% (Figure 4).

In addition to first-pass gadolinium stress perfusion techniques to identify ischemia, the LV myocardium can be imaged 10 minutes after contrast using a technique termed delayed enhancement imaging [23]. In this technique, scar tissue or areas of inflammation retain gadolinium within the extra-myocellular space. The retained gadolinium reduces the T1 (longitudinal) relaxation time such that on gradient echo inversion recovery images, healthy myocardium appears black and fibrosis related to prior myocardial infarction (MI) or residual viral infection appears white. As a result, this technique is useful for identifying prior infarcts in the setting of ischemic heart disease (Figure 5A).

Cardiac Computed Tomography

Over the past ten years, the implementation of gating algorithms has enabled the acquisition of high spatial resolution images of the heart. These high resolution images allow for the precise definition of coronary arterial anatomy including the determination of coronary arterial plaque presence, type, and severity of intraluminal obstruction [24-28]. The precision by which abnormalities of coronary artery anatomy can be defined facilitates identification of coronary arteriosclerosis that is associated with ischemic cardiomyopathy (Figure 6).

CCT can also be used to assess cardiac perfusion and delayed enhancement [29, 30]. Semi-quantitative per-segment analysis can be performed by simultaneous comparison of short-axis images obtained before and after intravenous vasodilator stress. To assess delayed enhancement with CCT, non-contrasted images are obtained ten minutes after a large contrast bolus (100-150 mL) or can be performed seven minutes after resting perfusion images.

Left ventricular volumes and systolic function are estimated with CCT using the Simpson's method [31, 32]. Left or right ventricular volumes may be measured by assessing the Hounsfield units to accurately delineate the LV end-diastolic and end-systolic volume. This technique has the added advantage of quickly and reliably separating the blood pool from

the papillary muscles. Cardiovascular CT derived measures of LV volumes and EF correlate well with CMR ($r = 0.97$) [31]; also, these techniques can be used to measure RV size and systolic function [31]. There are two limitations to measuring LV or RV volumes and EF with CCT. First, the temporal resolution of the images (~70 ms) may be insufficient to identify end systole. Second, since the methods utilize retrospective gating, the exposure to ionizing radiation increases.

The Evaluation of Cardiomyopathy

Heart Failure with a Reduced Left Ventricular Ejection Fraction

In those with reduced LVEF, it is important to identify ischemic heart disease. To this end, CMR and CCT often accomplish this identification using different processes. With CMR, the most common method is through the performance of a pharmacologic stress test with either a vasodilator (adenosine, regadenoson, or persantine) or a positive inotrope such as dobutamine [33]. Using either pharmacologic infusion protocol (vasodilator or inotrope), LV wall motion, stress and rest myocardial perfusion, and delayed gadolinium enhanced images are collected. In recent meta-analyses as well as large cohort studies of 500 to 1500 participants, the overall sensitivity and specificity of these stress modalities range from 83% to 95% for detecting >50% coronary arterial luminal narrowings [34].

In addition to diagnosing ischemia, CMR stress results can provide accurate estimates of cardiovascular prognosis [35, 36], and forecast the results of technically successful coronary artery revascularization procedures [37]. As an example, Steel, et al. demonstrated the prognostic utility of stress CMR myocardial perfusion and late gadolinium enhancement in 254 patients. The absence of a CMR-related perfusion defect or evidence of late gadolinium enhancement conferred a 98.1% negative annual event rate for cardiac death and MI within 17 months after the stress procedure [38]. In patients with chronic ischemic heart disease, those exhibiting greater than 50% transmural hyper-enhancement with late gadolinium imaging exhibit little evidence of recovery of systolic thickening after coronary arterial revascularization procedures [37].

Non-ischemic Cardiomyopathy

Over the last seven years, the role of CMR in evaluating patients with cardiomyopathies and heart failure with reduced LVEF has been expanded through the use of both delayed enhancement as well as T2 and T2* techniques for characterizing myocardial tissue. Using these tissue characterization capabilities, CMR is able to identify abnormalities of these tissue characteristics which are either highly suggestive or diagnostic of various etiologies of LV dysfunction.

For example, using the delayed enhancement technique, patients with evidence of myocarditis will demonstrate delayed enhancement that occurs along the epicardial surface of the LV myocardium in the regions of the visceral pericardium [39-41]. These epicardial patches of delayed hyper-enhancement (Figure 5D) help to also identify the cause of chest pain in patients presenting with LV dysfunction and a history consistent with myocarditis. Moreover, utilizing either T2-weighted images or T2 mapping processes, one can identify evidence of increased myocardial water associated with active inflammation and active myocarditis [41].

In those exposed to viral etiologies of their myocardial inflammation [41], delayed enhancement patterns consistent with mid-wall hyper-enhancement have also been appreciated in patients with dilated cardiomyopathies and residual fibrosis after treatment of their disease [41, 39]. Recent study results suggest that this mid-wall fibrosis that occurs

chronically after treatment for a viral infection may be associated with adverse cardiac prognosis.

Infiltrative processes associated with systemic amyloidosis or sarcoidosis also create patterns of delayed hyper-enhancement suggestive of the disease [42, 40, 43]. In patients with cardiac amyloid involvement, a characteristic sub-endocardial diffusely based delayed hyper-enhancement pattern is often appreciated (Figure 5F) [40]. In addition, identifying a point in which the myocardium can be appropriately “nulled” is difficult to achieve in patients with systemic amyloidosis. In individuals with sarcoid myocarditis, patchy accumulations of delayed enhancement are often observed, and in patients with active progressive sarcoid, these can continuously evolve over time (Figure 5E) [42, 43]. As shown in Figure 5, there are multiple patterns of delayed hyper-enhancement that can indicate the etiology of LV dysfunction in patients with non-ischemic cardiomyopathies [44].

It is important to note that in addition to delayed enhancement patterns, T2* evaluations are useful in assessing iron accumulation in patients with conditions such as hemochromatosis [45-47]. In this condition, a series of images is often collected in short axis views and the relationship between signal intensity and various echo times can produce a T2* value within the myocardium indicative of intramyocardial iron. Those individuals with T2* values less than 20 milliseconds often exhibit accumulations of myocardial iron warranting further therapy.

Cardiovascular MR also has the ability to provide detailed images of cardiac morphology. Another situation where CMR is useful is noncompaction cardiomyopathy (Figure 2) [48]. Several small studies have demonstrated that CMR can reliably demonstrate LV trabeculations more clearly than echocardiography [49]. In limited comparisons between echocardiography and CMR, several studies have suggested different criteria [50, 48]. However, more defined criteria are being investigated to avoid the confusion between imaging modalities. Cardiovascular CT can also image trabeculations, but due to increased radiation doses, most often only end-diastolic images are obtained. Cardiovascular MR can demonstrate both end-systolic and end-diastolic noncompacted:compacted ratios [51]. By accurately identifying patients with potential noncompaction cardiomyopathy, clinicians can make more informed decisions about whether anti-coagulation is appropriate (Figure 2).

Heart Failure and Preserved Left Ventricular Ejection Fraction

CMR provides important information in evaluating for potential hypertrophic cardiomyopathy. CMR can accurately assess wall thickness and location of asymmetric hypertrophy. This is particularly useful when assessing patients with the apical form of hypertrophic cardiomyopathy. In these situations, transthoracic echocardiography can often foreshorten the LV apex so that this condition would be missed. With CMR or CCT, identification of apical hypertrophic cardiomyopathy is readily obtained. A LV end-diastolic wall thickness of >15 mm or a LV end-diastolic wall thickness ratio of apical versus basal thickness >1.3 is diagnostic of the condition. When echocardiography is inconclusive, phase contrast imaging can accurately measure velocities to evaluate dynamic outflow tract obstruction [52]. Delayed enhancement can provide prognostic information related to risk of sudden cardiac death. In addition, it can indicate the presence of basal myocardial crypts and anterior mitral leaflet lengthening, two novel indicators to evaluate for subclinical disease [53, 54].

Valvular Heart Disease

Normally, aortic stenosis results in concentric hypertrophy, but in later stages, may result in a dilated cardiomyopathy. In situations where echocardiography is unable to assess aortic

stenosis, CMR with phase contrast imaging can be used to define aortic valve area and measure transvalvular gradient [55-58, 21].

Recently, the anatomic clarity of CCT has been used to facilitate management of patients with valvular heart disease. CCT can assess valve morphology, and measure aortic valve orifice area [59]. Increasingly, cardiac CT has an emerging role in the pre-procedural assessment for transcatheter aortic valve replacements [60]. To assess aortic regurgitation, CCT can measure aortic regurgitant orifice area [61], and further, it can provide LV volume assessments to help determine the significance of the valvular defect.

Cardiac Resynchronization Therapy

Once patients have developed severe LV dysfunction, a challenge related to their management involves determining which patients will benefit from cardiac resynchronization therapy. To date, echocardiographic measurements have not been overly helpful in identifying those individuals that will benefit above and beyond criteria obtained from 12-lead electrocardiography. Both CMR and CCT may have utility in this regard [62-64]. With either CMR or CCT, the identification of fibrotic scarred myocardium provides important information as to whether myocardial segments will improve contractility or develop synchronous contraction when a pacing lead is placed in close juxtaposition. Studies have indicated that placing pacing leads in regions with scarred myocardium will not facilitate the development of synchronous contraction [62, 64].

In addition, identifying coronary vein anatomy and correlating this coronary vein anatomy with regions of viable, non-fibrotic myocardium can be of use [65-67]. With CCT, images are obtained within seconds that provide accurate assessments of coronary venous anatomy that then can be co-registered with myocardial regions absent of scar to determine if coronary sinus lead placement will facilitate synchronous LV contraction (Figure 7) [65, 67].

Conclusion

Both CMR and CCT provide useful information to help identify the etiology of LV dysfunction in patients with congestive heart failure. Cardiovascular MR provides robust functional assessments and lends itself to serial studies due to the lack of exposing patients to ionizing radiation. Cardiovascular CT provides high spatial resolution images for visualizing coronary arteries, plaques and stenoses to help identify ischemic cardiomyopathy, but is limited in functional assessment due to increased radiation dose. Both CMR and CCT can be used to visualize scar tissue from prior infarctions and evaluate for inducible ischemia. The image clarity associated with CMR measures of delayed enhancement and both T1, T2, and T2* mapping provides a unique opportunity to characterize myocardial tissue and thereby establish the etiology of nonischemic causes of cardiomyopathy in individuals with heart failure and preserved or reduced LVEF. Functional CMR and anatomic CCT assessments can be useful when assessing patients with valvular heart disease. In those individuals under evaluation for cardiac resynchronization strategies, CCT or CMR can provide high-quality images of the coronary veins to help plan coronary sinus lead placement, and CMR may help further guide lead placement by direct visualization of the scarred myocardium by delayed enhancement imaging.

Acknowledgments

Research supported in part by National Institute of Health grants R33 CA121296-02, R01 HL076438-02, R01 CA167821-01, P30 AG21332 and P30 CA012197.

Dr. W. George Hundley reported receiving grants from NIH and Bracco (grants R33 CA121296-02, R01 HL076438-02, R01 CA167821-01, P30 AG21332 and P30 CA012197).

References

* Of importance

** Of great importance

1. Kitzman DW, Little WC, Brubaker PH, Anderson RT, Hundley WG, Marburger CT, et al. Pathophysiological characterization of isolated diastolic heart failure in comparison to systolic heart failure. *JAMA*. 2002; 288(17):2144–50. [PubMed: 12413374]
2. Hundley WG, Kitzman DW, Morgan TM, Hamilton CA, Darty SN, Stewart KP, et al. Cardiac cycle-dependent changes in aortic area and distensibility are reduced in older patients with isolated diastolic heart failure and correlate with exercise intolerance. *J Am Coll Cardiol*. 2001; 38(3):796–802. [PubMed: 11527636]
3. Dall'Armellina E, Morgan TM, Mandapaka S, Ntim W, Carr JJ, Hamilton CA, et al. Prediction of cardiac events in patients with reduced left ventricular ejection fraction with dobutamine cardiovascular magnetic resonance assessment of wall motion score index. *J Am Coll Cardiol*. 2008; 52(4):279–86. [PubMed: 18634983]
4. Lee, VS. *Cardiovascular MR Imaging: Physical Principles to Practical Protocols*. Lippincott Williams & Wilkins; 2006. Black-blood imaging; p. 274-82.
5. Lightfoot JC, D'Agostino RB Jr, Hamilton CA, Jordan J, Torti FM, Kock ND, et al. Novel approach to early detection of doxorubicin cardiotoxicity by gadolinium-enhanced cardiovascular magnetic resonance imaging in an experimental model. *Circ Cardiovasc Imaging*. 2010; 3(5):550–8. [PubMed: 20622140]
6. Lee, VS. *Cardiovascular MR Imaging: Physical Principles to Practical Protocols*. Lippincott Williams & Wilkins; 2006. Cine gradient echo imaging; p. 283-306.
- 7 **. Hundley WG, Bluemke DA, Finn JP, Flamm SD, Fogel MA, Friedrich MG, et al. ACCF/ACR/AHA/NASCI/SCMR 2010 expert consensus document on cardiovascular magnetic resonance: a report of the American College of Cardiology Foundation Task Force on Expert Consensus Documents. *J Am Coll Cardiol*. 2010; 55(23):2614–62. [PubMed: 20513610] This document represents a comprehensive review for clinical implementation of CMR.
8. Kuhl HP, Spuentrup E, Wall A, Franke A, Schroder J, Heussen N, et al. Assessment of myocardial function with interactive non-breath-hold real-time MR imaging: comparison with echocardiography and breath-hold Cine MR imaging. *Radiology*. 2004; 231(1):198–207. [PubMed: 14990805]
9. Bellenger NG, Davies LC, Francis JM, Coats AJ, Pennell DJ. Reduction in sample size for studies of remodeling in heart failure by the use of cardiovascular magnetic resonance. *J Cardiovasc Magn Reson*. 2000; 2(4):271–8. [PubMed: 11545126]
10. Kempny A, Fernandez-Jimenez R, Orwat S, Schuler P, Bunck AC, Maintz D, et al. Quantification of biventricular myocardial function using cardiac magnetic resonance feature tracking, endocardial border delineation and echocardiographic speckle tracking in patients with repaired tetralogy of Fallot and healthy controls. *J Cardiovasc Magn Reson*. 2012; 14:32. [PubMed: 22650308]
11. Li P, Meng H, Liu SZ, Vannan MA. Quantification of left ventricular mechanics using vector-velocity imaging, a novel feature tracking algorithm, applied to echocardiography and cardiac magnetic resonance imaging. *Chin Med J (Engl)*. 2012; 125(15):2719–27. [PubMed: 22931981]
12. Osman NF, Kerwin WS, McVeigh ER, Prince JL. Cardiac motion tracking using CINE harmonic phase (HARP) magnetic resonance imaging. *Magn Reson Med*. 1999; 42(6):1048–60. [PubMed: 10571926]
13. Pan L, Prince JL, Lima JA, Osman NF. Fast tracking of cardiac motion using 3D-HARP. *IEEE Trans Biomed Eng*. 2005; 52(8):1425–35. [PubMed: 16119238]
14. Diller GP, Radojevic J, Kempny A, Alonso-Gonzalez R, Emmanouil L, Orwat S, et al. Systemic right ventricular longitudinal strain is reduced in adults with transposition of the great arteries,

relates to subpulmonary ventricular function, and predicts adverse clinical outcome. *Am Heart J*. 2012; 163(5):859–66. [PubMed: 22607865]

15. Hor KN, Mazur W, Taylor MD, Al-Khalidi HR, Cripe LH, Jefferies JL, et al. Effects of steroids and angiotensin converting enzyme inhibition on circumferential strain in boys with Duchenne muscular dystrophy: a cross-sectional and longitudinal study utilizing cardiovascular magnetic resonance. *J Cardiovasc Magn Reson*. 2011; 13:60. [PubMed: 22011358]
16. Schuster A, Morton G, Hussain ST, Jogiya R, Kutty S, Asress KN, et al. The intra-observer reproducibility of cardiovascular magnetic resonance myocardial feature tracking strain assessment is independent of field strength. *Eur J Radiol*. 2013; 82(2):296–301. [PubMed: 23246014]
17. Korosoglou G, Futterer S, Humpert PM, Riedle N, Lossnitzer D, Hoerig B, et al. Strain-encoded cardiac MR during high-dose dobutamine stress testing: comparison to cine imaging and to myocardial tagging. *J Magn Reson Imaging*. 2009; 29(5):1053–61. [PubMed: 19388131]
18. Tandri H, Daya SK, Nasir K, Bomma C, Lima JA, Calkins H, et al. Normal reference values for the adult right ventricle by magnetic resonance imaging. *Am J Cardiol*. 2006; 98(12):1660–4. [PubMed: 17145230]
19. Lee, VS. *Cardiovascular MR Imaging: Physical Principles to Practical Protocols*. Lippincott Williams & Wilkins; 2006. Phase contrast flow quantification in the heart; p. 325–36.
20. Hundley WG, Li HF, Hillis LD, Meshack BM, Lange RA, Willard JE, et al. Quantitation of cardiac output with velocity-encoded, phase-difference magnetic resonance imaging. *Am J Cardiol*. 1995; 75(17):1250–5. [PubMed: 7778549]
21. Hundley WG, Li HF, Willard JE, Landau C, Lange RA, Meshack BM, et al. Magnetic resonance imaging assessment of the severity of mitral regurgitation. Comparison with invasive techniques. *Circulation*. 1995; 92(5):1151–8. [PubMed: 7648660]
22. Bernhardt P, Engels T, Levenson B, Haase K, Albrecht A, Strohm O. Prediction of necessity for coronary artery revascularization by adenosine contrast-enhanced magnetic resonance imaging. *Int J Cardiol*. 2006; 112(2):184–90. [PubMed: 16271778]
23. Lee, Vs. *Cardiovascular MR Imaging: Physical Principles to Practical Protocols*. Lippincott Williams & Wilkins; 2006. Tissue characterization.
24. Menke J, Unterberg-Buchwald C, Staab W, Sohns JM, Seif Amir Hosseini A, Schwarz A. Head-to-head comparison of prospectively triggered vs retrospectively gated coronary computed tomography angiography: Meta-analysis of diagnostic accuracy, image quality, and radiation dose. *Am Heart J*. 2013; 165(2):154–63. e3. [PubMed: 23351817]
25. Vorre MM, Abdulla J. Diagnostic Accuracy and Radiation Dose of CT Coronary Angiography in Atrial Fibrillation: Systematic Review and Meta-Analysis. *Radiology*. 2013; 267(2):376–86. [PubMed: 23382294]
26. Kim JS, Choo KS, Jeong DW, Chun KJ, Park YH, Song SG, et al. Step-and-shoot prospectively ECG-gated vs. retrospectively ECG-gated with tube current modulation coronary CT angiography using 128-slice MDCT patients with chest pain: diagnostic performance and radiation dose. *Acta Radiol*. 2011; 52(8):860–5. [PubMed: 21873509]
27. Martini C, Palumbo A, Maffei E, Rossi A, Rengo M, Malago R, et al. Dose reduction in spiral CT coronary angiography with dual-source equipment. Part I. A phantom study applying different prospective tube current modulation algorithms. *Radiol Med*. 2009; 114(7):1037–52. [PubMed: 19662339]
28. Yang CC, Mok GS, Law WY, Hsu SM, Wu TH, Chen LK. Potential dose reduction of optimal ECG-controlled tube current modulation for 256-slice CT coronary angiography. *Acad Radiol*. 2011; 18(6):731–7. [PubMed: 21420331]
29. Feuchtnner G, Goetti R, Plass A, Wieser M, Scheffel H, Wyss C, et al. Adenosine stress high-pitch 128-slice dual-source myocardial computed tomography perfusion for imaging of reversible myocardial ischemia: comparison with magnetic resonance imaging. *Circ Cardiovasc Imaging*. 2011; 4(5):540–9. [PubMed: 21862731]
30. Mendoza DD, Joshi SB, Weissman G, Taylor AJ, Weigold WG. Viability imaging by cardiac computed tomography. *J Cardiovasc Comput Tomogr*. 2010; 4(2):83–91. [PubMed: 20430338]

31. Raman SV, Shah M, McCarthy B, Garcia A, Ferketich AK. Multi-detector row cardiac computed tomography accurately quantifies right and left ventricular size and function compared with cardiac magnetic resonance. *Am Heart J*. 2006; 151(3):736–44. [PubMed: 16504643]
32. Sugeng L, Mor-Avi V, Weinert L, Niel J, Ebner C, Steringer-Mascherbauer R, et al. Quantitative assessment of left ventricular size and function: side-by-side comparison of real-time three-dimensional echocardiography and computed tomography with magnetic resonance reference. *Circulation*. 2006; 114(7):654–61. [PubMed: 16894035]
33. Wallace EL, Morgan TM, Walsh TF, Dall'Armellina E, Ntim W, Hamilton CA, et al. Dobutamine cardiac magnetic resonance results predict cardiac prognosis in women with known or suspected ischemic heart disease. *JACC Cardiovasc Imaging*. 2009; 2(3):299–307. [PubMed: 19356575]
- 34 *. Korosoglou G, Elhmidi Y, Steen H, Schellberg D, Riedle N, Ahrens J, et al. Prognostic value of high-dose dobutamine stress magnetic resonance imaging in 1,493 consecutive patients: assessment of myocardial wall motion and perfusion. *J Am Coll Cardiol*. 2010; 56(15):1225–34. [PubMed: 20883929] Prospective analysis of the use of CMR for assessment of cardiac prognosis in a large patient cohort.
35. Hundley WG, Morgan TM, Neagle CM, Hamilton CA, Rerkpattanapipat P, Link KM. Magnetic resonance imaging determination of cardiac prognosis. *Circulation*. 2002; 106(18):2328–33. [PubMed: 12403662]
36. Walsh TF, Dall'Armellina E, Chughtai H, Morgan TM, Ntim W, Link KM, et al. Adverse effect of increased left ventricular wall thickness on five year outcomes of patients with negative dobutamine stress. *J Cardiovasc Magn Reson*. 2009; 11:25. [PubMed: 19650895]
37. Kim RJ, Wu E, Rafael A, Chen EL, Parker MA, Simonetti O, et al. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med*. 2000; 343(20):1445–53. [PubMed: 11078769]
38. Steel K, Broderick R, Gandla V, Larose E, Resnic F, Jerosch-Herold M, et al. Complementary prognostic values of stress myocardial perfusion and late gadolinium enhancement imaging by cardiac magnetic resonance in patients with known or suspected coronary artery disease. *Circulation*. 2009; 120(14):1390–400. [PubMed: 19770399]
39. Mahrholdt H, Wagner A, Deluigi CC, Kispert E, Hager S, Meinhardt G, et al. Presentation, patterns of myocardial damage, and clinical course of viral myocarditis. *Circulation*. 2006; 114(15):1581–90. [PubMed: 17015795]
40. Mahrholdt H, Wagner A, Judd RM, Sechtem U, Kim RJ. Delayed enhancement cardiovascular magnetic resonance assessment of non-ischaemic cardiomyopathies. *Eur Heart J*. 2005; 26(15):1461–74. [PubMed: 15831557]
- 41 *. Thavendiranathan P, Walls M, Giri S, Verhaert D, Rajagopalan S, Moore S, et al. Improved detection of myocardial involvement in acute inflammatory cardiomyopathies using T2 mapping. *Circ Cardiovasc Imaging*. 2012; 5(1):102–10. [PubMed: 22038988] Excellent description and illustration of T2 color imaging to identify myocardial inflammation.
42. Cheong BY, Muthupillai R, Nemeth M, Lambert B, Dees D, Huber S, et al. The utility of delayed-enhancement magnetic resonance imaging for identifying nonischemic myocardial fibrosis in asymptomatic patients with biopsy-proven systemic sarcoidosis. *Sarcoidosis Vasc Diffuse Lung Dis*. 2009; 26(1):39–46. [PubMed: 19960787]
43. Patel MR, Cawley PJ, Heitner JF, Klem I, Parker MA, Jaroudi WA, et al. Detection of myocardial damage in patients with sarcoidosis. *Circulation*. 2009; 120(20):1969–77. [PubMed: 19884472]
44. White JA, Patel MR. The role of cardiovascular MRI in heart failure and the cardiomyopathies. *Cardiol Clin*. 2007; 25(1):71–95. vi. [PubMed: 17478241]
45. Lu MY, Peng SS, Chang HH, Yang YL, Chen CA, Jou ST, et al. Cardiac iron measurement and iron chelation therapy in patients with beta thalassaemia major: experience from Taiwan. *Transfus Med*. 2013; 23(2):100–7. [PubMed: 23448138]
46. Piga A, Longo F, Musallam KM, Cappellini MD, Forni GL, Quarta G, et al. Assessment and management of iron overload in beta-thalassaemia major patients during the 21st century: a real-life experience from the Italian Webthal project. *Br J Haematol*. 2013
47. Shamsian BS, Esfahani SA, Milani H, Akhlaghpour S, Mojtahedzadeh S, Karimi A, et al. Magnetic resonance imaging in the evaluation of iron overload: a comparison of MRI,

- echocardiography and serum ferritin level in patients with beta-thalassemia major. *Clin Imaging*. 2012; 36(5):483–8. [PubMed: 22920350]
48. Petersen SE, Selvanayagam JB, Wiesmann F, Robson MD, Francis JM, Anderson RH, et al. Left ventricular non-compaction: insights from cardiovascular magnetic resonance imaging. *J Am Coll Cardiol*. 2005; 46(1):101–5. [PubMed: 15992642]
 49. Alhabshan F, Smallhorn JF, Golding F, Musewe N, Freedom RM, Yoo SJ. Extent of myocardial non-compaction: comparison between MRI and echocardiographic evaluation. *Pediatr Radiol*. 2005; 35(11):1147–51. [PubMed: 16086159]
 50. Oechslin EN, Attenhofer Jost CH, Rojas JR, Kaufmann PA, Jenni R. Long-term follow-up of 34 adults with isolated left ventricular noncompaction: a distinct cardiomyopathy with poor prognosis. *J Am Coll Cardiol*. 2000; 36(2):493–500. [PubMed: 10933363]
 51. Stacey RB, Andersen MM, St. Clair SM, Hundley WG, Thohan V. Comparison of systolic and diastolic criteria for isolated left ventricular noncompaction in cardiac MRI. *JACC Cardiovasc Imaging*. 2013 (In press).
 52. van Dookum WG, Knaapen P, Hofman MB, Kuijter JP, ten Cate FJ, ten Berg JM, et al. Impact of alcohol septal ablation on left anterior descending coronary artery blood flow in hypertrophic obstructive cardiomyopathy. *Int J Cardiovasc Imaging*. 2009; 25(5):511–8. [PubMed: 19234870]
 53. Maron BJ, Lindberg J, Lesser JR. Ventricular septal crypt in hypertrophic cardiomyopathy. *Eur Heart J*. 2010; 31(15):1923. [PubMed: 20501477]
 54. Maron MS, Olivetto I, Harrigan C, Appelbaum E, Gibson CM, Lesser JR, et al. Mitral valve abnormalities identified by cardiovascular magnetic resonance represent a primary phenotypic expression of hypertrophic cardiomyopathy. *Circulation*. 2011; 124(1):40–7. [PubMed: 21670234]
 55. Defrance C, Bollache E, Kachenoura N, Perdrix L, Hrynchyshyn N, Bruguere E, et al. Evaluation of aortic valve stenosis using cardiovascular magnetic resonance: comparison of an original semiautomated analysis of phase-contrast cardiovascular magnetic resonance with Doppler echocardiography. *Circ Cardiovasc Imaging*. 2012; 5(5):604–12. [PubMed: 22798520]
 56. Tanaka K, Makaryus AN, Wolff SD. Correlation of aortic valve area obtained by the velocity-encoded phase contrast continuity method to direct planimetry using cardiovascular magnetic resonance. *J Cardiovasc Magn Reson*. 2007; 9(5):799–805. [PubMed: 17891618]
 57. Yap SC, van Geuns RJ, Meijboom FJ, Kirschbaum SW, McGhie JS, Simoons ML, et al. A simplified continuity equation approach to the quantification of stenotic bicuspid aortic valves using velocity-encoded cardiovascular magnetic resonance. *J Cardiovasc Magn Reson*. 2007; 9(6): 899–906. [PubMed: 18066750]
 58. Cawley PJ, Hamilton-Craig C, Owens DS, Krieger EV, Strugnell WE, Mitsumori L, et al. Prospective comparison of valve regurgitation quantitation by cardiac magnetic resonance imaging and transthoracic echocardiography. *Circ Cardiovasc Imaging*. 2013; 6(1):48–57. [PubMed: 23212272]
 59. Feuchtner GM, Muller S, Bonatti J, Schachner T, Velik-Salchner C, Pachinger O, et al. Sixty-four slice CT evaluation of aortic stenosis using planimetry of the aortic valve area. *AJR Am J Roentgenol*. 2007; 189(1):197–203. [PubMed: 17579171]
 60. Jilani H, Doctor N, Kashif M, Chakravarty T, Rafique A, Makar M, et al. Aortic annular sizing for transcatheter aortic valve replacement using cross-sectional 3-dimensional transesophageal echocardiography. *J Am Coll Cardiol*. 2013; 61(9):908–16. [PubMed: 23449425]
 61. Feuchtner GM, Dichtl W, Muller S, Jodocy D, Schachner T, Klauser A, et al. 64-MDCT for diagnosis of aortic regurgitation in patients referred to CT coronary angiography. *AJR Am J Roentgenol*. 2008; 191(1):W1–7. [PubMed: 18562710]
 - 62 *. Duckett SG, Chiribiri A, Ginks MR, Sinclair S, Knowles BR, Botnar R, et al. Cardiac MRI to investigate myocardial scar and coronary venous anatomy using a slow infusion of dimeglumine gadobenate in patients undergoing assessment for cardiac resynchronization therapy. *J Magn Reson Imaging*. 2011; 33(1):87–95. [PubMed: 21182125] Informative illustration regarding identification of coronary venous anatomy in preparation for multilead pacing systems.
 63. Leyva F, Foley PW, Chalil S, Ratib K, Smith RE, Prinzen F, et al. Cardiac resynchronization therapy guided by late gadolinium-enhancement cardiovascular magnetic resonance. *J Cardiovasc Magn Reson*. 2011; 13:29. [PubMed: 21668964]

64. Leyva F, Taylor RJ, Foley PW, Umar F, Mulligan LJ, Patel K, et al. Left ventricular midwall fibrosis as a predictor of mortality and morbidity after cardiac resynchronization therapy in patients with nonischemic cardiomyopathy. *J Am Coll Cardiol.* 2012; 60(17):1659–67. [PubMed: 23021326]
65. Malago R, Pezzato A, Barbiani C, Sala G, Zamboni GA, Tavella D, et al. Non invasive cardiac vein mapping: role of multislice CT coronary angiography. *Eur J Radiol.* 2012; 81(11):3262–9. [PubMed: 22480417]
66. Neizel M, Kramer N, Schutte A, Schnackenburg B, Kruger S, Kelm M, et al. Magnetic resonance imaging of the cardiac venous system and magnetic resonance-guided intubation of the coronary sinus in swine: a feasibility study. *Invest Radiol.* 2010; 45(8):502–6. [PubMed: 20531014]
67. Saremi F, Muresian H, Sanchez-Quintana D. Coronary veins: comprehensive CT-anatomic classification and review of variants and clinical implications. *Radiographics.* 2012; 32(1):E1–32. [PubMed: 22236907]

Figure 1. Dark blood T2-weighted short axis view of the left and right ventricles. On the image, the left and right ventricular cavities are shown with dark signal intensity. As noted by the arrows, there is increased signal intensity within the lateral wall of the left ventricle in a patient with confirmed regional myocarditis.

Figure 2.

End-diastolic frame from a cine white blood imaging sequence acquired within the 4 chamber plane. On these images, the cavitory blood appears white and the left ventricular myocardium appears gray. As shown by the arrows, there is noncompacted LV myocardium involving the apical segments of the left ventricle. This individual exhibited a reduced left ventricular ejection fraction due to noncompaction cardiomyopathy. This cine white blood imaging technique is useful for identifying both global (left ventricular ejection fraction and volumes) and regional left ventricular systolic function.

Figure 3. Four chamber view of the heart in a patient with arrhythmogenic right ventricular (RV) cardiomyopathy. The arrows denote RV aneurysms that represent a major criteria for establishing this diagnosis.

Figure 4.

T1-weighted first-pass gadolinium enhanced short axis view of the left and right ventricle. The left ventricular cavity appears bright, and the surrounding myocardium appears gray. As shown by the arrow, there is a region of low signal intensity in the inferior wall consistent with inducible myocardial ischemia after the administration of intravenous adenosine. This individual exhibited an 80% coronary arterial luminal narrowing in the right coronary artery on contrast coronary angiography.

Figure 5.

Characteristic patterns of late enhancement in specific cardiomyopathies. (A) Ischemic cardiomyopathy: regional thinning with 50% transmural scar in lateral wall and 100% transmural scar in inferoseptal wall. (B) Idiopathic dilated cardiomyopathy: midwall late enhancement in the basal septum. (C) HCM: patchy late enhancement within septum. (D) Myocarditis: epicardial-zone late enhancement in inferolateral and anteroseptal walls. (E) Sarcoidosis: dense epicardial-zone late enhancement. (F) Amyloidosis: diffuse late enhancement progressing from subendocardium to epicardium (pattern may also be seen in uremic cardiomyopathy and post heart transplantation). Reprinted from *Cardiology Clinics*, Volume 25(1), White JA et al., The role of cardiovascular MRI in heart failure and the cardiomyopathies, Pages 71-95, Copyright 2007, with permission from Elsevier

Figure 6. Contrast-enhanced computed tomography image of the coronary arteries of an individual with an ischemic cardiomyopathy. Higher signal intensity within the lumen of the coronary arteries appears bright. As shown by the arrow, there is reduced contrast in the vicinity of an 80% coronary arterial luminal narrowing in the left anterior descending coronary artery.

Figure 7.

3D volume rendered images from CCT to evaluate coronary veins. These high resolution images can be useful for guiding left ventricular pacing leads when considering patients with heart failure for resynchronization therapy. Reprinted from the *European Journal of Radiology*, Volume 81(11), Malago R et al., Non invasive cardiac vein mapping: role of multislice CT coronary angiography, Pages 3262-9, Copyright 2012, with permission from Elsevier